Sudan University of Sciences & Technology

College of graduate studies

Characterization of Heart Wall Changes in Diabetes Mellitus Patients type II Using Echocardiography

توصيف تغييرات جدار القلب لمرضى السكري النوع الثاني باستخدام الموجات الفوق قلب

A Thesis Submitted for the Fulfillment of Ph.D. Degree in Medical Diagnostic Ultrasound

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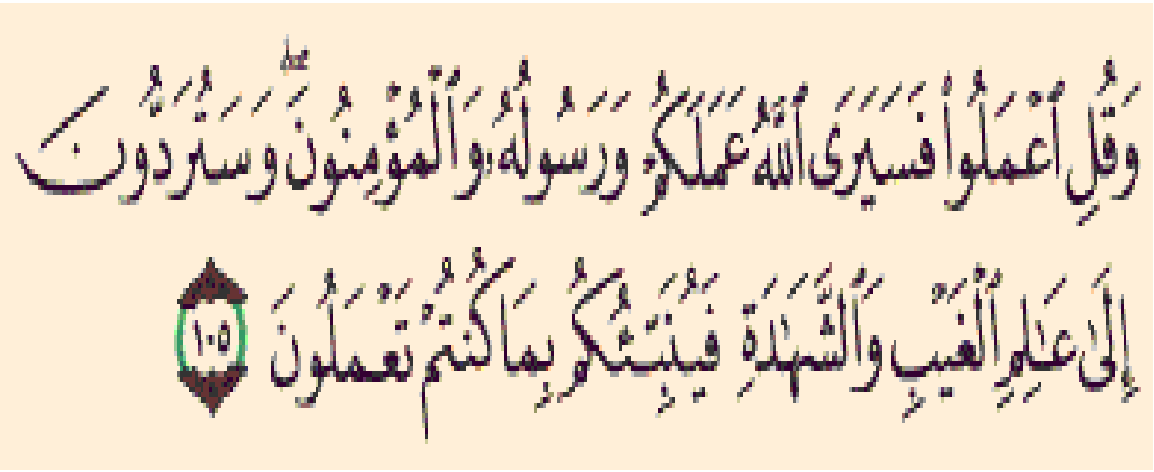
الآية الكريمة



الآية 105 التوبة

صدق الله العظيم

Dedication

To the Soul of my father Abd ALLAH

Elhaj

To my mother Zinab Mohammed

Ahmed

To my wife Nousiba Yousef,

To my brothers

To my Sisters

To my children

To my friends and

To my colleagues

With love and

respect

Acknowledgement

First, my thanks to ALLAH who made me courageous and gave me strength to conduct this study as I planned and wish.

I would like to give my special thanks to my supervisor **Dr. Elsafi Abdullah**. I'm greatly indebted to his valuable guidance.

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Special gratefulness to the administration and the staff of El Ribeat national teaching hospital; echocardiography department.
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Abstract

Diabetes mellitus is a group of diseases characterized by elevated blood glucose concentration. It may be a consequence of either the body does not produce enough insulin or because cells do not respond to the insulin that is produced. Diabetes is major cause to cardiovascular disease which lead to morbidity and mortality. The objective of this study was to Characterization of Heart wall changes in patients with type II Diabetes Mellitus using Echocardiography, in this study a total of 243 individual divided into two groups (130 control group we named Non-diabetes and 113 diabetes patients. Age ranged from 30 to 85 years with mean 55.8 years ±SD 13.6. The study corned only in echocardiography department in El Rebat educational Hospital in Khartoum state, in Sudan. The biographic data collected include (age, gender, job

status, past medical history and duration of diabetes 1to 20 years with mean 9.2 years ±5) and the other main echo parameters and findings, All were collected in data sheet collection and the data was analyzed using IBM SPSS(Standard Statistical Package for the Social Sciences 22). The result of this study they revealed the Compare Means of Echocardiography parameters between Non-diabetes and diabetes patients Type II as body mass index the non-diabetes mean was 23.2 Kg/m2 ±4.7 and diabetes mean was 25.4Kg/m2 ± 5.4 with p-value .001, Interventricular septal thickness the non-diabetes mean 11.06 mm ± 1.8 diabetes mean 12.08 mm ± 2 with p-value .000 and Left ventricular posterior wall Diameter non-diabetes mean 9.61 mm ±1.56 diabetes mean 10.39 mm ± 1.4 with p-value .000. On the other hand the studies manifested the Compare Means of Echocardiography parameters Diabetes patients Type II between male and female as Body mass index the male diabetes mean was 23.1Kg/m2 ± 2.9 and female diabetes mean was 27.0Kg/m2 ± 6.2 with p-value 0.000101, Aortic root diameter the male diabetes mean was 31.09 mm ±3.4 and female diabetes mean was 28.15 mm ±3.6 the p-value 0.000026, left atrial diameter the male diabetes mean was 36.87 mm ±4.2 and female diabetes mean was 34.47 mm ± 3.7 the p-value 0.001856, Left ventricular diastolic diameter the male diabetes mean was 46.87 mm ± 5.2 and female diabetes mean was 44.47 mm ± 5.5 p-value 0.021and Left Ventricle systolic diameter the male diabetes mean was 30.64 mm ± 5.3 and female diabetes mean was 28.6 mm ± 5 p-value 0.046. Lastly they showed the correlation of Echocardiography finding parameters in diabetic patients Type II, between Inter ventricular septal diameter and Left ventricular posterior wall Diameter Pearson Correlation 0.739 with p-value 0.000, between Inter ventricular septal diameter and Aortic root diameter Pearson Correlation .360 p-value 0.000, between Inter ventricular septal diameter and Left ventricular diastolic diameter Pearson Correlation - 0.224 p-value and 0.017, between Inter ventricular septal diameter and Ejection fracture Pearson Correlation .186 and p-value 0.048, Left ventricular diastolic diameter and Ejection fracture Pearson Correlation - 0.326 and p-value 0.000, between Left Ventricle systolic diameter and Ejection fracture Pearson Correlation -.644 and p-

value 0.000. The study confirm that echocardiography is a valuable tool for evaluation of patients with Diabetes Mullets Type II. This study confirmed that; age, gender, body mass index, and echocardiography parameters show significant association with Heart failure risk worldwide (p 0.001- 0.05).

المستخلص

داء السكري يعرف بانه مجموعة من الأمراض التي توصف بارتفاع تركيز السكر في الدم قد يكون ذلك لما الجسم لا ينتج ما يكفي من الأنسولين أو ان الخلايا لا تستجيب للأنسولين الذي يتم

انتاجه. مرض. السكري. هو السبب. الرئيسي لأمراض. القلب. والأوعية. الدموية. التي تؤدي إلى. الإصابة بالأمراض. والوفيات. والهدف. من. هذه الدراسة. هو وصيف. تغييرات. جدار. القلب. في. المرضى الذين. يعانون. من. السكري. من. النوع 2 السكري. باستخدام. الموجات. فوق. الصوتية. للقلب. في. هذه الدراسة. تم. دراسة. مجموع. 243 فرد. تنقسم. إلى. مجموعتين (130. المجموعة. السليمة. تسمي. غير. مرض. السكري. و 113 مرضى السكري. تراوحت. الاعمار. من. ≥ 30 حتى 85 عاما. بمتوسط. 55.76 عاما. الانحراف. المعياري. ± 13.632). وقد. تمت. الدراسة. في. آلة. تخطيط. صدى. القلب. في. مستشفى. الرباط. التعليمية. بولاية الخرطوم. في. السودان. وقد. اوضحت. المعلومات. الاساسية. وهي. كالاتي. (العمر. الجنس. حالة. المرضية. والشكوي. الطبية. والتاريخ المرضي ومدة. مرض. السكري. 1 الي. 20 عاما. بمتوسط. زمني 9.2 سنوات. 5). وغيرها. من. القراءات. الرئيسية. وكل. النتائج، قد. جمعت. في. ورقة. البيانات. وتم. تحليلها. بواسطة. برنامج IBM الاحصائيات. SPSS النسخة. 22. في. الدراسة. تم. توزيع الفئات. العمرية. وجد. أن. الفئة. العمرية. الاكثر. تاثرا. بمرض. السكري. هي. من. 50-60 سنة. وقد. كان. عدد المرضي 36 مريض سكري. 28 إناث. و 8 ذكور. وكشفت. مقارنة. متوسطات. قراءات. اضربات. القلب. بين المجوعتين الاولي. السليمة. والثانية مرضى السكري. من. النوع II، أول. مؤشر كتلة. الجسم. بين المجوعتين المجوعة. السليمة كان 23.2 كجم / م ^{2} 4.7 وكانت مجموعة مرضي السكري 25.4 كجم / م ^{2} ± 5.5 القيمة الاحتمالية 0.001، سماكة الحاجز بين البطينين فى السليمة يساوي 11.06 ملم ± 1.83السكري يساوي 12.08 ملم ± 2 و 0.000 ومقدار سماكة الجدار الخلفي البطين. للسليمين يساوي و 9.61 ملم ± 1.6 وفى مرضي. السكري قياسه 10.39 ملم ± 1.4و 0.000. من ناحية أخرى تتجلى في الدراسة مقارنة متوسطات قراءات مقايس الموجات الصوتية لضربات القلب لمرضى. داء السكري. من. النواعين النكور. والإناث. وكانت. النتيجة. مؤشر كتلة. الجسم. في. المتوسط. مرضي السكري. النكور. كان. 23.1 كجم/م. 2 ± 3 عند مرضي السكري. الإناث. كان. 27.0 كجم/م. 2 ± 6.2 و 0.001، وكانت. قطر. جنرالأبهر. كان. المتوسط. لدي. مرضي السكري. النكور. 31.09 ملم. وانحراف. معياري. ± 3.4 و لدي. مرضي السكري. الإناث. كان. 28.15 ملم. وانحراف. معياري. ± 3.6 و 0.000026، مقياس. قطر. الأذين. الايسر. لدي. مرضي النكور. كان. 36.87 ملم. ± 4.2 ومرضي الإناث. 34.47 ملم. ± 3.7 و 0.001856، كان. قطر. البطين الأيسر الانبساطي. لمرضي النكور. متوسطه. 46.87 ملم. ± 5.2 ومرضي الإناث. كان. 44.47 ملم. ± 5.6 و 0.021567، و قطر. البطين الأيسر الانقباضي كان. متوسط. مرضي النكور. كان. 30.64 ملم. ± 5.3 و متوسطه. الإناث. السكري. 28.64 ملم. ± 5.1 و 0.045766. أظهرت. الدراسة. لارتباط. متوسطات. قراءات. مقاييس. الموجات. الصوتية. اضربات. القلب. لمرضى داء السكري. من. النوع الثاني. وكانت. على. النحو. الاتي. : بين قطر. حاجز. البطين. الداخلي. و قطر. جدار. البطين الأيسر. الخلفي. الارتباط. الناتي. 0.739 مع القيمة. الاحتمالية. 0.000، بين قطر. حاجز. البطين الداخلي. وقطر. جنر. الأبهر. الارتباط. الناتي. 0.360 القيمة. الاحتمالية. 0.000، بين قطر. حاجز. البطين الداخلي. وقطرللبطين الأيسر. الانبساطي. الارتباط. الناتي. -0.224 القيمة. الاحتمالية. 0.017، بين قطر. حاجز. البطين الداخلي. وكسر طرد. الارتباط. الناتي. 0.186 والقيمة. الاحتمالية. 0.048،

قطر البطين الأيسر للانبساطي وكسر طرد الارتباط الناتي -0.326 والقيمة الاحتمالية 0.000، بين قطر البطين الأيسر الانقباضي وكسر طرد الارتباط الناتي -.644 والقيمة الاحتمالية 0.000، واكدت الدراسة أن تخطيط صدى القلب بالموجات الفوق الصوتية هو أداة ذات قيمة لتشخيص المرضى الذين يعانون من مرض السكري النوع الثاني وأثبتت هذه الدراسة بأن؛ العمر والجنس ومؤشر كتلة الجسم ومقاييس الموجات الفوق الصوتية للقلب تظهر لرتباط كبير مع مخاطر فشل وقصور عضلة القلب في جميع أنحاء العالم القيمة الاحتمالية الموجبة او السالبة (0.001 - 0.05)

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List of Abbreviations

Phrase	Abbreviation
Two-dimensional	2 D
Acute coronary syndrome	ACS
Acute myocardial infarction	AMI
Atrioventricular	AV
Body Mass Index	BMI
Body surface area	BSA
Coronary artery disease	CAD
Color-flow mapping	CFM
Congestive Heart Failure	CHF
Chronic obstructive pulmonary disease	COPD
Computed tomography	CT
Cardiovascular disease	CVD
Continuous-wave	CW
Diabetes Mellitus	DM
Diabetes ketoacidosis	DKA
Early acute pericarditis	EAP
Electrocardiogram	ECG

Ethylenediaminetetraacetic acid	EDTA
High-density lipoprotein	HDL
Insulin-dependent diabetes mellitus	IDDM
Ischemic Heart Disease	IHD
Inferior vena cava	IVC
Interventricular septum	IVS
Left atrium	LA
Low-density lipoprotein	LDL
low and middle income countries	LMIC
Left ventricle	LV
Left ventricular ejection fraction	LVEF
Left Ventricular Outflow Tract	LVOT
Metropolitan area	MA
Megahertz	MHz
Myocardial Infarction	MI
Motion mode	M-mode
Magnetic resonance imaging	MRI
Mitral Valve	MV
Non-insulin dependent diabetes mellitus	NIDDM
Pulmonary artery	PA
peripheral arterial disease	PAD
Percutaneous coronary intervention.	PCI
Pericardial effusion	PE
Parasternal long –axis	PLA
Point of maximal impulse	PMI
Posterior Wall	PW
Pulsed-wave	PW
Right atrium	RA
Right ventricle	RV
Sinoatrial	SA
Standard Statistical Package for the Social Sciences	SPSS
Superior vena cava	SVC
Transient Ischemic Attack	TIAs
Tricuspid Valve	TV
Ventricular septal defect	VSD

CHAPTER ONE

Introduction

Chapter One

:Introduction .1.1

Diabetes is a group of diseases characterized by elevated blood glucose concentration. It may be a consequence of either the body does not produce enough insulin or because cells do not respond to the insulin that is produced, it can be classified into three major classes: Type I, Type II and Gestational diabetes mellitus) (Jameson, 2006). Type I diabetes known as insulin-dependent diabetes (Astorri et al., 1997), childhood diabetes or also known as juvenile diabetes, is characterized by loss of the insulin producing beta cells of the islets of Langerhans of the pancreas leading to a severe deficiency of insulin. (Gardner and Greenspan, 2007) (Type II diabetes mellitus known as adult-onset diabetes or non-insulin dependent diabetes mellitus (NIDDM) is characterized by insulin resistance which may be combined with relatively reduced insulin secretion .The defective responsiveness of body tissues to insulin is believed to involve the insulin receptor (Benedict , 2004).

Gestational diabetes mellitus resembles type II diabetes in several aspects, involving a combination of inadequate insulin secretion and responsiveness. It occurs in about 2% to 5% of all pregnancies and may improve or disappear after delivery (Lawrence et al., 2008).

Due to Diabetes Mellitus type II that affect many organs on human, the most important ones is the heart, the heart is vital organ in the boy whenever any disease affect the heart is the threaten life (Lawrence et al., 2008).

We studied the characterization of heart wall disease which is generally affected by Diabetes Mellitus well improve the management of patients with type II DM and to dedicate their aerially heart problem and shorten the circle of diagnosis and safe patients life (Awad, 2006).

While Diabetes, is the most common non- communicable disease in the country, is having an increasing impact on rates of morbidity and mortality in Sudan.(Awad, 2006, Rahamtalla et al., 2012)

The spread of sedentary lifestyles and adoption of western dietary habits – high in refined carbohydrates and fat – are driving an increase in the number of people with obesity-related type II diabetes (Awad, 2006).

Knowledge of the diabetes epidemic in Sudan is limited. The most recent data come from a small scale study that was carried out in 1996. The results of the study indicated a prevalence of 3.4% (Elbagir et al., 1996) .

But recent estimates place the diabetes population at around one million – around 95% of whom have type II diabetes. Patients with diabetes make up around 10% of all hospital admissions in Sudan (Awad, 2006).

The principal cause for admission is diabetes ketoacidosis – inadequate insulin resulting in high blood glucose levels and accumulation of organic acids and ketones in the blood. The lack of access to affordable insulin and other diabetes supplies in Sudan exacerbates the severe shortcomings in diabetes care. Diabetes provokes more deaths each year than any other non-communicable disease 10% of hospital mortality. (Rahamtalla et al., 2012).

Ketoacidosis is the principal killer; other causes of death include diabetic foot related septicemia and end stage renal disease and ischemic heart disease. (Rahamtalla et al., 2012)

Diabetes mellitus (DM) is closely associated with cardiovascular (CV) diseases. These are the main cause of death in patients not only with type II but also type I diabetes. Apart from the traditional risk factors such as arterial hypertension, dyslipidemia and obesity, hyperglycemia is an independent risk factor for the development of ischemic heart disease (IHD). A number of experimental, pathologic, and epidemiologic studies support the existence of diabetic cardiomyopathy, the clinical diagnosis of which is made when systolic and diastolic left ventricular dysfunction

are present in diabetic patients without other known cardiac disease (Awad, 2006).

Diabetic cardiomyopathy may be an important contributor to the susceptibility of diabetic subjects to the development of heart failure and to their worse outcome with this condition (Francis, 2001).

The problem of study .1.2

Heart attack is become the main cause of sudden death among patients with type II diabetic, in many cases of diabetics due to ischemic heart disease. The echocardiography will be the best examination for checking a heart condition for alarming the diabetic patient and be care for their full up. Due to Diabetes Mellitus type II that affect many organs on human, mainly the important one is the heart, heart is vital organ in the .body whenever been affect it threaten life

Objectives .1.3

General Objective .1.3.1

To characterized the heart wall changes in diabetes mellitus patient's type II using echocardiography.

Specific Objectives .1.3.2

- To find the correlation between the left ventricle hypertrophy and (Aortic root diameter, left atrial diameter, Left ventricular diastolic diameter, Left Ventricle, systolic diameter, Interventricular septal thickness, Left ventricular posterior wall Diameter and Ejection .(fracture
- To find the correlation between the left ventricular diastolic .diameter and Ejection fraction
- To find the correlation between the body mass index age, gender, duration of DM and left ventricular diastolic diameter among the .diabetes patients and non-diabetes

:Significance of the study .1.4

This study will have a significant importance for the diabetic’s patients, because due to sudden death form the heart problem it is the first study in this field according my knowledge. Its results may contribute in health promotion program in Sudan.

This study will improve the quality of the patient’s life and get them back to living without complication of heart disease due to diabetic.

Overview of the study .1.5

This study was consisted of five chapters, with chapter one is an introduction which is include overview of the case under study, problem of the study, objective and significant of the study. While chapter two contain scholar literature about the problem of the study as well chapter three include the material used to collect the data and method of data collection. Chapter four include result presentation using tables and figure and finally chapter five will present discussion of the presented result as well as conclusion and recommendation.

CHAPTER TWO
Literature Review

Chapter Two

Litterature Review .2

The global burden of diabetes mellitus .2.1

The most recent International Diabetes Federation (IDF) estimates from 2013 are that 8.3% of adults that is, 382 million people worldwide have diabetes. This number has doubled over the past 20 years, and notably 80% of people with diabetes live in low and middle income countries (LMIC) (Guariguata et al., 2014). Diabetes already contributes significantly to morbidity and mortality in Africa. The highest global age-specific mortality rate is recorded in this continent (Alli, 2013), (Steyn, 2006), (Guariguata, 2011), (Werfalli, 2014) and (Naidoo, 2010).

All countries in Africa fall into the LMIC category, and predominantly the low income category. The rise in the number of individuals with type 2 diabetes in Africa, similar to LMIC has been attributed to ageing of the population and relatively rapidly changing environmental factors (Guariguata et al., 2014). These include urbanization, the adoption of health behaviors favoring sedentariness and unhealthy eating patterns. While unhealthy behavior patterns and obesity are potentially modifiable, ageing, one of the major drivers for diabetes, is not (Sobngwi et al., 2001).

In 2013, the majority of individuals with diabetes in Africa were reported to be under 60 years of age with the highest proportion (43.2%) in people aged 40–59 years (Sobngwi et al., 2001) The relatively small proportion of individuals aged 60–79 years in the region is likely to account for the estimate that only 18.8% of patients with diabetes fall in this age group (Guariguata et al., 2014).

Diabetes Mellitus .2.2

Diabetes is a group of diseases characterized by elevated blood glucose concentration .It may be a consequence of either the body does not produce enough insulin or because cells do not respond to the insulin that

is produced, it can be classified into three major classes: Type, I Type II and Gestational diabetes mellitus (Nunoda et al., 1985). Type I diabetes known as insulin-dependent diabetes mellitus (IDDM), childhood diabetes or also known as juvenile diabetes, is characterized by loss of the insulin producing beta cells of the islets of Langerhans of the pancreas leading to a severe deficiency of insulin.(Jameson, 2006) Type II diabetes mellitus known as adult-onset diabetes or non-insulin dependent diabetes mellitus(NIDDM)is characterized by insulin resistance which may be combined with relatively reduced insulin secretion .The defective responsiveness of body tissues to insulin is believed to involve the insulin receptor (Gardner and Greenspan, 2007).

Gestational diabetes mellitus resembles type II diabetes in several aspects, involving a combination of inadequate insulin secretion and responsiveness. It occurs in about 2% to 5% of all pregnancies and may improve or disappear after delivery (Benedict C., 2004).

Chronic disorder of glucose metabolism with hyperglycemia, triggered by conditions associated with a relative or absolute insulin deficiency. Patients have diabetes mellitus means the body cannot control the level of glucose in their blood. There are two type of DM, type I and type II; in type 1 diabetes mellitus, the lack of control of glucose is due to absence of insulin production, and in type II diabetes mellitus, it is due to tissue insulin resistance (Benedict C., 2004).

Diabetes mellitus Type I .2.2.1

The Epidemiology of type I diabetes mellitus: Approximately 10% of cases; usually occurs in younger patients, but can occur at any age. The Pathogenesis: β-cell destruction, which is a possible autoimmune process with T lymphocytes reactive against islet cells. Some cases of type I diabetes mellitus may be caused by a viral infection (Kemp. et al., 2008).

Diabetes ketoacidosis (DKA): can be diagnosis by the following: Hyperglycemia (250 mg/dL), ketosis and metabolic acidosis with anion gap. The Precipitating factors; as Infection, new-onset diabetes mellitus, stress, and insulin deficiency (insulin noncompliance). The Pathogenesis

of DKA; DKA is characterized by a combination of insulin deficiency and relative glucagon excess. Physiologic stress such as infection contributes to the development of DKA by increasing catecholamine release, which in turn increases the physiologic insulin requirement. Decreased peripheral glucose uptake and utilization caused by insulin deficiency, coupled with excess glucagon, results in hyperglycemia. Insulin-sensitive lipase is activated, resulting in hepatic mobilization of free fatty acids and conversion to ketone bodies for insulin-independent use. The β-hydroxybutyrate produced by ketosis, combined with lactic acidosis, produces an anion gap acidosis. Osmotic diuresis from hyperglycemia causes severe volume depletion and often leads to electrolyte disturbances. Insulin deficiency, and to a lesser extent acidosis, causes a shift of potassium from the intracellular compartment to the serum. Thus, serum potassium is usually normal or elevated despite heavy urinary losses and depletion of total body potassium stores. Rapid insulin infusion can result in profound hypokalemia; the Clinical presentation of DKA; the Symptoms: Nausea, vomiting, thirst, abdominal pain, weakness, and fatigue and its signs: Tachycardia; poor skin turgor and warm and dry skin (due to dehydration). Patients also have ketones on breath and altered mental status. The Laboratory findings: Hyperglycemia, anion gap metabolic acidosis, and serum ketones (Kemp. et al., 2008).

.2.2.2 :Diabetes mellitus Type II

The Epidemiology of type II diabetes mellitus represents 80–90% of cases of diabetes mellitus. It usually occurs in older patients (> 40 years) and obese individuals, but can occur in children as young as 6 years of age. Risk factors for its development include sedentary lifestyle, poor nutrition, and overweight and obesity (Kemp. et al., 2008).

Pathogenesis of type II diabetes mellitus: Genetic factors play a more important role in type II diabetes mellitus than in type I diabetes mellitus (e.g., 50–90% concordance rate for type II diabetes mellitus among identical twins). Type II diabetes mellitus is due to inadequate secretion of insulin and peripheral resistance to insulin. The Clinical presentation**;**

Weakness, weight loss, and susceptibility to infections the important point: It is commonly taught that type I diabetics develop DKA and type II diabetics develop hyperosmolar nonketotic coma. In practice, DKA is very common in both type I and type II diabetics, whereas hyperosmotic coma is unusual. The signs of the Hyperosmolar nonketotic coma as marked hyperglycemia; no metabolic acidosis or ketonemia, more dehydration than DKA and low free fatty acid levels; no ketone bodies and serum hyperosmolality (>325 mOsm/L) (Kemp. et al., 2008).

Diagnostic criteria for diabetes mellitus .2.2.2.1

The single random glucose level > 200 mg/dL in the presence of appropriate symptoms is sufficient for determining the diagnosis of diabetes mellitus, two random glucose tolerance tests with a level >200 mg/dL in the absence of symptoms, two 2-hour (75 g glucose) glucose tolerance tests with a level >200 mg/dL and a fasting glucose level > 126 mg/dL and lastly an important point the Hemoglobin A1C is a determination of the amount of glycosylated hemoglobin and is used for monitoring the disease process; it is not used for diagnostic purposes (Kemp. et al., 2008).

Pathogenesis and complication of diabetic complications .2.2.2.2

The pathogenesis as: Activation of polo pathway with accumulation of sorbitol, Production of advanced glycosylation end products and from nonenzymatic glycosylation of proteins, increased oxidative damage and platelet dysfunction associated with increased aggregation (Kemp. et al., 2008).

On the other hand the complications of diabetes mellitus as: Important point: Diabetes mellitus is the number one cause of end-stage renal disease, blindness, and nontraumatic lower extremity amputations, pancreas: Reduction in number and size of islets (type I diabetes mellitus); amyloid deposition (type 2 diabetes mellitus) and Vessels: Diabetes mellitus is a contributor to atherosclerosis in large vessels (i.e., macrovascularzsaas damage), in small vessels, diabetes mellitus produces hyaline arteriolosclerosis (microvascular damage), which has a

similar appearance to that seen in hypertension. Macrovascular damage (atherosclerosis) leads to infarcts (e.g., heart, brain). Diabetics can also develop hypertension due to hyperglycemia- induced endothelial dysfunction (Kemp. et al., 2008).

The Kidney: Microalbuminuria (30–300 mg/24 hours), which is associated with 10 to 20 times the increased risk of progression to diabetic nephropathy. Diabetic nephropathy includes diffuse glomerulosclerosis and nodular glomerulosclerosis (Kimmelstiel-Wilson lesion); diabetics are also at risk for pyelonephritis with risk of development of papillary necrosis. The Eye: nonproliferative and proliferative retinopathy and cataracts (Kemp. et al., 2008).

- Nonproliferative retinopathy is due to increased capillary permeability, dilation of venules, and presence of microaneurysms.

- Proliferative retinopathy is due to retinal ischemia and hypoxia-induced neovascularization. The new vessels are fragile, and hemorrhage can cause sudden loss of vision.

The peripheral nervous system: Peripheral neuropathies (sensory loss more than motor loss); decreased sensation causes diabetics to be more prone to injury.

The skin and soft tissue of extremities: Diabetics often develop ulcers and gangrene of the legs, requiring amputation this leads to decreased sensation causes diabetics to be prone to injury (Kemp. et al., 2008).

These patients are unable to feel the damage occurring and associated damage to vessels leads to poor perfusion that impairs healing lastly to pregnancy: Large-for-gestational age infants are often born to diabetic mothers (Kemp. et al., 2008).

Anatomy and physiology of the heart .2.3

Overview of the Heart .2.3.1

The heart is a muscular organ that acts like a pump to continuously send blood throughout your body. The heart is at the center of the circulatory system. This system consists of a network of blood vessels, such as arteries, veins, and capillaries. These blood vessels carry blood to and from all areas of the body.

An electrical system regulates the heart and uses electrical signals to contract the heart's walls. When the walls contract, blood is pumped into the circulatory system. A system of inlet and outlet valves in the heart chambers work to ensure that blood flows in the right direction. The heart is vital to your health and nearly everything that goes on in the body. Without the heart's pumping action, blood can't circulate within the body. Blood carries the oxygen and nutrients that your organs need to work normally. Blood also carries carbon dioxide, a waste product, to your lungs to be passed out of the body and into the air. A healthy heart supplies the areas of the body with the right amount of blood at the rate needed to work normally. If disease or injury weakens the heart, the body's organs won't receive enough blood to work normally (Tortora and Derrickson, 2012).

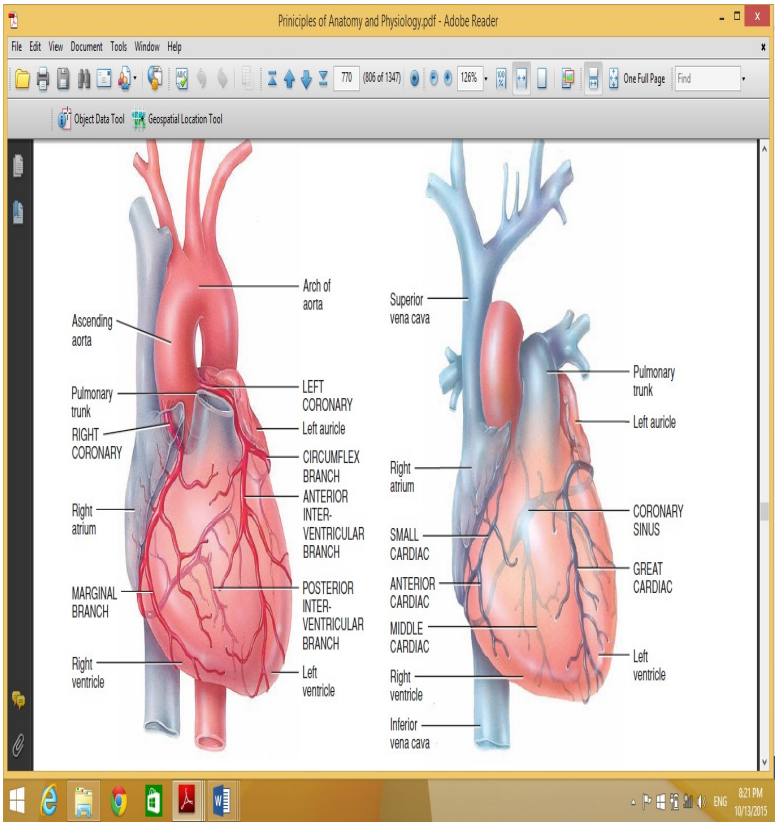


Figure 2.1 Structure of the heart: surface features.

(Tortora and Derrickson, 2012)

Location, Size and Shape of the Heart .2.3.2

The heart is located underneath the sternum in a thoracic compartment called the mediastinum, which occupies the space between the lungs. It is approximately the size of a man's fist (250-350grams) and is shaped like an inverted cone. The narrow end of the heart is called the apex. It is directed downward and to the left and lie just above the arch of the diaphragm at the approximate level of the fifth or sixth rib. The broad end of the heart is called the base and gives rise to the major blood vessels, which is directed upwards and to the right and lies at the approximate level of the second rib (Tortora and Derrickson, 2012).

Surrounding the hearts is a fibrous sac called the pericardium, which performs several functions. Fluid within the sac lubricates the outer wall of the heart so it can beat without causing friction. It also holds the heart in place forms a barrier against infections and helps keep the heart from over expanding (Tortora and Derrickson, 2012).

The pericardium is made up of a coronal section which comprises of two walls and a thin intervening space. The outer wall is thickest and consists of two tissue layers. The external layer is formed by a dense irregular connective tissue and is often called the fibrous pericardium. This layer protects the heart and anchors it to nearby organs (Tortora and Derrickson, 2012).

At the roots of the major blood vessels, the parietal pericardium reflects back over the surface of the heart to form the inner wall of the pericardium, the visceral pericardium. Because it is the outer layer of the heart wall, the visceral pericardium is referred to as the epicardium. Together, the parietal and visceral pericardial layers are also called the serous pericardium. Between the walls of the serous pericardium is the pericardial cavity. This narrow space is normally filled with a few (10-15) milliliters of pericardial fluid, which is secreted by the serous membranes. The fluid reduces friction between membranes as they glide past one another during heartbeats. The heart wall is composed of three tissue layers. Covering the outer surface of the heart is the epicardium. It is also referred to as the visceral pericardium, which is the inner layer of the pericardium. The epicardium is a serous membrane that consists of an external layer of simple squamous and an inner layer of areolar tissue (loose connective tissue).The squamous cells secrete lubricating fluids into the pericardial cavity (Tortora and Derrickson, 2012).

The thick middle layer of the heart wall is called the myocardium. It consists of numerous layers of cardiac muscle fibers that wrap around the heart wall. Contraction of the myocardium pumps blood out of the heart into the aorta and pulmonary trunk arteries. Covering the outer surface of the heart wall is the endocardium. This layer also covers the heart valves and tendons and is continuous with the endothelium that lines the major blood vessels that attach to the heart. The endocardium is made up of thin layer of simple squamous cells and areolar tissue, similar to the epicardium. Secretions from the squamous cells help regulate the activity of the myocardium (Tortora and Derrickson, 2012).

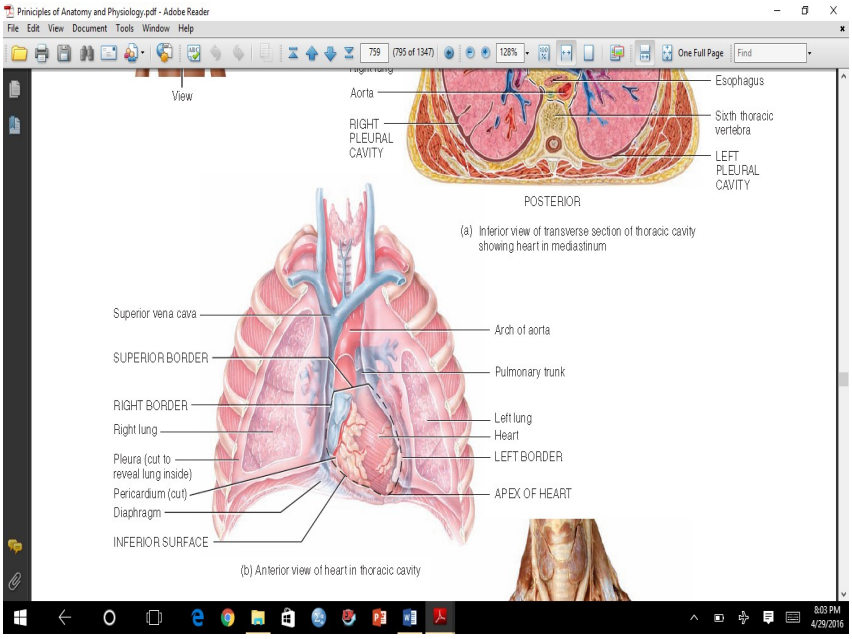


Figure 2.2 Location, Size and Shape of the Heart

([Tortora and Derrickson, 2012])

The Chambers of the Hearts .2.3.3

The heart has four chambers. The two superior receiving chambers are the atria, and the two inferior pumping chambers are the ventricles. The paired atria receive blood from blood vessels returning blood to the heart, called veins, while the ventricles eject the blood from the heart into blood vessels called arteries. On the anterior surface of each atrium is a wrinkled pouch like structure called an auricle, so named because of its resemblance to a dog's ear. Each auricle slightly increases the capacity of an atrium so that it can hold a greater volume of blood. Also on the surface of the heart are a series of grooves, called sulci that contain coronary blood vessels and a variable amount of fat. Each sulcus marks the external boundary between two chambers of the heart. The deep coronary sulcus encircles most of the heart and marks the external boundary between the superior atria and inferior ventricles. The anterior interventricular sulcus, is a shallow groove on the anterior surface of the heart that marks the external boundary between the right and left ventricles. This sulcus continues

around to the posterior surface of the heart as the posterior interventricular sulcus, which marks the external boundary between the ventricles on the posterior aspect of the heart (Tortora and Derrickson, 2012).

