



Sudan University of Sciences & Technology



College of graduate studies

**Evaluation of the Morphology and Function
of Heart In Hypertensive Patients Using
Echocardiography**

تقويم شكل ووظيفة القلب لمرضى إرتفاع ضغط الدم باستخدام الموجات فوق الصوتية للقلب

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الآية

بسم الله الرحمن الرحيم

اقْرَأْ بِاسْمِ رَبِّكَ الَّذِي خَلَقَ (1) خَلَقَ الْإِنْسَانَ مِنْ عَلَقٍ (2) اقْرَأْ وَرَبُّكَ

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سورة العلق (1-5)

صدق الله العظيم

Detected

To :

My father

My mother

My brothers

My sisters

My husband

My children

My Friends

My colleagues

With love

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First of all, my thanks should go to ALLAH who creates us from nothing.

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Abstract

The objective of this study was to evaluate the morphology and function of the heart chambers in hypertensive patients using echocardiography. In this study, a total of 200 primary or essential hypertensive patients were selected and divided into two groups (100 patients with controlled hypertensive patients and other 100 patients with uncontrolled hypertensive patients. Ages ranged from ≤ 50 up to 95 years. The studies were done by echocardiography machine in King Abdul-Aziz Specialist hospital-Taif in Saudi Arabia. The bio- data, included (age ,gender and period of hypertension in general (5-10)years), were collected by collected data sheet. Data analyzed by SPSS (version 16) .

The result of this study revealed that:

-The effectiveness of advancing age and treatment of hypertension on both sample's groups in age ≤ 50 was (11 %) in uncontrolled group and 18% in controlled group .While in age ranging (50-65) in uncontrolled was (59%) and (51%) in controlled group although in age ranging (66-95%) was (30%) in uncontrolled group and (31%) in controlled group.

- The effects of hypertension on different rates of heart ejection fraction on both sample's groups. And the result was demonstrated the there was indicator of heart failure in case of low (3%) and normal (90%) ejection fraction in uncontrolled group while in controlled group was (3%) in case of low and (92%) in case of normal ejection fraction which called heart failure with normal ejection fraction or heart failure preserved ejection fraction and there was indicator of hypertrophic cardiomyopathy in uncontrolled (7%) more than controlled group (5%).

- There was increased percentage of high EF specially in middle age groups (**50-65**) years in uncontrolled of hypertension which represented (57.1%) more than controlled (40%) while the percentage in age groups (65-95) years was formed

(42.1%) in uncontrolled group and (60%) in controlled and there were no patients in age < 50 years .

-The left ventricular Hypertrophy had affected in different rates of ejection fraction in both sample`s group and there was indicator of heart failure in low ejection fraction (66.7%) and heart failure preserved ejection fraction in normal ejection fraction (45.6%) in uncontrolled group. And in controlled group the percentage was (66.7%) in low ejection fraction and (60.9%) in normal ejection fraction. But there was no directly relationship between left ventricular hypertrophy and high ejection fraction although there was indicator of hypertrophic cardiomyopathy can be developed by hypertension in uncontrolled group the percentage was (28.6%) and in controlled group was (80%) .

- The relationship between the left ventricular hypertrophy with advancing age and gender on both sample`s group especially in middle age ranging (50-65) years (69.1%) in uncontrolled and (50%). There was increase in percentage of female more than male in both sample`s group. In uncontrolled group represented (53.3%) and in controlled group represented (51.6%).

- The left ventricular diastolic dysfunction had effect in different rates of heart ejection fraction on both sample`s group and there was indicator of heart failure in low ejection fraction (66.7%) was equalized percentage on both sample`s . The indicator of heart failure preserved ejection fraction in normal ejection fraction was represented (75.6%) in uncontrolled group and (69.6%) in controlled group.

- In left ventricular diastolic dysfunction indicator of hypertrophic cardiomyopathy in case of high ejection fraction in uncontrolled group was (85.7%) and in controlled was (100%).

-The left ventricular diastolic dysfunction had relationship with age and gender. The percentage was increased in middle age ranging (50-65) years on both sample`s group in uncontrolled group was (64.5) and in controlled group was

(50.7%) . And there was increased in females more than males in uncontrolled group (51.3%) in females and (48.7%) in males while vice versa in controlled was (42.3%) in female and 57.7%) in males.

-The involving of left ventricular diastolic dysfunction with left ventricular hypertrophy in controlled was (49.3%) in nil left ventricular hypertrophy and (50.7%) in case of left ventricular hypertrophy. And sometimes the left diastolic dysfunction could occur without left ventricular hypertrophy especially in uncontrolled group (67.1%) in nil left ventricular hypertrophy and (32.9%) in left ventricular hypertrophy.

In conclude, the study showed that the patients with common complications of hypertension on the heart can be evaluated by using noninvasive method transthoracic echocardiography easily without radiation hazard like can be acquired from in Nuclear medicine investigations.

المستخلص

تهدف هذه الدراسة لتقييم شكل ووظيفة بطينات للقلب لمرضى ارتفاع ضغط الدم باستخدام الموجات فوق الصوتية للقلب . في هذه الدراسة (200 مريض) مصابين بارتفاع ضغط الدم الأولي أو الأساسي تم إختيارهم وتقسيمهم إلى (100مرضى غير محكمين في أخذ علاج ارتفاع ضغط الدم و100 مرضى متحكمين في أخذ علاج ارتفاع ضغط الدم) . معدل الأعمار $50 \geq 95$ سنة. الحالات تم عمل فحوصات لها بجهاز الموجات فوق الصوتية للقلب بمستشفى الملك عبد العزيز بالطائف -المملكة العربية السعودية . البيانات الحيوية للمرضى، أضمنت (العمر والجنس وفترة ارتفاع ضغط الدم بصورة عامة 5-10 سنة)، حيث كان تجميعها بواسطة نموذج جمع البيانات. وحلت بواسطة برنامج التحليل الإحصائي (SPSS) نسخة (16).

النتائج لهذه الدراسة كشفت عن :

- مدى تأثير تقدم العمر وعلاج ارتفاع ضغط الدم في كلا من مجموعتي الدراسة في الأعمار ما بين أقل من أو يساوي 50 بنسبة (11%) في مجموعة غير المتحكمين في أخذ علاج ارتفاع ضغط الدم و (18%) في مجموعة المتحكمين في أخذ علاج ارتفاع ضغط الدم ، بينما في مدى العمر (50-65) سنة في مجموعة غير المتحكمين في أخذ علاج ارتفاع ضغط الدم كانت النسبة (59%) و(51%) في مجموعة المتحكمين في أخذ علاج ارتفاع ضغط الدم ، بالرغم من أن مدى العمر (66-95) سنة كانت النسبة من (30%) في مجموعة غير المتحكمين في أخذ علاج ارتفاع ضغط الدم و(31%) في مجموعة المتحكمين في أخذ علاج ارتفاع ضغط الدم.
- كان هنالك التأثير من ارتفاع ضغط الدم على مختلف معدلات كفاءة القلب في كلا من مجموعتي الدراسة مع وجود مؤشر إلى فشل القلب في حالة انخفاض كفاءة القلب بنسبة (3%) وفي الحالة الطبيعية لمعدل كفاءة القلب بنسبة (90%) في مجموعة غير المتحكمين في علاج ارتفاع ضغط الدم ، بينما في مجموعة المتحكمين في علاج ارتفاع ضغط الدم كانت بنسبة (3%) في حالة انخفاض معدل كفاءة القلب و(92%) في الحالة الطبيعية لمعدل كفاءة القلب والتي تسمى بفشل القلب مع المعدل الطبيعي لكفاءة القلب أو فشل القلب الوقائي لكفاءة القلب ، كما أن هنالك مؤشر لتضخم القلب الإعتلالي في مجموعة غير المتحكمين في علاج ارتفاع ضغط الدم بنسبة (7%) أكثر من مجموعة المتحكمين في علاج ارتفاع ضغط الدم بنسبة (5%).
- هنالك زيادة في نسبة ارتفاع معدل كفاءة القلب خصوصا في منتصف الأعمار (50-65) سنة للمجموعتين حيث مجموعة غير المتحكمين في علاج ارتفاع ضغط الدم ، مثلوا (57.1%) وكانوا أكثر من مجموعة المتحكمين في ارتفاع ضغط الدم (40%) ، بينما النسبة في الأعمار بين (65-95) سنة كانت (42.9%) في مجموعة غير المتحكمين في علاج ارتفاع ضغط الدم و(60%) في مجموعة المتحكمين في علاج ارتفاع ضغط الدم ، كما لا يوجد مرضى في عمر أقل من 50 سنة.
- تضخم البطين الأيسر لديه أثر على مختلف معدلات كفاءة القلب في كلا من المجموعتي الدراسة كما يوجد مؤشر لفشل القلب في حالة انخفاض معدل كفاءة القلب (66.7%) وفشل القلب الوقائي في الحالة الطبيعية لكفاءة القلب (45.6%) في مجموعة غير المتحكمين في علاج ارتفاع ضغط الدم،

في مجموعة المتحكمين في علاج ارتفاع ضغط الدم النسبة كانت (66.7%) في حالة انخفاض معدل كفاءة القلب و(60.9%) في الحالة الطبيعية لكفاءة القلب. لكن لا توجد هناك علاقة مباشرة بين تضخم البطين الأيسر وارتفاع معدل كفاءة القلب بالرغم من أن هنالك مؤشر لتضخم القلب الإعتلالي والذي يمكن أن يتطور بواسطة ارتفاع ضغط الدم في كلا من مجموعتي الدراسة حيث (53.3%) في مجموعة غير المتحكمين في علاج ارتفاع ضغط الدم و(28.6%) في مجموعة المتحكمين في علاج ارتفاع ضغط الدم .

- هنالك علاقة بين تضخم البطين الأيسر مع تقدم العمر والجنس في كلا من مجموعتي الدراسة ، وخصوصا في منتصف الأعمار ما بين (50-65) سنة حيث كانت (66.1%) في مجموعة غير المتحكمين في علاج ارتفاع ضغط الدم و(50%) في مجموعة المتحكمين في علاج ارتفاع ضغط الدم. هنالك زيادة في نسبة النساء أكثر من الرجال في كلا مجموعتي الدراسة . مجموعة غير المتحكمين في علاج ارتفاع ضغط الدم مثلت (53.3%) نساء و (46.7%) رجال بينما مجموعة المتحكمين في علاج ارتفاع ضغط الدم مثلت (51.6%) نساء و(48.4%) رجال .

- صعوبة الأتساع للبطين الأيسر لديه تأثير على مختلف معدلات كفاءة القلب في كلا مجموعتي الدراسة، وأن هنالك مؤشر لفشل القلب في حالة انخفاض كفاءة القلب (66.7%) مع تساوي النسبة في كلا من مجموعتي الدراسة، هنالك مؤشر لفشل القلب الوقائي لكفاءة القلب في الحالة الطبيعية لكفاءة القلب والذي مثل (75.6%) في مجموعة غير المتحكمين في علاج ارتفاع ضغط الدم و(69.6%) في مجموعة المتحكمين في علاج ارتفاع ضغط الدم .

- في صعوبة الأتساع للبطين الأيسر هنالك مؤشر لتضخم القلب الإعتلالي في حالة ارتفاع كفاءة القلب (في مجموعة غير المتحكمين كانوا (85.7%) و في مجموعة المتحكمين في علاج ارتفاع ضغط الدم كانوا 100%) .

- صعوبة الأتساع للبطين الأيسر لديه علاقة مع العمر والجنس. وكانت النسبة متزايدة في منتصف الأعمار (50-65) سنة في كلا مجموعتي الدراسة ، في مجموعة غير متحكمين في علاج ارتفاع ضغط الدم كانوا (64.5%) و في مجموعة المتحكمين في علاج ارتفاع ضغط الدم كانوا (50.7%) وهنالك كانت الزيادة في النساء أكثر من الرجال في مجموعة غير المتحكمين، (51.3%) في النساء و(48.7%) في الرجال ، بينما في مجموعة المتحكمين (42.3%) في النساء و(57.7%) في الرجال.

- هنالك ارتباط بين صعوبة الأتساع للبطين الأيسر مع تضخم البطين الأيسر في مجموعة المتحكمين (49.3%) في حالة عدم وجود تضخم البطين الأيسر (50.7%) في حالة وجود تضخم للبطين الأيسر، بعض الأحيان صعوبة الأتساع للبطين الأيسر يمكن أن يحدث من غير حدوث تضخم في البطين الأيسر، خصوصا في مجموعة غير المتحكمين (67.1%) في حالة عدم وجود تضخم للبطين الأيسر و (32.9%) في حالة وجود تضخم للبطين الأيسر .

- خلّصت هذه الدراسة الى ايضاح أن المرضى الذين لديهم المضاعفات الأكثر شيوعا لإرتفاع ضغط الدم مثل (تضخم البطين الأيسر وفشل البطين الأيسر الإنبساطي) يمكن أن يتم تقييمهم بإستخدام طريقة غير ضارة من خلال خارج الصدر بجهاز الموجات فوق الصوتية للقلب بسهولة ومن غير التعرض للإشعاع كالتالي يمكن أن يتعرضوا لها في فحوصات الطب النووي.

List of abbreviations

2 D	Two dimensional
3 D	Three dimensional
4 D	Four dimensional
A II	Angiotensin II
AHA	American Heart Association
A-mode	Amplitude mode
AO	Aorta
ASE	American Society of Echocardiography
A-V	Atrioventricular Valve
AV node	Aterioventricular node
B-mode	Brightness mode
Bp	Blood Pressure
BSE	British Society Echocardiography
Ca	Atrial Compliance
CHF	Congestive or Heart Failure
CKD	Chronic Kidney disease
CM	Centimeter
CPR	C-Creative protein
CSA	Cross-sectional Area

CW	Continuous wave Doppler
DCM	Dilated Cardiomyopathy
E/A ratio	The ratio of the early (E) to late (A) ventricular filling velocities
EABV	Effective Arterial Blood Volume
ECG	Electrocardiography
Echo	Echocardiography
EF	Ejection Fraction
Equ	Equation
ESV	End Systolic Volume
EVD	End Diastolic Volume
FDA	Food and Drugs Administration
Fig	Figure
G	Gram
HCM	Hypertrophic Cardiomyopathy
HF	Heart Failure
HFNEF	Heart failure with preserved ejection fraction
HFpEF	Heart failure with preserved ejection fraction
HFREF	heart failure with reduced ejection fraction
HNT	Hypertension
ISH	Isolated Systole Hypertension

IVC	Inferior Vena Cava
IVRT	Isovolumic Relaxation Time
IVS	Interventricular Septum
JNC7	Seventh Report of the Joint National Committee
KAASH	King AbdulAziz Specialist Hospital
KHz	Kilo Hertiz
kPa	Kilo Pascal
LA	Left Atrium
LAX	Long Axis
LBB	Left Bundle Branch
LV	Left Ventricle
LVDD	Left Ventricular Diastolic Dysfunction
LVEF	Left Ventricular Ejection Fraction
LVEF	Left ventricle
LVH	Left Ventricular Hypertrophy
LVOT	Left Ventricular Out flow
MI	Mechanical Index
mm HG	Millimeter of mercury
M-mode	Motion mode
MV	Mitral Valve

NHANES	National Health and Nutrition Examination
NHS	National Health Service
Nil	Non
O ₂	Oxygen
P	Pressure
PA	Pulmonary artery
pEF	Preserved Ejection Fraction
Pts	Patients
PW	Power wave Doppler
RA	Right Atrium
RBB	Right Bundle Branch
RV	Right Ventricle
RVOT	Right Ventricular outflow
S ₃	Third heart Sound
SA node	Sinuatrial node
SAX	Short Axis
SEK	Swedish krona
SPSS	Statistical Package for the Social Sciences
SPTA	Special Peak Temporal average
SV	Stroke Volume

TAPSE	Tricuspid Annular Plane Systolic Excursion
TDI	Tissue Doppler Imaging
TGC	Time Gain Compensator
TOE	Transoesophageal
TPR	Total Peripheral Resistance
TTE	Transthoracic Echocardiography
UK	United Kingdom
V	Volume
VSD	Ventricular Septal defect
VTI	Velocity Time integral

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Chapter One

Introduction

Chapter One

1.1 Introduction:

Hypertension (HTN) is considered the silent killer cause of death all over the world. It is a serious health condition that can lead to serious life-threatening problems . Monitoring of early pathological changes in heart chambers, valve and myocardium muscles may reduce the severe complications which lead to death.

Hypertensive heart disease includes a number of complications of high blood pressure that affect the heart. While there are several definitions of hypertensive heart disease in the medical literature, the term is most widely used in the context of the International Classification of Diseases (ICD) coding categories. The definition includes heart failure and other cardiac complications of hypertension when a causal relationship between the heart disease and hypertension is stated or implied on the death certificate. In addition Hypertensive heart disease is the result of structural and functional adaptations leading to left ventricular hypertrophy, diastolic dysfunction, CHF . In 2013 hypertensive heart disease resulted in 1.07 million deaths up to 630,000 in 1990(*Donnelly , 2013*).

Echocardiography is a unique noninvasive method for imaging the living heart. It is based on detection of echoes produced by a beam of very high frequency ultrasound pulses transmitted into the heart.

From its introduction in 1954 to the mid 1970's, most echocardiographic studies employed a technique called M-mode, in which the ultrasound beam is aimed manually at selected cardiac structures to give a graphic recording of their positions and movements.

M-mode recordings permit measurement of cardiac dimensions and detailed analysis of complex motion patterns depending on transducer angulation. They also facilitate analysis of time relationships with other physiological variables such as ECG, heart sounds, and pulse tracings, which can be recorded simultaneously.

A more recent development uses electromechanical or electronic techniques to scan the ultrasound beam rapidly across the heart to produce two-dimensional tomographic images of selected cardiac sections. This gives more information than M-mode about the shape of the heart and also shows the spatial relationships of its structures during the cardiac cycle.

A comprehensive echocardiographic examination, utilizing both M-mode and two dimensional recordings, therefore provides a great deal of information about cardiac anatomy and physiology, the clinical value of which has established echocardiography as a major diagnostic tool (*Rani Gera,2015*).

1.2 Problem of the study:

Hypertension can affected heart chambers and valves especially if it takes long duration or does not treated or controlled.

heart studying can be performed, using different modalities e.g nuclear medicine techniques and cardiac catheterization . But all were invasive and of high radiation dose risk. so this study will be an attempt for replacing that method with another noninvasive technique .

1.3 General objective:

To Evaluate the morphology and function of heart in hypertensive patients using echocardiography.

1.3.1 Specific objectives:

- To evaluate or characterize the common complications of hypertension on the heart chambers.
- To assess ejection fraction rate.
- To measure all variables of echocardiography related to patient age ,gender
- To classify the findings according to the causes(cross tabulation).

1.4 Outlines of Thesis :

In chapter one, the introduction, objectives, problem of study and the aim of study are presented. This included general introduction concepts about the effect of hypertension on heart and the role of echocardiography in diagnosis. In chapter two, basic theoretical background is given. It includes an overview of anatomy, physiology, pathology, Physical characteristic of echocardiography machine and techniques. The materials and methods are presented in Chapter three. In chapter four the results are included, followed by discussion, conclusions and recommendations in chapter five.

Chapter Two

Theoretical Background and Previous study

Chapter Two

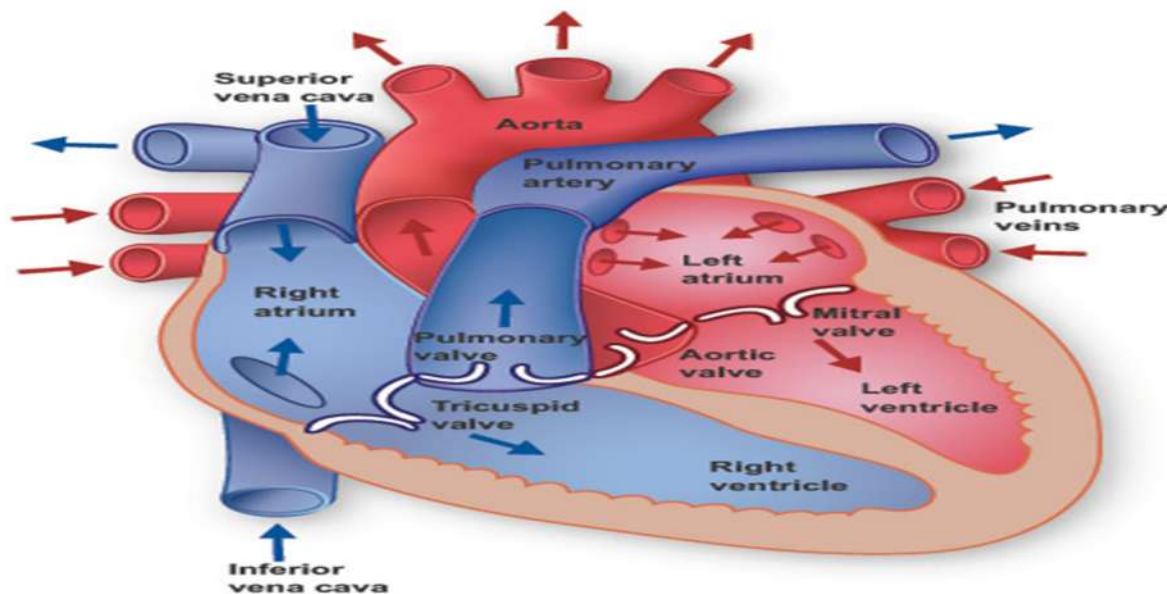
Theoretical Background

2.1 Anatomy

2.1.1 The heart:

2.1.1.1 Introduction:

The heart is a pair of valved muscular pumps combined in a single organ (**Figure 2.1,A**). Although the fibromuscular framework and conduction tissues of these pumps are structurally interwoven, each pump (the so-called 'right' and 'left' hearts) is physiologically separate, and is interposed in series at different points in the double circulation. Despite this functional disposition in series, the two pumps are usually described tomographically in parallel (*Susan Standring , 2008*).



(**Figure 2.1,A**) Structure of the heart, and course of blood flow through the heart chambers and heart valves (*Arthur C. Guyton, M.D & JOHN e. Hall ,ph.D, 2006*).

2.1.1.2 Position of the heart :

The heart is a hollow, fibromuscular organ conical or pyramidal form, with a base, apex and a series of surfaces and 'borders'. Enclosed in the pericardium, it

occupies the middle mediastinum between the lungs and their pleural coverings (Figure.2.1,A B and C). It is placed obliquely behind the body of the sternum and the adjoining costal cartilages and ribs .Approximately one-third of the mass lies to the right of the midline(*Susan Standring , 2008*).

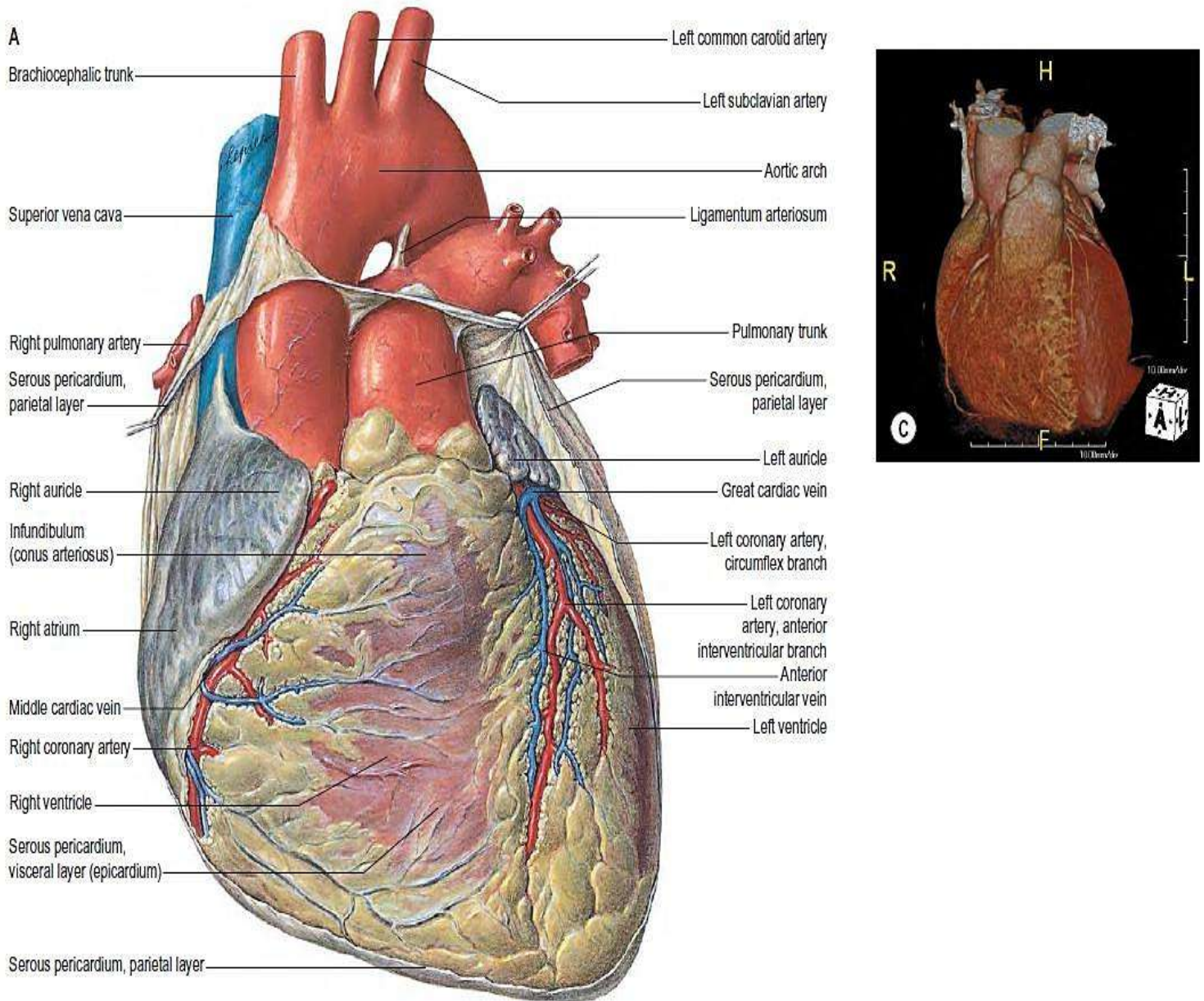


Figure 2.1,B The heart and great vessels : B, Anterior Postreior views with corresponding three-dimentional reconstructions from multislice CT scanning . (main figuers from Sobitta 2006) (*Susan Standring , 2008*).

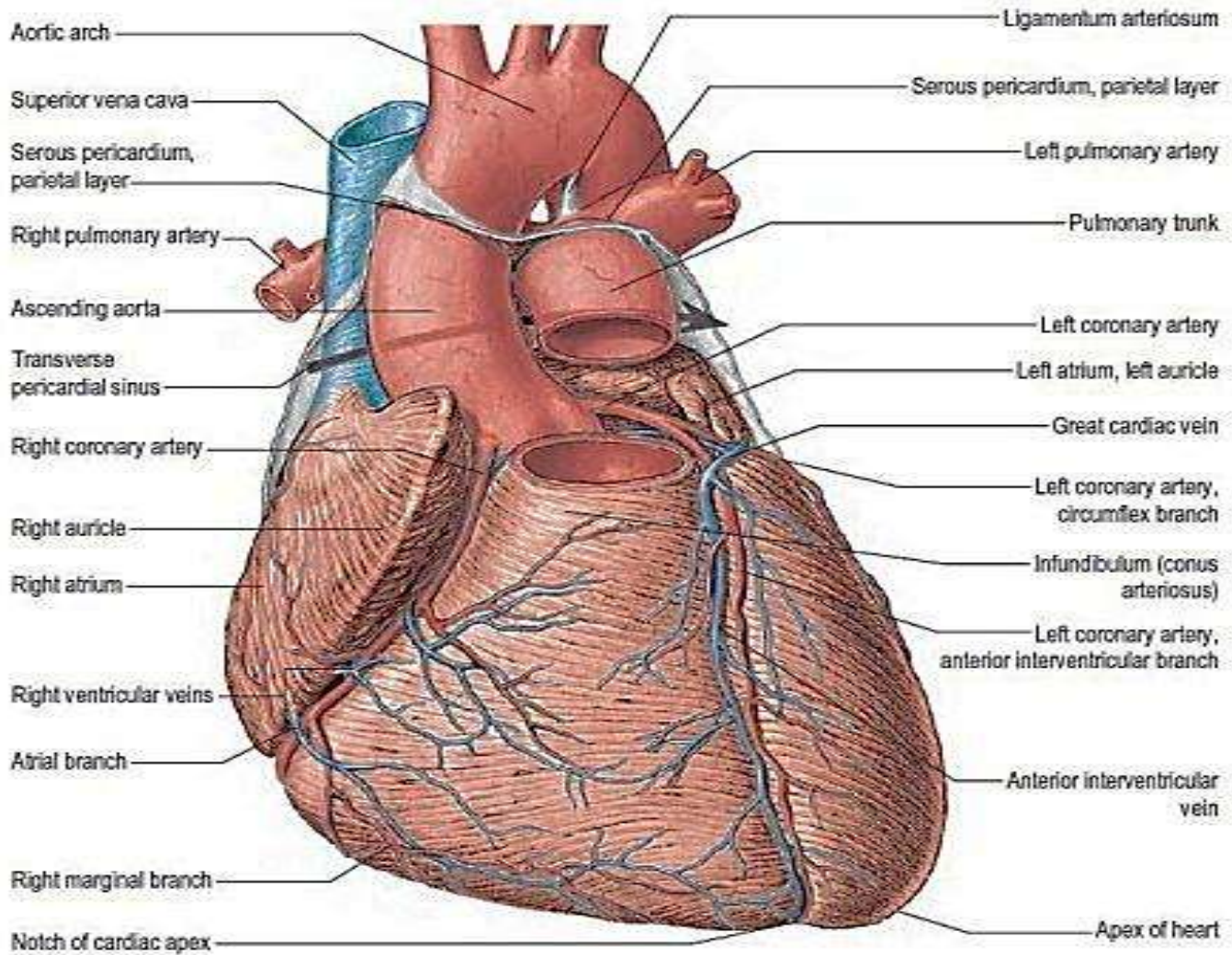


Figure 2.1,C The anterior or sternocostal surface of the heart (From Sobotta 2006)(Susan Standring , 2008).

2.1.1.3 Pericardium

The pericardium contains the heart and the juxtacardiac parts of its great vessels. It consists of two components, the fibrous and the serosal pericardium. The fibrous pericardium is a sac made of tough connective tissue completely surrounding the heart without being attached to it.

The serosal pericardium consists of two layers of serosal membrane, one side the other: the inner (viseral) one adheres to the heart and forms its outer covering known as the epicardium, whereas the outer (parietal) one lines the

internal surface of the fibrous pericardium. The two serosal surfaces are apposed and separated by a film of fluid. This allows movement of the inner membrane and the heart adhering to it. The separation of the two membranes of the serosal pericardium creates a narrow space, the pericardial cavity, which provides a complete cleavage between the heart and its surroundings and so allows it some freedom to move and change shape (Figure 2.2, A and B) (Susan Standring, 2008).

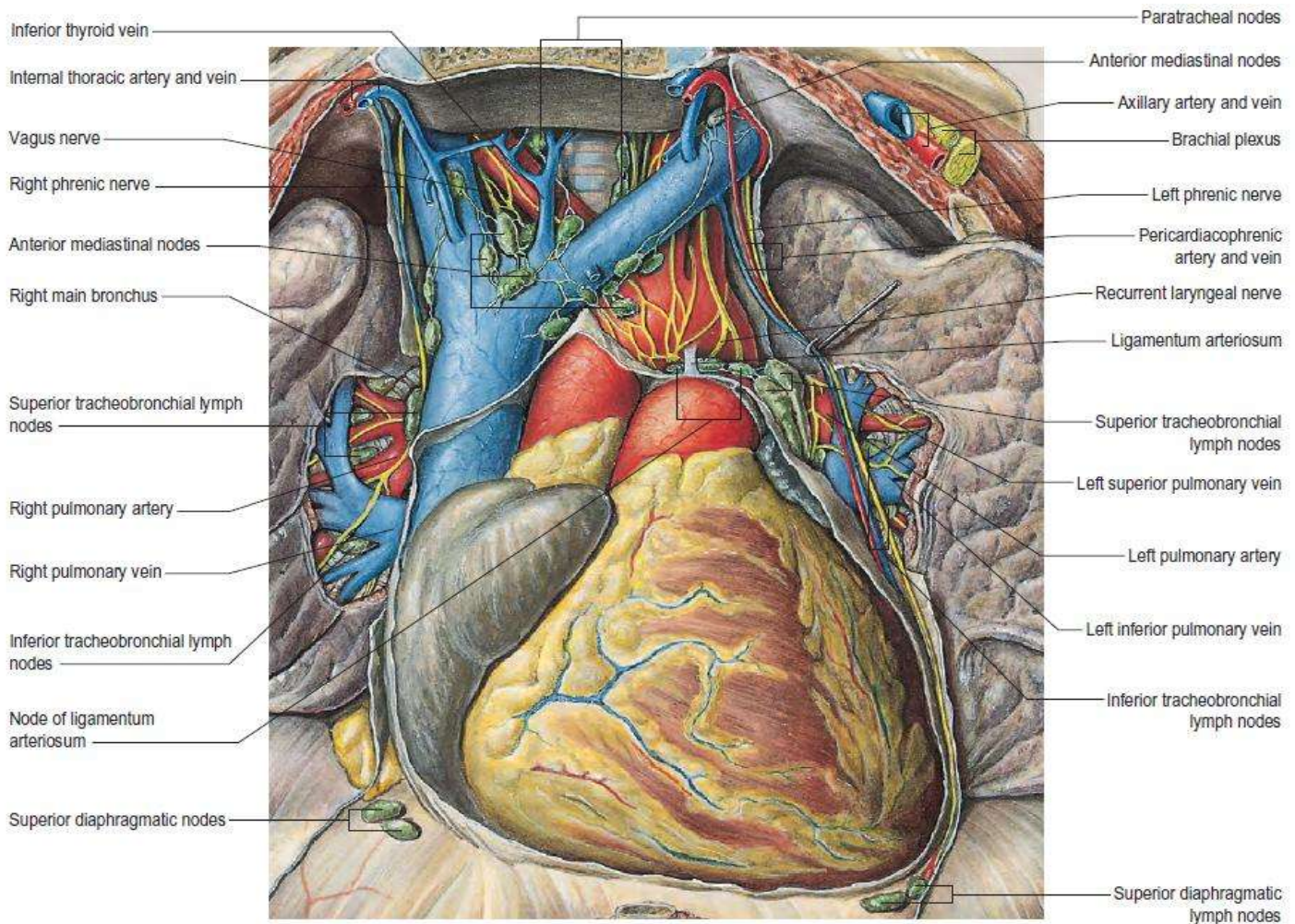


Figure 2.2, A Dissection that displays the heart, the great vessels and the lungs in situ: the manubrium sterni has been retracted cranially and the thymus has been removed totally. The pericardium has been partially removed and the helium of the lung has been dissected to expose the tracheobronchial lymph nodes (From Sobotta 2006) (Susan Standring, 2008).

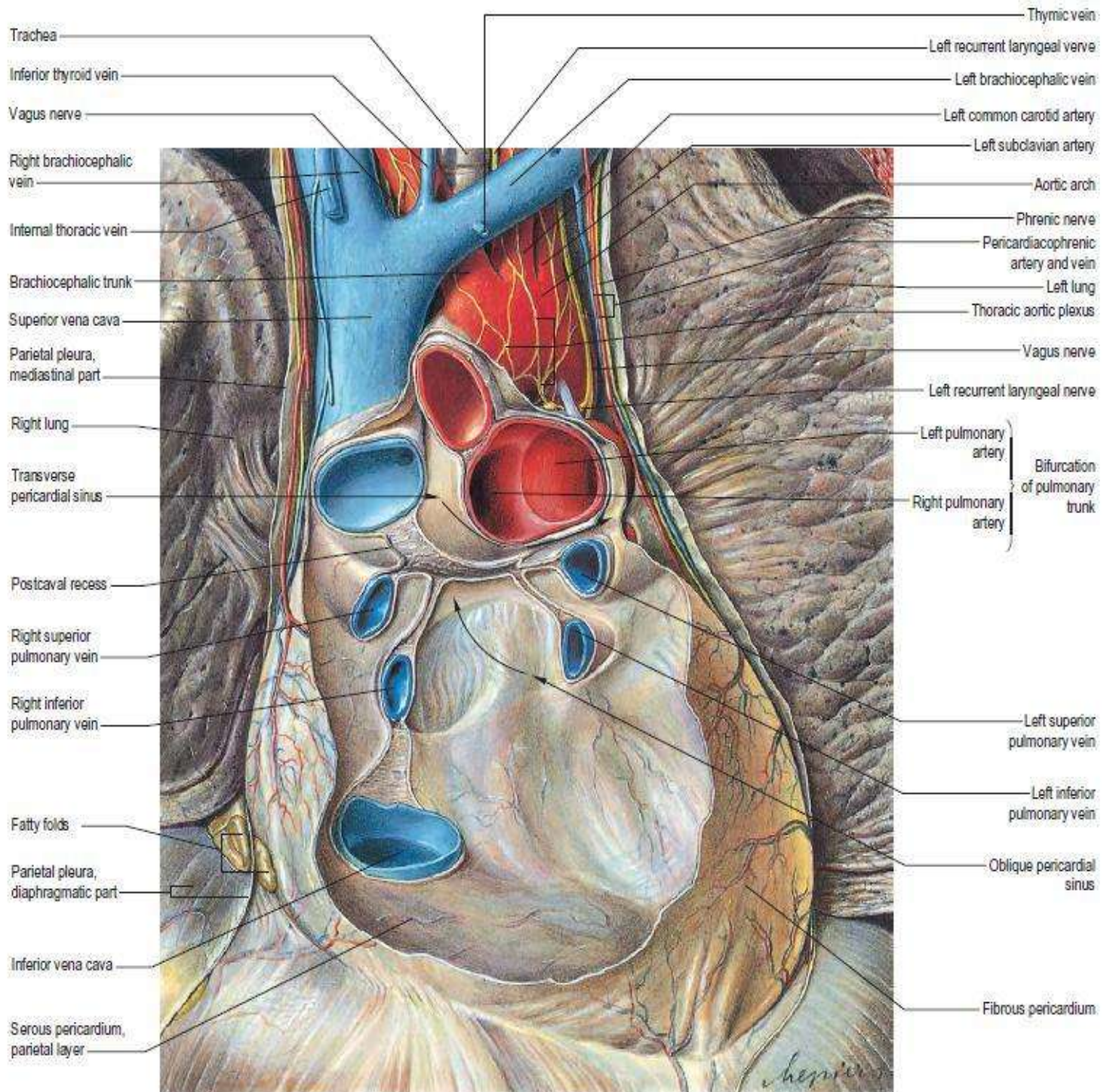


Figure 2.2, B Inferior of the serosal pericardial sac after section of the large vessels at their cardiac origin and removal of the heart (see from the front). See text for additional named recesses of the general serosal pericardial cavity and its transverse sinus (From Sobotta 2006) (Susan Standring, 2008).

2.1.1.4 Cardiac size , shape and external features :

An average adult heart is 12 cm from base to apex ,8-9 cm at its broadest transverse diameter and 6 cm anteroposteriorly. Its weight varies from 280 to 340 g (average 300 g) in males and from 230 to 280 g (average 250 g) in females. Adult weight is achieved between the ages 17 and 20 years. The oblique position of the heart may be emphasized by comparing it to a rather deformed pyramid, with the base facing posteriorly and to the right , and the apex anteriorly and to the left .

The heart is described as having a base and apex, its surfaces being designated as sternocostal (anterior) diaphragmatic(inferior) and right and left(pulmonary) . Its borders are termed upper, inferior ('acute' margin or border) and left ('obtus' margin or border)(*Chummy S-Sinnatamby,2006*).

The heart is placed obliquely in the thorax .The atrial and ventricular septal structures are virtually in line, but inclined forwards and to the left at 45° to a sagittal plane. The planes of the mitral and tricuspid valves, although vertical and not precisely co-planar . The right atrium , therefore, is not only to the right , but also anterior and inferior to the left atrium. It is also partly anterior to the left ventricle , an important atrioventricular septum intervening . The right ventricle forms most of the anterior aspect of the ventricular mass (**Figure 2.1, A and B**) , only its inferior end is to the right of the left ventricle, its upper left extremity (pulmonary orifice) is to the left and superior relative to the aortic valve . The left atrium forms most of the posterior aspect of the heart, whereas the left ventricle is only prominent inferiorly ,running along the left margin to reach the apex. These general dispositions are of the greater importance in planning or interpreting radiographs, scans, angiocardiograms and echocardiograms(*Chummy S-Sinnatamby,2006*).

2.1.1.5 Cardiac base, apex, surfaces and borders:

Posterior aspect of the heart The true cardiac base is somewhat quadrilateral , with curved lateral extensions. It faces back and to the right, separated from the thoracic vertebrae (fifth to eight in the recumbent, sixth to ninth in the erect posture) by the pericardium ,right pulmonary veins ,oesophagus and aorta.It is formed mainly by the left atrium,and only partly by the posterior part of the right atrium (**Figure2.3**).It extends superiorly to the bifurcation of the pulmonary trunk and inferiorly to the posterior part of the atrioventricular groove,which contains the coronary sinus and branches of the coronary arteries. It is limited to the right and left by the rounded surfaces of the corresponding atria. These are separated by the shallow interatrial groove.

Two pulmonary veins on each side open into the left atrial part of the base,whereas the superior and the inferior vena cava open into the upper and lower parts of the right atrial basal region.The area of the left atrium between the opening of right and left pulmonary viens forms the anterior wall of the oblique pericardial sinus (**Figure2.3**)*From Gray's anatomy (Susan Standring , 2008).*

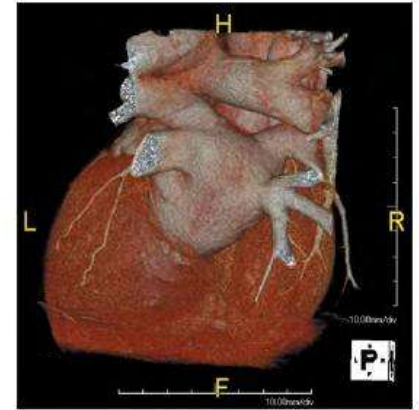
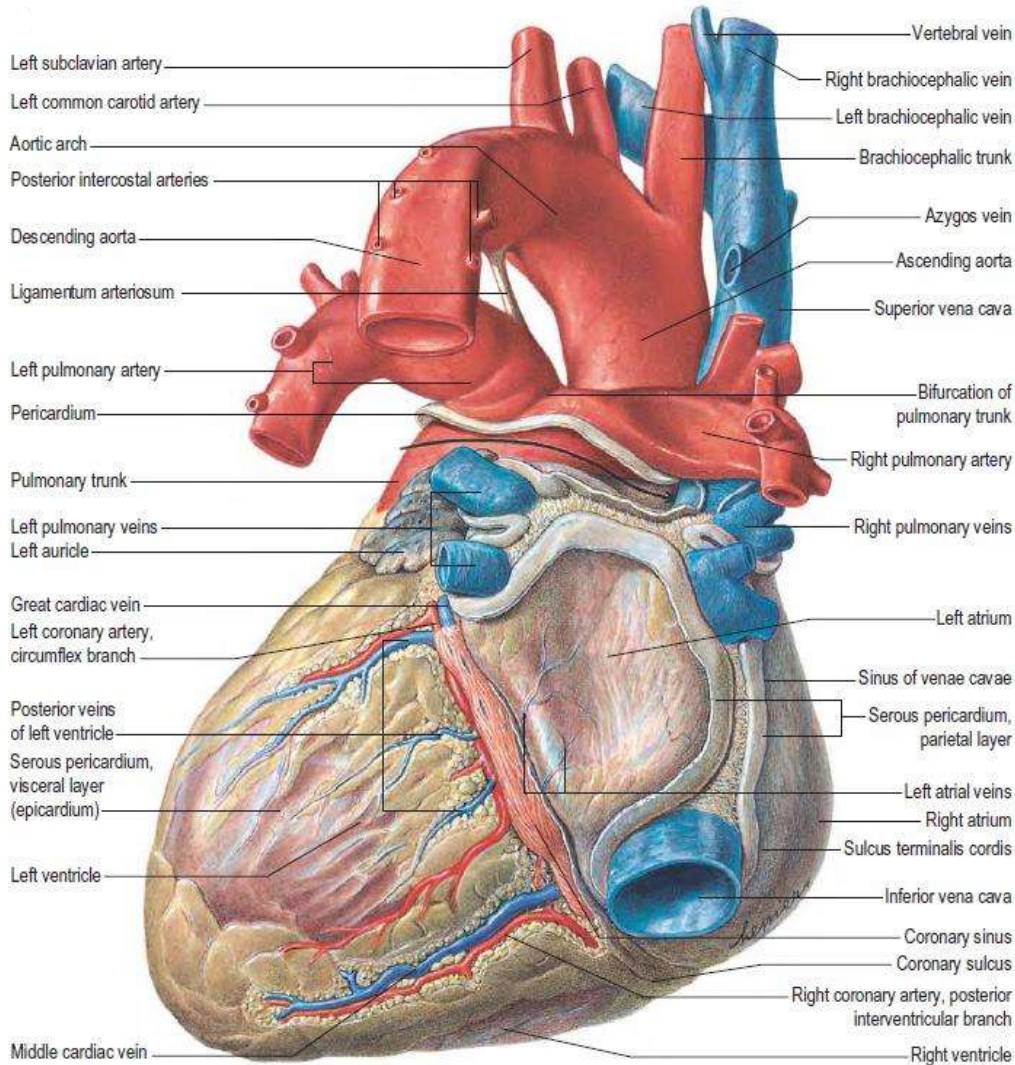


Figure 2.3 The heart and great vessels : A Postreior views (main figures from Sobotta 2006)(Susan Standing , 2008) .

2.1.1.5.1 Anatomical apex of the heart

This is the apex of the conical left ventricle, which is directed down, forwards and to the left . It is overlapped by the left lung and pleura. The apex is located most commonly behind the fifth left intercostal space, near or a little medial to the midclavicular line.

Anterior surface , Sternocostal surface of the heart Facing forwards and upwards, the anterior surface has an acute right and a more gradual left convexity . It consist of an atrial area above and to the right , and a ventricular part below and

to the left of the atrioventricular groove. The atrial area is occupied almost entirely by the right atrium. The left atrium is largely hidden by the ascending aorta and pulmonary trunk. Only a small part of the left appendage projects forwards to the left of the pulmonary trunk. Of the ventricular region, about one-third is made up by the left and two-thirds by the right ventricle. The site of the septum between them is indicated by the interventricular groove. The sternocostal surface is separated by the pericardium from the body of the sternum, the sternocostal muscles and the third to the sixth costal cartilages. Inferior, diaphragmatic surface of the heart Largely horizontal, the inferior surface of the heart slopes down and forwards a little towards the apex (**Figure.2.1,C**). It is formed by the ventricles (chiefly the left) and rests mainly upon the central tendon but also, apically, on a small area of the left muscular part of the diaphragm.

Left surface of the heart Facing up, back and to the left, the left surface consists almost entirely of the obtuse margin of the left ventricle, but a small part of the left atrium and its auricle contribute superiorly.

Right surface of the heart The right surface is rounded and formed by the right atrial wall. It is separated from the mediastinal aspect of the right lung by the pericardium and the pleural coverings. Its convexity merges below into the short intrathoracic part of the inferior vena cava and above into the superior vena cava.

Upper border of the heart This atrial (mainly the left atrium). Anterior to it are the ascending aorta and the pulmonary trunk (**Figure.2.1,C**).

Right border of the heart Corresponding to the right atrium, the profile of the right border is slightly convex to the right and it approaches the vertical.

Inferior border of the heart Also known as the acute margin of the heart, the inferior border is sharp, thin and nearly horizontal. It extends from the lower limit of the right border to the apex and it is formed mainly by the right ventricle, with a small contribution from the left ventricle near the apex.

Left border of the heart Also known as the obtuse margin, the left border separates the sternocostal and left surfaces. It is round and mainly formed by the left ventricle but slightly extent superiorly, convex to the left from the auricle to the cardiac apex(*Susan Standring , 2008*) and (*Chummy S-Sinnatamby,2006*).

2.1.1.6 Right atrium :

The interatrial septum (or atrial septum) is oblique, so the right atrium is anterior and to the right of the left atrium (**Figure.2.4,A**), The superior vena cava opens into its upper anterior part the inferior vena cava into its lower posterior part. An extensive muscular pouch ,the auricle,projects anteriorly to overlap the right side of the ascending aorta.The junction between the venous part (sinus venosus) and the atrium proper is marked externally by a shallow groove , the sulcus terminalis extending between the right sides of the openings of the two venae cavae.

Anteriorly , the right atrium is related to anterior part of the mediastinal surface of the right lung . Laterally , the atrium is also related to the mediastinal surface of the right lung ,but anterior to its hilum and separated from it by the pleura,right phrenic nerve and pericardiophrenic vessels and pericardium .Posteriorly and to the left , the atrial septum and the surrounding infolded atrial walls separate the right from the left atrium (the mural infolding is indicated by an extensive interatrial groove). Posteriorly and to the right are the right pulmonary veins. Medially are the ascending aorta and to a lesser extent the root of the pulmonary trunk and its bifurcation(*Chummy S-Sinnatamby,2006*).