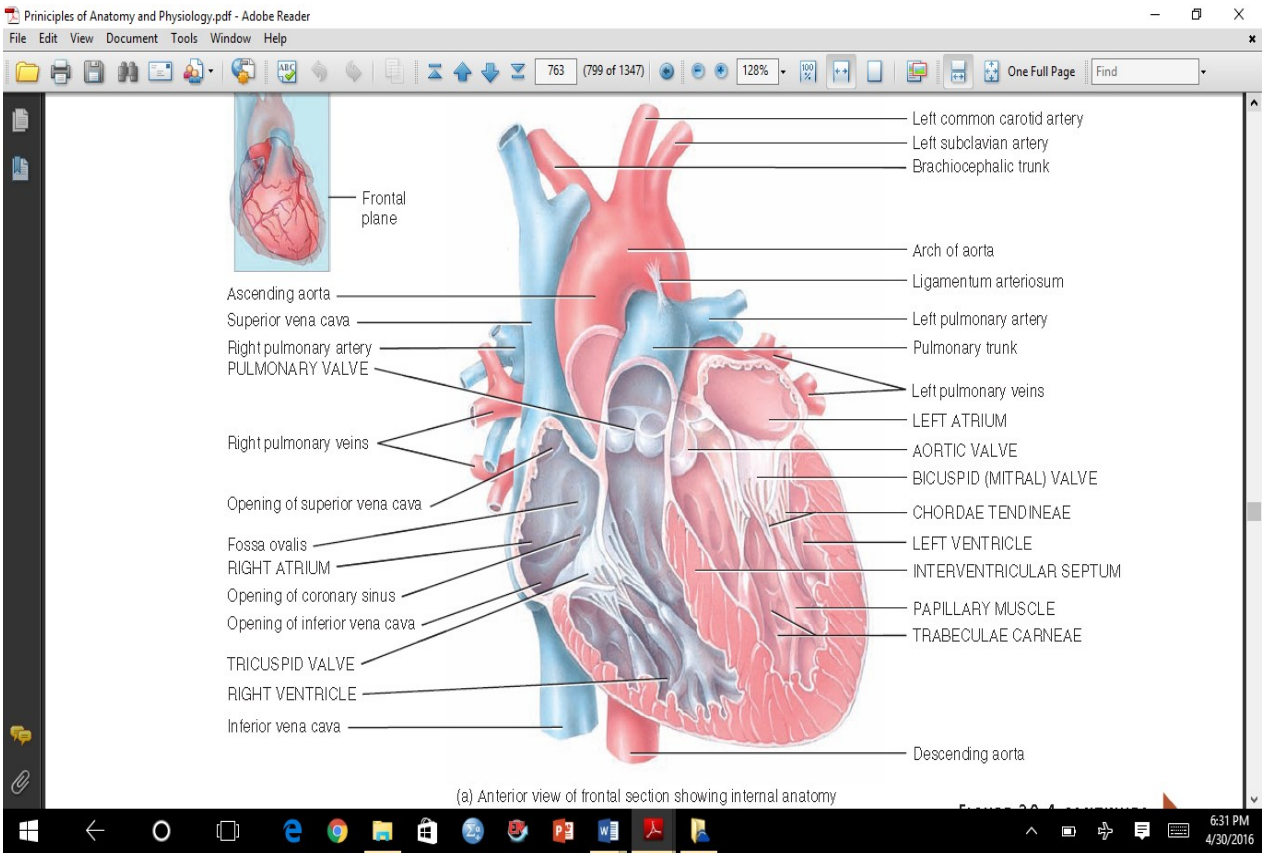


Figure 2.3 Anterior view of frontal section showing internal anatomy (Tortora and Derrickson, 2012)

The Circulation System .2.3.4

The major vessels of the heart are the large arteries and veins that attach to the atria, ventricles and transport blood to and from the systemic circulatory system and pulmonary circulation system. Blood is delivered to the right atrium from the systemic circulatory system by two veins. The superior vena cava transport oxygen-depleted blood from the upper extremities, heard and neck. The inferior vena cava transport oxygen-depleted blood from the thorax, abdomen and lower extremities (Consultants, 2006).

Blood exits the right ventricles through the pulmonary trunk artery. Approximately two inches superior to the base of the heart, this vessel branches into the left and right pulmonary arteries, which transport blood into the lungs. The left pulmonary veins and right pulmonary veins return oxygenated blood from the lungs to the left atrium (Consultants, 2006).

Blood passes from the left atrium into the left ventricle and then is pumped into the systemic circulatory system through a large elastic artery called the aorta (Consultants, 2006).

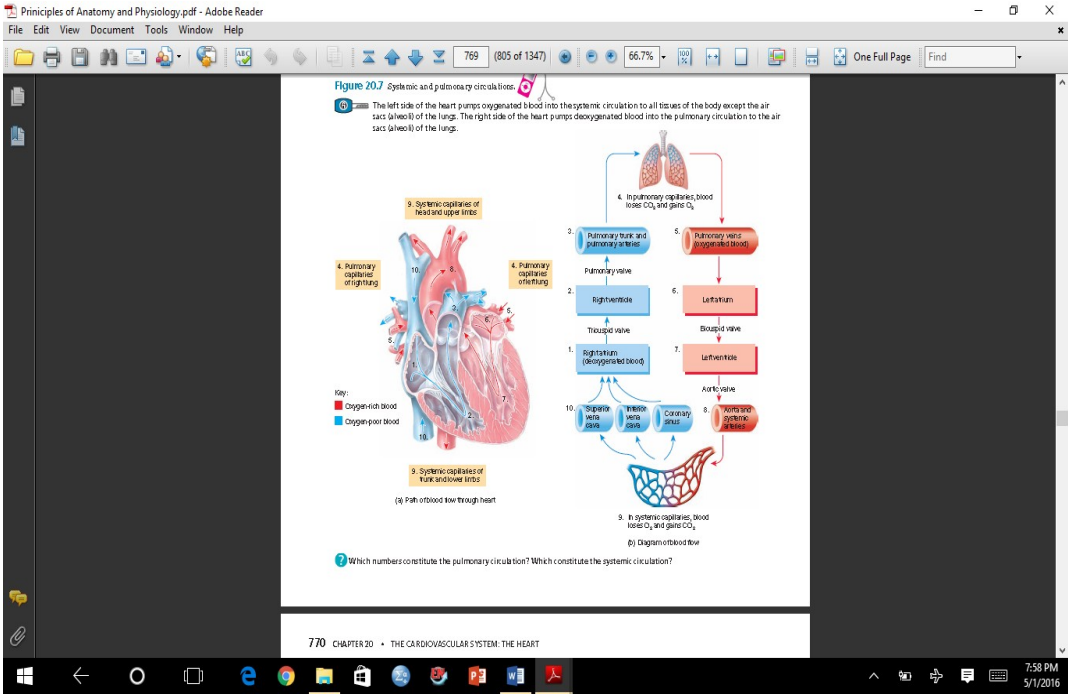


Figure 2.4 Systemic and pulmonary circulations (Tortora and Derrickson, 2012)

The Heart Valve Anatomy .2.3.5

Four valves maintain the unidirectional flow of blood through the heart. The valves are located between each atrium and ventricle and in the two arteries that empty blood from the ventricle. These valves are primarily composed of fibrous connective tissues that originate and extend from the heart walls. The external surfaces of the valves are covered by endocardium (Consultants, 2006).

The Tricuspid valve (right atroventricular) is composed of three caps or flaps and controls blood flow from the right atrium to the right ventricle.

The bicuspid valve is made up of two cusps or flaps and controls blood flow from the left atrium to the left ventricle. The term mitral valve is also commonly applied because the left AV valve is shaped somewhat like a bishop's miter (Cleveland Clinic 2009, 2011).

Thin tendon like cord called chordae tendineae connect the AV valves to cone shaped papillary muscles that extend upward from the myocardium. The chordae tendineae and papillary muscles tether the AV valves to the ventricular walls. This allows the valves to close properly and not bulge (or prolapse) into the atria (Cleveland Clinic 2009, 2011).

Semilunar valves direct blood flow from the ventricles into the aorta and pulmonary trunk artery. The valves are located in the vessels just above the opening to ventricles. Each consists of three cusps that curve upwards to from small pockets (Cleveland Clinic 2009, 2011).

The four heart valves open and close in response to pressure changes that occur in the ventricles during each cardiac cycle. When the ventricles relax their pressures drop below those of the atria, pulmonary trunk artery and aorta. This allows the AV valves to open as their cusps passively drop downwards. The pressure change additionally permits blood to flow into the ventricles from the atria without restriction(Consultants, 2006).

The semilunar valves close during this same period as blood flowing toward the ventricles collects in the pockets of the cusps. Closure of the semilunar valves prevents blood from re-entering the ventricles while they are relaxing. After filling with blood, the ventricles contract and their rising pressures forces blood up towards the atria and into the pulmonary trunk and aorta (Consultants, 2006).

Blood pushing up under the cusps causes the atrioventricular valves to close. As a result, blood enters the atria from the pulmonary veins but not from the ventricles. At the same time, rising pressure in pulmonary trunk artery and aorta forces the semilunar valves to open and blood flow into systemic and pulmonary circulatory systems. When the ventricles begin to relax, pressure in the chambers drop again and a new cardiac cycle begins (Hatchett, 2007).

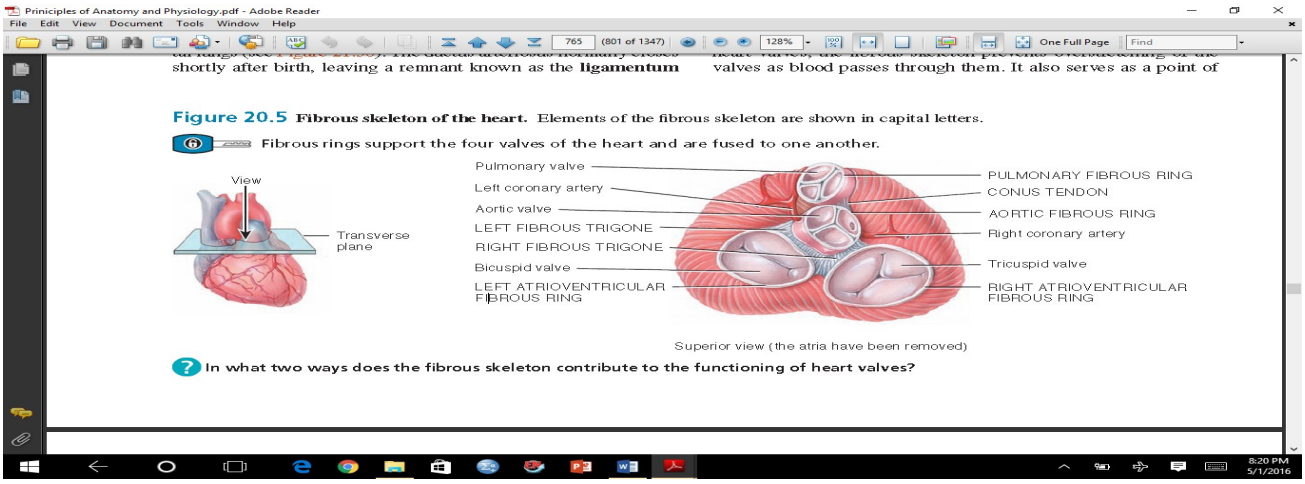


Figure 2 .5 Fibrous skeleton of the heart valve from the superior.

(Tortora and Derrickson, 2012)

The development of the heart .2.3.6

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 begin working before its development is complete (MMcKinley and O'Loughlin, 2008).

By day 19 (middle of week 3), two heart tubes (or endocardial 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 (MMcKinley and O'Loughlin, 2008).

The primitive heart is a single tube which soon shows grooves demarcating the sinus venosus, atrium, and ventricle and bulbus cord is 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 venous 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 (Harold, 2006).

The division of the primitive atrium into two is a complicated process but an important one in the understanding of congenital septal defects. Apartition, the septum primum, 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 scandium in the septum primum (Harold, 2006).

A second membrane, the septum secundum, then develops to the right of the primum 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 primum and hence to close it (Harold, 2006).

The two overlapping defects in the septa form the valve-like foramen ovale which shunts blood from the right to left heart in the fetus. After birth, this foramen usually becomes completely fused leaving only the fossa ovalis on the septal wall of the right atrium as 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 venosus absorbs into the right atrium so that the venae cavae 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).

Blood Supply .2.3.7

:The Arterial Supply of the Heart .2.3.7.1

The arterial supply of the heart is provided 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 subepicardial connective tissue (Snell, 2005).

The right coronary artery arises 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, 2005).

Branches

1. The right conus artery supplies the anterior surface of the pulmonary conus (infundibulum of the right ventricle) and the upper part of the anterior wall of the right ventricle (Snell, 2005).
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 to reach the apex (Snell, 2005).
3. The posterior ventricular branches are usually two in number and supply the diaphragmatic surface of the right ventricle (Snell, 2005).
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 a branch from the left coronary artery.

5. The atrial branches supply the anterior and lateral surfaces of the right atrium. One branch supplies the posterior surface of both the right and left atria. The artery of the sinuatrial node supplies the node and the right and left atria; in 35% of individuals it arises from the left coronary artery (Snell, 2005).

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 ventricle, and 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 a circumflex branch (Snell, 2005).

Branches

1. The anterior interventricular (descending) branch. Its branch supplies the right and left ventricles 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 conus artery supplies the pulmonary conus (Snell, 2005).

2. The circumflex artery is the same size as the anteriorinterventricular 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 branches supply the left ventricle. Atrial branches supply the left atrium (Tortora and Derrickson, 2012).

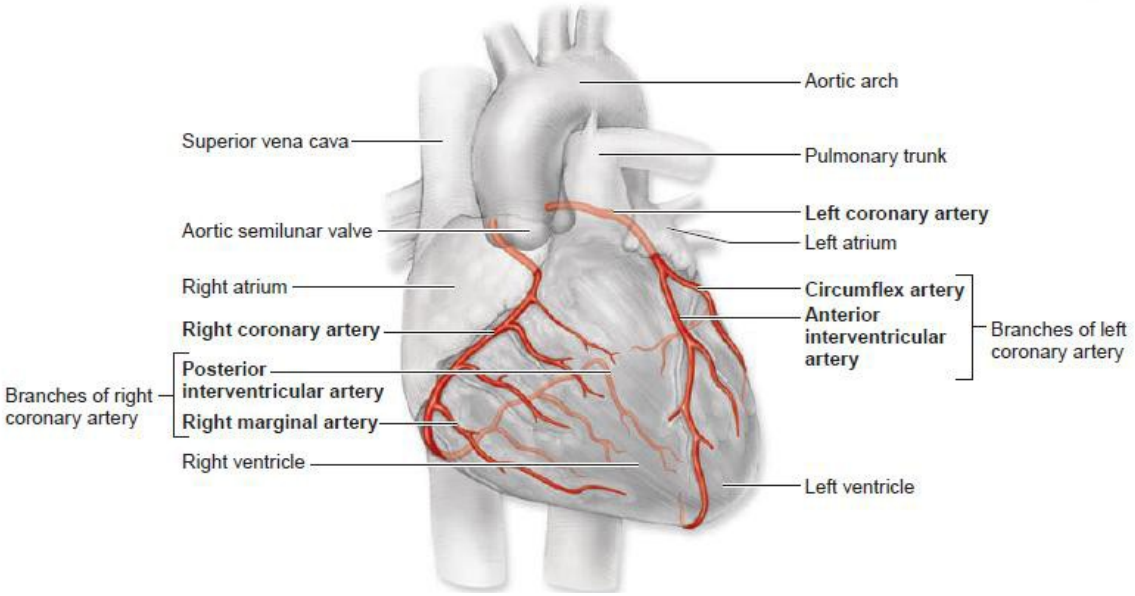


Figure. 2.6 Coronary Circulation. Anterior view of (a) coronary arteries (Tortora and Derrickson, 2012).

:Venous Drainage .2.3.7.2

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 drains by means of small veins (venae cordis minimae) 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 veins (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).

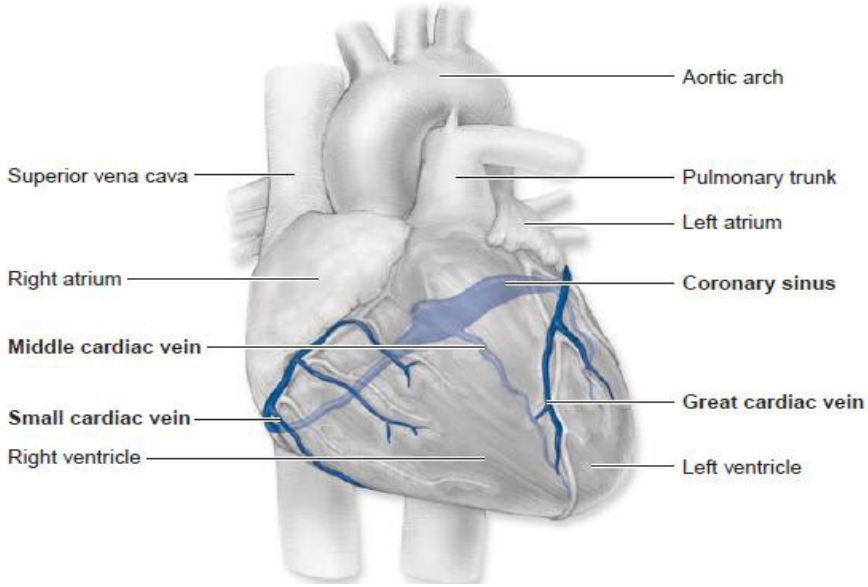


Figure. 2.7 Coronary Circulation. Anterior view of (a) coronary veins. (Tortora and Derrickson, 2012)

:Lymph Drainage .2.3.8

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).

Nerves Supply .2.3.9

The nerve supply of the heart is derived from the vagus (cardio-inhibitor) and the cervical and upper 5 thoracic sympathetic ganglia (cardioaccelerator) by way of superficial and deep cardiac plexuses (McMinn, 2009).

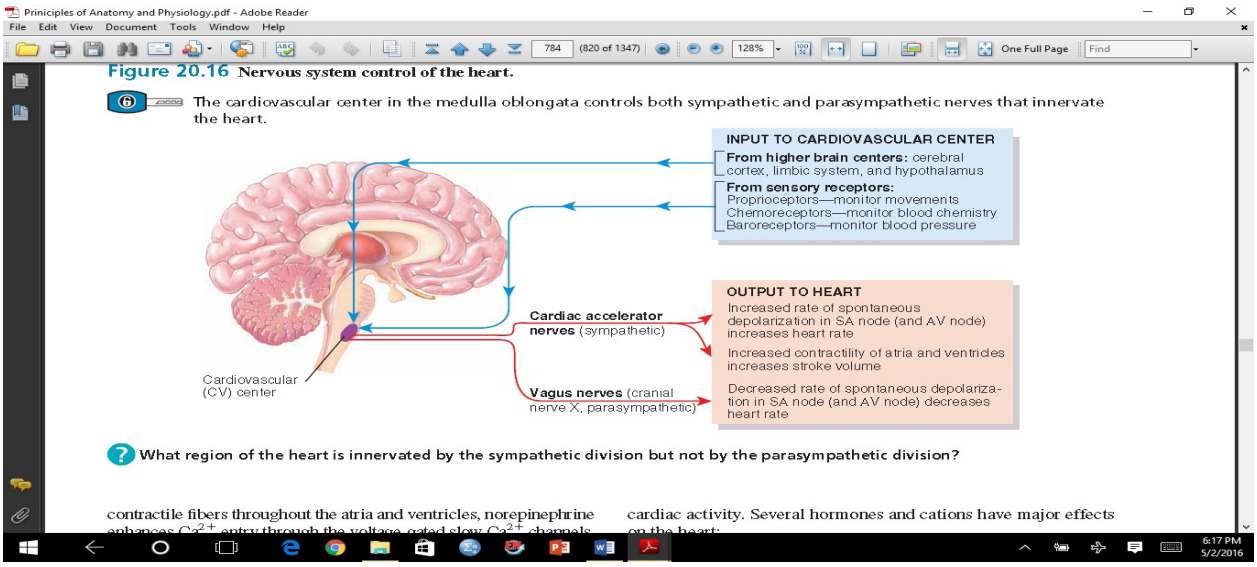


Figure 2.8 Nervous system control of the heart (Tortora and Derrickson, 2012).

Physiology of the heart .2.4

The parts of the heart normally beat in orderly sequence: Contraction of the atria (atrial systole) is followed by contraction of the ventricles (ventricular systole), and during diastole all four chambers 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 internodal atrial pathways, the atrioventricular node (AV node), the bundle of His and its branches, and the Purkinje system. The various parts of the conduction system and, under abnormal conditions, 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 the Purkinje system to the ventricular muscle (Tortora and Derrickson, 2012).

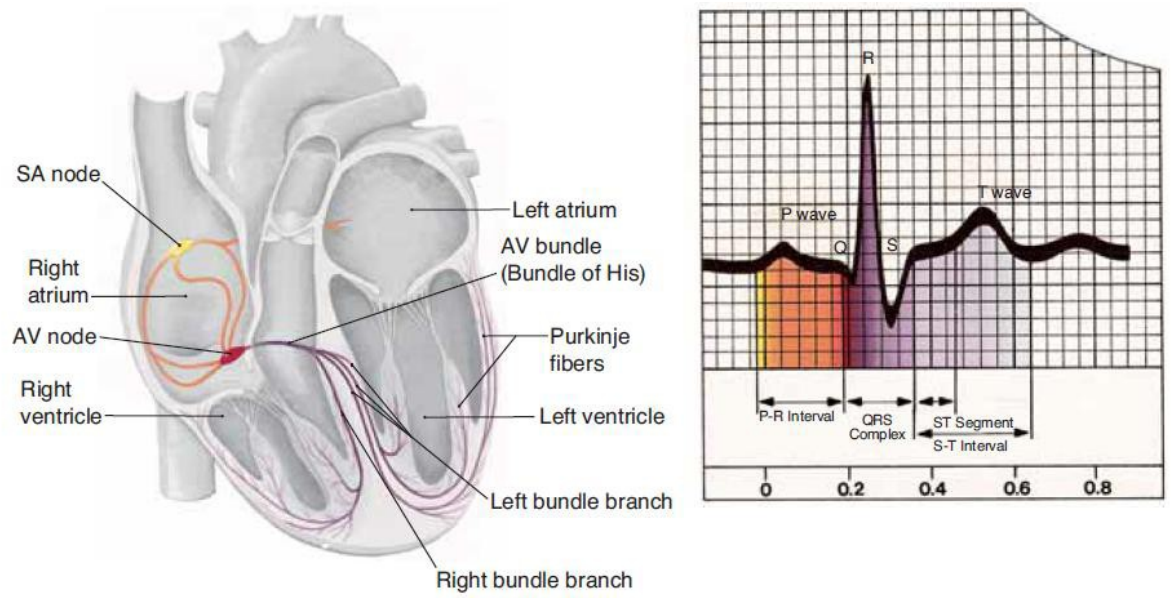


Figure. 2.9 Conduction pathway of the heart, anterior view of the interior of the heart, the electrocardiogram tracing is of one normal heartbeat. (Tortora and Derrickson, 2012)

Cardiac Muscle Tissue .2.4.1

Cardiac muscle cells make up the myocardium portion of the heart wall. They are relatively short, branched fibers that measure approximately 10-20 micrometers in diameter and 50 to 100 micrometers in length. Typically each cardiac myocyte contains a single nucleus, which is centrally positioned. Thick and thin myofilaments are present and prganized into myofibrils. Their overlapping arrangement creates alternating dark (A) and light (I) bands or striations, similar to those seen in skeletal muscle tissue. Sarcoplasmic reticulum tubules surround the myofibrils. However, there are not well organised and do not have termial cisternae. T-tubules are also present, but run along the Z-discs (instead of the myofilament overlap zones). The mitichondria in cardiac myocytes are large and numerous. They supply the ATP needed for repeated contraction of the heart (Tortora and Derrickson, 2012).

Unlike other types of muscle tissues, cardiac myocytes are joined end to end by intercalated discs. These complex, highly convoluted couplings

contain both anchoring junctions and electrical junctions. Forming the anchoring junctions are fascia adherens and desmosomes, which arrach the adjacent myocyte. The electrical junctions are composed of connexon protein channels, which usually occur in clusters referred to as gap junctions. Connexon proteins span the distance between adjacent plasma membranes and ions can travel through the channel pores. The ion movement allows action potentials to pass directly from cell to cell. This property makes the entrie myocardium act like a single cell (Tortora and Derrickson, 2012).

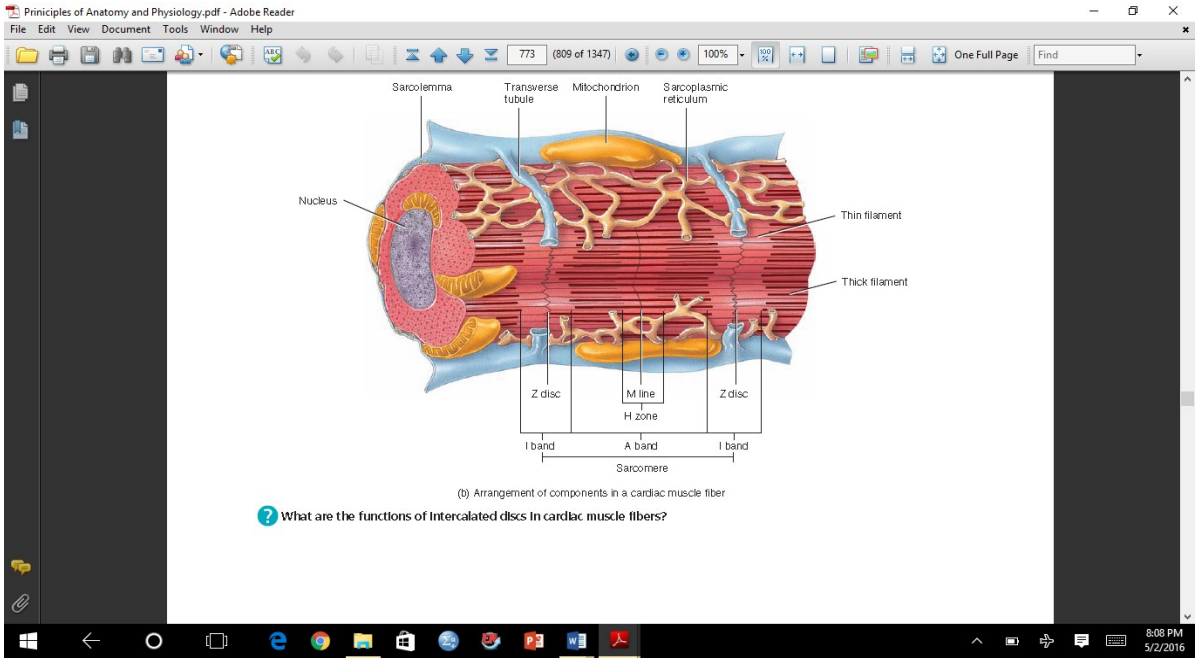


Figure 2.10 Histology of cardiac muscle tissue.

(Tortora and Derrickson, 2012)

Origin and spread of cardiac excitation .2.5

The Conduction System .2.5.1

The conducting system of the heart consists of cardiac muscle cells and conducting fibers (not nervous tissue) that are specialized for initiating impulses and conducting them rapidly through the heart. They initiate the normal cardiac cycle and coordinate the contractions of cardiac chambers (Woods, 2005).

The conducting system provides the heart its automatic rhythmic beat. For the heart to pump efficiently and the systemic and pulmonary circulations to operate in synchrony, the events in the cardiac cycle must be coordinated (Woods, 2005).

The sinoatrial (SA) node is a spindle-shaped structure composed of a fibrous tissue matrix with closely packed cells. It is 10-20 mm long, 2-3 mm wide, and thick, tending to narrow caudally toward the inferior vena cava. The SA node is located less than 1 mm from the epicardial surface, laterally in the right atrial sulcus terminalis at the junction of the anteromedial aspect of the superior vena cava (SVC) and the right atrium (RA) (Woods, 2005).

The middle internodal tract begins at the superior and posterior margins of the sinus node, travels behind the SVC to the crest of the interatrial septum, and descends in the interatrial septum to the superior margin of the AV node.

The posterior internodal tract starts at the posterior margin of the sinus node and travels posteriorly around the SVC and along the crista terminalis to the eustachian ridge and then into the interatrial septum above the coronary sinus, where it joins the posterior portion of the AV node. These groups of internodal tissue are best referred to as internodal atrial myocardium, not tracts, as they do not appear to be histologically discrete specialized tracts (Woods, 2005).

In 85-90% of human heart, the arterial supply to the AV node is a branch from the right coronary artery which originates at the posterior intersection of the AV and interventricular groove. In the remaining 10-15% of the heart, a branch of the left circumflex coronary artery provides the AV nodal artery. Fibers in the lower part of the AV node may exhibit automatic impulse formation. The main function of the AV node is modulation of the atrial impulse transmission to the ventricles to coordinate atrial and ventricle contractions (Woods, 2005).

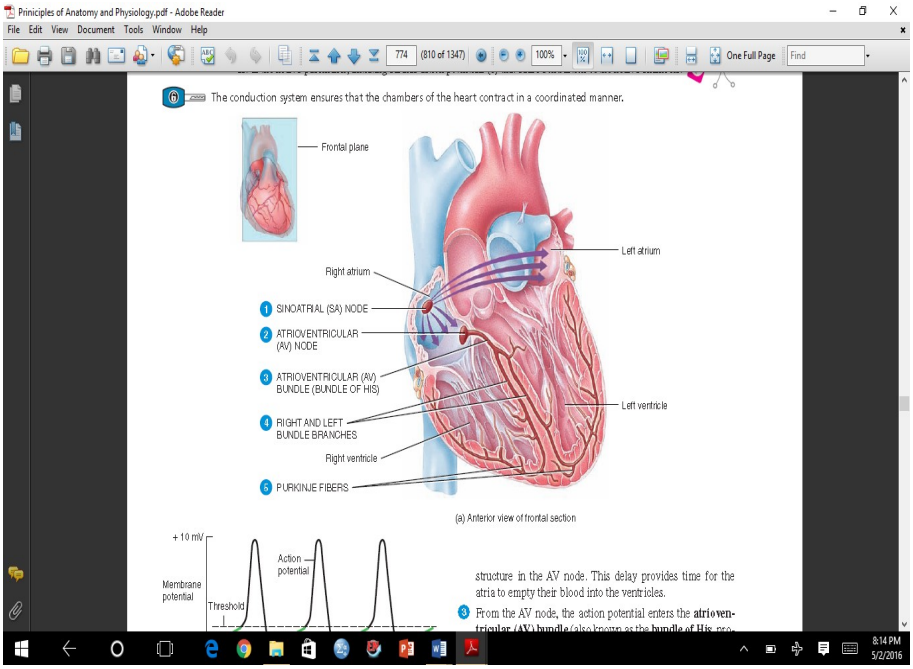


Figure 2.11 The conduction system of the heart.

(Tortora and Derrickson, 2012)

Bundle of His .2.5.2

The bundle of His is a structure that connects with the distal part if the compact AV node, perforates the central fibrous body and continues through the annulus fibrous, where it is called the non-branching portion as it penetrates the membranous septum. Connective tissue of the central fibrous body and membranous septum encloses the penetrating portion of the AV bundle, which may send out extensions into the central fibrous body (Woods, 2005).

Proximal cells of the penetrating portion are heterogeneous and resemble those of the compact AV node; distal cells are similar to cells in the proximal bundle branches. Branches from the anterior and posterior descending coronary arteries supply the upper muscular interventricular septum with blood, which makes the conduction system at this site more impervious to the ischemic damage, unless the ischemia is extensive (Tortora and Derrickson, 2012).

Bundle branches .2.5.3

The bundle branches originate at the superior margin of the muscle interventricular septum, immediately below the membranous septum with the cells of the left bundle branch cascading downward as a continuous sheet onto the septum beneath the noncoronary aortic cusp. The right bundle branch continues intramycardially as an unbranched extension of the AV bundle down the right side of the interventricular septum to the apex of the right ventricle and base of the anterior papillary muscle. The anatomy of the left bundle branch system may be variable and may not conform to a constant bifascicluar division (Tortora and Derrickson, 2012).

Purkinje fibers .2.5.4

Punkinje fibers connect with the ends of the bundle branches to form interwearving network on the endocardia surface of both ventricles. These fibers transmit the cardiac impulse almost simultaneously to the entire right and left ventricle endocardium. Punkinje fibers tend to be less concentrated at the base of the ventricles and the papillary muscle tips and only penetrate the inner third of the endocardium. They appear to be more resistant to ischemia than ordinary myocardial fibers (Hatchett, 2007).

Cardiac Cycle .2.5.5

The cardiac cycle is the sequence of events that occur when the heart beats. The cycle has two main phases: diastole – when the heart ventricles are relaxed and systole – when the ventricles contract. In a cardiac cycle, blood enters the right atrium of the heart from the superior and inferior vena cavae, and flows across the tricuspid valve into the right ventricle. From the right ventricle the blood flows into the pulmonary artery, which is separated from the ventricle by the pulmonary valve (Hatchett, 2007).

After oxygenation in the lungs, blood returns to the heart via four pulmonary veins that enter the left atrium. From the left atrium, blood flows across the mitral valve and into the left ventricle. From the left

ventricle blood is ejected across the aortic valve into the aorta. Together, the mitral and tricuspid valves are known as the atrioventricular valves and the aortic and pulmonary valves as the semilunar valves (Hatchett, 2007).

From a mechanical point of view, the cardiac cycle is due to blood movement as result of pressure differences within the chambers of the heart. In order for blood to flow through a blood vessel or through a heart valve, there must be force acting on the blood. This force is provided by the difference in blood pressure (a pressure gradient) across these structures by the contractions of the heart. Each heartbeat, or cardiac cycle, is divided into two phases of contraction and relaxation, stimulated by elctricla impulses from the sinoatrial node (SA node). The time during which ventricular contraction occurs is called systole. The time between ventricular contraction, during which ventricular filling occurs is called diastole also known as the relaxation phase) (Woods, 2005).

In early diastole, the ventricles relax, the semilunar valve close, the atrioventricular valves open and the ventricles fill with blood. In mid diastole, the atria and ventricles are relaxed, the semilunar valves are closed, the atrioventricular valves are open and the ventricles keep filling with blood. In late diastole, the SA node sends and electrical impulse to the atria, this causes the atria to contract and the ventricles to fill with more blood. The electrical signal that causes contraction moves from the atria toward the ventricles. Before it does, it reaches the atrioventricular node (AV node). The AV node delays the signal so that the ventricles can contract all at once rather than a little bit at a time (Woods, 2005).

Prior to systole, the electrical signal passes from the AV node down the AV bundl, also known as the bundle of His to the Punkinje fibers. The fibers allow the fast spread of the electrical signal to all parts of the ventricles and the electrical signal causes the ventricles to contract. Systole begins with the closure if the atrioventricular valves. During systole, the ventricles contract, the semi-lunar valves open and bloods is pumped from the ventricles to the aorta (Woods, 2005).

Blood pressure is highest during systole and lowest during diastole. It has two components, the systole and diastole pressure. Normal systole pressure for an adult is estimates at 120 mm/hg and normal diastole pressure is estimated at 80 mm/hg (Hatchett, 2007).

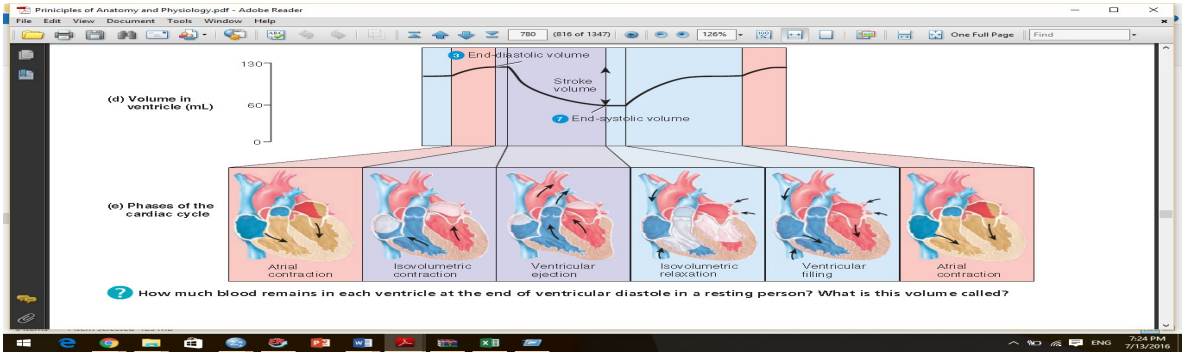


Figure 2.12: A cardiac cycle is composed -Phases of the cardiac cycle (Tortora and Derrickson, 2012)

Pathophysiology of the heart .2.6

Diseases of the heart fit into several general categories: congenital heart disease, ischemic heart disease, valvular diseases, and diseases of the myocardium (i.e., cardiomyopathies). Pericardial diseases and cardiac tumors are an additional small subset of conditions affecting the heart (Kemp. et al., 2008).

Congenital heart disease .2.6.1

There are three main categories of congenital heart disease: conditions causing a right-to-left shunt; conditions causing a left-to-right shunt; and conditions causing obstruction (Kemp. et al., 2008).

:Right-to-Left shunt .2.6.1.1

Deoxygenated blood from the right side of the heart goes to the left side; thus, deoxygenated blood is delivered to the body. This type of shunt usually results in cyanosis at the time of birth.

The causes of right-to-left shunt are found in those cases Tetralogy of Fallot, Tricuspid atresia, Truncus arteriosus, totally anomalous pulmonary venous return and Transposition of the great vessels (Kemp. et al., 2008).

:Left -to- Right shunt .2.6.1.2

Increases the amount of blood delivered to the right side of the heart and will result in hypertrophy and dilation of the right atrium or right ventricle (or both), depending upon the type of shunt. Eventually, the pressure in the right side of the heart increases and surpasses that in the left side of the heart, resulting in a reversal of the shunt from left-to-right to a right-to-left shunt. This change is called Eisenmenger syndrome (Kemp. et al., 2008).

The causes of left-to-right shunt are the Ventricular septal defect (VSD), atrial septal defect (ASD), patent ducts arteriosus (PDA) and atrioventricular septal defect (Kemp. et al., 2008).

Obstruction .2.6.2

An abnormally formed valve or vessel leads to pressure overload of the involved atrium or ventricle. The causes of obstruction as pathophysiologic mechanism of congenital heart disease are aortic stenosis, pulmonary stenosis and coarctation of the aorta (Kemp. et al., 2008).

Ischemic Heart Disease .2.6.3

The meaning of ischemic heart disease refers to inadequate perfusion of the myocardium, most commonly due to atherosclerosis of the coronary arteries.

Ischemia causes functional disturbances of the heart, including those like impaired relaxation, which occurs first, causing diastolic dysfunction, impaired contraction, which occurs second, causing systolic dysfunction, Myocardial stunning, a prolonged (hours to days) but reversible dysfunction after an acute ischemic event, Myocardial hibernation, which occurs when oxygenation is adequate to maintain viability of the myocardium but cannot support normal function (Kemp. et al., 2008).

Acute Coronary Syndromes .2.6.4

Differentiation of acute coronary syndromes are happened by two causes one if blood flow is restored within 20 minutes, no necrosis occurs: this is unstable angina and second If the blood flow is not restored within 20–40 minutes, necrosis can occur: this is an acute myocardial infarct (Kemp. et al., 2008).

Myocardial Infarcts .2.6.5

Death of a segment of myocardium due to prolonged ischemia, usually due to obstruction of a coronary artery they are two first is STEMI (previously called transmural or Q wave infarct): The full thickness of the wall of the ventricle is affected, and second is NSTEMI (previously called subendocardial or non-Q wave infarct): The full thickness of the wall is not affected; only the subendocardial myocytes are affected (Kemp. et al., 2008).