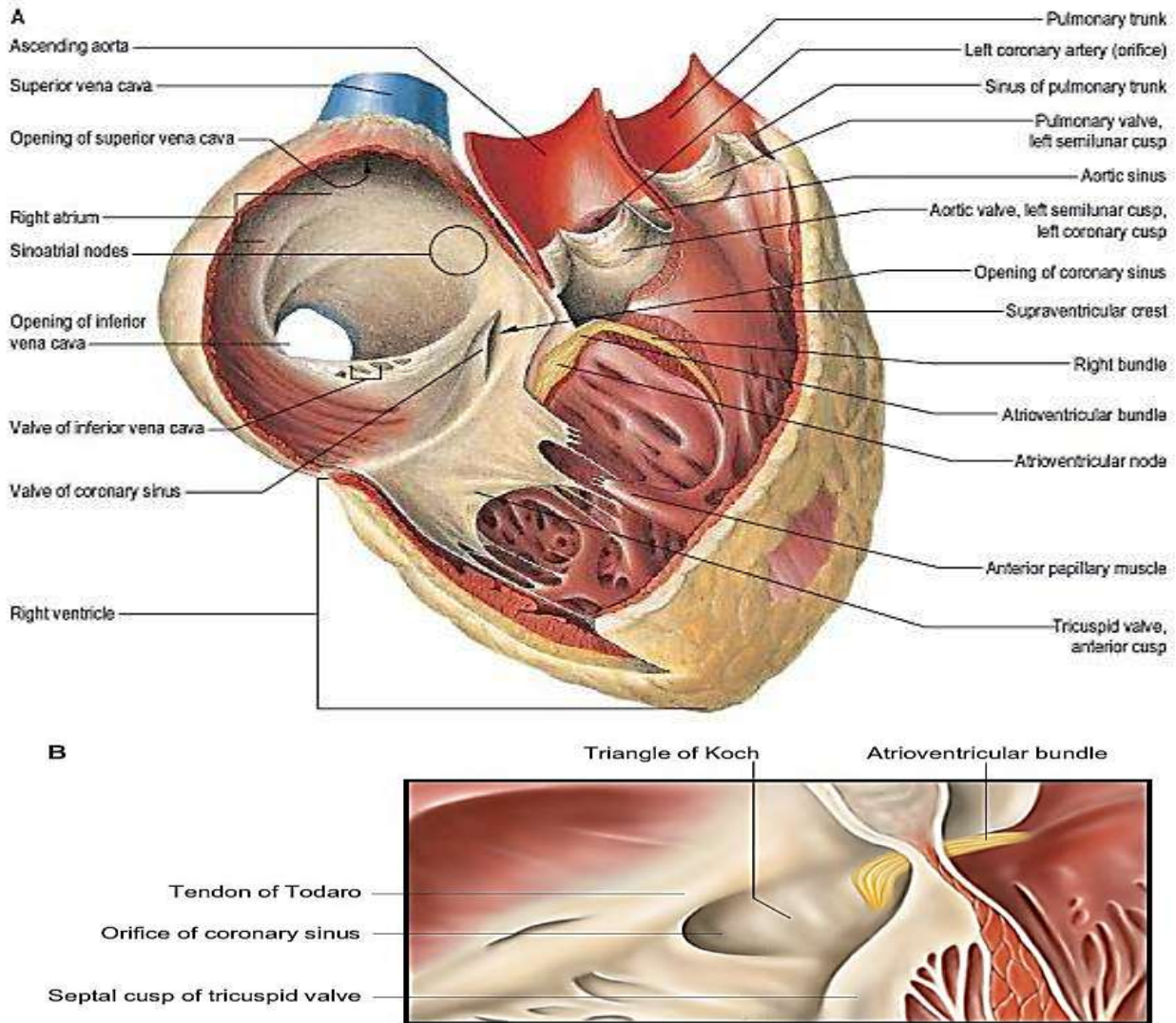


Figure.2.4,A,B The inferior of the heart, revealed by incising it along its right and part of the lower surface. The rest of the heart has been turned over to the left. **B**, the triangle of koch, which is defined by the tendon of Todaro, orifice of the coronary sinus and the septal cusp of the tricuspid valve. (A from Sobotta 2006) (Susan Standring , 2008) .

2.1.1.6.1 Interior surface

The interior surface of the right atrium can be divided into three regions: a smooth-walled venous component posteriorly that leads anteriorly to the vestibule of the tricuspid valve and the auricle (**Figure.2.5**). The wall of the vestibule is smooth, but its junction with the auricle is redged all round the atrioventricular junction. The smooth-walled part receives the opening of the venae cava and the

coronary sinus. It represents the venous component (sinus venosus) of the developing heart. The wall of the vestibule has a ridged surface and that of the auricle is trabeculated. Both are derived from the embryonic atrium proper.

The superior and inferior vena cava open into the venous component. The superior vena cava returns blood from head, neck and upper limb through an orifice that faces inferoanteriorly and has no valve, and also receives blood from the chest wall and the oesophagus via the azygos system. The inferior vena cava is larger than its superior counterpart: it drains blood from all structures below and including the diaphragm into the lowest part of the atrium. Anterior to its orifice is a flap-like valve, the Eustachian valve or valve of the inferior vena cava (**Figure.2.6,A**). Of varying size, this valve is found along the lateral or right margin of the vein.

The coronary sinus opens into the venous atrial component between the orifice of the inferior vena cava, the fossa ovalis and the vestibule of the atrioventricular opening (**Figure2.4,A,B**).

The atrium proper and the auricle are separated from the venous sinus by the crista terminalis. The sinoatrial node is located within the superior part of the atrium, lateral to and extending below the orifice of the superior vena cava (*Richard S. Snell, 1992*).

The pectinate muscles (musculi pectinati) that form the auricle almost parallel muscular ridges, extend anterolaterally from the terminal crest and reach into the auricle, where they form several trabeculations.

The septal wall presents the fossa ovalis, an oval depression above and to the left of the orifice of the inferior vena cava. Foramen during embryological period of fetus, which remains patent in up to one-third of all normal hearts.

Anteroinferior in the right atrium is the large, oval vestibule leading to the orifice of the tricuspid valve. A triangular zone, the triangle of Koch, is defined

between the attachment of the septal cusp of the tricuspid valve, the anteromedial margin of the ostium of the coronary sinus, and the round, collagenous, palpable, subendocardial tendon of Todaro (**Fig2.4,B**) . The triangle is a landmark of particular surgical importance, indicating the site of the atrioventricular node and its atrial connections (*Susan Standring , 2008*).

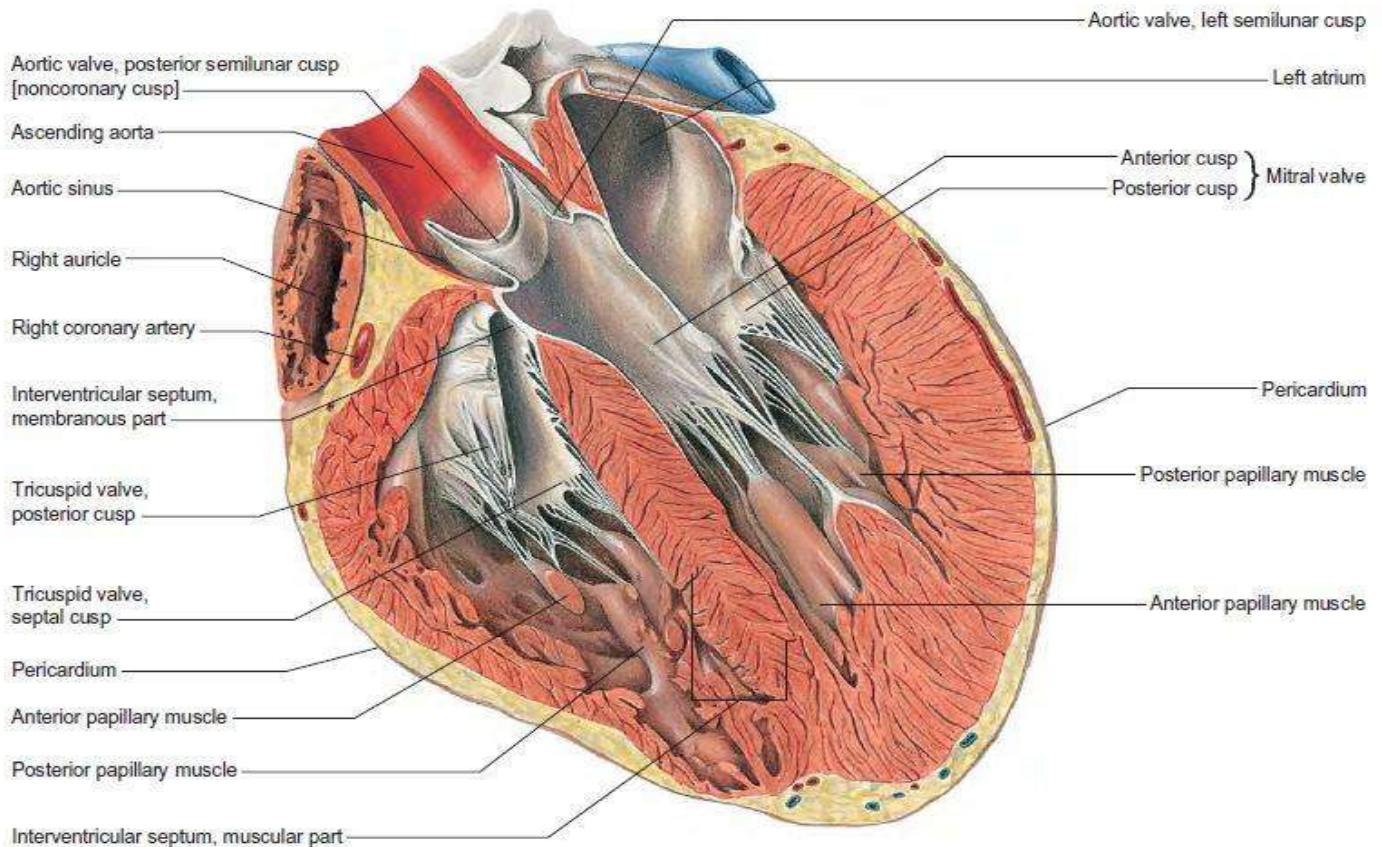


Figure 2.5dissection opening the ventricles, viewed from the front (from Sobotta 2006) (*Susan Standring , 2008*) .

2.1.1.7 Right ventricle

The right ventricle extends from the right atrioventricular (tricuspid) orifice nearly to the cardiac base . It then ascends to the left to become the infundibulum, or conus arteriosus, reaching the pulmonary orifice and supporting the cusps of pulmonary valve. Topographically, the ventricle possesses an inlet component which supports and surrounds the tricuspid valve(*Susan Standring , 2008*).

2.1.1.7.1 External features

The convex anterosuperior surface of the right ventricle makes up a large part of the sternocostal aspect of the heart, and is separated from the thoracic wall only by the pericardium (**Figure 2.2,C**) and (**Figure 2.3**). The left pleura and, to a lesser extent, the anterior margin of the left lung are interposed above and to the left. The inferior surface is flat and is related mainly, with the interposition of the pericardium, to the central tendon and a small adjoining muscular part of the diaphragm. The left and posterior wall is the ventricular septum. This slightly curved and bulges into the right ventricle so that, in sections across the cardiac axis, the outline of the right ventricle is crescentic. The wall of the right ventricle is significantly thinner (3-5 mm on average) than that of the left, the ratio of the thickness of the two walls usually being 1:3 in thickness (*Susan Standring , 2008*).

2.1.1.7.2 Internal features

The inlet and outlet components of the ventricle, supporting and surrounding the cusps of the tricuspid and pulmonary valves respectively are separated in the roof of the ventricle by the prominent supraventricular crest (crista supraventricularis) (**Figure 2.5**). The crest is a thick, muscular highly arched structure, extending obliquely forwards and to the right from a septal limb high on the interventricular septal wall to a mural or parietal limb on the anterolateral right ventricular wall. The posterolateral aspect of the crest provides a principal attachment for the anterosuperior cusp of the tricuspid valve. The septal limbs of the crest may be continuous with, or embraced by, the septal limbs of the septomarginal trabecula. The inlet and outlet regions extend apically into and from the prominent coarsely trabeculated component of the ventricle. The inlet component is itself also trabeculated, whereas the outlet component (or infundibulum) has predominantly smooth walls. The trabeculated appearance is caused by a myriad of regular muscular ridges and protrusions, which are known collectively as trabeculate

carneae, and are lined by endocardium. These protrusions and intervening grooves impart great variation in wall thickness; the protrusions vary in extent from mere ridges to trabeculae, which are fixed at both ends but otherwise free. Other conspicuous protrusions are the papillary muscles, which are inserted at one end onto the ventricular wall and are continuous at the other end with collagenous cords, the chordae tendineae, inserted on the free edge and elsewhere on the free aspect of the atrioventricular valves.

The atrioventricular valvular complex, in both right and left ventricles, consists of the orifice and its associated anulus, the cusps, the supporting chordae tendineae of various types and the papillary muscles. Harmonious interplay of all these, together with the atrial and ventricular myocardial masses, depends on the conduction tissues and the mechanical cohesion provided by the fibroelastic cardiac skeleton. All parts change substantially in position, shape, angulation and dimensions during a single cardiac cycle (*Susan Standring, 2008*) and (*Chummy Sinnatamby, 2006*).

2.1.1.8 Tricuspid valve cusps

The tricuspid valve guards the right atrioventricular orifice. It has three cusped and admits the tips of three fingers. The three cusps, called anterior, posterior and septal, are attached by their bases to the fibrous atrio-ventricular ring and lie against the sternocostal, diaphragmatic and septal wall of the ventricle. The cusps of both tricuspid and mitral valves often appear to be subdivided but without forming complete additional leaflets. The edges and ventricular surfaces of the cusps receive the attachments of the chordate tendinea, collagenous cord which diverge from the papillary muscles and prevent the cusps from being everted when the ventricle contracts. The main papillary muscles are anterior, inferior (posterior) and septal in location and each is connected to more than one cusps.

The cavity of the ventricle continues upwards into a narrowing funnel-shaped approach to pulmonary orifice. The walls of this part, the infundibulum or conus, are thin and smooth (*Richard S.Snell,1992*).

Never forming a simple complete fibrous ring . The free margin of each cusp contains a central localized thickening of collagen, the nodule of the semilunar `cusp` (nodule of Arantius). Performations within the cusps close to the free margin and near the commissures are frequently present, but are of the no functional significance . Each semilunar cusp is contained within one of the three sinuses of the pulmonary trunk (*Susan Standring , 2008*).

2.1.1.9 Pulmonary valve

The pulmonary valve , guarding the outflow from the right ventricle , surmounts the infundibulum and is situated at some distance from the other three cardiac valves (**Figure 2.6,A and B**) and (**Figure 2.7,A and B**).Its general plane faces superiorly to the left and slightly posteriorly. It has three semilunar cusps attached by convex edges partly to the infundibular wall of the right ventricle and partly to the origin of the pulmonary trunk. The line of attachments is curved , rising at the periphery of each cusp near their zones of apposition and reaching the sinutubular ridge of the pulmonary trunk(**Figure 2.7,B**)(*Susan Standring , 2008*) and (*Richard S. Snell ,1992*).

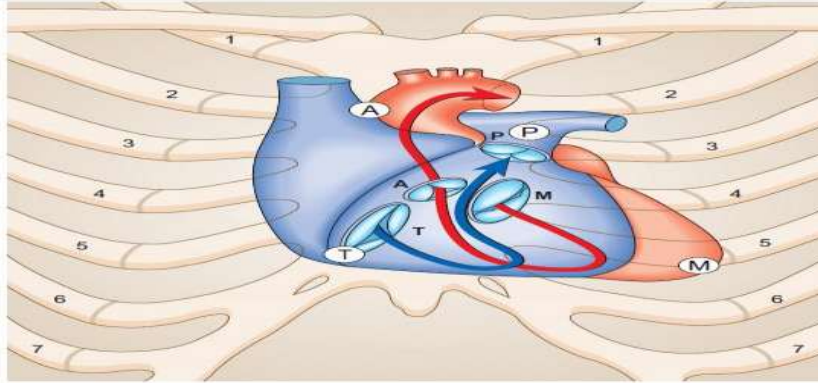


Figure 2.6.A Relation of the sternocostal surface and valves of the heart to the thoracic cage. The right heart is blue, the arrow denotes the inflow and outflow channels of the right ventricle; the left heart is treated similarly in red. The positions, planes and relative sizes of the cardiac valves are shown. The position of the letters A, P, T and M indicate respectively the aortic, pulmonary, tricuspid and mitral auscultation areas of clinical practice. Note that, for the purpose of illustration, the orifices of the aortic, mitral and tricuspid valves are shown with some separation between them. In reality, the cusps of the three valves are in fibrous continuity (see Figure.2.5) (*Susan Standring , 2008*) .

Removal of the cusps shows that the fibrous semilunar attachments enclose three crescents of infundibular musculature within the pulmonary sinuses, whereas three roughly triangular segments of arterial wall are incorporated within the ventricular outflow tract beneath the apex of each commissural attachment. There is, thus, no proper circular `anulus` supporting the cusps of the valve , and the fibrous semilunar attachment is an essential requisite for snug closure of the nodules and lunules of the cusps (see below) during ventricular diastole. It is difficult to name the cusps and corresponding sinuses of the pulmonary valve and trunk precisely according to the coordinates of the body, because the valvular orifice is obliquely positioned.

The official nomenclature (*Terminologia Anatomica 1998*) refers to an anterior , a posterior and a septal cusp, based on their position in the fetus. The

position changes with development and in the adult there is one anterior semilunar cusp, and right and left semilunar cusps. Each semilunar cusp is contained within one of the three sinuses of the pulmonary trunk. Coronary arteries and cardiac veins can be seen *From Gray's anatomy (Susan Standring , 2008) and from Clinical Anatomy for student (Richard S. Snell ,1992).*

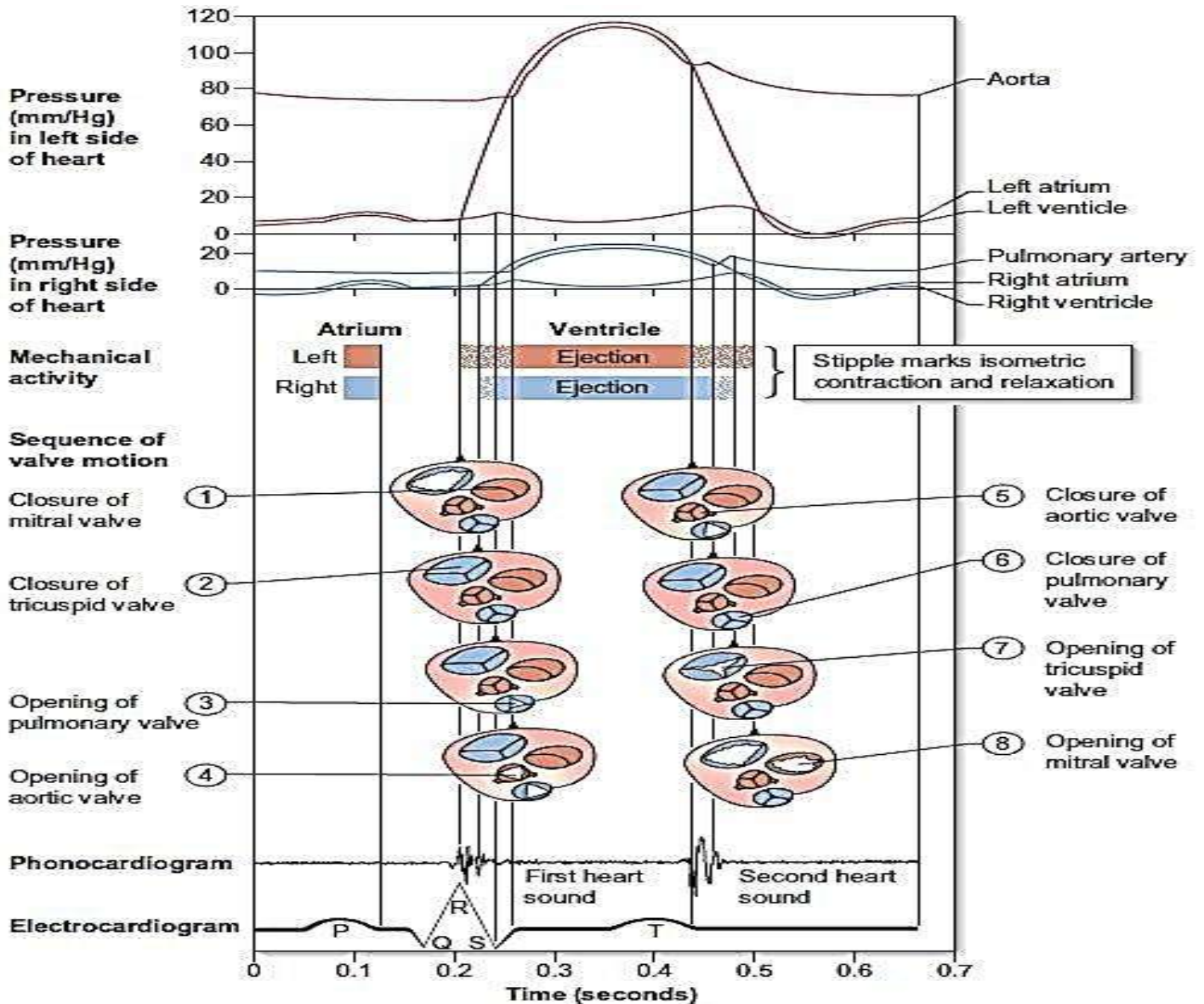


Figure 2.6B Summary of some of the principal events that occur in the cardiac cycle .Systole beings at the onset of the second heart sound, when diastole beings and this cycle repeats itself continuous *(Susan Standring , 2008) .*

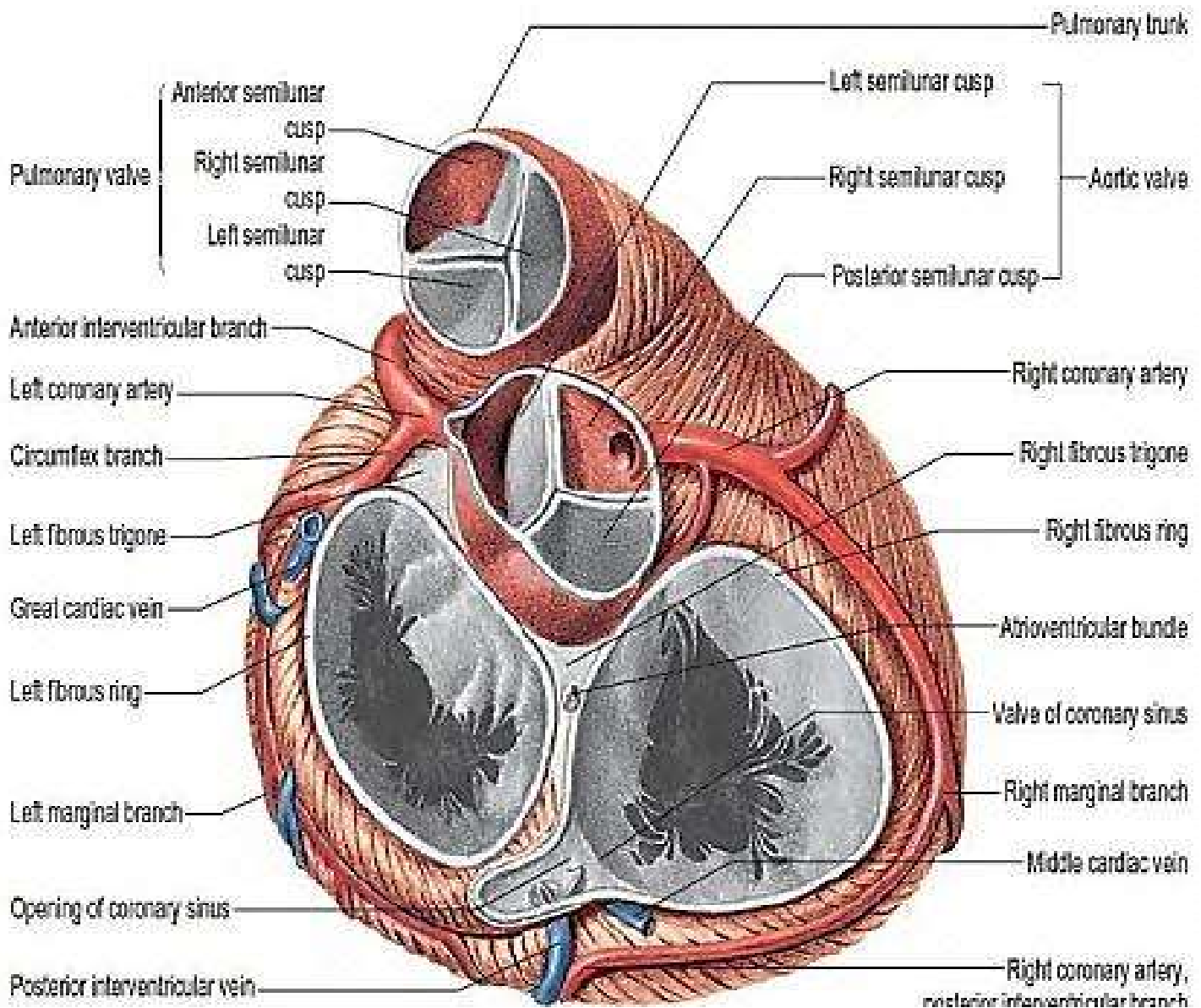


Figure 2.7,AThe base of the ventricles. after removal of the atria and the pericardium (Susan Standing , 2008) .

The Opening of the pulmonary valve during diastole ,the pulmonary valve is closed and all three cusps of the valve are tightly apposed. The pulmonary valve is difficult to visualize at echocardiography and usually only the posterior cusp is visible when the valve is closed; atrial systole may cause a slight posterior movement of the valve cusps. The pulmonary valve opens passively during

ventricular systole and then closes rapidly at the end of systole(Figure 2.7,B) (*Susan Standring , 2008*).

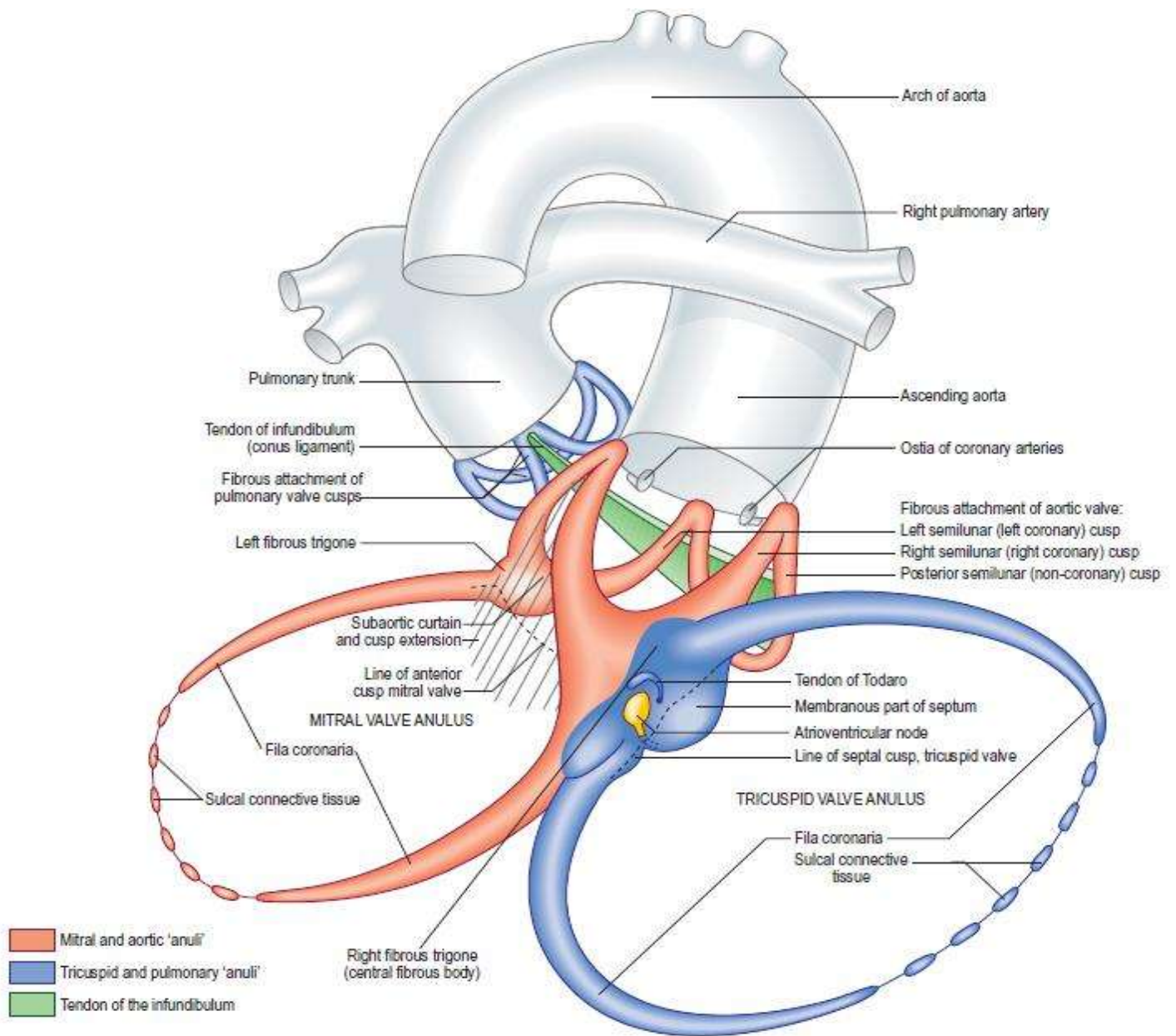


Fig. 2.7,B Principal elements of the fibrous skeleton of the heart . For clarity , the view is from the right posterosuperior aspect. Perspective causes the pulmonary anulus to appear smaller than the aortic anulus, whereas in fact the reverse is the case. Consult text for an extended discussion. (Copyright from The royal college of Surgeons of England.Reproduced with permission) (*Susan Standring , 2008*).

2.1.1.10 Left atrium

Although smaller in volume than the right , the left atrium has thicker walls (3 mm on average). It forms the posterior surface (base) of the heart and lies behind the right atrium the inferior margin of the left atrium lies a little above that of the right atrium, whose posterior wall here receives the coronary sinus. From the left atrium the left ventricle slopes away to the apex. A small, bent left auricle projects from its upper and curves round to the front on the left side of the infundibulum. The four pulmonary veins enter the left the left atrium symmetrically, one above the other on each side.

The cavity of the left atrium is smooth-walled except in the auricle, here the muscular ridges indicate that the appendage was original auricular chambers of the embryonic heart. All the smooth-walled portion is derived by incorporation of the embryonic pulmonary veins in to the atrial cavity. The bicuspid **mitral valve** admits the tips of the two fingers.

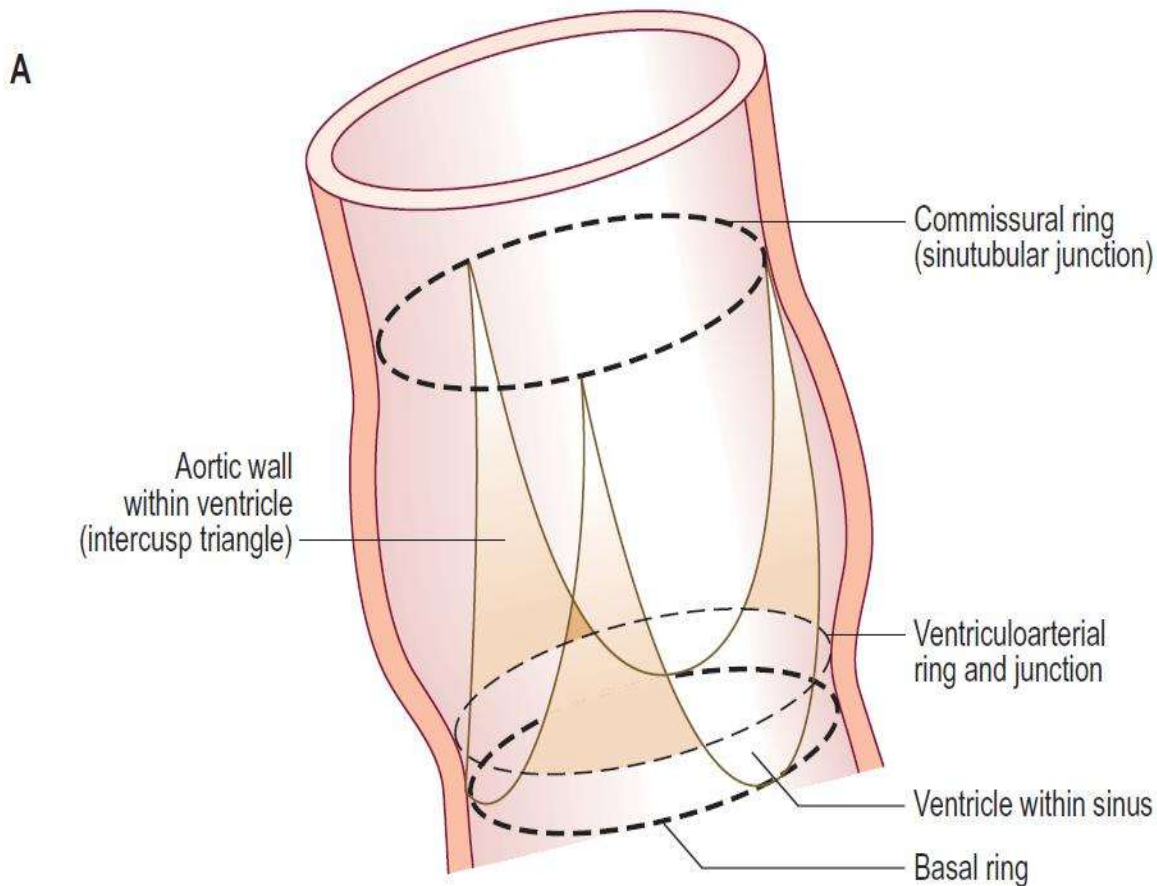
The cusped are name anterior and posterior. The base of the anterior cusp is attached to one-third, and that of the posterior cusped to two-third of the margin of the fibrous atrioventricular ring, but sometimes they fail to meet and a small accessory cusp fills the gap between them. The anterior cusp of the mitral valve is thicker and more ridged than the posterior cusp. The anterior cusp lies between the mitral and aortic orifices and thus lies between the inflow and outflow tracts of the left ventricle (**Figure 2.7,B**) and (**Figure 2.8,C**)(*Chummy S-Sinnatamby,2006*).

2.1.1.11 Left ventricle

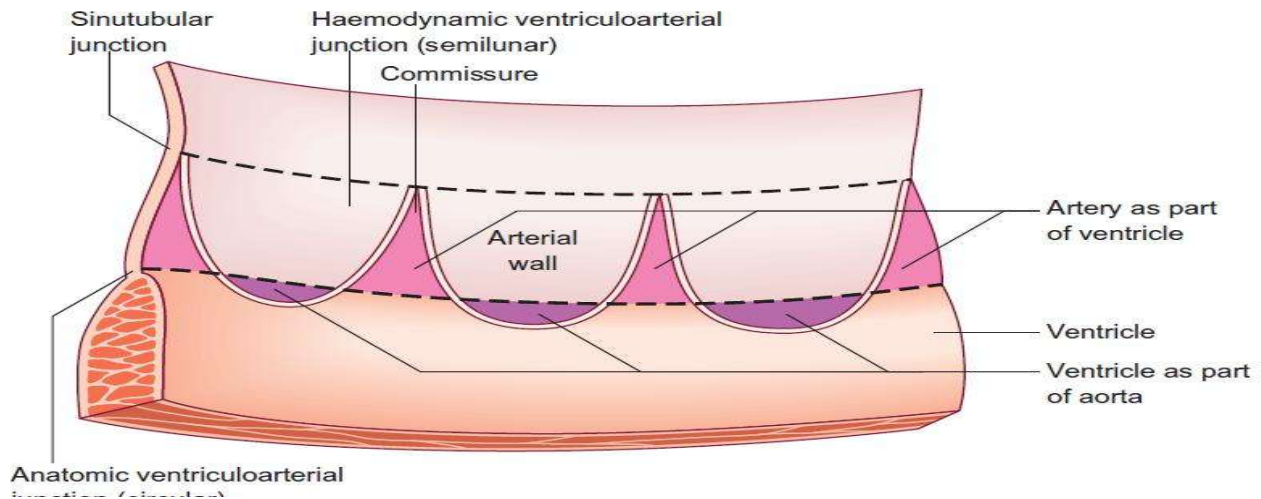
The left ventricle is constructed in accordance with its role as a powerful pump that sustains pulsatile flow in the high-pressured systemic arteries . Variouslly described as half-ellipsoid or cone-shaped, it is longer and narrower than the right ventricle, extending from its base in the plane of the atrioventricular groove to the cardiac apex(*Chummy S-Sinnatamby,2006*)

2.1.1.11.1 Internal features

The left ventricle has an inlet region, guarded by the mitral valve (ostium venosum), an outlet region, guarded by the aortic valve (ostium arteriosum), and an apical trabecular component. The left atrioventricular orifice admits atrial blood during diastole, flow being towards the cardiac apex. After closure of the mitral cusps, and throughout the ejection phase of systole, blood is expelled from the apex through the aortic orifice. In contrast to the orifices within the right ventricle, those of the ventricle are in close contact, with fibrous continuity between the cusps of the aortic and mitral valves (the sub aortic curtain (**Figure 2.8,A,B and C**). The inlet and outlet turns sharply round this fibrous curtain (**Figure 2.7,A**).



B



C

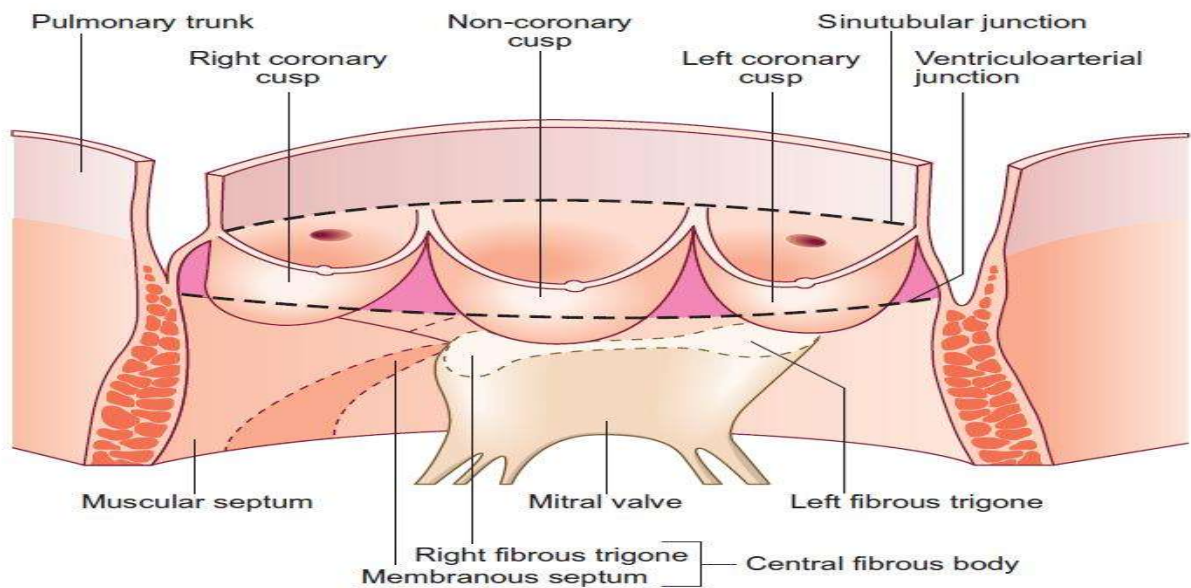


Figure 2.8,A,Band C The structure of the aortic root is best conceptualized in terms of a three-pronged content (A): there are at least three rings within this coronet, but none supports the entirety of the attachments of the valvular cusps (compare with C). In B, the cusps have been resected at their attachment to the aortic wall. Note the relationship of the cusp insertions and the ventriculo-arterial junction. In C, the root of the semilunar cusps. Note the zone of fibrous continuity between the cusps of the aortic and mitral valves and their relationship to the fibrous trigones, and the semilunar attachment of the cusps (compare with B). (Redrawn by courtesy of professor RH Anderson ,Institue of Child Health , University College. London)(*Susan Standing , 2008*) .

The anterolateral wall is the concave-convex ventricular septum, a muscular wall the convexity of which is the posteromedial profile of the right ventricle as seen in section. It thus completes the circular outline of the left ventricle (**Figure 2.8,A,B and C**). Towards the aortic orifice, the septum becomes the thin, collagenous interventricular component of the membranous septum, an oval or round area below and confluent with the fibrous triangle separating the right and the non-coronary cusps of the aortic valve.

Between the lower limits of the free margins of the cusps of the mitral valve and the apex of the ventricle, the muscular walls are deeply trabeculated. These trabeculae carneae are finer and more intricate than those of the right ventricle, but similar in structure. Trabeculation is characteristically well developed near the apex, whereas the upper reaches of the septal surface are smooth (**Figure 2.9**)(*Susan Standring , 2008*).

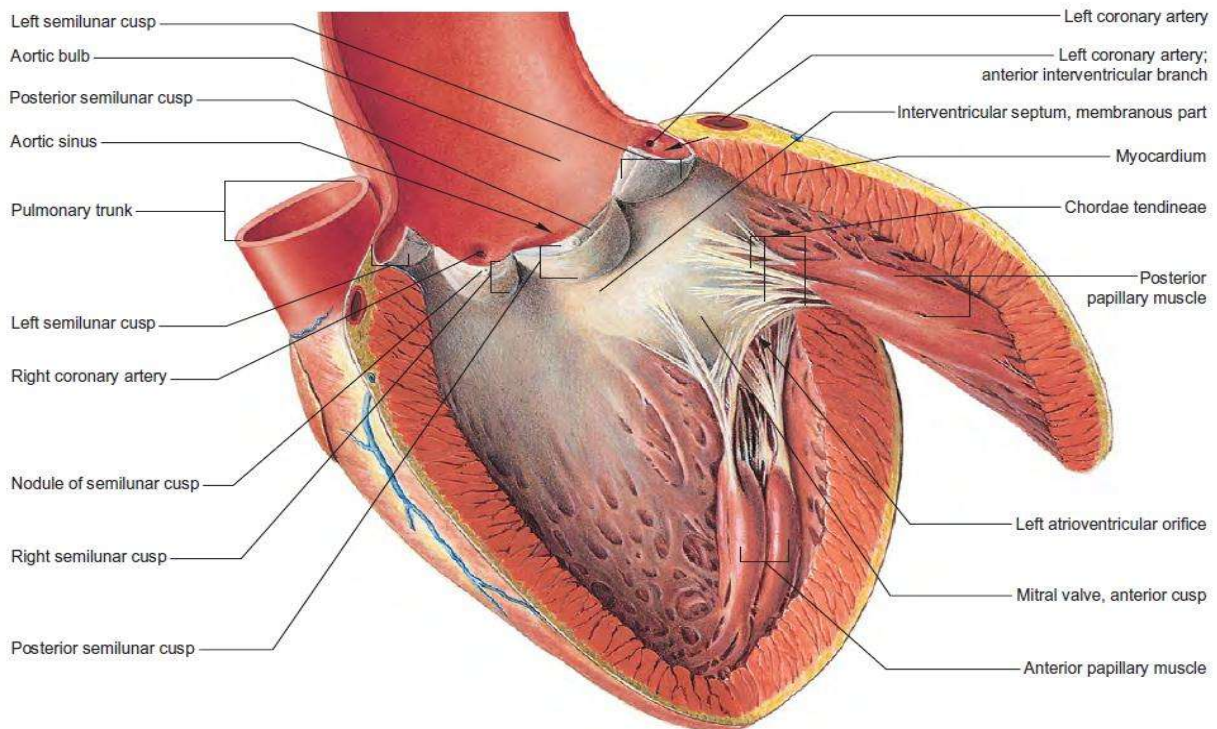


Figure 2.9The aortic orifice opened from the front to show the cusps of the aortic valves, their nodules, lunules, commissures and the triple-scalloped line of anular attachment. (From Sobotta 2006.)(Susan Standring , 2008).

2.1.1.12 Mitral valve:

The general comments already made in respect to the tricuspid valve apply equally to the mitral. The valve has an orifice with its Supporting anulus, cusps and a variety of chordae tendineae and papillary muscles

The mitral orifice is a well-defined transitional zone between the atrial wall and the bases of the cusps. It is smaller than the tricuspid orifice(mean circumference is 9.0 cm in males, 7.2 cm in females).

The anulus of the valve is not a simple fibrous ring, but is made up of fibrocollagenous elements of varying consistency from which the fibrous core of the cusps take origin. These variations allow major changes in the shape and dimensions of the anulus at different stages of the cardiac cycle and ensure optimal efficiency in valvularaction(*Chummy S-Sinnatamby,2006*).

2.1.1.12.1 Mitral valve cusps :

Since the earliest descriptions, the mitral valvular cusps have been described as paired structures. Hence, the name 'bicuspid valve' is more explicit, When the valve is laid open, the anterior cusp (aortic, septal, `greater` or anteromedial) is seen to guard one-third of the circumference of the orifice and to be semicircular or triangular, with few or no marginal indentations . Its fibrous core (lamina fibrosa is continuous, on the outflow aspect , beyond the margins of the fibrous subaortic curtain.

The anterior cusp has no basal zone,continuous instead into the valvular curtain . Hinging on its anularattachment , and continuous with the subaorticcurtain,it is critically placed between the inlet and the outlet of the ventricle .

The posterior cusp (mural,ventricular smaller or posterolateral)usually has two or more minor indentations. Lack of definition of major intervalvular commissures has previously led to disagreement and confusion concerning the

territorial extent of this cusp and the possible existence of accessory 'scallop'. Examination of the valve in the closed posterior cusp can conveniently be regarded as all the valvular tissue posterior to the anterolateral and posteromedial ends of the major zone of apposition with the aortic cusp.

Papillary muscles The two muscles supporting the cusps of the mitral valve also vary in length and breadth and may be bifid. The anterolateral muscle arises from the sternocostal mural myocardium, the posteromedial from the diaphragmatic region (**Figure 2.9**). Chordae tendineae arise mostly from the tip and apical one-third of each muscle, but sometimes take origin near their base. The chordae from each papillary muscle diverge and are attached to corresponding areas of closure on both valvular cusps (*Richard S. Snell, 1992*).

The aortic orifice is guarded by the aortic valve, at the entrance of aorta. It lies at a lower level than the pulmonary orifice, to its right side (**Figure 2.9**) and is more obliquely placed. It has three semilunar cusps. In the adult heart these cusps are in anterior, left posterior and right posterior positions (*Chummy S-Sinnatamby, 2006*).

2.1.1.13 Aortic sinuses (of Valsva):

The aortic sinuses are more prominent than those in the pulmonary trunk. The upper limit of each sinus reaches considerably beyond the level of the free border of the cusp and forms a well-defined complete circumferential sino tubular ridge when viewed from the aortic aspect (**Figure 2.9**). Coronary arteries usually open near this ridge within the upper part of the sinus, but are thus markedly variable in their origin. The walls of the sinuses are largely collagenous near the attachment of the cusps, but the amount of lamellated elastic tissue increases with distance from the zone of attachment. Strands of myocardium may enter this fibroelastic wall.

At the mid-level of each sinus, its wall is about half the thickness of the supra valvular aortic wall and less than one-quarter of the thickness of the sino

tubular ridge. At this level, the mean luminal diameter of the beginning of the aortic root is almost double that of the ascending aorta. These details are functionally significant in the mechanism of valvular motion *Fro (Chummy S-Sinnatamby,2006)*

2.1.1.14 Opening of the aortic valve :

During diastole, the closed aortic valve supports an aortic column of blood at high but slowly diminishing pressure (**Figure 2.9**). Each sinus and its cusp form a hemispherical chamber. The three nodules are opposed and the margins and lunular parts of adjacent cusps are tightly apposed on their ventricular aspects. From the aortic aspect, the closed valve is triradiate, three pairs of closely compressed lunules radiating from their nodules to their peripheral commissural attachments at the sinu tubular junction. As ventricular systolic pressure increases, it exceeds aortic pressure and the valve is passively opened. The fibrous wall of the sinuses nearest the aortic vestibule is almost inextensible but, in the upper parts of sinuses, the wall is fibroelastic. Under left ventricular ejection pressure, the radius here increases 16% in systole. Hence the commissures move apart, making the orifice triangular when fully open.

The free margins of the cusps then become almost straight lines between peripheral attachments. However, they do not flatten against the sinus walls, even at maximal systolic pressure, which is probably an important factor in subsequent closure. During ejection, most blood enters the ascending aorta, but some enters the sinuses, forming vortices that help to maintain the triangular 'mid position' of the cusp during ventricular systole and probably initiate their approximation with the end of systole. Tight and full closure ensues, with the rapid decrease in ventricular pressure in diastole. Commissures narrow, nodules aggregate and the valve reassumes its triradiate form. Experiments indicate that 4% of ejection blood regurgitates through a valve with normal sinuses, whereas 23% regurgitates

through a valve without them. The normal structure of the aortic sinuses also promotes non-turbulent flow into the coronary arteries(*Chummy S-Sinnatamby,2006*) and (*Cunningham,1987*).

2.1.1.15 Vascular Supply of the heart:

2.1.1.15.1 Arterial Supply of the Heart

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 (**Figure 2.10,A,Band C**). The coronary arteries and their major branches are distributed over the surface of the heart, lying within subepicardial connective tissue(*Richard S.Snell,1992*).

2.1.1.15.1.1 The Right Coronary Artery:

The right coronary artery arises from the anterior aortic sinus of the ascending aorta (**Figure.2.7,A**). It descends 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(*Richard S.Snell,1992*).

2.1.1.15.1.1.1 Branches of the Right Coronary Artery:

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.

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.

The posterior ventricular branches are usually two in number and supply the diaphragmatic surface of the right ventricle.

The posterior interventricular (descending) artery runs toward the apex in the posterior interventricular groove (**Figure 2.7,A**) . It gives off branches to the right and left ventricles, including its inferior wall. It supplies branches to the posterior part of the ventricular septum but 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.

The arterial 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 the left atria; in 35% of individuals, it arises from the left coronary artery (*Richard S.Snell,1992*).

2.1.1.15.1.2 Left Coronary Artery:

The left coronary artery is usually larger than the right coronary artery. It arises from the left posterior aortic sinus of the ascending aorta and passes forward between the pulmonary trunk and the left auricle (**Figure 2.7,A**) .It then enters the atrioventricular groove and divides into an anterior interventricular branch and a circumflex branch. The left coronary artery supplies the major part of the heart, including the greater part of the atrium , left ventricle, and ventricular septum (*Richard S.Snell,1992*).

2.1.1.15.1.2.1 Branches of the left coronary Artery:

The anterior interventricular (descending) branch runs downward in the anterior interventricular groove to the apex of the heart (**Figure 2.10,A,B,C**)In most individuals, it then passes around the apex of the heart to enter the posterior interventricular groove and anastomoses with the terminal branches of the right coronary artery . In one third of individuals, it ends at the apex of the heart. The anterior intervrentricular 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.

The circumflex artery is the same size as the anterior interventricular artery (**Figure 2.10,C**).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 (*Richard S.Snell,1992*).

2.1.1.15.1.3 Variations in the Coronary Arteries:

Variations in the blood supply to the heart do occur, and the most common variations affect the blood supply to the diaphragmatic surface of both ventricles. Here, the origin, size, and distribution of the posterior interventricular artery are variable . In right dominance , the posterior interventricular artery is a large branch of the right coronary artery . Right dominance is present in most individuals (90%) . In left the posterior interventricular artery is a branch of the circumflex branch of the left coronary artery (10%)(*Richard S.Snell,1992*).

2.1.1.15.1.4 Coronary Artery Anastomoses :

Anastomoses between the terminal branches of the right and left coronary arteries (collateral circulation) exist, but they are usually not large enough to provide an adequate blood supply to the cardiac muscle should one of the large branches become blocked by disease. A sudden block of one of the larger branches of either coronary artery usually leads to myocardial death (myocardial infarction), although sometimes the collateral circulation is enough to sustain the muscle(*Richard S.Snell,1992*) .