Sudden Cardiac Death .2.6.6

Sudden cardiac death is due to cardiac pathology. The time frame required to call a death “sudden cardiac death” is < 24 hours if the patient is untreated, and <1 hour if patient is treated).Most cases are due to an arrhythmia, usually ventricular tachycardia or fibrillation (Kemp. et al., 2008).

Chronic Ischemic Heart Disease .2.6.7

Chronic ischemic heart disease is a condition resulting from ischemic injuries occurring over time (i.e., in multiple episodes). The main Complications: Congestive heart failure (Woods, 2005).

Hypertensive Cardiovascular Disease .2.6.8

Some Cardiac disease occurring because of longstanding hypertension. The Cardiac hypertrophy is Increase in the weight of the heart and thickness of the wall of the left ventricle. Complications of hypertensive cardiovascular disease: CHF, lethal cardiac arrhythmias and atrial fibrillation secondary to left atrial dilation (Kemp. et al., 2008).

Congestive Heart Failure .2.6.9

The heart can no longer pump out enough blood to supply the body's needs. Types and terminology of CHF: Most cases of CHF are Systolic dysfunction, Diastolic dysfunction, Left-sided heart failure, Right-sided heart failure, Low-output failure and either Acute or chronic failure (Kemp. et al., 2008).

The morphology of left-sided heart failure: Recurrent bouts of pulmonary edema and increased pulmonary venous pressure lead to hemorrhage and hemosiderin-laden macrophages (i.e., "heart failure cells") in the lung.

The morphology of right-sided heart failure leads to Hepatosplenomegaly; increased venous pressure due to increased resistance to portal flow results in passive congestion of blood in the liver and spleen and Ascites and peripheral pitting edema (Kemp. et al., 2008).

General Valvular Disease .2.6.10

Conditions specifically related to the aortic and mitral valve, endocarditis and rheumatic fever **Endocarditis;** It is infection (usually bacterial) involving the valve cusps or the adjacent endocardium, or both. The type of endocarditis is Infective endocarditis and Nonbacterial thrombotic endocarditis. **And Rheumatic Fever;** Group A β-hemolytic streptococcal pharyngitis causes production of antibodies that cross react with cardiac antigens. These antibodies produce a disease known as acute rheumatic fever. Acute rheumatic fever occurs most commonly in children aged 4 to 9 years (Riede and Werner, 2004).

Aortic Valvular Disease .2.6.11

Aortic Stenosis; Obstruction of blood flow through the aortic valve. The main Causes of aortic stenosis is Tricuspid aortic valve degenerative calcification, congenital bicuspid aortic valve degenerative calcification and chronic rheumatic valvulitis**. And Aortic Regurgitation;** Leakage of blood back through closed valve. The causes of chronic aortic regurgitation is dilation of aortic valve ring as occurs in Marfan syndrome, Healed endocarditis and Bicuspid aortic valve (Riede and Werner, 2004).

Mitral Valvular Disease .2.6.12

Mitral Stenosis. The cause: In adults, mitral stenosis is almost always due to chronic rheumatic valvulitis. **Mitral Regurgitation;** Causes of mitral regurgitation: Three of the main causes of mitral regurgitation are a myxomatous mitral valve, mitral annular calcification, and endocarditis (Riede and Werner, 2004).

Cardiomyopathies .2.6.13

Cardiomyopathy (cardio meaning heart, myopathy meaning muscle disease) refers to a group of disorders that directly damage the muscle of the heart walls. In these disorders, all chambers of the heart are affected. The heart's function as a pump is disrupted, leading to an inadequate blood flow to organs and tissues of the body. Depending on the nature of the injury or abnormality in the heart muscle and the resulting structural changes in the heart chambers, one of three types of nonischemic (not caused by heart attack) heart muscle disease may be present dilated congestive, hypertrophic and restrictive (Riede and Werner, 2004).

Hypertrophic cardiomyopathy .2.6.14

It is Asymmetric thickening of the interventricular septum; fibrosis of the aortic outflow tract corresponding to thick interventricular septum striking the anterior leaflet of the mitral valve. **Dilated cardiomyopathy as** the gross morphology is External features is Globular heart and internal features is Dilation of all four chambers. The causes of dilated cardiomyopathy are Idiopathic, Genetic causes, Toxin-induced, Metabolic causes, Sarcoidosis and hemochromatosis. Restrictive **cardiomyopathy;** Basic description: The myocardium is partially infiltrated by noncontractile tissue or extracellular material (e.g., collagen, amyloid). This infiltration of the myocardium impairs the ability of the heart to dilate (Kemp. et al., 2008).

Pericardial Disease .2.6.15

This section will cover pericarditis, pericardial effusion, and cardiac tamponed. Forms of pericarditis: Can be categorized two ways, by nature

of transudate or exudate or by general etiology. **Pericardial Effusion;** Basic description: Accumulation of fluid in the pericardial sac (Kemp. et al., 2008).

Cardiac Tamponade .2.6.16

Basic description: Compression of the heart due to an increase in fluid within the pericardial sac (Kemp. et al., 2008).

:Effect of diabetes to the cardiovascular system .2.7

When the glucose increases in the blood it can cause serious complications including: Cardiomyopathy, nephropathy, neuropathy, and retinopathy (Cheung et al., 2007).

Diabetes is a major risk factor for coronary artery disease and cardiovascular disease (Somaratne et al., 2011). Is the most important cause of morbidity and mortality in patients with type 2 diabetes, accounting for approximately two-thirds of total mortality (Srivastava et al., 2008). Diastolic dysfunction has been described as an early sign of diabetic heart muscle disease preceding systolic damage it is associated with future occurrence of heart failure, is a predictor of cardiovascular morbidity and mortality in the general population (Ike and Ikeh, 2006).

Diabetes mellitus is associated with a multitude of cardiovascular complications, e.g., increased incidence of atherosclerotic coronary artery disease, myocardial infarction, congestive heart failure, coronary microangiopathy, and systemic arterial hypertension. In addition, structural myocardial involvement termed as diabetic cardiomyopathy may be there which is suggested by clinical, epidemiological, and histological studies done till date in large number of diabetics (Galderisi et al., 1991). Myocardial involvement in diabetics may occur relatively early in the course of disease, initially impairing early diastolic relaxation and when more extensive, it causes decreased myocardial contraction. Prior to the development of symptomatic congestive heart failure,

subclinical left ventricular dysfunction (systolic or diastolic) exists for some time (Thanikachalam, 1981; Mittal, 1983 and Ahmed, 1975). However, frequency of progression from pre-clinical to clinically evident myocardial dysfunction is not established. Further, role of metabolic control in primary prevention or reversal of myocardial dysfunction had not been much studied. With the availability of echocardiography and Doppler, it is now possible to fully elucidate the natural history of cardiac involvement from pre-clinical to clinical stage in patients with diabetes (Mathew, 1992 and Studdard, 1989).

Two major types of heart and blood vessel disease, also called cardiovascular disease, are common in people with diabetes: coronary artery disease (CAD) and cerebral vascular disease. People with diabetes are also at risk for heart failure. Narrowing or blockage of the blood vessels in the legs, a condition called peripheral arterial disease, can also occur in people with diabetes (Alwan et al., 2014).

Coronary Artery Disease .2.7.1

The term coronary artery disease (CAD) describes heart disease caused by impaired coronary blood flow. In most cases, CAD is caused by atherosclerosis, which affects not only the coronary arteries but arteries in other areas of the body. Diseases of the coronary arteries can cause myocardial ischemia and angina, myocardial infarction or heart attack, cardiac arrhythmias, conduction defects, heart failure, and sudden death. Heart attack is the single largest killer of men and women in the United States, Canada, and other industrialized countries. Each year, more than 1.6 million Americans have new or recurrent myocardial infarctions; one third of those die within the first 24 hours, and many of those who survive suffer significant morbidity. In spite of these numbers, the overall death rate from CAD has declined over the past several decades (Porth C.M & Mattfin G, 2009).

Major risk factors for CAD include cigarette smoking, elevated blood pressure, elevated serum total and low-density lipoprotein (LDL) cholesterol, low serum high-density lipoprotein (HDL) cholesterol,

diabetes, advancing age, abdominal obesity, and physical inactivity. Individuals with diabetes and the metabolic syndrome are at particularly increased risk for development of cardiovascular disease (Porth C.M & Mattfin G, 2009).

CAD is a disorder of impaired coronary blood flow, usually caused by atherosclerosis. Myocardial ischemia occurs when there is a disparity between myocardial oxygen supply and demand and can present as chronic ischemic heart disease or ACS. Diagnostic methods for CAD include ECG, exercise stress testing, nuclear imaging studies, CT, MRI, and angiographic studies in the cardiac catheterization laboratory (Porth C.M & Mattfin G, 2009).

The chronic ischemic heart diseases include chronic stable angina, silent myocardial ischemia, and variant (vasospastic) angina. Chronic stable angina is associated with a fixed atherosclerotic obstruction and pain that is precipitated by increased work demands on the heart and relieved by rest. Variant angina can result from spasms of the coronary arteries or other dysfunctions. Silent myocardial ischemia occurs without symptoms (Porth C.M & Mattfin G, 2009).

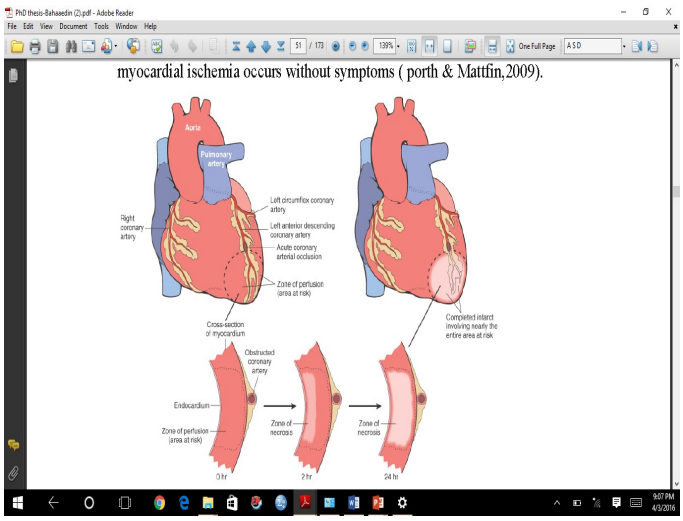


Figure. 2.13 Schematic representation of the progression of myocardial necrosis after coronary artery occlusion. Necrosis begins in a small zone of the myocardium beneath the endocardial surface in the

center of the ischemic zone (Porth C.M & Mattfin G, 2009).

Cerebral Vascular Disease .2.7.2

Cerebral vascular disease affects blood flow to the brain, leading to strokes and TIAs. It is caused by narrowing, blocking, or hardening of the blood vessels that go to the brain or by high blood pressure (Porth C.M & Mattfin G, 2009).

Stroke .2.7.3

A stroke results when the blood supply to the brain is suddenly cut off, which can occur when a blood vessel in the brain or neck is blocked or bursts. Brain cells are then deprived of oxygen and die. A stroke can result in problems with speech or vision or can cause weakness or paralysis. Most strokes are caused by fatty deposits or blood clots—jelly-like clumps of blood cells—that narrow or block one of the blood vessels in the brain or neck. A blood clot may stay where it formed or can travel within the body. People with diabetes are at increased risk for strokes caused by blood clots (Somaratne et al., 2011).

A stroke may also be caused by a bleeding blood vessel in the brain. Called an aneurysm, a break in a blood vessel can occur as a result of high blood pressure or a weak spot in a blood vessel wall. The level of disability varies from patient to patient according to the type of stroke suffered, the part of the brain affected, and the size of the damaged area. Approximately two million brain cells die every minute during a stroke which increases the risk of brain damage, disability, and death. Stroke is the third leading causes of death in United States and UK after heart disease and cancer, and the number one cause of adult disability. In the United States, over 160,000 American adults die of stroke each year. In Europe, approximately 650,000 people die of stroke. The risk of having a stroke more than doubles each decade after the age of 55. According to the World Health Organization, 15 million people worldwide have a stroke ever year, of which 5 million die and 5 million are permanently disabled.

However, medical research shows that every year 80 percent of the all cases of stroke can be prevented (Adams, 2007).

Guidelines for the early management of adults with ischemic stroke: A guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. Stroke, Cost of Stroke is estimated direct and indirect cost of stroke in the US for 2010 was $ 73.7 billion. The estimated cost of stroke in Europe in 2010 was approximately € 64.1 billion (Fang et al., 2000). The two major class of strokes is ischemic stroke and hemorrhagic stroke other forms of stroke cardiac arrest and hematomas adjacent to the brain (Fang et al., 2000).

There are 2 types of risk factors for stroke: controllable and uncontrollable. Controllable risk factors generally fall into two categories: lifestyle risk factors or medical risk factors. Lifestyle risk factors can often be changed, while medical risk factors can usually be treated. Both types can be managed best by working with a doctor, who can prescribe medications and advise on how to adopt a healthy lifestyle. Uncontrollable risk factors include being over age 55, being male, being African American, Hispanic or Asian/Pacific Islander, or having a family history of stroke or transient ischemic attack (TIA) (Conditions, 2006).

The stroke can be diagnosis by the following first clinically do neurological examination and the other tools like CTS, lumbar puncture, MRI, Transcranial Doppler, Cerebral Angiography, Carotid Stenosis Treatments, Electrocardiogram, Transthoracic echocardiogram, Leg Ultrasound and blood test.

It is very difficult to regenerate brain cells when they have died. There has been a lot of work done in this area and there are glimmers of hope. Strokes damage brain cells and the amount of damage and the severity can vary enormously. At one extreme it could be physical movement

affected, but at the other memory and reasoning are lost. The final danger will always be the death of the victim from the effects of the stroke (Conditions, 2006).

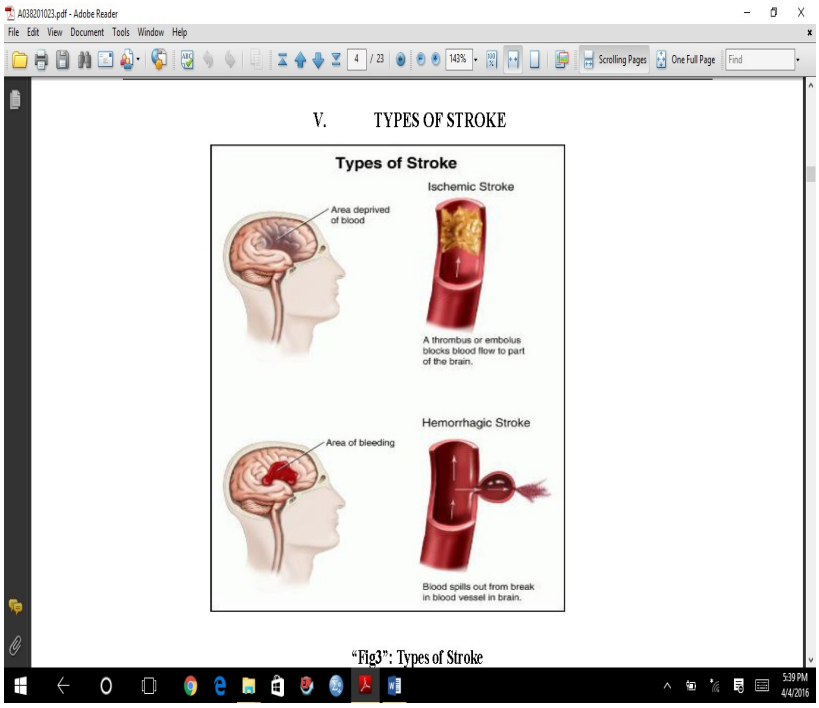


Figure: 2.14 Type of stroke(Conditions, 2006).

(Transient Ischemic Attack (TIAs .2.7.4

TIAs are caused by a temporary blockage of a blood vessel to the brain. This blockage leads to a brief, sudden change in brain function, such as temporary numbness or weakness on one side of the body. Sudden changes in brain function also can lead to loss of balance, confusion, blindness in one or both eyes, double vision, difficulty speaking, or a severe headache. However, most symptoms disappear quickly and permanent damage is unlikely. If symptoms do not resolve in a few minutes, rather than a TIA, the event could be a stroke. The occurrence of a TIA means that a person is at risk for a stroke

sometime in the future. Similarly, diabetes increases the risk of stroke (Folsom, 1999 and Jamrozik, 2000). For example, the risk of stroke among patients taking hypoglycemic medications was increased 3-fold among the nearly 350 000 men in the Multiple Risk Factor Intervention Trial (Stamler et al., 1993). In the Baltimore-Washington Cooperative Young Stroke Study, stroke risk increased more than 10-fold in diabetic patients younger than 44 years of age, ranging as high as 23-fold in young white men (Rohr J et al., 1996). Diabetes also increases stroke related mortality, doubles the rate of recurrent stroke, and trebles the frequency of stroke-related dementia (Luchsinger, 2001; Hankey, 1998 and Tuomilehto J, 1996).

Heart Failure .2.7.5

Heart failure is a chronic condition in which the heart cannot pump blood properly—it does not mean that the heart suddenly stops working. Heart failure develops over a period of years, and symptoms can get worse over time. People with diabetes have at least twice the risk of heart failure as other people. One type of heart failure is congestive heart failure, in which fluid builds up inside body tissues. If the buildup is in the lungs, breathing becomes difficult.

Blockage of the blood vessels and high blood glucose levels also can damage heart muscle and cause irregular heartbeats. People with damage to heart muscle, a condition called cardiomyopathy, may have no symptoms in the early stages, but later they may experience weakness, shortness of breath, a severe cough, fatigue, and swelling of the legs and feet. Diabetes can also interfere with pain signals normally carried by the nerves, explaining why a person with diabetes may not experience the typical warning signs of a heart attack (Jamrozik et al., 2000).

Peripheral Arterial Disease .2.7.6

Another condition related to heart disease and common in people with diabetes is peripheral arterial disease (PAD). With this condition, the blood vessels in the legs are narrowed or blocked by fatty deposits, decreasing blood flow to the legs and feet. PAD increases the chances of a heart attack or stroke occurring. Poor circulation in the legs and feet also raises the risk of amputation. Sometimes people with PAD develop pain in the calf or other parts of the leg when walking, which is relieved by resting for a few minutes (Jamrozik et al., 2000).

Diabetes increases the incidence and severity of limb ischemia approximately 2- to 4-fold (Abbott RD et al., 1990). Data from the Framingham cohort and Rotterdam studies show increased rates of absent pedal pulses, femoral bruits, and diminished ankle-brachial indices (Abbott RD, 1990; Meijer, 1998 and Hiatt WR, 1995). Diabetic peripheral arterial disease often affects distal limb vessels, such as the tibial and peroneal arteries, limiting the potential for collateral vessel development and reducing options for revascularization (Jude et al., 2001). As such, patients with diabetes are more likely to develop symptomatic forms of the disease, such as intermittent claudication and critical limb ischemia, and undergo amputation. In the Framingham cohort, the presence of diabetes increased the frequency of intermittent claudication by more than 3-fold in men and more than 8-fold in women.4 Diabetes is the No. 1 cause of nontraumatic amputations in the United States. For patients aged 65 to 74 years, diabetes heightens the risk of amputation more than 20-fold, putting these patients at great risk for limb loss (Rep, 1998).

:Cardiac Disease Due Diabetes mellitus .2.8

Diabetes mellitus (DM) causes damaging effects on the cardiac function; these effects can be observed on the diastolic performance of the heart reflected on the change in transmitral blood velocity, the cardiac wall and septum thickness. Impaired relaxation despite up regulated calcium-handling protein atrial myocardium from type 2 diabetic patients with preserved ejection fraction (Lamberts et al., 2014).

Impaired cardiac function during diastole is one of the early manifestations of cardiac dysfunction in type 2 diabetes (Jain et al., 1996), and it makes type 2 diabetic patients prone to developing clinical features of heart failure, with both preserved and reduced ejection fraction (From et al., 2010).Diastolic function deteriorates progressively over time (From et al., 2009), but the exact mechanisms underlying this clinically important increased stiffness and/or impaired relaxation, especially in diabetes, remain unclear. In contrast to systolic dysfunction, treatments for diastolic dysfunction are limited, which is likely due to this lack of knowledge regarding the pathophysiology of diastolic dysfunction (Paulus and van Ballegoij, 2010).

Impact of Diabetes on Cardiac Structure and Function The Strong Heart Study (Devereux et al., 2000).

Accordingly, the present study was undertaken to assess LV structure and function in individuals with and without DM among American Indians participating in the Strong Heart Study (SHS).14–16 this population includes tribes with various prevalence rates of both DM and coronary heart disease. The specific objectives were to determine whether (Kannel and DL., 1979). Non–insulin-dependent DM is associated with LV hypertrophy and dysfunction in a population-based sample of middle-aged to elderly adults, (Heyden et al., 1980). Observed associations are independent of major correlates of LV hypertrophy (body size, BP, sex, and age), and. DM is associated with more severe LV abnormalities in women than in men (Kleinman et al., 1988).

Patients with Early Diabetic Heart Disease Demonstrate a Normal Myocardial Response to Dobutamine (Fang, 2003). A number of experimental, pathologic, and epidemiologic studies support the existence of diabetic cardiomyopathy (Francis, 2001), the clinical diagnosis of which is made when systolic and diastolic left ventricular (Lamberts et al.) dysfunction are present in diabetic patients without other known cardiac disease . As with other conditions where new cardiac imaging technologies have identified subclinical heart disease myocardial backscatter and strain characteristics in patients with diabetes mellitus

have been shown to be abnormal (Di Bello et al., 1995) and (Fang et al., 2003b).

2.9. Sonography of the Heart (echocardiography):

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 noninvasive, 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 two-dimensional echocardiography has allowed cardiac structures to be visualized in a real time fashion. Thus, the echocardiographers can now assess intracardiac lesions, observe contractility, and estimate valvular function. The combination two-dimensional and M-mode studies provides an extremely accurate means of evaluating wall thickness, valvular orifice an chamber size, and contractility of the left ventricle (Sandra et al., 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 techniques. The standard M-mode examination is presented first, followed by evaluation of the heart by combined two-dimensional and M-mode techniques (Sandra et al., 2011).

2.9.1. Patient preparation

A basic EKG is attached to the patient assist with the timing of the cardiac cycle. Leads are attached to the right chest left chest and left hip region avoiding hair if possible(Tempkin, 1999).

2.9.2. Patient position

- Left lateral decubitus for most views with the left arm extended above the head and the right arm at the patients side (Tempkin, 1999).
- Sub xiphoid: left lateral decubitus or supine bend the knees to relax the stomach muscles if needed (Tempkin, 1999).
- Supra sterna: the patient is supine with the neck extended A pillow can also be placed under the shoulder allowing the head to drop back hyper extending the neck even further (Tempkin, 1999).

2.9.3. Types of Transducers used

Several types of transducers are available for echocardiographic techniques. Ideally one should use as high a frequency as possible to improve the resolution of returning echoes. However, the higher the frequency, the less the penetration; therefore compromises have to be made to obtain the best possible image (Sandra et al., 2011).

Many echocardiographers working with adults use a 3.5-MHz transducer with a medium focus. The larger patient may require a 2.25-MHz transducer, whereas a barrel-chested, emphysematous patient needs a 1.6-MHz transducer. The pediatric patient generally requires a 5.0- or a 7-5-MHz transducer for improved resolution and near-field definition (Sandra et al., 2011).

Although many transducers are internally focused to improve resolution by shaping the beam and reducing distortion, most cardiac transducers are of medium focus to concentrate the maximum resolution in the area of the mitral valve (Sandra et al., 2011).

The smaller crystal or diameter of the transducer allows better skin contact between the rib interspaces and also gives more freedom to “sweep the beam.” Thus the transducer remains in one interspace, but the beam angle is swept obliquely from the right shoulder to the left hip to record cardiac structures (Sandra et al., 2011).

Breathing techniques .2.9.4

For the majority of patients normal respiration. When ribs or lungs interfere having the patients either hold their breath or expel all their air and not breathe may improve the image .you may also need to slide an interspace to follow the movement of the heart. Experiment to find the best possible picture (Tempkin, 1999).

Transducers orientation .2.9.5

NOTE: Hold the transducer like a pencil, keeping two fingers on the patient at all times. This contact helps to prevent unintentional sliding and allows the sonographer to know how much pressure he or she is applying.

NOTE: To simplify the discussion of transducer orientation, imagine a clock on the patient's chest. The indicator on the transducer, which is some type of mark or indentation, will be directed anywhere from one to twelve o'clock. (To check indictor orientation side. There should be movement on the left side of the sector. Adjust L/R invert if necessary) (Tempkin, 1999).

NOTE: Most movements are very slight once the proper interspace is found (Tempkin, 1999).

NOTE: Remember the heart sits on an angle between the right shoulder and left hip (Tempkin, 1999).

Heart survey using echocardiography .2.10

The 2 D Examination .2.10.1

:The purpose of the 2 D examination is to

a) Identify the chambers and walls valves of the heart, and evaluated their size, thickness, and motion (Tempkin, 1999).

Assess the anatomical relationships of structures to rule out congenital (b
.(defects (Tempkin, 1999

Document the presence of any pathology including tumors or fluid surrounding the heart, or thrombi within (Tempkin, 1999).

Parasternal views .2.10.2

1. Begin with parasternal long axis by placing the transducer to the left side of the sternum in the second to third intercostal space with the indicator on the transducer directed towards 10 o'clock. Evaluate the sizes of the LA, LV, aortic root, and the RV. Assess for thickness and motion of the AV, MV, IVS, and posterior wall of the LV (Tempkin, 1999).
2. Maintaining the same interspace and 10 o'clock orientation, angle the transducer inferior and medial towards the belly button. This produces the right ventricular inflow view and visualizes the more anterior structure of the heart: the RA, TV, and RV. A remnant of the Eustachian valve (a normal variant) may also be observed in the RA (Tempkin, 1999).
3. To obtain the right ventricular out flow view, the transducer is now angled superior and lateral towards the left shoulder. The indicator is still directed towards10 o'clock. This will open the pulmonary artery and allow for assessment of the pulmonic valve (Tempkin, 1999).
4. The transducer 90 degrees clock wise towards 1 o'clock maintaining the same inter space as above and keeping the transducer close to the sternum. The parasternal short axis views are observed here, beginning with aortic valve level. Tilt the transducer towards the right shoulder .start by visualizing the area above the aortic valve for the presence of pathology, then slowly sweep towards the level of the aortic valve. The AV should be in the center of the screen with the LA, RV, RA, and PA surrounding it. Evaluate for the presence of three aortic cusps and note the thickness and motion of all the valve (Tempkin, 1999).
5. Continue to slowly sweep laterally through the left ventricular out flow tract region towards the mitral valve. Only the angle of the transducer has changed and the ultra sound beam is now pointing almost directly

anterior to posterior. Both leaflets of the mitral valve should be observed as well as its biphasic motion (Tempkin, 1999).

6. Slowly angle the transducer further lateral, towards the left hip. The cross section of the LV appears round with the papillary muscles indenting the inner surface, giving the cavity a mushroom –like appearance. Assess LV function for focal or global abnormalities. Continue to sweep laterally, beyond the papillary muscles as deep in to the ventricles as possible allowing for further assessment of LV function (Tempkin, 1999).

NOTE: Occasionally, you need to slide an intercostal space to obtain the different level of short axis, though angling is usually sufficient (Tempkin, 1999).

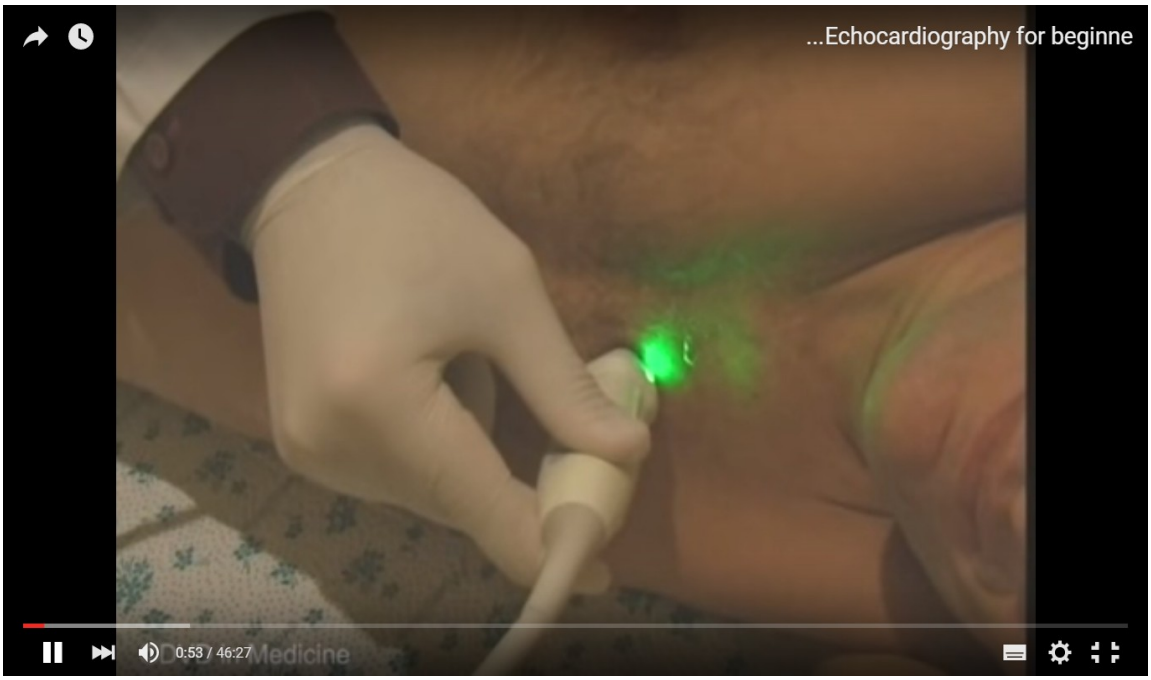


Figure. 2.15 Parasternal Long axis views. (parvliuk and povalishko, 2013)

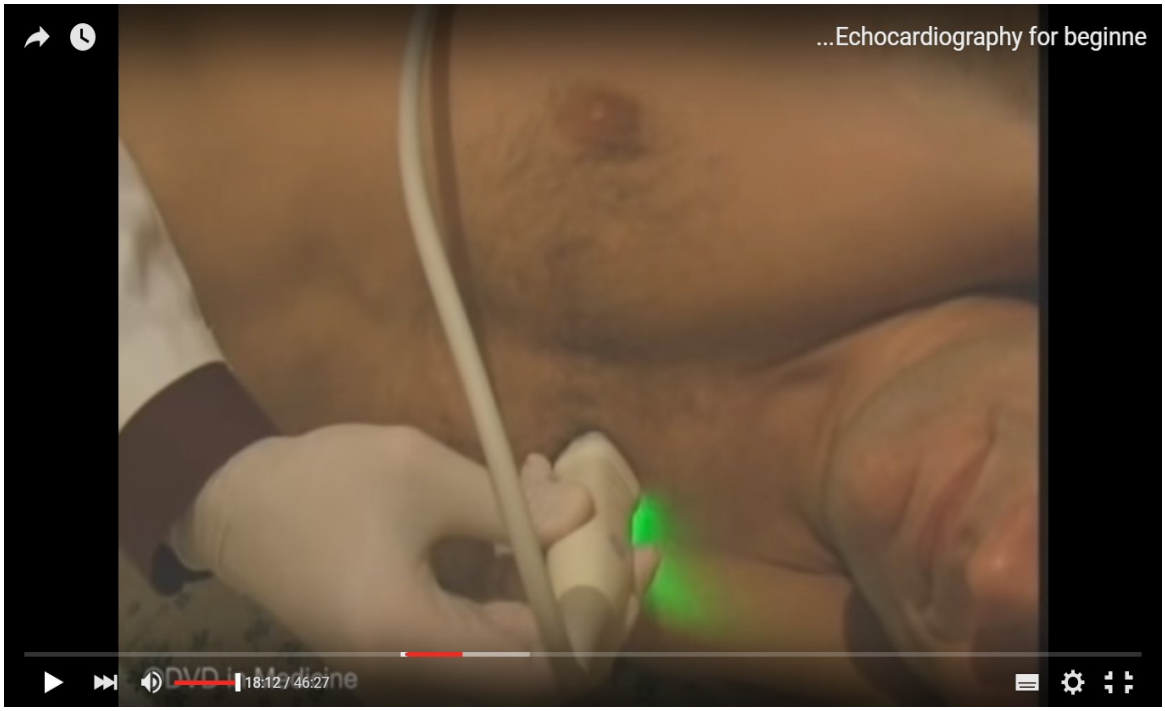


Figure. 2.16 Parasternal short axis views. (parvliuk and povalishko, 2013)

Apical Views .2.10.3

1. Place the transducer on the left flank, lateral to the left breast and point up wards, in the direction of the right shoulder. The indicator is oriented to 3 o'clock. The heart is transected from apex to base and the four chambers, MV, TV, interventricular septum, and lateral walls in the apical region are now visualized. Each structure is evaluated in respect to its size, thickness, and motion. This is known as the apical four chamber view (Tempkin, 1999).

TE: If unable to find the proper apical inter space, locate the PMI (point of maximal impulse) by placing two fingers on the left side of the chest and feeling for the heartbeat. The transducer is placed at this position. This is the apex of the heart (Tempkin, 1999).

NOTE: It is important to visualize the endocardium of the LV in order to assess function. Evaluate the walls to see if they are thickening and there are any focal or global ischemic abnormalities. When estimating motion it is easiest to segmentalize the ventricle, looking first at the proximal, mid, then distal walls, and then to check overall function (Solomon, 2007).

2. For the apical five – chamber view, angle the transducer slightly superior to open the LVOT and the aortic valve. The MV and TV become obscured. Evaluate for the presence of any obstruction in the outflow tract region.

3. Rotate the transducer counterclockwise towards 12 o'clock while still pointing towards the right shoulder. The MV, LA, and LV are visualized and thus called the apical two –chamber view. The inferior, anterior, and apical walls of the LV can now be assessed (Tempkin, 1999).

4. Rotate the transducer further counterclockwise towards 11 o'clock opening up the apical long axis view. The structures seen in parasternal long axis are visualized again in this view, but due to the different orientation, the apical region of the heart is now observed (Leeson and Becher, 2007).

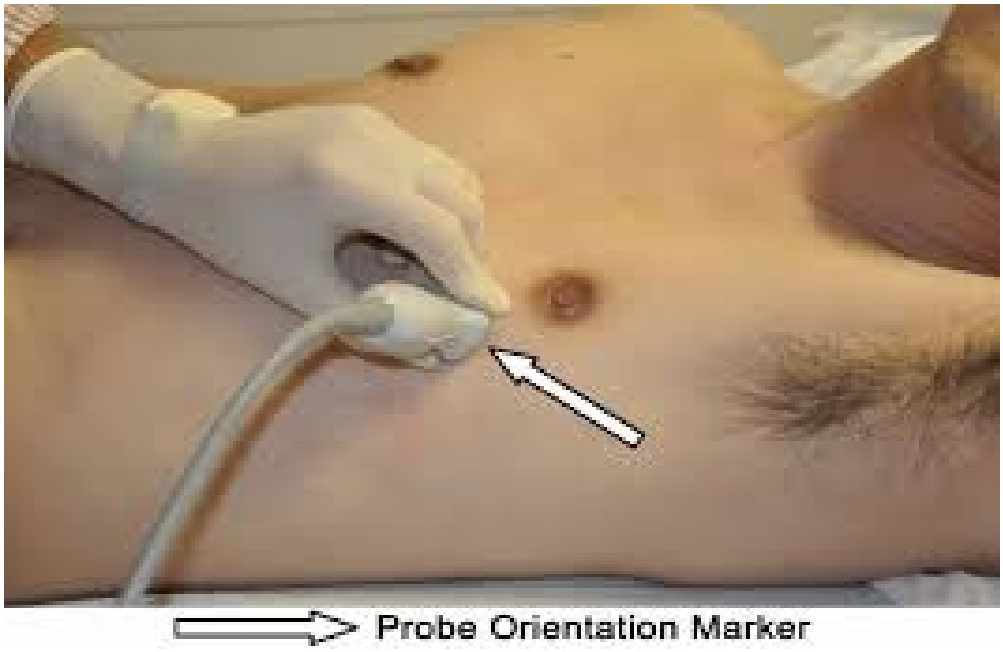


Figure. 2.17 Apical Views. (parvliuk and povalishko, 2013)

Subxiphoid views .2.10.4

1. To obtain the subxiphoid long axis, place the transducer below the xiphoid process and slightly to the right of midline away from the stomach on a softer portion of the abdomen. Using the liver as a window, point the transducer toward the left shoulder. Hold the hand above the

transducer, rather than like a pencil. These enable the transducer to be angled under the ribs and prevent the hand from interfering with the scan. The indicator is pointed towards 3 o'clock. The four chambers of the heart are visualized and assessed for relative sizes. If the chambers appear foreshortened, the transducer should be rotated the transducer should be rotated accordingly. The area around the heart should also be evaluated for the presence of pericardial fluid, tumors, and masses. The interatrial septum is also best evaluated in this view (Edmund, 2004).

2. For the subxiphoid short axis views, rotate the transducer 90 degrees counterclockwise towards 12 o'clock. Sweeping the transducer from the direction of the left shoulder to direction of the right shoulder procedures the same three levels as the parasternal short axis views (papillary muscles, mitral valve, and aortic valves) but with the heart on a slightly different tilt. In addition, the hepatic veins and IVC can be seen to enter the RA by pointing more rightward, beyond the aortic valve level. Make sure the IVC is clear with no thrombi (Edmund, 2004).

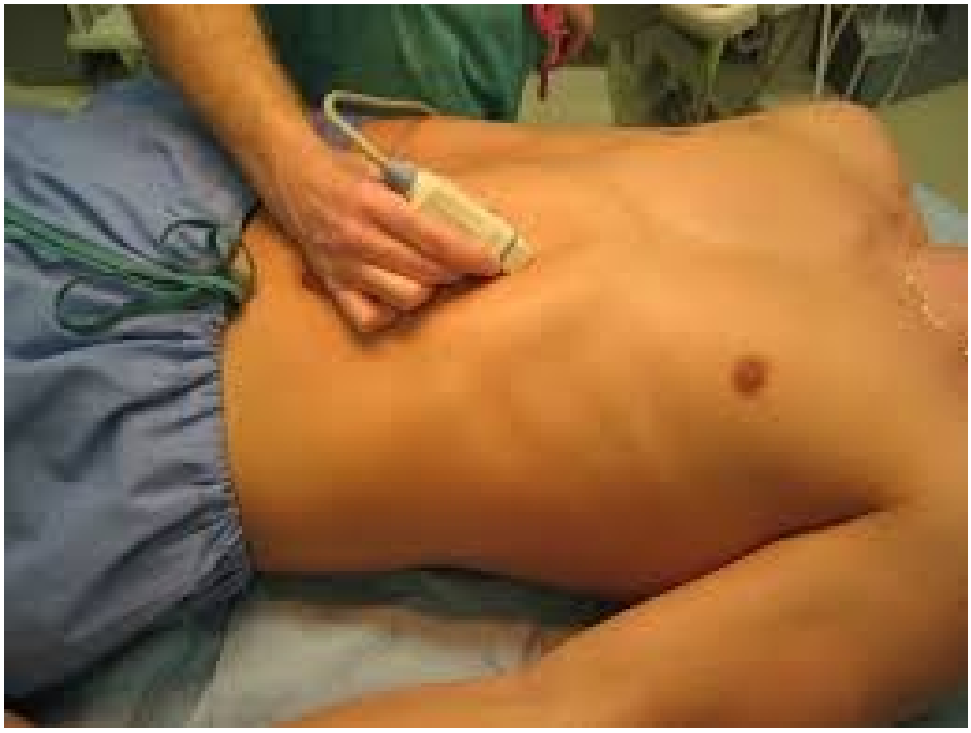


Figure. 2.18 Subxiphoid short axis views.(parvliuk and povalishko, 2013)

Suprasternal view .2.10.5

1. With the patient in the supine position, neck extended place the transducer at the sternoclavicular groove and angle inferior towards the heart. This will visualize the aortic arch and its branches, along with a cross section of the right pulmonary artery. The transducer is oriented towards 12o'clock.
2. For a short axis of the aorta, rotate the transducer 90 degrees clockwise to 3 o'clock. A longitudinal section of the right pulmonary artery may also be seen anterior to the LA (Leeson and Becher, 2007).

NOTE: this view should be used when questions involving the aorta arise, such as in dissection or Marfans syndrome (Tempkin, 1999).

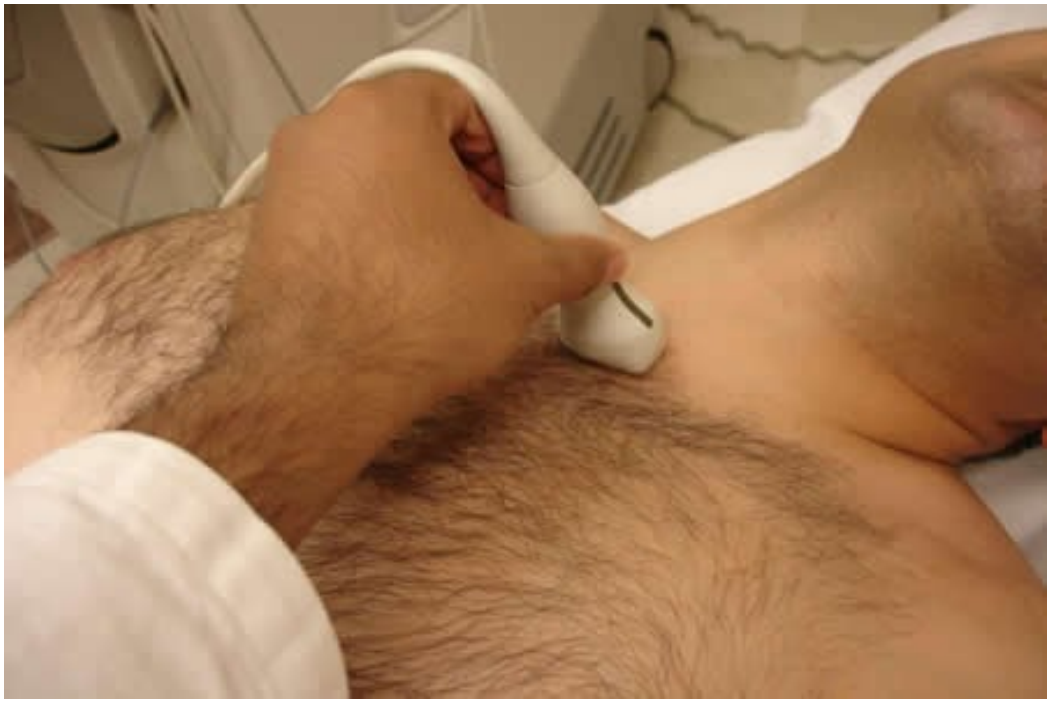


Figure. 2.19 Suprasternal view. (parvliuk and povalishko, 2013)

Sonographic Appearance .2.11

- .The pericardium is most reflective structure and appears almost white
- The papillary muscles and myocardium are medium gray and homogeneous in echotexture (Tempkin, 1999) and (Edmund, 2004).
- The valves are slightly more echogenic than the walls when perpendicular to the ultrasound beam (Leeson and Becher, 2007).

- The area within the chambers and great vessels as well as any other fluid space is anechoic (Edmund, 2004) and(Kerut et al., 2008).

Display of normal Heart patents .2.12

The patient is generally examined in the supine or left lateral semidecubitus position. The cardiac window is usually found between the third to fifth intercostal spaces, slightly to the left of the sternal border. The cardiac window may be considered that area on the anterior chest where the heart is just beneath the skin surface, free of lung interference. With initial high-gain settings we have found it more advantageous to cover a larger area along the sternal border in the search for typical echocardiographic patterns in an effort to determine which intercostal space is the best window. When the transducer is placed along the left sternal border, the examiner should run the transducer up and down (between the third to fifth intercostal spaces) the chest wall to define the pericardial echo with the strongest or loudest echo reflection. After the pericardium is defined, one can search for the mitral and aortic valve patterns and determine which interspace is best for demonstrating the continuity of the cardiac structures. The cardiac sonographer must keep in mind that different body shapes require variations in transverse heart, and thus a slight lateral movement from the sternal border may be needed to record cardiac structures. A thin patient may have a long and slender heart, requiring a lower, more medial transducer position. Barrel chested patients may present with echography difficulties because of the lung absorption interference. It may be necessary to turn these patients completely on their left sides or even prone to eliminate this lung interference. Sometimes the upright or slightly bent-forward position is useful in forcing the heart closer to the anterior chest wall (Solomon, 2007).

The following techniques guidelines for the average patient. In the initial echocardiographic study, moving the transducer freely along the left sterna border until all the cardiac structures are easily identified is a

better practice than restricting it to one interspace. This saves time and gives the examiner a better understanding of cardiac relationships. If there is difficulty examining the patient in the supine position, a semidecubitus position should be used. Sometimes, if the heart is actually very medial, the best study is performed with the patient completely on the left side. If too much lung interference clouds the study, the patient should exhale for as long as possible. This usually gives the examiner enough time to record pieces of a valid study (Sandra et al., 2011).

The gain or power settings are usually increases for the initial search period and then decreases to obtain a clear tracing. The highest gain is used in the area of the left ventricle and mitral valve, with the more anterior structures, such as the aorta, tricuspid, and pulmonary valve, requiring less gain (Sandra et al., 2011).

Echocardiography Normal mitral valve .2.12.1

Echography, the mitral valve is one of the easiest cardiac structures to recognize. The transducer should be directed perpendicular to the patient's chest wall, slightly away from the left sternal border, in approximately the fourth intercostal space. With proper gain settings, the M-mode tracings are often the most sensitive recorder of initial mitral valve motion. The cardiac sonographer may recognize the initial echo of the right ventricular wall, the echo-free cavity of the right ventricular cavity, the anterior and posterior walls of the interventricular cavity, the anterior and posterior walls of the interventricular septum, and, finally, the mitral valve apparatus as shown in the left atrial or left ventricular cavity (depending on transducer angulation). The mitral valve pattern is usually seen 6 to 9 cm from the patient's skin surface. It has the greatest amplitude and excursion and can be unquestionably recognized by its "double" or biphasic kick. This is caused by the initial opening of the valve in ventricular diastole and the atrial contraction at end diastole (Sandra et al., 2011).

Echocardiography Normal Aortic Valve and Left Atrium .2.12.2

To examine the aortic root, semilunar cusps, and left atrial cavity, the transducer should be directed cephalad toward the right shoulder, from the landmark area of the mitral valve. The cardiac sonographer should be able to identify the anterior leaflet of the mitral valve blending with the posterior aortic wall at the same time as the interventricular septum blends into the anterior aortic wall. Often there is a double parallel echo appearance along the anterior and posterior aortic walls, denoting wall thickness. Care should be taken to record both wall echoes to ensure proper measurement of the aortic root dimensions. Adjustment of the near-gain control allows excellent visualization of the anterior wall. The echoes recorded from the aortic root should be parallel, moving anteriorly in systole and posteriorly in diastole (Leeson and Becher, 2007).

As the transducer is angled slightly medial, two of the three semilunar cusps can be visualized. On M-mode tracings, the right coronary cusp is shown anterior and the non-coronary posterior. When seen, the left coronary cusp is shown in the midline between the other two cusps. The onset of systole causes the cusps to open to the full extent of the aortic root. The extreme force of blood through this opening causes fine flutter to occur during systole. As the pressure relents in the ventricle, the cusps begin to drift to a closed position until they are fully closed in diastole (Solomon, 2007).

The chamber posterior to the aortic root is the left atrium, which can recognized by its immobile posterior wall. As one sweeps from the mitral apparatus medially and superiorly, the left ventricular wall blends into the atrioventricular groove and finally into the left atrial wall. Thus the sweep demonstrates good contractility in the left ventricle, with anterior wall motion in systole to the atrioventricular area where the posterior wall starts to move posteriorly in systole, and then to the left atrium, where there is no movement (Solomon, 2007).

Sometimes it is possible to record the left pulmonary vein within the left atrial cavity. This appears as thin, double –walled vessel and can be a problem in determining left atrial measurements. Care should be taken to sweep from the mitral valve to the aortic root and back to the mitral

apparatus several times to note the continuity of the posterior ventricular wall with the left atrial wall to avoid confusion. The pulmonary vein never appears continuous with the left ventricular wall (Solomon, 2007).

Other structures posterior to the left atrial cavity that may lead to confusion in the identification of the left atrial wall are the atrial appendage and descending aorta. The left atrial appendage may appear very prominent posterior to the left atrial wall if there is severe enlargement of the left atrial cavity (especially seen in patients with severe mitral valve disease). Real time evaluation with the transducer in the apical four –chamber position clarifies the atrial appendage as a separate structure. The descending aorta may also be recognized as a parallel pulsating tubular structure posterior to the left atrial cavity. The aorta is not continuous with the left ventricular wall as the left atrial is; thus the cardiac sonographer should be able to distinguish this echo reflection as normal anatomy (Edmund, 2004)

Echocardiography Interventricular septum .2.12.3

The Interventricular septum divides the right ventricle from the left ventricle. As the transducer is angled slightly inferior and lateral to the mitral valve, the septum moves somewhat anteriorly in early systole and posteriorly at the end of systole and early diastole (Leeson and Becher, 2007).

Both sides of the septum should move symmetrically. If they do not, the transducer should be placed more medial on the chest wall or the patient should be rolled into a slightly steeper decubitus position. Echo reflections from the chordate tendineae of the papillary muscle apparatus in the right heart may be mistaken for the right side of the septum, and care should be used to accurately identify the true right side of the septum (Sandra et al., 2011).

The septum thickens in systole at the midportion of the ventricular cavity. The measurement and evaluation of septal thickness and motion should be made at this point. Normal septal thickness should match that of the posterior left ventricular wall and not exceed 1.2 cm (Edmund, 2004).

Echocardiography Left Ventricle .2.12.4

The determination of left ventricular volume and function may be made with a routine M-mode sweep. The patient is generally examined in the left semidecubitus position to best define septal motion and left ventricular posterior wall motion. The anterior leaflet of the mitral valve should first be located and then the beam angled slightly inferior and lateral (toward the left hip) to record the left ventricular chamber. Correct identification of this chamber may be made when both sides of the septum are seen to contract with the posterior heart wall. If the septum is not well defined or does not appear to move well, a more medical placement of the transducer along the sternal border with a lateral angulation may permit better visualization of this structure (Solomon, 2007).

The three layers of the posterior heart wall-endocardium (inner layer), myocardium (middle layer), and epicardium (outer layer)-should be identified separately from the pericardium. Sometimes it is difficult to separate the epicardium from the pericardium until the gain is reduced. The myocardium usually has a fine scattering of echoes throughout its muscular layer. The endocardium may be a more difficult structure to record, since it reflects a very weak echo pattern. Sometimes the multiple chordate tendieae are difficult to separate from the endocardium, and the posterior wall must be carefully evaluated. The chordae are much denser structures than the endocardium. They generally are shown in the systolic segment along the anterior surface of the endocardium. As the ventricle contracts, the endocardial velocity is greater than the chordae tendieae velocity (Solomon, 2007).

Small pieces of the mitral apparatus seen in the left ventricle ensure that the correct dimension is being evaluated. Posterior papillary muscles are shown near the apex of the ventricle. These appear as a dense, fuzzy echo band and make it difficult to evaluate the posterior wall clearly. If the ventricular volume is to be determined, these muscles are a clue that the transducer is directed too far inferior to the desired point of measurement and the cavity size would be underestimated (Sandra et al., 2011).

Echocardiography Right Ventricle .2.12.5

The right ventricle is the most anterior chamber of the heart. Its anterior wall may be demonstrated with proper near-gain settings adjusted so the first moving echo shown after the immobile main bang and chest wall echoes represents the right ventricular wall. If this echo is not clearly defined, Popp has suggested an arbitrary measurement of 0.5 cm from the last nonmoving echo to serve as the right ventricular wall for right ventricular size determination. Most ventricular measurements are made in the supine position and thus must be slightly adjusted if the patient is examined in an upright or decubitus position (Edmund Kenneth Kerut, 2004).

Echocardiography Right Atrium .2.12.6

The right atrium is best seen on the longitudinal, sub-costal, two-dimensional display as the inferior vena cava sternal into. It may also be seen on the parasternal long-axis two-dimensional view as the cardiac sonographer angles the transducer medially from the level of the mitral valve to visualize the right ventricle, tricuspid valve, and right atrial cavity. The apical four-chamber view is another excellent position for evaluating the size of the right atrium (Leeson and Becher, 2007).

Often fine linear echoes may be recorded within the right atrial cavity, which probably represent remnants of the Chiari network (these linear echoes are located near the interatrial septum) and the Eustachian valve (the valve found at the exit of the inferior vena cava) (Sandra et al., 2011).

Echocardiography Normal Tricuspid Valve .2.12.7

The tricuspid valve is not as easily identified as the mitral valve because of its substernal location in most patients. Recordings are easily made if the right ventricle is slightly enlarged or if the heart is recorded the mitral apparatus; the beam should be angled slightly medially, under the sternum, to record the tricuspid valve. It is fairly easy to identify the whipping motion of the anterior valve in systole and early diastole. However, the complete diastolic period reveals the pathologic changes of

stenosis and regurgitation; careful angulation may allow this phase to be recorded. An alternate method of recording the valve is to locate the aortic root. The transducer beam should sweep inferiorly and medially toward the patient's right foot to record the valve leaflet (Edmund, 2004). Sometimes on M-mode scan it may be confusing to differentiate the tricuspid valve from the pulmonary valve. In the normal person the tricuspid valve is always inferior and medial to the aortic root, whereas the pulmonary valve is superior and lateral to the aorta. The other difference is that the tricuspid valve moves anteriorly with atrial contraction and the pulmonary valve dips, posteriorly (Solomon, 2007).

Echocardiography Normal Pulmonary Valve .2.12.8

The pulmonary valve was the last of the four cardiac valves to be adequately visualized by ultrasound. Gramiak and Nanda were the first to document its echographic pattern through the aid of contrast studies. Although it is a semilunar three-cusp valve, only the left or posterior cusp can be adequately demonstrated echocardiographically (Sandra et al., 2011).

A slow sweep from the aortic valve area, laterally and superiorly toward the left shoulder, should allow visualization of the pulmonary valve area. The parallel aortic echoes serve as a landmark in the sweep to the pulmonary valve. The anterior aortic root forms the posterior boundary of the pulmonary valve area. There should be a 2- to 4-cm space beneath the anterior chest wall and in front of this posterior border in which to visualize the pulmonary valve. Gramiak identified these posterior structures as the junction of the right ventricular outflow tract with the pulmonary artery and the atriopulmonary sulcus (with the left atrium posterior) (Sandra et al., 2011).

When this structure complex is identified, small adjustments in beam position and direction usually pass the beam through the left pulmonary valve cusp. The appearance of the cusp is similar to that of the aortic cusp and requires very slight angulations of the beam to demonstrate fully. We have not found it easier to locate this valve in one particular position. Generally we search for the cusp with the patient in a

semidecubitus position. With two dimensional capabilities, the optimal view is generally a high-parasternal short-axis view with a slight angulation of the beam toward the left shoulder (Sandra et al., 2011).

Gramiak described the physiologic parameters as shown on the M-mode echocardiogram. At the beginning of diastole, the pulmonary valve is displaced downward and is represented anteriorly on the ultrasound recording. The low transducer position with upward beam angulation, together with the vertical inclination of the pulmonary ring, results in the examination of the valve from below. All elevations of the pulmonary valve in the stream of flow are represented as posterior movements on the echo. Likewise, downward movements are represented by anterior cusp positions on the trace (Sandra et al., 2011).

Other Methods of Echocardiographic Examination .2.13

In a small percentage of the patients scanned the examiner cannot record adequate information from the conventional left sternal approach. This may be a function of lung interference, an unusual angulation of the cardiac structures, or relational pathology surrounding the cardiac structures. Therefore other useful approaches should be used to obtain the echographic information (Solomon, 2007).

Suprasternal Approach. The suprasternal technique was first described by Goldberg. A special angulated transducer is placed in the suprasternal notch with the beam directed caudate toward the aortic arch. The transducer beam passes through the left brachiocephalic artery, aortic arch, right pulmonary artery, and left atrium. This technique has proven useful in the further detection of aneurismal growth, tumor invasion, and in determining accurate great vessel dimensions (Sandra et al., 2011).

Subxiphoid Approach. Chang first described the subxiphoid approach as an alternative method in the evaluation of cardiac structures obscured by lung tissue. The transducer is directed in a cephalic angulation from the subxiphoid approach. Recording can then be made of the left ventricular wall, mitral valve, and aortic valve. Although measurements cannot be obtained from this tangential approach and compared with “normal” measurements from the semidecubitus approach, this method has

proven a useful technique in ruling out certain cardiac problems such as valvular disease, pericardial effusion, and tumor formation (Leeson and Becher, 2007).

General Evaluation of the Heart with 2D Echocardiography .2.14

The widespread clinic acceptance of the real-life 2D image has tremendously aided the diagnostic results of a typical echocardiographic examination. Improved transducer design, resolution capabilities, focus parameters, gray-scale differentiation, gain control factors, cine loop functions, and other computer capabilities have aided the cardiac sonographer in the attempt to record consistent, high-quality images from the multiple scan planes necessary to obtain a composite image of the cardiac structures. In addition, most two-dimensional transducers have the combined function of imaging and selecting one crystal to perform and M-mode or a Doppler study simultaneously. Thus the addition of these functions has increased the accuracy of the M-mode and Doppler studies. The ability to actually image the cardiac anatomy has also improved the speed with which an M-mode study can be performed. Most equipment has the capability of computing dimensions of various cardiac structures in either two-dimensional or M-mode. More advanced echocardiographic equipment has the added feature of combined pulsed-wave (PW) and continuous-wave (CW) Doppler capabilities, with the further ability to actually steer the CW Doppler beam (Solomon, 2007).

The recent addition of color-flow Doppler has added a new dimension for the cardiac sonographer in detecting in tracardiac shunt flow, mapping out regurgitate pathways, and in determining obstructive flow pathways (Sandra et al., 2011).

The procedures of Two-Dimensional Echocardiography .2.14.1

Nomenclature and Image Orientation. The Committee on Nomenclature and Standards in Two-Dimensional Echocardiography of the American

Society of Echocardiography recommends the following nomenclature and image orientation standards for transducer location: (Leeson and Becher, 2007).

Suprasternal Transducer placed in the suprasternal notch.

Subcostal transducer located near body midline and beneath costal margin.

Apical transducer located over cardiac apex (at the point of maximal impulse) (Edmund, 2004).

Parasternal transducer placed over the area bounded superiorly by left clavicle, medially by sternum, and inferiorly by apical region (Leeson and Becher, 2007).

Imaging planes these planes are described by the manner in which the two- dimensional transducer transects the heart.

Long axis transects heart perpendicular to dorsal and ventral surfaces of body and parallel with long axis of heart (Sandra et al., 2011).

Short axis transects heart perpendicular to dorsal and ventral surfaces of body and perpendicular with long axis of heart.

Four chamber transects heart approximately parallel with dorsal and ventral surfaces of body (Solomon, 2007).

The routine two dimensional examination .2.14.2

A routine two-dimensional examination for the adult and pediatric patient usually begins with the patient in a semileft lateral decubitus position. This position allows the heart to move away from the sternum and closer to the chest wall, thus allowing a better cardiac window (Leeson and Becher, 2007).

Parasternal long –Axis Views. The parasternal long –axis (Hippisley-Cox and Coupland) view should be used first in the ethnographic examination. An attempt should be made to record as many of the cardiac structures as possible (from the base of heart to apex). Generally this is accomplished by placing the transducer slightly to the left of the sternum in about the fourth intercostal space. When the bright echo reflection of the pericardium is noted, the transducer is gradually rotated until a long-axis view of the heart is obtained. If it is not possible to record the entire

long axis on a single scan, the transducer should be gently rocked cephalad to caudad in an “ice-pick” fashion to record all the information from the base to the apex of the heart (Sandra et al., 2011).

The cardiac sonographer should observe the following structures and functions in the PLA view:

- Composite size of the cardiac chambers
- Contractility of the right and left ventricles
- Thickness of the right ventricular wall
- Continuity of the interventricular septum with the anterior wall of the aorta
- Pliability of the atrioventricular and semilunar valves
- Coaptation of the atrioventricular valves
- Presence of increased echoes on the atrioventricular and semilunar valves
- Systolic clearance of the aortic cusps
- Presence of abnormal echo collections in the chambers or attached to the valve orifice
- Presence and movement of chordal-papillary muscle structure
- Thickness of the septum and posterior wall of the left ventricle
- Uniform texture of the endocardium and myocardium
- Size of the aortic root

Respective M-mode tracings should then be made in these areas:

- .Record the aortic root at the level of the cusp opening
- .Record the size of the left atrium
- :Sweep the beam from the aortic root to the mitral valve
- Demonstrate right side of interventricular septum to anterior .aortic wall continuity
- Demonstrate posterior aortic wall to anterior leaflet mitral .valve continuity
- Show transition from left atrial wall to atrioventricular groove .(to posterior wall of the left ventricle (Leeson and Becher, 2007

- Record the anterior leaflet of the mitral valve at the tip of the leaflet.
- Record both leaflets of the mitral valve.
- Record the left ventricle at an area inferior to the papillary muscles.

Parasternal Short-Axis (PSA) View. The transducer should be rotated 90 degrees from the parasternal long-axis view to obtain multiple transverse short-axis views of the heart at particularly these four levels:

- High PSA view to demonstrate the pulmonary valve, right ventricular outflow tract and aorta:
  - Typical sausage-shaped right ventricular outflow tract and pulmonary artery draped anterior to circular aorta
  - Semilunar cusp thickness and mobility
  - Presence of calcification, extraneous echoes, or both in right ventricle or valve areas
  - Pulmonary valve mobility and thickness

Respective M-mode tracings should then be made in these areas:

- Record the mobility of the pulmonary cusps.
- Moderate to high PSA view to demonstrate the right ventricle, tricuspid valve, aortic cusps, coronary arteries, right and left atria:
  - Size of right ventricle and left atrium
  - Presence of mass lesions in right or left atrium
  - Mobility and thickness of tricuspid and aortic valves
  - Continuity of interatrial septum
  - Right ventricular wall thickness
  - Presence of trileaflet aortic valve

Respective M-mode tracings should then be made in these areas:

- Record the right ventricular outflow tract, aorta with cusps, and left atrial size.
- Record the right ventricle and tricuspid valve.
- Mid PSA view to demonstrate the right ventricle, left ventricular outflow tract, and anterior and posterior leaflets of the mitral valve:

- Size of the left ventricular outflow tract
- Size of the septum and posterior wall
- Presence of mass lesions in left or right ventricle
- Mobility and thickness of the mitral valves
- Presence of a flutter on the septum or anterior leaflet of the mitral valve or both
- Systolic apposition of mitral valve leaflets
- Contractility of septum and posterior wall

Respective M-mode tracings should then be made in these areas:

- Record the right ventricle, interventricular septum, anterior leaflet of the mitral valve, and left ventricle.
- Record both leaflets of the mitral valve in the left ventricular cavity
- Low PSA should demonstrate the right ventricle, left ventricle, and papillary muscles (chordal echoes may also be seen):
  - Contractility of septum and posterior wall of the left ventricle
  - Thickness of the septum and posterior wall
  - Size of the left ventricle
  - Presence or absence of mitral thrombus or other mass
  - Presence or absence of pericardial fluid, constriction, or restriction
  - Presence of increased echo density in posterior wall
  - g .Number of papillary muscles and their location within the left ventricular cavity

Respective M-mode tracing should then be made in these areas:

- Record the right ventricle, interventricular septum (chordate tendineae), left ventricle, endocardium, myocardium, epicardium, and pericardium. Reduce gain to show bright pericardial echo reflection.
- Sweep from left ventricle to mitral valve to aorta.

Apical Views. Two apical views are very useful: the four – chamber view and the apical long –axis vies, or two chamber view. The cardiac sonographer should palpate the patient’s chest to detect the point of

maximal impulse (PMI). The transducer should then be directed in a transverse plane at the PMI and angled sharply cephalad to record the four chambers of the heart. If there is too much lung interference, then the proper cardiac window has not been found and care should be taken to adjust the patient's position or the transducer position to adequately see all four chambers of the heart. Many laboratories have found it useful to use a very thick mattress with a large hole cut out of the mattress at the level of the apex of the heart. This allows the transducer more flexibility in recording the four chamber view (Solomon, 2007).

This view is excellent for assessing cardiac contractility, size of cardiac chambers, presence of mass lesions, alignment of atrioventricular valves, coaptation of atrioventricular valves, septal or posterior wall hypertrophy, chordal attachments, and the presence of pericardial effusion. It is not a good view to evaluate the presence of an atrial septal defect, since the beam is parallel to the thin foramen ovale and the septum commonly appears as a defect in this view. The subcostal four-chamber view is much better to evaluate the presence of such a defect (Sandra et al., 2011).

Generally no M-mode tracings are made in the apical views: therefore the cardiac sonographer should observe the following structures:

- Size of the cardiac chambers
- Contractility of right and left ventricles
- Septal and posterior wall thickness, contractility, and continuity
- Coaptation of atrioventricular valves
- Alignment of atrioventricular valves
- Presence of increased echoes on valve apparatus
- Presence of mass or thrombus in cardiac chambers
- Entrance of pulmonary veins into left atrial cavity
- Size of left ventricular outflow tract, signs of obstruction, mobility of aortic cusps and absence of subaortic membrane
- Entrance of inferior and superior vena cava into the right atrium

The apical long-axis view is very useful to evaluate the left ventricular cavity and aortic outflow tract. Once the apical four-chamber view is obtained, the transducers should be rotated 90 degrees to visualize the left ventricle, left atrium, and aorta with cusps. This view permits the cardiac sonographer to evaluate the wall motion of the posterior basal segment of the left ventricle, the anterior wall, and the apex of the left ventricle. It also permits another view of the left ventricle outflow tract, which may be useful in determining aortic cusp motion or the presence of a subvalvular membrane (Leeson and Becher, 2007).

Subcostal View. The subcostal view also has multiple windows in the four-chamber and short-axis planes. Many of the views are only available in the pediatric patient (because of the flexible abdominal muscles).The subcostal four-chamber view is generally a useful view in many adults and may serve as an alternate view if the apical four-chamber view is unobtainable. The transducer should be placed in the subcostal space, and with a moderate amount of pressure, angled steeply toward the patient's left shoulder. The plane of the transducer is transverse to visualize the four chambers of the heart (Leeson and Becher, 2007).

It is usually easy to follow the inferior vena cava into the right atrium of the heart. With careful angulation the interatrial septum may be visualized between the anterior right atrial chamber and posterior left atrial chamber. It usually is more difficult to open up the right ventricular cavity in this view; therefore no size assessment should be made. This view is usually very good to evaluate the presence of pericardial effusion, especially since it surrounds the anterior segment of the right heart (Sandra et al., 2011).

Suprasternal View. In the suprasternal view, the transducer is directed transversely in the patient's suprasternal notch and angled steeply toward the arch of the aorta. This view is only useful if the design of the transducer is small enough to fit well into the suprasternal notch. The patient best prepared if several towels or a pillow is placed under the shoulders in an effort to flex the neck enough to avoid interference with the neck of the transducer and cable. The Patient's head should be

turned to the right, again to avoid interference with the cable. With careful angulation, the cardiac structures visualized are the aortic arch, brachiocephalic vessels, right pulmonary artery, left atrium, and left main bronchus (Sandra et al., 2011)

This view is especially useful in determining supravalvular enlargement of the aorta, coarctation of the aorta, or dissection of the aorta (Leeson and Becher, 2007).

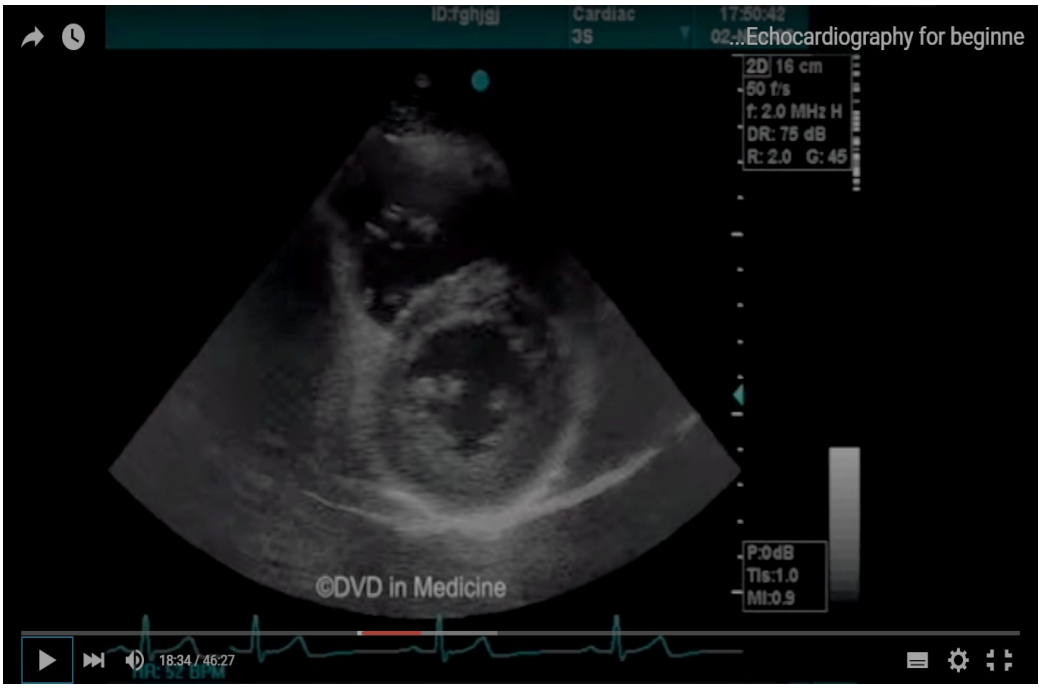


Figure. 2.20 Parasternal short –Axis Views (Leeson and Becher, 2007).

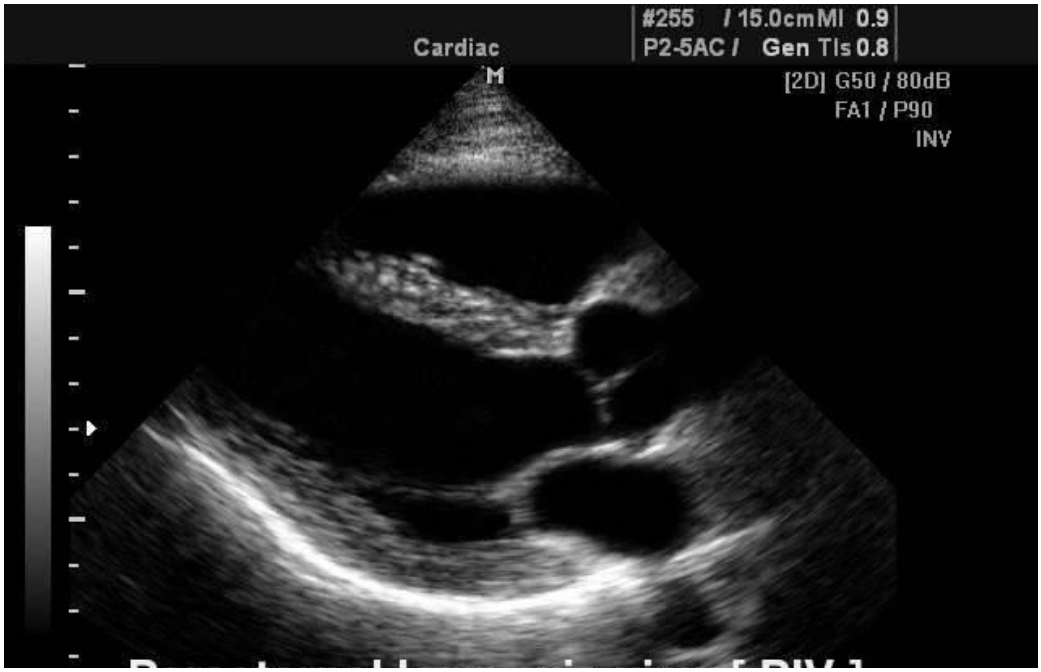


Figure. 2.21 Parasternal long –Axis Views (parvliuk and povalishko, 2013)

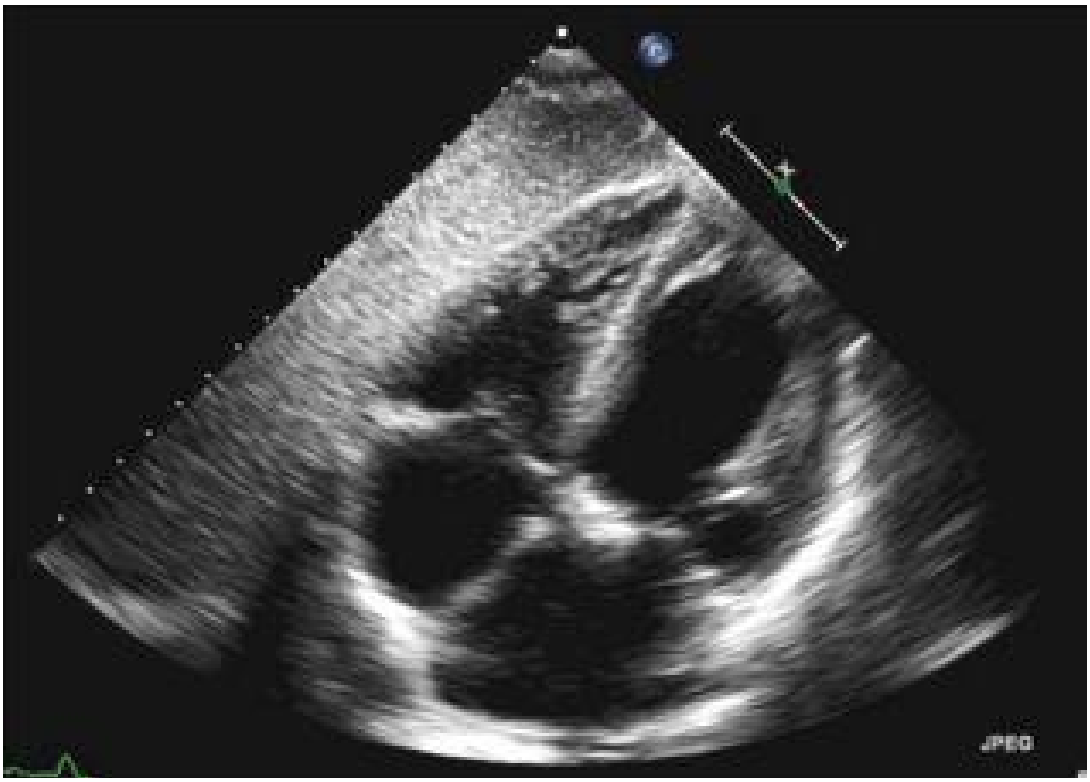


Figure. 2.22 Subcostal view (Solomon, 2007).