2.1.1.15.1.5 Summary of the Overall Arterial Supply to the Heart in Most Individuals:

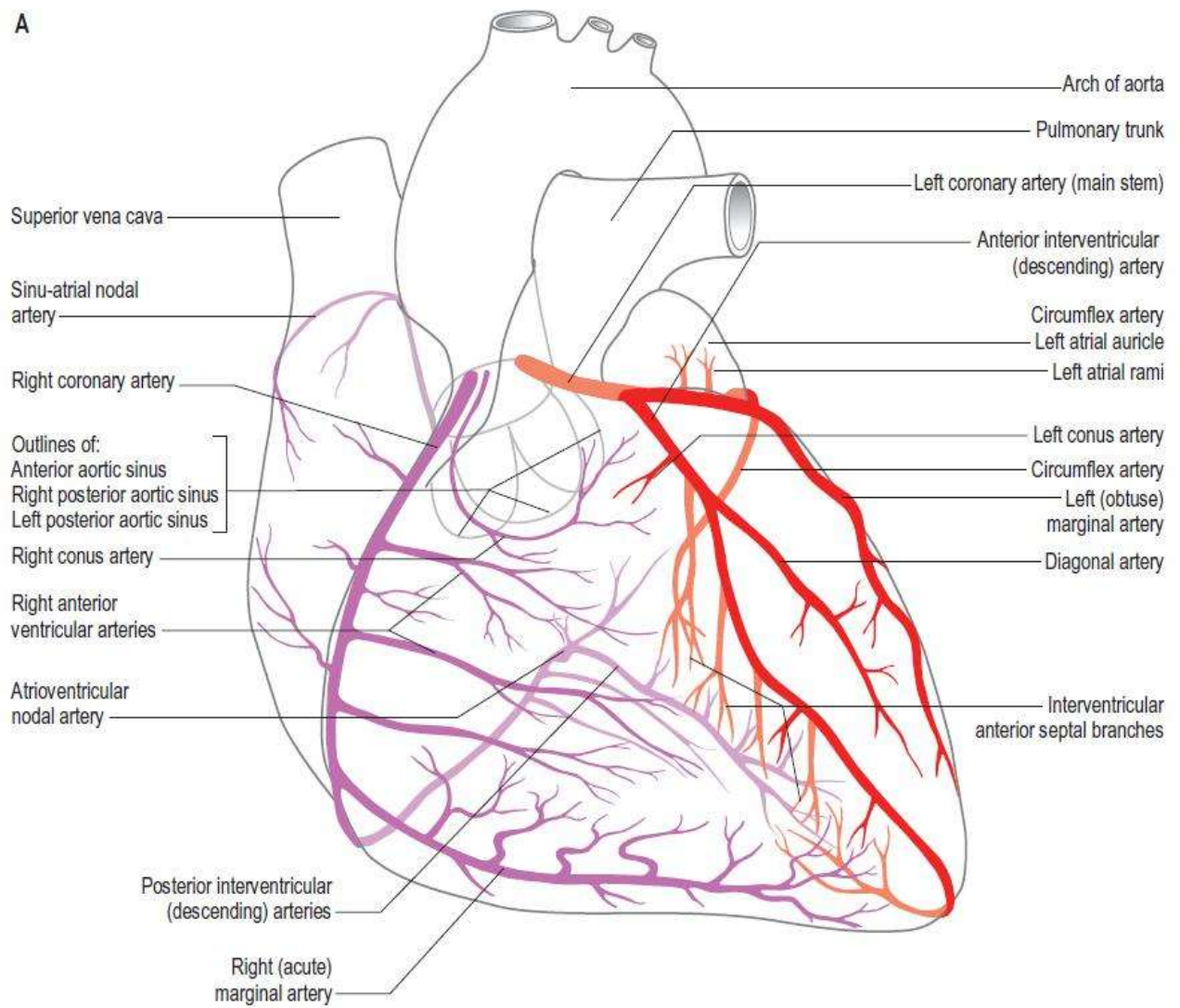
The right coronary artery supplies all of the right ventricle (except for the small area to the right of the anterior interventricular groove), the variable part of the diaphragmatic surface of the left ventricle , the posteroinferior third of the ventricular septum , the right atrium and part of the left atrium, and the sinuatrial node and the atrioventricular node and bundle. The LBB also receives small branches.

The left coronary artery supplies most of the left ventricle, a small area of the right ventricle to the right of the interventricular groove , the anterior two thirds of the ventricular septum , most of the left atrium , the RBB, and the LBB (*Richard S.Snell,1992*) .

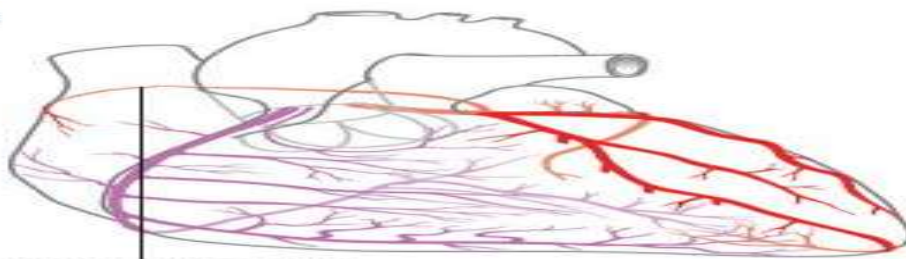
2.1.1.15.1.6 Arterial Supply to the Conducting System :

The sinuatrial node is usually supplied by the right coronary artery but is sometimes supplied by the left coronary artery. The atrioventricular node and the atrioventricular bundle are supplied by the right coronary artery. The RBB of the atrioventricular bundle is supplied by the left coronary artery; the LBB is supplied by the right and left coronary arteries (*Richard S.Snell,1992*).

A

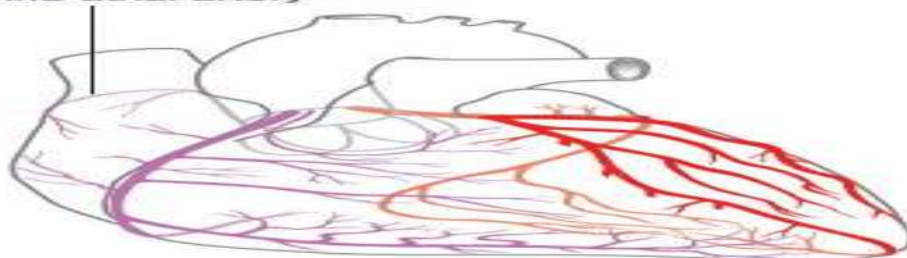


B



Sinu-atrial artery

C



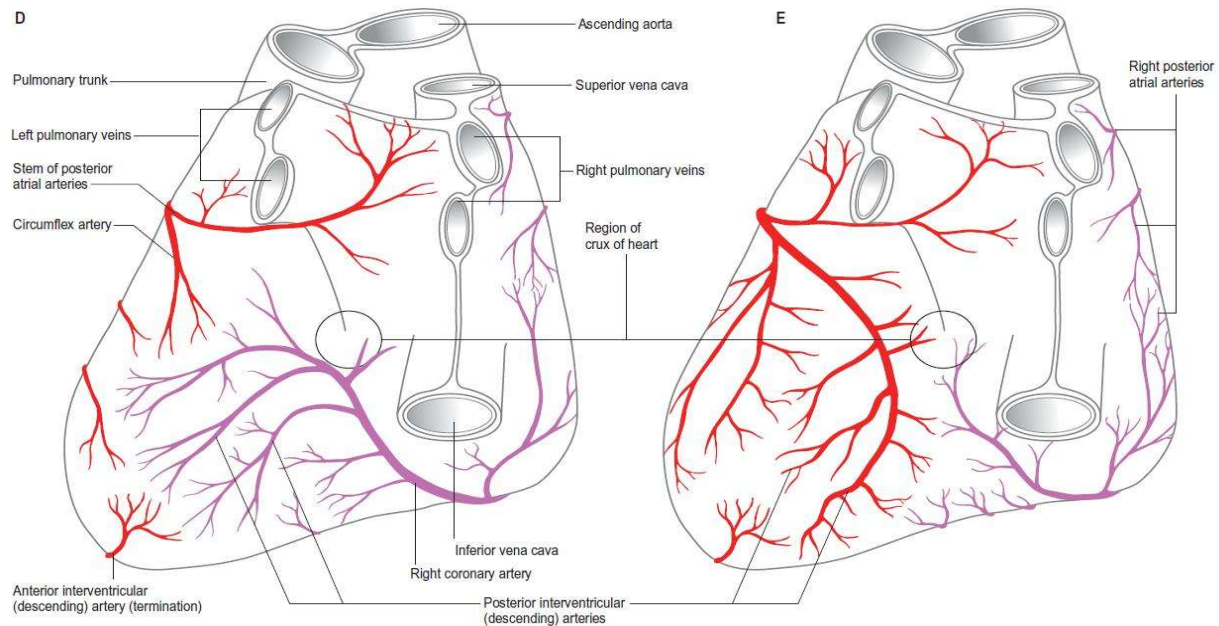


Figure 2.10,A,B,C,D,E Anterior views of the coronary arterial system ,with the principal variations .The right coronary arterial tree is shown in magenta , the left in full red. In both cases posterior distribution is shown in a paler shade. A, The most common arrangement.B, A common variatin in the origin of the sinoatrial nodal artery.C, An example of left`dominance` by the left coronary artery, showing also an uncommon origin of the sinu-atrial artery.Posterior views of the coronary arterial system. The right coronary arterial tree is shown in magenta, the left in full red.D, An example of the more normal distribution in right `dominance`. E, A less common form of left `dominance`*(Richard S.Snell,1992)*.

2.1.1.15.2 Venous Drainage of the Heart:

Most blood from the heart wall drains into the right atrium through the coronary siuns(**Figure 2.11**), which lies in the posterior part of the atrioventricular groove and is a continuation of the great cardiac vein. It opens into the right atrium to the left of the inferior vena cava. The small and middle cardiac veins are tributaries of the coronary sinus. The remainder of the blood is returned to the right atrium by the anterior cardiac vein and by small veins that open directly into the heart chambers *From Clinical Anatomy for student (Richard S.Snell,1992) and From Text book of Anatomy (Cunningham,1987)*.

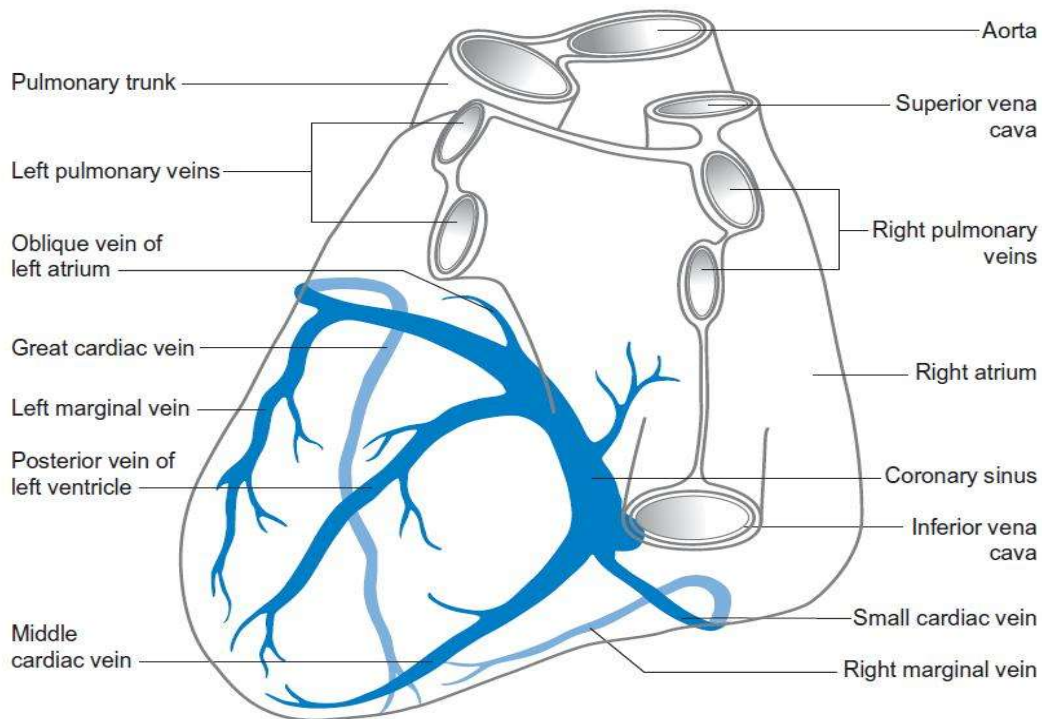


Fig.2.11 Principle veins of the heart *From Clinical Anatomy for student (Richard S.Snell,1992).*

2.1.1.16 Nerve Supply :

2.1.1.16.1 Cardiac plexus:

The cardiac plexus consist of sympathetic , parasympathetic and afferent fibers and small ganglia. It is divided into superficial and deep parts, but functionally they are one. Their branches enter the pericardium to accompany the coronary arteries (vasomotor) and to reach the myocardium, in particular the SA and AV nodes (cardio inhibitor) and cardio accelerator).

The **superficial part** of the cardiac plexus lies in front of the ligamentumarteriosum . The **deep part** of the cardiac plexus is larger and lies to the right of the ligamentumarteriosum ,infront of the bifurcation of the trachea and behind the aortic arch.

The cardiac plexus receives sympathetic fibers from the three cervical and the upper four or five thoracic sympathetic ganglia of both sides, and

parasympathetic fibers from both vagi in their cervical course and both recurrent laryngeal nerves. The sympathetic fibers accelerate the heart and dilate the coronary arteries ; the parasympathetic fibers slow the heart and constrict the coronary arteries.

The vagi carry afferent fibers concerned with cardiovascular reflexes. Pain fibers run with sympathetic nerves, reaching any of the cervical and upper thoracic sympathetic ganglia. The pain fibers pursue the usual pathway to the central nervous system , passing through the sympathetic ganglia to the spinal nerves via white rami communicantes. The connection with cervical and thoracic spinal nerves presumably explains the referral of cardiac pain to the arm, chest or neck(*Richard S.Snell,1992) and (Ellis,1986).*

2.1.1.16.2 Conducting (conduction) system:

The conducting system of the heart consists of the sinoatrial node (SA node) , the atrioventricular node (AV node), the atrioventricular bundle (of His), the right and left limbs or branches of the bundle, and the subendocardial Purkinje fibers. From the SA node , which, like AV node and its extensions, is composed of a specialized type of cardiac muscle fibers (not nervous tissue) , impulses are conducted to the AV node by atrial cardiac muscle fibers. The AV node , bundle, branches and subendocardial fibers form one continuous mass of conduction tissue (*Susan Standring , 2008) and (Ellis,1986).*

The sinoatrial node, or pacemaker of the heart , is a small mass of histologically distinctive myocardial cells. It is subepicardially situated in the wall of the right atrium , just below the superior vena cava , at the top of the sulcus terminalis. It has no macroscopic or palpable features that indicate its location. The atrioventricular node is also a small mass of specialized myocardial cells. It is situated in the right atrium on the interatrial septum , above the attachment of the

septum cusp of the tricuspid valve, to the left of the opening of the coronary sinus **(Figure 2.12)***(Chummy S-Sinnatamby,2006)and (Ellis,1986).*

From AV node, the AV bundle runs along the inferior border of the membranous part of the interventricular septum , where it divides into right and left branches. Since the fibrous framework of the heart separates the muscles of the atria from those of the ventricles , the bundle is the only means of conducting the contractile impulse from atria to ventricles . The right branch runs at first within the muscle of the septum and then becomes subendocardial on the right side of the septum.

Much of it continues into the septomarginal trabecular (moderator band) to reach the anterior papillary muscle and the anterior wall of the ventricle, and its Purkinje fibres then spread out beneath the endocardium . The left branch reaches the septal endocardium of the left ventricle and rapidly breaks up into a sheaf of branches which spread out subendocardially over the septum and the rest of the ventricular wall *(Richard S.Snell,1992).*

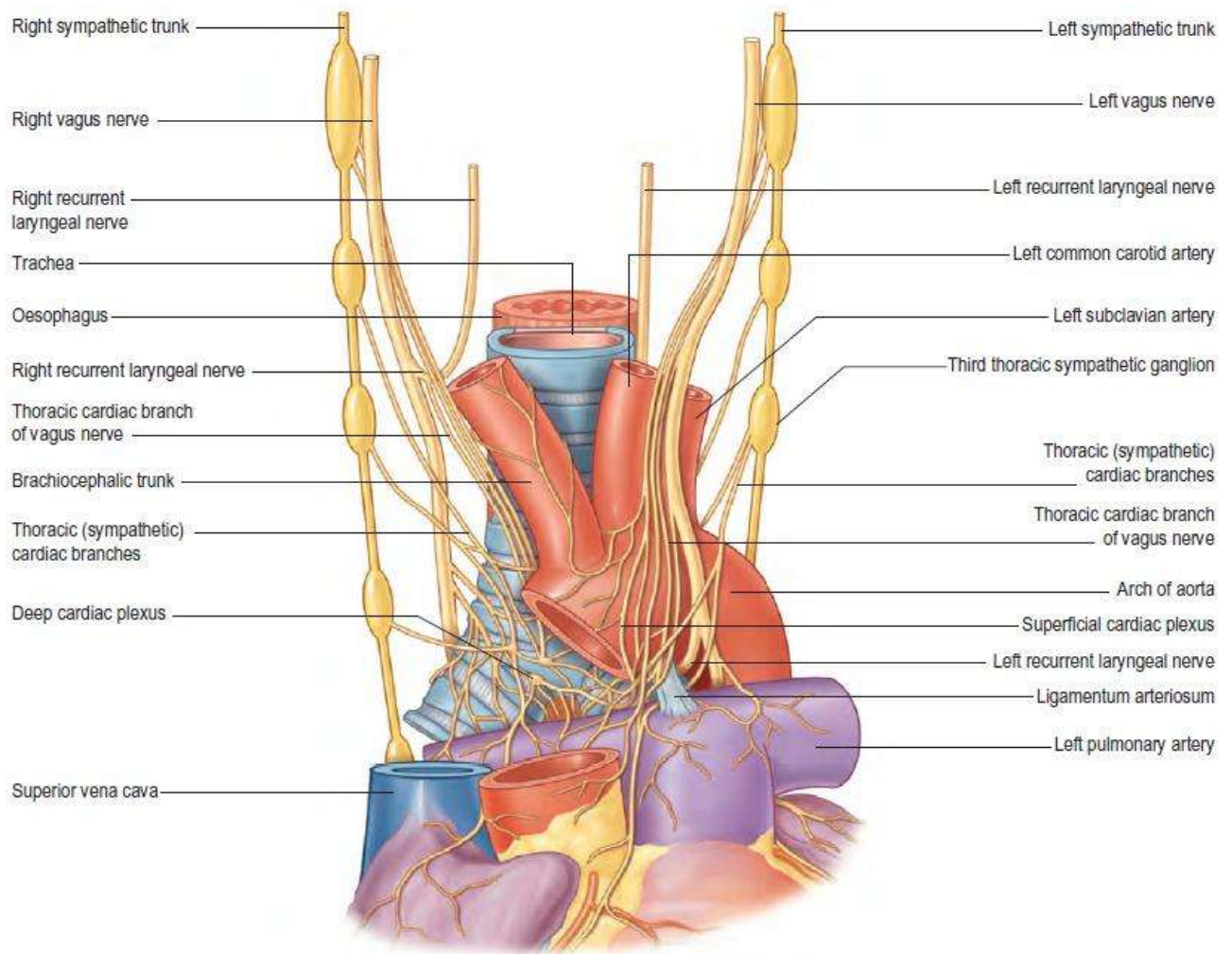


Figure 2.12 The human cardiac plexus:its source from the cervical parts of the vagus nerves and sympathetic trunks and its extensions, the pulmonary , atrial and coronary plexuses. Note the numerous junctins between sympathetic and parasympathetic (vagal) branches that form the plexus (Chummy S-Sinnatamby,2006).

2.1.1.17 Surface anatomy of the Heart:

The heart has three surfaces: sternocostal (anterior), diaphragmatic (inferior), and a base (posterior).It also has an apex, which is directed downward, forward, and to the left

The **sternocostal surface** is formed mainly by the right atrium and the right ventricle, which are separated from each other by the vertical

atrioventricular groove (**Figure 2.13,A**). The right border is formed by the right atrium ; and the left border,by the left ventricle and part of the left auricle.The right ventricle is separated from the left border , by the anterior interventricular groove.

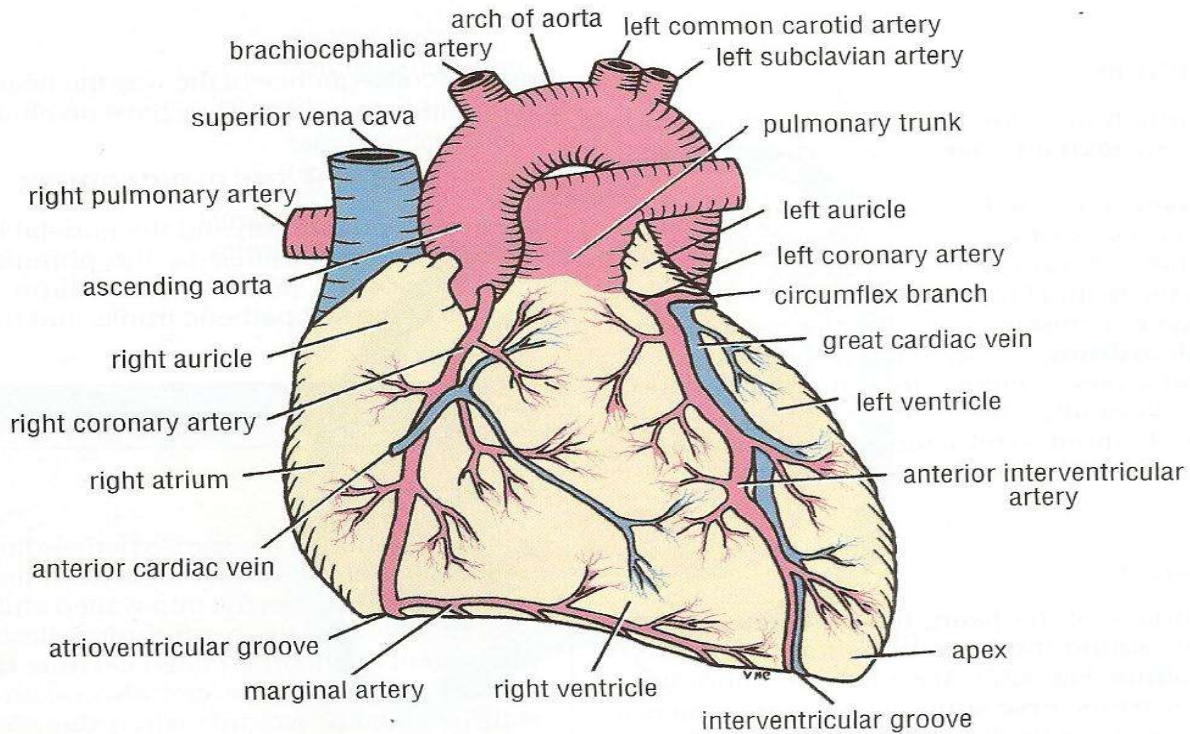


Figure 2.13,AThe anterior surface of the heart and the great vessels. Note the course of the coronary arteries and the cardiac veins (*Richard S.Snell,1992*).

The **diaphragmatic surface** of the heart is formed mainly by the right and left ventricles separated by the posterior interventricular groove. The inferior surface of the right atrium , into which the inferior vena cava opens, also forms part of this surface.

The **base of the heart** , or the posterior surface , is formed mainly by the left atrium , into which open the four pulmonary veins (**Figure 2.13,B**). The base of the heart lies opposite the apex.

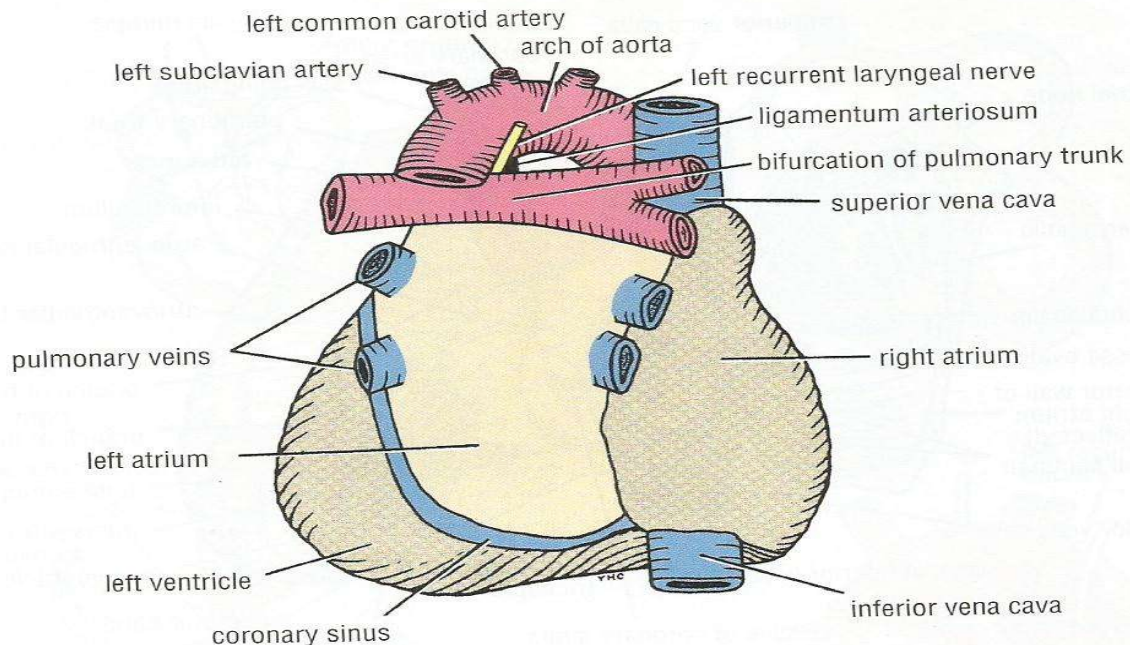


Figure 2.13,BThe posterior surface , or the base, of the heart (*Richard S.Snell,1992*).

The **apex of the heart** , formed by the left ventricle , is directed downward, forward, and to the left (**Figure2.13,B**). It lies at the level of the fifth left intercostal space, 3.5 cm in.(9 cm) from the midline. In the region of the apex, the apex beat can usually be seen and palpated in the living patient.

Note that the base of the heart is called the base because the heart is pyramid shaped ; the base lies opposite the apex. The heart does not rest on its base; it rests on its diaphragmatic (inferior) surface(*Chummy S-Sinnatamby,2006*) and (*Richard S.Snell,1992*).

2.1.1.18The Valves

The surface markings of the heart valves are as follows (Figure 214):

The **tricuspid valve** lies behind the right half of the sternum opposite the four intercostal space.

The **mitral valve** lies behind the left half of the sternum opposite the fourth costal cartilage .

The **pulmonary valve** lies behind the medial end of the third left costal cartilage and the adjoining part of the sternum.

The **aortic valve** lies behind the left half of the sternum opposite the third intercostal space (*Richard S.Snell,1992*).

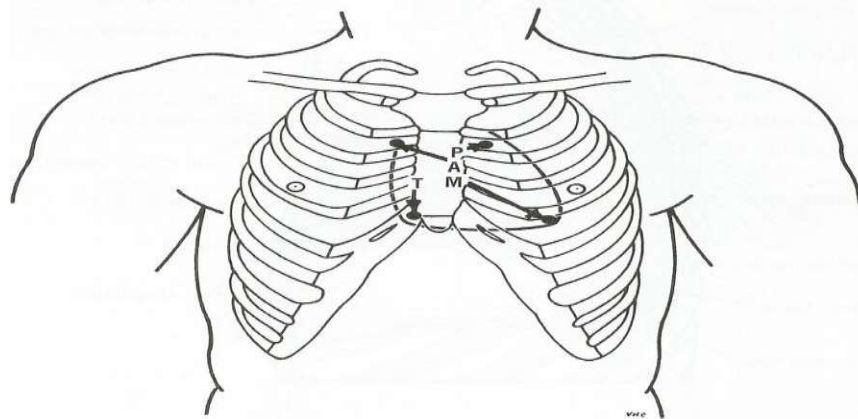


Figure 2.14 Position of the heart valves. *P*, Pulmonary valve ;*A* , aortic valve ; *M* , mitral valve; *T*, tricuspid valve. Arrows indicate position where valves may be heard with last interference (*Richard S.Snell,1992*) .

2.1.2 Major Blood vessels in the thorax.

The major blood vessels comprise the pulmonary trunk , ascending aorta, the aortic arch, the thoracic aorta and its branches, the azygous vein the superior and inferior venae cava and their tributaries(*Susan Standring , 2008*) .

2.1.2.1 Arteries

2.1.2.1.1 Pulmonary trunk

The pulmonary trunk, or pulmonary artery, conveys deoxygenated blood from the right ventricle to the lungs. About 5 cm in length and 3 cm in diameter , it is the most anterior of the cardiac vessels and arises from the base of the right ventricle above and to the left+ of the supraventricular crest. It slopes up and back, at first in front of the ascending aorta, then to its left. Below the aortic arch it divides , level with the fifth thoracic vertebra and to the left of the midline , into right and left pulmonary arteries of almost equal size. The pulmonary trunk

bifurcation lies below, in front and to the left of the tracheal bifurcation (which is also associated with the inferior tracheobronchial lymph nodes and the deep cardiac nerve plexus). In the fetus, at the level of the bifurcation the pulmonary artery is connected to the aortic arch by the ductus arteriosus, which lies in the same direction as the pulmonary artery.

During fetal life, when blood pressure is similar in the pulmonary artery and the aorta, the structure of the vessels is similar. After birth, the lungs expand and pulmonary arteries dilate, and so pulmonary vascular resistance decreases, whereas blood flow increases. The systolic pressure in the pulmonary artery consequently decreases and this is accompanied by a structural remodelling of its wall. The thickness of the wall of the aorta is about twice that of the pulmonary artery (*Susan Standring, 2008*).

2.1.2.1.2 Ascending aorta :

The ascending aorta is typically 5 cm long and begins at the base of the left ventricle, level with the lower border of the third left costal cartilage; it ascends obliquely, curving forwards and to the right, behind the left half of the sternum to the level of the upper border of the second left costal cartilage. At its origin, proximal to the aortic anulus, the sectional profile is larger and not circular because of three almost hemispherical outward bulges, one posterior (non-coronary), one left and one right, which correspond to the three cusps of the aortic valve (see above). Distal to the aortic anulus there are three aortic sinuses, beyond which the calibre of the vessel is slightly increased by a bulging of its right wall. This aortic bulb gives the vessel an oval section. (**Figure 2.15**) and (**Figure 2.20,A**) (*Susan Standring, 2008*).

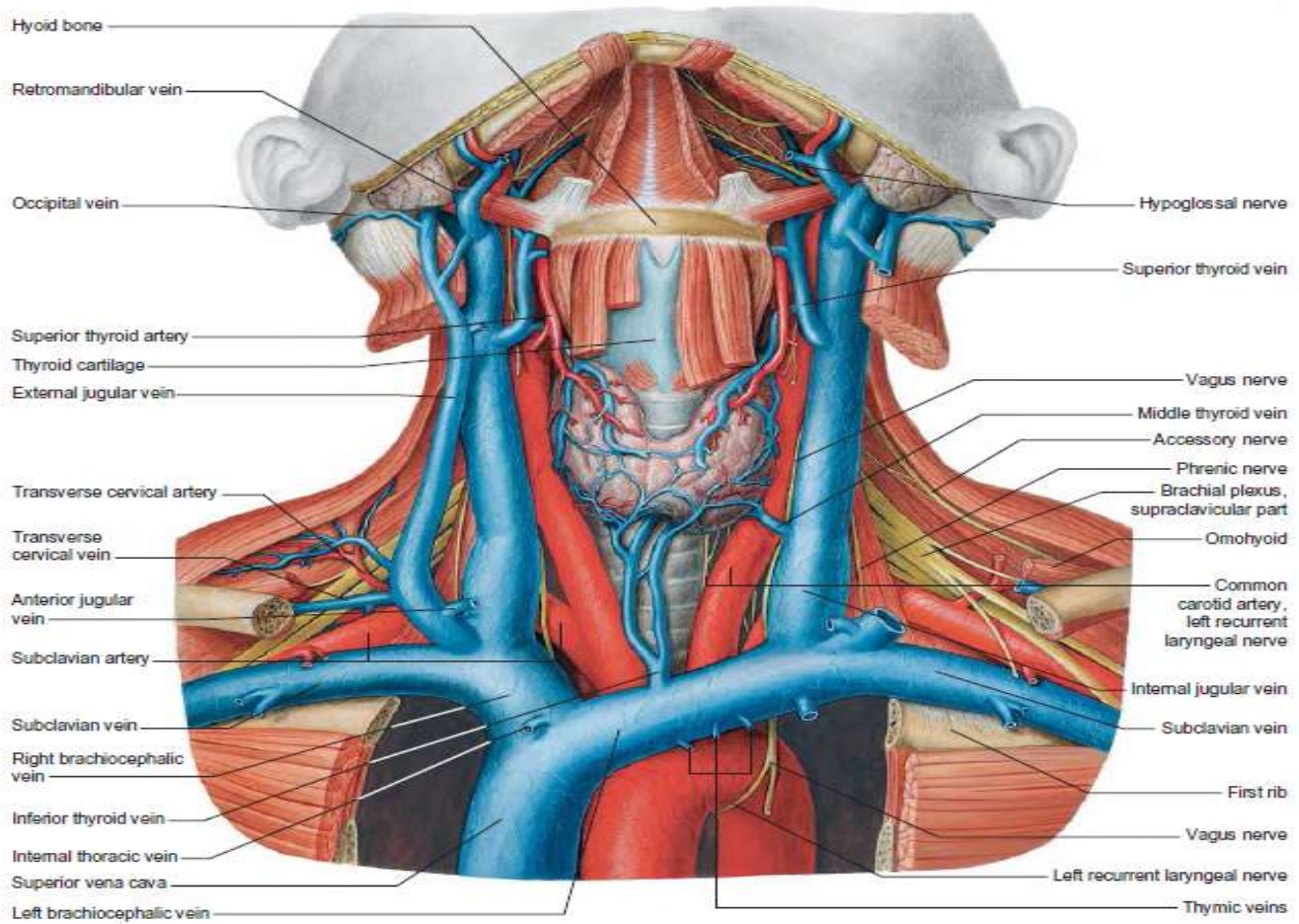
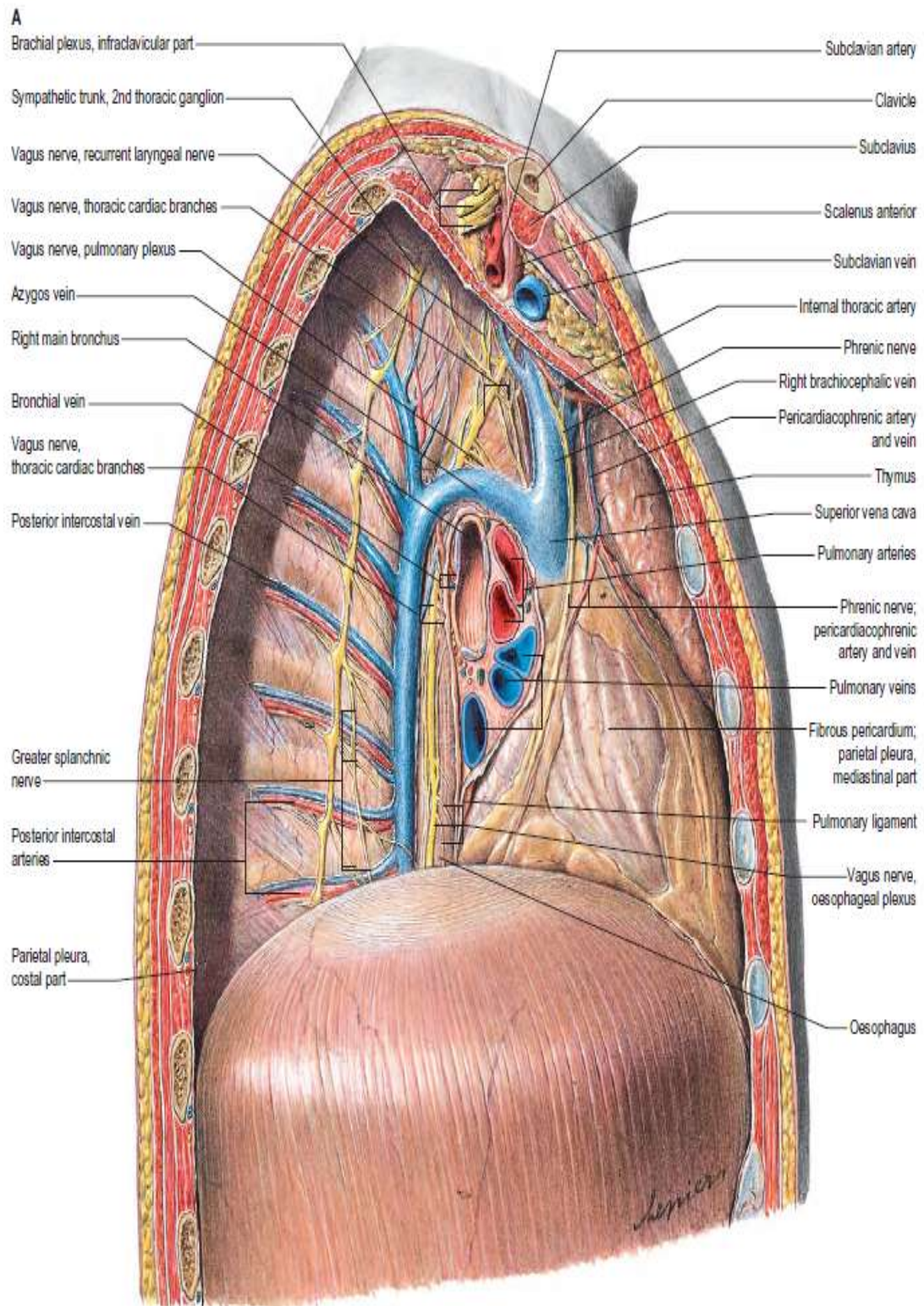


Figure 2.15 The veins of the neck, seen from the front and at a deep level. Both sternocleidomastoids have been removed and additional dissection has exposed the thyroid gland and some of the structures that pass through the upper thoracic aperture. (From Sobotta 2006) (Susan Standring, 2008).



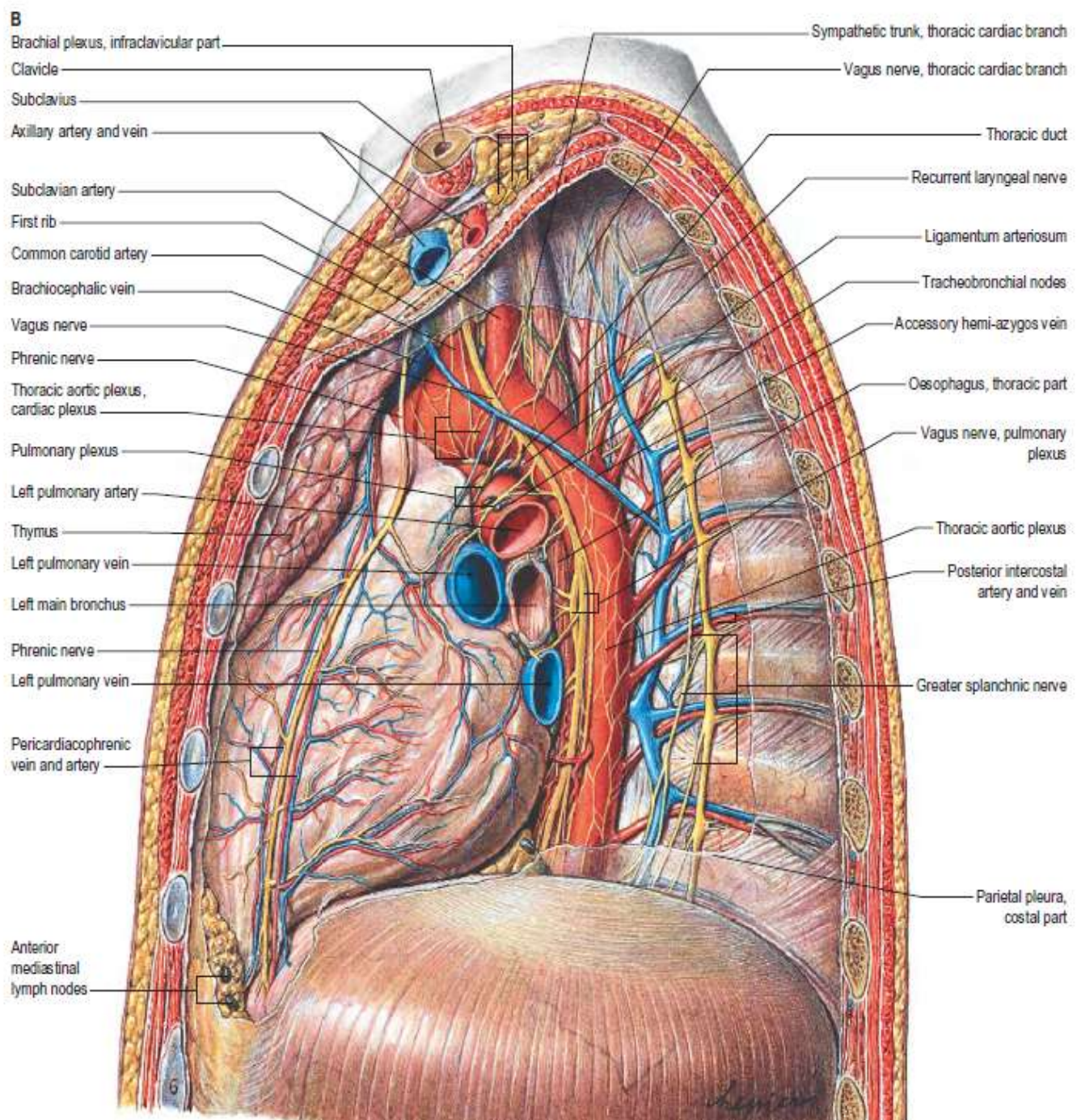


Figure 2.16,A and B The mediastinum A.Right lateral aspect.shows azygos vein SVC (From Sobotta 2006.)B. Left lateral aspect.thoracic aorta . (From Sobotta 2006) (Susan Standing , 2008).

2.1.2.1.3 Aortic arch :

The aortic arch continuous from the ascending aorta (**Figure 2.17**), its origin, slightly to the right , at level of the upper border of the second right sternocostal joint. The arch first ascends diagonally back and to the left over the anterior surface of the trachea, then back across its left side and finally descends to the left of the fourth thoracic vertebral body , continuing as the descending thoracic aorta. It ends level with the sternal end of the second, left costal cartilage. Thus the aortic arch lies wholly in the superior mediastinum. It curves around the hilum of the left lung, and extends upwards to the mid-level of the manubrium of the sternum. The shadow of the arch is easily identified in anteroposterior radiographs and its left profile is sometimes called the `aortic knuckle` (**Figure 2.18**).

The arch may also be visible in left anterior oblique views enclosing a pale space, `the aortic window`, in which shadows of the pulmonary trunk and its left branch may be discerned .its diameter at the origin is the same as in the ascending aorta, 28 mm , but it is reduced to 20 mm at the end , after the issue of its large collateral branches. At the border with the thoracic aorta, a small stricture (aortic isthmus), followed by a dilation , can be recognized. In fetal life the isthmus lies between the origin of the left subclavian artery and the opening of the ductusarteriosus.

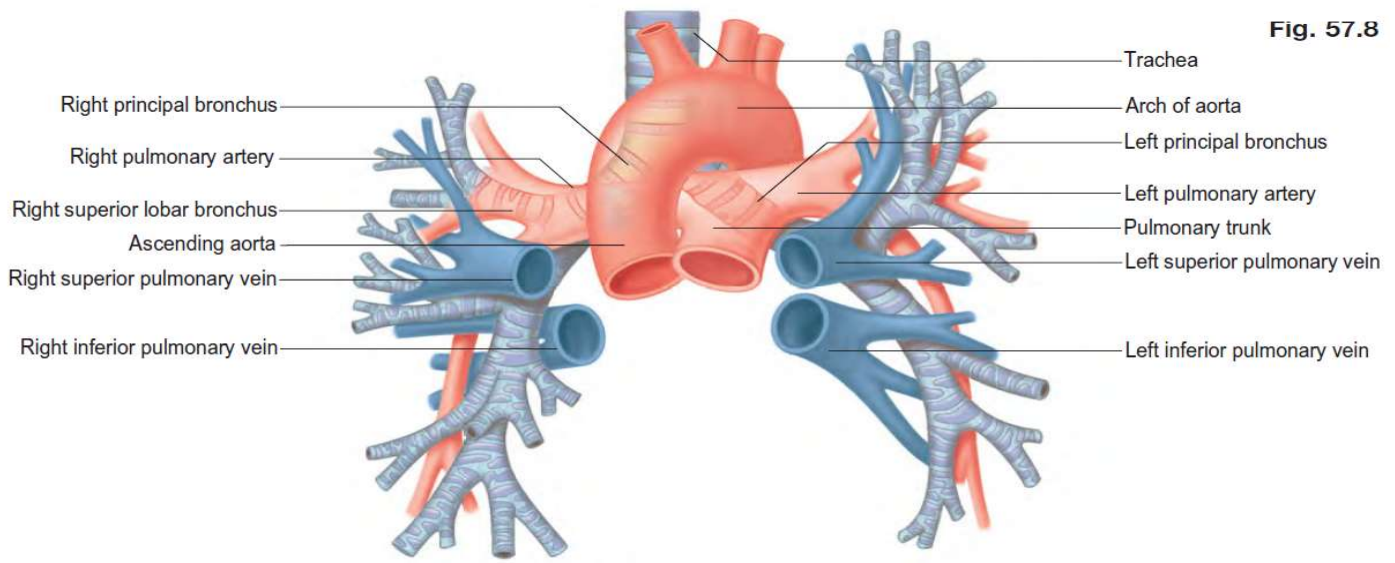


Figure 2.17 The relationship between the central airways and the pulmonary vessels .aortic arch and branches(*Susan Standring , 2008*) .

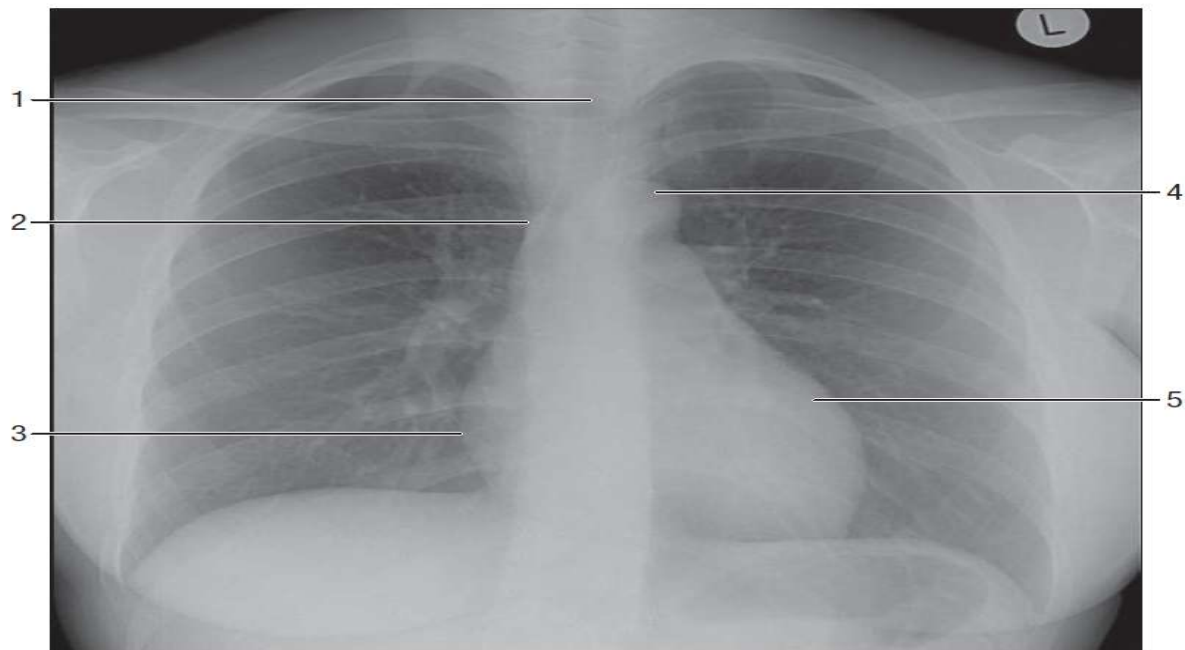


Figure 2.18 Radiograph of chest of adult female, Posteroanterior view. 1. Trachea. 2. Azygos vein. 3. Right atrial border. 4. Aortic arch,(aortic knuckle) 5. Left ventricular border (*Susan Standring , 2008*) .

Branches, three branches arise from the convex aspect of the arch : the bronchiocephalic trunk, left common carotid and left subclavian arteries (**Figure 2.17**). They may branch from the beginning of the arch or the upper part of the ascending aorta. The distance between these origins varies, the most frequent being approximation of the left common carotid artery to the bronchiocephalic trunk. one or both bronchial arteries and the thyroidea artery.

An analysis of variation in branches from 1000 aortic arches showed the usual pattern in 65%; a left common carotid shared the brachiocephalic trunk in 27% and the four large arteries branched separately in 2.5% . The remaining 5% showed a great variety of patterns, the most common (1.2%) being symmetric right and left brachiocephalic trunks *From Gray's anatomy (Susan Standring , 2008)*.

2.1.2.1.3.1 Brachiocephalic artery :

The brachiocephalic (innominate) artery, the largest branch of the aortic arch. It arises from the convexity of the arch posterior to the centre of the manubrium of sternum , and ascends posteriolaterally to the right , at first anterior to the trachea, then on its right . Level with the upper border of the right sternoclavicular joint. it divides into the right common carotid and right subclavian arteries.

Branches, the brachiocephalic artery usually has only terminal branches, the right common carotid and right subclavian artery. Occasionally a thymic or bronchial branch, or a thyroidea artery arises from it. The thyroidea artery is a small and inconstant artery which may arise from the aorta , right common carotid, subclavian or internal thoracic arteries; it ascends on the trachea to the thyroid isthmus, where it terminates (*Susan Standring , 2008*) .

2.1.2.1.3.2 Subclavian arteries

2.1.2.1.3.2.1 Right subclavian artery:

The right subclavian artery arises from the brachiocephalic trunk. The right subclavian artery is formed behind the upper border of the right sternoclavicular joint. It ascends above the clavicle superomedial and then posterior to scalenus anterior. It next descends laterally to scalenus anterior, to the outer border of the first rib, where it becomes the axillary artery (*Susan Standring , 2008*).

2.1.2.1.3.2.2 Left subclavian artery

In the majority of individuals, the left subclavian artery originates independently from the aortic arch after the origin of the brachiocephalic trunk and left common carotid artery. The left subclavian artery arises from the aortic arch below the left common carotid artery and rises into the neck lateral to the medial border of scalenus anterior, cross behind this muscle and then descends towards the outer border of the first rib, where it becomes the axillary artery. A common origin exists occasionally between the left subclavian artery and left vertebral artery (*Susan Standring , 2008*).

2.1.2.1.3.2.3 Common carotid arteries:

The right and left common carotid arteries differ in length and origin. The right common carotid is exclusively cervical, and arises from the brachiocephalic trunk behind the right sternoclavicular joint. The left common carotid originates directly from the aortic arch immediately posterolateral to the brachiocephalic trunk and therefore has both thoracic and cervical parts (*Susan Standring , 2008*).

2.1.2.1.4 Descending thoracic aorta :

The thoracic aorta is the segment of descending aorta confined to the posterior mediastinum (**Figure 2.19**) It begins at level with the lower border of the fourth thoracic vertebra, continuous with the aortic arch, and ends anterior to the lower border of the 12th thoracic vertebra in the diaphragmatic aortic aperture. At

its origin it is left of the vertebral column , as it descends it approaches the midline, and at its termination is directly anterior to it(*Susan Standring , 2008*).

2.1.2.1.4.1 Branches the thoracic aorta

Provides visceral branches to the pericardium , lungs, bronchi and oesophagus , and parietal branches to the thoracic wall. Pericardial branches A few small vessels are distributed to the posterior aspect of the pericardium(*Susan Standring , 2008*).

2.1.2.1.4.1.1 Bronchial arteries

Bronchial arteries vary in number, size and origin . There is usually only one right bronchial artery which arises either from the third posterior intercostal or upper left bronchial artery, and runs posteriorly on the right bronchus. Its branches supply these structures, in addition to the pulmonary areolar tissue and the bronchopulmonary lymph nodes, pericardium and esophagus. The left bronchial arteries , usually two, arise from the thoracic aorta, the upper near the fifth thoracic vertebra , the lower below the left bronchus, and run posteriorly to the left bronchus; they are distributed as on the right (*Susan Standring , 2008*).

2.1.2.1.4.1.2 Mediastinal branches

Numerous small vessels supply lymph nodes and areolar tissue in the posterior mediastinum(*Susan Standring , 2008*).

2.1.2.1.4.1.3 Phrenic branches

Phrenic branches arise from the lower thoracic aorta and are distributed posteriorly to the superior diaphragmatic surface. They anastomose with the musculophrenic and pericardiophrenicarteries*From Gray`s anatomy (Susan Standring , 2008)*.

2.1.2.1.4.1.4 Subcostal arteries

Subcostal arteries are the last paired branches of the thoracic aorta, in series with the posterior intercostal arteries, and below the twelfth ribs. Each runs laterally anterior to the twelfth thoracic vertebral body and posterior to the splanchnic nerves, sympathetic trunk, pleura and diaphragm .

The right is also posterior to the thoracic duct and azygos vein, the left is posterior to the accessory hemiazygos vein. Each then enters the abdomen at the lower border of the 12th rib, accompanied by the 12th thoracic (subcostal) nerve, lying posterior to the lateral arcuate ligament and kidney, and anterior to quadratus lumborum.

The right artery courses posterior to the ascending colon, the left posterior to the descending colon. Piercing the aponeurosis of transversus abdominis, each proceeds between this and internal oblique, and anastomoses with the superior epigastric, lower posterior intercostal and lumbar arteries. Each has dorsal branch, distributed like those of the posterior intercostal arteries (*Susan Standring, 2008*).

2.1.2.2 The Veins

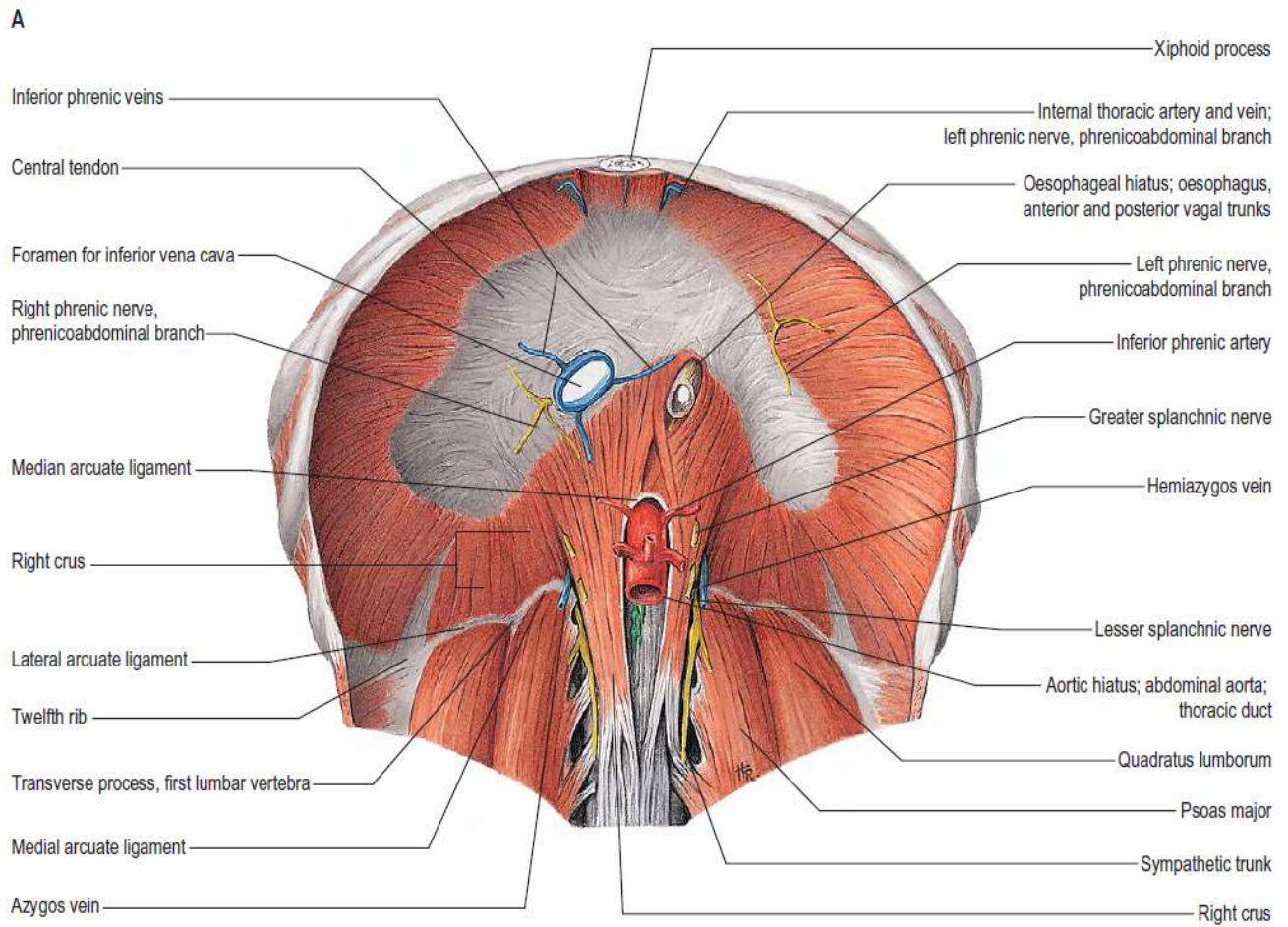
2.1.2.2.1 Superior vena cava:

The superior vena cava returns blood to the heart from the tissues above the diaphragm. It is approximately 7 cm in length and is formed by the junction of the branchiocephalic veins behind the lower border of the first right costal cartilage near the sternum. It descends vertically behind the first and second intercostal spaces, and ends in the upper right atrium behind the third right costal cartilage (**Figure 2.15**). Its inferior half is within the fibrous pericardium, which it pierces level with the second costal cartilage. Covered anterolaterally by serous pericardium (from which a retrocaval recess projects), it is slightly convex to the right. The superior vena cava has no valves (*Susan Standring, 2008*).

2.1.2.2.2 Inferior vena cava:

The inferior vena cava returns blood to the heart from the tissues below the diaphragm. It passes through the diaphragm between the right leaf and central area of the central tendon of the diaphragm at the level of the eighth and ninth thoracic vertebrae, and drains into the inferoposterior part of the right atrium (**Figure 2.19, A and B**).

The thoracic part is very short, and is partly inside and partly outside the pericardial sac. The extrapericardial part is separated from the right pleura and lung by the right phrenic nerve, and the interpericardial part is covered, except posteriorly, by inflected serous pericardium (*Susan Standring, 2008*).



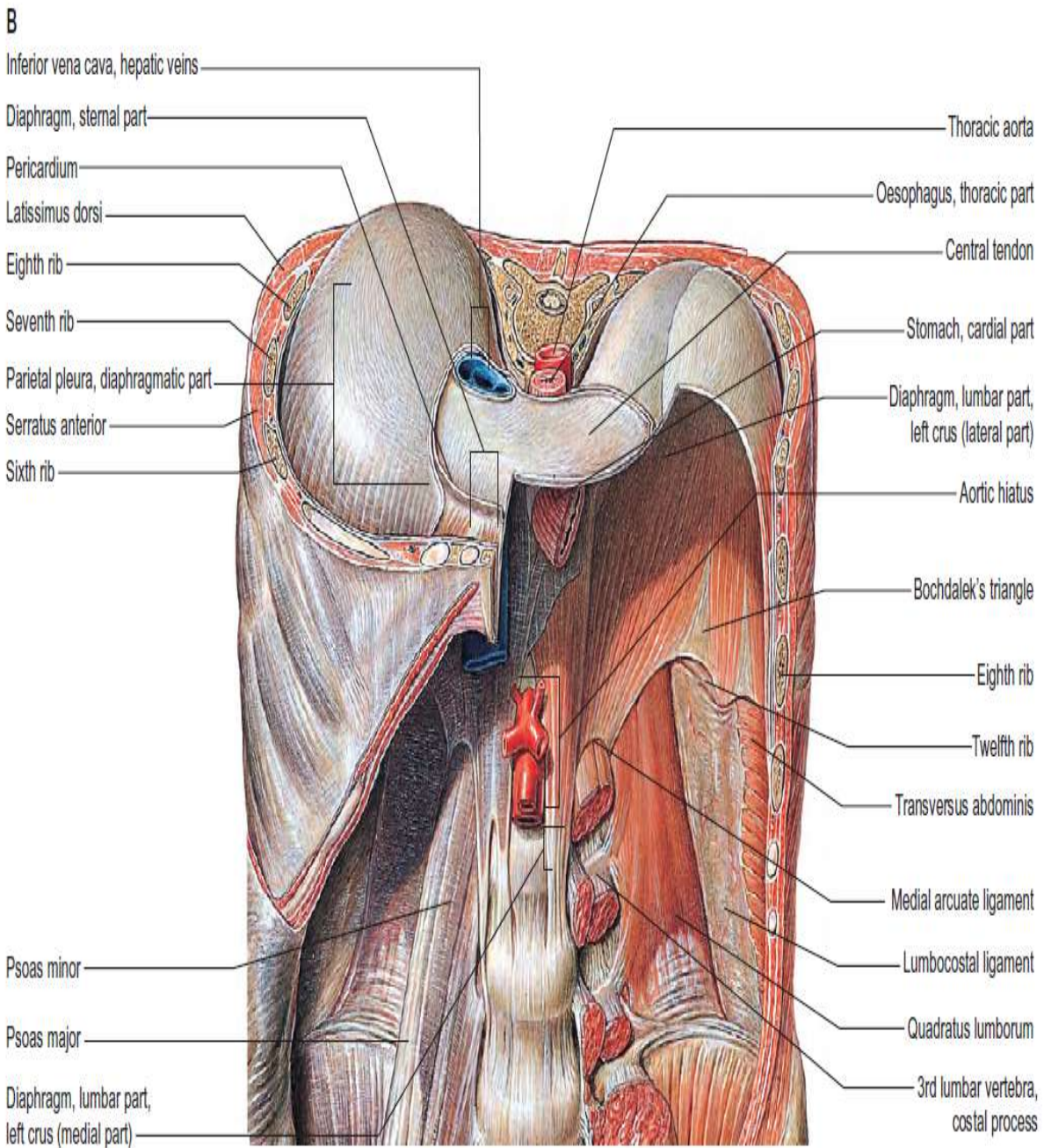


Fig. 58.1 **he**
oesophagus and inferior vena cava and the aortic hiatus. The medial part of the right crus
frequently consist of three components and extend further distally than the left crus. B,
Trunk sectioned at the level of the tenth thoracic vertebra. (From Sobotta 2006) (Susan
Standring , 2008).

2.1.2.2.3 Brachiocephalic veins :

The right and left brachiocephalic veins join to form the superior vena cava (*Susan Standring , 2008*).

2.1.2.2.3.1 Right brachiocephalic veins :

The right brachiocephalic vein is about 2.5 cm long, and begins posterior to the sternal end of the right clavicle. It descends almost vertically to join the left brachiocephalic vein, forming the superior vena cava posterior to the lower border of the first right costal cartilage, near the right sternal border. It is anterolateral to the brachiocephalic artery and right vagus nerve; the right pleura, phrenic nerve and internal thoracic artery are posterior to it above, and become lateral below (**Figure 2.19**) (*Susan Standring , 2008*).

2.1.2.2.3.2 Left brachiocephalic vein :

The left brachiocephalic vein is longer than the right, being some 6 cm long. It begins posterior to the sternal end of the left clavicle, anterior to the cervical pleura, and descends obliquely to the right, posterior to the upper half of the manubrium sterni, to the sternal end of the first right costal cartilage, where it joins the right brachiocephalic vein to form the superior vena cava. It is separated from the left sternoclavicular joint and manubrium by sternohyoid and sternothyroid, the thymus or its remains, and areolar tissue; terminally it is overlapped by the right pleura. It crosses anterior to the left internal thoracic, subclavian, brachiocephalic and common carotid arteries, left phrenic and vagus nerves, and the trachea. The aortic arch is inferior to it (*Susan Standring , 2008*).

2.2 Physiology

2.2.1 The Heart :

The circulation system transports and distributes essential substances to tissues and removes metabolic byproducts. This system also participates in homeostatic mechanisms such as regulation of body temperature, maintenance of fluid balance, and adjustment of O₂ and nutrient supply under various physiological states.

The cardiovascular system that accomplishes these tasks is composed of a pump (the heart), a series of distributing and collecting tubes (blood vessels), and an extensive system of thin vessels (capillaries) that permit rapid exchange between the tissues and vascular channels. Blood vessels throughout the body are filled with a heterogeneous fluid (blood) that is essential for the transport processes performed by the heart and blood vessels (*Koeppen and Stanton, 2010*).

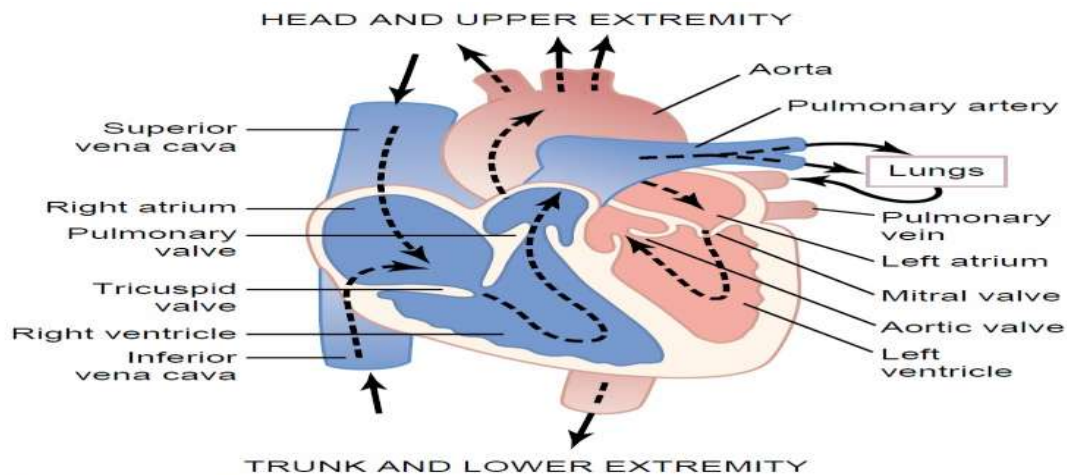


Figure 2.20 Structure of the heart, and course of blood flow through the heart chambers and heart valves(*Guyton, & Hall , 2006*).

2.2.2 The Cardiac Cycle:

The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called the *cardiac cycle*. Each cycle is initiated by

spontaneous generation of an action potential in the *sinus node*(Guyton, & Hall , 2006).

2.2.2.1 Diastole and Systole :

The cardiac cycle consists of a period of relaxation called *diastole*, during which the heart fills with blood , followed by a period of contraction called *systole*. (Figure 2.21) shows the different events during the cardiac cycle for the left side of the heart.

The top three curves show the pressure changes in the aorta, left ventricle ,and left atrium, respectively.

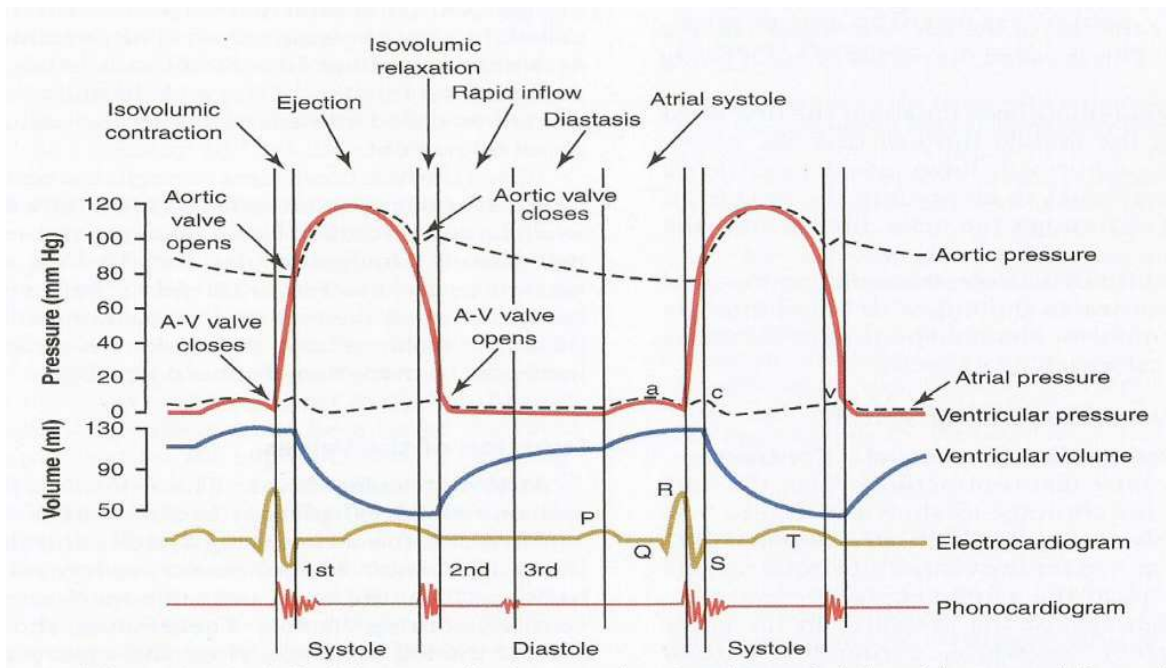


Figure 2.21 Events of the cardiac cycle for left ventricular function, showing changes in left atrial pressure, left ventricular pressure, aortic pressure ,ventricular volume, the electrocardiogram, and the phonocardiogram (Guyton, & Hall , 2006).

The fourth curve depicts the changes in left ventricular volume, the fifth the electrocardiogram, and the sixth a phonocardiogram ,which is a recording of the sounds produced by the heart—mainly by the heart valves—as it pumps. It is

especially important that the reader study in detail this figure and understand the causes of all the events shown (*Guyton, & Hall, 2006*).

2.2.2.2 Relationship of the Electrocardiogram to the Cardiac Cycle :

The electrocardiogram in (**Figure 2.21**) shows the *P, Q,R, S,* and *T waves*. They are electrical voltages generated by the heart and recorded by the electrocardiograph from the surface of the body.

The *P wave* is caused by *spread of depolarization* through the atria, and this is followed by atrial contraction ,which causes a slight rise in the atrial pressure curve immediately after the electrocardiographic P wave.

About 0.16 second after the onset of the P wave, the *QRS waves* appear as a result of electrical depolarization of the ventricles, which initiates contraction of the ventricles and causes the ventricular pressure to begin rising, as also shown in the figure. Therefore, the QRS complex begins slightly before the onset of ventricular systole.

Finally, one observes the *ventricular T wave* in the electrocardiogram. This represents the stage of repolarization of the ventricles when the ventricular muscle fibers begin to relax. Therefore, the T wave occurs slightly before the end of ventricular contraction (*Guyton, & Hall, 2006*).

2.2.2.3 Function of the Atria as Primer Pumps :

Blood normally flows continually from the great veins into the atria; about 80 per cent of the blood flows directly through the atria into the ventricles even before the atria contract.

Then, atrial contraction usually causes an additional 20 per cent filling of the ventricles. Therefore, the atria simply function as primer pumps that increase the ventricular pumping effectiveness as much as 20 per cent. However, the heart can continue to operate under most condition even without this extra 20 per cent

effectiveness because it normally has the capability of pumping 300 to 400 per cent more blood than is required by the resting body.

Therefore, when the atria fail to function, the difference is unlikely to be noticed unless a person exercises; then acute signs of heart failure occasionally develop, especially shortness of breath (*Guyton, & Hall, 2006*).

2.2.2.4 Pressure Changes in the Atria—The a, c, and v Waves :

In the atrial pressure curve of (**Figure 2.21**) three minor pressure elevations, called the a, c, and v atrial pressure waves, are noted .

The a wave is caused by atrial contraction. Ordinarily, the right atrial pressure increases 4 to 6 mm Hg during atrial contraction, and the left atrial pressure increases about 7 to 8 mm Hg.

The c wave occurs when the ventricles begin to contract; it is caused partly by slight backflow of blood into the atria at the onset of ventricular contraction but mainly by bulging of the A-V valves backward toward the atria because of increasing pressure in the ventricles.

The v wave occurs toward the end of ventricular contraction; it results from slow flow of blood into the atria from the veins while the A-V valves are closed during ventricular contraction.

Then, when ventricular contraction is over, the A-V valves open, allowing this stored atrial blood to flow rapidly into the ventricles and causing the v wave to disappear (*Guyton, & Hall, 2006*).

2.2.2.5 Function of the Ventricles as Pumps :

2.2.2.5.1 Filling of the Ventricles, During ventricular systole:

Large amounts of blood accumulate in the right and left atria because of the closed A-V valves.

Therefore, as soon as systole is over and the ventricular pressures fall again to their low diastolic values, the moderately increased pressures that have

developed in the atria during ventricular systole immediately push the A-V valves open and allow blood to flow rapidly into the ventricles, as shown by the rise of the left *ventricular volume curve* in **(Figure 2.21)**.

This is called the *period of rapid filling of the ventricles*. The period of rapid filling lasts for about the first third of diastole. During the middle third of diastole, only a small amount of blood normally flows into the ventricles; this is blood that continues to empty into the atria from the veins and passes through the atria directly into the ventricles.

During the last third of diastole, the atria contract and give an additional thrust to the inflow of blood into the ventricles; this accounts for about 20 per cent of the filling of the ventricles during each heart cycle *(Guyton, & Hall, 2006)*.

2.2.2.6 Emptying of the Ventricles During Systole:

2.2.2.6.1 Period of Isovolumic (Isometric) Contraction:

Immediately after ventricular contraction begins, the ventricular pressure rises abruptly, as shown in **(Figure 2.21)**, causing the A-V valves to close. Then an additional 0.02 to 0.03 second is required for the ventricle to buildup sufficient pressure to push the semilunar (aortic and pulmonary) valves open against the pressures in the aorta and pulmonary artery.

Therefore, during this period, contraction is occurring in the ventricles, but there is no emptying. This is called the period of *isovolumic* or *isometric contraction*, meaning that tension is increasing in the muscle but little or no shortening of the muscle fibers is occurring *(Guyton, & Hall, 2006)*.

2.2.2.6.2 Period of Ejection:

When the left ventricular pressure rises slightly above 80 mm Hg (and the right ventricular pressure slightly above 8 mm Hg), the ventricular pressures push the semilunar valves open.

Immediately , blood begins to pour out of the ventricles, with about 70 per cent of the blood emptying occurring during the first third of the period of ejection and the remaining 30 per cent emptying during the next two thirds.

Therefore, the first third is called the *period of rapid ejection*, and the last two thirds, the *period of slow ejection* (Guyton, & Hall , 2006).

2.2.2.6.3 Period of Isovolumic (Isometric) Relaxation:

At the end of systole, ventricular relaxation begins suddenly, allowing both the right and left *intra ventricular pressures* to decrease rapidly.

The elevated pressures in the distended large arteries that have just been filled with blood from the contracted ventricles immediately push blood back toward the ventricles, which snaps the aortic and pulmonary valves closed.

For another 0.03to 0.06 second, the ventricular muscle continues to relax, even though the ventricular volume does not change, giving rise to the period of *isovolumic or isometric relaxation*. During this period, the intra ventricular pressures decrease rapidly back to their low diastolic levels. Then the A-V valves open to begin anew cycle of ventricular pumping(Guyton, & Hall , 2006).

2.2.2.7 End-Diastolic Volume, End-Systolic Volume, and Stroke Volume Output:

During diastole, normal filling of the ventricles increases the volume of each ventricle to about 110 to120 milliliters. This volume is called the *end-diastolic volume*. Then, as the ventricles empty during systole, the volume decreases about 70 milliliters, which is called the *stroke volume output*. The remaining volume in each ventricle, about 40 to 50 milliliters, is called the *end-systolic volume*.

The fraction of the end-diastolic volume that is ejected is called the *ejection fraction*—usually equal to about 60 per cent. When the heart contracts strongly, the end-systolic volume can be decreased to as little as 10 to 20 milliliters.

Conversely, when large amounts of blood flow into the ventricles during diastole, the ventricular end diastolic volumes can become as great as 150 to 180 milliliters in the healthy heart. By both increasing the end-diastolic volume and decreasing the end-systolic volume, the stroke volume output can be increased to more than double normal (*Guyton, & Hall, 2006*).

2.2.2.8 Aortic Pressure Curve:

When the left ventricle contracts, the ventricular pressure increases rapidly until the aortic valve opens. Then, after the valve opens, the pressure in the ventricle rises much less rapidly, as shown in (**Figure 2.21**) , because blood immediately flows out of the ventricle into the aorta and then into the systemic distribution arteries.

The entry of blood into the arteries causes the walls of these arteries to stretch and the pressure to increase to about 120 mm Hg.

Next, at the end of systole, after the left ventricle stops ejecting blood and the aortic valve closes, the elastic walls of the arteries maintain a high pressure in the arteries, even during diastole. A so-called *incisura* occurs in the aortic pressure curve when the aortic valve closes.

This is caused by a short period of backward flow of blood immediately before closure of the valve, followed by sudden cessation of the backflow. After the aortic valve has closed, the pressure in the aorta decreases slowly throughout diastole because the blood stored in the distended elastic arteries flows continually through the peripheral vessels back to the veins.

Before the ventricle contracts again, the aortic pressure usually has fallen to about 80 mm Hg (diastolic pressure), which is two thirds the maximal pressure of 120 mm Hg (systolic pressure) that occurs in the aorta during ventricular contraction.

The pressure curves in the *right ventricle* and *pulmonary artery* are similar to those in the aorta, except that the pressures are only about one sixth as great as **(Figure 2.21)**(*Guyton, & Hall, 2006*).

2.2.3 Properties of the Vasculature:

The vascular consists of a closed system of tubes or vessels that distributes blood from the heart to the tissues and return blood from the tissues to the heart.

It can be divided into three components: the **arterial system**, which takes blood from the heart and distributes it to the tissues; **the venous system** ; which returns blood from the tissues to the heart; and **the micro circulation** , which separates the arterial and venous systems and is the site where nutrients and cellular waste products are exchanged between blood and tissues.

In addition, the properties of blood flow to specific vascular beds and tissues are considered . As an introduction to this material , the physics of blood/fluid flow through the vasculature (i.e, hemodynamics) is reviewed (*Koeppen, and Stanton ,2010*).

2.2.3.1. Arterial Elasticity :

The systemic and pulmonary arterial system distribute blood to the capillary beds throughout the body. The arteries are high-resistance vessels of this system that regulate the distribution of flow to the various capillary beds. The aorta, the pulmonary artery , and their major branches have a large amount of elastin in their walls, which makes these vessels highly distensible (i.e., compliant).

This distensibility serves to dampen the heart pumping blood intermittently. When blood is ejected from the ventricles during systole, these vessels distend, and during diastole, they recoil back and propel the blood forward **(Figure 2.22)** Thus, the intermittent output of the heart is converted to a steady flow through the capillaries.

The elastic nature of the large arteries also reduces the work of the heart. If these arteries were rigid rather than compliant, the pressure would rise dramatically during systole. This increased pressure would require the ventricles to pump against a large load (i.e., after load) and thus increase the work of the heart. Instead, as blood is ejected into these vessels, they distend, and the resultant increase in systolic pressure, and thus the work of the heart, is reduced (Koeppen, and Stanton, 2010).

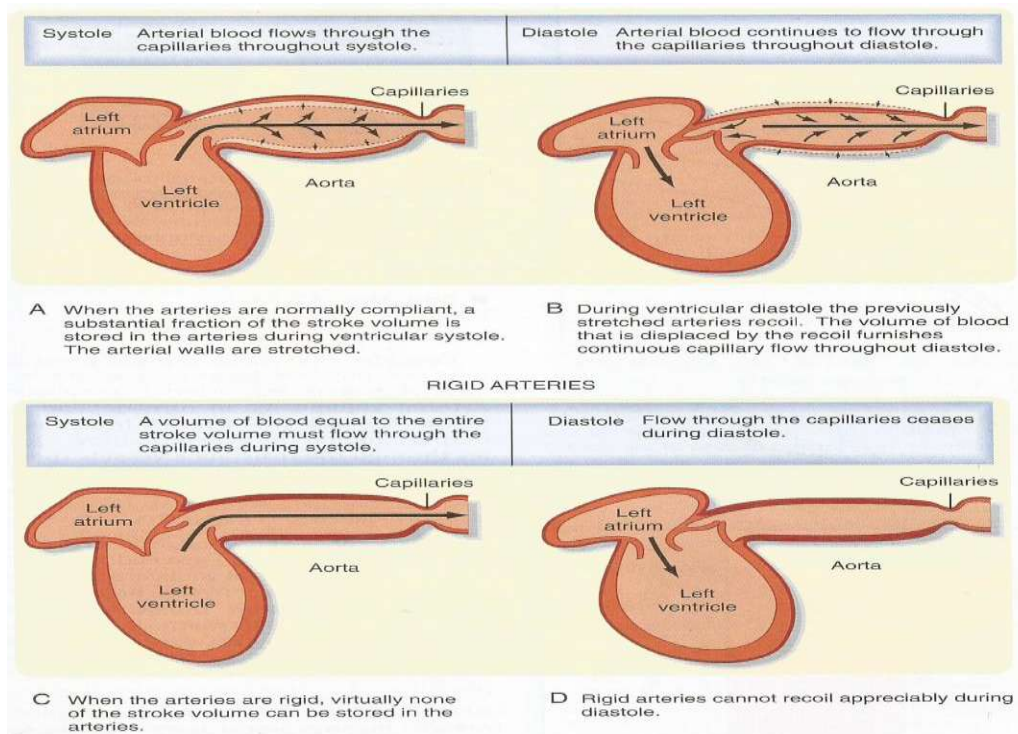


Figure 2.22A to D, when the arteries are normally compliant, blood flows through the capillaries throughout the cardiac cycle. When the arteries are rigid, blood flows through the capillaries during systole, but flow ceases during diastole (Koeppen, and Stanton, 2010).

2.2.4 Blood pressure:

Blood pressure is the force or pressure that the blood exerts on the walls of the blood vessels. Systemic arterial blood pressure maintains the essential flow of blood into and out of the organs of the body. Keeping blood into sure within normal limits is very important, If it becomes too high, blood vessels can be

damaged, causing clots or low, then blood flow through tissue beds may be inadequate. This is particularly dangerous for such essential organs as the heart, brain or kidneys.

The systemic arterial blood pressure, usually called simply arterial blood pressure, is the result of the discharge of blood from the left ventricle into the already full aorta.

Blood pressure varies according to the time of day, the posture, gender and age of the individual. Blood pressure falls at rest and during sleep. It increases with age and is usually higher in women than in men. *(Anne Waugh and Allison Grant, 2010)*

2.2.4.1 Systolic and diastolic pressure :

When the left ventricle contracts and pushes blood into the aorta, the pressure produced within the arterial system is called the systolic blood pressure. In adults it is about 120 mmHg or 16 kPa. When complete cardiac diastole occurs and the heart is resting following the ejection of blood, the pressure within the arteries is much lower and is called diastolic blood pressure. In an adult this is about 80 mmHg or 11 kPa. *(Anne Waugh and Allison Grant, 2010)*

The difference between systolic and diastolic blood pressures is the pulse pressure. Arterial blood pressure is measured with a sphygmomanometer and is usually expressed with the systolic pressure written above the diastolic pressure :

$$\text{Bp} = 120 / 80 \text{ mmHg} \text{ or } \text{Bp} = 16 / 11 \text{ kPa.} \quad \dots\dots\dots \text{Equation 1}$$

(Anne Waugh and Allison Grant, 2010).

2.2.4.2 Mean arterial pressure :

Mean arterial pressure, P , may be estimated from arterial blood pressure tracing by measuring the area under the pressure curve dividing this area by the time interval involved **(figure 2.23)** *(Koeppen, and Stanton, 2010).*

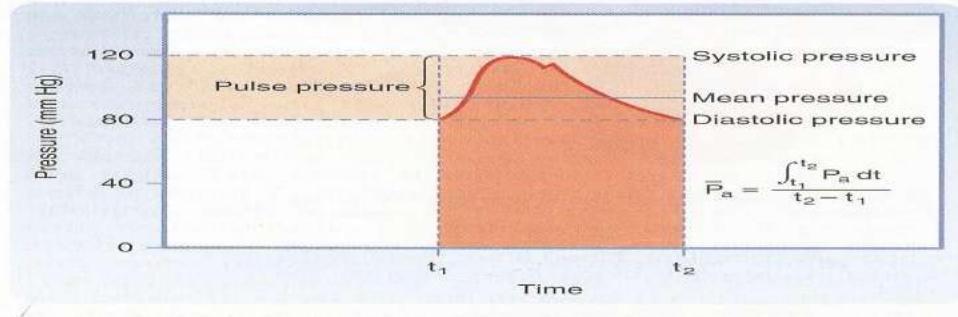


Figure 2.23 Arterial systolic , diastolic , pulse, and mean pressure . Mean arterial pressure(\bar{P}_a) represents the area under the arterial pressure curve (shaded area) divided by the duration of the cardiac cycle(t_2-t_1)(*Koeppen, and Stanton ,2010*).

2.2.4.3. Arterial pulse pressure :

Arterial pulse pressure is systolic pressure minus diastolic pressure. It is principally a function of just one physiological factor, stroke volume , which determines the change in arterial blood volume (a physical factor) during ventricular systole.

This physical factor, pulse a second physical factor (arterial compliance) , determines the arterial pulse pressure (**Figure 2.24**).

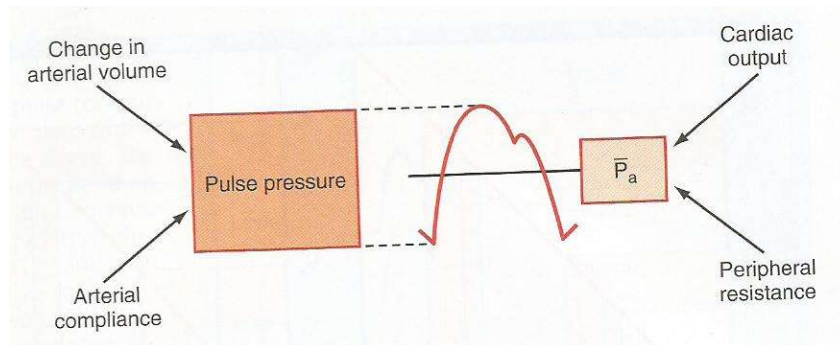


Figure 2.24The two physical determinants of pulse pressure are arterial compliance (C_a) and the change in arterial volume. Two physiological determinants of mean arterial pressure (\bar{P}_a) are cardiac output and total peripheral resistance.(*Koeppen, and Stanton ,2010*).

Stroke Volume. As described previously , mean arterial pressure depends on cardiac output and peripheral resistance. During the rapid ejection phase of systole, the volume of blood introduced into the arterial system exceeds the volume that

exits the system through the arterioles. Arterial pressure and volume therefore rise to a peak pressure, which is systolic pressure.

During the remainder of the cardiac cycle (i.e., ventricular diastole), cardiac ejection is zero, and peripheral runoff now greatly exceeds cardiac ejection. The resultant decrement in arterial blood volume thus causes pressure to fall a minimum, which is diastolic pressure. The effect of stroke volume on pulse pressure when arterial compliance is constant is illustrated in **(Figure 2.25)**(Koeppen, and Stanton, 2010).

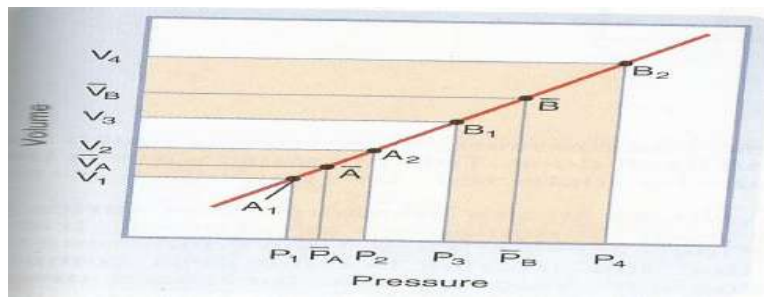


Figure 2.25 Effect of a change in stroke volume on pulse pressure in a system in which arterial compliance remains constant over the prevailing range of pressures and volumes. A large volume increment $[V_4 - V_3] \cdot [V_2 - V_1]$ results in a greater mean pressure ($P_B > P_A$) and a greater pulse pressure $[(P_4 - P_3) \cdot (P_2 - P_1)]$ (Koeppen, and Stanton, 2010).

2.2.5 Maintenance factors of Atrial blood pressure :

The determinants of arterial blood pressure are arbitrarily divided into “physical” and “physiological” factors **(Figure 2.26)**. The two physical factors or fluid mechanical characteristics are fluid volume (i.e., blood volume) within the arterial system and the static elastic characteristics (compliance) of the system. The physiological factors are cardiac output (which equals heart rate x stroke volume) and peripheral resistance (Koeppen, and Stanton, 2010).

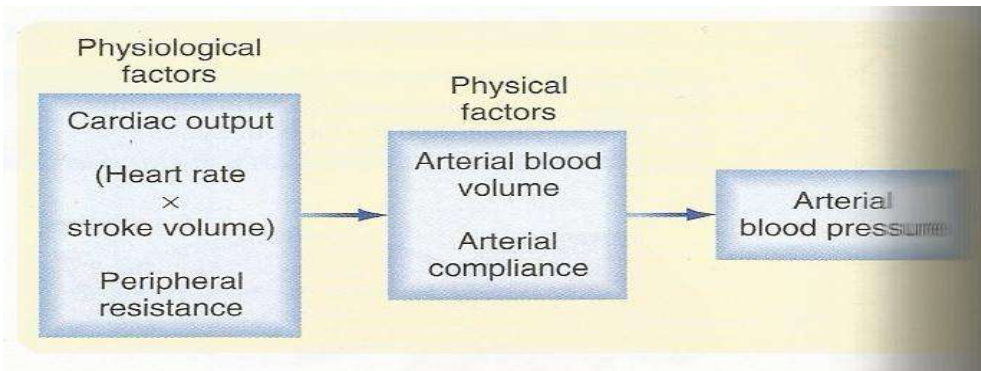


Figure 2.26 Arterial blood pressure is determined directly by two major physical factors: arterial blood volume and arterial compliance. These physical factors in turn are affected by certain physiological factors, namely, cardiac output (heart rate x stroke volume) and peripheral resistance(Koeppen, and Stanton ,2010).

2.2.5.1 Work Output of the Heart:

The *stroke work output* of the heart is the amount of energy that the heart converts to work during each heart beat while pumping blood into the arteries. *Minute work output* is the total amount of energy converted to work in 1 minute; this is equal to the stroke work output times the heart rate per minute. Work output of the heart is in two forms.

First, by far the major proportion is used to move the blood from the low-pressure veins to the high-pressure arteries. This is called *volume-pressure work* or *external work*.

Second, a minor proportion of the energy is used to accelerate the blood to its velocity of ejection through the aortic and pulmonary valves. This is the *kinetic energy of blood flow* component of the work output.

Right ventricular external work output is normally about one sixth the work output of the left ventricle because of the six fold difference in systolic pressures that the two ventricles pump.

The additional work output of each ventricle required to create kinetic energy of blood flow is proportional to the mass of blood ejected times the square of velocity of ejection.

Ordinarily, the work output of the left ventricle required to create kinetic energy of blood flow is only about 1 per cent of the total work output of the ventricle and therefore is ignored in the calculation of the total stroke work output.

But in certain abnormal conditions, such as aortic stenosis, in which blood flows with great velocity through the stenosed valve, more than 50 per cent of the total work output may be required to create kinetic energy of blood flow (*Guyton & Hall, 2006*).

2.2.5.1.1 Graphical Analysis of Ventricular Pumping :

(**Figure 2.31**) shows a diagram that is especially useful in explaining the pumping mechanics of the *left* ventricle. The most important components of the diagram are the two curves labeled “diastolic pressure” and “systolic pressure.” These curves are volume-pressure curves.

The diastolic pressure curve is determined by filling the heart with progressively greater volumes of blood and then measuring the diastolic pressure immediately before ventricular contraction occurs, which is the *end diastolic pressure* of the ventricle.

The systolic pressure curve is determined by recording the systolic pressure achieved during ventricular contraction at each volume of filling. Until the volume of the non-contracting ventricle rises above about 150 milliliters, the “diastolic” pressure does not increase greatly. Therefore, up to this volume, blood can flow easily into the ventricle from the atrium.

Above 150 milliliters, the ventricular diastolic pressure increases rapidly, partly because of fibrous tissue in the heart that will stretch no more and partly because the pericardium that surrounds the heart becomes filled nearly to its limit.

During ventricular contraction, the “systolic” pressure increases even at low ventricular volumes and reaches a maximum at a ventricular volume of 150 to 170 milliliters.

Then, as the volume increases still further, the systolic pressure actually decreases under some conditions, as demonstrated by the falling systolic pressure curve in (Figure 2.27), because at these great volumes, the actin and myosin filaments of the cardiac muscle fibers are pulled apart far enough that the strength of each cardiac fiber contraction becomes less than optimal .

Note especially in the figure that the maximum systolic pressure for the normal *left* ventricle is between 250 and 300 mm Hg, but this varies widely with each person’s heart strength and degree of heart stimulation by cardiac nerves.

For the normal *right* ventricle ,the maximum systolic pressure is between 60 and 80 mm Hg(Guyton & Hall , 2006) .

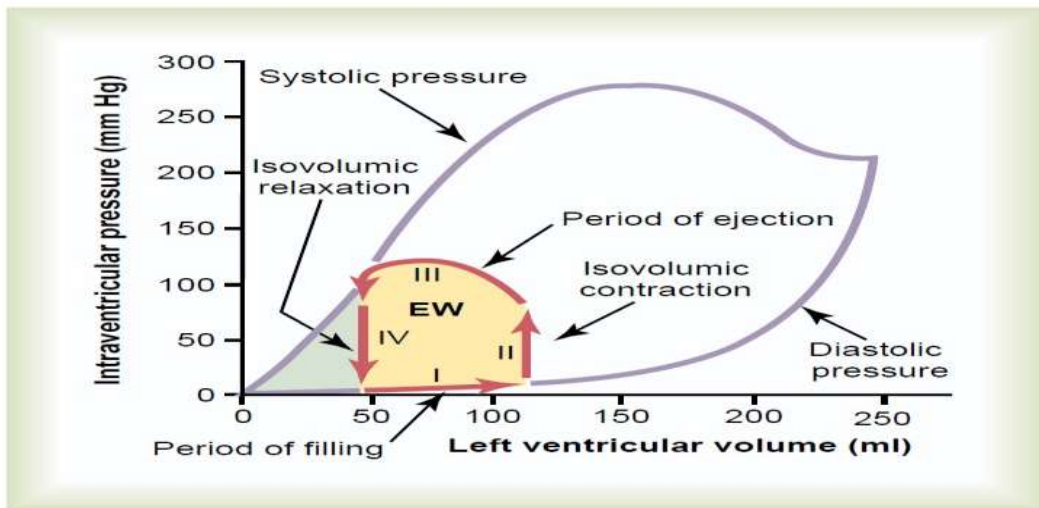


Figure 2.31 Relationship between left ventricular volume and intra ventricular pressure during diastole and systole. Also shown by the heavy red lines is the “volume-pressure diagram,” demonstrating changes in intra ventricular volume and pressure during the normal cardiac cycle. EW, net external work. (Guyton, & Hall , 2006).

2.2.5.1.2 “Volume-Pressure Diagram” During the Cardiac Cycle; Cardiac Work Output :

The red lines in **(Figure 2.24)** form a loop called the *volume-pressure diagram* of the cardiac cycle for normal function of the *left* ventricle. It is divided into four phases.

2.2.5.1.2.1 Phase I. Period of filling:

This phase in the volume pressure diagram begins at a ventricular volume of about 45 milliliters and a diastolic pressure near 0 mm Hg. Forty-five milliliters is the amount of blood that remains in the ventricle after the previous heartbeat and is called the *end-systolic volume*.

As venous blood flows into the ventricle from the left atrium, the ventricular volume normally increases to about 115 milliliters, called the *end-diastolic volume*, an increase of 70 milliliters.

Therefore, the volume-pressure diagram during phase I extends along the line labeled “with the volume increasing to 115 milliliters and the diastolic pressure rising to about 5 mm Hg”.

2.2.5.1.2.2 Phase II. *Period of isovolumic contraction:*

During isovolumic contraction, the volume of the ventricle does not change because all valves are closed. However, the pressure inside the ventricle increases to equal the pressure in the aorta, at a pressure value of about 80 mm Hg, as depicted by the arrow end of the line labeled “II”.

2.2.5.1.2.3 Phase III. *Period of ejection:*

During ejection, the systolic pressure rises even higher because of still more contraction of the ventricle. At the same time, the volume of the ventricle decreases because the aortic valve has now opened and blood flows out of the ventricle into the aorta.

Therefore, the curve labeled “III” traces the changes in volume and systolic pressure during this period of ejection.

2.2.5.1.2.4 Phase IV. *Period of isovolumic relaxation:*

At the end of the period of ejection, the aortic valve closes, and the ventricular pressure falls back to the diastolic pressure level. The line labeled “IV” traces this decrease in intra ventricular pressure without any change in volume.

Thus, the ventricle returns to its starting point, with about 45 milliliters of blood left in the ventricle and at an atrial pressure near 0 mmHg.

Readers well trained in the basic principles of physics should recognize that the area subtended by this functional volume-pressure diagram (the tan shaded area, labeled EW) represents the *net external work output* of the ventricle during its contraction cycle. In experimental studies of cardiac contraction, this diagram is used for calculating cardiac work output.

When the heart pumps large quantities of blood, the area of the work diagram becomes much larger. That is it extends far to the right because the ventricle fills with more blood during diastole, it rises much higher because the ventricle contracts with greater pressure, and it usually extends farther to the left because the ventricle contracts to a smaller volume—especially if the ventricle is stimulated to increased activity by the sympathetic nervous system (Guyton & Hall, 2006).

2.2.5.1.3 Concepts of Preload and Afterload:

In assessing the contractile properties of muscle, it is important to specify the degree of tension on the muscle when it begins to contract, which is called the *preload*, and to specify the load against which the muscle exerts its contractile force, which is called the *afterload*.

For cardiac contraction, the *preload* is usually considered to be the end-diastolic pressure when the ventricle has become filled.

The *afterload* of the ventricle is the pressure in the artery leading from the ventricle. In (**Figure 2.21**), this corresponds to the systolic pressure described by the phase III curve of the volume-pressure diagram. (Sometimes the afterload is loosely considered to be the resistance in the circulation rather than the pressure.)

The importance of the concepts of preload and after load is that in many abnormal functional states of the heart or circulation, the pressure during filling of the ventricle (the preload), the arterial pressure against which the ventricle must contract (the afterload), or both are severely altered from normal (*Guyton & Hall, 2006*).

2.2.5.1.4 Chemical Energy Required for Cardiac Contraction :

2.2.5.1.4 .1 Oxygen Utilization by the Heart :

Heart muscle, like skeletal muscle, uses chemical energy to provide the work of contraction. This energy is derived mainly from oxidative metabolism of fatty acids and, to a lesser extent, of other nutrients, especially lactate and glucose.

Therefore, the rate of oxygen consumption by the heart is an excellent measure of the chemical energy liberated while the heart performs its work (*Guyton & Hall, 2006*).

2.2.5.1.4.2 Efficiency of Cardiac Contraction :

During heart muscle contraction , most of the expended chemical energy is converted into *heat* and a much smaller portion into *work output*. The ratio of work output to total chemical energy expenditure is called the *efficiency of cardiac contraction*, or simply *efficiency of the heart*. Maximum efficiency of the normal heart is between 20 and 25 percent. In heart failure, this can decrease to as low as 5 to 10 per cent (*Guyton & Hall, 2006*).

2.2.5.1.5 Regulation of Heart Pumping:

When a person is at rest, the heart pumps only 4 to 6 liters of blood each minute. During severe exercise ,the heart may be required to pump four to seven

times this amount. **The basic means by which the volume pumped by the heart is regulated are :**

(1)intrinsic cardiac regulation of pumping in response to changes in volume of blood flowing into the heart and

(2)control of heart rate and strength of heart pumping by the autonomic nervous system(*Guyton & Hall ,2006*).

2.2.5.1.5.1 Intrinsic Regulation of Heart Pumping—The Frank-Starling

Mechanism :

The amount of blood pumped by the heart each minute is determined almost entirely by the rate of blood flow into the heart from the veins, which is called *venous return*. That is, each peripheral tissue of the body controls its own local blood flow, and all the local tissue flows combine and return by way of the veins to the right atrium.

The heart, in turn, automatically pumps this incoming blood into the arteries, so that it can flow around the circuit again.

This intrinsic ability of the heart to adapt to increasing volumes of inflowing blood is called the *Frank-Starling mechanism of the heart*, in honor of Frank and Starling, two great physiologists of a century ago.

Basically , the Frank-Starling mechanism means that the greater the heart muscle is stretched during filling ,the greater is the force of contraction and the greater the quantity of blood pumped into the aorta. Or, stated another way: **Within physiologic limits, the heart pumps all the blood that returns to it by the way of the veins** (*Guyton &Hall ,2006*).

2.2.5.1.5.2 What Is the Explanation of the Frank-Starling Mechanism?

When an extra amount of blood flows into the ventricles ,the cardiac muscle itself is stretched to greater length.

This in turn causes the muscle to contract with increased force because the actin and myosin filaments are brought to a more nearly optimal degree of overlap for force generation.

Therefore, the ventricle, because of its increased pumping, automatically pumps the extra blood into the arteries. This ability of stretched muscle, up to an optimal length, to contract with increased work output is characteristic of all striated muscle and is not simply a characteristic of cardiac muscle.

In addition to the important effect of lengthening the heart muscle, still another factor increases heart pumping when its volume is increased.

Stretch of the right atrial wall directly increases the heart rate by 10 to 20 per cent; this, too, helps increase the amount of blood pumped each minute, although its contribution is much less than that of the Frank-Starling mechanism (Guyton & Hall, 2006).

2.2.5.1.5.3 Ventricular Function Curves :

One of the best ways to express the functional ability of the ventricles to pump blood is by *ventricular function curves*, as shown in (Figure 2.28) and (Figure 2.29) shows a type of ventricular function curve called the *stroke work output curve* (Guyton & Hall, 2006).