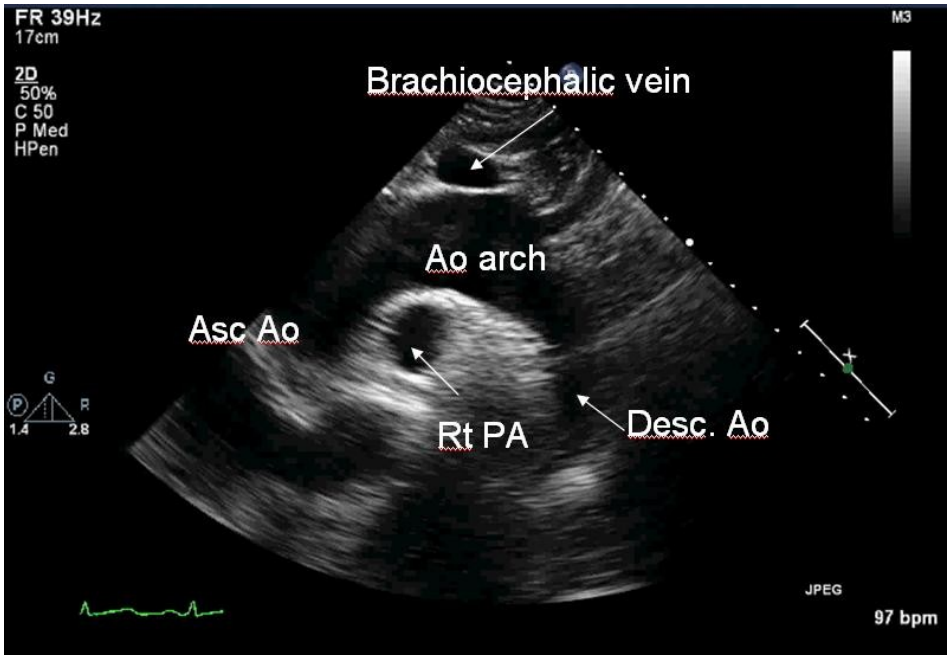


Figure. 2.23 Suprasternal view (Solomon, 2007).

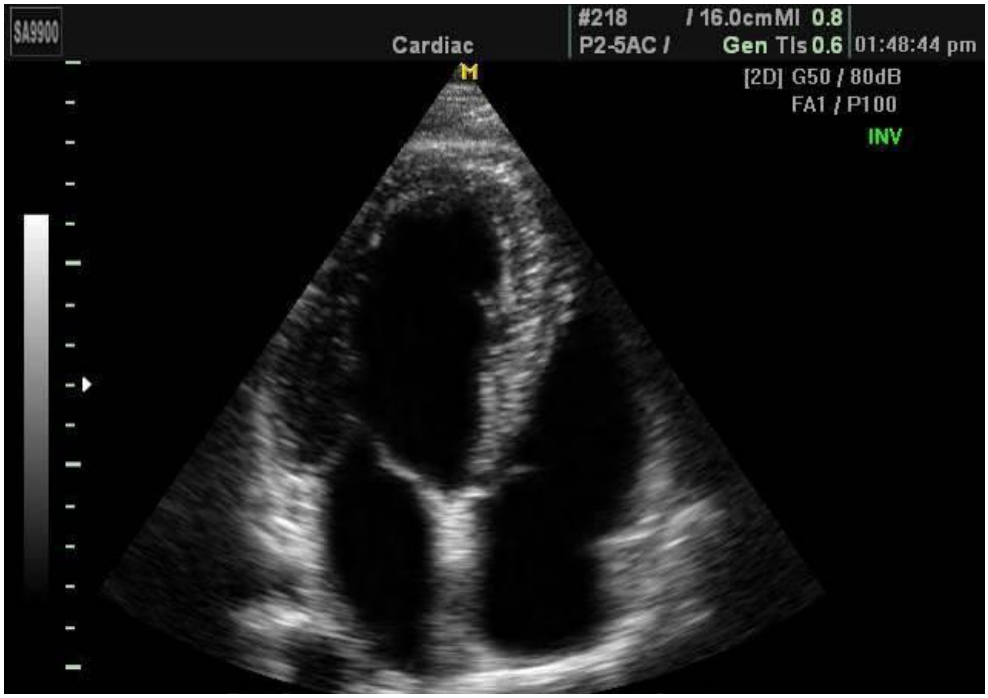


Figure. 2.24 Apical 4 chamber view (parvliuk and povalishko, 2013).

Doppler Applications and technique .2.15

The Doppler Effect, first described by Christian Johann Doppler, is demonstrated on an echocardiogram as red blood cells move from a lower –frequency sound source at rest toward a higher-frequency sound

source. The change in frequency is called the Doppler shift in frequency, or the Doppler frequency (Edmund, 2004).

Normal Doppler Examination and Techniques .2.15.1

Doppler echocardiography has emerged as a valuable noninvasive tool in clinical cardiology to provide hemodynamic information about the function of the cardiac valves and chambers of the heart. When combined with conventional two-dimensional and M-mode echocardiography, Doppler techniques may be focused to produce specific information on the flows of a particular area within the heart. Advances in Doppler technology have made it possible to provide steerable continuous-wave Doppler along with pulsed-wave Doppler. The ability to be qualitative and quantitative in evaluating valvular function, intracardiac shunts, dysfunction of a prosthetic valve, obstruction of a surgically inserted shunt, and to record normal cardiac blood flow patterns has contributed to the understanding and diagnostic capability of the Doppler technique in cardiology. However, to record this information, the cardiac sonographer and physician should master cardiac physiology and hemodynamics. In addition, the operator must clearly understand Doppler principles, artifacts, and pitfalls to produce a quality study. Although cardiac instrumentation is fundamentally similar to imaging echocardiography, the approach to Doppler especially color Doppler, varies considerably from one company to another; a solid understanding of the instrumentation is necessary to produce a valid examination (Sandra et al., 2011).

Normal Doppler Flow Patterns .2.15.2

It is important to understand the relationship between the two-dimensional study and the Doppler-flow study. Real time two-dimensional imaging allows one to assess the cardiac anatomy and function. On the other hand, Doppler-flow analysis allows one to study blood flow rather than cardiac anatomy. The Doppler principle on which this technique is based involves the backscatter of transmitted ultrasonic waves from circulating red blood cells. The difference in frequency between

transmitted and backscattered sound waves (Doppler shift) is used to quantify forward or backward blood-flow velocity (Sandra et al., 2011).

Pulsed-Wave Doppler .2.15.3

A PW transducer is constructed with a single crystal that sends bursts of ultrasound at a rate called the pulsed repetition frequency. Sound waves backscattered from moving red blood cells are received by the transducer during a limited time between transmitted pulses. A time-gating device is then used to select the precise depth from which the returning signal has originated, since the signals return from the heart at different times. The pulsed-wave probe is built into the two-dimensional imaging probe. The particular site undergoing Doppler evaluation is referred to as the sample volume. The sample volume and directional line placement of the beam are moved through use of the trackball. The exact size and location of the sample volume can be adjusted at the area of interest. Some instruments have a fixed sample volume size, whereas others allow the operator to select the size that would be appropriate for the particular study. Velocities under 2 m per sec are usually easily recorded without an alias pattern. However, pulsed Doppler is limited in its ability to record high-velocity patterns. The maximum frequency shift that can be measured by a pulsed Doppler system is called the Nyquist limit, and is one half the pulsed repetition frequency. Velocities that exceed this limit are known to produce an aliasing pattern. Normal cardiac structures do not exceed the Nyquist limit and are very easily measured with the pulsed Doppler system (Solomon, 2007).

Continuous-Wave Doppler .2.15.4

The continuous-wave probe differs from the pulsed-wave probe in that it requires two crystals. If it is part of a two dimensional imaging transducer, the sample direction can sometimes be steered by use of the trackball. Some instruments have a fixed CW sample direction, which means that the area of interest must be aligned with a stationary line on the screen to obtain the best signal. Since the diameter of the probe is smaller, it allows greater flexibility to reach in between small rib

interspaces or to obtain signals from the suprasternal notch. Many patients allow the small independent probe to be angled within their suprasternal notch rather than the bulky imaging probe. The independent probe is often more sensitive and therefore produces better Doppler signals. The audio portion of the Doppler exam becomes a critical factor in this study, since there is no two-dimensional image to guide in the transducer location. Sometimes it is useful to use a combination of both probes to perform the study. Once the proper transducer position is found with the imaging transducer, the angulation and window are marked for proper placement of the CW transducer. The audio sound and spectral wave pattern are then used to guide the correction angulation of the beam for maximum velocity recordings. There is not a particular sample volume site within the CW beam. Velocities are recorded from several points along the linear beam. The techniques has the ability to record maximum velocities without alias patterns. This is especially useful for very-high-velocity patterns (Solomon, 2007).

The Doppler Examination: Techniques and Transducer .2.16 Position

The Doppler examination is generally performed after a complete two-dimensional study of the cardiac structures. During this conventional study, the operator noted structures that may need special attention during the Doppler examination (e.g., a redundant mitral valve leaflet may indicate the need to search for mitral regurgitation). Throughout the Doppler study, various patient positions and transducer rotations are necessary to place the sample volume parallel to blood flow. The patient should be forewarned about the audio sounds produced by the Doppler signal because some find them quite alarming (Sandra et al., 2011)

Examination the Normal Cardiac Color -Flow .2.17

The color-flow mapping (CFM) examination is generally performed after the conventional two-dimensional examination. The advantage of CFM is its ability to rapidly investigate flow direction and movement within the cardiac chambers. Flow toward the transducer is recorded in red, whereas

flow away from the transducer is blue. As the velocities increase, the flow pattern in the variance mode turns from a red color to various shades of red, orange, and yellow before it aliases. Likewise the blue color turns to various shades of blue, turquoise, and green before it aliases. Depending on the location of the transducer the flow signals from various structures within the heart appear as different colors. An understanding of cardiac hemodynamics helps the examiner understand the flow patterns. Although normal cardiac flows are difficult to accurately time during the CFM examination because of its slow frame rate, the use of color M-mode (with a faster frame rate) allows one to precisely determine specific cardiac events in correlation with the ECG. The color M-mode is made in the same manner as a conventional M-mode study. The cursor is placed through the area of interest and the flow is evaluated using an autocorrelation technique. The operator must thoroughly understand the color instrument settings to produce a high-quality image. Familiarity with the color-flow maps provided in the software of the equipment is necessary to understand the alias pattern and turbulent flow parameters (Solomon, 2007)

The CFM examination is generally performed in the same planes used for conventional Doppler examination. How-ever, to provide a basic understanding of the color Doppler image, we first describe flow pattern with the conventional two-dimensional view (Sandra et al., 2011).

Table 2.1: Reference limits and values and partition values of left ventricular

	Women				Men			
	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal
Left Ventricle size	3.9–5.3	5.4-5.7	5.8-6.1	≥6.2	4.2-5.9	6.0-6.3	6.4-6.8	≥6.9
IVS thickness	0.6-0.9	1.0-1.2	1.3-1.5	≥1.6	0.6-1.0	1.1-1.3	1.4-1.6	≥1.7
Posterior wall thickness	0.6-0.9	1.0-1.2	1.3-1.5	≥1.6	0.6-1.0	1.1-1.3	1.4-1.6	≥1.7
Ejection Fraction	≥55	45-54	30-44	<30	≥55	45-54	30-44	<30

Table 2.2: Normal Individuals valves velocity (m/sec)

	Children	Adults

Mitral	0.8-1.3	0.6-1.3
Tricuspid	0.5-0.8	0.3-0.7
Pulmonary	0.7-1.1	0.6-0.9
Aorta	1.2-1.8	1.0-1.7

Previous studies .2.18

In the realm of echocardiography of heart wall disease among adult type II diabetic patients, minimum authors and ultrasound practitioners have wrote about. First study by Rajesh et al. (2002) was studied (Rajput et al., 2002): Diabetes mellitus is a chronic progressive metabolic disease. It involves myocardium at a relatively early stage even before clinical manifestation(s) become obvious. The present study was undertaken to assess cardiac functions by echocardiography and Doppler in patients of NIDDM before and after control of hyperglycemia. He found that; included thirty patients of uncomplicated type-2 diabetes mellitus (duration > 1 year) and thirty, age and sex matched, healthy subjects. Pre-treatment blood sugar, fasting (182.46 ± 33.92 mg %) and post-prandial (245.76 ± 37.87 mg %), was significantly higher than the post-treatment values (101.93 ± 10.17 mg % and 152.75 ± 15.42 mg% respectively). Systolic functions of LV were within normal range in all patients. Diastolic dysfunction of LV was very common and was detected in 63% of patients (A/E > 1.0). None of the control subjects had systolic or diastolic dysfunction. Diastolic dysfunction persisted even after control of hyperglycemia over three-month duration. It is suggested that all patients of NIDDM should be routinely and repeatedly subjected to 2D-echocardiography and Doppler assessment of cardiac functions in long term management of this metabolic disease. Long-term glycemic control

may result in decrease/ reversal of diastolic dysfunction or development of overt cardiomyopathy.

Fadia et al. (2012) (Alwan, 2014) : they reported that the Diabetes mellitus (DM) causes damaging effects on the cardiac function; these effects can be observed on the diastolic performance of the heart reflected on the change in transmitral blood velocity, the cardiac wall and septum thickness. The group of study involved 97 patients (35 male and 62 female of average age of 56.2 ±10.755) of type 2 diabetes mellitus (DM), they were investigated for their left ventricle performance and compared with 51 normal individuals “the control group” (20 male and 31 female of average age of 41.4 ± 13.196). Their results reveal differences in these parameters for patients group relative to controls, in IVRT, ET, E, A, E/A, EF%, IMP, LVIDs, PWTd and IVSTd all are strongly significant with p value <0.001and for FS% p value = 0.0029 except for IVCT the change was 9.342% with p value 0.188 and the change in LVIDd -3.586%, p value 0.052 were not significant. The Conclusion: Diabetes mellitus can cause a deleterious effect on the myocardium. The effect causes impairment in the cardiac diastolic performance and muscle contractility caused by the damage inflicted by hyperglycemia (high blood sugar). Also results show that IMP is increased in type 2 DM patients. This increase may be an early sign of diabetic cardiomyopathy in diabetic patients.

Boonman et al (2012) (Winter et al., 2012); they studied and assessed the prevalence of (unknown) heart failure and left ventricular dysfunction in older patients with type 2 diabetes. He founded in his Results among of 581 patients studied, 161 (27.7%; 95% CI 24.1%, 31.4%) were found to have previously unknown heart failure: 28 (4.8%; 95% CI 3.1%, 6.6%) with reduced ejection fraction, and 133 (22.9%; 95% CI 19.5%, 26.3%) with preserved ejection fraction. The prevalence of heart failure increased steeply with age. Heart failure with preserved ejection fraction was more common in women. Left ventricular dysfunction was diagnosed in 150 patients (25.8%; 95% CI 22.3%, 29.4%); 146 (25.1%; 95% CI 21.6%, 28.7%) had diastolic dysfunction.

Zhi et al. (2003) (Fang et al., 2003) studied echocardiographic detection of early diabetic myocardial disease, he founded that all patient groups (DM, DH, LVH) showed reduced systolic function compared with controls, evidenced by lower peak strain ($p < 0.001$) and strain rate ($p = 0.005$). Calibrated integrated backscatter IB, signifying myocardial reflectivity, was greater in each patient group than in controls ($p < 0.05$). Peak strain and strain rate were significantly lower in the DH group than in those in the DM alone ($p < 0.03$) or LVH alone ($p = 0.01$) groups.

Richard et al., (1999) (Devereux et al., 2000) studied impact of diabetes on cardiac structure and function the strong heart study he founded that echocardiography was used in the Strong Heart Study, a study of cardiovascular disease in American Indians, to compare LV measurements between 1810 participants with DM and 944 with normal glucose tolerance. Participants with DM were older (mean age, 60 versus 59 years), had higher BMI (32.4 versus 28.9 kg/m2) and systolic blood pressure (133 versus 124 mm Hg), and were more likely to be female, to be on antihypertensive treatment, and to live in Arizona (all P,0.001). In analyses adjusted for covariates, women and men with DM had higher LV mass and wall thicknesses and lower LV fractional shortening, midwall shortening, and stress-corrected midwall shortening (all P, 0.002). Pulse pressure/stroke volume, a measure of arterial stiffness, was higher in participants with DM (P, 0.001 independent of confounders).

Isabelle et al., (2015) (Pham et al., 2015) studied the evidence for a specific diabetic cardiomyopathy; an observational retrospective echocardiographic study; the aim was to assess the prevalence of subclinical diabetic cardiomyopathy, occurring among diabetic patients without hypertension or coronary artery disease (CAD) They examined 656 asymptomatic patients with type 2 diabetes for 14 ± 8 years (359men, 59.7 ± 8.7 years old, HbA1c 8.7 ±2.1%) and at least one cardiovascular risk factor had a cardiac echography at rest, a stress cardiac scintigraphy to screen for silent myocardial ischemia (SMI), and, in case of SMI, a coronary angiography to screen for silent CAD. They founded that SMI was diagnosed in 206 patients, and 71 of them had

CAD. In the 157 patients without hypertension or CAD, left ventricular hypertrophy (LVH: 24.1%) was the most frequent abnormality, followed by left ventricular dilation (8.6%), hypokinesia (5.3%), and systolic dysfunction (3.8%). SMI was independently associated with hypokinesia (odds ratio 14.7 [2.7– 81.7], $p < 0.01$) and systolic dysfunction (OR 114.6 [1.7–7907], $p < 0.01$), while HbA1c (OR 1.9 [1.1–3.2], $p < 0.05$) and body mass index (OR 1.6 [1.1–2.4], $p < 0.05$) were associated with systolic dysfunction. LVH was more prevalent among hypertensive patients and hypokinesia in the patients with CAD. Conclusion. In asymptomatic type 2 diabetic patients, diabetic cardiomyopathy is highly prevalent and is predominantly characterized by LVH. SMI, obesity, and poor glycemic control contribute to structural and functional LV abnormalities.

Peter et al., (2015) (Blomstrand et al., 2015) studied the Left ventricular diastolic function, assessed by echocardiography and tissue Doppler imaging, is a strong predictor of cardiovascular events, superior to global left ventricular longitudinal strain, in patients with type 2 diabetes. They founded that, four hundred and six patients had adequate visualization of the left ventricle (≥15 segments) to allow calculation of global strain. In all, 19 major cardiovascular events were documented during the follow-up period, including 12 myocardial infarctions (1 fatal) and 7 strokes (1 fatal). Another 17 patients died during the follow-up period, 8 because of cancer, 3 of trauma, and 6 related to other reasons. Seven patients were hospitalized for congestive heart failure. The majority of the patients had preserved left ventricular systolic function, 51 (13%) had a GLS of less than 215% and 11 (3%) of less than 213%. Eighty-four patients had an estimated glomerular filtration rate of, 60 mL/min/1.73 m2. A higher E/e ratio was significantly associated with cardiovascular events [hazards ratio (HR) 1.12; 95% confidence interval (CI) 1.06–1.18, P < 0.001]. In a multivariate Cox proportional hazard regression analysis, we confirmed that the E/e′ ratio was associated with major cardiovascular events after adjusting for age, sex, medical history, blood chemistry, LVEF, GLS, and pulse pressure (pp). ROC analyses revealed that E/e′ and HbA1c were the strongest predictors followed by LVEF and GLS. To examine the

incremental prognostic value of the risk factors, we performed a nested Cox proportional hazard regression analysis. The addition of E/e′, pp, and LVEF to HbA1c significantly increased the predictive value of the model, but GLS did not improve it further. A total of 139 patients (34%) had an E/e′ ratio≤15. These patients were predominately female, had higher body mass index (BMI), lower GLS, and a higher aortic pulse wave velocity compared with patients with an E/e′ ratio ≤15. An elevated E/e′ ratio, defined as≤15, was also predictive of major cardiovascular events using a Kaplan–Meyer analysis. The cumulative probability of the development of an event during the follow-up period for patients with an E/e′ ratio .15 was 8.6% compared with 2.6% for patients with an E/e′ ratio ≤15, P = 0.021. In a multivariate Cox proportional hazard regression analysis, we determined that an elevated E/e′ ratio of .15 was associated with cardiovascular events after adjusting for age and sex (HR 3.05; 95% CI 1.18–7.85, P = 0.011).

Stefano et al., (2012) (Bonapace et al., 2012) the aim of the study, nonalcoholic fatty liver disease is associated with left ventricular diastolic dysfunction in patients with type 2 diabetes they studied 50 consecutive type 2 diabetic individuals without a history of ischemic heart disease, hepatic diseases, or excessive alcohol consumption, in whom NAFLD was diagnosed by ultrasonography. A tissue Doppler echocardiography with myocardial strain measurement was performed in all patients. They founded thirty-two patients (64%) had NAFLD, and when compared with the other 18 patients, age, sex, BMI, waist circumference, hypertension, smoking, diabetes duration, microvascular complication status, and medication use were not significantly different. In addition, the left ventricular (Lamberts et al.) mass and volumes, ejection fraction, systemic vascular resistance, arterial elasticity, and compliance were also not different. NAFLD patients had lower e′ (8.26 + 1.5 vs. 9.96 + 1.9 cm/s, P < 0.005) tissue velocity, higher E-to- e′ ratio (7.90 + 1.3 vs. 5.59 + 1.1, P , 0.0001), a higher time constant of isovolumic relaxation (43.1+10.1 vs. 33.2+12.9 ms, P,0.01), higher LV–end diastolic pressure (EDP) (16.5 + 1.1 vs. 15.1 + 1.0 mmHg, P < 0.0001), and higher LV

EDP/end diastolic volume (0.20+0.03 vs. 0.18+0.02mmHg, P<0.05) than those without steatosis. Among the measurements of LV global longitudinal strain and strain rate, those with NAFLD also had higher All of these differences remained significant after adjustment for hypertension and other cardiometabolic risk factors.

CHAPTER THREE

Materials and Methods

Chapter Three

Materials and Methods .3

:Study design and population .3.1

This study is a descriptive cross-sectional study was carried out in Echocardiography room recruiting patients from wards and outpatient clinics of ElRibat Teaching hospital in Khartoum. Study population comprises a total of 243 adults patient who underwent cardiac ultrasound scanning were enrolled in this descriptive study, Sudanese participants were included in this study.

:Instrumentation Information .3.2

Echocardiography studies were performed using SIEMENS (ACUSON CV70™ Cardiovascular System Germany) with high frequency probe (2 to 3.5 MHz). (Manufactured date of February 2000), convex face with small footprint for "peeking" in between rib interspaces. Printing facility issued through the ultrasound-digital graphic-printer (serial number of 3-619-GBI-01 made by Sony Corporation- Japan), 100 V; 1.5 A; and 50/60 Hz. 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 the surface of the patient. This fluid is called an acoustic coupling agent, often referred to as "gel", the coupling agent is best applied using a squeeze bottle, from which it can be squirted onto 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 the coupling agent. If there is an open wound, a 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 Procedures of Heart echocardiography .3.3

Transthoracic echocardiography was performed to all patients. The examination was performed in 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 lingual of the left lung out of the way, and opens the intercostal 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 over the chest to provide a detailed view of the heart and its structures. At least 4 separate standard transducer positions which allow for different portions of the heart to be visualized in detail.

The standard positions were used:

Subxiphoid 4-chamber view .3.3.1

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 view the heart.

Parasternal long-axis (Hippisley-Cox and Coupland) .3.3.2 view

The probe should be placed in the parasternal fourth or fifth intercostal space with the transducer indicator directed pointed to the patient's right shoulder, This allows for typical identification of the right ventricle, left atrium, left ventricle, aortic valve, aortic root, aortic outflow tract, and surrounding pericardium.

Parasternal short-axis view .3.3.3

From the PLA position, rotate the probe clockwise 90° 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 to the right ventricle and appears circular.

Apical 4-chamber view .3.3.4

If possible, have the patient raise the left arm up over his or her head to try and spread the ribs. Palpate for the cardiac point of maximal impulse (PMI) and place the probe there with the indicator pointed toward the left axilla and the probe in a coronal plane relative to the heart, as shown below, aimed toward the base of the heart. This allows for identification of the left ventricle, right ventricle, left atrium, right atrium, and pericardium. Direct the transducer-probe up toward the base of the heart. If the probe is directed anteriorly, the left ventricular outflow tract and aortic valve can often be seen; this is known as an apical 5-chamber view.

Septum thickness was measured using the parasternal long-axis view. M-mode measurements were made according to the recommendations of the American Society of Echocardiography. Thus, the line of measuring was perpendicular to the endocardium of the septum and the posterior wall. With the patient in the recumbent position, the transducer was placed in the fourth intercostal space at the left sterna border and directed posteriorly, laterally, and inferiorly to obtain a group of strong echoes from the posterior left ventricular wall.

Figure.3.1 SIEMENS Cardiovascular System (ACUSON CV70™)

Figure.3.2 Phased array probe 2.5 MHz

:Data collection .3.4

Structured questionnaires were administered and physical examinations were done in all cases. Information was obtained about demographic factors, socioeconomic status of the residential neighborhood, drug described, risk factors, and personal and family history of diabetes history. Both weight and height were measured with standardized protocols. The sonographic data was obtained through direct ultrasound scanning of heart.

:Statistical analysis .3.5

Data were initially summarized into means, standard deviations (SD); mean ±SD and percentages in a form of comparison tables and graphs. Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS, USA) version 22 for windows and (P-value) was used for significance, the result is Significant $P < 0.05$. The smaller the P-value obtained the more significant are the results.

:Ethical considerations .3.6

Special consideration was given to the right of confidentiality and anonymity for all participants. Anonymity was achieved by using number

for each participant to provide link between the collected information and the participants.

In addition confidentiality was obtained by making the collected data accessible only to the researcher and the supervisor. Justice and human dignity was 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

Chapter Four

Results

Table 4.1: Gender and diabetes mellitus status of the participants

Gender * Diabetes mellitus status Cross tabulation

gender		Diabetes mellitus status		
		Non-Diabetes	Diabetes	Total
Male	Count	71	47	118
	Total %	29.2%	19.3%	48.6%
Female	Count	59	66	125
	Total %	24.3%	27.2%	51.4%
	Count	130	113	243
	Total %	53.5%	46.5	100.0%

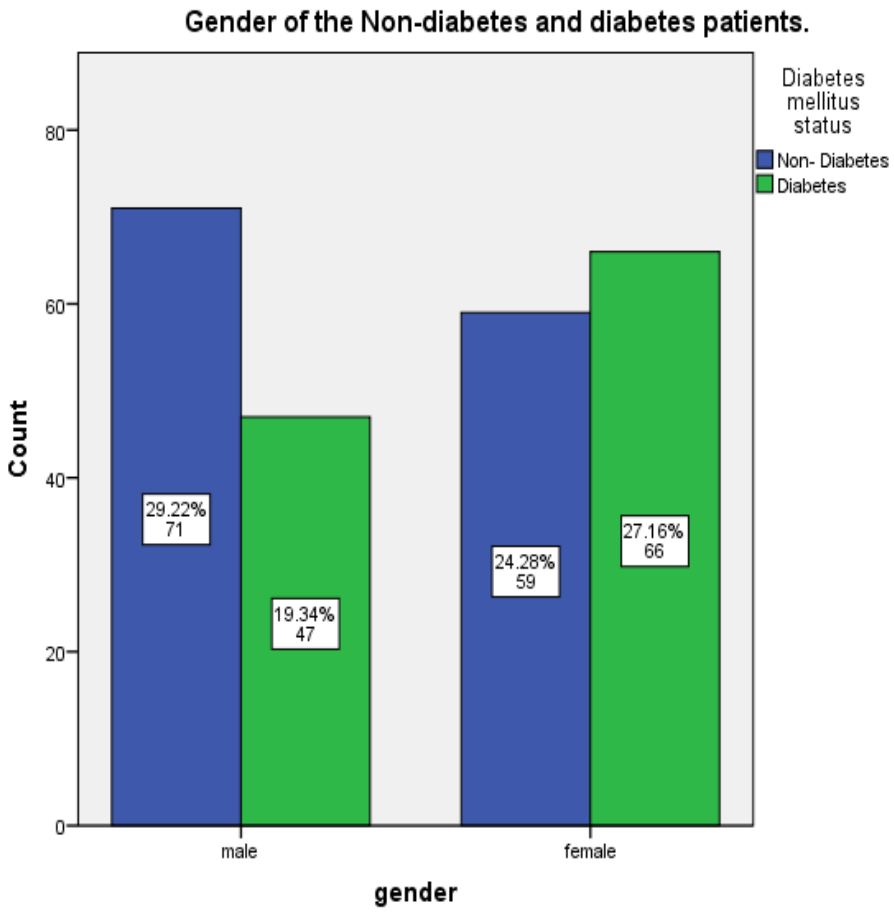


Figure 4.1 Gender and diabetes status of the patients.

Table 4.2: Distribution of age groups among gender Diabetes mellitus status * Age groups * gender Cross tabulation

Age (years)			Rage	Mean	±SD
			30-85	55.76	13.632

gender			Age groups					Total
			from 30 to 40	from 40 to 50	from 50 to 60	from 60 to 70	from 70 to 85	
male Count	Diabetes mellitus status	Non-Diabetes	17	5	8	19	22	71
		Diabetes	1	14	8	15	9	47
	Total		18	19	16	34	31	118
Female Count	Diabetes mellitus status	Non-Diabetes	17	14	10	11	7	59
		Diabetes	3	11	28	14	10	66
	Total		20	25	38	25	17	125
Total	Diabetes mellitus status	Non-Diabetes	34	19	18	30	29	130
		Percentage	14%	7.82%	7.4%	12.34%	11.93%	53.50%
		Diabetes	4	25	36	29	19	113
		Percentage	1.64%	10.28%	14.81%	11.93%	7.82%	46.50%
	Total		38	44	54	59	48	243
			15.64%	18.1%	22.2%	24.27%	19.75%	100%

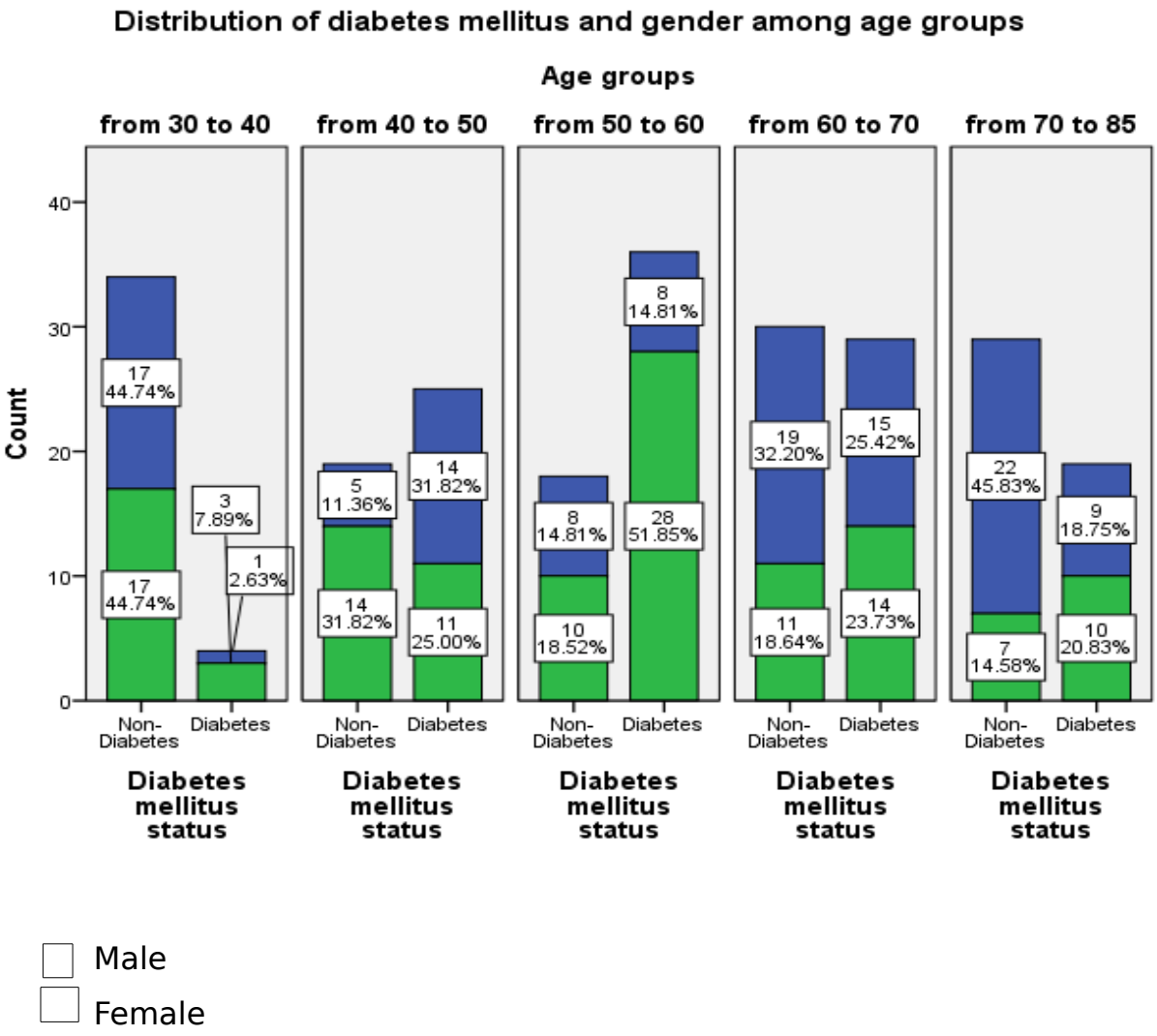


Figure .4.2 Distribution of diabetes mellitus and gender among age groups

Table 4.3: Body Mass Index among Diabetes mellitus status and gender

Diabetes mellitus status * dist. of body Mass index * gender Cross tabulation

Count

Diabetes mellitus status	Mean	N	Std. Deviation
Non diabetes	23.19244	130	4.663368
diabetes	25.38572	113	5.419364
Total	24.21236	243	5.136788

gender			Dist. of body Mass index				Total
			Loss of weight	Normal weight	Overweight	Obese	
Male Count	Diabetes mellitus status	Non diabetes	13	45	10	3	71
		diabetes	1	36	9	1	47
	Total		14	81	19	4	118
Female Count	Diabetes mellitus status	Non diabetes	5	28	16	10	59
		diabetes	2	28	19	17	66
	Total		7	56	35	27	125
Total	Diabetes mellitus status	Non diabetes	18	73	26	13	130
		diabetes	3	64	28	18	113
	Total		21	137	54	31	243

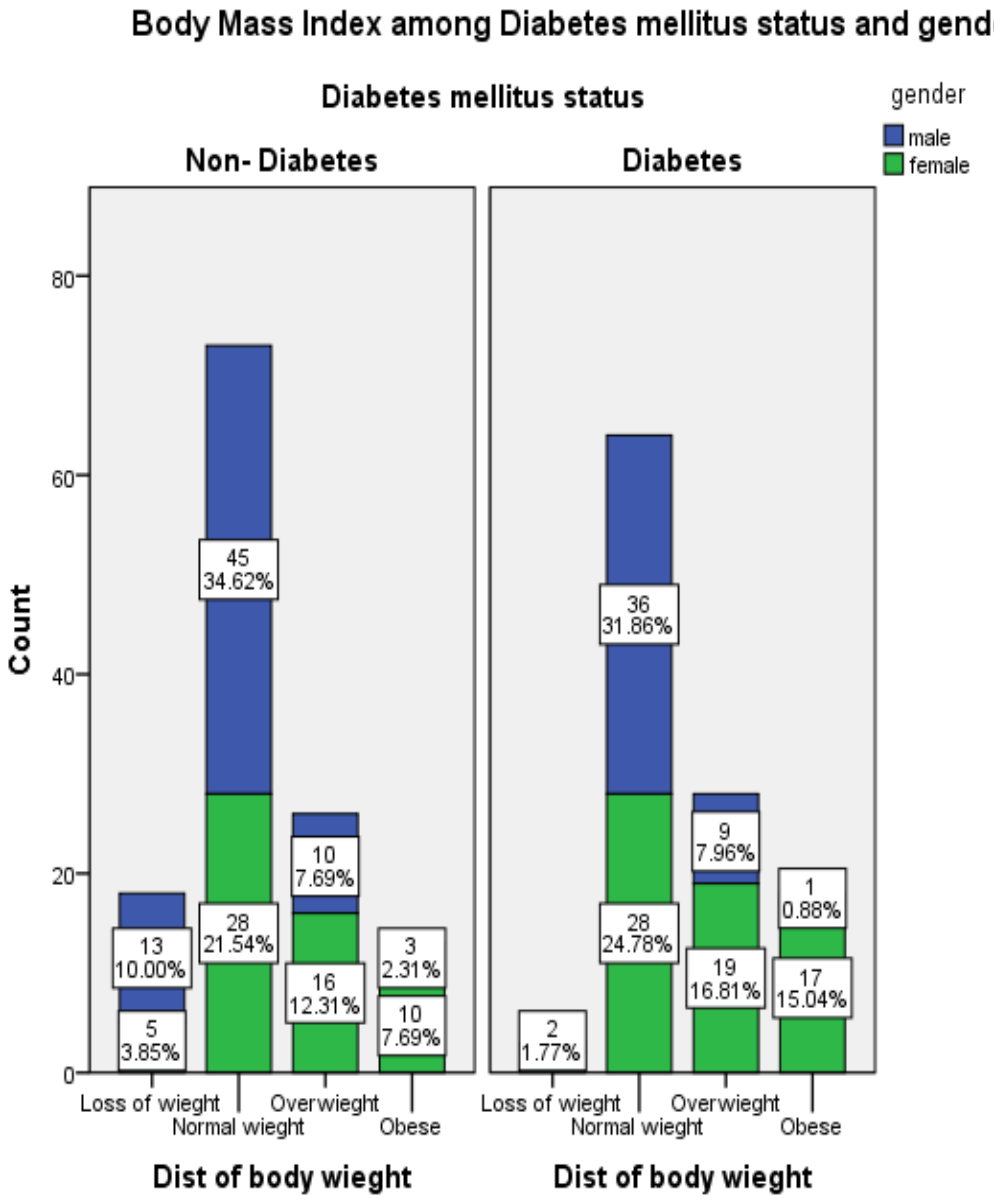


Figure: 4.3: Distribution Body Mass Index among Diabetes mellitus status and gender