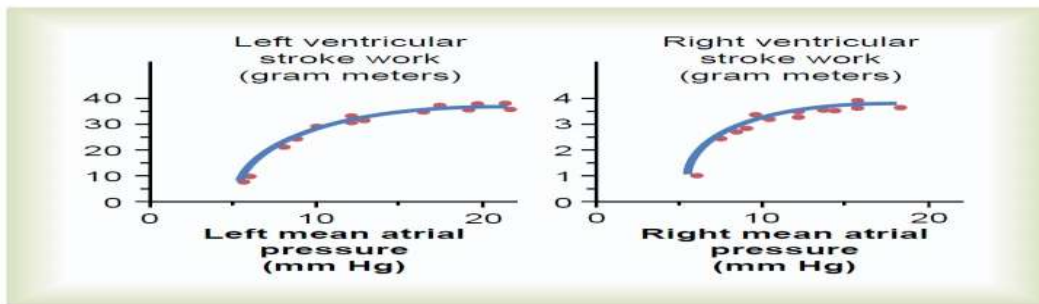


Figure 2.28 Left and right ventricular function curves recorded from dogs, Depicting *ventricular stroke work output* as a function of left and right mean atrial pressures. (Curves reconstructed from data in Sarnoff SJ: Myocardial contractility as described by ventricular function curves. *Physiology Rev* 35:107, 1955) (Guyton & Hall, 2006).

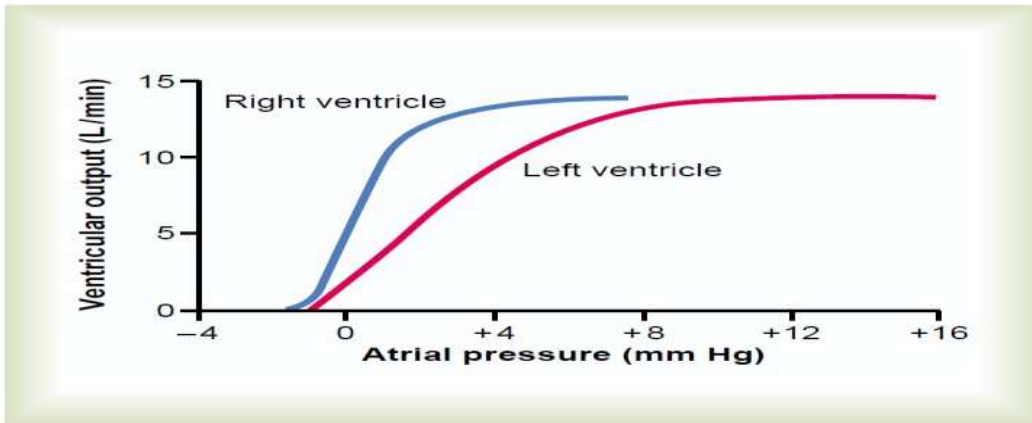


Figure 2.29 Approximate normal right and left *ventricular volume output* Curves for the normal resting human heart as extrapolated from data obtained in dogs and data from human beings (Guyton & Hall, 2006).

Note that as the atrial pressure for each side of the heart increases, the stroke work output for that side increases until it reaches the limit of the ventricle's pumping ability. (Figure 2.29) shows another type of ventricular function curve called the *ventricular volume output curve*.

The two curves of this figure represent function of the two ventricles of the human heart based on data extrapolated from lower animals. As the right and left atrial pressures increase, the respective ventricular volume outputs per minute also increase.

Thus, *ventricular function curves* are another way of expressing the Frank-Starling mechanism of the heart. That is, as the ventricles fill in response to higher atrial pressures, each ventricular volume and strength of cardiac muscle contraction increase, causing the heart to pump increased quantities of blood into the arteries (Guyton & Hall, 2006).

2.2.5.1.6 Control of the Heart by the Sympathetic and Parasympathetic Nerves :

The pumping effectiveness of the heart also is controlled by the *sympathetic* and *parasympathetic (vagus)* nerves, which abundantly supply the heart, as shown

in (Figure 2.30) . For given levels of input atrial pressure, the amount of blood pumped each minute (*cardiac output*) often can be increased more than 100per cent by sympathetic stimulation.

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

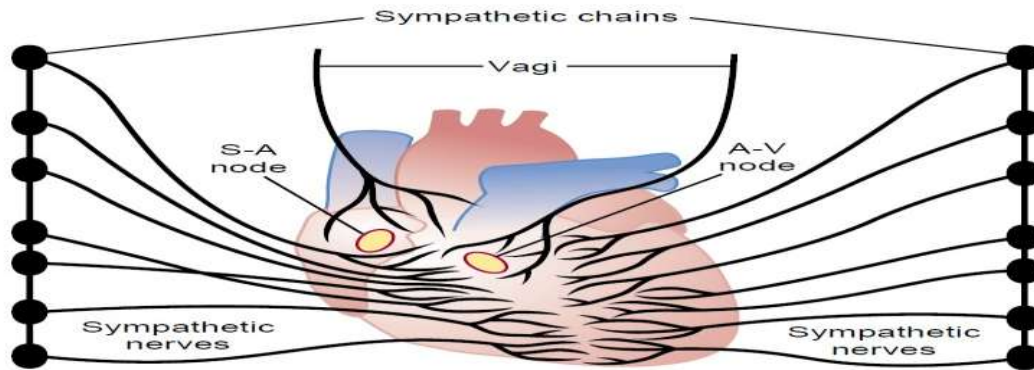


Figure 2.30 Cardiac sympathetic and parasympathetic nerves. (The vagusnerves to the heart are parasympathetic nerves) (Guyton &Hall ,2006).

2.2.5.1.6.1 Mechanisms of Excitation of the Heart by the Sympathetic Nerves :

Strong sympathetic stimulation can increase the heart rate in young adult humans from the normal rate of 70 beats per minute up to 180 to 200 and, rarely ,even 250 beats per minute.

Also, sympathetic stimulation increases the force of heart contraction to as much as double normal, thereby increasing the volume of blood pumped and increasing the ejection pressure.

Thus, sympathetic stimulation often can increase the maximum cardiac output as much as twofold to three fold ,in addition to the increased output caused by the Frank-Starling mechanism already discussed.

Conversely, *inhibition* of the sympathetic nerves to the heart can decrease cardiac pumping to a moderate extent in the following way:

Under normal conditions, the sympathetic nerve fibers to the heart discharge continuously at a slow rate that maintains pumping at about 30 per cent above that with no sympathetic stimulation.

Therefore, when the activity of the sympathetic nervous system is depressed below normal, this decreases both heart rate and strength of ventricular muscle contraction, thereby decreasing the level of cardiac pumping as much as 30 per cent below normal (*Guyton & Hall, 2006*).

2.2.5.1.6.2 Parasympathetic (Vagal) Stimulation of the Heart:

Strong stimulation of the parasympathetic nerve fibers in the vagus nerves to the heart can stop the heartbeat for a few seconds, but then the heart usually “escapes” and beats at a rate of 20 to 40 beats per minute as long as the parasympathetic stimulation continues.

In addition, strong vagal stimulation can decrease the strength of heart muscle contraction by 20 to 30 per cent.

The vagal fibers are distributed mainly to the atria and not much to the ventricles, where the power contraction of the heart occurs. This explains the effect of vagal stimulation mainly to decrease heart rate rather than to decrease greatly the strength of heart contraction.

Nevertheless, the great decrease in heart rate combined with a slight decrease in heart contraction strength can decrease ventricular pumping 50 per cent or more (*Guyton & Hall, 2006*).

2.2.5.1.6.3 Effect of Sympathetic or Parasympathetic Stimulation on the Cardiac Function Curve.

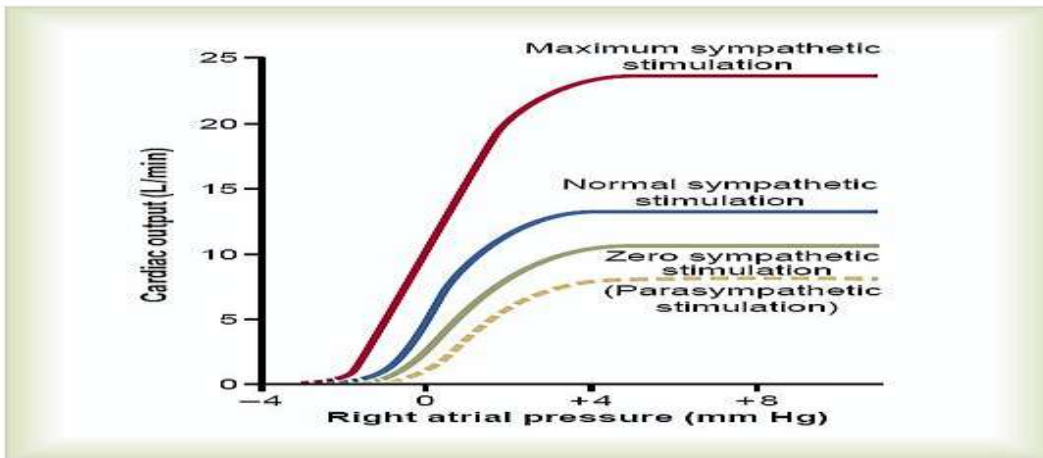


Figure 2.31 Effect on the cardiac output curve of different degrees of sympathetic or parasympathetic stimulation (Guyton & Hall, 2006).

(Figure 2.31) shows four cardiac function curves. They are similar to the ventricular function curves of (Figure 2.29)

However, they represent function of the entire heart rather than of a single ventricle ; they show the relation between right atrial pressure at the input of the right heart and cardiac output from the left ventricle into the aorta.

The curves of (Figure 2.31) demonstrate that at any given right atrial pressure, the cardiac output increases during increased sympathetic stimulation and decreases during increased parasympathetic stimulation.

These changes in output caused by nerve stimulation result both from changes in heart rate and from changes in contractile strength of the heart because both change in response to the nerve stimulation (Guyton & Hall, 2006).

2.2.5.1.7 Effect of electrolytes and temperature on Heart function:

- Effect of Potassium Ions.
- Effect of Calcium Ions.
- Effect of Sodium Ions.
- Effect of Temperature .

(Guyton & Hall, 2006).

2.2.5.1.8 Increasing the Arterial Pressure Load(up to a Limit) Does Not Decrease the Cardiac Output:

Note in (Fig. 2.2.5.1.8.1) that increasing the arterial pressure in the aorta does not decrease the cardiac output until the mean arterial pressure rises above about 160 mm Hg.

In other words, during normal function of the heart at normal systolic arterial pressures (80 to 140 mm Hg), the cardiac output is determined almost entirely by the ease of blood flow through the body tissues, which in turn controls *venous return* of blood to the heart *From Textbook of Medical Physiology (Arthur C. Guyton, M.D. & John E. Hall, Ph.D., 2006)*

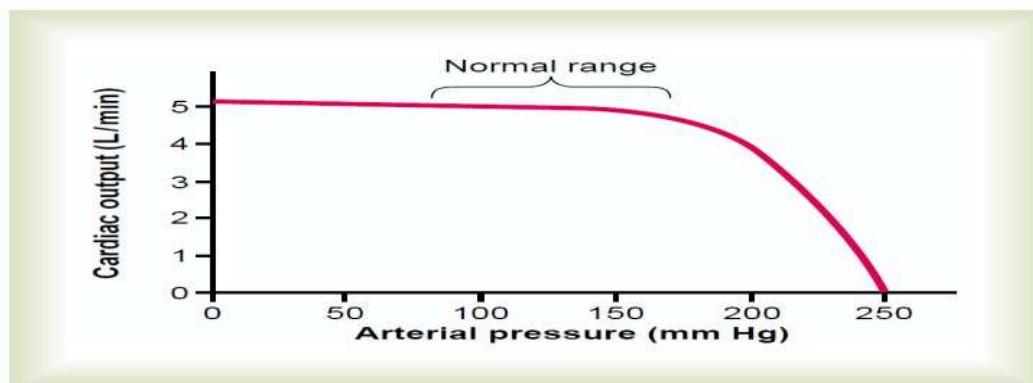


Figure 2.32 shows direct relationship between CO and arterial blood pressure (Guyton & Hall, 2006).

2.2.5.2. Total Peripheral Resistance and Arterial Diastolic Pressure:

As previously discussed, if the heart rate and stroke volume remain constant, an increase in TPR will increase mean arterial pressure. When arterial compliance is constant, an increase in TPR leads to proportional increases in systolic and diastolic pressure such that the pulse pressure is unchanged (Figure 33, A).

However, arterial compliance is not linear. As mean arterial pressure increases and the arterial is stressed, compliance decreases (Figure 2.33, B).

Because of the decrease in arterial compliance with increase when arterial pressure is elevated (*Koeppen and Stanton ,2010*).

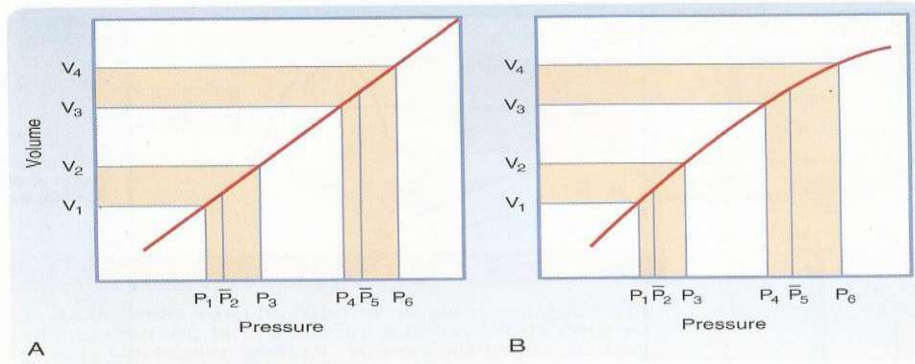


Figure 2.33,A and B Comparison of the effects of a given change in peripheral resistance on pulse pressure when the pressure-volume curve for the arterial system is either rectilinear (A) or curvilinear (B). The increment in arterial volume is the same for both conditions. $[(V_4-V_3)=(V_2-V_1)]$ (*Koeppen and Stanton,phD,2010*).

2.2.5.3.Effective Arterial Blood Volume:

Effective arterial blood volume (EABV) refers to the adequacy of the arterial blood volume to "fill" the capacity of the arterial vasculature. Normal EABV exists when the ratio of cardiac output to peripheral resistance maintains venous return and cardiac output at normal levels.

EABV can be reduced, therefore, by factors which reduce actual arterial blood volume (hemorrhage, dehydration), increase arterial vascular capacitance (cirrhosis, sepsis) or reduce cardiac output (congestive heart failure). EABV can be reduced in the setting of low, normal, or high actual blood volume. Whenever EABV falls, the kidney is triggered to retain sodium and water as (**figure 2.34**).
<http://edemainformation.blogspot.ca/2005/11/edema-pathophysiology-and-treatment.htm>,2015

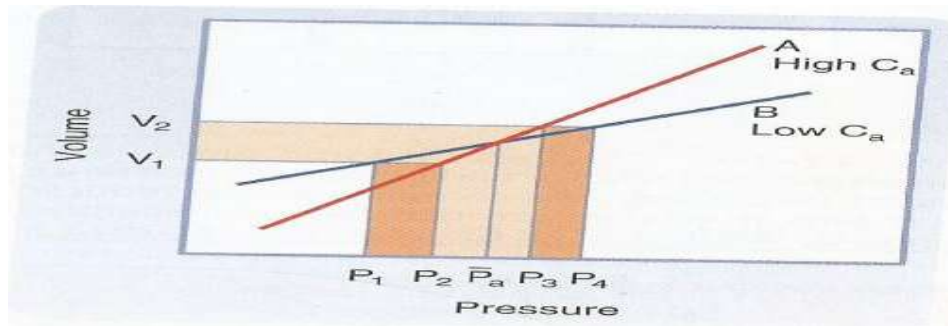


Figure 2.34 For a given volume increment (V_2-V_1), reduced arterial compliance (compliance B, compliance A) results in increased pulse pressure $[(P_4-P_1)/(P_3-P_2)]$ (Koeppen and Stanton, 2010).

2.2.5.4 Arterial Compliance:

Arterial compliance also affects pulse pressure. This relationship is illustrated in (Figure 2.34). When cardiac output and TPR are constant, a decrease in arterial compliance results in an increase in pulse pressure.

Diminished arterial compliance also imposes a greater workload on the left ventricle (i.e., increased after load), even if stroke volume, TPR, and mean arterial pressure are equal in the two individuals.

Total Peripheral Resistance and Arterial Diastolic Pressure: As previously discussed, if the heart rate and stroke volume remain constant, an increase in TPR will increase mean arterial pressure.

When arterial compliance is constant, an increase in TPR leads to proportional increases in systolic and diastolic pressure such that the pulse pressure is unchanged (Figure 2.35, A).

However, arterial compliance is not linear. As mean arterial pressure increases and the arterial is stressed, compliance decreases (Figure 2.35, B). Because of the decrease in arterial compliance with increase when arterial pressure is elevated (Koeppen and Stanton, 2010).

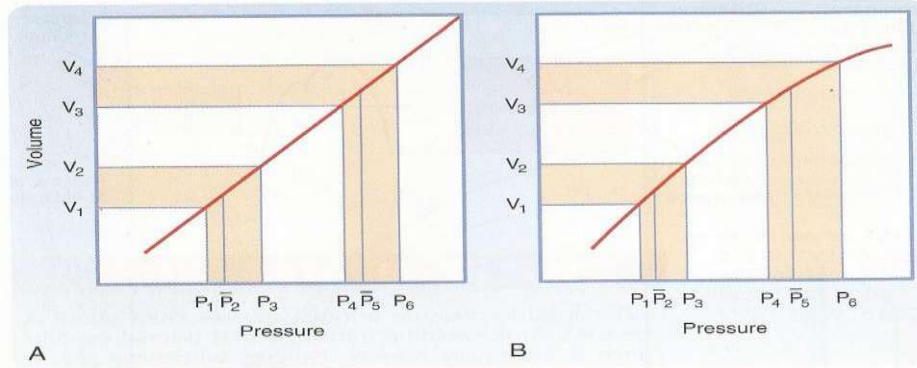
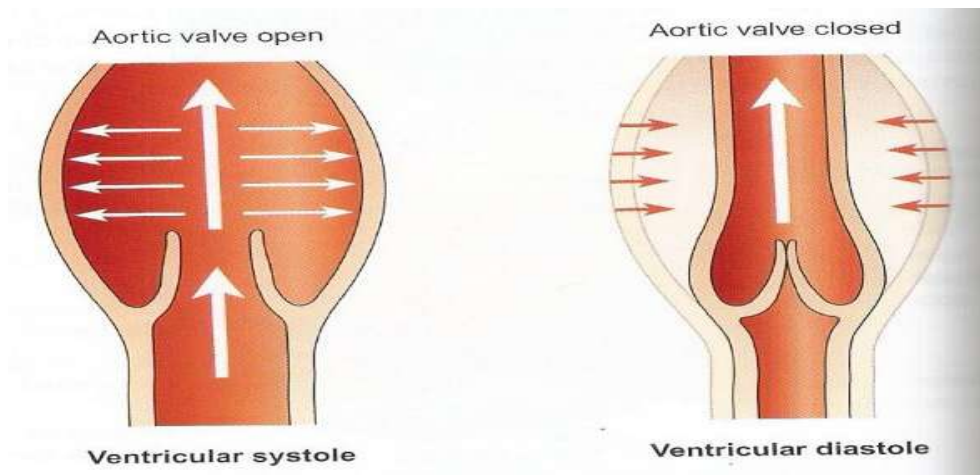


Figure 2.35, A and B Comparison of the effects of a given change in peripheral resistance on pulse pressure when the pressure-volume curve for the arterial system is either rectilinear (A) or curvilinear (B). The increment in arterial volume is the same for both conditions. $[(V_4 - V_3) = (V_2 - V_1)]$ (Koeppen and Stanton, 2010).

There is a considerable amount of elastic tissue in the arterial walls, especially in large arteries.

Therefore, when the left ventricle ejects blood into the already full aorta, the aorta expands to accommodate it, and then recoils because of the elastic tissue in the wall. This pushes the blood forwards, into the systemic circulation.

This distension and recoil occurs throughout the arterial system. During cardiac diastole the elastic recoil of the arteries maintains the diastolic pressure. (figure 2.36) (Waugh and Grant, 2010).



(Fig 2.36) The elasticity of the wall of the aorta (Waugh and Grant, 2010).

2.3. Pathology

2.3.1 Introduction :

Affecting 70 million Americans and 1 billion people worldwide, hypertension remains the most common, readily identifiable, and reversible risk factor for myocardial infarction, stroke, heart failure, atrial fibrillation, aortic dissection, and peripheral arterial disease. Because of escalating obesity and population aging, the global burden of hypertension is rising and projected to affect 1.5 billion persons—one third of the world's population—by the year 2025. Currently, high blood pressure (BP) causes about 54% of stroke and 47% of ischemic heart disease worldwide.¹ Half of this disease burden is in people with hypertension; the other half is in people with lesser degrees of high BP (hypertension). Thus, high BP remains the leading cause of death worldwide and one of the world's great public health problems (*Bonow et al 2012*).

2.3.2 Hypertension:

Although hypertension is a common health problem with sometimes devastating consequences, it often remains asymptomatic until late in its course. It is one of the most important risk factors in both coronary heart disease and cerebrovascular accidents; in addition, hypertension may also lead to cardiac hypertrophy and heart failure (hypertensive heart disease), aortic dissection, and renal failure.

A sustained diastolic pressure greater than 90 mm Hg, or a sustained systolic pressure in excess of 140 mm Hg, is considered to constitute hypertension. Reduction of blood pressure dramatically reduces the incidence and death rates from IHD, heart failure, and stroke.

Ninety percent to 95% of hypertension is idiopathic (essential hypertension), which is compatible with long life, unless a myocardial infarction, cerebrovascular

accident, or other complication supervenes. Most of the remainder of "benign hypertension" is secondary to renal disease or, less often, to narrowing of the renal artery, usually by an atheromatous plaque (renovascular hypertension). Infrequently, hypertension is secondary to diseases of the adrenal glands, such as primary aldosteronism, Cushing syndrome, pheochromocytoma, or other disorders. Primary or essential hypertension is more common in adolescents and has multiple risk factors, including obesity and a family history of hypertension.

About 5% of hypertensive persons show a rapidly rising blood pressure that if untreated leads to death within 1 or 2 years. Termed accelerated or malignant hypertension, the clinical syndrome is characterized by severe hypertension (diastolic pressure over 120 mm Hg), renal failure, and retinal hemorrhages and exudates, with or without papilledema. It may develop in previously normotensive persons but more often is superimposed on preexisting benign hypertension, either essential or secondary (See table 2.3.2.1) (*Burn and Kumer,2007*).

Table 2.1 Classification of HTN :

Classification	Systolic BP (mmHg)	Diastolic BP (mmHg)
Normal	<120	<i>And</i> <80
Prehypertension	120-139	<i>Or</i> 80-89
Stage 1 hypertension	140-159	<i>Or</i> 90-99
Stage 2 hypertension	≥160	<i>Or</i> ≥100

Based on Seventh Report of the Joint National Committee (JNC 7), patients with sustained hypertension are further divided into stage 1 hypertension (systolic BP 140-159 or diastolic BP 90-99 mmHg), stage 2 hypertension (systolic BP ≥160 or diastolic BP ≥100 mmHg), and those with compelling indications that include diabetes, cardiovascular disease, and renal disease. The JNC 7 recommended a blood pressure goal of <140/90 mmHg for patients with hypertension and more

intense lowering (a BP target of <130/80 mmHg) in hypertensive patients with diabetes or kidney disease. In recent years however, large clinical trials performed in patients with kidney disease and diabetes have failed to demonstrate clear benefit with intense blood pressure control www.nhlbi.nih.gov/guidelines/hypertension/ (accessed April 25, 2013).

2.3.2.1 Pathogenesis of Hypertension :

Hemodynamic Subtypes: Primary hypertension can be divided into three distinctly different hemodynamic subtypes that vary sharply by age (*Bonow et al 2012*).

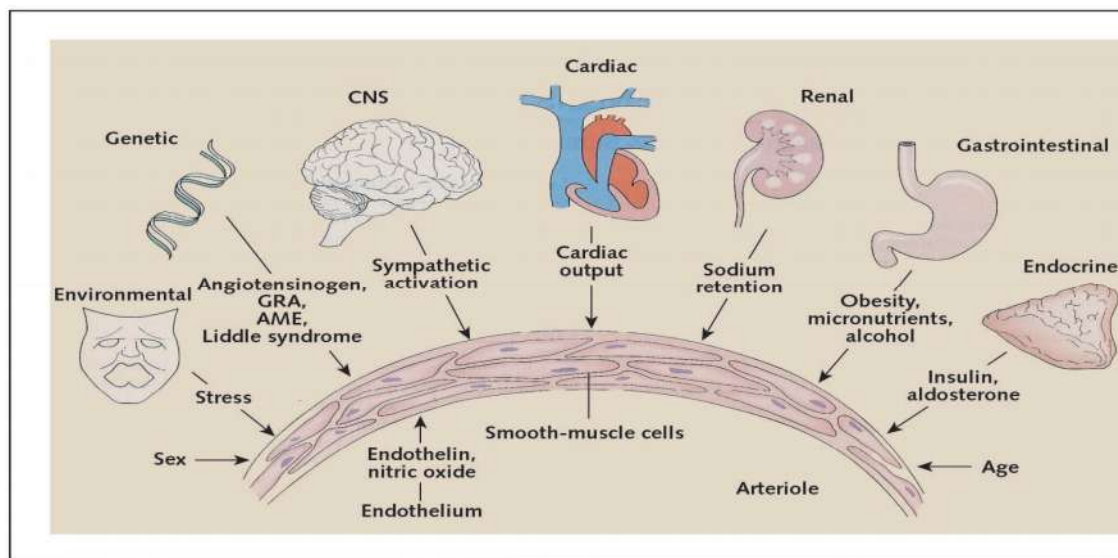


Figure 2.37 Pathophysiologic mechanisms of hypertension From (*Suzanne et al , 2003*).

2.3.2.1.1 Systolic Hypertension :

At one end of the age spectrum is isolated systolic hypertension (ISH) in young adults (typically 17 to 25 years of age). The key hemodynamic abnormalities are increased cardiac output and a stiff aorta, both presumably reflecting an overactive sympathetic nervous system (*Bonow et al 2012*).

2.3.2.1.2 Diastolic Hypertension In Middle age

When hypertension is diagnosed in middle age (typically 30-50 years of age), the most common BP pattern is elevated diastolic pressure, with systolic pressure

being normal (isolate diastolic hypertension) or elevated (combined systolic-diastolic hypertension) (*Bonow et al 2012*).

The fundamental hemodynamic fault is an elevated systemic vascular resistance coupled with an inappropriately normal cardiac output. Vasoconstriction at the level of the resistance arterioles results from increased neurohormonal drive and an autoregulatory reaction of vascular smooth muscle to an expanded plasma volume, the latter because of impairment in the kidneys ability to excrete sodium(*Bonow et al 2012*).

2.3.2.1.3 Isolated Systolic Hypertension In Older Adults :

After the age 55 years, ISH (systolic BP >140 mm Hg and diastolic BP <90 mm Hg) is the most common form. In developed countries, systolic pressure rises steadily with age; in contrast, diastolic pressure rises until about 55 years of age, then falls progressively thereafter. The resultant widening of pulse pressure indicates stiffening of the central aorta and a more rapid return of reflected pulse waves from the periphery, causing an augmentation of systolic aortic pressure. A multitude of neurohormonal, renal, and vascular mechanisms interact to varying degrees in contributing to these different hemodynamic forms of hypertension (*Bonow et al 2012*).

2.3.2.2 Neural Mechanisms :

In young adults, primary hypertension consistently is associated with increased heart rate and cardiac output, plasma and urinary norepinephrine levels, regional norepinephrine spillover, peripheral postganglionic sympathetic nerve firing, and alpha-adrenergic receptor-mediated vasoconstrictor tone in the peripheral circulation (*Bonow et al 2012*).

Sympathetic over activity has been demonstrated in early primary hypertension and in several other forms of **established**, human hypertension,

including hypertension associated with obesity, sleep apnea, early type 2 diabetes mellitus and pre diabetes, chronic kidney disease (CKD) , heart failure, and immunosuppressive therapy with calcineurin inhibitors such as cyclosporine (*Robert O. BONOW et al 2012*).

In these conditions, centric sympathetic outflow can be driven by deactivation of inhibitory neural inputs (e.g., baroreceptors), activations of excitatory neural inputs (e.g., carotid body chemoreceptor's, renal afferents), or circulating angiotensin II (A II), which activates pools of excitatory brain-stem neurons without a blood-brain barrier (*Bonow et al 2012*).

2.3.2.2.1 Baroreceptors And Hypertension :

In hypertension, the baroreceptors reset to defend a higher level of BP. Complete baroreflex failure rarely causes labile hypertension, most often seen in throat cancer survivors as a late complication of radiation therapy, which causes a gradual destruction of the baroreceptor nerves. In contrast, partial baroreceptor dysfunction is common in elderly hypertensive and typically is manifested with a triad of orthostatic hypotension, supine hypertension, and symptomatic postprandial hypotension, the last initiated by splanchnic pooling after carbohydrate-rich meals (*Bonow et al 2012*).

2.3.2.3 Vascular Mechanism:

Alterations in the structure and function of small and large arteries play a pivotal role in the pathogenesis and progression of hypertension (*Bonow et al 2012*).

2.3.2.3.1 Endothelial cell Dysfunction:

The endothelial lining of blood vessels is critical to vascular health and constitutes a major defense against hypertension. Dysfunctional endothelium is characterized by impaired release of endothelium-derived relaxing factors (e.g., nitric oxide, endothelium-derived hyperpolarizing factor) and enhanced release of

endothelium-derived constricting, pro inflammatory, pro thrombotic, and growth factors (*Bonow et al 2012*).

The endothelium of all blood vessels expresses the enzyme nitric oxide synthase, which can be activated by bradykinin, acetylcholine, or cyclic laminar shear stress. Nitric oxide synthase generates nitric oxide that diffuses to the adjacent vascular smooth muscle and activates a series of G kinases that culminate in vasodilatation (*Bonow et al 2012*).

In humans, endothelium-dependent vasodilatation can be assessed by measuring increases in the large artery diameter after intra-arterial infusion of acetylcholine or release of ischemia. Mounting evidence indicates that smoldering vascular inflammation plays a central role in the genesis and complications of high BP. C-reactive protein (CPR) , an easily measured serum biomarker, reports on inflammation(*Bonow et al 2012*).

Cross-sectional studies show strong correlations between elevated CRP and arterial stiffness and elevated pulse pressure. Longitudinal studies implicate elevated CRP levels as a risk marker (or risk factor) for new onset of hypertension and accelerated progression of hypertensive target organ disease (*Bonow et al 2012*).

Oxidative stress also contributes to endothelial cell vasodilator dysfunction in hypertension. Generation of reactive oxygen species by xanthine oxidase accounts for the association of hyperuricemia with endothelial dysfunction and hypertension (*Bonow et al 2012*).

2.3.2.3.2Vascular Remolding

Over time, endothelial cell dysfunction. neurohormonal activation , and elevated BP cause remodeling of blood vessels, which further perpetuates hypertension .An increase in the medial thickness relative to lumen diameter (increased media-to-lumen ratio) is the hallmark of hypertensive remodeling in

small and large arteries. Vasoconstriction initiates small artery remodeling, which normalize wall stress (*Bonow et al 2012*).

2.3.3 Hypertensive Heart Disease :

Chronic hypertension is a common disorder associated with considerable morbidity. Inadequately controlled hypertension has serious effects on many organs, including the heart, brain, and kidneys. The diagnosis of hypertensive heart disease is based on the presence of **left ventricular hypertrophy** in an individual with a history of hypertension and in whom other causes of ventricular hypertrophy have been excluded(*Bonow et al 2012*).

2.3.3.1 Left ventricular hypertrophy :

The stimulus to ventricular hypertrophy in patients with hypertension is a sustained pressure load on the left ventricular myocardium. The cellular events leading to myocardial hypertrophy are incompletely understood but appear to involve both local mechanical effects and growth factors, which lead in turn to changes in the genes controlling the expression of myosin, actin, and other cellular constituents (*Bonow et al 2012*).

The metabolic requirements of hypertrophic myocardium, understandably, are greater than those of normal myocardium. With increasing degrees of hypertrophy, the metabolic requirements continue to increase but the ability of the heart to meet these demands decreases. This occurs because hypertrophy renders the myocardium stiff, thus increasing wall tension while simultaneously decreasing diastolic filling and stroke volume (*Bonow et al 2012*).

Capillary density in the hypertrophic myocardium does not increase sufficiently to meet the metabolic demands of the myocytes. The distance over which oxygen and other nutrients delivered by the capillaries must diffuse is increased. To make matters worse, chronic hypertension also predisposes to atherosclerosis. In concert, these various changes predispose the hypertrophic

myocardium to ischemic injury, eventually resulting in the development of congestive heart failure (*Bonow et al 2012*).

2.3.3.1.1 Morphology :

The essential feature of hypertensive heart disease is left ventricular hypertrophy. The weight of the heart usually exceeds 450 g. The hypertrophy typically involves the ventricular wall in a symmetric, circumferential pattern termed concentric hypertrophy with free wall thicknesses exceeding 2.0 cm. On occasion, particularly in long-standing cases, hypertrophy may be more pronounced in the septal area, mimicking the appearance of hypertrophic cardiomyopathy (*Burn and Kumer,2007*).

The size of the chamber is normal in the early stages of hypertensive heart disease, but in long-standing cases some degree of dilation is common. As left ventricular failure progresses, right ventricular hypertrophy and dilation may also develop (*Burn and Kumer,2007*).

Microscopically, the cardiac myocytes are enlarged and contain large, hyperchromatic, rectangular "boxcar"-shaped nuclei. Superimposed ischemic changes, including interstitial fibrosis and recent or remote infarcts, are common (*Burn and Kumer,2007*).

2.3.3.1.2.Clinical features :

In its early stages, while the cardiac output is maintained at normal levels, hypertensive heart disease may cause no symptoms. In these patients, the diagnosis is usually based on the detection of left ventricular enlargement in chest radiographs or echocardiograms or by electrocardiographic evidence of left ventricular hypertrophy (*Burn and Kumer,2007*).

As the left ventricle begins to fail, the clinical manifestations of heart failure appear. Heart failure in the setting of hypertension is associated with a poor prognosis. Signs and symptoms of myocardial ischemia, such as angina pectoris, often punctuate the course of hypertensive cardiac disease. In addition, progressive

renal damage or cerebrovascular accidents may occur and contribute to both morbidity and mortality. The risk of sudden cardiac death is also increased. There is substantial evidence that effective control of hypertension can prevent or lead to regression of hypertrophy and its associated risks (*Burn and Kumer,2007*).

2.3.3.2. Cardiomyopathy:

Although myocardial dysfunction can occur secondary to ischemic, valvular, hypertensive or other heart diseases, the term cardiomyopathy implies a principal cardiac dysfunction (*Richard Mitchell et al ,2012*) .

Causes of such myocardial disease can be primary (i.e., predominantly affecting heart) or secondary (i.e., part of a larger systemic disorder):

- Infections (e.g., viral, bacterial, fungal, protozoal).
- Toxic exposure (e.g., alcohol, cobalt, chemotherapeutic agents).
- Metabolic disorders (e.g., hyperthyroidism, nutritional deficiency).
- Genetic abnormalities in cardiomyocytes (e.g., storage disorders, muscular dystrophies).
- Infiltrative lesions (e.g., sarcoid, carcinoma, radiation –induced fibrosis).

Immunologic disorders (e.g., autoimmune myocarditis, rejection) (*Richard Mitchell et al ,2012*).

Cardiomyopathy is divided into three main functional and pathologic patterns:

2.3.3.2.1.Dilated Cardiomyopathy

Dilated cardiomyopathy (DCM) is characterized by gradual four chamber hypertrophy and dilation; there is systolic dysfunction with hypo contraction . It typically presents as indolent, progressive CHF(*Richard Mitchell et al ,2012*).

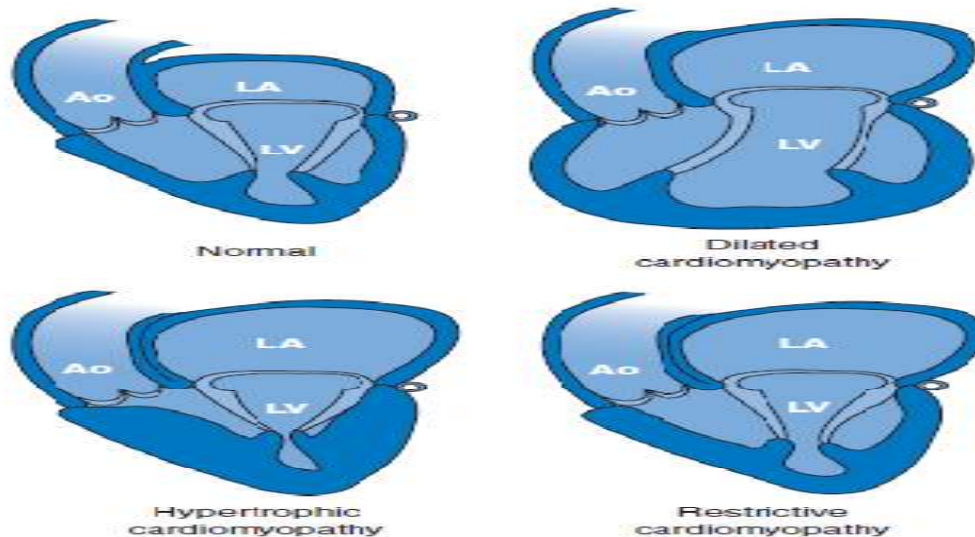


Figure 2.38 Schematics of the three forms of cardiomyopathy; each from can have a variety of causes. AO, Aorta; LA, Left Atrium; LV, Left Ventricle (*Richard Mitchell et al ,2012*).

Although the cause is frequently unknown (idiopathic DCM), certain pathologic mechanisms can contribute:

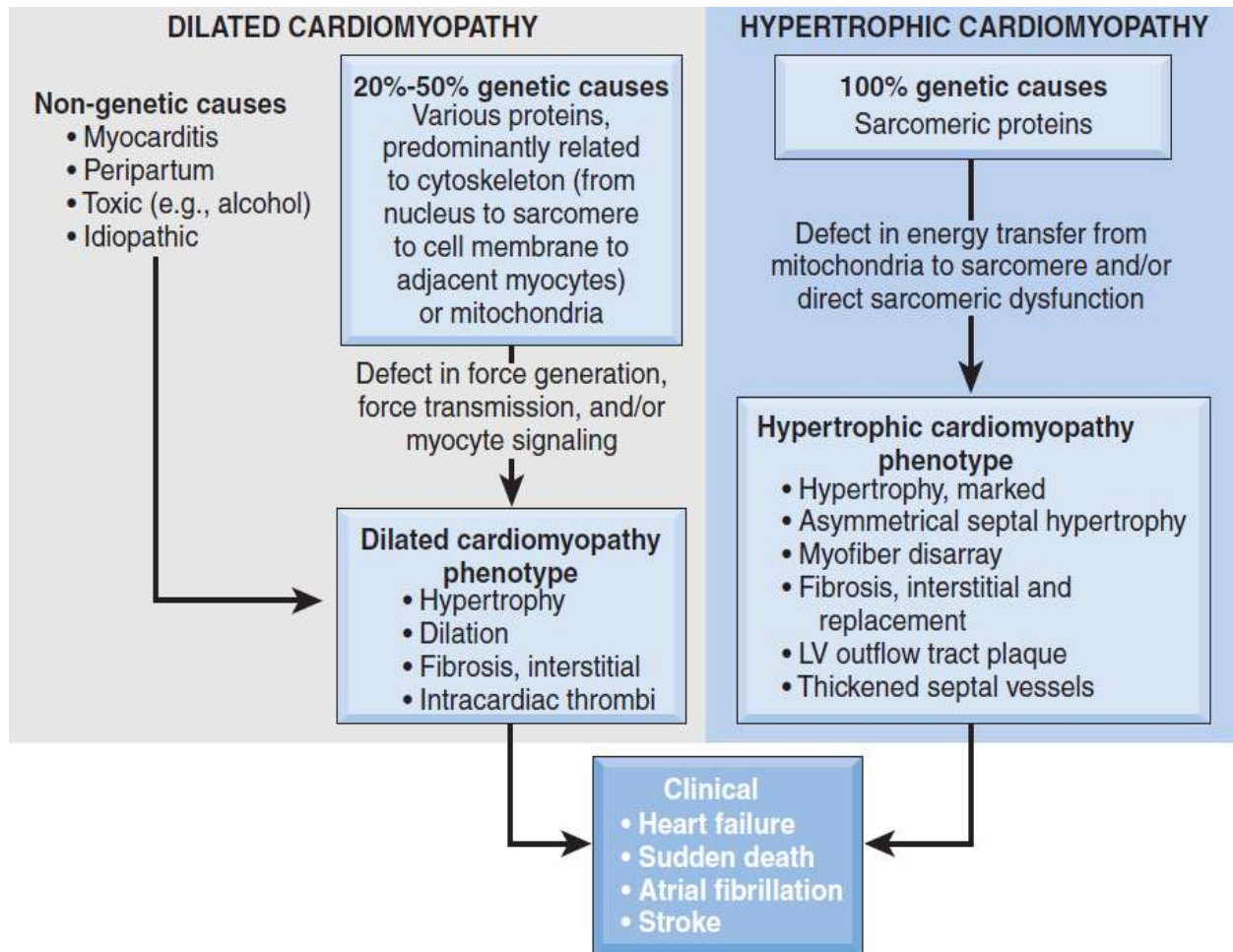
- Genetic abnormalities commonly involve cytoskeletal proteins. Others involve mutations of enzymes involved in fatty acid β -oxidation or mitochondrial gene deletions causing abnormal oxidative phosphorylation.
- DCM is attributed to direct toxicity of alcohol or a metabolite on the myocardium. No morphologic features distinguish alcohol-induced cardiac damage from other forms of idiopathic DCM or chronic thiamine deficiency.
- DCM is discovered within several months before or after delivery. Although the mechanism is uncertain, the association with pregnancy suggests possible etiologies of chronic hypertension, volume overload, nutritional deficiency, metabolic derangement, or immunologic response.

Myocarditis: even after resolution of the infection, injury related to myocarditis can progress to DCM(*Richard Mitchell et al ,2012*).

Table 2.2 Cardiomyopathy and Indirect Myocardial Dysfunction: Functional Pattern and causes (Richard Mitchell et al ,2012) .

Functional Pattern	Left Ventricular Ejection Fraction*	Mechanisms of Heart Failure	Causes	Indirect Myocardial Dysfunction (Not Cardiomyopathy)
Dilated	<40%	Impairment of contractility (systolic dysfunction)	Idiopathic; alcohol; peripartum; genetic; myocarditis; hemochromatosis; chronic anemia; doxorubicin (Adriamycin); sarcoidosis	Ischemic heart disease; valvular heart disease; hypertensive heart disease; congenital heart disease
Hypertrophic	50% to 80%	Impairment of compliance (diastolic dysfunction)	Genetic; Friedreich ataxia; storage diseases; infants of diabetic mothers	Hypertensive heart disease; aortic stenosis
Restrictive	45% to 90%	Impairment of compliance (diastolic dysfunction)	Idiopathic; amyloidosis; radiation-induced fibrosis	Pericardial constriction

Table 2.3 Causes and consequences of dilated cardiomyopathy. Some dilated cardiomyopathy and virtually forms of hypertrophic cardiomyopathy are genetic in origin. The genetic causes of dilated myopathy involve mutations predominantly in cytoskeletal proteins but also molecules in the Sarcomer, mitochondria, and morphologic phenotypes, they share a final common pathway of clinical complications . LV, Left Ventricle (Richard Mitchell et al ,2012).



2.3.3.2.1.1 Morphology:

Grossly, the heart is flabby with cardiomegaly; wall thickness may not reflect the degree of hypertrophy due to chamber dilation .Poor contractile function and stasis predisposing to mural thrombi. Valves and coronary arteries are generally normal. Microscopic changes in DCM are often subtle and entirely nonspecific; most commonly there is diffuse myocyte hypertrophy and variable interstitial fibrosis (Richard Mitchell et al ,2012).

2.3.3.2.1.2 Arrhythmogenic Right Ventricular cardiomyopathy:

It is recently recognized cardiomyopathy with distinct presentation and morphology. It is an autosomal dominant disorder characterized by predominantly right-sided failure and arrhythmia. The defect is most commonly caused by defective adhesive molecules in desmosomes .Morphologically; the right ventricular wall is severely thinned with myocyte loss and profound fatty infiltration. Death occurs secondary to progressive CHF or fatal arrhythmias (*Richard Mitchell et al ,2012*).

2.3.3.2.2 Hypertrophic cardiomyopathy:

Classically , there is disproportion thickening of the interventricular, The left ventricular cavity is compressed into a banana-like configuration by the asymmetric of the mitral valve compromises left ventricular systolic outflow by contact of the anterior mitral leaflet with the , this cause hypertrophic obstructive cardiomyopathy , reflected by a fibrous plaque on the septum (*Richard Mitchell et al ,2012*).

Microscopically, there is marked myofiber hypertrophy, classically with helter-skelter myocyte disarray, accompanied by myofilament disorganization within muscle cells, most prominent in the interventricular septum. There is also patch interstitial and replacement fibrosis (*Richard Mitchell et al ,2012*).

2.3.3. 2.2.1 Pathogenesis:

HCM is predominantly caused by mutation of sarcomeric proteins, most are autosomal dominant mutations with variable penetrance .Prognosis varies widely depending on the specific mutations. The pathogenic sequence leading from specific mutations to disease manifestations is not understood. Different mutations in the same gene can even give rise to DCM or HCM(*Richard Mitchell et al ,2012*).

2.3.3. 2.2.2 Clinical Features:

The major feature is reduced stroke volume due to a combination of impaired diastolic filling and left ventricular outflow tract. Focal myocardial ischemia is common due to increased ventricular pressures , massive myocyte hypertrophy, diminished stroke volume, and frequently abnormal intramyocardial arterioles(*Richard Mitchell et al ,2012*).

HCM can be entirely asymptomatic. Symptomatic disease usually presents in young adults with dyspnea, angina, and/or syncope. The clinical course can be highly variable; HCM is one of the most common causes of sudden death in young athletes(*Richard Mitchell et al ,2012*).

2.3.3.2.3 Restrictive Cardiomyopathy:

Relatively rare and with multiple etiology, this entity is marked by a restriction of ventricular filing leading to reduced cardiac output. Contractile function is usually normal. Ventricle size is normal, although there is typically bilateral dilation. Non- specific interstitial myocardial fibrosis is usually present(*Richard Mitchell et al ,2012*).

2.3.3.2.3.1 Biopsy frequently reveals a specific etiology, Causes include:

Endomyocardial fibrosis is a disease mainly of African children and young adults; the cause is unknown. It is characterized by dense ventricular subendocardial fibrosis extending from the apex upward, often with superimposed organizing mural thrombus (*Richard Mitchell et al ,2012*).

Loffler endocarditis is morphologically similar to endomyocardial fibrosis but is classically associated with peripheral eosinophilia and eosinophilic infiltration of multiple organs (especially the heart). The cardiac changes are probably due to toxic products of eosinophils, and the course can be rapidly fatal (*Richard Mitchell et al ,2012*).

Endocardialfibroelastosis is an uncommon disorder of obscure etiology, characterized by focal to diffuse, fibro elastic thickening of the endocardial, and left ventricle greater than right. It occurs at all ages but is most common in patients less than 2 years old (*Richard Mitchell et al ,2012*).

2.3.3.3 Congestive Heart Disease:

Congestive heart failure is a multisystem derangement that occurs when the heart is no longer able to eject the blood delivered to it by the venous system. Excluded from this definition are conditions in which inadequate cardiac output occurs because of blood loss or some other process that impairs the return of blood to the heart (*Burn and Kumer,2007*).

In an additional minority of cases, heart failure may result because of greatly increased demands for blood by the tissues, a process sometimes referred to as high output failure. Inadequate cardiac output, also termed forward failure, is almost always accompanied by increased congestion of the venous circulation (backward failure), because the failing ventricle is unable to eject the normal volume of venous blood delivered to it during diastole. This results in an increase in the volume of blood in the ventricle at the end of diastole, an elevation in end-diastolic pressure within the heart, and, finally, elevated venous pressure (*Burn and Kumer,2007*).

The most common causes of left-sided cardiac failure are systemic hypertension, mitral or aortic valve disease, ischemic heart disease, and primary diseases of the myocardium. The most common cause of right-sided heart failure is left ventricular failure, with its associated pulmonary congestion and elevation in pulmonary arterial pressure. Right-sided failure may also occur in the absence of left-sided heart failure in patients with intrinsic diseases of the lung parenchyma and/or pulmonary vasculature (corpulmonale) and in patients with pulmonic or

tricuspid valve disease. It sometimes follows congenital heart diseases, in which there is a left-to-right shunt (*Burn and Kumer,2007*).

As the heart begins to fail, a number of local adaptive responses are triggered in an attempt to maintain normal cardiac output. These include neurohormonal reactions as well as molecular and morphologic changes within the heart. One of the earliest neurohormonal responses to decreased cardiac output is an increase in the activity of the sympathetic nervous system (*Burn and Kumer,2007*).

Catecholamines cause both a more forceful contraction of the heart muscle and an increase in heart rate. Over time, the overburdened heart may respond to increased demands by undergoing various forms of "remodeling," including hypertrophy and dilatation. Because cardiac muscle fibers in the adult no longer have the ability to proliferate to any significant degree, the major initial structural adaptation to a chronically increased workload is hypertrophy of individual muscle fibers (*Burn and Kumer,2007*).

The development of hypertrophy initially serves as a positive, adaptive response. Oxygen requirements of the hypertrophic myocardium are increased, owing to increased myocardial cell mass and increased tension of the ventricular wall. Because the myocardial capillary bed does not always increase sufficiently to meet the increased oxygen demands of the hypertrophic muscle fibers, the myocardium becomes vulnerable to ischemic injury (*Burn and Kumer,2007*).

Increased cardiac workload of any type predisposes to the development of cardiac dilatation, or chamber enlargement, when increased sympathetic activity and myocyte hypertrophy prove insufficient to expel all the venous blood that drains into the heart. As cardiac failure progresses, end-diastolic pressure increases, causing individual cardiac muscle fibers to stretch, ultimately increasing the volume of the cardiac chamber (*Burn and Kumer,2007*).

In accordance with the Frank-Starling relationship, these lengthened fibers initially contract more forcibly, thereby increasing cardiac output. If the dilated ventricle is able to maintain cardiac output at a level that meets the needs of the body, the patient is said to be in compensated heart failure (*Burn and Kumer,2007*). However, cardiac dilatation, like hypertrophy, has certain deleterious effects on the heart. Increasing degrees of dilatation result in an increase in wall tension of the affected chamber, which causes, in turn, an increase in the oxygen requirements of an already-compromised myocardium. With time, the failing myocardium is no longer able to propel sufficient blood to meet the needs of the body, even at rest. At this point, patients enter a phase termed decompensated heart failure(*Burn and Kumer,2007*).

Heart failure causes changes in other organs as well. Cardiac failure inevitably includes an element of backward failure, the result of which is congestion of the venous circulation. In a patient with left-sided failure, this results in passive congestion of the pulmonary circulation. As left ventricular failure progresses, the hydrostatic pressure in the pulmonary vasculature increases sufficiently to cause leakage of fluid and, occasionally, erythrocytes into the interstitial tissue and airspaces of the lungs to produce pulmonary edema (*Burn and Kumer,2007*).