Table 4.4: gender * past medical history * Diabetes mellitus status

gender * past medical history * Diabetes mellitus status Cross tabulation

			Count						
			past medical history						Total
Diabetes mellitus status			TB	cancer	orthopedic surgery	cholecystectomy	urological disease	medical checkup	
Non-Diabetes	gender	male	1	1	4	4	4	57	71
		femal e	0	2	1	0	1	55	59
	Total		1	3	5	4	5	112	130
Diabetes	gender	male		1	0	2	3	41	47
		femal e		0	3	1	0	62	66
	Total			1	3	3	3	103	113
Total	gender	male	1	2	4	6	7	98	118
		femal e	0	2	4	1	1	117	125
	Total		1	4	8	7	8	215	243
	Percentage		0.41	1.64	3.29	2.88	3.29	88.48	100

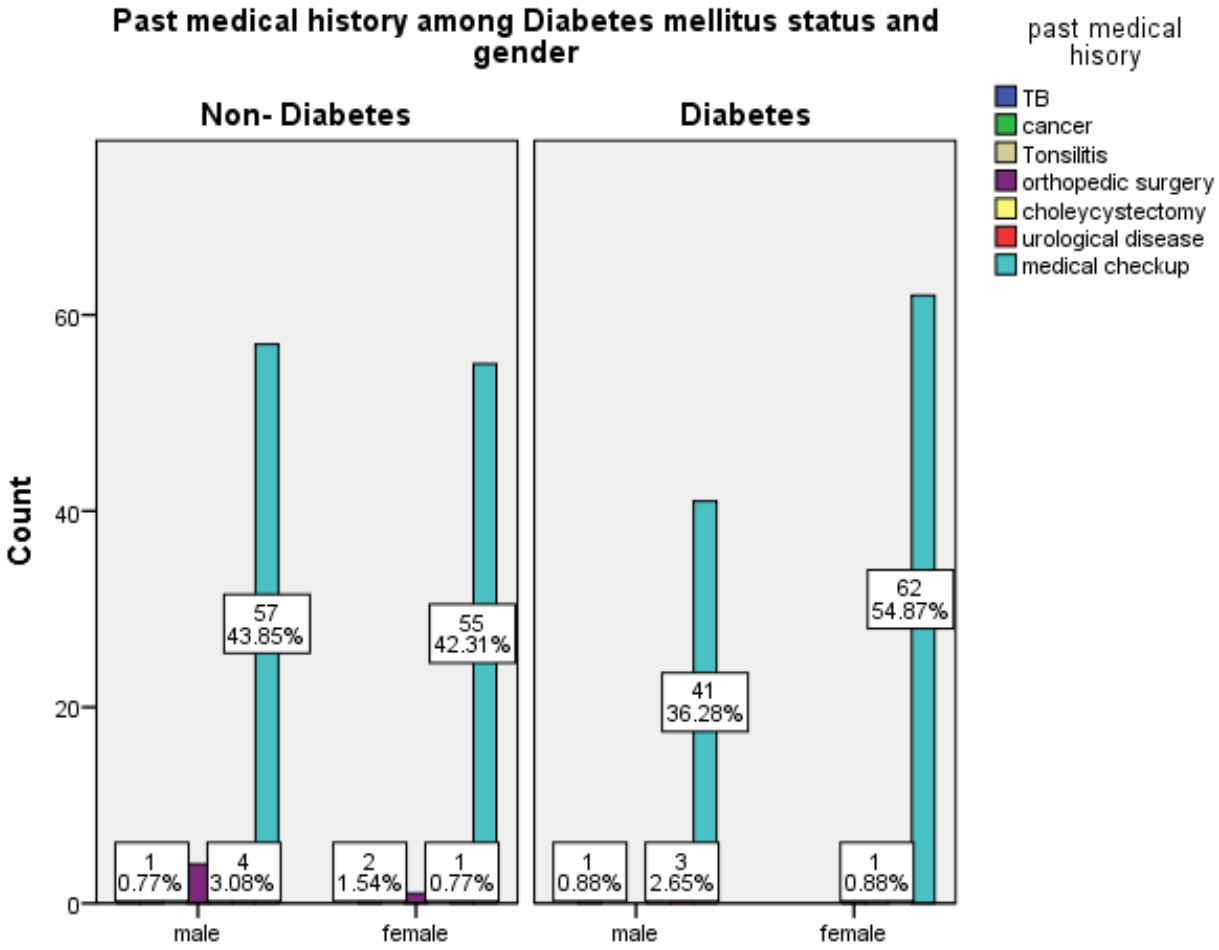


Figure: 4.4 Distribution of past medical history among the diabetes status

Table4.5: frequency distribution of job status

		Frequency	Percent
Valid	house wife	103	42.5
	student	4	1.6
	Police	79	32.5
	officer	3	1.2
	Workers	13	5.3
	retired	41	16.9
	Total	243	100.0

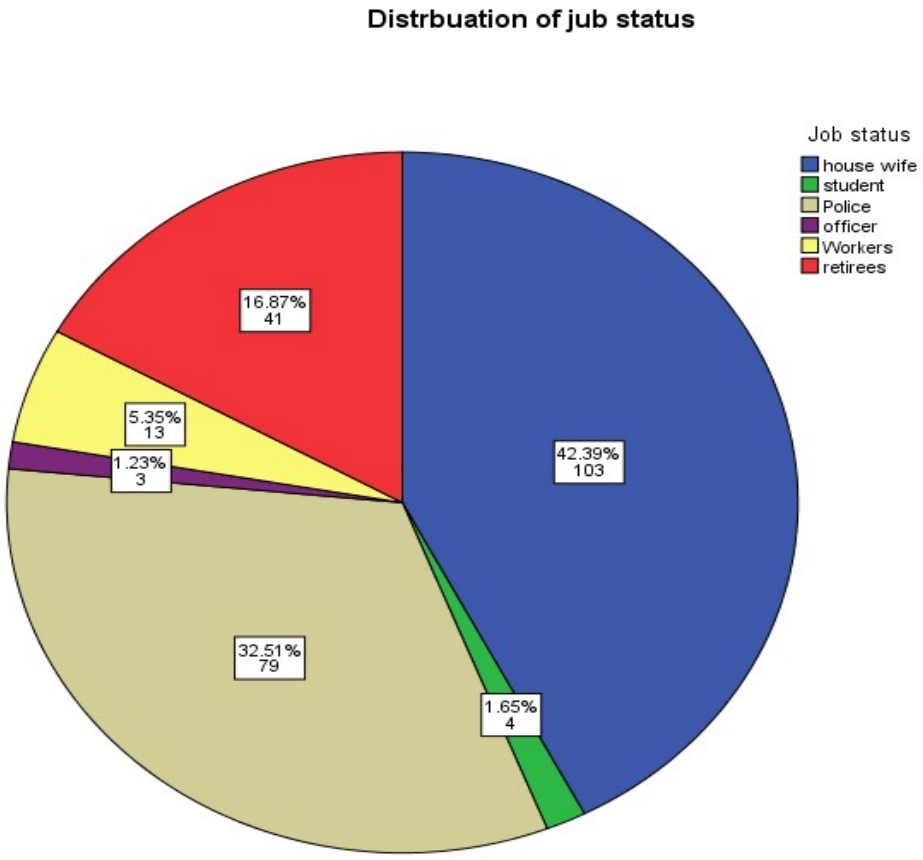


Figure 4.5*:* Frequency distribution of job status

Table4.6: frequency distribution of Mitral valve conditions

Valid	Non diabetes	diabetes	Frequency	Percent
normal	95	86	181	74.5
degenerative changes	5	13	18	7.4
stenosis	1	0	1	.4
regurgitation	3	4	7	2.9
degenerative and regurgitation	20	7	27	11.1
stenosis and regurgitation	6	3	9	3.7
Total	130	113	243	100.0

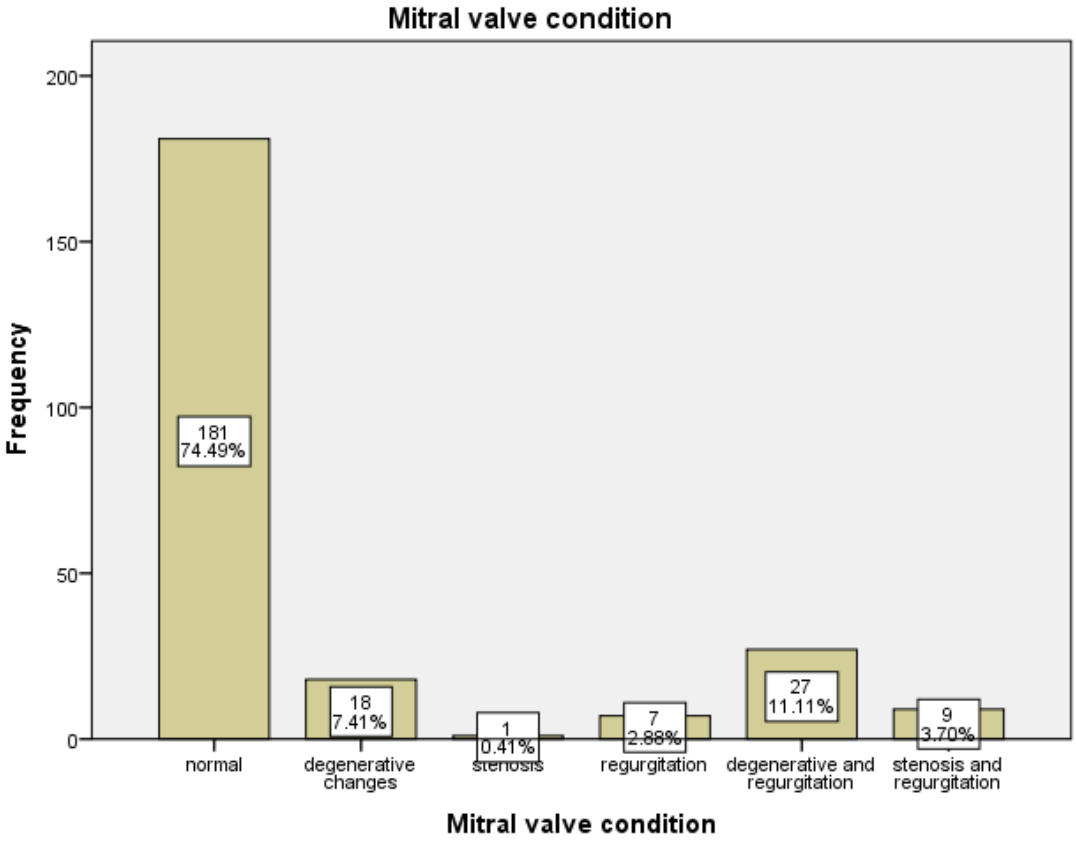


Figure 4.6: Frequency distribution of Mitral valve Condition

Table4.7: frequency distribution of Aortic valve conditions

Valid	Non diabetes	diabetes	Frequency	Percent
Normal	106	96	202	83.1
degenerative change	7	7	14	5.8
stenosis	1	0	1	.4
regurgitation	2	2	4	1.6
degenerative and regurgitate	13	8	21	8.6
stenosis and regurgitation	1	0	1	.4
Total	130	113	243	100.0

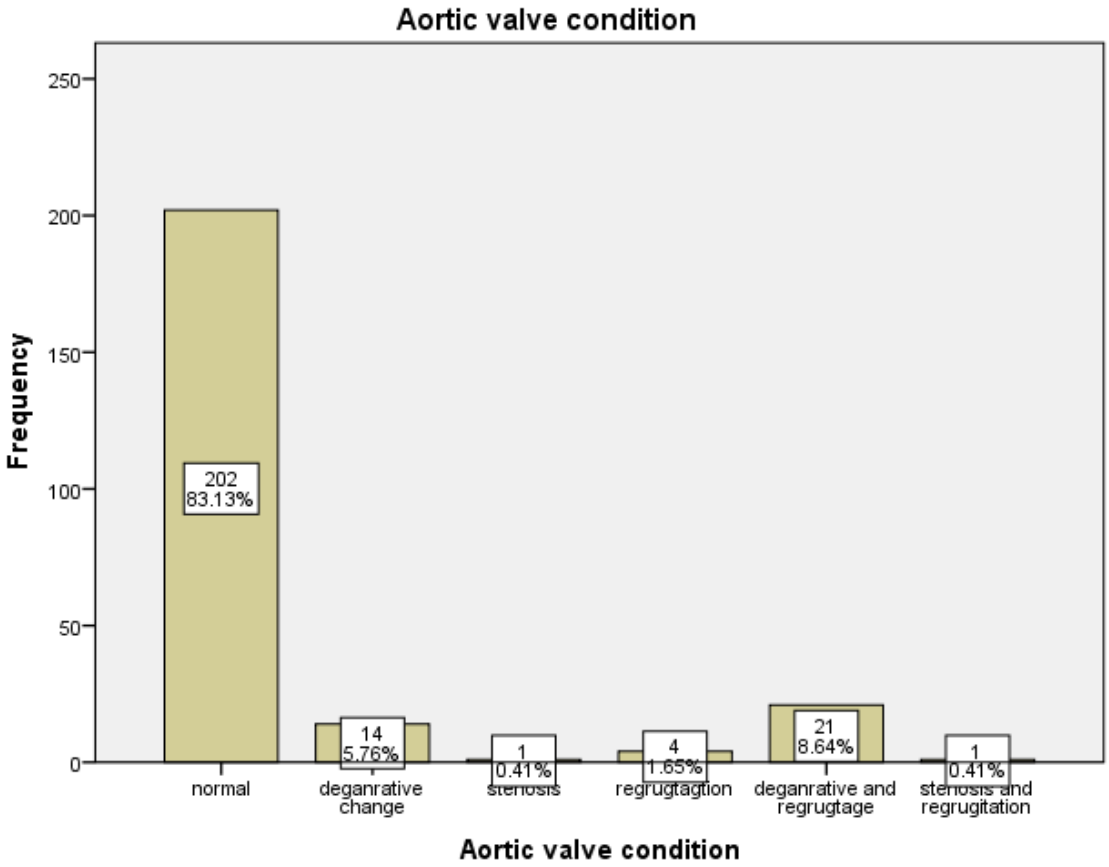


Figure 4.7: Frequency distribution of Aortic valve Condition

Table4.8: frequency distribution of Tricuspid valve conditions

Valid	Non diabetes	diabetes	Frequency	Percent
Normal	113	93	206	84.8
Degenerative change	7	12	19	7.8
stenosis	1	0	1	.4
regurgitation	1	3	4	1.6
degenerative and regurgitation	8	4	12	4.9
stenosis and regurgitation	0	1	1	.4
Total	130	113	243	100.0

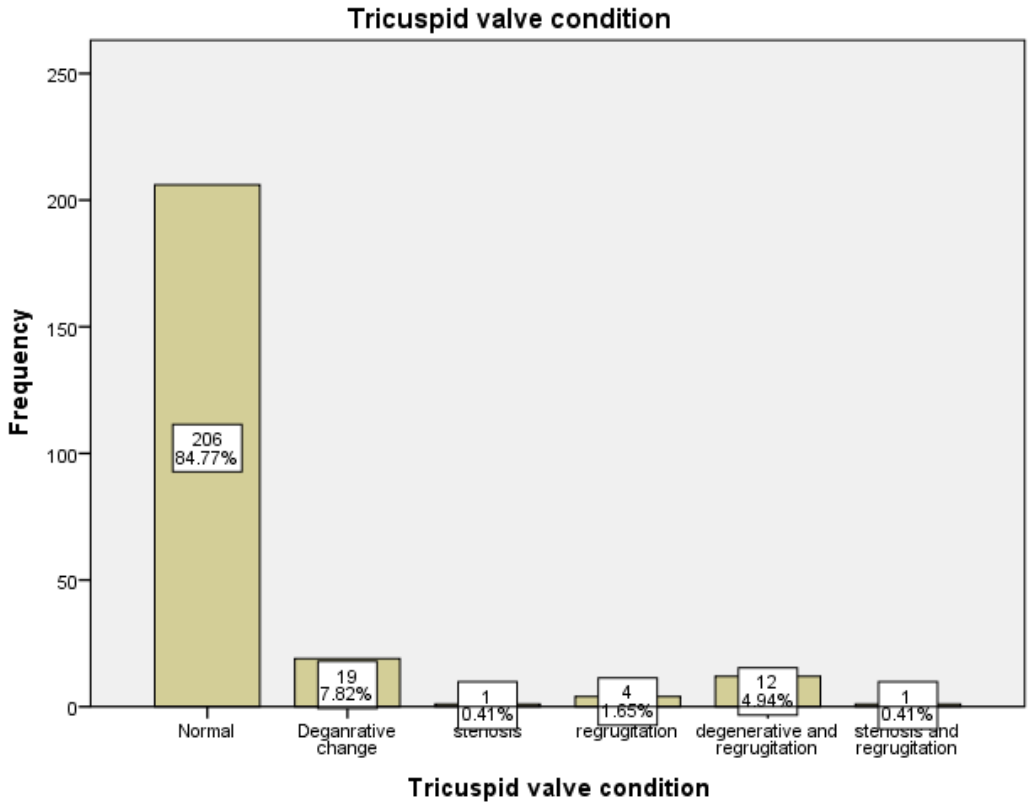
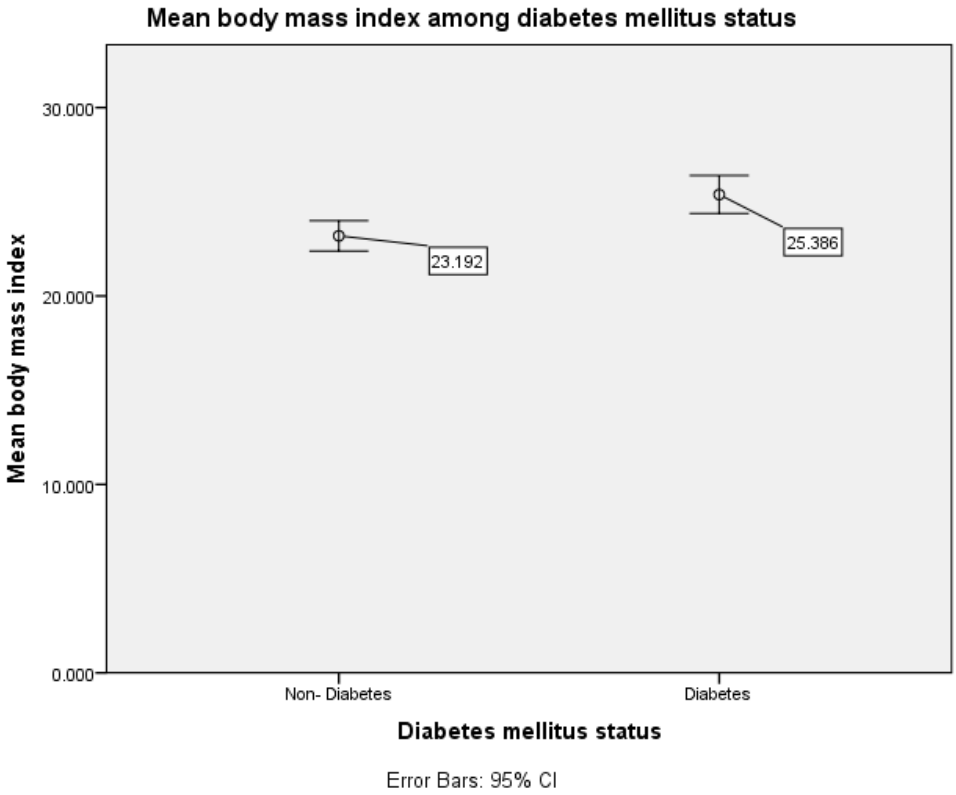


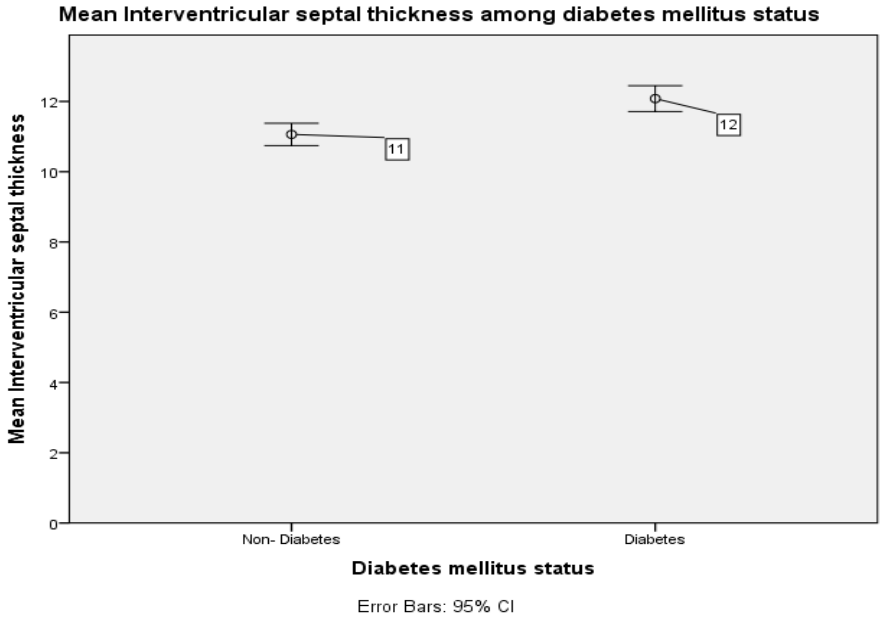
Figure 4.8: Frequency distribution of Tricuspid valve Condition

Parameters	Mean Non-diabetes	Mean Diabetes	T- value	P -values
body mass	23.19244	25.38572	- 3.391	.001
Duration	0	9.23	- 21.107	.000
Inter ventricular septal thickness Diameter	11.06	12.08	- 4.113	.000
Left ventricular posterior wall Diameter	9.61	10.39	- 4.167	.000

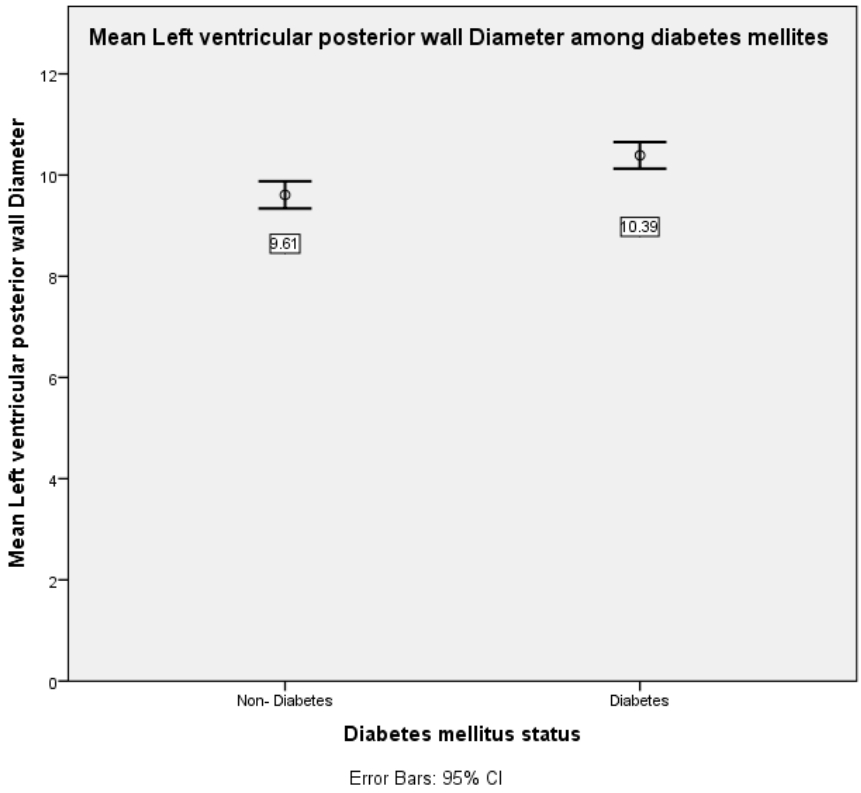
Table 4.9: Compare Means of Echocardiography parameters between Non- diabetes and diabetes patients Type



Figures 4.9: Compare Means of BMI between Non- diabetes and diabetes patients.



Figures 4.10: Compare Means of Echocardiography parameters between Non-diabetes and diabetes patients (Interventricular septal thickness)

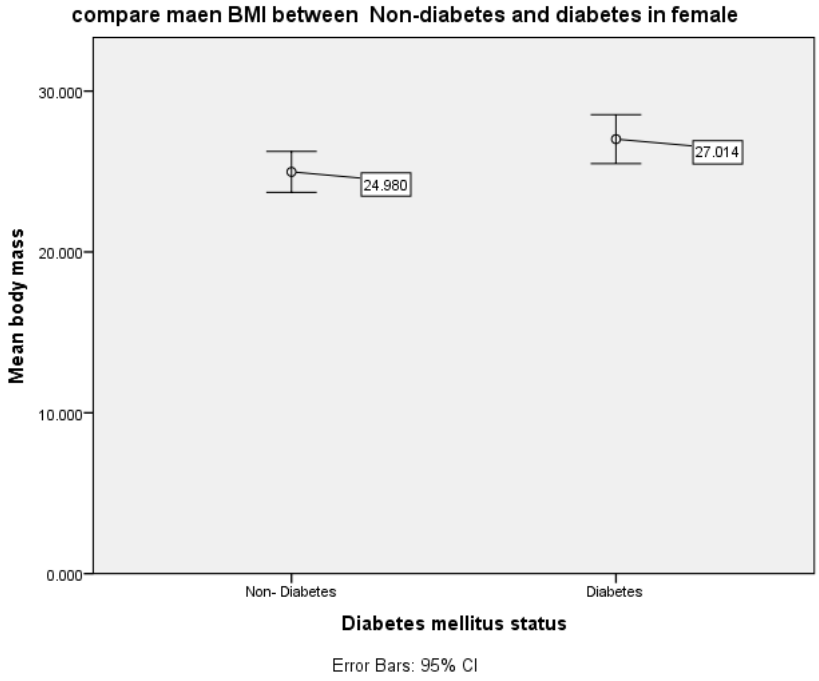


Figures 4.11: Compare Means of Echocardiography parameters between Non- diabetes and diabetes patients (left ventricular Posterior wall diameter)

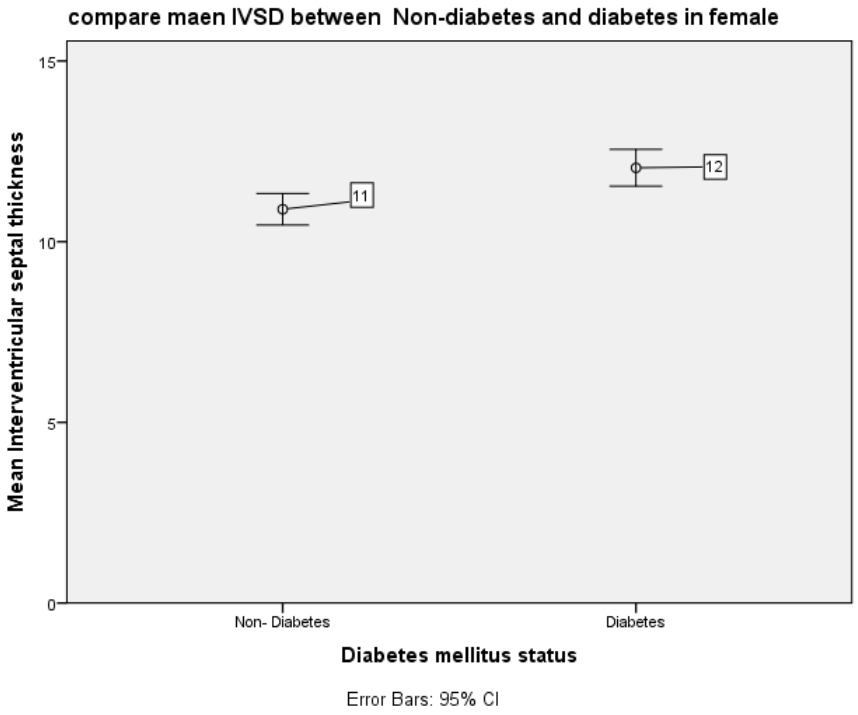
Table 4.10: Compression of mean between female non-diabetes and diabetes

	Diabetes mellitus status	Mean	T -test	P- value
body mass	Non- Diabetes	24.98043	-2.025	.043
	Diabetes	27.01367		
Duration of Diabetes	Non- Diabetes	.00	-2.025	.000
	Diabetes	9.39		
Aortic root diameter	Non- Diabetes	27.78	-.614	.540
	Diabetes	28.15		
left atrial diameter	Non- Diabetes	34.98	.563	.574
	Diabetes	34.47		
Left ventricular	Non- Diabetes	44.92	.481	.631

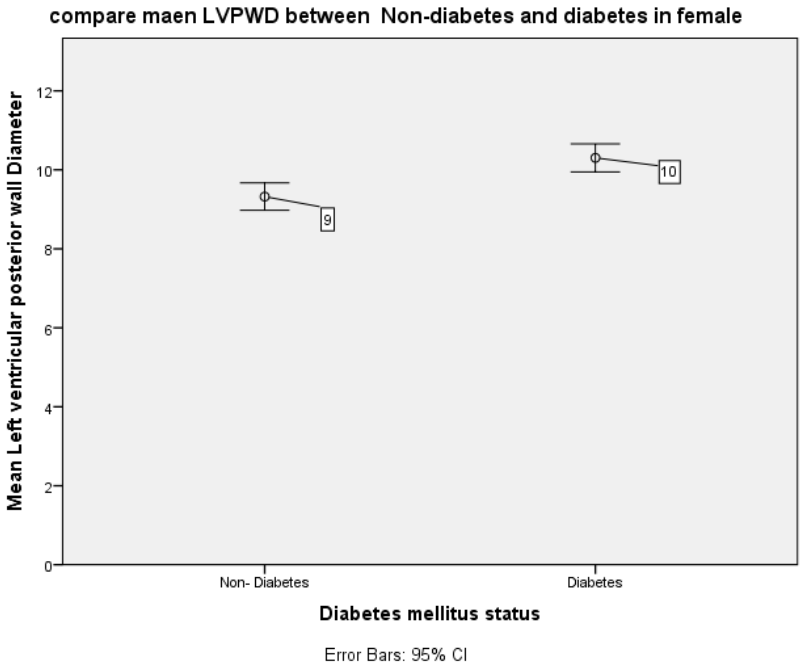
diastolic diameter Left Ventricle	Diabetes	44.47		
	Non- Diabetes	28.66	.028	.978
systolic diameter Interventricular	Diabetes	28.64		
	Non- Diabetes	10.90	-3.377	.001
septal thickness Left ventricular	Diabetes	12.05		
	Non- Diabetes	9.32	-3.929	.000
posterior wall Diameter	Diabetes	10.30		
Ejection fracture	Non- Diabetes	65.23	1.953	.051
	Diabetes	63.38		



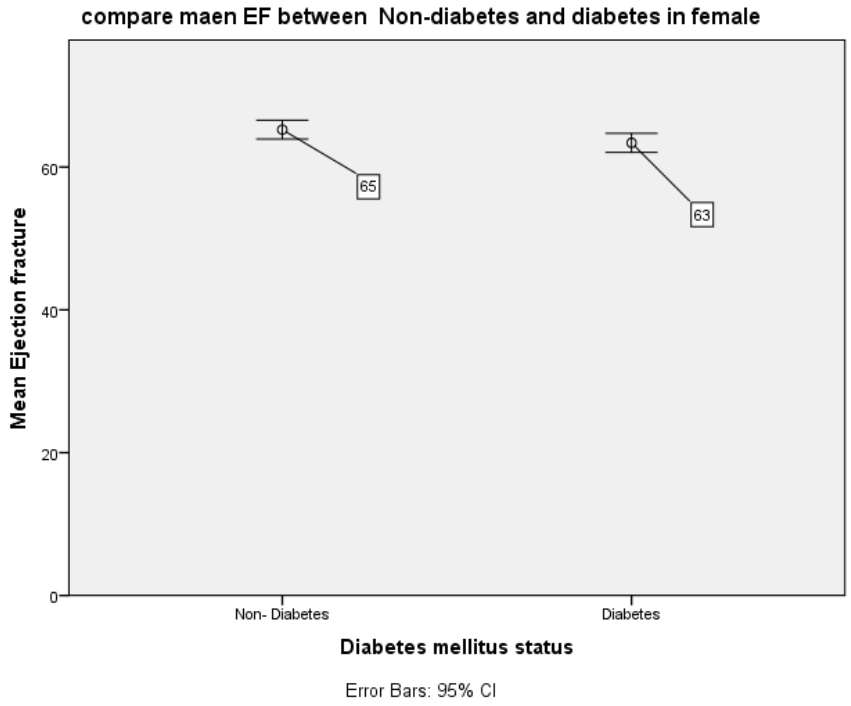
Figures 4.12: Compare means of BMI between Non- diabetes and diabetes female patients.



Figures 4.13: Compare means of IVSD between Non- diabetes and diabetes female patients.



Figures 4.14: Compare means of LVPWD between Non- diabetes and diabetes female patients.

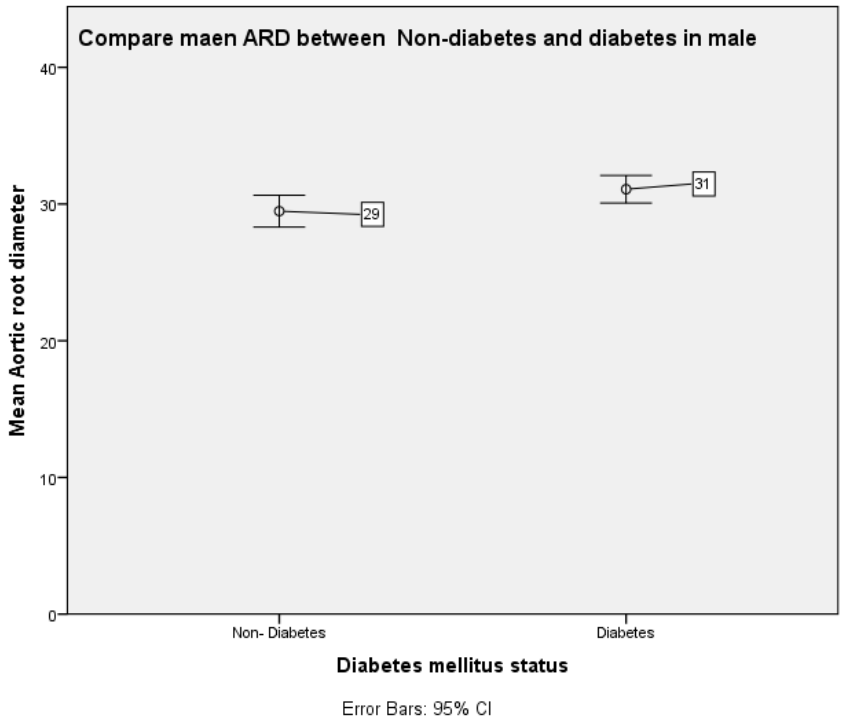


Figures 4.15: Compare means of EF between Non- diabetes and diabetes female patients.

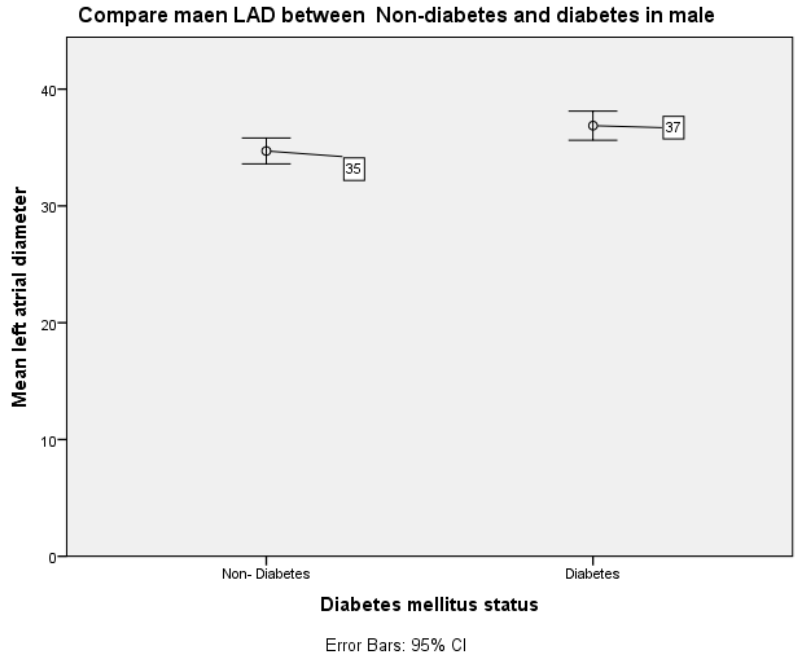
Table4.11: showed the compression of mean between male non-diabetes and diabetes

	Diabetes mellitus status	N	Mean	T -test	p- value
body mass	Non-Diabetes	71	21.70664	-2.084	.034
	Diabetes	47	23.09967		
Duration of Diabetes	Non-Diabetes	71	.00	-14.050	.000
	Diabetes	47	9.00		
Aortic root diameter	Non-Diabetes	71	29.48	-1.942	.047
	Diabetes	47	31.09		
left atrial diameter	Non-Diabetes	71	34.70	-2.556	.011
	Diabetes	47	36.87		

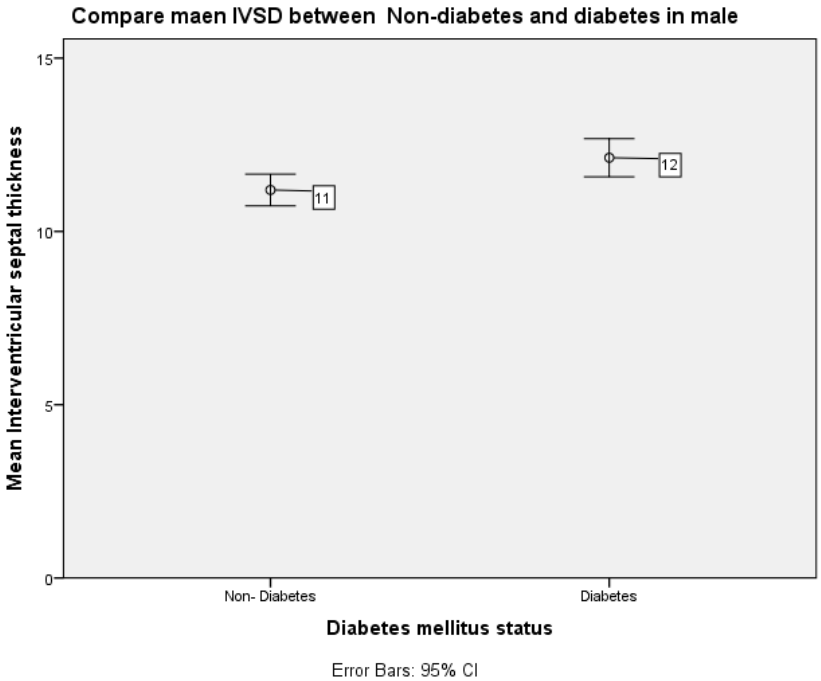
Left ventricular diastolic diameter	Non-Diabetes	71	46.86	-.011	.991
	Diabetes	47	46.87		
Left Ventricle systolic diameter	Non-Diabetes	71	31.13	.394	.694
	Diabetes	47	30.64		
Interventricular septal thickness	Non-Diabetes	71	11.20	-2.587	.011
	Diabetes	47	12.13		
Left ventricular posterior wall Diameter	Non-Diabetes	71	9.85	-2.258	.023
	Diabetes	47	10.51		
Ejection fracture	Non-Diabetes	66	62.83	-1.387	.168
	Diabetes	40	64.38		



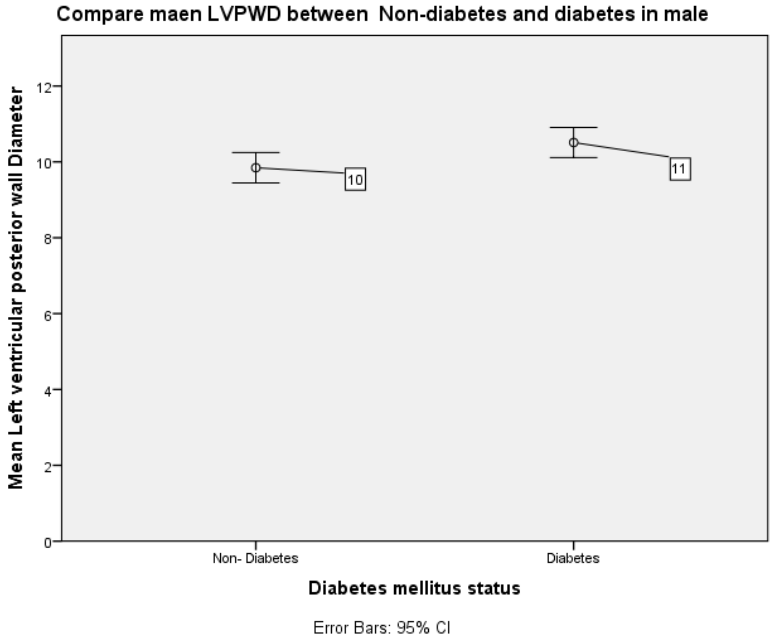
Figures 4.16: Compare means of ARD between Non- diabetes and diabetes male patients.



Figures 4.17: Compare means of LAD between Non- diabetes and diabetes male patients.



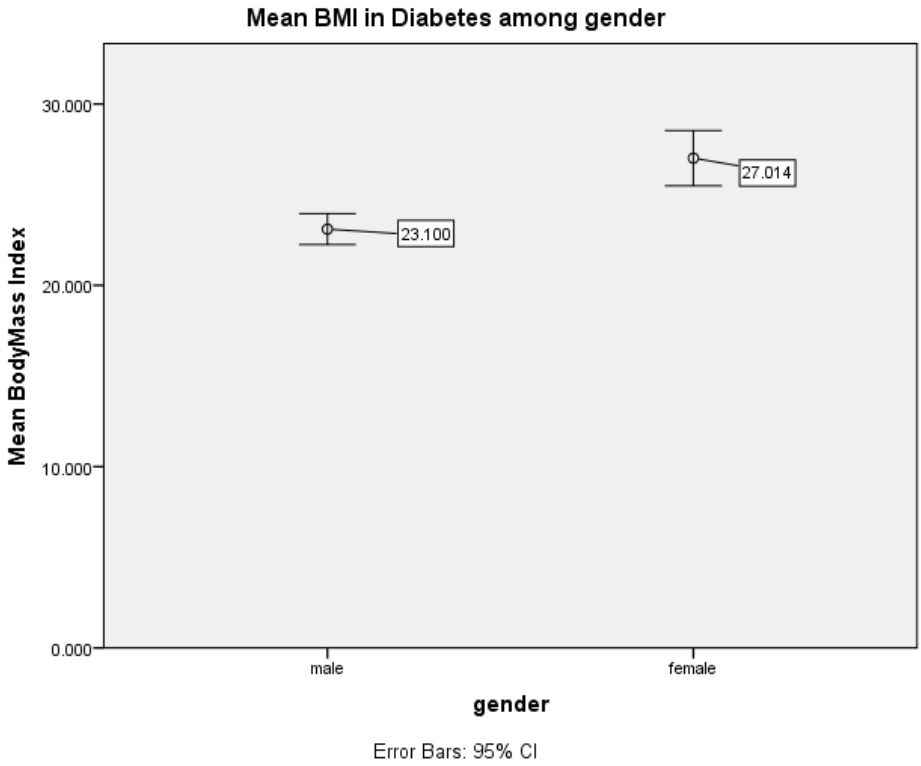
Figures 4.18: Compare means of IVSD between Non- diabetes and diabetes male patients.



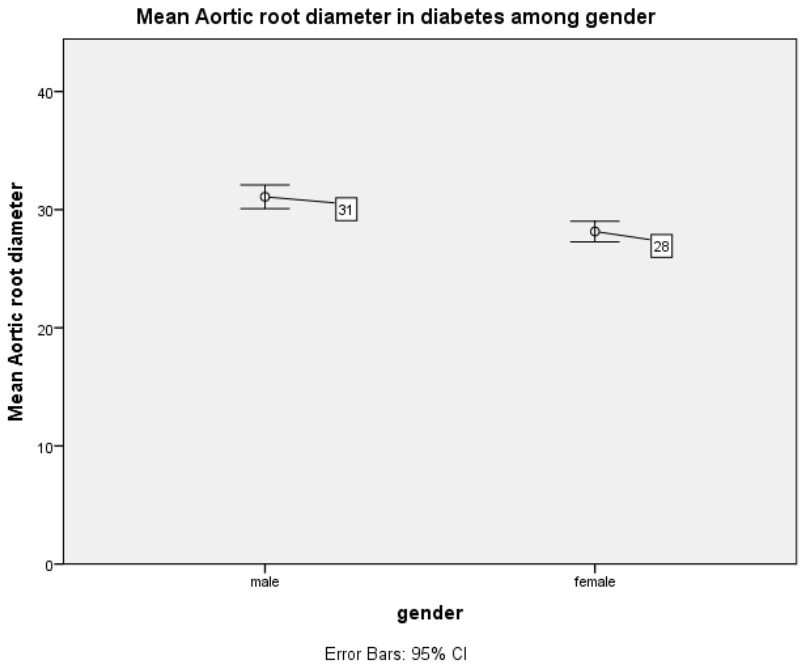
Figures 4.19: Compare means of LVPWD between Non- diabetes and diabetes male patients.

Table 4.12: Compare Means of Echocardiography parameters between Diabetes patients Type II according to gender

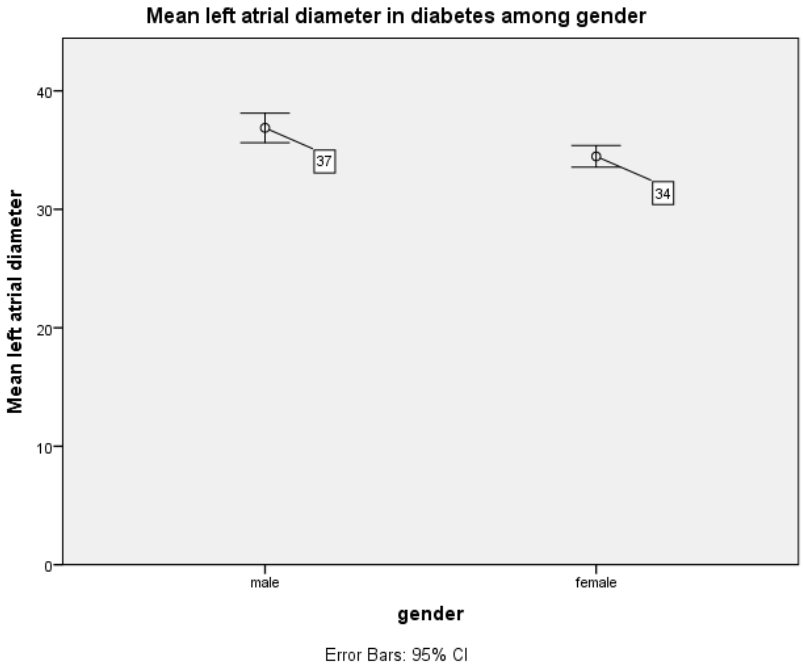
Parameters	Mean male diabetes	Mean female Diabetes	T -test	p-values
body mass	23.09967	27.01367	-4.034	.0001
Aortic root diameter	31.09	28.15	4.391	.0001
left atrial diameter	36.87	34.47	3.189	0.002
Left ventricular diastolic diameter	46.87	44.47	2.331	0.022
Left Ventricle systolic diameter	30.64	28.64	2.020	0.047



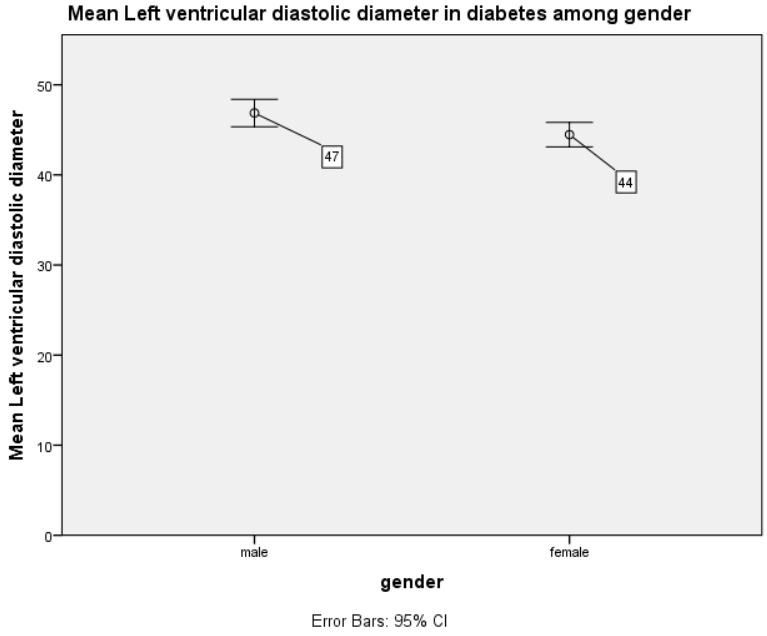
Figures 4.20: Compare Means of BMI between Diabetes patients according to gender.



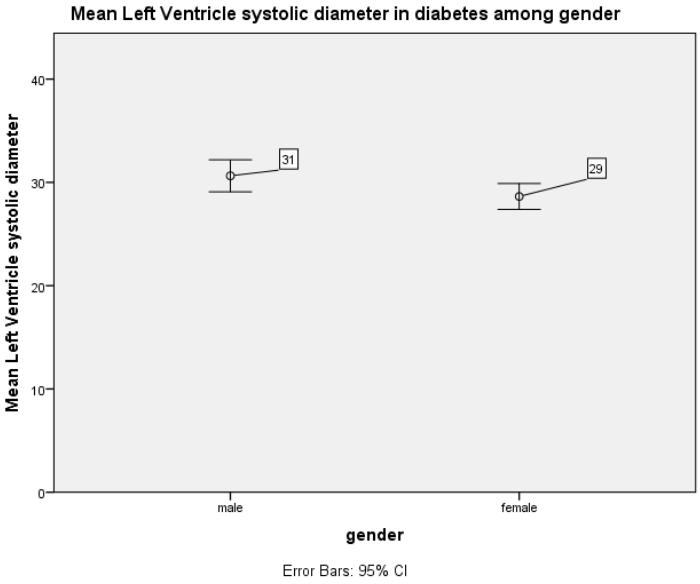
Figures 4.21: Compare Means of Aortic root diameter between Diabetes patients according to gender



Figures 4.22: Compare Means of Left atrial diameter between Diabetes patients according to gender



Figures 4.23: Compare Means of Left ventricular diastolic diameter between Diabetes patients according to gender



Figures 4.24: Compare Means of Left Ventricle systolic diameter between Diabetes patients according to gender

Table 4.13: The correlation of Echocardiography finding parameters in diabetic patients Type II

No	P1	P2	Pearson Correlation	p-values
1	Inter ventricular septal thickness diameter	Left ventricular posterior wall Diameter	.739**	.000
2	Inter ventricular septal thickness diameter	Aortic root diameter	.360**	.000
3	Inter ventricular septal thickness diameter	Left ventricular diastolic diameter	-.224*	.017
4	Inter ventricular septal thickness diameter	Ejection fracture	.186*	.048
5	Left ventricular diastolic diameter	Ejection fracture	-.326**	.000
6	Left Ventricle systolic diameter	Ejection fracture	-.644**	.000

7	Left ventricular diastolic diameter	Left Ventricle systolic diameter	.778**	.000
8	Left ventricular diastolic diameter	left atrial diameter	.295**	.001
9	Left ventricular posterior wall Diameter	Aortic root diameter	.244**	.009
10	left atrial diameter	Aortic root diameter	.190*	.044

R^2Linear=0.546

Figure 4.25: Correlation between Inter ventricular septal diameter and Left ventricular posterior wall Diameter in Diabetes patients

Figure 4.26: Correlation between Inter ventricular septal diameter and Aortic root diameter.

Figure 4.27: Correlation between Inter ventricular septal diameter and Left ventricular diastolic diameter.

Figure 4.28: Correlation between Inter ventricular septal diameter and Ejection fracture

Figure 4.29: Correlation between Left ventricular diastolic diameter and Ejection fracture

Figure 4.30: Correlation between Left Ventricle systolic diameter and Ejection fracture

Figure 4.31: Correlation between Left ventricular diastolic diameter and Left Ventricle systolic diameter

Figure 4.32: Correlation between Left ventricular diastolic diameter and left atrial diameter

Figure 4.33: Correlation between Left ventricular posterior wall Diameter and Aortic root diameter

Figure 4.34: Correlation between left atrial diameter and Aortic root diameter

CHAPTER FIVE
Discussion, Conclusion and Recommendations

Chapter Five
Discussion, Conclusion and Recommendations

5-1 Discussion

Cardiovascular disease remains the leading cause of death in the world as well as in the Sudan territories. Approximately 80% of all cardiovascular-related deaths occur in low- and middle-income countries and at a younger age in comparison to high-income countries (B.Gersh et al. (2010).

Two-dimensional echocardiography has recently gained popularity as a noninvasive diagnostic aid in the evaluation of various forms of heart disease. Although M-mode echocardiography is valuable in detecting wall motion changes related to ischemia (Corya et al. (1975),

We examined 243 subject 130 non-diabetes male 71 and female 59 and 113 subject was diabetes male 47 and 66 female in present study, the body mass index was found to be very highly significant at p value 0.001 in diabetes more than non-diabetes, This finding was in counterbid with Ramzi Ali Mansour (2014) study that found the High population attributable risks were related to excess weight (BMI ≥25) for the outcomes hypertension (26% men; 28% women), angina pectoris (26% men; 22% women), and CAD (23% men; 15% women) (Mansour (2014).

The study revealed the range age (30-85years) mean 55.76 years stander Deviation 13.632 the big number of diabetes was female patients in age group 50-60, this would agree with study done by Mahmoud Werfalli et al (2013) (Werfalli et al. (2014), the majority of individuals with diabetes in Africa were reported to be under 60 years of age with the highest proportion (43.2%) in people aged 40–59 years.

This study manifested the Compare Means of Echocardiography parameters Diabetes patients Type 2 between male and female as fowled first Body mass index the male diabetes mean was 23.09967Kg/m2 St. D 2.898752 and female diabetes mean was 27.01367Kg/m2 St. D 6.179767

the p-value .000101, second was Aortic root diameter the male diabetes mean was 31.09 mm St. D 3.425 and female diabetes mean was 28.15 mm St. D 3.553 the p-value .000026, third was left atrial diameter the male diabetes mean was 36.87 mm St. D 4.241 and female diabetes mean was 34.47 mm St. D 3.726 the p-value 0.001856, the forth one was the Left ventricular diastolic diameter the male diabetes mean was 46.87 mm St. D 5.195 and female diabetes mean was 44.47 mm St. D 5.542 the p-value 0.021567 and lastly the Left Ventricle systolic diameter the male diabetes mean was 30.64 mm St. D 5.306 and female diabetes mean was 28.64 mm St. D 5.110 the p-value 0.045766.

On other hand, this study revealed that mitral, tricuspid, pulmonary and aortic valve velocity within standard normal limits indicated by the international literature in most cases. These findings were in agreement with Garot et al.,(1999) who reported that our data confirmed that systolic and diastolic myocardial regional velocities, as assessed by colour M-mode Doppler tissue imaging, were significantly reduced in the infarct region when compared with the corresponding walls in healthy subjects.(Corya et al. (1975)

Current study found that patients Inter ventricular septal thickness diameters and Left ventricular posterior wall Diameter both was strongly significant in diabetes more than in non-diabetes subject (p-value 0.000) this agreement with Fadia J. Alwan et al (2012) who reported that indicate the LV muscle at the beginning of lack in LV performance Alwan et al. (2014), this also Similar as study by Current study revealed that Ejection fracture, Left ventricular diastolic diameter, Left Ventricle systolic diameter they had no significance difference between diabetes, and non-diabetes this well be clinic significance because there's different in dimension between them , this contributed with study, Zhi You Fang et al (2003) was reported that Experimental and clinical evidence has shown that diabetes is associated with LV systolic and diastolic dysfunction This study shows that sensitive indexes of systolic and diastolic function are abnormal in diabetic patients with a normal ejection fraction and without coronary heart disease and LV hypertrophy However, the absence of an

abnormal response to stress argues against a role for ischemia in the etiology of this dysfunction (Fang et al. (2003a). Aigbe, I. F. et al (2012) reported that the main finding of this study is high prevalence of LVDD in black normotensive type 2 DM patients. The study showed that 72% of the patients evaluated had one form of LVDD or the otherAigbe et al. (2012). Similar studies in USA involving both White and Black type 2 DM individuals reported comparable prevalence of LVDD. (Zabalgoitia et al. (2001), Boyer et al. (2004)) In the same vein, Osunkwo and Okeahialam had found high prevalence of LVDD in DM patients seen in Jos, (Nigeria.Osunkwo and BN (2001).

The current study revealed that the compared between female diabetes and non-diabetes we found that the diabetes had effect on same echo parameters like body mass index is more in female diabetes than in female control with mean 27.01Kg vs. 24.98 Kg and p-value 0.043, Duration of Diabetes mean 9.39 year std. deviation 4.7 with (p- value 0.000), Aortic root diameter and Ejection fraction In non-diabetes 65.23 vs. diabetes 63.38 were slightly high in diabetes with no mathematically significant, Interventricular septal thickness in non-diabetes 10.90, this in diabetes 12.05 with p-value 0.001 Left ventricular posterior wall Diameter (non-diabetes 9.32 vs. diabetes 10.30) with p-value 0.000. this similar as Amal Al Marayati found (Amal Al Marayati and MuhammedMD, 2007). similarity as Richard B. (Devereux et al, 2000) it is reported that the BMI was more in women diabetes than mean and Interventricular septal and posterior LV wall thicknesses were greater in diabetic than the non-diabetes (p-value 0.0001) (Devereux et al., 2000).

On the other hand the study showed that the compared between male diabetes and non-diabetes we found that the diabetes had effect on same echo parameters like body mass index, Duration of Diabetes, Aortic root diameter, left atrial diameter, Interventricular septal thickness and Left ventricular posterior wall diameter were different in diabetes vs. non-diabetes as (23.09967 vs.21.70664 with p-value 0.34), (9.00 years p-value 0.000), (31.09 vs. 29.48 with p-value 0.47), (34.70 vs. 36.87 with p-

value 0.011), (12.13vs.11.20 with p-value 0.011) and (10.51vs.9.85 with p-value 0.023) this similar as Devereux et al.

Current study showed the correlation in DM patients between male and female were found the BMI was more in female than male with high significance (p-value 0.0001), (Bakris et al. (2004).

On the other hand the other echo finding parameters are more in male than female, as Left ventricular diastolic diameter, Aortic root diameter, left atrial diameter and Left Ventricle systolic diameter that findings demonstrate that pre-clinical diastolic dysfunction is common in patients with DM. Pre-clinical diastolic dysfunction has been broadly defined as diastolic dysfunction in patients with normal systolic function, and no symptoms of heart failure (HF). This agreed with studied Veranda C. Patil et al (2011)Patil et al. (2011) they reported that the study reveals high burden of diastolic dysfunction in cohort of type 2 DM population. Also similarity as study by, L. J. M. Boonman (Adeoye et al.) Boonman-de Winter et al. (2012) he reported that the older patients is more with type 2 diabetes showed that the prevalence of previously unknown heart failure is very high (Boonman-de Winter et al.), Our study in a large representative group of older patients steeply increases with age, and is overall higher in women (31.0%) than men (24.8%). The prevalence is significantly higher in patients with a BMI≥30 kg/m2 (38.7% vs. 23.4%). Also agreed with Isabelle Pham (Blomstrand et al.) Pham et al. (2015) prevalence of systolic dysfunction in asymptomatic patients is not often studied and as in our results concerns less than 10% of the diabetic population. And so similar as study of Akdemir O, et al (Akdemir et al.)) Akdemir et al. (2001) his result, that Among the groups, left ventricular diastolic dimension-index, and fractional shortening, were showed no statistically significant differences, while septum and posterior wall thickness and posterior wall.

In this study the last point we found the correlation in diabetes between many echo parameters as reveled first between Inter ventricular septal thickness diameters and Left ventricular posterior wall Diameter with highly Pearson Correlation 0.739 and highly significance (P value 0.000)

This study showed the correlation of Echocardiography finding parameters in diabetic patients Type 2, they was as fowled between Inter ventricular septal diameter and Left ventricular posterior wall Diameter Pearson Correlation 0.739 with p-value 0.000, between Inter ventricular septal diameter and Aortic root diameter Pearson Correlation 0.360 p-value 0.000, between Inter ventricular septal diameter and Left ventricular diastolic diameter Pearson Correlation -.224 p-value and 0.017, between Inter ventricular septal diameter and Ejection fracture Pearson Correlation .186 and p-value 0.048, Left ventricular diastolic diameter and Ejection fracture Pearson Correlation -.326 and p-value 0.000, between Left Ventricle systolic diameter and Ejection fracture Pearson Correlation -.644 and p-value .000, between Left ventricular diastolic diameter and Left Ventricle systolic diameter Pearson Correlation 0.778 and p-value 0.000, between Left ventricular diastolic diameter and left atrial diameter Pearson Correlation .295 and p-value 0.001, between Left ventricular posterior wall Diameter and Aortic root diameter Pearson Correlation .244 and p-value 0.009 and left atrial diameter and Aortic root diameter Pearson Correlation .190 and p-value 0.044.

5-2 Conclusion

This study had been carried out in echocardiography department of Al Rebate teaching Hospital in Khartoum-Sudan.

The aim of this study was to measure the preservative factors of diabetes (V.S), BMI, left ventricle size IVS thickness, posterior wall thickness, pericardium, valves velocity and ejection fraction, to predict the occurrence of diastolic dysfunction of the LV, to correlate between the age and early changes in heart muscles, to determine the incidence of LV hypertrophy in gender. Two hundred and forty three subjects were included in this study.

All participants of the study were subjected to the following:

- History taking and complete clinical examination.
- Calculation of BMI.
- Echocardiogram.

Current study confirm that echocardiography is a valuable tool for evaluation of patients with diabetes mullet's type 2.

As regard age; this study showed that the medial age patients were more than young patients. Majority of patients are in between 50-60 age group. Diabetes were is more common in females than males due to the sample size rest of it are house wife.

As regard treatment of BMI, this study found that BMI shows significant association with diabetes type 2 risk worldwide. BMI is a useful statistical tool to track the body size trends in a population.

As regard echocardiography findings, current study showed that the means and range for the measurements of left ventricular diastolic dimension, IVS thickness, posterior wall thickness, pericardium and valves velocity found in this survey were within standard normal limits indicated by the international literature.

The study revealed that the patients with DM without overt heart disease display subclinical observed LV systolic function even after adjustment for blood pressure, age, and BMI.

The prevalence of type 2 diabetes mellitus is increasing about 9% of the adult U.S. and in Sudan 7-10 % of population currently has this disorder. Diabetes is a leading cause of blindness, renal disease, and amputation

and leads to increased mortality, primarily from cardiovascular events which lied to sudden HF.

In the present study, DM was the strongest independent factor for LV diastolic dysfunction. This study confirms that asymptomatic diastolic dysfunction is more prevalent in subjects with type 2-DM.

Non–insulin-dependent DM has independent adverse cardiac effects, including increased LV mass and wall thicknesses, reduced LV systolic chamber and myocardial function, and increased arterial stiffness. These findings identify adverse cardiovascular effects of DM, independent of associated increases in BMI and arterial pressure that may contribute to cardiovascular events in diabetic individuals.

5-3 Recommendation

- The echocardiography should be route in patient DM type 2 to prevent in subclinical over heart manly in patient above 40 years old.
- They recommends screening for type 2 diabetes in asymptomatic adults with sustained blood pressure (either treated or untreated) greater than 135/80 mm Hg. This is a grade B recommendation.
- The Government and community should by alarmed about the diabetes harmful, so to take accent to prevent their people.
- They concludes that the current evidence is insufficient to assess the balance of benefits and harms of routine screening for type 2 diabetes in asymptomatic.
- Echo parameters were not changed that not main patient is healthy, but fowl up must be considered.
- In order to improve the current poor prognosis in subjects with DM, the treatment of diastolic HF must be optimized. Subjects with DM type 2 should be screened for sub clinical diastolic dysfunction by echocardiography.
- The future studies should including blood sugar immediately test.

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APPENDICES

Appendix (A)

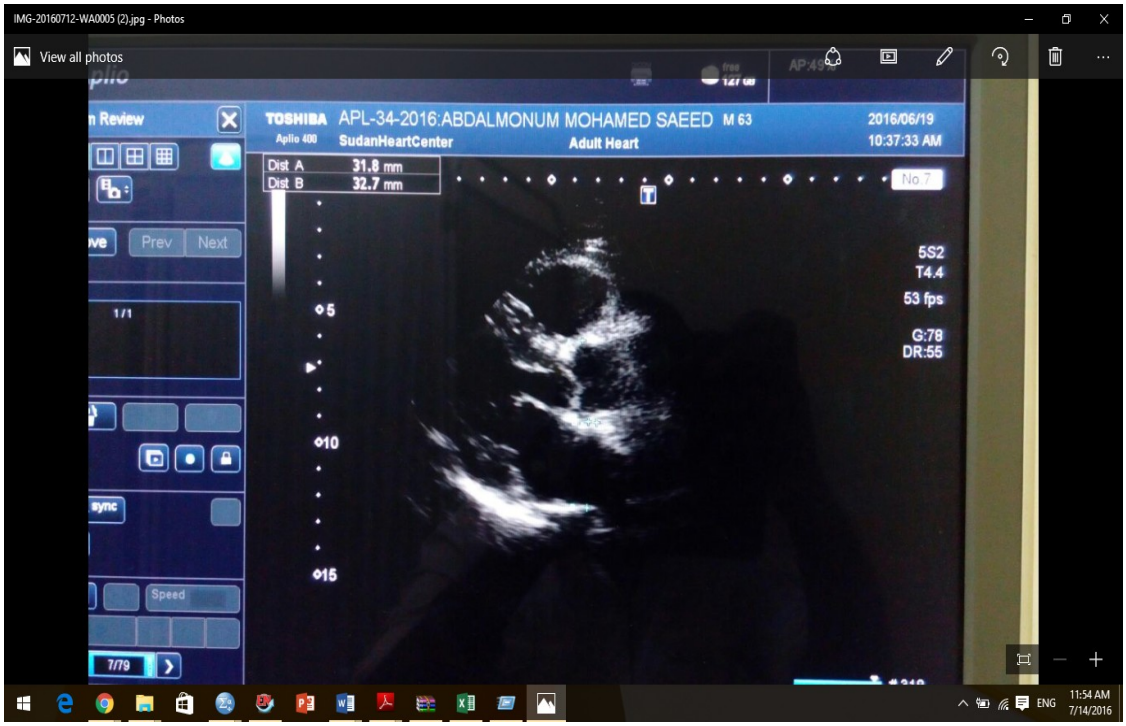


Figure: 5.1 Dist. 1 measure of Ascending Aorta and Dist. 2 left Atrium

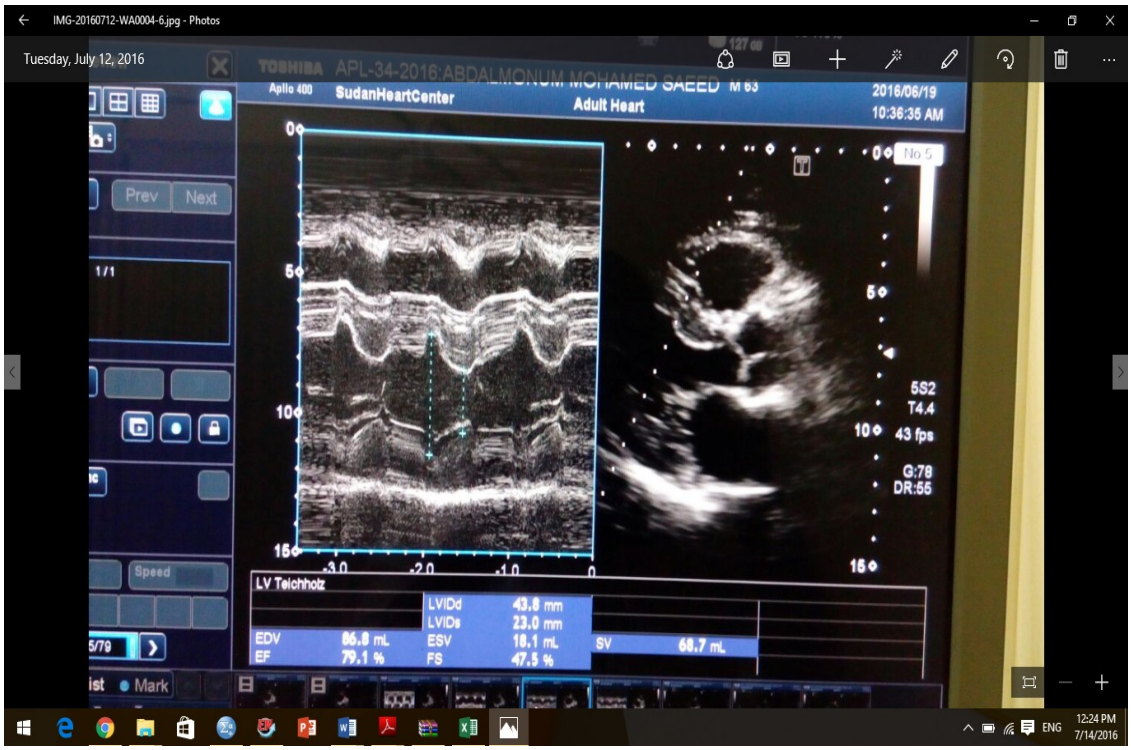
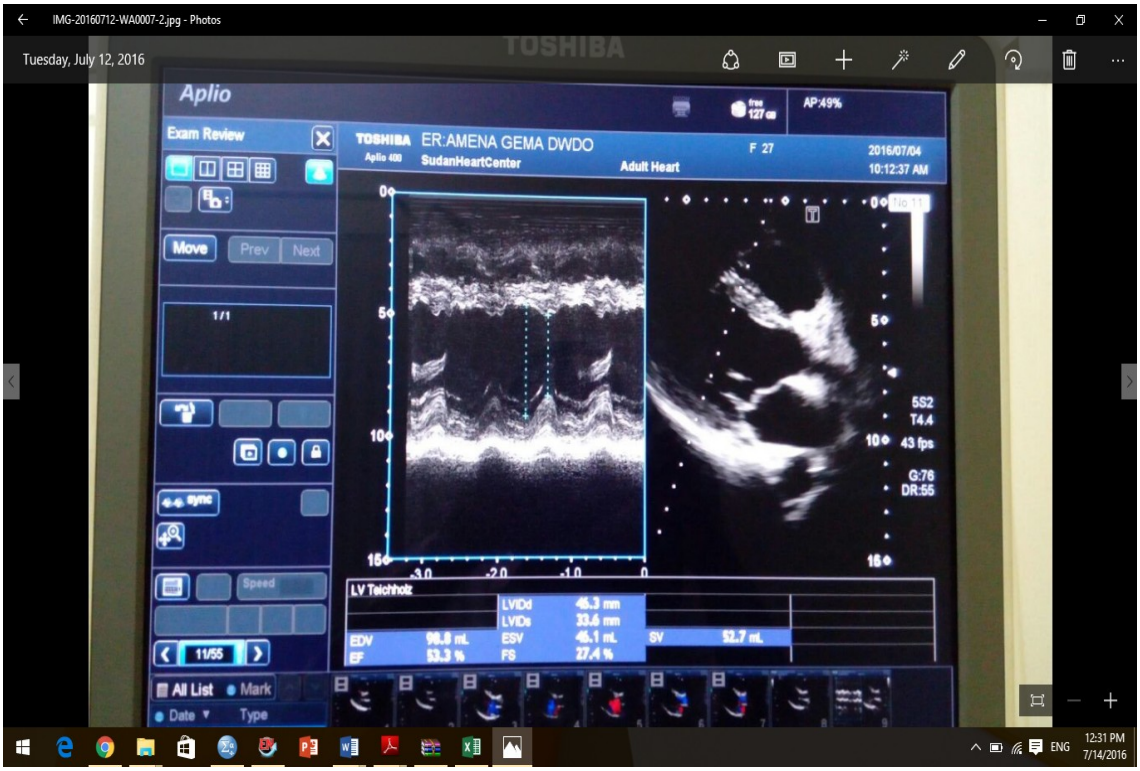


Figure: 5.2 Measure of LVIDd, LVIDs and Ejection fracture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LVIDs 33.6 mm
EDV 98.8 mL
ESV 46.1 mL
SV 52.7 mL
EF 53.3 %
FS 27.4 %
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Figure: 5.3 Measure of LVIDd, LVIDs and Ejection fracture in DM patient

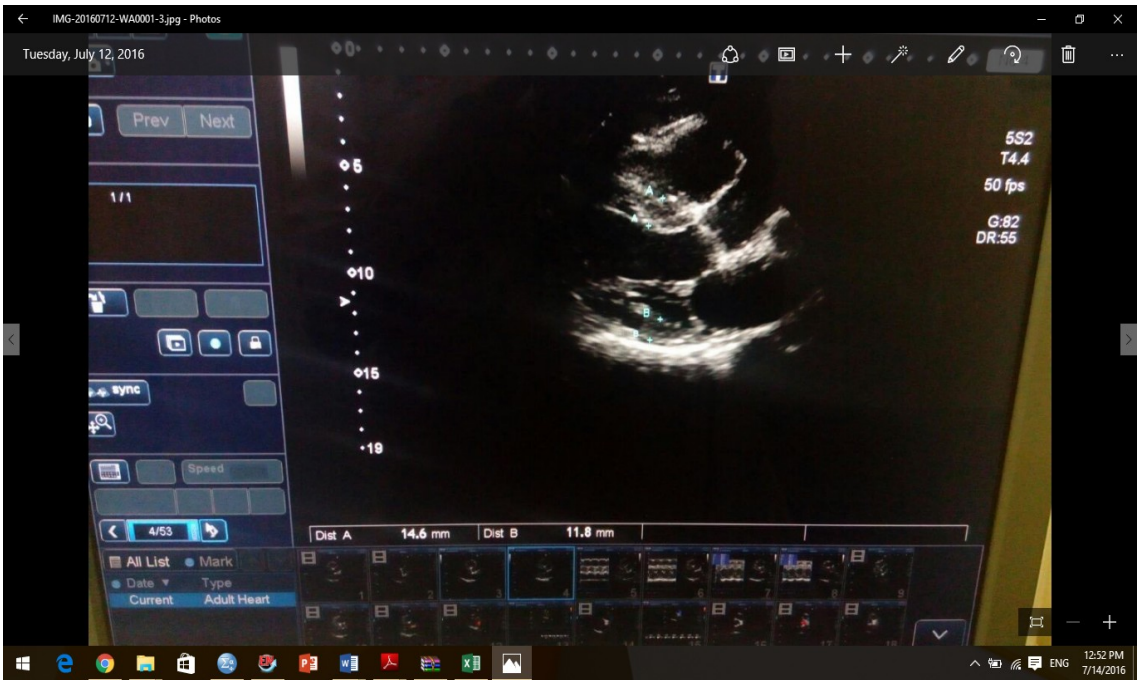


Figure: 5.4 Measure of Inter ventricular septal and Left ventricular posterior wall Diameters