Congestion of the pulmonary circulation also causes an increase in pulmonary vascular resistance and, with it, an increased workload on the right side of the heart. This increased burden, if sustained and severe, may ultimately cause the right side of the heart to fail also. Failure of the right side of the heart, in turn, contributes to the development of systemic venous congestion and soft tissue edema (*Burn and Kumer,2007*).

As the heart fails, a number of systemic alterations also occur that serve to maintain cardiac output at near-normal levels. Decreased left ventricular output

(forward failure) is associated with decreased perfusion of the kidneys, which in turn causes local activation of the renin-angiotensin system. Aldosterone released in response to activation of the renin-angiotensin system causes the renal tubules to resorb both sodium and water. This sequence of events, sometimes called secondary hyperaldosteronism, increases the total plasma volume of extracellular fluid (*Burn and Kumer,2007*).

However, unless the performance of the cardiac pump improves, the failing heart is unable to pump the increased intravascular volume, which remains pooled in the veins, thereby adding further to systemic and pulmonary venous congestion. This ultimately contributes further to both pulmonary and soft tissue edema (*Burn and Kumer,2007*).

2.3.3.3.1 Morphology :

The failing cardiac chambers are dilated and are usually hypertrophied as well. In left-sided failure, the lungs are boggy and congested and the cut surface exudes a frothy mixture of surfactant-rich fluid and blood (*Burn and Kumer,2007*).

Microscopically, the pulmonary alveolar capillaries are congested. There is transudation of fluid, initially limited to perivascular interstitial spaces, causing widening of the alveolar septa. In time, it overflows into the alveoli (pulmonary edema) (*Burn and Kumer,2007*).

Long-standing right-sided heart failure is associated with congestion of the abdominal viscera, soft tissue edema, and, in some cases, fluid in the pleural, pericardial, and abdominal cavities. Changes in the liver include chronic passive congestion, characterized by atrophy of hepatocytes around the central veins(*Burn and Kumer,2007*).

2.3.3.3.2 Clinical Features :

The most common manifestation of left ventricular failure is dyspnea, or a sense of breathlessness. This is caused predominantly by decreased lung compliance

resulting from pulmonary edema and congestion and by increased activity of autonomic stretch receptors within the lung. Dyspnea is most noticeable during periods of physical activity (exertional dyspnea). It is also prominent when the person is lying down (orthopnea) because of the increased amount of venous blood returned to the thorax from the lower extremities and because the diaphragm is elevated in this position. Paroxysmal nocturnal dyspnea is an especially dramatic form of dyspnea that awakens the patient with sudden, severe shortness of breath, accompanied by coughing, a choking sensation, and wheezing (*Burn and Kumer,2007*).

Other manifestations of left ventricular failure include muscle fatigue, an enlarged heart, tachycardia, a third heart sound (S3), and fine rales at the lung bases, produced by the flow of air through edematous pulmonary alveoli. With progressive ventricular dilation, the papillary muscles are displaced laterally, causing mitral regurgitation and a high-pitched systolic murmur (*Burn and Kumer,2007*).

Chronic dilation of the left atrium may also occur, which is often associated, in turn, with the development of atrial fibrillation, manifested by an "irregularly irregular" heartbeat(*Burn and Kumer,2007*).

As stated earlier, right-sided heart failure is most often caused by left-sided failure. Its major consequences are systemic venous congestion and soft tissue edema. Systemic venous congestion is manifested clinically by distended neck veins and an enlarged, sometimes tender liver. It is also associated with an increased frequency of deep venous thrombi and pulmonary embolism(*Burn and Kumer,2007*).

Edema causes weight gain and usually becomes apparent first in the dependent areas of the body, such as the feet and lower legs. With more severe degrees of ventricular failure, the edema may become generalized. Pleural

effusions are common, particularly on the right side, and may be accompanied by pericardial effusions and ascites(*Burn and Kumer,2007*) .

2.4.1 Echoacrdiography

2.4.1.1 Introduction:

Echocardiogram, often referred to as a **cardiac echo** or simply an **echo**, is a sonogram of the heart. (It is not abbreviated as Electrocardiography (ECG), which in medicine usually refers to an electrocardiogram.) Echocardiography uses standard two-dimensional, three-dimensional, and Doppler ultrasound to create images of the heart.

Echocardiography has become routinely used in the diagnosis, management, and follow-up of patients with any suspected or known heart diseases. It is one of the most widely used diagnostic tests in cardiology. It can provide a wealth of helpful information, including the size and shape of the heart (internal chamber size quantification), pumping capacity, and the location and extent of any tissue damage. An Echocardiogram can also give physicians other estimates of heart function such as a calculation of the cardiac output, ejection fraction, and diastolic function (how well the heart relaxes).

Echocardiography can help detect cardiomyopathies, such as hypertrophic cardiomyopathy, dilated cardiomyopathy, and many others. The use of Stress Echocardiography may also help determine whether any chest pain or associated symptoms are related to heart disease. The biggest advantage to echocardiography is that it is noninvasive (doesn't involve breaking the skin or entering body cavities) and has no known risks or side effects.

Not only can an echocardiogram create ultrasound images of heart structures, but it can also produce accurate assessment of the blood flowing through the heart, using pulsed or continuous wave Doppler ultrasound. This allows assessment of both normal and abnormal blood flow through the heart. Color Doppler as well as spectral Doppler is used to visualize any abnormal communications between the left and right side of the heart, any leaking of blood

through the valves (valvular regurgitation), and to estimate how well the valves open (or do not open in the case of valvular stenosis) (*Donnelly,2013*)

Echocardiography is now a fully grown tree. It has numerous clinical applications, with various forms of ultrasound technology being used throughout the entire field of cardiovascular medicine.

This mature ultrasound tree has grown from a seed planted more than 50 years ago. Since then, the tree has been trimmed and nourished carefully by many pioneers to serve the needs of patients and clinicians (*James and Jamil,2006*).

The first application of diagnostic ultrasound in medicine was in the late 1930s, when Karl Dussik, an Austrian psychiatrist and neurologist, became interested in the potential use of ultrasound for brain imaging. Ultrasound was already in use at that time by mariners for underwater imaging and also by engineers for flaw detection in metals. The piezoelectric effect was already well known, having been discovered more than half a century earlier, and the concept of using a piezoelectric crystal both to transmit and receive ultrasound was described in 1917 (*Houghton,2014*).

Echo transmission was also the first ultrasound technique used for cardiac imaging, by the German physiologist Wolf-Dieter Keidel, in order to make measurements of the heart and thorax. Echo reflection was first used by IngeEdler and Carl Hellmuth Hertz in Sweden (*Houghton,2014*).

One weekend in 1953 they borrowed an industrial device, used to detect flaws in metals by the Kockum shipyard in Malmo, to conduct their work on human subjects (*Houghton,2014*).

By a fortunate coincidence the frequency of the echo transducer happened to be one that was suitable for cardiac imaging. The image of the heart they produced was known as an A-mode scan and was thought to show the posterior wall of the left ventricle (LV) (*Houghton,2014*).

They were soon granted an ultrasound machine of their own and began to produce M-mode scans, with which they were able to examine the mitral valve and also detect atrial thrombus, myxoma, and pericardial effusion (*Houghton,2014*).

Nonetheless, it was not until the early 1960s that the potential value of cardiac ultrasound became more widely recognized. The first dedicated cardiac ultrasound machine, developed by Jack Reed and Claude Joyner, appeared at this time and the term ‘echocardiography’ was coined for the first time (*Houghton,2014*).

Real-time two dimensional (2D) echo followed in the 1960s, spurred on by advances in electronics, and by the early 1970s mechanical transducers were available that could produce 2D images by steering the transducer back and forth, sweeping the ultrasound beam across the heart. Phased-array transducers soon followed, in which the mechanical beam-steering mechanism was replaced by solid-state electronics (*Houghton,2014*).

The 1970s also saw rapid developments in the use of Doppler techniques, and by the early 1980s colour Doppler imaging was becoming a common feature of echo studies (*Houghton,2014*).

During the 1980s, the technique of transoesophageal echo started to enter clinical practice, initially with monoplane probes but later with biplane probes, multiplane probes and, ultimately, the use of three dimensional (3D) transoesophageal imaging (*Houghton,2014*).

The 1990s saw a gradual change in archiving methods, with a move away from recording studies on videotape towards more versatile digitally based archiving. There were also refinements in the quality of echo, with the introduction of harmonic imaging and the growing use of echo contrast agents to enhance endocardial border definition (*Houghton,2014*).

Tissue Doppler imaging entered mainstream practice towards the end of the 1990s, adding a new modality that has proven particularly valuable in the assessment of LV diastolic function (*Houghton,2014*).

The new millennium saw the increasing adoption of three and four dimensional (3D/4D) echo, both in transthoracic and transoesophageal (TOE) studies. The use of speckle tracking echo has provided valuable insights into myocardial mechanics and is gradually moving from the research setting into routine clinical practice. Meanwhile, echo machines have gradually shrunk, initially to the size of laptop computers, and subsequently to the size of handheld devices, greatly increasing the portability and availability of echo technology (*Houghton,2014*).

2.4.1.2 Physics and instrumentation:

Echocardiography uses ultrasound to examine the structure and function of the heart. **A firm understanding of the physics of ultrasound gives the sonographer:**

- an understanding of the capabilities and limitations of their echo machine
- the confidence to adjust the machine's controls to optimize the images (*Houghton,2014*).

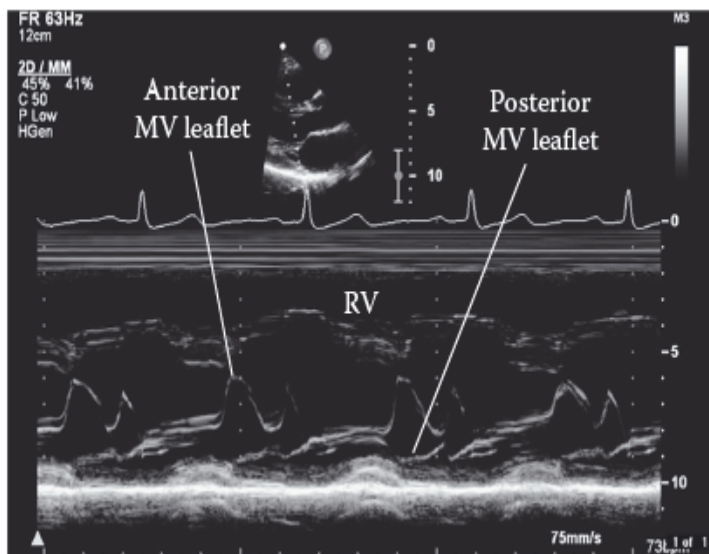
2.4.1.3 Imaging modalities :

The earliest echo modality was amplitude mode (A-mode) imaging, which simply plotted the amplitude of the reflected ultrasound (as a 'spike' with a certain amplitude) versus the distance of the reflected signal from the transducer.

Brightness mode (B-mode) imaging was similar in principle, but rather than plotting the returning signals as a row of spikes of varying sizes, it represented the amplitude of the returning signal by the brightness of a dot. A-mode and B-mode imaging have been superseded by M-mode and 2D imaging (*Houghton,2014*).

2.4.1.3.1 M-mode imaging :

M-mode (or motion mode) imaging records motion along a single ‘line of sight’, selected by careful positioning of the on-screen cursor across a region of interest (**Figure 2.45**) . Once the cursor is in place, activation of M-mode imaging produces a scrolling display of movement (along the vertical y axis), as it occurs along the cursor (**Figure 2.45**) .Positioning of the cursor for an M-mode study of the mitral valve. From this, and from a knowledge of the propagation velocity of ultrasound in soft tissue, the echo machine can calculate the distance between the transducer and the reflector. The transducer can also determine the intensity of the returning signal, and use this information in building up the image display. Other features of the returning signal, such as its frequency and any frequency shift compared to the transmitted signal (*Houghton,2014*).

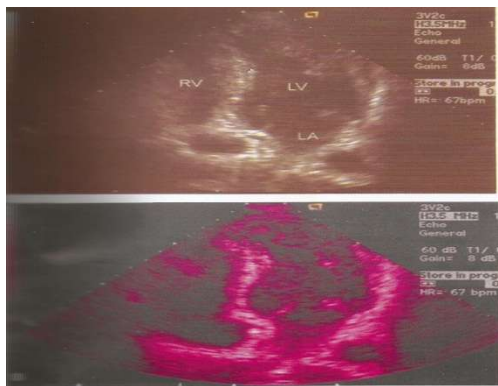


(Figure 2.39) M-mode study of the mitral valve (MV – mitral valve; RV – right ventricle) (*Houghton,2014*).

2.4.1.3.2 Color B-mode Scanning:

For routine two-dimensional imaging (B-mode), the image typically is displayed in gray scale . Although first-generating scanners were limited to 16 shades of gray and later scanners to 64 shades, current instrument display 256

shades of gray. This degree of gray scale range exceeds the eye's ability to discern differences. An alternative mode of display is to convert the gray scale assignments to a range of color or hue within a color (color B-mode)(**Figure 2.40**) and (**Figure 2.41**). Studies have suggested that this may enhance detection of subtle soft tissue density targets. This display format has seen most acceptance in display of spectral Doppler signals and of three-dimensional echocardiographic image , where hue may be used to denote depth. Note the more obvious nature of the apical hypertrophy in the B-mode color image (*William and Ryan ,2010*).



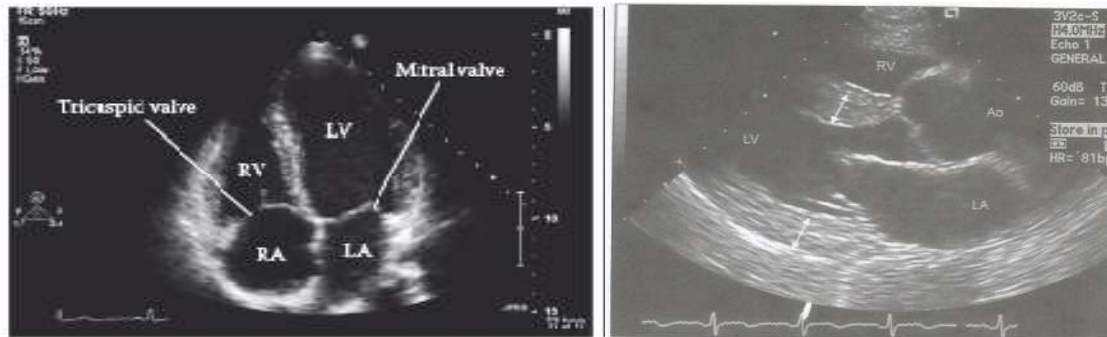
(**Figure 2.40**) (A) Apical four chamber view recorded in a patient with the apical variant of hypertrophic cardiomyopathy in routine gray scale (B) B-mode color (*William and Ryan ,2010*).

2.4.1.3.3 Two-dimensional imaging :

Whereas in M-mode imaging the heart is imaged along just a single scan line, in 2D imaging a picture of the heart is built up from a series of scan lines side by side. In 2D imaging the ultrasound probe sweeps a beam across the heart around 20–30 times per second, creating a series of scan lines (usually around 120) each time it makes a sweep, in order to build up a 2D image (**Figure 2.41**).

The probe has to transmit and receive an ultrasound pulse for each scan line of the image, and there is therefore a limit on how many image ‘frames’ can be generated each second, determined by the number of scan lines that make up the image (the sector width) and the depth of the image. Reducing the sector width

and/or depth will reduce the time taken to generate an image frame, increasing the number of image frames that can be generated each second (‘frame rate’) (*William and Ryan ,2010*).



(Figure 2.41) Normal 2D echo (LA – left atrium; RV – right ventricle) (*Houghton,2014*) and (*William and Ryan ,2010*).

2.4.1.3.4 Three dimensional Echocardiography:

Three- dimensional echocardiography is a technique that remains in evolution . The eventual goal is a real-time, three-dimensional display of cardiac anatomy incorporating all the modalities mentioned above including Doppler flow imaging , M-mode echocardiography , and Doppler tissue imaging .Several approaches have been taken to acquire and display three-dimensional information .

The basic problems with three dimensional echocardiography can be divided into those of acquisition of the three-dimensional data set and subsequent display of the images. A limitation has been that the information either acquired as or reconstructed into a three-dimensional data set must subsequently be displayed as a two-dimensional image (*William and Ryan ,2010*).

2.4.1.4 Display and recording methods

The returning echo signal at the transducer undergoes a series of initial processing steps which include amplification, time gain compensator (TGC) and filtering. The video signal is then sent to a scan converter, which converts the signal into a ‘rectangular’ format suitable for display. The resulting data undergo

further processing ('post-processing') and can then be stored in a digital format and/or can undergo digital-to-analogue conversion to create a video signal for display on a monitor (and/or archiving onto videotape).

This process occurs so rapidly that the acquired data can be displayed on a monitor almost in 'real time'. Storage of echo studies can be on videotape, which is relatively inexpensive, although it rapidly becomes cumbersome to store (and to review) studies when large numbers are archived in this way.

Digital archiving is now more commonly used, with storage on hard drives or optical disks. This makes accessing studies easier and allows greater flexibility in image processing after the study has been completed.

However, the quantity of digital data generated by an echo study can be considerable, so high volume storage media (and 'lossless' data compression techniques) are required if large numbers of studies are to be archived (*Houghton,2014*).

2.4.1.5 Acquisition of cardiac Ultrasound Information:

2.4.1.5.1 Transthoracic echocardiogram:

A standard echocardiogram is also known as a transthoracic echocardiogram (TTE), or cardiac ultrasound. In this case, the echocardiography transducer (or probe) is placed on the chest wall (or thorax) of the subject, and images are taken through the chest wall. This is a non-invasive, highly accurate and quick assessment of the overall health of the heart (*Medical Imaging Radiology,2016*).

2.4.1.5.2 Transesophageal echocardiogram

This is an alternative way to perform an echocardiogram. A specialized probe containing an ultrasound transducer at its tip is passed into the patient's esophagus. This allows image and Doppler evaluation from a location directly behind the heart. This is known as a transoesophageal echocardiogram, or TOE (TEE in the United States).

Trans esophageal echocardiograms are most often utilized when transthoracic images are suboptimal and when a more clear and precise image is needed for assessment. This test is performed in the presence of a cardiologist, registered nurse, and ultrasound technician. Conscious sedation and/or localized numbing medication, may or may not be used in order to make the patient more comfortable during the procedure (*Medical Imaging Radiology,2016*).

2.4.1.5.3 Three-dimensional echocardiography:

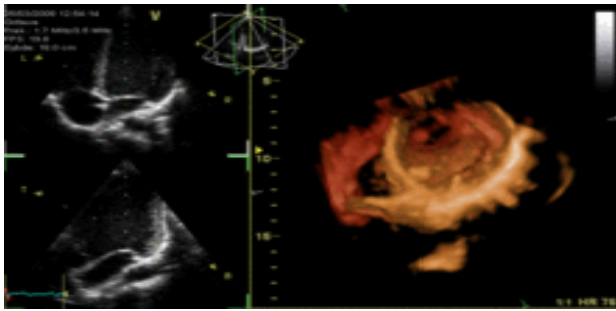
3D echocardiography (also known as 4D echocardiography when the picture is moving) is now possible, using a matrix array ultrasound probe and an appropriate processing system. This enables detailed anatomical assessment of cardiac pathology, particularly valvular defects, and cardiomyopathies.

The ability to slice the virtual heart in infinite planes in an anatomically appropriate manner and to reconstruct three-dimensional images of anatomic structures make 3D echocardiography unique for the understanding of the congenitally malformed heart.

Real Time 3-Dimensional echocardiography can be used to guide the location of biotomes during right ventricular endomyocardial biopsies, placement of catheter delivered valvular devices, and in many other intraoperative assessments. The 3D Echo Box developed by the European Association of Echocardiography offers a complete review of Three Dimensional Echocardiography (**Figure 2.42**) .

Three-dimensional echocardiography technology may feature Anatomical Intelligence, or the use of organ modeling technology to automatically identify anatomy based on generic models. All generic models reference a dataset of anatomical information that uniquely adapts to variability in patient anatomy to perform specific tasks. Built on feature recognition and segmentation algorithms, this technology can provide patient-specific three-dimensional modeling of the

heart and other aspects of the anatomy including the brain, lungs, liver, kidney, rib cage and vertebra column (*Medical Imaging Radiology,2016*).



(Figure 2.42)Three-dimensional echocardiogram of a heart viewed from the apex (*Medical Imaging Radiology,2016*).

2.4.1.5.4 Contrast echocardiography:

Contrast echocardiography, or Contrast-enhanced ultrasound is the addition of ultrasound contrast medium, or imaging agent, to traditional ultrasonography. The ultrasound contrast is made up of tiny micro bubbles filled with a gas core and protein shell. This allows the micro bubbles to circulate through the cardiovascular system and return the ultrasound waves creating a highly reflective image. The most commonly used types of ultrasound contrast are known as Definity®(definityimaging.com) and Option ® (optisonimaging.com). Both have been approved by the Food and Drug Administration (FDA) . There are multiple applications in which contrast-enhanced ultrasound can be useful. The most commonly used application is in the enhancement of LV endocardial borders for assessment of global and regional systolic function.

Contrast may also be used to enhance visualization of wall thickening during stress echocardiography, for the assessment of LV thrombus, or for the assessment of other masses in the heart. Contrast echocardiography has also been used to assess blood perfusion throughout myocardium in the case of coronary artery disease. The Contrast Echo Box developed by the European Association of Echocardiography, and the American Society of Echocardiography Contrast

Zoneboth offer a complete review of Contrast Echocardiography (*Medical Imaging Radiology,2016*).

2.4.1.5.5 Epicardial Imaging :

The fidelity of image registration is greatest when the amount of intervening tissue is minimal . For this reason , application of an ultrasound probe directly to the heart provides a high resolution , non-obstructive view of cardiac structures.

Because the very near field of most ultrasound transducers is subject to distortion , probes have been designed specifically for epicardial application. Because these probes are placed directly on the beating heart or vascular , they must be either sterilized or more commonly placed in a sterile insulating sheath before use (*William and Ryan ,2010*) .

2.4.1.5.6 Intracardiac Echocardiography:

This technique is applicable only in the invasive laboratory setting and requires large-bore intravascular access (*William and Ryan ,2010*) .

2.4.1.5.7 Intravascular Ultrasound:

The technique was instrumental in determining the optimal methods for intracoronary stent deployment . It is usually most often used to define the true anatomic severity of “intermediate” coronary lesions noted on angiography or for highly precise evaluation of coronary arterial wall anatomy (*William and Ryan ,2010*).

2.4.1.6 Safety of ultrasound:

Ultrasound involves the delivery of external energy to body tissues, and so it is important to consider the potential adverse biological effects that this could entail. The intensity of exposure to ultrasound is expressed as power per unit of area (watts/cm²) expressed as the maximum intensity within the ultrasound beam (the spatial peak) averaged over the duration of exposure (temporal average), the **spatial peak temporal average (SPTA)**. There are two main biological effects of

exposure to ultrasound energy: **thermal (heating) and mechanical (e.g. cavitation)** (*Houghton,2014*).

2.4.1.6.1 Thermal effects :

Thermal effects are caused by conversion of the mechanical energy of the ultrasound into heat energy as it passes through the tissues. The amount of heating is hard to predict but relates to several factors including transducer frequency, transmit power, focus and depth. Thermal effects are most relevant to TOE where the probe may remain stationary in the oesophagus for long periods, particularly during intraoperative studies. Heat may be generated not just by the ultrasound but also directly by the probe itself. It is prudent to keep imaging time to a minimum and to ensure that the TOE probe is repositioned regularly, and to monitor the temperature of the probe (*Houghton,2014*).

2.4.1.6.2 Mechanical effects :

Mechanical effects include cavitation, in which gas bubbles are created as ultrasound passes through the tissues. It is not thought to be a problem during standard transthoracic studies, but is important when bubble contrast agents are used as it can cause resonance and even disruption of the bubbles .

Mechanical effects of ultrasound can also be measured by **mechanical index (MI)**, which is the peak negative (rarefactional) pressure divided by the square root of the transducer frequency. **An MI of 1 is considered safe** (*Houghton,2014*).

2.4.2 Technique of Transthoracic echocardiography

2.4.2.1 Indications for transthoracic echo :

The versatility of transthoracic echocardiography (TTE) means that it can play a useful role in a diverse range of clinical situations. The British Society of Echocardiography (BSE) has published guidance on the appropriate clinical indications for TTE. The American College of Cardiology Foundation has also produced guidance (jointly with a number of other societies).

The two sets of guidelines are broadly similar and describe echo as being an appropriate investigation in the assessment of patients with:

- symptoms, signs or previous tests that indicate possible structural heart disease
- heart murmurs when associated with symptoms or when structural heart disease is suspected, and the follow-up of those with known significant valvular stenosis or regurgitation.
- prosthetic valves (except asymptomatic patients with mechanical valves or those in whom no further intervention would be undertaken)
- suspected or proven infective endocarditis.
- known or suspected ischaemic heart disease (e.g. diagnostic stress echo, assessment following myocardial infarction).
- known or suspected cardiomyopathy.
- suspected pericarditis, pericardial effusion, cardiac tamponade or pericardial constriction, and follow-up of patients with known moderate or large pericardial effusions (or small effusions if there has been a clinical change)
- suspected or possible cardiac masses (and follow-up of patients following surgical excision of a cardiac mass)
- pulmonary disease (with cardiac involvement)
- pulmonary hypertension
- thromboembolism
- neurological disorders (with cardiac involvement)
- arrhythmia, palpitations and syncope (with suspected/possible structural heart disease)
- prior to cardioversion (unless the patient is on long-term anticoagulants at a therapeutic level and there is no suspicion of structural heart disease)
- hypertension (if left ventricular hypertrophy (LVH)/dysfunction or aortic coarctation are suspected)

- aortic disease (e.g. monitoring of aortic root dimensions in Marfan Syndrome)
- known or suspected congenital heart disease.

Transthoracic echo is also indicated for pre-operative assessment in patients awaiting elective or semi-urgent surgery if they have:

- known ischaemic heart disease with a reduced functional capacity
- unexplained breathlessness (with an abnormal electrocardiogram and/or chest X-ray).
- a murmur (with suspected structural heart disease or in the presence of cardiac or respiratory symptoms).

It is essential that echo requests contain adequate clinical data both to judge the appropriateness of the request and also to allow the sonographer to place the echo findings into an appropriate clinical context.

Echo requests must therefore carry appropriate clinical details and contain information about known cardiac diagnoses or previous cardiac interventions/surgery (e.g. prosthetic valves)(*Houghton,2014*).

2.4.2.2 Patient preparation :

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

Reassure patients that having an echo is safe and painless. Patients can eat and drink normally before attending for a standard TTE, and they can take their medication as usual. It is good practice to offer patients an information leaflet before they attend (and to make available large-print/Braille and translated versions as appropriate).

The patient information leaflet and/or appointment letter can also invite the patient to bring a friend or relative if they wish to have someone accompany them during the echo. If a friend or relative does not accompany the patient when they attend, offer the patient a chaperone in line with hospital policy.

Prior to performing the echo study, it is good practice to record the patient's height and weight, as this will allow the indexing of echo measurements for body surface area.

You should also ideally record the patient's heart rate and blood pressure. Once you have checked that the patient understands the test that is about to be performed, ask the patient to undress to the waist for the echo study. Always offer female patients a gown to wear during the echo (even if the sonographer is female).

Ask the patient to sit on the echo couch and recline at 45°, rolling on to their left side. The patient should then raise their left arm and place their left hand behind their head. Be sure to check if the patient has any physical limitations that may make it difficult or uncomfortable for them to adopt this position. If so, you may need to adapt the patient's position until they are comfortable. Sonographers who prefer to scan left-handed will also need to adapt the patient's positioning accordingly.

When the patient is in a comfortable position, apply the ECG electrodes and ensure that a clear ECG tracing is visible on the screen of the echo machine. You may need to adjust the electrodes and/or the ECG gain setting to obtain a good trace. Ensure that the correct patient identification and clinical details are entered into the echo machine, and then perform and report the study as described in the sections that follow.

At the end of the study, explain to the patient that you will be writing a report which will be sent to the referring clinician. Patients may ask you what the

study has shown, but you should not discuss the study findings at this stage and it is usually better to redirect persistent requests for information to the referring clinician (*Houghton,2014*).

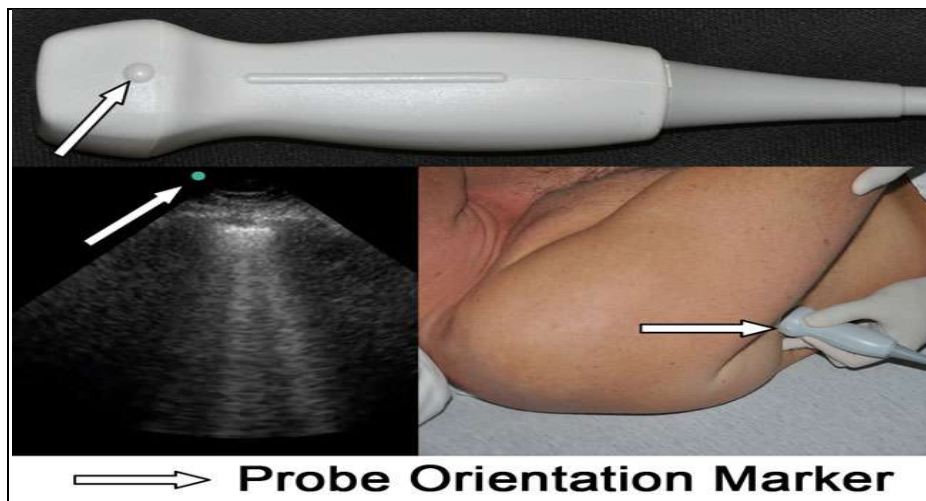
2.4.2.3 Preparing machine and probe :

- Set up an ergonomic orientation of machine , patient , and operator . This will depend on your preferred operator position. A standard way is to have the operator sitting behind the patient on the edge of the couch with their right arm holding the probe and wrapped over the patient. The machine is then operated by the left hand . Another standard method is to site facing the patient holding the probe against the chest wall with the left arm (arm rested on the couch) and with machine operated by the right hand.
- Check thoracic image settings on machine , with harmonic imaging (if available) and your preferred image post-processing options selected . Set overall gain , compress , and transverse or lateral gain controls to standard positions.
- Make sure there is an ECG tracing on the echo machine and patient details are entered.
- Make sure there is an storage is possible (to magneto-optical disc, download to image server or videotape).
- Take the appropriate transthoracic probe, apply gel to transducer , and start imaging (*Paul and Andrew,2012*).

2.4.2.4 Probe handling and image quality:

The probe should be held in one hand and pressured firmly against the chest wall. Varying the pressure will alter image quality .Ensure sufficient pressure to optimize the image but not too much to make it uncomfortable for the patient .The usual problem while learning is not applying enough pressure to gel good image quality.

A layer of gel ensures good contact between probe and chest wall . It excludes any air(that would degrade image quality). However , too much gel makes it difficult to keep a stable position so , once a layer is established , try not to apply more gel as image quality will not improve.The probe has a dot on one side to orientate the probe in your hand with the image on the screen (**Figure 2.43**).



(Figure 2.43) The probe has a dot on one side to orientate the probe in your hand with the image on the screen (*Paul and Andrew,2012*) .

The probe can be moved in multiple directions but the four key movements are :

- rotation around a point;
- rocking back and forwards;
- rocking side to side; and
- sliding across the chest. Movements needed to improve image quality are usually quite small and, with experience , hand movements are almost subconscious.

Remember that image quality may be improved by different patient or heart positions. These can be altered by physically rolling the patient a little , one way or the other , readjusting the patient to ensure they are sitting up , or asking the patient to breathe in or out to move the diaphragm(and heart)

- Imaging well is hard work and requires concentration . If it is proving difficult to find , keep , or return to an image during a study remember a short break can help. Remove the probe from the chest , re-apply gel and start again (*Paul and Andrew,2012*).

2.4.2.5 Standard windows and views :

The BSE has produced a guidance document, entitled *A Standard Transthoracic Echocardiogram*, which provides a framework for performing a comprehensive transthoracic echo study.

This document forms the basis of the approach outlined, and identifies minimum requirements and recommendations (in terms of views and measurements).

A comprehensive echo study should include not only the minimum requirements but also the recommendations, and this is particularly important in individuals who are being scanned for the first time.

Moreover, if pathology is found, then additional views/measurements (over and above the minimum requirements and recommendations) may be appropriate (*Houghton,2014*).

There are five TTE windows (Figure 2.50) , each providing one or more views of the heart. The right parasternal window is optional and can be used when other views are suboptimal or when additional information is needed:

➤ Left parasternal window

- Parasternal long axis view
- Parasternal right ventricular (RV) inflow view
- Parasternal RV outflow view
- Parasternal short axis view (base, mid-cavity, apex)

➤ (Right parasternal window)

- Apical window

- Apical 4-chamber view
- Modified apical 4-chamber view (to assess the right heart)
- Apical 5-chamber view
- Apical 2-chamber view
- Apical 3-chamber (long axis) view

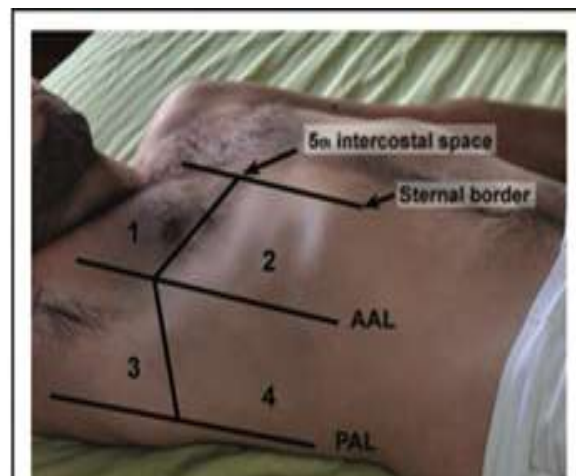
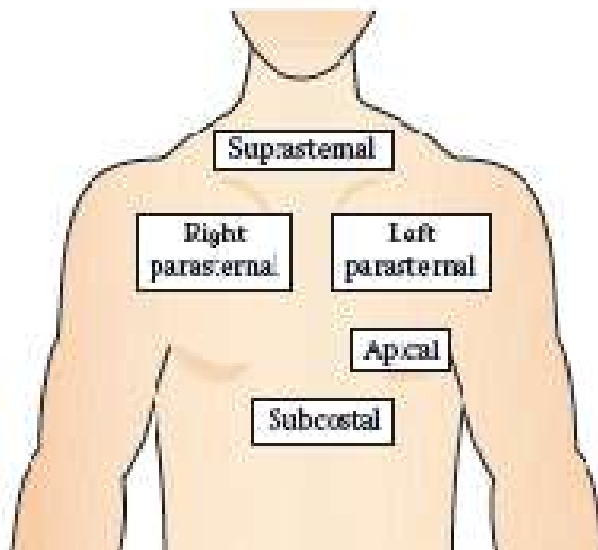
➤ **Subcostal window**

- Subcostal long axis view
- Subcostal short axis view

➤ **Suprasternal window**

- Aorta view.

(Houghton,2014).



line; PAL, posterior axillary line.

(Figure 2.44) Transthoracic echo window (Figure 2.45) The four Volpicelli's zones. AAL, anterior axillary line ; PAL- posterior axillary line (Houghton,2014) and (Paul and Andrew,2012) .

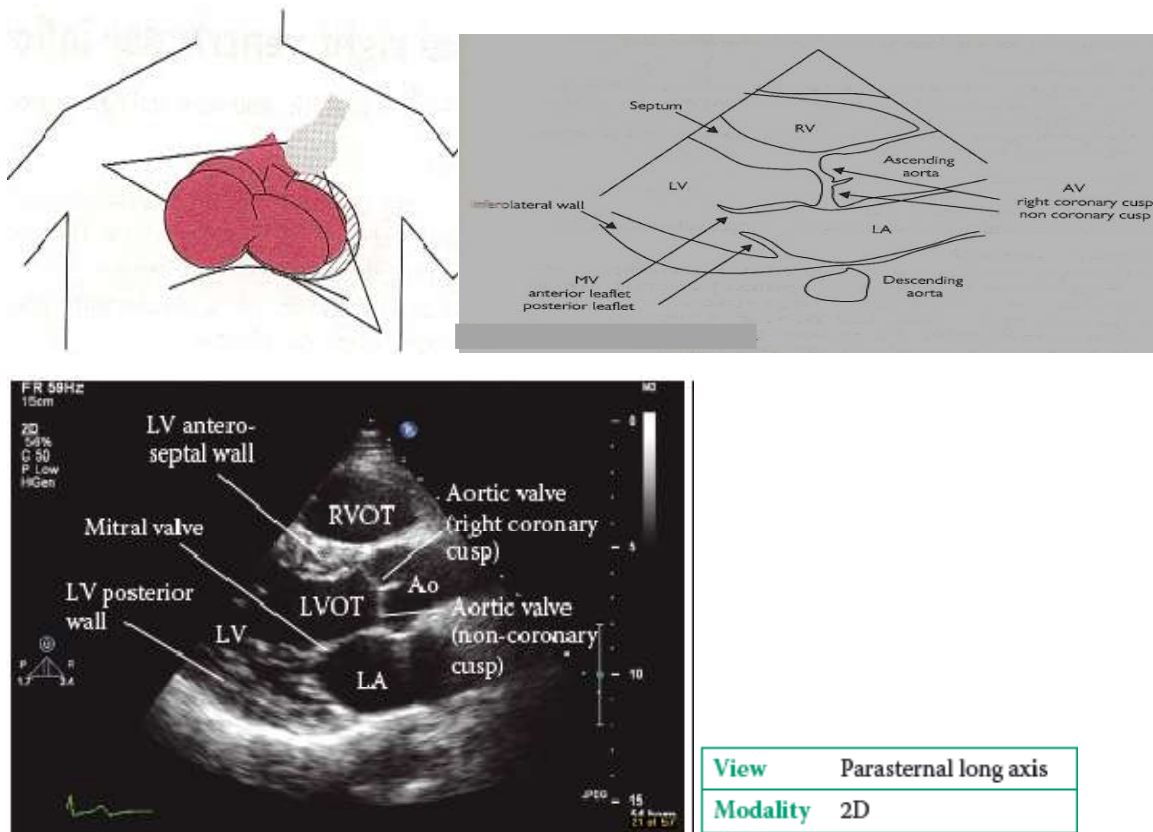
2.4.2.5.1 Left parasternal window

The left parasternal window is located to the left of the sternum, usually in the third or fourth intercostal space, but in some patients you may need to adjust the

position to optimize the image by moving the probe up/down a rib space or further towards/away from the sternum. From the left parasternal window a number of views can be obtained (Houghton,2014) .

2.4.2.5.1.1 Parasternal long axis view:

The parasternal long axis (LAX) view is shown in (Figure 2.46). To obtain the view with the probe in the left parasternal window, rotate the probe so that the probe’s ‘reference point’ (sometimes a ‘dot’) is pointing towards the patient’s right Shoulder .



(Figure 2.46) Normal parasternal long axis view(Ao = aorta; LA = left atrium; LV = left ventricle; LVOT = left ventricular outflow tract; RVOT = right ventricular outflow tract) (Houghton,2014) and (Paul and Andrew,2012) .

For an optimal view, aim to position the probe so that the view cuts through the centre of the mitral and aortic valves, without foreshortening the left ventricle (LV) or ascending aorta (Houghton,2014) .

2.4.2.5.1.1.1 In this view Use 2D to:

- measure LV cavity size and wall thickness (this can be done using M-mode if you prefer)
- assess LV radial function (thickening and motion of the anteroseptal and inferolateral (also known as posterior) wall)
- inspect the appearance of the left atrium (LA) and measure its size at end systole.
- assess structure and mobility of the mitral valve – in this view, the A2 and P2 segments are visible
- assess structure and mobility of the aortic valve. The right and non-coronary cusps are visible and normally have a central closure line – an eccentric closure line suggests bicuspid aortic valve
- inspect the appearance of the left ventricular outflow tract (LVOT) and measure its diameter (no more than 1 cm below the aortic valve annulus)
- measure the diameter of the right ventricular outflow tract (RVOT)
- inspect and measure the aortic root (at the level of the aortic annulus, sinuses of Valsalva and sinotubular junction)
- inspect and measure the proximal ascending aorta (tilt the probe superiorly to view the mid-ascending aorta)
- look at the descending aorta as it runs behind the LA – this is a useful landmark for assessing a pericardial/pleural effusion
- assess the pericardium and check for any pericardial (or pleural) effusion .
(Houghton,2014) .

2.4.2.5.1.1.2 Use M-mode:

- with the cursor placed at the level of the aortic valve cusp tips to measure aortic root diameter (at end-diastole) and LA diameter (at end-systole), as an alternative to 2D measurement
- with the cursor placed at the level of the mitral valve leaflet tips to measure

mitral valve E point septal separation (the distance between the E point of the anterior mitral leaflet and the septum).

- with the cursor placed just distal to the mitral valve leaflet tips to measure LV wall thickness and cavity size, as an alternative to 2D measurement.

(Houghton,2014) .

2.4.2.5.1.1.3 Use colour Doppler to:

- assess the aortic valve for stenosis or regurgitation (if regurgitation is present, measure the vena contracta and the width of the jet in relation to the diameter of the LVOT).
- assess the mitral valve for stenosis or regurgitation (if regurgitation is present, measure the vena contracta).
- check for flow acceleration in the LVOT in association with septal hypertrophy
- check the integrity of the interventricular septum (IVS).

(Houghton,2014) .

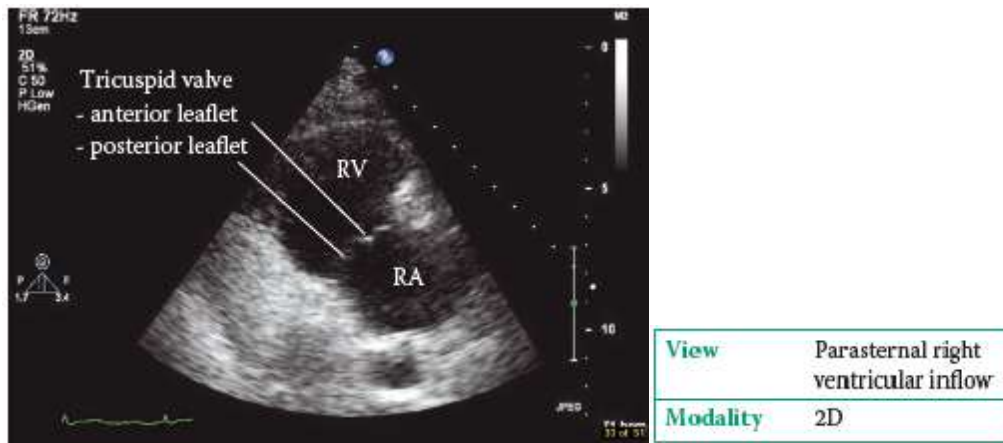
2.4.2.5.1.2 Parasternal right ventricular inflow view:

This view is obtained from the left parasternal window by tilting the probe so that it points more medially and towards the patient's right hip, bringing the right atrium (RA), tricuspid valve and RV into view (**Figure 2.47**) *(Houghton,2014) .*

2.4.2.5.1.2.1 Use 2D to

- assess size and function of the RV
- inspect the structure of the RA. In this view it may be possible to see the coronary sinus and the inferior and superior vena cava as they join the RA. There may be a prominent Eustachian valve at the junction with the inferior vena cava (IVC)
- assess the structure and mobility of the tricuspid valve (the two leaflets seen are the anterior and posterior leaflets).

- Use colour Doppler to examine tricuspid valve inflow and check for regurgitation.
- Use continuous wave (CW) Doppler to assess tricuspid valve function. If tricuspid regurgitation is present, measure the maximum velocity to assess RV systolic pressure.



(Figure 2.47) Normal right ventricular inflow view (RA = right atrium; RV = right ventricle) *From Making Sense of Echocardiography (Houghton,2014)* .

2.4.2.5.1.3 Parasternal right ventricular outflow view :

This view is obtained from the left parasternal window by tilting the probe so that it points more laterally and towards the patient's left shoulder, bringing the RVOT, pulmonary valve and pulmonary artery into view (**Figure 2.48**) . It may be possible to see the pulmonary artery bifurcation *From Making Sense of Echocardiography (Houghton,2014)* .

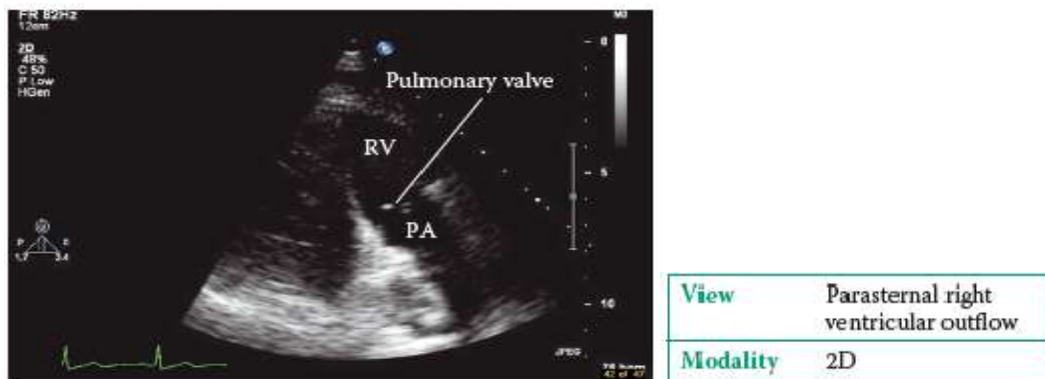
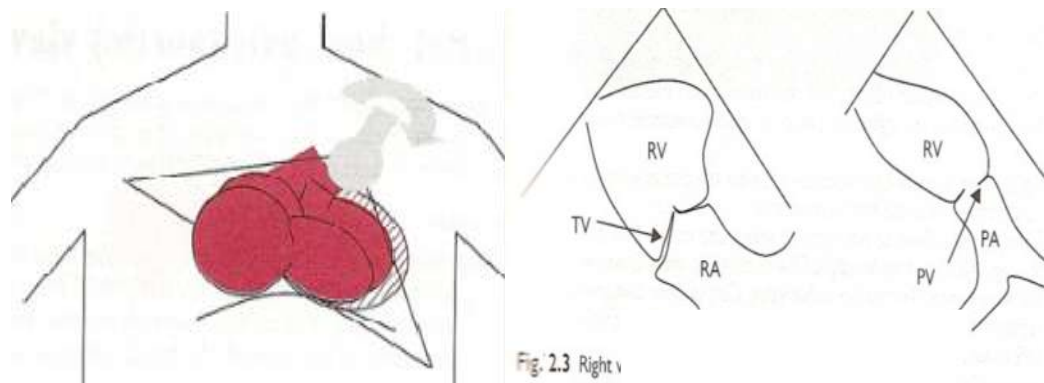
2.4.2.5.1.3.1 Use 2D to:

- assess the structure of the RVOT and main pulmonary artery; check for the presence of thrombus (pulmonary embolus)
- measure RVOT diameter at pulmonary valve annulus level (known as RVOT2,)
- assess the structure and mobility of the pulmonary valve.
- Use colour Doppler to examine flow in the RVOT and pulmonary artery, and to

assess the pulmonary valve for stenosis or regurgitation. It may be possible to detect the abnormal jet of a persistent ductus arteriosus by examining the pulmonary artery with colour Doppler in this view (*Houghton,2014*) .

2.4.2.5.1.3.2 Use CW and PW Doppler to:

- assess flow in the RVOT and pulmonary artery.
- assess the pulmonary valve for stenosis or regurgitation.



(Figure 2.48) Normal right ventricular outflow view (PA = pulmonary artery; RV = right ventricle) (*Houghton,2014*) and (*Paul and Andrew,2012*) .

2.4.2.5.1.4 Parasternal short axis view :

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

There are actually four SAX views, obtained by sweeping the probe along the axis of the heart from the level of the aortic valve down to the apex (*Houghton,2014*).

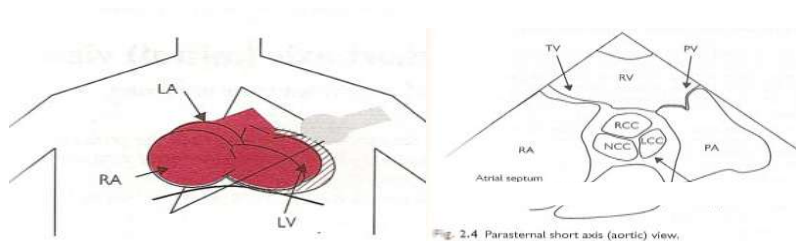
2.4.2.5.1.4.1 The standard SAX views are:

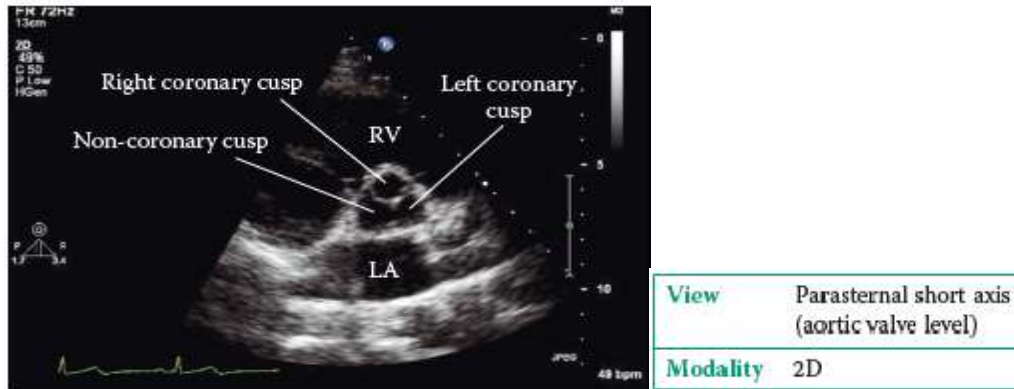
- aortic valve level (sometimes called the RV ‘outflow’ level)
- mitral valve level (also known as the ‘base’)
- papillary muscle level (also known as ‘mid’)
- apical level.

(*Houghton,2014*).

2.4.2.5.1.4.1.1 At the aortic valve level (Figure 2.49) Use 2D to:

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





(Figure 2.49) Normal parasternal short axis view (aortic valve level(LA =Left Atrium ; RV=Right Ventricle) (Houghton,2014) and (Paul and Andrew,2012) .

You may be able to inspect the origins of the left main stem and right coronary artery arising just above the aortic valve cusps (Houghton,2014) .

2.4.2.5.1.4.1.2 Use colour Doppler to:

- examine the aortic valve for regurgitation
- check the integrity of the interatrial septum
- examine tricuspid valve inflow and check for regurgitation
- examine the pulmonary valve for stenosis or regurgitation.

It may be possible to detect the abnormal jet of a ventricular septal defect (VSD) or a persistent ductus arteriosus with colour Doppler in this view.

- Use PW Doppler to assess flow in the RVOT, just proximal to the pulmonary valve.

(Houghton,2014) .

2.4.2.5.1.4.1.3 CW Doppler to:

- assess the pulmonary valve for stenosis or regurgitation. If pulmonary regurgitation is present, assess pulmonary artery diastolic pressure
- assess tricuspid valve function. If tricuspid regurgitation is present, assess RV systolic pressure (Houghton,2014) .