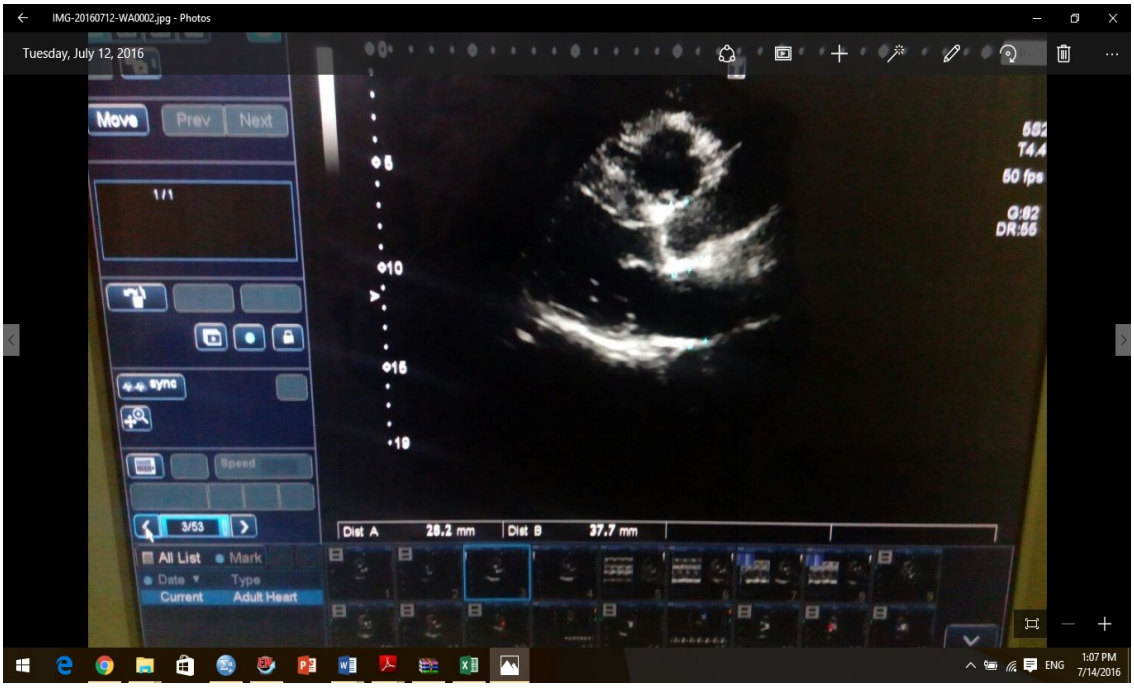


Figure: 5.5 Dist.1 measure of Ascending Aorta and Dist.2 left Atrium in diabetic patient

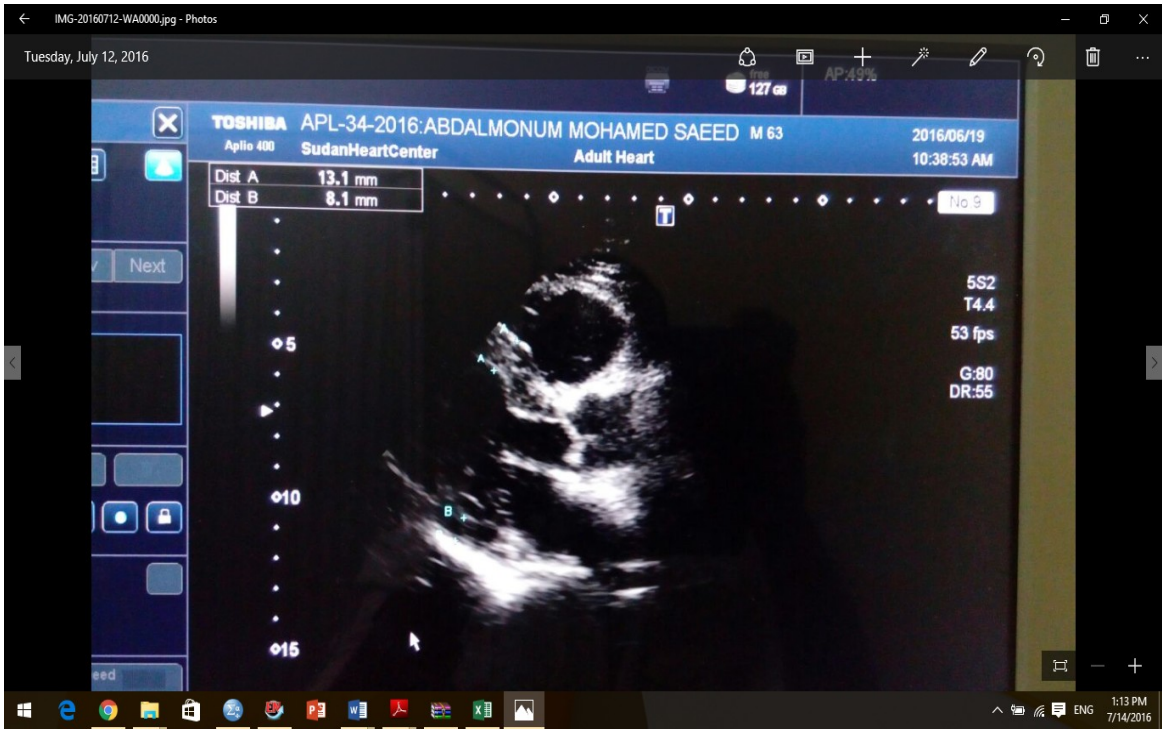


Figure: 5.6 Measure of Inter ventricular septal and Left ventricular posterior wall Diameters

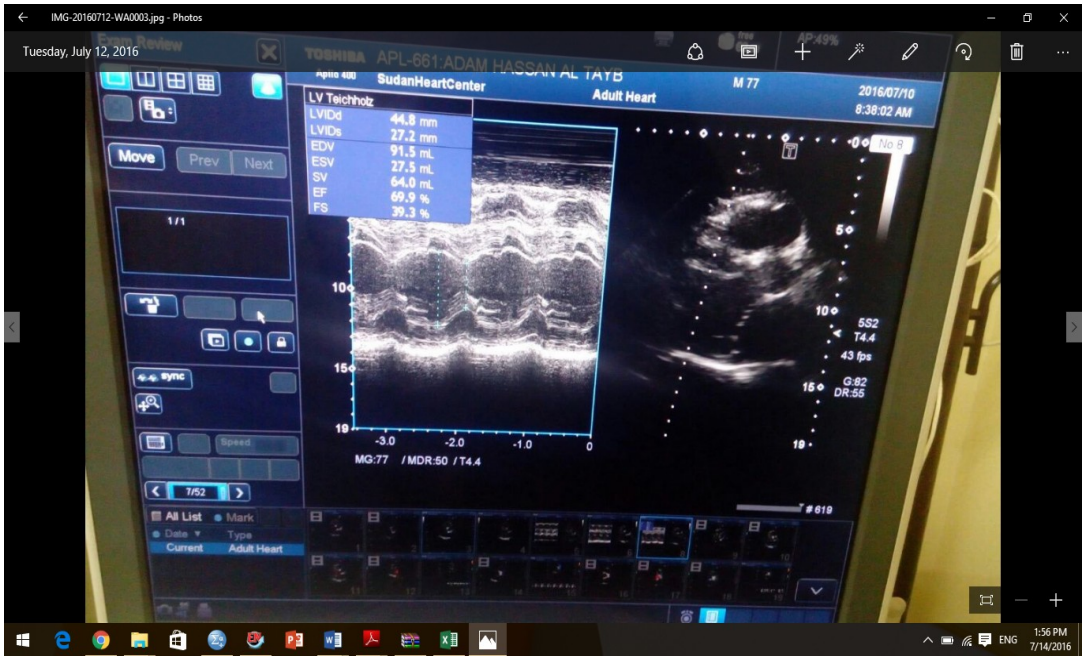


Figure 5.7 M mode measuring LVIDd, IVIDs, EF and FS

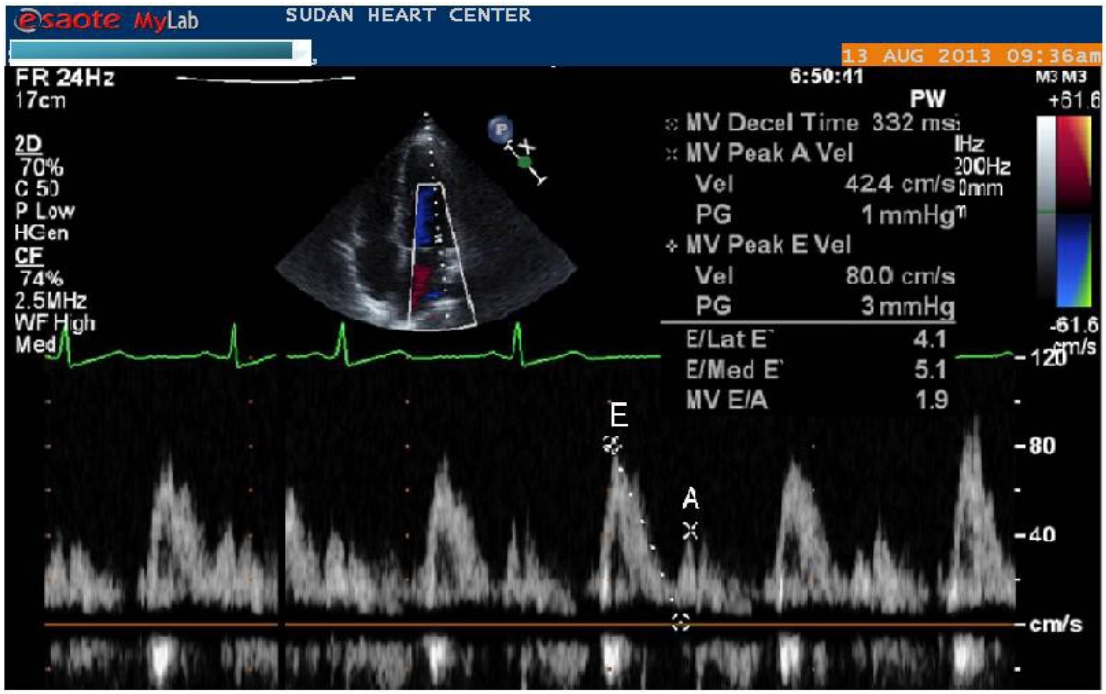


Figure 5.8 Normal Mitral Valve Inflow Pattern.

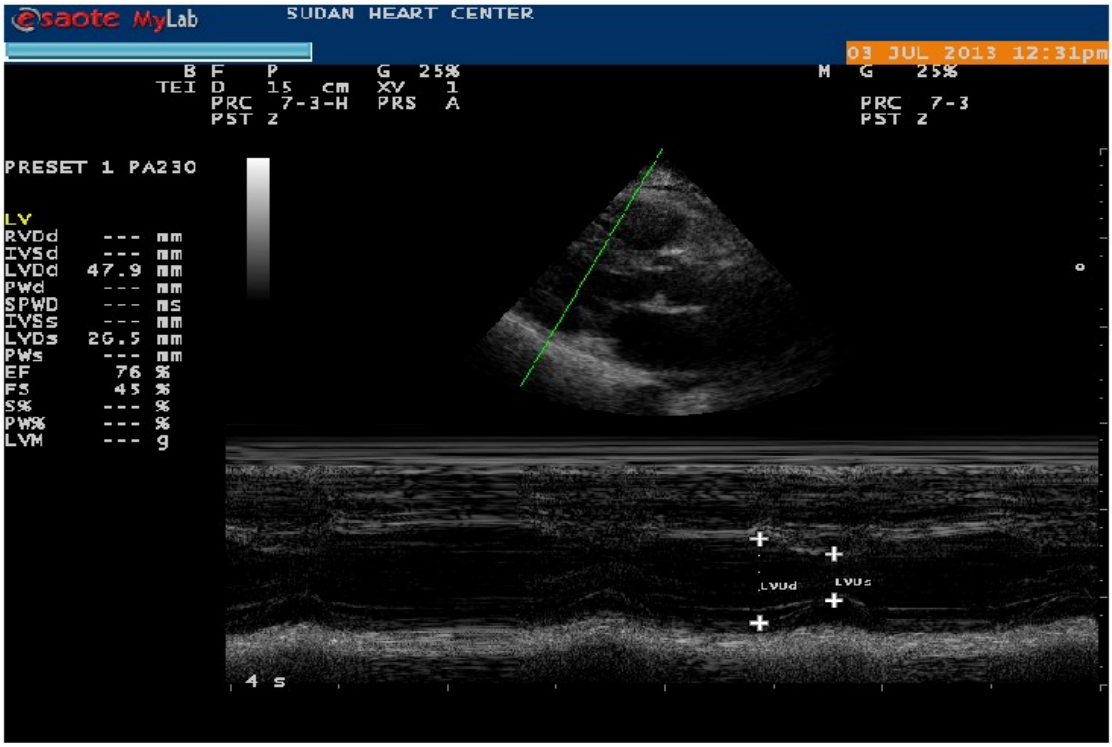


Figure 5.9 Same patient the LV diastolic measurement is 47.9 mm and the systolic measurement is 26.5 mm and EF76%.

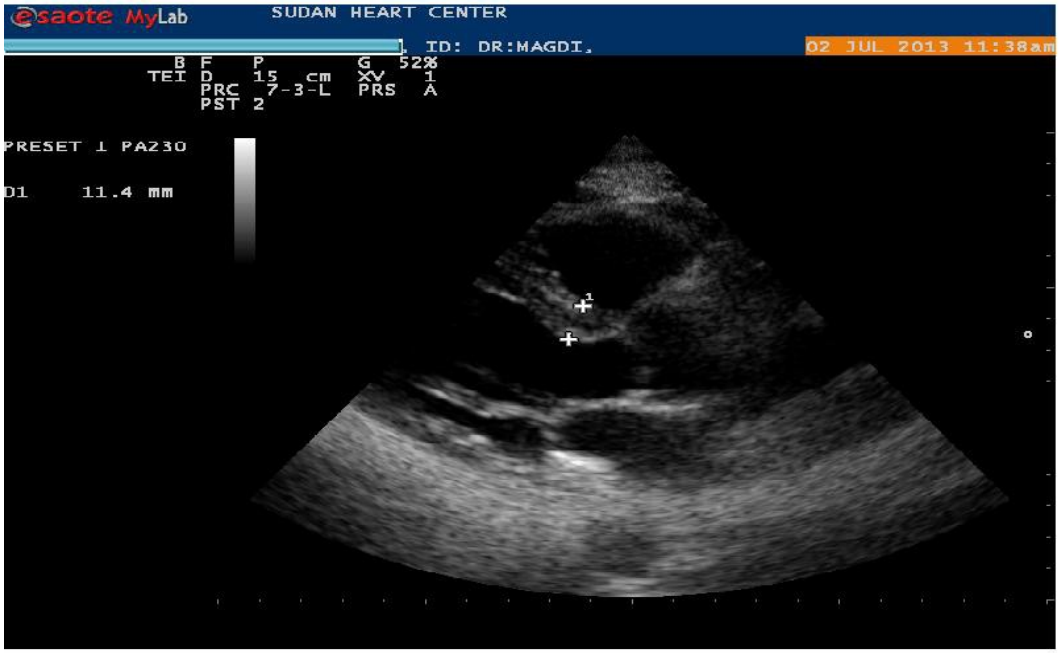


Figure 5.10 Parasternal long axis shows IVS in mildly abnormal (11.4mm).

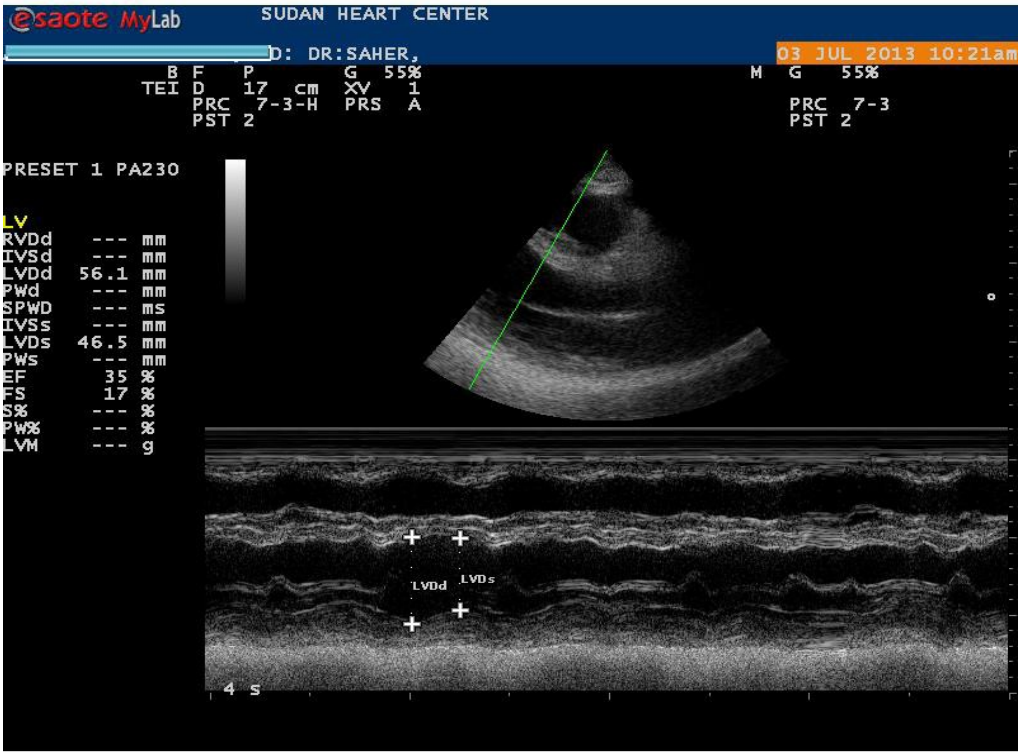


Figure 5.11 In the M-mode below, the LV diastolic measurement is 56.1mm and the systolic measurement is 46.5 mm, both of which are normal.

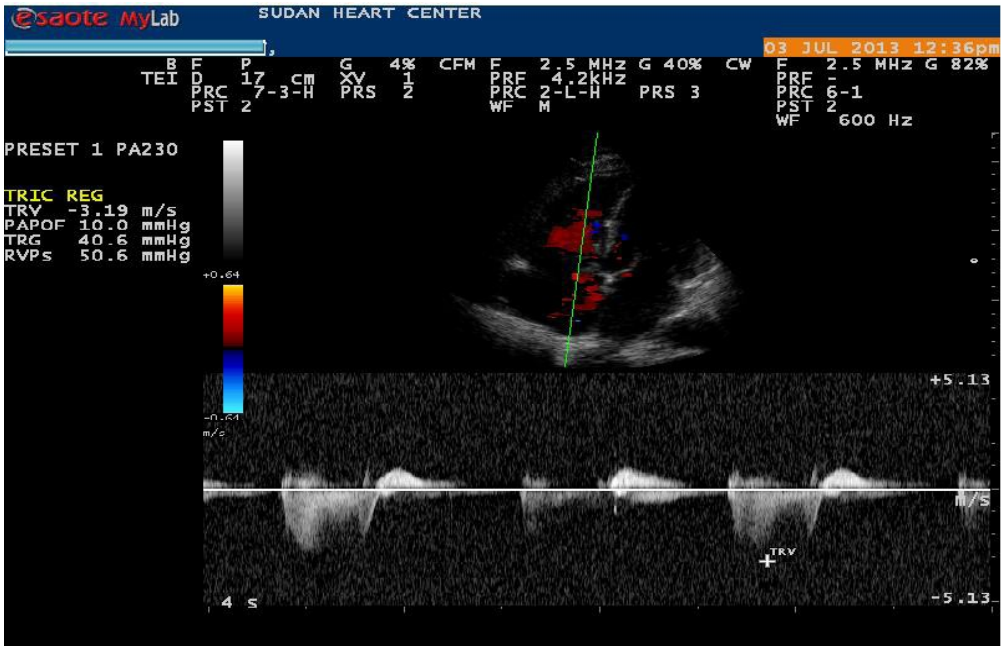


Figure 5.12 Continuous wave Doppler signal of tricuspid with a peak velocity of approximately 3.19 m/s.

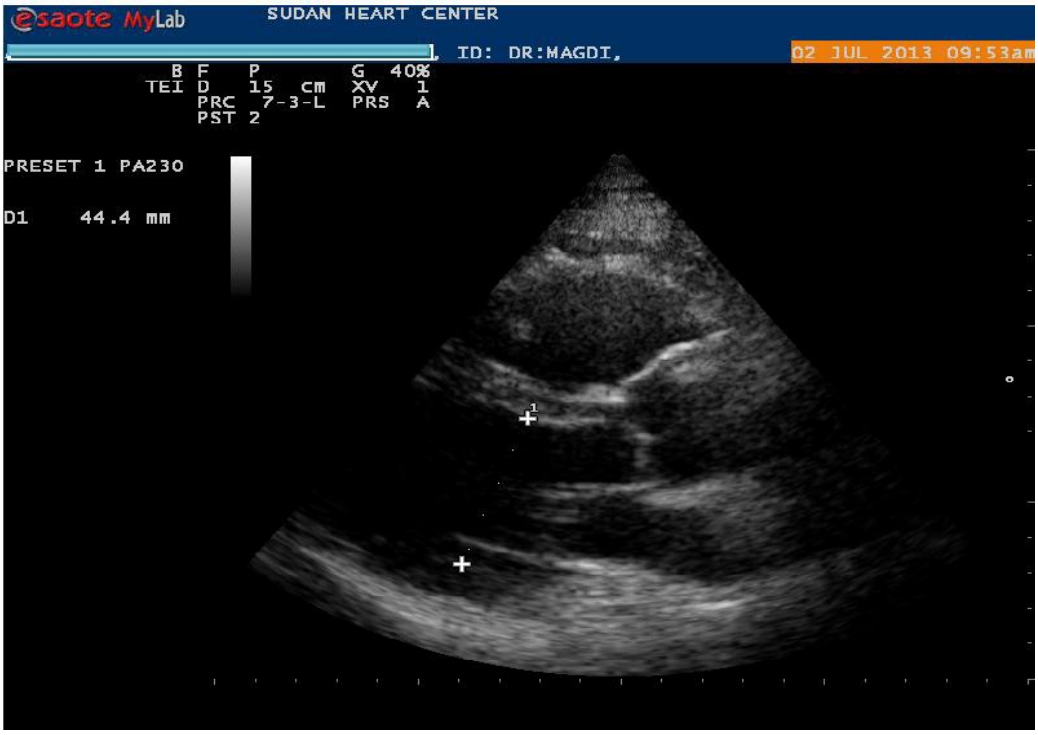


Figure 5.13 The LV diastolic measurement is 44.4mm.

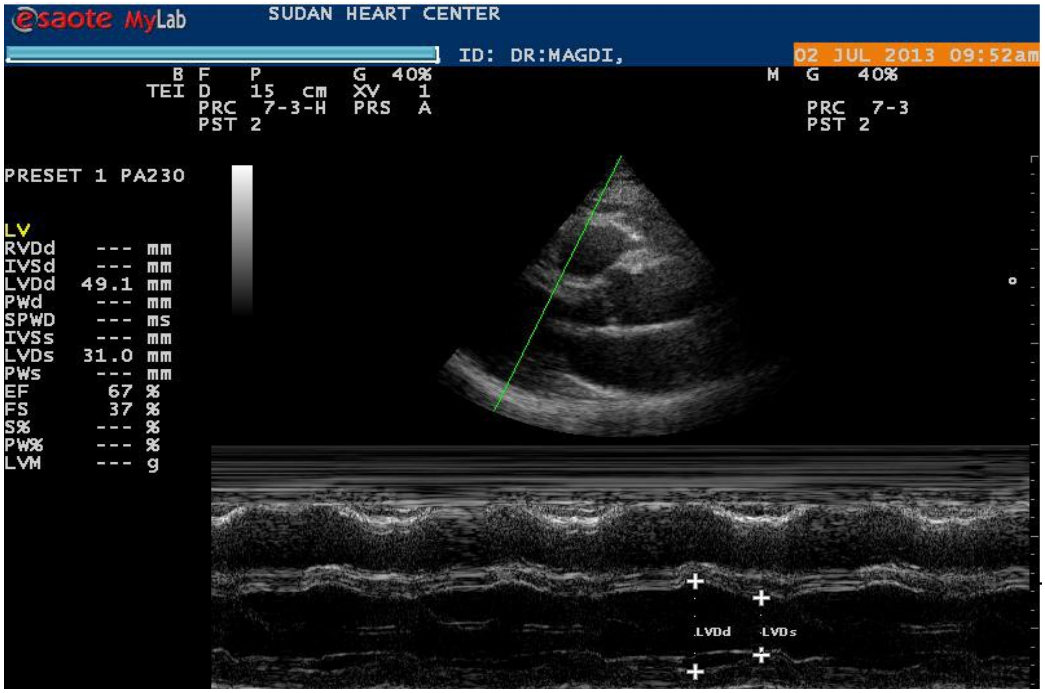
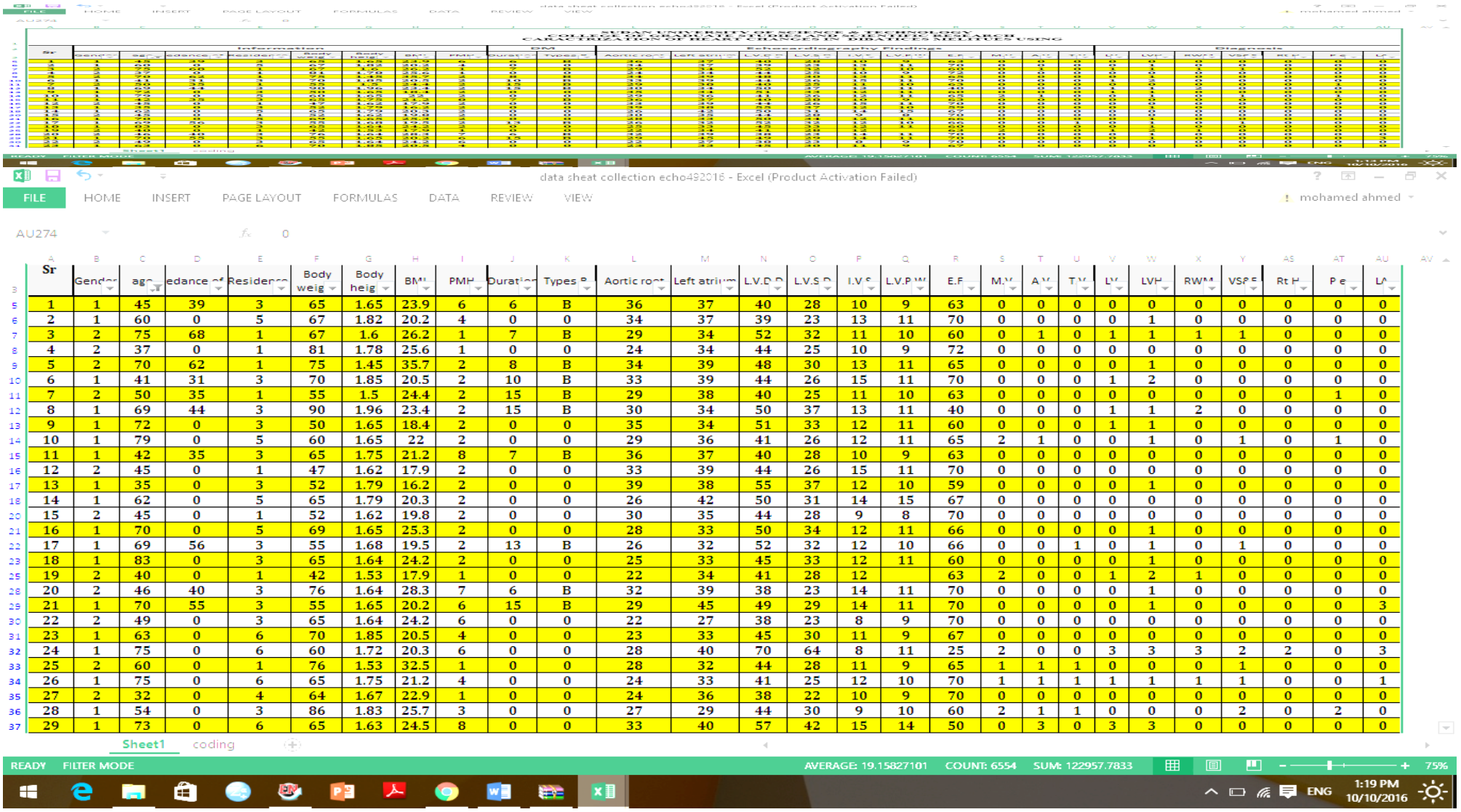


Figure 5.14 In the M-mode the LV diastolic measurement is 49.1mm and the systolic measurement is 31.0 mm, EF 67% all of which are normal.

Appendix (B) Data Collection Sheet



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1	1	45	39	3	65	1.65	23.9	6	6	B	36	37	40	28	10	9	63	0	0	0	0	0	0	0	0	0	0
2	1	60	0	5	67	1.82	20.2	4	0	0	34	37	39	23	13	11	70	0	0	0	0	1	0	0	0	0	0
3	2	75	68	1	67	1.6	26.2	1	7	B	29	34	52	32	11	10	60	0	1	0	1	1	1	1	0	0	0
4	2	37	0	1	81	1.78	25.6	1	0	0	24	34	44	25	10	9	72	0	0	0	0	0	0	0	0	0	0
5	2	70	62	1	75	1.45	35.7	2	8	B	34	39	48	30	13	11	65	0	0	0	0	1	0	0	0	0	0
6	1	41	31	3	70	1.85	20.5	2	10	B	33	39	44	26	15	11	70	0	0	0	1	2	0	0	0	0	0
7	2	50	35	1	55	1.5	24.4	2	15	B	29	38	40	25	11	10	63	0	0	0	0	0	0	0	0	1	0
8	1	69	44	3	90	1.96	23.4	2	15	B	30	34	50	37	13	11	40	0	0	0	1	1	2	0	0	0	0
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27	2	32	0	4	64	1.67	22.9	1	0	0	24	36	38	22	10	9	70	0	0	0	0	0	0	0	0	0	0
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Sheet1 coding

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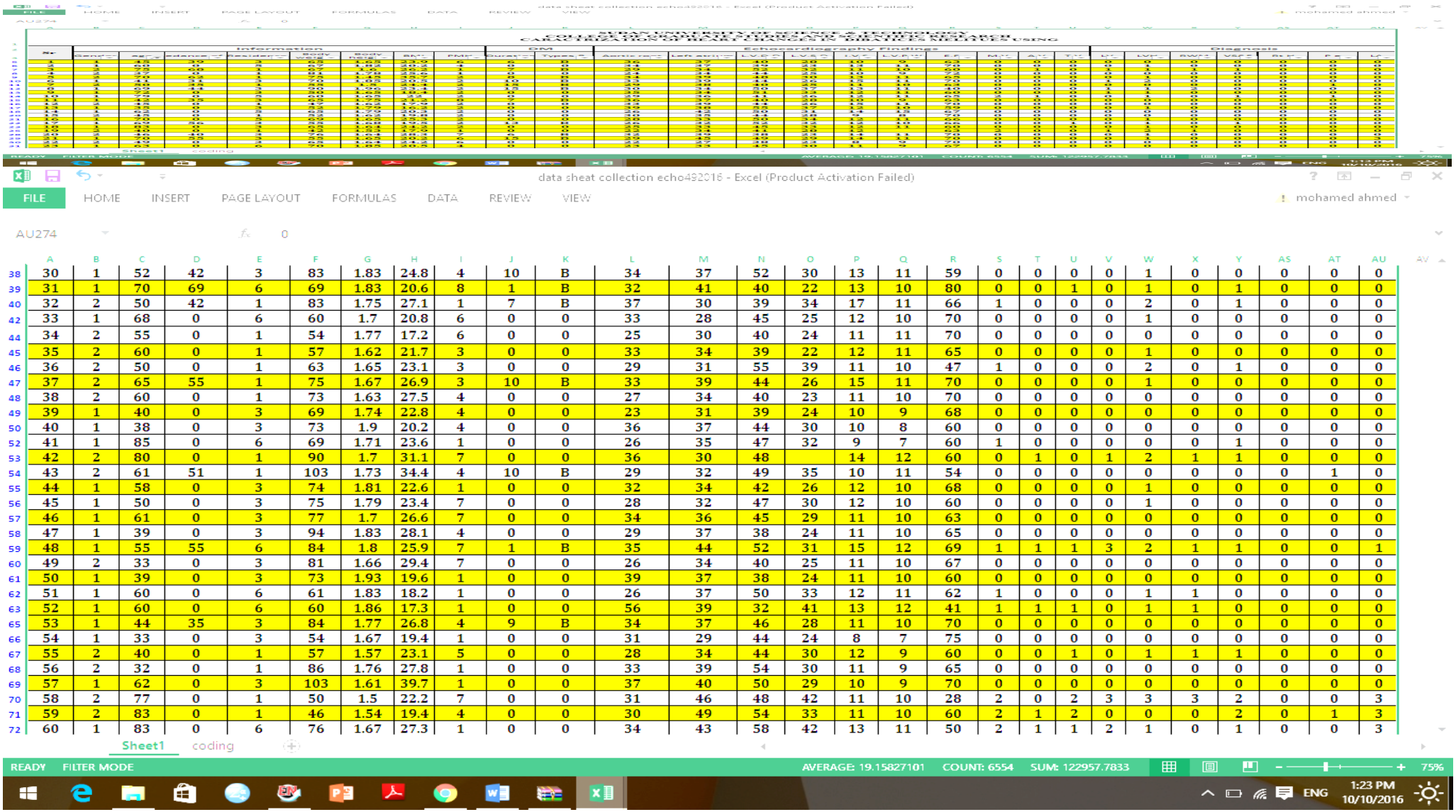
ENG 1:19 PM 10/10/2016



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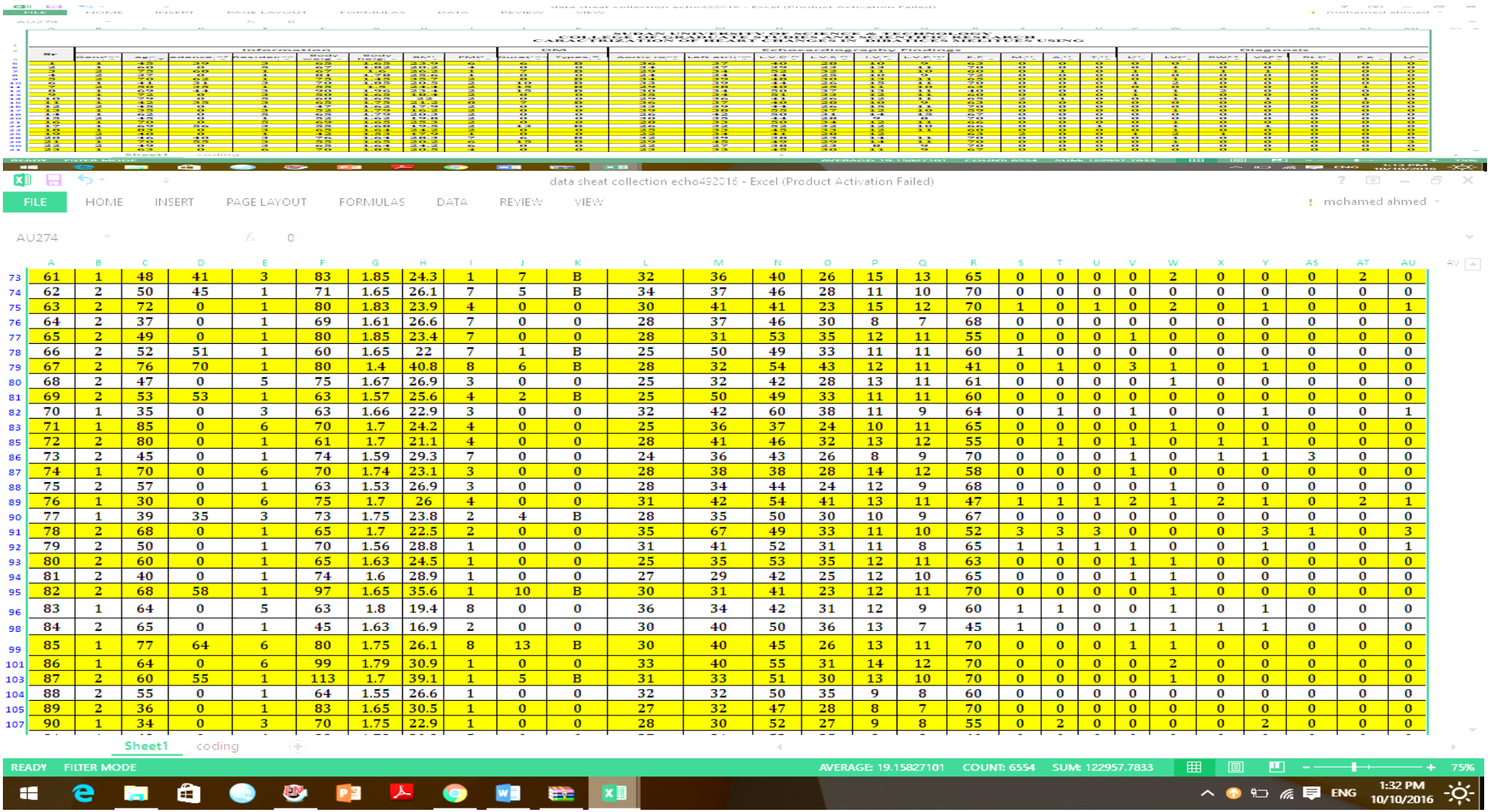

data sheet collection echo492016 - Excel (Product Activation Failed)

FILE HOME INSERT PAGE LAYOUT FORMULAS DATA REVIEW VIEW

mohamed ahmed

AU274 0

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Sheet1 coding

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ENG 1:32 PM 10/10/2016

Appendix (C) Published papers

Two papers were published:

1- Mohammed A. Alhaj, Moawia Gameraddin, AlsafiAhmed, Mahmoud S. Babiker. The assessment of echocardiographic findings of diabetes in Sudanese, Scholars Journal of Applied Medical Sciences (SJAMS), DOI: 10.21276/sjams.2016.4.8.13

2- Asma I. Ahmed, Mohamed A. Elhaj, Elsafi A. Abdullah, Mohamed Omer, Moawia G. Elddin, A. Hassan A. B Characterization of Heart wall Diseases in Patients with Type-2 Diabetes Mellitus using Echocardiography International Journal of Science and Research (IJSR) ISSN (Online): 2319-7064