2.4.2.5.1.4.1.4 At the mitral valve level (Figure 2.50) Use 2D to:

- inspect the MV leaflets, mitral annulus and subvalvular apparatus. The anterior and posterior leaflets are visible as is the classical mitral valve orifice, which can be plain metered to measure orifice area
- assess the mobility of the mitral valve leaflets
- assess LV radial function and look for any regional wall motion abnormalities at the basal level
- assess RV size and function.

(Houghton,2014) .

2.4.2.5.1.4.1.5 Use colour Doppler to:

- examine mitral valve inflow
- check for mitral regurgitation and identify precisely where it occurs in relation to the leaflet scallops
- check the integrity of the IVS.

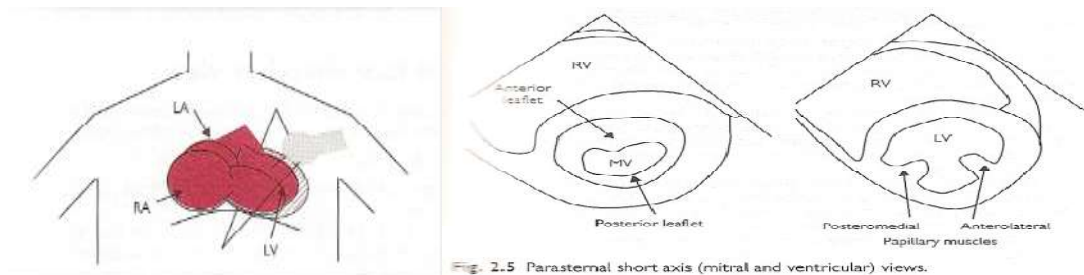
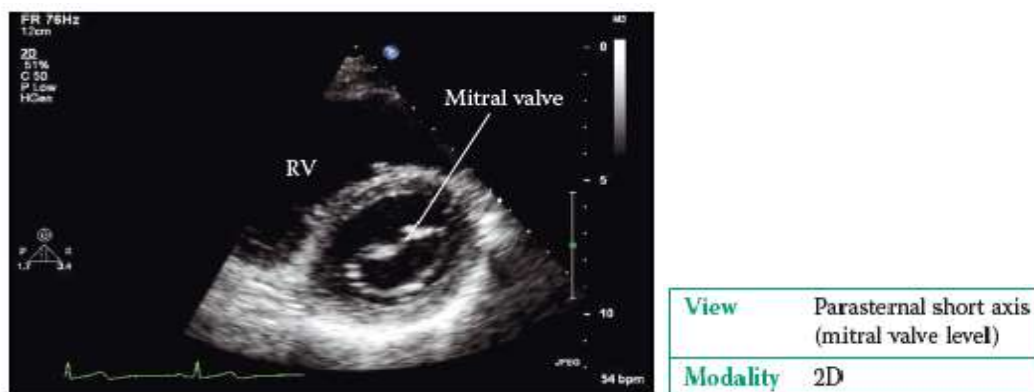


Fig. 2.5 Parasternal short axis (mitral and ventricular) views.



(Figure 2.50) Normal parasternal short axis (Mitral valve level) (RV = Right Ventricle)

(Houghton,2014) and (Paul and Andrew,2012) .

2.4.2.5.1.4.1.6 At the papillary muscle level (Figure 2.51) Use 2D to:

- assess the structure of the posteromedial and anterolateral papillary muscles
- measure LV wall thickness
- assess LV radial function and look for any regional wall motion abnormalities at the mid-ventricle level
- assess RV size and function

(Houghton,2014) .

2.4.2.5.1.4.1.7 Use colour Doppler to :

- check the integrity of the IVS.

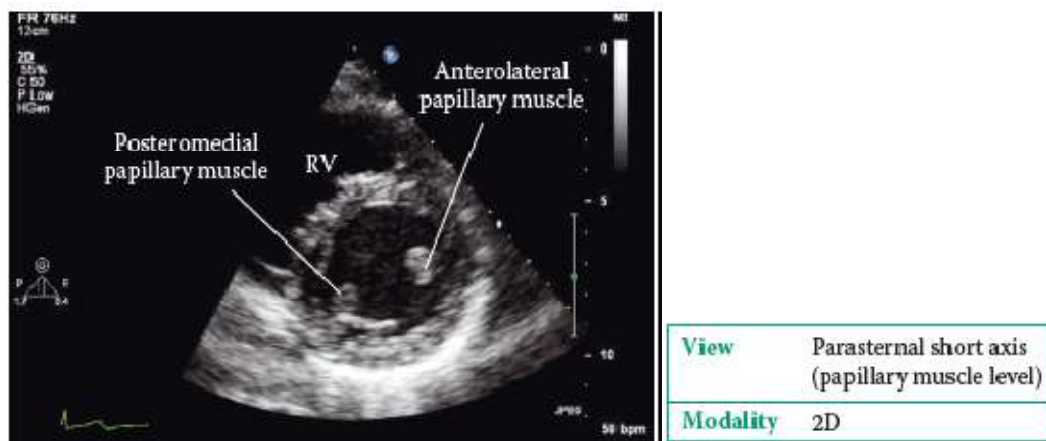
2.4.2.5.1.4.1.8 Finally, sweep the probe down towards the apical level and Use 2D to:

- assess LV radial function and look for any regional wall motion abnormalities at the apical level
- assess RV size and function.

(Houghton,2014) .

2.4.2.5.1.4.1.9 Use colour Doppler to :

- check the integrity of the IVS.



(Figure 2. 51) Normal parasternal short axis view (papillary muscle level) (RV = Right ventricle) (Houghton,2014) .

2.4.2.5.2 Right parasternal window:

The right parasternal window is ‘optional’ but can be useful for assessing flow in the ascending aorta. With the patient lying on their right-hand side, place the probe to the right of the sternum in the third intercostal space (some adjustment may be required, as with the left parasternal window) and angle the probe downwards and pointing towards the heart.

It is a challenging view, but it may be possible to visualize the ascending aorta and assess colour Doppler within it. This view is most useful for undertaking CW Doppler assessment of the aortic valve, particularly with a standalone pencil probe (*Houghton,2014*).

2.4.2.5.2.1 Apical window :

The apical window is located at the LV apex. This is normally in the mid-clavicular line and the fifth intercostal space, but may be displaced downwards and to the left if the heart is enlarged. From the apical window a number of views can be obtained (*Houghton,2014*).

2.4.2.5.2.1.1 Apical 4-chamber view :

To obtain this view, place the probe in the apical position with the ‘dot’ pointing towards the patient’s left. For an optimal view, aim to position the probe exactly at the apex to avoid distortion or foreshortening of the cardiac structures. The interatrial and interventricular septa should be in line with the probe and lie vertically on the screen (**Figure 2.52**) (*Houghton,2014*).

2.4.2.5.2.1.1.1 In this view Use 2D to:

- measure LV cavity size and wall thickness
- assess LV radial and longitudinal function, looking carefully for any regional wall motion abnormalities (inferoseptal and anterolateral wall)
- assess structure and mobility of the mitral valve – in this view, the P1, A2 and A3 segments are visible

- inspect the appearance of the LA and measure its size at end-systole
- assess atrial septal mobility
- assess the pericardium and check for any pericardial (or pleural) effusion.
- with the cursor placed at the lateral tricuspid annulus, measure tricuspid annular plane systolic excursion (TAPSE)
- with the cursor placed at the lateral mitral annulus, measure mitral annular *(Houghton,2014)* .

2.4.2.5.2.1.1.2 Use colour Doppler to:

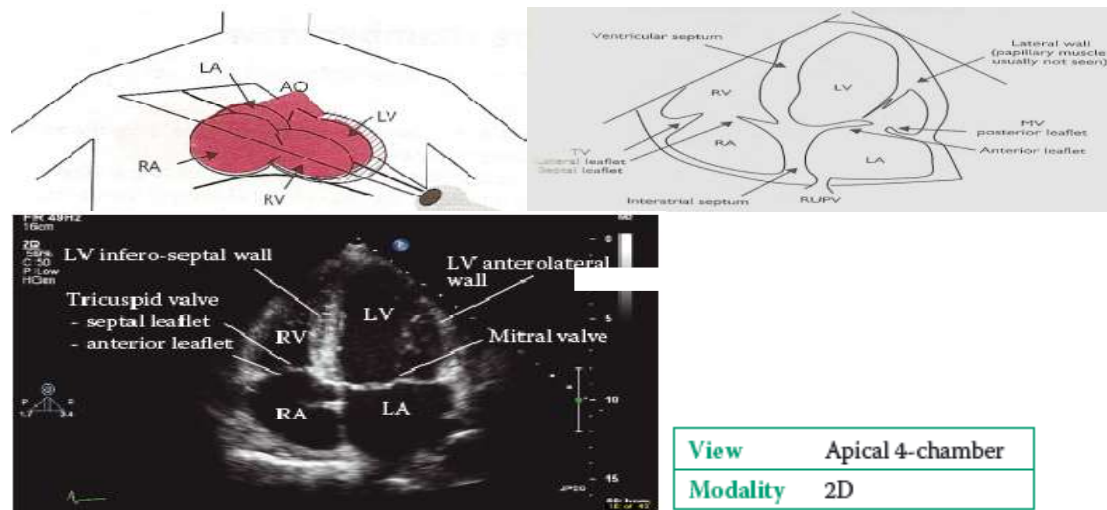
- assess the mitral valve for stenosis or regurgitation
- assess flow in the pulmonary veins (the right upper pulmonary vein is usually the easiest to locate)
- check the integrity of the interatrial and ventricular septa *(Houghton,2014)* .

2.4.2.5.2.1.1.3 Use PW Doppler to:

- assess LV inflow at the level of the mitral valve tips.
- assess flow in the pulmonary veins *(Houghton,2014)* and *(Paul and Andrew,2012)* .

2.4.2.5.2.1.1.4 Use CW Doppler to:

- assess mitral stenosis or regurgitation
- if mitral regurgitation is present, assess LV systolic function by measuring dP/dt .
- Use tissue Doppler imaging of the mitral annulus to:
- assess LV diastolic function *(Houghton,2014)* .



(Figure 2.52) Normal apical 4-chamber view (LA=Left atrium; LV= Left ventricle ; RV= Right ventricle) (Houghton,2014) and (Paul and Andrew,2012) .

2.4.2.5.2.1.1.5 Modified apical 4-chamber view :

To obtain an optimal view of the right heart, it is best to slightly adjust the standard apical 4-chamber view to centre the right heart on the screen and to ensure that there is no foreshortening. This is known as the ‘modified’ apical 4-chamber view (Houghton,2014) .

2.4.2.5.2.1.1.5.1 In this view Use 2D to:

- measure RV cavity size
- assess RV systolic function
- assess structure and mobility of the tricuspid valve – in this view, the anterior and septal tricuspid leaflets are visible
- inspect the appearance of the RA and measure its size at end-systole (Houghton,2014) .

2.4.2.5.2.1.1.5.2 Use colour Doppler to:

- assess the tricuspid valve for stenosis or regurgitation (Houghton,2014) .

2.4.2.5.2.1.1.5.3 Use PW Doppler to:

- assess RV inflow at the level of the tricuspid valve tips (Houghton,2014) .

2.4.2.5.2.1.1.5.4 Use CW Doppler to:

- assess tricuspid stenosis or regurgitation
- if tricuspid regurgitation is present, assess RV systolic pressure by measuring the tricuspid regurgitation Vmax(*Houghton,2014*).

2.4.2.5.2.1.2 Apical 5-chamber view

From the apical 4-chamber view, maintain the same window but angle the probe anteriorly so that the aortic valve and aortic root (the ‘fifth chamber’) come into view (**Figure 2.59**). This view is used principally to assess the LVOT and aortic valve, and it is important to align these with the ultrasound beam so that reliable Doppler traces can be obtained (*Houghton,2014*).

2.4.2.5.2.1.2.1 Use 2D to:

- assess LV cavity size, wall thickness and systolic function
 - inspect the LVOT (any signs of asymmetrical hypertrophy?)
 - assess structure and mobility of the aortic valve
- (*Houghton,2014*).

2.4.2.5.2.1.2.2 Use colour Doppler to:

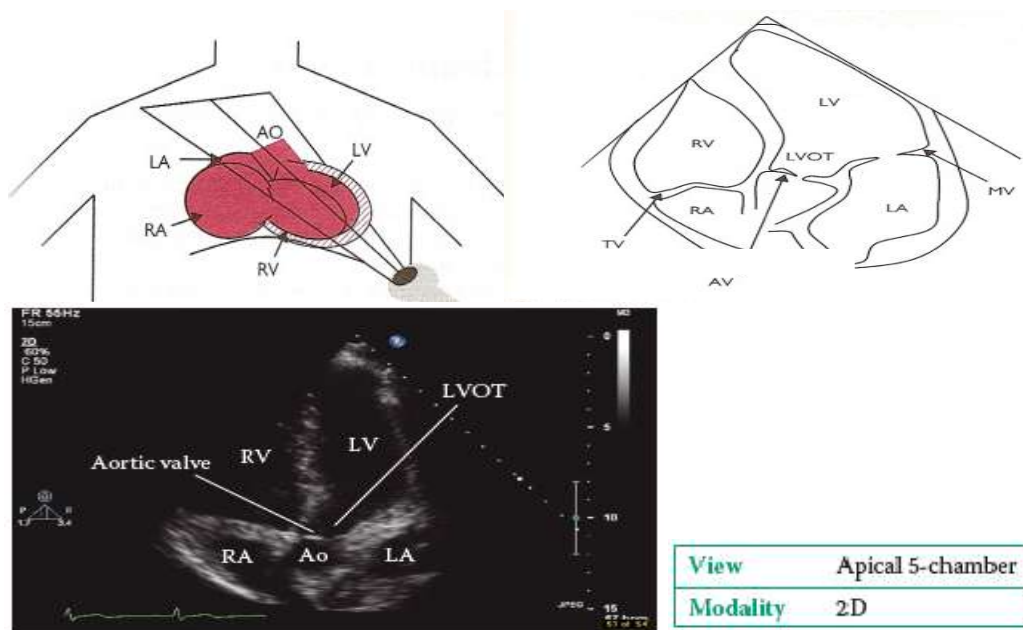
- check for flow acceleration in the LVOT in association with septal hypertrophy
 - assess the aortic valve for regurgitation
 - check for a perimembranous VSD
- (*Houghton,2014*).

2.4.2.5.2.1.2.3 Use PW Doppler to:

- assess flow in the LVOT.
- (*Houghton,2014*).

2.4.2.5.2.1.2.4 Use CW Doppler to:

- assess aortic stenosis or regurgitation
- assess any subvalvular or supra- valvular obstruction
- measure isovolumic relaxation time (IVRT) (*Houghton,2014*).



(Figure 2.53) Normal apical 5-chamber view (AO=aorta; LA=Left atrium; LV=Left Ventricle; LVOT=Lefventricleoutflow tract; RV=Right ventricle (Houghton,2014) .

2.4.2.5.2.1.3 Apical 2-chamber view :

Return to the apical 4-chamber view and maintain the same window but rotate the probe about 60° anticlockwise so that the ‘dot’ points approximately towards the patient’s left shoulder. Stop rotating the probe before the LVOT comes into view, and ensure that the mitral valve is centred in the image (Figure 2.54)(Houghton,2014) .

2.4.2.5.2.1.3.1 Use 2D to:

- measure LV cavity size and wall thickness
- assess LV radial and longitudinal function, looking carefully for any regional wall motion abnormalities (anterior and inferior wall)
- assess structure and mobility of the mitral valve – in this view, the P1, A2 and P3 segments are visible
- inspect the appearance of the LA and measure its size at end-systole (the LA

appendage may be visible as a small ‘pocket’ to the right of the mitral valve, and the coronary sinus may be visible as a circular structure to the left of the mitral valve.

(Houghton,2014) .

2.4.2.5.2.1.3.2 Use colour Doppler to:

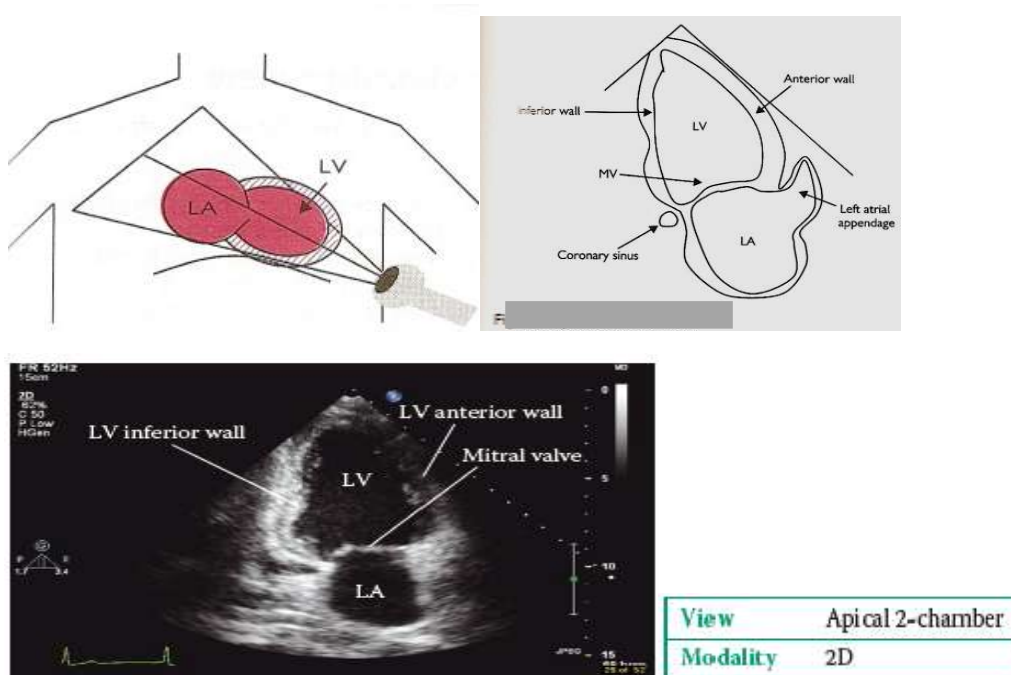
- assess the mitral valve for stenosis or regurgitation (Houghton,2014) .

2.4.2.5.1.3.3 Use PW Doppler to:

- assess LV inflow at the level of the mitral valve tips (Houghton,2014).

2.4.2.5.2.1.3.4 Use CW Doppler to:

- assess mitral stenosis or regurgitation.



(Figure 2.54)Normal apical 2-chamber view (LA=Left atrium; LV= Left ventricle) (Houghton,2014) and (Paul and Andrew,2012) .

2.4.2.5.2.1.4 Apical 3-chamber (long axis) view

From the apical 2-chamber view, maintain the same window but rotate the probe a further 60° anticlockwise so that the ‘dot’ now points approximately towards the patient’s right shoulder. Stop rotating the probe once the LVOT comes

into view, and ensure that the mitral and aortic valves are centred and not foreshortened (**Figure 2.55**). This view is the apical equivalent of the parasternal LAX view (*Houghton,2014*) .

2.4.2.5.2.1.4.1 Use 2D to:

- measure LV cavity size and wall thickness
- assess LV radial and longitudinal function, looking carefully for any regional wall motion abnormalities (anteroseptal and inferolateral (posterior) wall)
- assess the appearance of the LVOT (any signs of asymmetrical hypertrophy?)
- assess structure and mobility of the aortic valve
- assess structure and mobility of the mitral valve – in this view, the A2 and P2 segments are visible
- inspect the appearance of the LA

(*Houghton,2014*) .

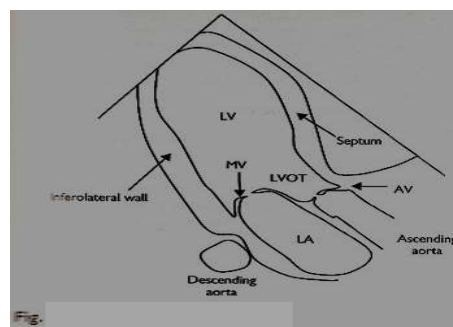
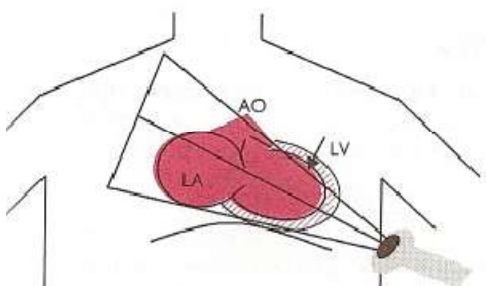
2.4.2.5.2.1.4.2 Use colour Doppler to:

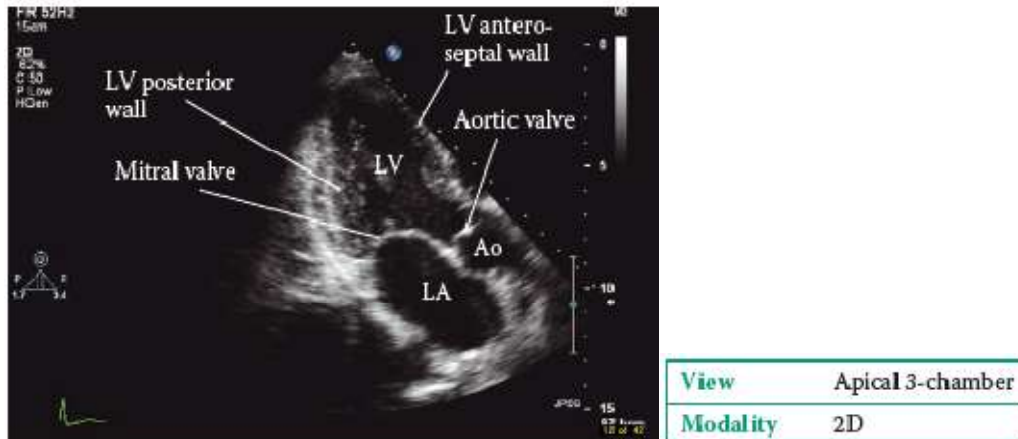
- assess the mitral valve for stenosis or regurgitation
- assess the aortic valve for regurgitation
- check for flow acceleration in the LVOT in association with septal hypertrophy

(*Houghton,2014*) .

2.4.2.5.2.1.4.3 Use PW Doppler to:

- assess LV inflow at the level of the mitral valve tips.
- assess flow in the LVOT.





(Figure 2.55) Normal apical 3-chamber view (AO=aorta; LA=Left atrium ; LV=Left ventricle) (Houghton,2014) and (Paul and Andrew,2012) .

2.4.2.5.2.1.4.5 Use CW Doppler to:

- assess mitral stenosis or regurgitation
- assess aortic stenosis or regurgitation
- assess any subvalvular or supra-ventricular obstruction

(Houghton,2014) .

2.4.2.5.3 Subcostal window

The subcostal window is obtained with the patient lying supine with their arms by their sides. It is important that the abdominal wall is relaxed, and asking the patient to lie with their knees bent can help this. Place the probe just below the xiphisternum and angle it up towards the heart, with the ‘dot’ to the patient’s left. From the subcostal window a number of views can be obtained (Houghton,2014) .

2.4.2.5.3.1 Subcostal long axis view :

To optimize this view, ensure that the interatrial septum is perpendicular to the ultrasound beam (i.e. lies horizontally across the screen) with no foreshortening of the chambers (Figure 2.56) (Houghton,2014) .

2.4.2.5.3.1.1 Use 2D to:

- assess RV dimensions and function
- assess RA dimensions

- assess LV dimensions and function
- assess LA dimensions
- assess the structure of the interatrial septum
- assess the pericardium and check for any pericardial effusion

(Houghton,2014) .

2.4.2.5.3.1.2 Use colour Doppler to:

- check the integrity of interatrial and interventricular septa

(Houghton,2014) .

2.4.2.5.3.1.3 Use CW and PW Doppler to:

- assess flow across any septal defect

(Houghton,2014) .

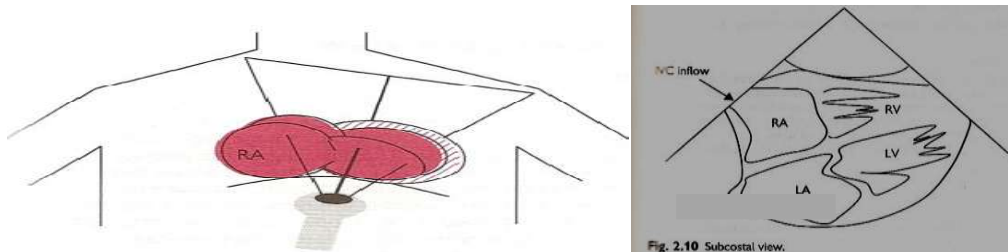
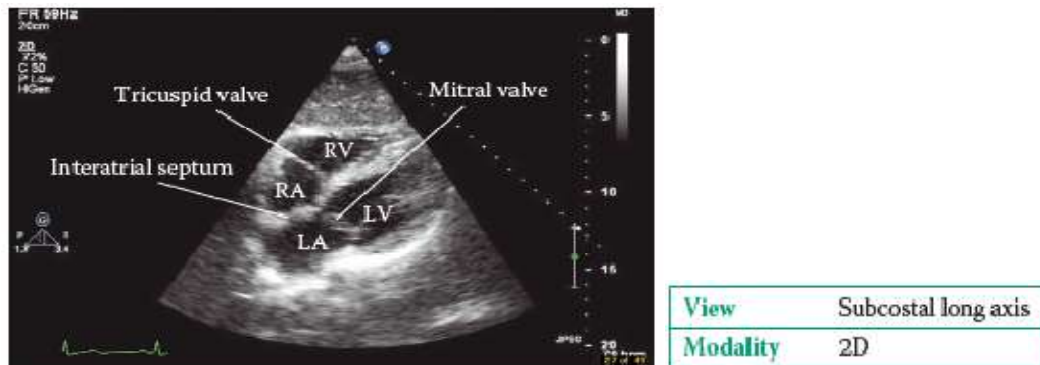


Fig. 2.10 Subcostal view.



(Figure2.56) Normal subcostal long axis view(LA=Left atrium; LV=Left ventricle; RA=Right atrium; RV=Right ventricle) (Houghton,2014) and (Paul and Andrew,2012) .

2.4.2.5.3.2 Subcostal short axis view:

Keeping the probe in the subcostal window rotate the probe 90° to obtain a SAX view (Figure 2.63) (Houghton,2014) .

2.4.2.5.3.2.1 Use 2D to:

- assess IVC dimensions (check for respiratory variation by taking measurements in inspiration and expiration)
- assess hepatic veins (congested?)

(Houghton,2014) .

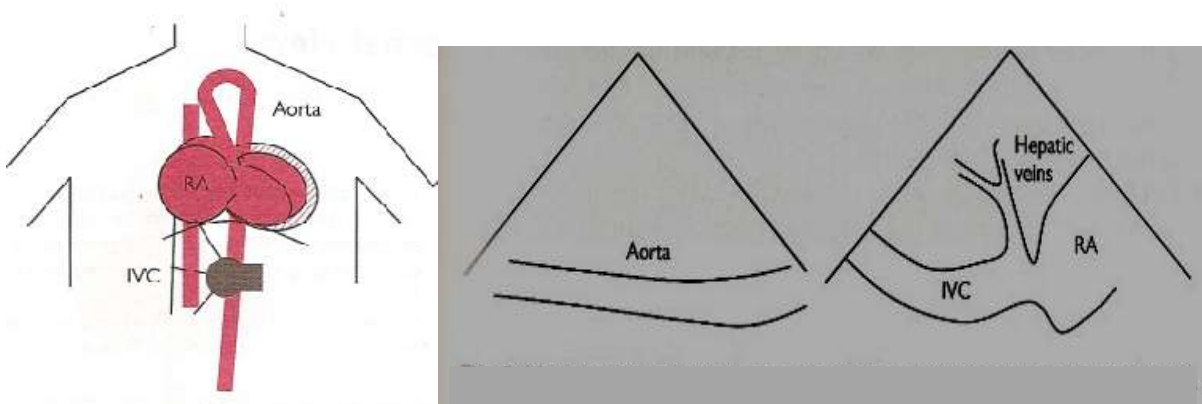
2.4.2.5.3.2.2 Optionally, you can also use 2D to:

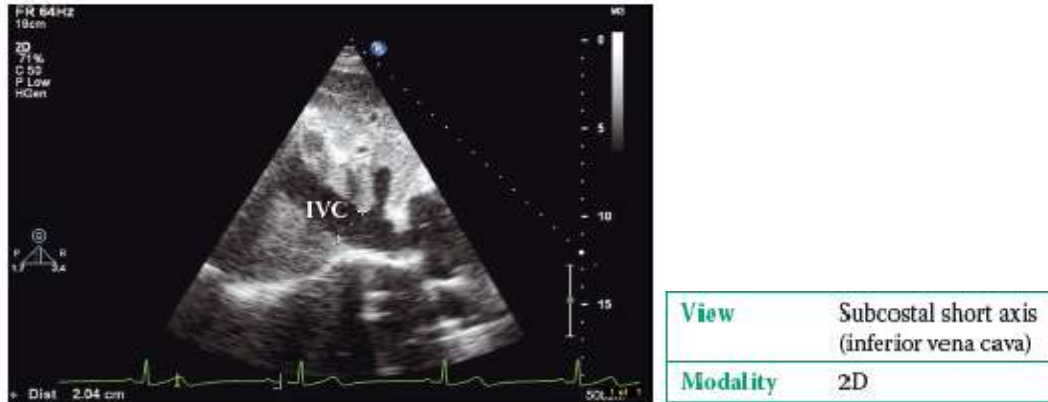
- inspect the interatrial septum
- inspect the tricuspid valve
- inspect the RVOT
- inspect the pulmonary valve
- inspect the pulmonary arteries
- inspect the abdominal aorta (modified view)

(Houghton,2014) .

2.4.2.5.3.2.3 Use M-mode to:

- assess IVC dimensions (check for respiratory variation by taking measurements in inspiration and expiration) *(Houghton,2014)* .





(Figure 2.57) Normal subcostal short axis (Inferior vena cava (IVC)) view (Houghton,2014) and (Paul and Andrew,2012) .

2.4.2.5.3.2.4 Use colour Doppler to:

- assess flow in the IVC and hepatic veins
- check the integrity of the interatrial septum

(Houghton,2014) .

2.4.2.5.3.2.5 Optionally, you can use PW Doppler to:

- assess flow in the hepatic veins
- assess flow in the descending aorta

(Houghton,2014).

2.4.2.5.4 Suprasternal window :

The suprasternal window is located in the suprasternal notch. Ask the patient to lie supine and to raise their chin. Place the probe in the notch and angle it downwards into the chest. Be mindful that some patients find this uncomfortable. This view shows the aortic arch in LAX (Figure 2.58). A similar view can, if needed, be obtained from the right supraclavicular position (Houghton,2014) .

2.4.2.5.4.1 Aorta view :

2.4.2.5.4.1.1 Use 2D to :

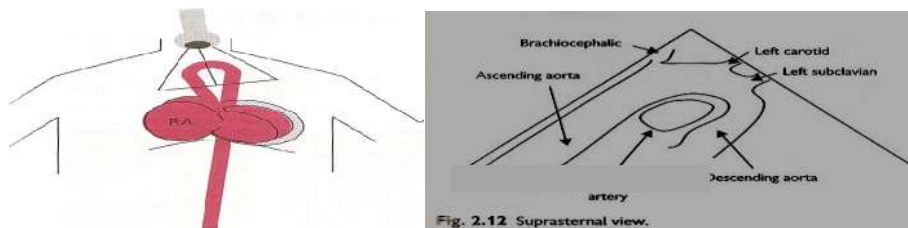
assess the appearances and dimensions of the aortic arch (Houghton,2014) .

2.4.2.5.4.1.2 Use colour Doppler to :

assess flow in the aorta, looking in particular for evidence of coarctation or persistent ductus arteriosus (*Houghton,2014*).

2.4.2.5.4.1.3 Use CW Doppler to:

assess flow in the descending aorta in the presence of a coarctation (it may be better to use a non-imaging ‘pencil’ probe if alignment is difficult using an imaging probe) (*Houghton,2014*) .



(Figure 2.58) Normal suprasternal aorta view (*Houghton,2014*) and (*Paul and Andrew,2012*) .

2.4.2.6 Left ventricle

2.4.2.6.1 Normal anatomy :

The left ventricle is a cavity with muscle walls that contains the papillary muscles and their chordal attachments. The anatomical characteristics of the size and thickness can vary significantly with pathology ,and many cardiac and

systemic processes are associated with cardiac dilation or hypertrophy (*Paul and Andrew,2012*).

2.4.2.6.2 Normal findings:

2.4.2.6.2.1 Views

The left ventricle is seen in virtually all windows. The minimal views are parasternal long and short axis and the apical 4-2 and 3-chamber views (*Paul and Andrew,2012*).

2.4.2.6.2.2 Findings:

Parasternal long axis, The basal and mid-segments of the septum and posterior wall (in some publications it is also referred to as the inferolateral wall) are visible. This view is used for linear measures of wall thickness and cavity dimensions. The left ventricular outflow tract can also be assessed. Parasternal short axis.

By angling the probe back and forth, the whole of the left ventricle can be scanned in cross-section. The key ventricle views are mid-ventricle (mid-papillary) and apical. The mid-ventricle level is used for linear and area measures of wall and cavity. Regional wall motion abnormalities can also be assessed in (in clockwise order septum, anterior, lateral and inferior walls.

Apical 4-chamber. Provides best views of apex, septum (on left), and lateral (on right) walls for regional assessment. Suitable for tracing ventricular area and left ventricular length.

Apical 2-chamber. Focuses on inferior (on left) and anterior walls (on right).

Apical 3-chamber. The parasternal long axis view but from the apex looks at posterior (inferolateral) wall and septum. Subcostal provides an alternative view of the left ventricle but is not essential (*Paul and Andrew,2012*).

2.4.2.6.3 Left ventricular assessment:

Accurate left ventricular assessment (diameters, volumes, wall thickness, mass, and function) is critical in practice. Measurements can be altered by virtually all cardiovascular pathologies. The most common indication for echocardiography is evaluation of left ventricular function with ejection fraction being the most sought parameter. Assessments are frequently visually estimated but there is significant inter observer variability and dependence on interpreter skill. Quantitative measures are recommended to ensure diagnostic accuracy (*Paul and Andrew,2012*).

2.4.2.6.3.1 Assessment

Start with an overview of the ventricle in all views (parasternal and apical) and gather an impression of appearance, size, and function.

Comment on obvious structural changes:

- Ventricular shape, aneurysms, wall thinning, wall hypertrophy, wall character (speckling .etc.).

Report quantitative measures of size and a general summary: normal , mild, moderate , or server dilatation : normal , mild, moderate , or sever

hypertrophy. If hypertrophy , give an idea of the paternbased on appearance and relative wall thickness. I.e. eccentric, concentric, asymmetric (septal, apical) .

2D and M-mode quantification of left ventricular size and mass have been well validated but both have advantages and disadvantages.

M-mode measures in parasternal views are widely used. They are very dependent on M-mode alignment and take no account of left ventricular shape or regional wall motion abnormalities. The alignment problem is reduced with 2D guided or direct 2D-measures.

In general, left ventricular shape changes are best accounted for by using the volumetric biplane Simpson`s method for volumes and the truncated ellipsoid

method for left ventricular mass. These methods should therefore be used to provide accurate assessment of left ventricular volume and mass, respectively.

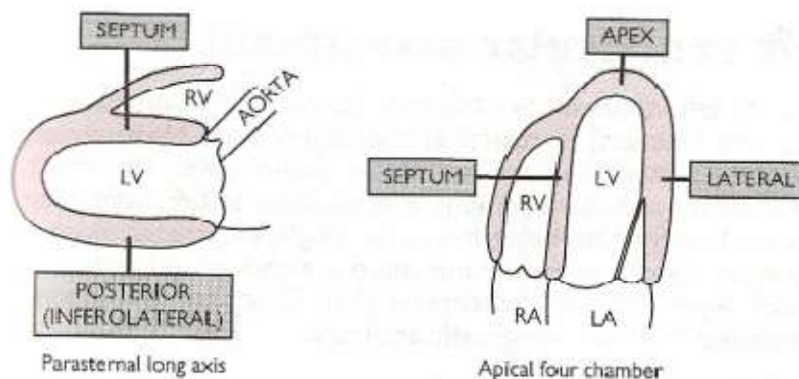
Reference ranges are dependent on gender and body habitus . Ideally, height and weight should be recorded and body surface area used to correct left ventricular dimensions.

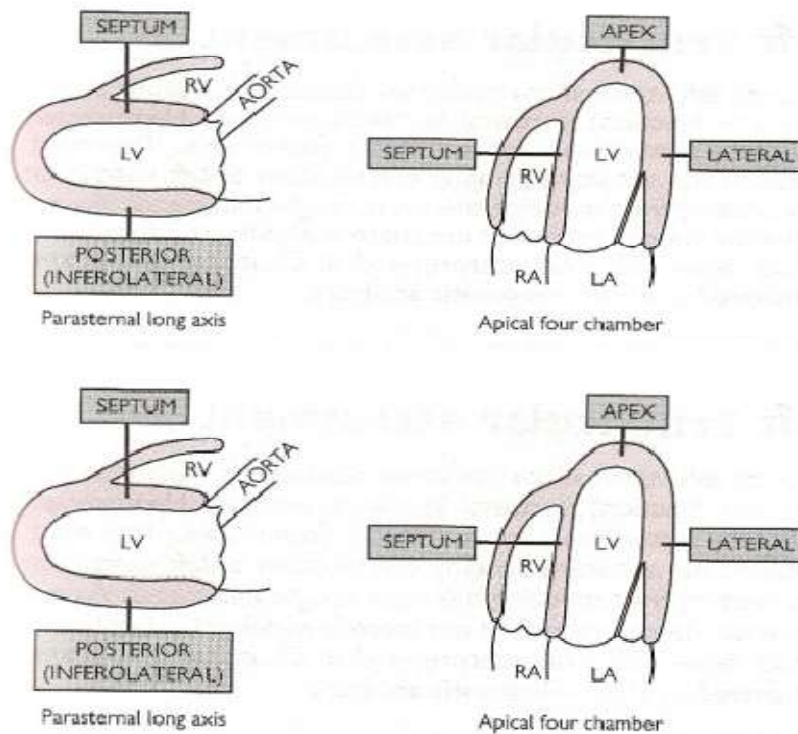
Using the measures of left ventricular size, report a quantitative assessment of systolic function or mild, moderate, or severe systolic dysfunction .

From apical and parasternal views look at changes in regional wall motion (normal, hypokinesis ,akinesis, dyskinesis, aneurysmal).Report abnormalities .

When relevant , assess and report left ventricular diastolic function from changes in mitral valve inflow and tissue Doppler imaging .

Finally , ensure you have reported fully pathologies that might relate to the changes you have identified in the left ventricle (e.g. valve disease) (*Paul and Andrew,2012*) .





(Figure 2.59) Key views to assess the left ventricle with walls marked (*Paul and Andrew,2012*).

2.4.2.6.4 Left ventricular size:

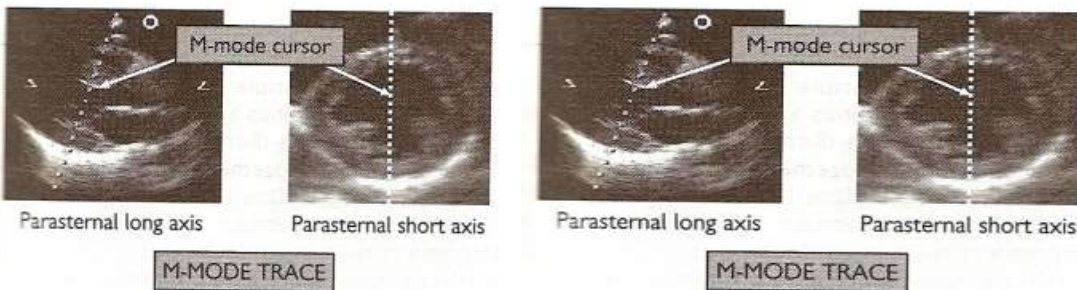
Because it is difficult to quantify a 3D structure using 2D image, the techniques that have developed rely on measuring the ventricle in standard places. The measures are then reported directly (linear methods) or used in mathematical equations to model on assumed shape for the ventricle (volumetric measures). In principle, the more measures of the left ventricle in the more planes the more accurate the assessment.

Conversely, the fewer measures the more assumptions have to be made and the more likely it is that regional pathology is overlooked. Sometimes - such as in a normal heart-simple linear measures are adequate .However, if there is pathology accuracy is required (*Paul and Andrew,2012*).

2.4.2.6.4.1 Linear measures:

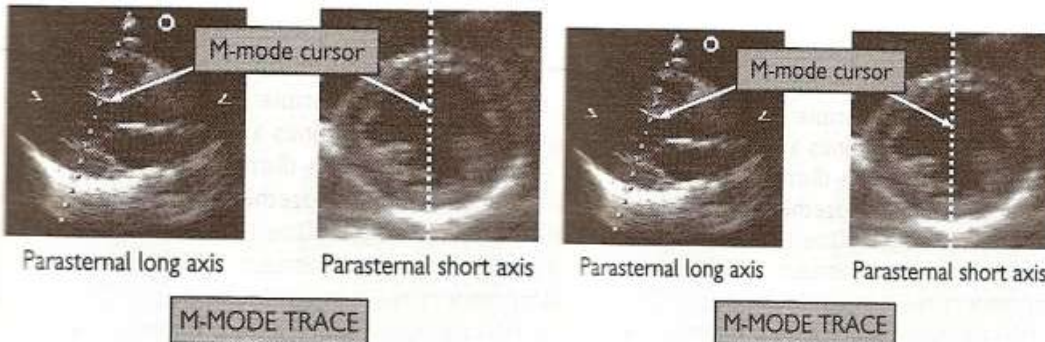
2.4.2.6.4.1.1 M-mode

This is based on change in size in a single plane at the mid-ventricle level in a parasternal view (**Figure 2.60**). Recent guidelines suggest this should be a parasternal short axis view (*Paul and Andrew,2012*).



(Figure 2.60) Measurement using M-mode in parasternal views (*Paul and Andrew,2012*).

2.4.2.6.4.1.2 Two dimension -imaging:



(Figure 2.61) Examples of 2D measures in parasternal views (*Paul and Andrew,2012*).

2.4.2.6.4.2 Volumetric measures :

2.4.2.6.4.2.1 Simpson`s method:

Simpson`s method is based on the principle of slicing the left ventricle from apex down to mitral valve annuls into a series of discs. The volume of each disc is then calculated (using the diameter and thickness of each slice). All the disc volumes are added together to provide the total left ventricular volume. If done in

single plane (based on apical 4-chamber view) it is assumed the left ventricle is circular at each level .

Accuracy is improved by using diameters in two perpendicular planes (biplane-apical 4-and 2-chamber) so that the disc surface area is more precisely defined although this can be done `by hand` by measuring the diameter at multiple levels, in reality , you trace the outline of the ventricle and the machine or off-line software automatically calculates the volume. In the apical 4-chamber view obtain a clear image of the left ventricular cavity with a clear endocardial border.

Record a loop and scroll through to find the end-diastolic image (usually just before the aortic valve opens or on the R-wave of the ECG). This image should have the largest left ventricular volume. Trace around the endocardial border going from one side of the mitral valve annulus to the other and joining the two ends with a straight line. Record the left ventricular end-diastolic volume.

Measure the length of the left ventricle from apex to middle of mitral valve. Depending on the machine. identification of the apex may be automatic after tracing the border. Record left ventricular long axis. Scroll through the loop again and find the smallest left ventricular volume at end-systole (usually just before the mitral valve opens or on the T-wave of the ECG). Trace around the endocardialborder , as before , and record left ventricular end-systolic volume.

The method above will provide single plane measures of left ventricular volumes. For biplane measures repeat the process for diastolic and systolic images using an optimized apical 2-chamber view (*Paul and Andrew,2012*) .

2.5 Previous studies

In study performed by **Manickavasagam in 2009 reported** “Patients with HF may present either with reduced or normal ejection fraction”.

The another study by **Sharief et al , 2016 said** “This study showed the probability of sign of HF in low, high and normal EF in uncontrolled HTN with normal LV and LV function and also deference grades of them.

The study of **Sharief et al , 2015 reported** “the probability of sign of HF in low, high and normal EF in uncontrolled HTN with normal LV and LV function and also deference grades of them”.

The study performed by William , 2008 that reported “more than three quarters of patients with heart failure (HF) have antecedent hypertension. Hypertension appears to play an especially important role in HF associated with a preserved ejection fraction (EF) >0.50 (HFPEF)”.

The study of Hossein et al , 2014 reported “There is a U shape relationship between LVEF and outcomes. In both patients with or without HF , Those with an LVEF $\geq 70\%$ have higher mortality and hospitalization rates than those with normal LVEF even after adjustment for comorbidities”.

The other study from **Maeder&David, 2009 said** “HFNEF, used as a term to describe a condition associated with HF symptoms and normal LVEF, and without obvious explanation for the symptoms (e.g., coronary artery disease, valvular heart disease), is typically associated with concentric LV hypertrophy or concentric LV remodeling, increased left atrial size, and LV diastolic dysfunction “

The a novel study by American Heart Association (AHA) , 2015 reported “HCM also can develop over time because of high blood pressure or aging”.

The another study by Cleveland Clinic , 2016 reported "Heart failure affects about 2.5 million women in the United States. Women tend to develop congestive heart failure at an older age than men "

Anne et al in 2006 they reported "In the general population of Dallas County, women had higher LVEF than did men, reflecting a higher stroke volume for a given EDV".

Libhaber et al , 2013 who said "inappropriate LVH is strongly and inversely related to variations in ejection fraction independent of and more closely than LVM or LVMI in a community sample of black African ancestry".

The another study by **Gregory , 2001**who said " Indeed, LVH is probably the most visible manifestation of hypertensive target organ damage".

Jeffrey , 2004said "Compensatory" hypertrophy may prolong the time to symptom development in patients with hypertension and valvular heart diseases.

Myoclinic , 2015reported "Left ventricular hypertrophy is more common in people who have uncontrolled high blood pressure. But no matter what your blood pressure is, developing left ventricular hypertrophy puts you at higher risk for a heart attack and stroke. Treating high blood pressure can help ease your symptoms and may reverse left ventricular hypertrophy".

Also the study of **Cuspidi et al , 2012** said "LVH is a highly prevalent organ damage in essential hypertensive, particularly in the elderly"

Also the **Richard et al , 1987** said "All published studies have reported that left ventricular hypertrophy is more closely related to blood pressure recorded in the patient's natural setting during normal activity or exercise"

The other study by **Thrainsdottir et al , 2003** said "We conclude that the presence of LVH and its appearance is associated with age and increased blood pressure amongst both genders.

The study performed by **Nikitin et al , 2003** that result with their report “Left atrial conduit function deteriorates with age while reservoir and pump function are maintained ”.Also By **Cleveland clinic , 1995** in a report reported “There is ongoing debate about whether some medications for high blood pressure can cause LVH to improve”

The study performed by AHA, 2015 which reported echocardiographic LA enlargement is commonly found in hypertensive patients with electrocardiographic LV hypertrophy. In such patients, LA enlargement is particularly prevalent in older and more obese patients, as well as in women and patients with eccentric LV geometry, independent of the degree of LV hypertrophy or the presence of additional atrial fibrillation and mitral valve regurgitation.

The a novel study by **Stojanov et al ,2012** reported “Women had higher left atrial index (Lai) due to smaller body surface area.

The study performed by **Avdićetal ,2007**said“The number of patients with LVH was significantly higher and they were significantly older, with more women patients. Patients with LVH have higher LV mass compare to patients without LVH and longer hypertension endurance.

The study of **Jakovljevic et al , 2010** reported left ventricular hypertrophy was more often present in hypertensive men than women .

The another study **Pasquale Palmiero, 2014** reported “ Doppler echocardiography is the best tool for early LVDD diagnosis.

The study of **Mooiet al ,** report “In summary,older age, poorer blood pressure control, presence of left ventricular hypertrophy ,central obesity, and plasma glucose levels were significantly associated with higher risk of LVDD”.

The study performed by **Brunotteet al in 2003**in **Journal of Human Hypertension (JHH)** who reported “Heart failure in many patients is due to left

Ventricular Diastolic Dysfunction (LVDD), but little is known about its prevalence among hypertensive adults, especially in the primary care setting”.

The study of **Palmiero et al , 2014**said“LV diastolic function is influenced .There is a strong relationship between arterial stiffness and the diastolic properties of the left ventricle.

The performed by **Zhao et al in Jan 2014** reported “The prevalence of left ventricular diastolic dysfunction (LVDD) sharply increases in women after menopause and may lead to heart failure.

This study a novel by**Baloch et al , 2010 reported** “ LVDD is a frequent finding in patients with left ventricular hypertrophy . It is more frequent in female patients with advancing age and smaller body surface area”.

The study of **Simone et al ,2011**said “Obesity influences left ventricular geometry substantially more in women than in men possibly due to biological factors specifically associated with female adiposity.

The study of **Ogah, 2012** which said ” The prevalence is similar in men and women. Awareness, treatment and control of hypertension were generally low with attendant high burden of hypertension related complications.

Also the study of **Avdićet al ,2007 reported** “In all hypertensive patients all mentioned left ventricular diastolic function parameters have to be determined, particularly IVRT duration for left ventricular diastolic function detection in all hypertensive patients with or without LVH and in case of co-joined atrial fibrillation.

Cheng et al , 2014said“In our sample of individuals with hypertension and diastolic dysfunction, older compared to younger adults experienced less improvement in diastolic function in response to similar reductions in SBP.

Adamu et al , 2010 said “The study has shown that LV diastolic dysfunction is common in newly diagnosed Nigerians with systemic hypertension, and effort should be made to screen for them routinely.LV diastolic dysfunction occurred in hypertensives without LVH, hence its early detection may lead to additional risk stratification and may guide the choice of antihypertensive drugs(*Adamu et al , 2010*).

Masugata, 2011 said “Among hypertensive patients with LVH, those with concentric LVH may, therefore, have more severe LV diastolic dysfunction than those with eccentric LVH even if their LVMI, which reflect the degree of LVH, are similar (*Masugata,2011*).

Algazouly,2014 reported “Using echo cardiograph the effects of hypertension on cardiac character and ejection fraction (EF) during systole and diastole was studied and the relation was found to be significant(*Algazouly,2014*).

Chapter Three

Materials and Methods

Chapter Three

Materials and Methods

3.1. Type of the study :

It is a cross sectional hospital based study deals with 200 patients (100 male, 100 female), all of them had primary or essential hypertension for more than 5 years, Blood pressure, systolic blood pressure should be more than 120 mm Hg, diastolic pressure should be more than 80 mm Hg and all these patients were undergo echocardiography study to identify the common changes in heart chambers due to HTN.

Consents were taken from all patients included in this study, after explaining the whole procedures.

3.2 Area and duration of the study:

The study was carried in the Kingdom of Saudi Arabia (King AbdulAziz specialist Hospital- Al-Taif (KAASH) during 39 months from Nov 2012-March 2015.

3.3 Material :

In this study the ultrasound machine used is MyLab 50 which is Esaote's mid-range shared-service ultrasound machine. The MyLab 50 includes high end imaging technologies such as stress echo, TEE, 3D/4D obstetrics, strain imaging, and is compatible with more than 30 transducers for nearly every study, including Esaote's ergonomic apple probes. Esaote ultrasound systems are particularly good at cardiac, vascular.

3.4 Method:

3.4.1 Echocardiography used

The gross anatomy of the heart can be evaluated by two-dimensional Transthoracic (TTE) echocardiography in the para-sternal, apical, suprasternal

and subcostal position. The standardized planes used are long axis, short axis and four-chamber.

3.4.2 Technique used

TTE by using the long axis view is obtained by placing the ultrasound transducer in the left apicosternal position and provides detailed images of the left ventricle, aorta, left atrium, and mitral and aortic valves. Angling the beam towards the right also allows assessment of the right atrium, right ventricle and tricuspid valves. Rotating the transducer by 90° in the clockwise direction produces the short-axis view, which allows assessment of the left ventricle, papillary muscles, chordae tendineae and mitral valves. The four-chamber view demonstrates the ventricles, atria and mitral and tricuspid valves. Rotation of the transducer allows two-chamber views of the heart and more detailed assessment of the aorta and aortic valves demonstrated.

The EF and volumes were measured with 2D-biplane Simpson's method, 2D-triplane, and 3-dimensional echocardiography (3DE) by 2 investigators blinded to any clinical data. By using the protocol of echo which was established by British Society of Echocardiography Education Committee which state that for viewing the HF a long-axis view is used applying of this view was obtained by put the echo transducer (probe) in the left apicosternal position and provides detailed images of many parts of heart specially the left ventricle, aorta, left atrium, and mitral and aortic valves. The echocardiography machine that used for this study was my lab 50 from Esaote Company with (2.5 MHz) phased Array (PA) transducer.

3.5 The study variables:

The variables used were :

1. Age and gender
2. Ejection fraction

3. The main complications of hypertension

-Left ventricular hypertrophy .

-Left ventricular diastolic dysfunction .

3.6 Data analysis:

Statistical Package for the Social Sciences SPSS was used.

Chapter four

Results

Chapter Four

4.1 Results

Table 4.1 Age groups of studied samples illustrates:

			Studied groups				Total		P value of difference
			(Uncontrolled untreated hypertensive patients)		Control (treated hypertension)				
Age groups	<50 years	N0.	11	11%	18	18%	29	14.5%	P=0.32 NS
	50 - 65 years	N0.	59	59%	51	51%	110	55%	
	66-95 years	N0.	30	30%	31	31%	61	30.5%	
		N0.	100	100%	100	100%	200	100%	

Table 4.2 Gender distribution of studied sample illustrates:

			Studied groups				Total		P value of difference
			(Uncontrolled untreated hypertensive patients)		Control (treated hypertension)				
Gender	Male	N0.	50	50%	50	50%	100	50%	P=1.0 NS
	Female	N0.	50	50%	50	50%	100	50%	
Total		N0.	100	100%	100	100%	200	100%	

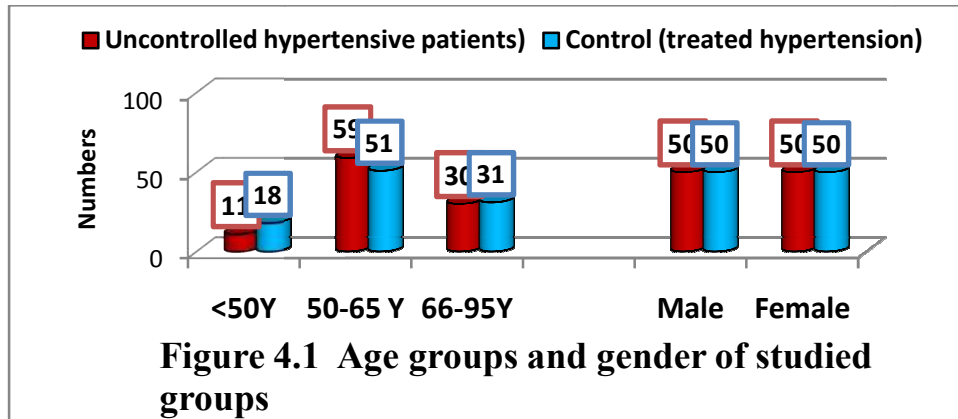


Table 4.3 Ejection fraction groups of studied illustrates :

		Studied groups				Total		P value of difference	
		(Uncontrolled untreated hypertensive patients)		Control (treated hypertension)					
Ejection fraction groups	Low (<50)	N0.	3	3%	3	3%	6	3%	P=0.83 NS
	Normal (50-75)	N0.	90	90%	92	92%	182	91%	
	High (>75)	N0.	7	7%	5	5%	12	6%	
Total		N0.	100	100%	100	100%	200	100%	

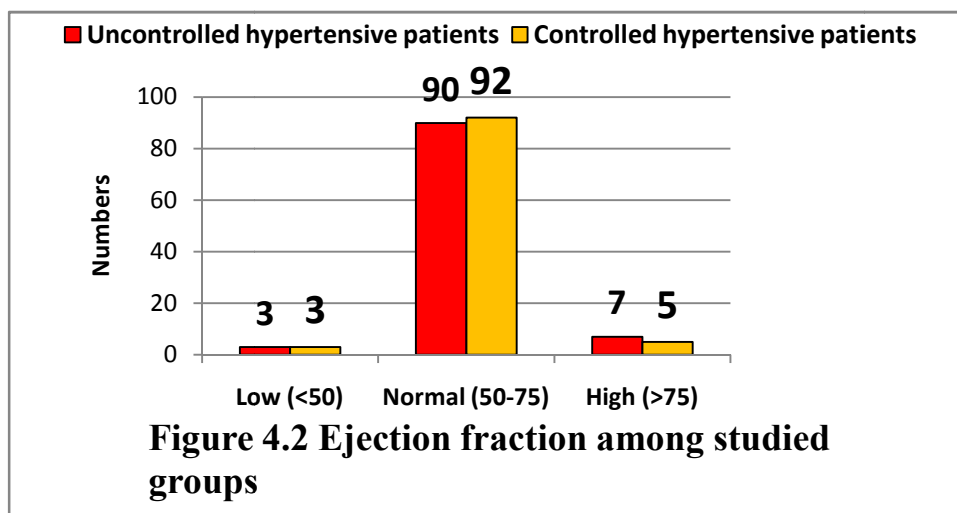


Table 4.4 Ejection fraction groups distributed by age groups of uncontrolled hypertensive patients illustrates:

Age groups	Ejection fraction groups						Total	P value of difference
	Low (<50)		Normal (50-75)		High (>75)			
<50 years	0	0%	11	12.2%	0	0%	11	11%
50 - 65 years	3	100%	52	57.8%	4	57.1%	59	59%
66-95 years	0	0%	27	30%	3	42.9%	30	30%
Total	3	100%	90	100%	7	100%	100	100%

* Likelihood ratio

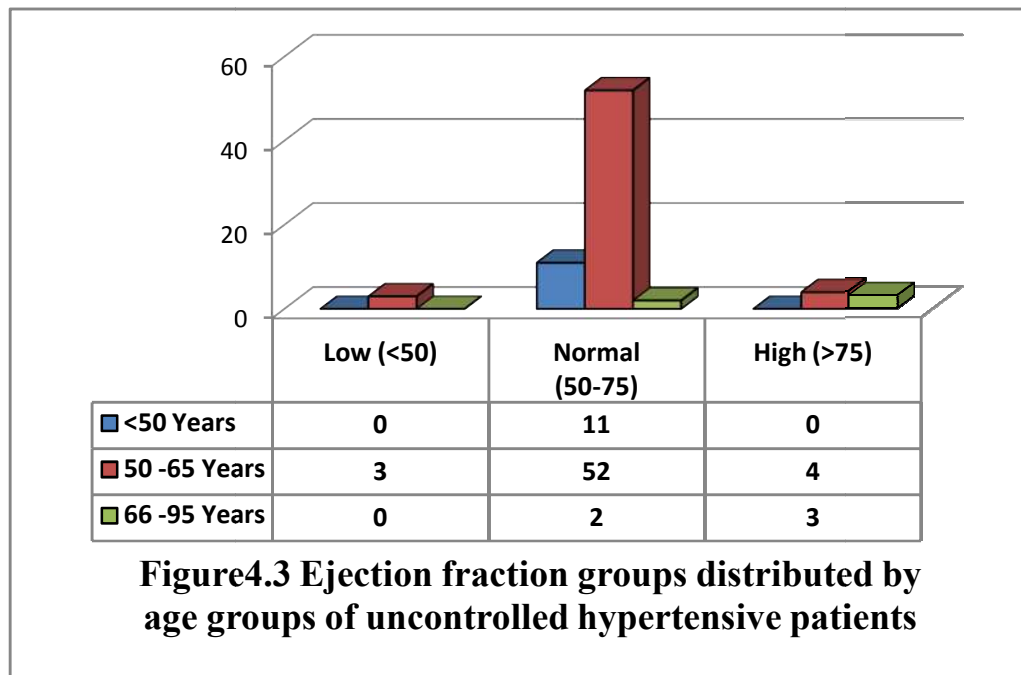


Table 4.5 Ejection fraction groups distributed by age groups of controlled hypertensive patients illustrates :

Age groups	Ejection fraction groups						Total	P value of difference
	Low (<50)		Normal (50-75)		High (>75)			
<50 years	0	0%	18	19.6%	0	0%	18 18%	* P=0.35 NS
50 - 65 years	2	66.7%	47	51.1%	2	40%	51 51%	
66-95 years	1	33.3%	27	29.3%	3	60%	31 31%	
Total	3	100%	92	100%	5	100%	100 100%	

* Likelihood ratio

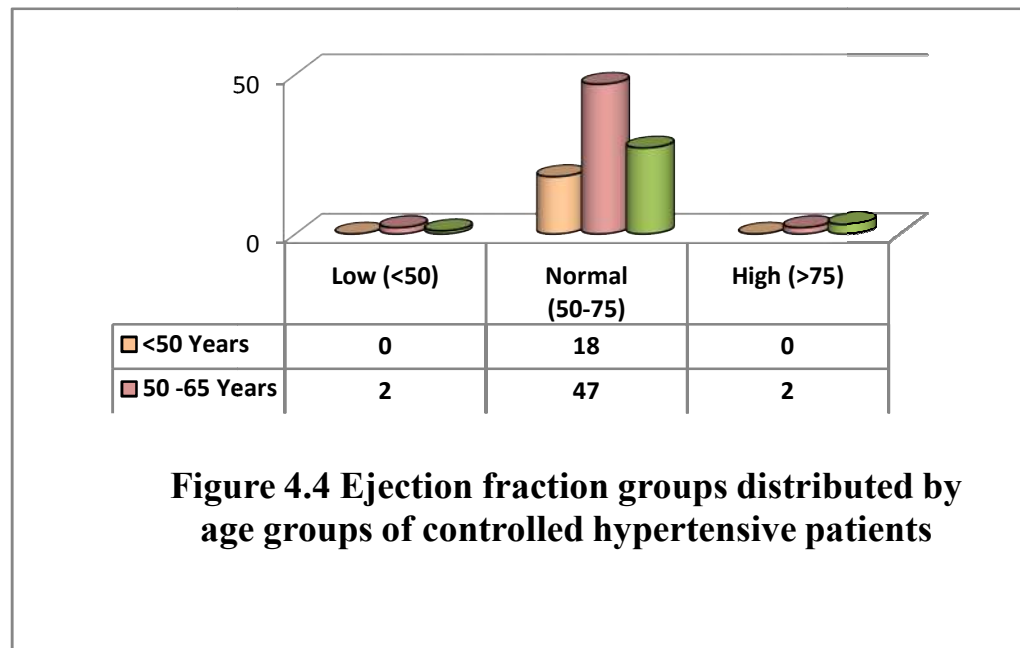


Table 4.6 Ejection fraction groups of uncontrolled hypertensive patients distributed by gender illustrates:

Gender	Ejection fraction groups						Total	P value of difference
	Low (<50)		Normal (50-70)		High (>75)			
Male	1	33.3%	46	51.1%	3	42.9%	50 50%	* P=0.76 NS
Female	2	66.7%	44	48.9%	4	57.1%	50 50%	
Total	3	100%	90	100%	7	100%	100 100%	

* Likelihood ratio

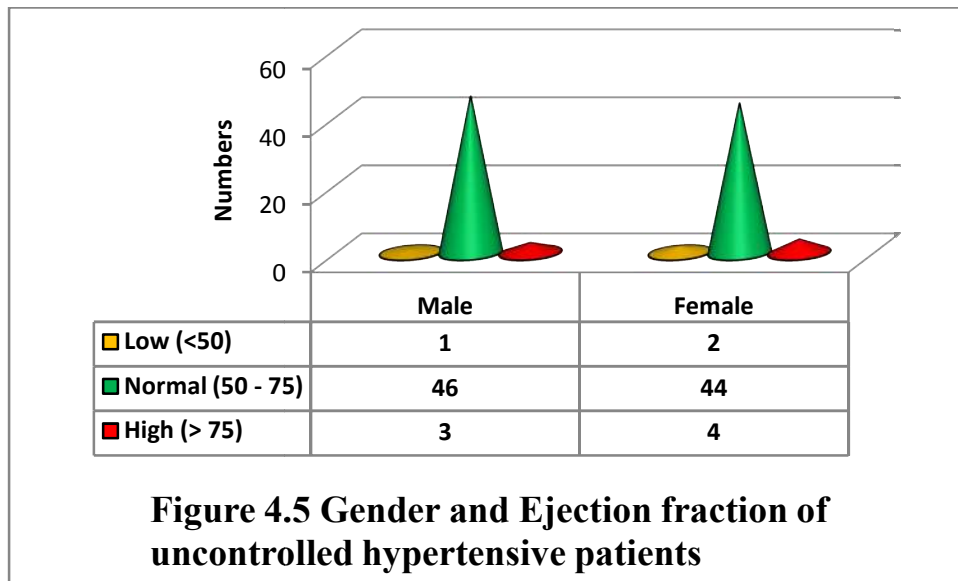


Table 4.7 Ejection fraction groups of controlled hypertensive patients distributed by gender illustrates:

Gender	Ejection fraction groups						Total	P value of difference
	Low (<50)		Normal (50-70)		High (>75)			
Male	1	33.3%	47	51.1%	2	40%	50 50%	* P=0.74 NS
Female	2	66.7%	45	48.9%	3	60%	50 50%	
Total	3	100%	92	100%	5	100%	100 100%	

* Likelihood ratio

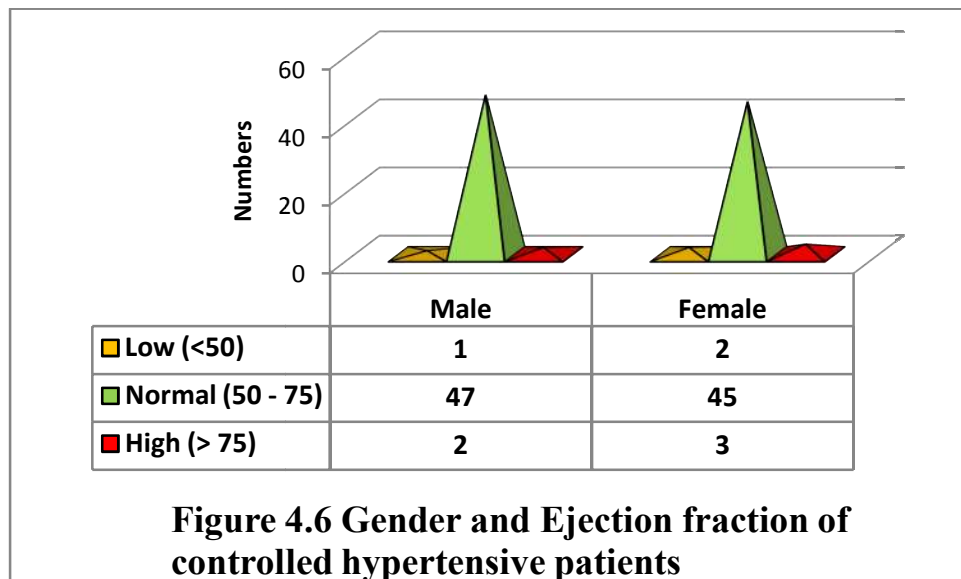


Table 4.8 Ejection fraction groups distributed by LVH as a cause of hypertension among uncontrolled hypertensive patients illustrates :

	Ejection fraction groups						Total N=100	P value	
	Low (<50) N=3		Normal (50-70) N=90		High (>75) N=7				
LVH:									
NIL	1	33.3%	49	54.4%	5	71.4%	55	55%	*P=0.50 NS
LVH	2	66.7 %	41	45.6 %	2	28.6%	45	45%	

*Likelihood ratio

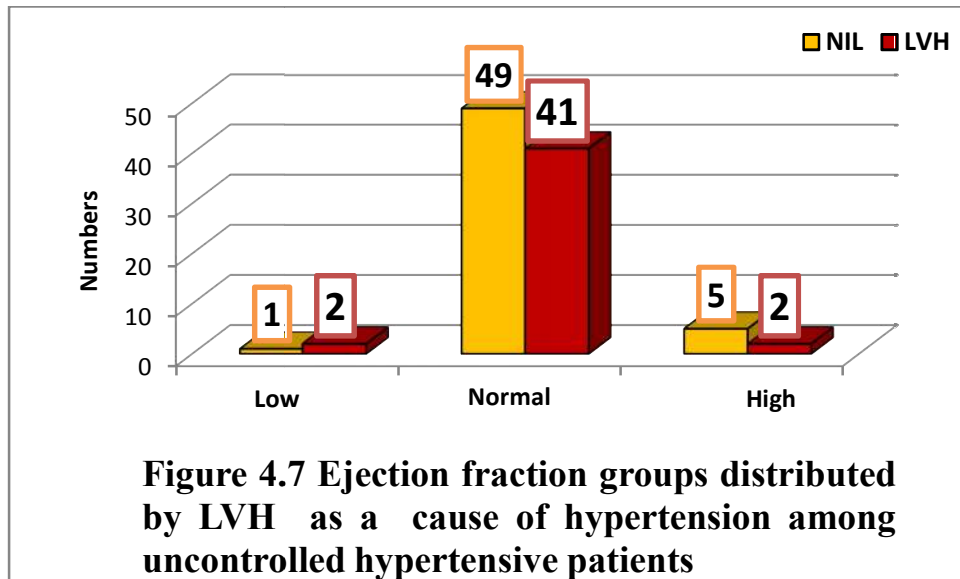


Table 4.9 Ejection fraction groups distributed by LVH as a cause of hypertension among controlled hypertensive patients illustrates:

	Ejection fraction groups						Total N=100	P value	
	Low (<50) N=3		Normal (50-70) N=92		High (>75) N=5				
LVH:									
NIL	1	33.3%	36	39.1%	1	20%	38	38%	*P=0.56 NS
LVH	2	66.7 %	56	60.9 %	4	80%	62	62%	

*Likelihood ratio

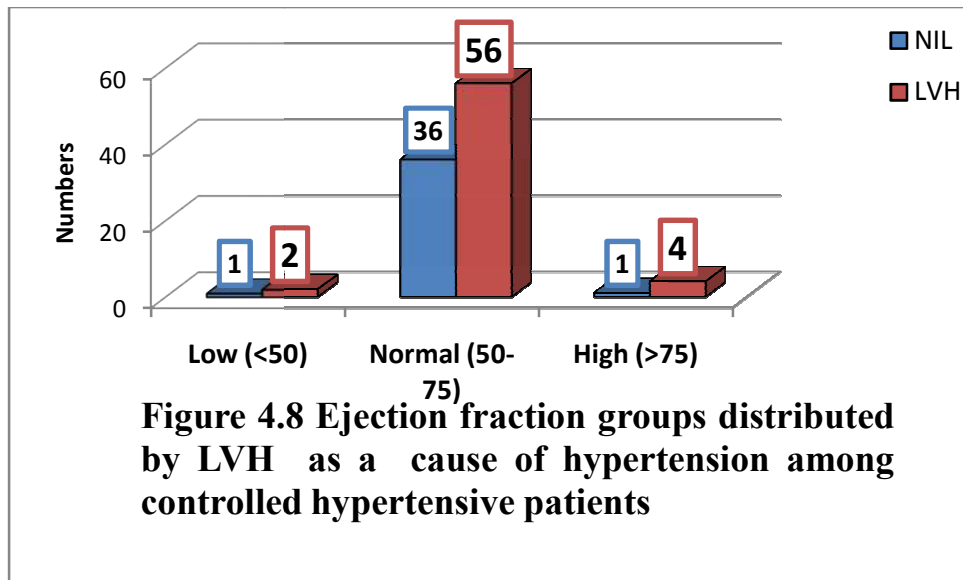


Table. 4.10: LVH distributed by age groups of studied 100 Uncontrolled hypertensive patients illustrates :

Age groups	LVH groups				Total		P value of difference
	NIL		LVH				
<50 years	3	5.4%	8	17.8%	11	11%	*P=0.04 Sig.
50 - 65 years	38	69.1%	21	46.7%	59	59%	
66-95 years	14	25.5%	16	35.6%	30	30%	
Total	55	100%	45	100%	100	100%	

*Likelihood ratio

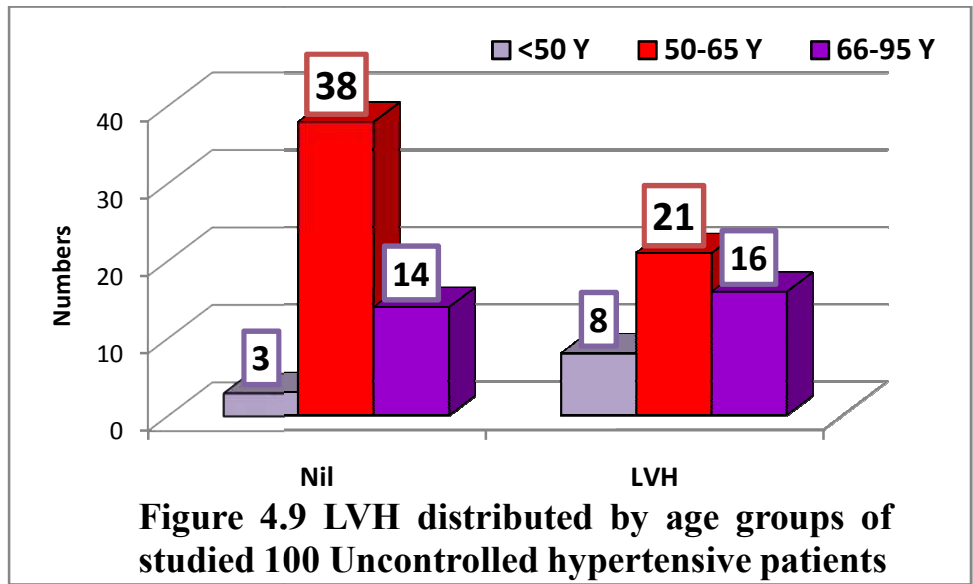


Table 4.11 LVH distributed by age groups of studied 100 controlled hypertensive patients illustrates :

Age groups	LVH groups				Total		P value of difference
	NIL		LVH				
<50 years	2	5.3%	16	25.8%	18	18%	*P=0.01 Sig.
50 - 65 years	19	50%	32	51.6%	51	51%	
66-95 years	17	44.7%	14	22.6%	31	31%	
Total	38	100%	62	100%	100	100%	

*Likelihood ratio

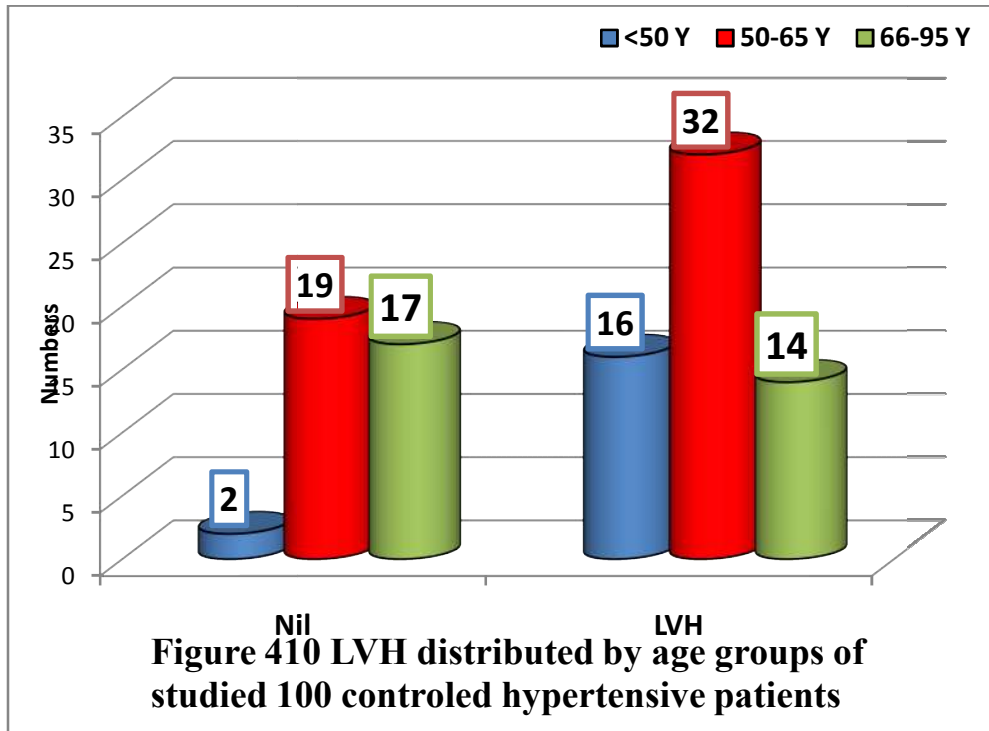


Table 4.12 LVH groups distributed by gender of uncontrolled hypertensive patients illustrates :

Gender	LVH groups				Total		P value of difference
	NIL		LVH				
Male	29	52.7%	21	46.7%	50	50%	*P=0.54 NS
Female	26	47.3%	24	53.3%	50	50%	
Total	55	100%	45	100%	100	100%	

*Likelihood ratio

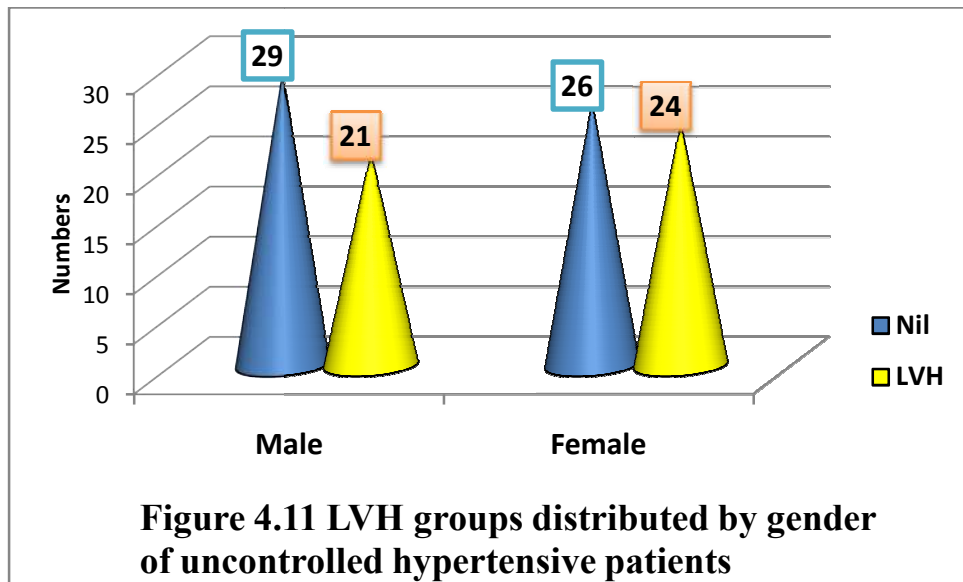


Table 4.13 LVH groups distributed by gender of controlled hypertensive patients illustrates:

Gender	LVH groups				Total		P value of difference
	NIL		LVH				
Male	20	52.6%	30	48.4%	50	50%	*P=0.67 NS
Female	18	47.4%	32	51.6%	50	50%	
Total	38	100%	62	100%	100	100%	

*Likelihood ratio

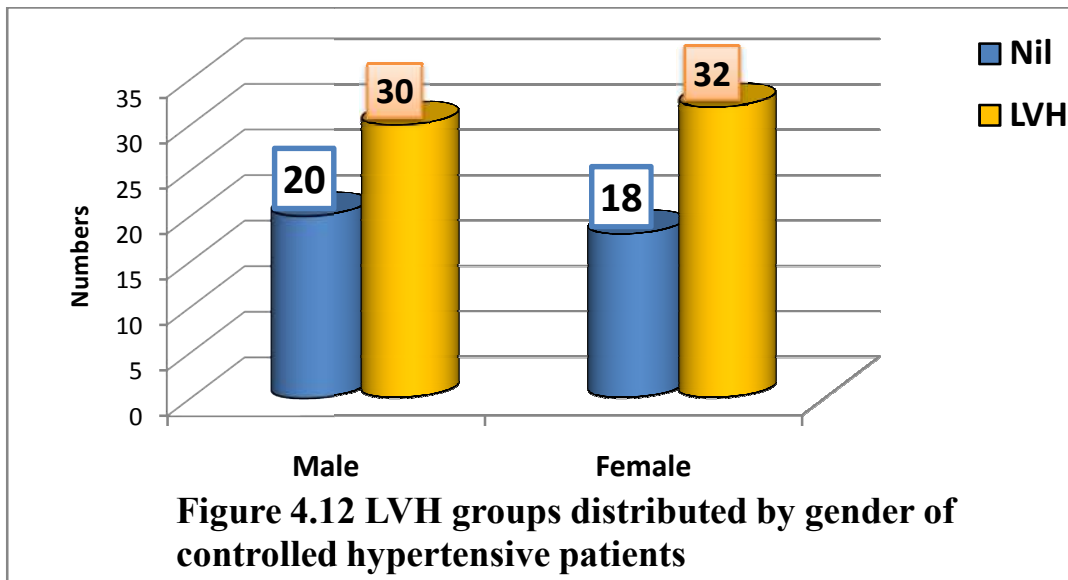


Table 4.14 Ejection fraction groups distributed by LVDD among uncontrolled hypertensive patients illustrates :

	Ejection fraction groups						Total N=100	P value	
	Low (<50) N=3		Normal (50-70) N=90		High (>75) N=7				
<u>LV diastolic dysfunction:</u>									
NIL	1	33.3%	22	24.4%	1	14.3%	24	24%	*P=0.76 NS
LVDD	2	66.7 %	68	75.6 %	6	85.7%	76	76%	
Total	3	100%	90	100%	7	100%	100	%	

*Likelihood ratio

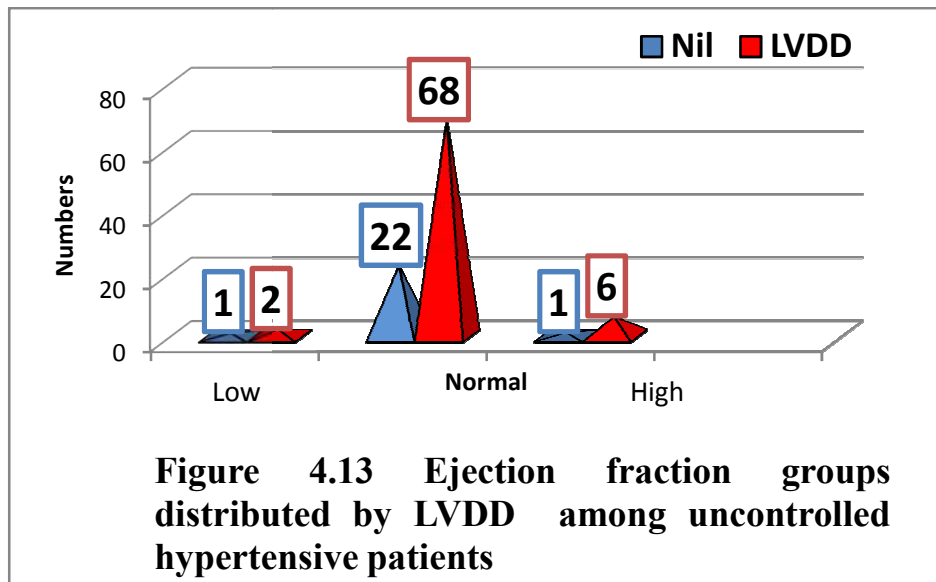


Table 4.15 Ejection fraction groups distributed by LVDD among controlled hypertensive patients illustrates :

	Ejection fraction groups						Total N=100	P value	
	Low (<50) N=3		Normal (50-70) N=92		High (>75) N=5				
<u>LV diastolic dysfunction:</u>									
NIL	1	33.3%	28	30.4%	0	0%	29	29%	P= 0.17 NS
LVDD	2	66.7 %	64	69.6 %	5	100%	71	71%	
Total	3	100%	92	100%	5	100%	100	%	

*Likelihood ratio

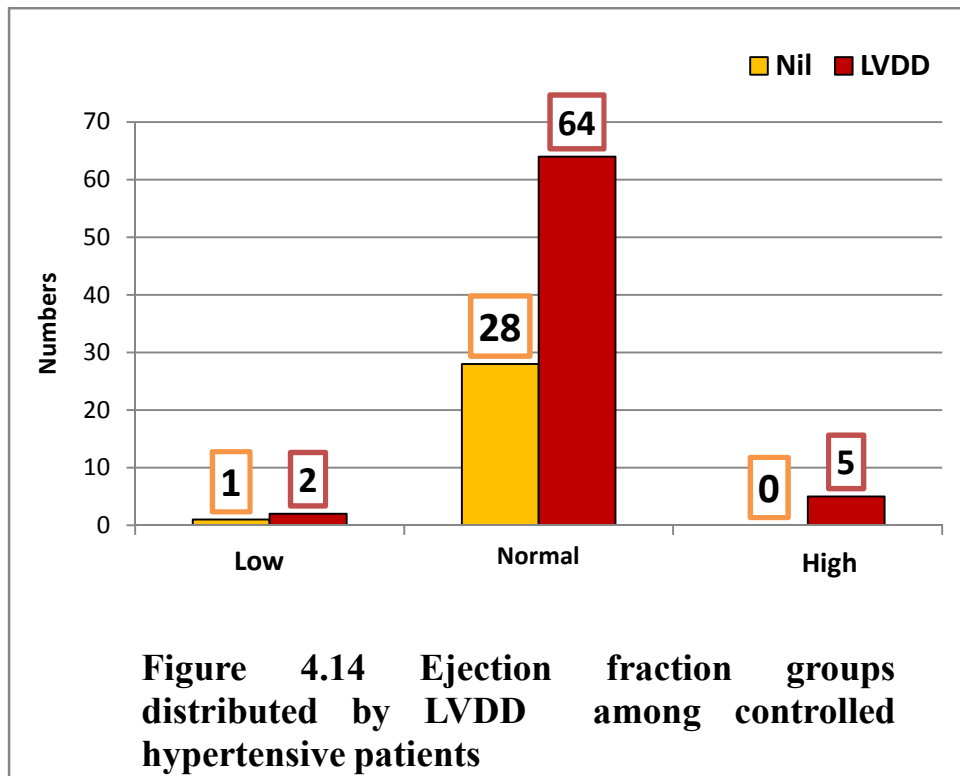


Table 4.16 LVDD groups distributed by age groups of uncontrolled hypertensive patients illustrates :

Age groups	LVDD groups				Total		P value of difference
	NIL		LVDD				
<50 years	6	25%	5	6.6%	11	11%	*P=0.03 Sig.
50 - 65 years	10	41.7%	49	64.5%	59	59%	
66-95 years	8	33.3%	22	28.9%	30	30%	
Total	24	100%	76	60%	100	100%	

*Likelihood ratio

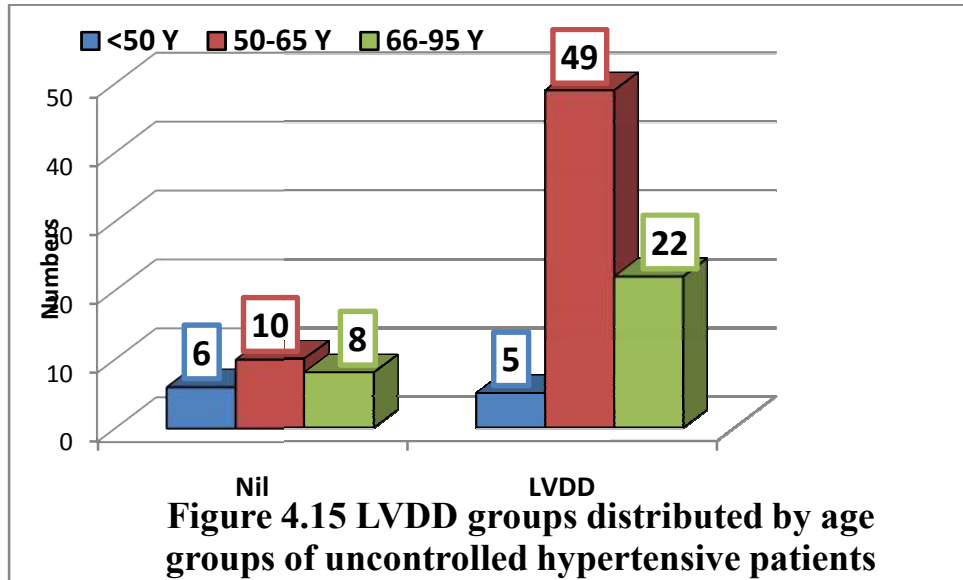


Table 4.17 LVDD groups distributed by age groups of controlled hypertensive patients illustrates :

Age groups	LVDD groups				Total		P value of difference
	NIL		LVDD				
<50 years	12	41.4%	6	8.5%	18	18%	P=0.000 High Sig.
50 - 65 years	15	51.7%	36	50.7%	51	51%	
66-95 years	2	6.9%	29	40.8%	31	31%	
Total	29	100%	71	100%	100	100%	

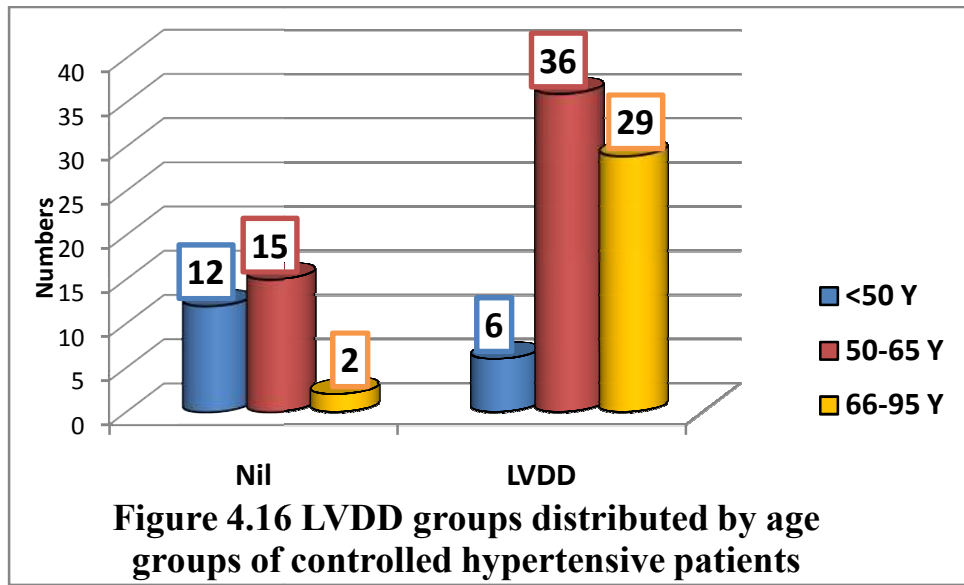


Table 4.18 LVDD groups distributed by gender of uncontrolled hypertensive patients illustrates :

Gender	LVDD groups				Total		P value of difference
	NIL		LVDD				
Male	13	54.2%	37	48.7%	50	50%	P=0.64 NS
Female	11	45.8%	39	51.3%	50	50%	
Total	24	100%	76	100%	100	100%	

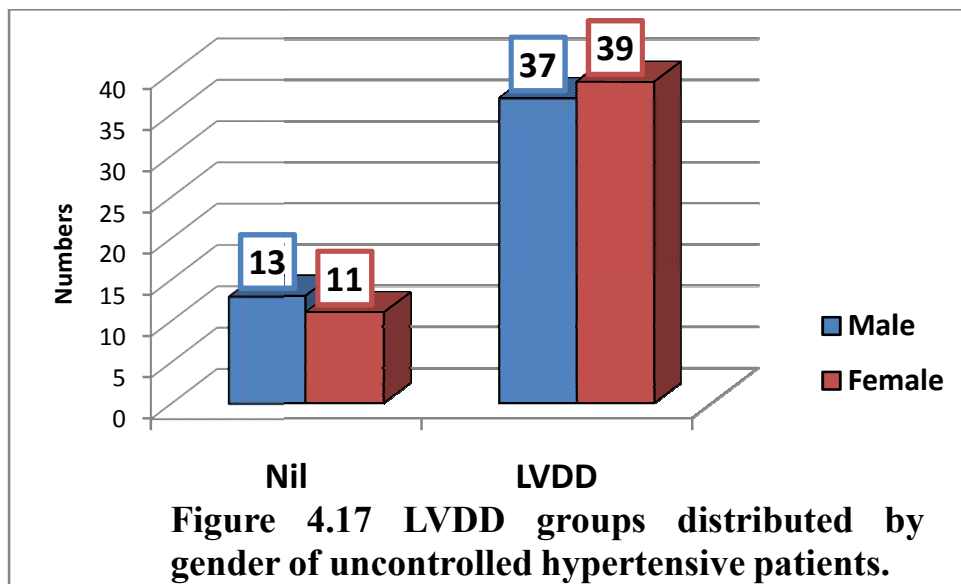


Table 4.19 LVDD groups distributed by gender of controlled hypertensive patients illustrates :

Gender	LVDD groups				Total		P value of difference
	NIL		LVDD				
Male	9	31%	41	57.7%	50	50%	P=0.02 Sig.
Female	20	69%	30	42.3%	50	50%	
Total	29	100%	71	100%	100	100%	

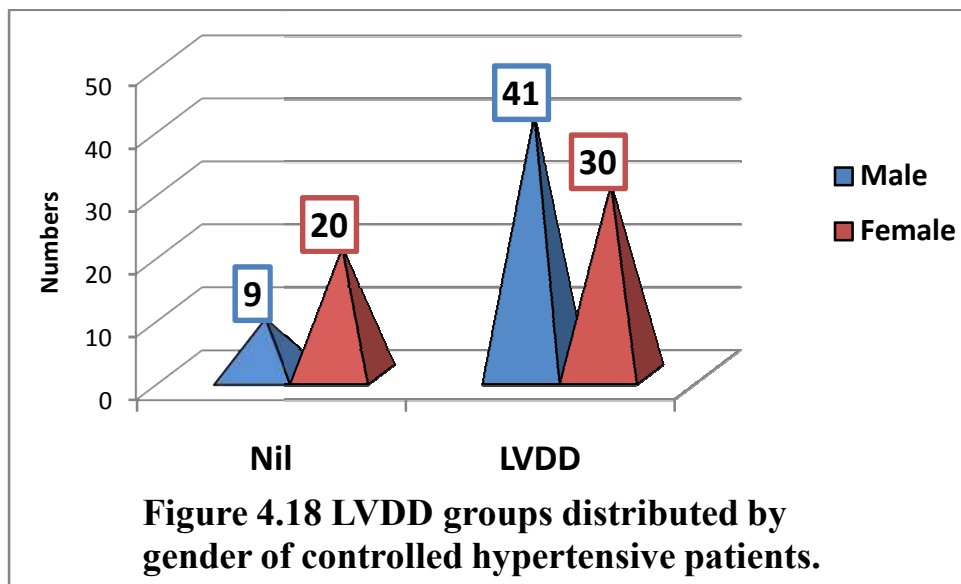


Table 4.20 Relation between LVH groups and LV diastolic dysfunction groups among uncontrolled hypertensive patients illustrates :

			LV diastolic dysfunction groups				Total		P value
			NIL		LVDD				
LVH groups	NIL	N0.	4	16.7%	51	67.1%	55	55%	P=0.000 *HS
	LVH	N0.	20	83.3%	25	32.9%	45	45%	
Total		N0.	24	100%	76	100%	100	100%	

HS = High significant

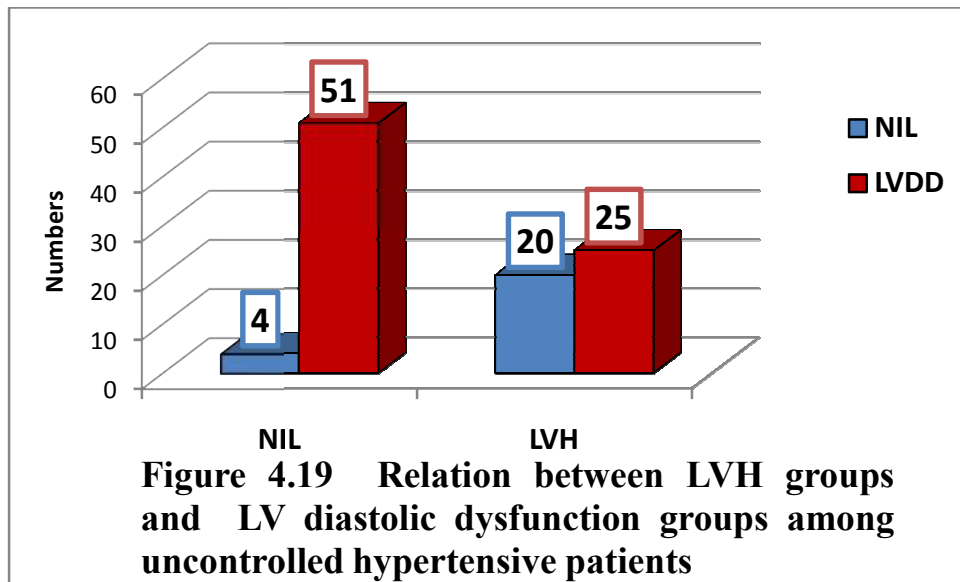
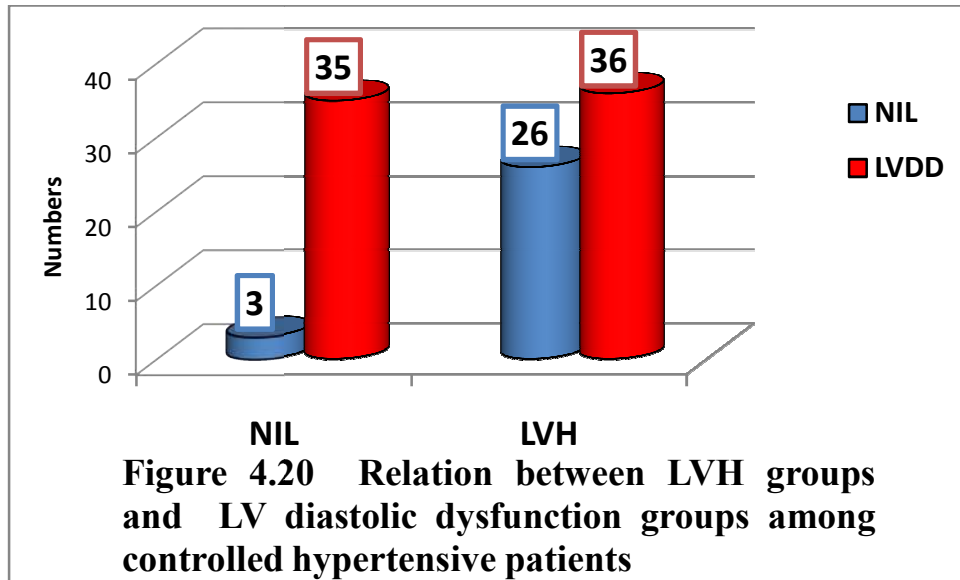


Table 4.21 Relation between LVH groups and LV diastolic dysfunction groups among controlled hypertensive patients illustrates :

			LV diastolic dysfunction groups				Total		P value
			NIL		LVDD				
LVH groups	NIL	N0.	3	10.3%	35	49.3%	38	38%	P=0.000 *HS
	LVH	N0.	26	89.7%	36	50.7%	62	62%	
Total		N0.	29	100%	71	100%	100	100%	

HS = High significant



Chapter five
Discussion, Conclusion and
Recommendations

Chapter Five

Discussion, conclusion and recommendations

5.1 Discussion

This cross-sectional hospital based study used echocardiography to study effect of hypertension on cardiac ejection fraction, LVH and diastolic dysfunction through age and gender. 200 patients underwent echocardiography. 100 of participants had control hypertension (HTN) and 100 of them had uncontrolled hypertension (HTN). Half of each group was male and other half was female.

The study demonstrated the effect of HTN on ejection fraction (EF) of heart. That touched through the percentage which was increased in normal EF in controlled group 92% more than uncontrolled 90%. Also the increasing was represented in uncontrolled group 7% in high EF more than controlled 5%. Although there was equalized in percentage in low EF in both group sample 3% may be that due to two probabilities, either the controlling was not good in control group or may be the uncontrolled group was included new discovered which may be they were not affect the EF so the number of uncontrolled was less than controlled (*figure 4.2*). This is agree with study done by (*Manickavasagam 2009*) and (*Sharief et al 2016*).

The study demonstrated that the advancing in age reduced ejection fraction for both controlled and uncontrolled hypertensive as general, although there was no one in group with age ranging (65-95) in uncontrolled hypertension had low ejection fraction as expected, this may because these patients had short duration of hypertension which not enough to produce structural change in left ventricle which affect the ejection fraction and there are more factors can contributed in that. (Type of medication, life style and management of life). The study also showed normal ejection fraction for all patients with age ≤ 50 up to 95 years. There was equalized percentage in age ranging (66-95) years for both group sample but it there was high percentage in age (≤ 50) years in control (19.6%) more than

uncontrolled hypertensive 12.2% and in age ranging (50-65)years the percentage was (57.78%) in uncontrolled more than in controlled which was (51.1%) . For uncontrolled HTN as general in both ages the patients there were two probabilities, either may because really were normal due to short duration and some of them new discovered of HTN which not enough to produce structural changes in left ventricle which affect the ejection fraction, and they also not controlled well because they took medication but not regularly .Nor they had a sign of HF because there is a type of HF with normal EF called HFpEF. Although in age range (66-95) years were equalized in controlled and uncontrolled HTN in number of patients. but they both may be really normal or the uncontrolled group suffering from HFpEF. This study also represented the high EF in different age groups and increased in age between (50-65) years in uncontrolled (57.1%) than controlled (40%) while equalized in age between (65-95) years in both group and there was no patients in age (≤ 50) years high EF . May be that due to small age which considered there was no effect on heart of them. And for other ages may be they suffering from Hypertrophic cardiomyopathy (HCM) which involved with high EF and it can be developed with HTN (*figure 4.3 and 4.4*) and this is match with studies by (*Sharief et al , 2015.*) ,(*William , 2008*), (*Hosseini et al,2014*),(*Maeder&David,2009*),(*Aronow et al,1998*) ,(*Algazouly,2014*)and (*American Heart Association (AHA),2015*).

This study showed the female was affected more than male on all groups controlled and uncontrolled in low and high EF may be due to the female have high left ventricle EF (*figure 4.5 and 4.6*). Women tend to develop congestive heart failure at an older age than men. This is consistent with study done by(*Cleveland Clinic , 2016*)and(*Anne et al 2006*) .

This study showed that there is increasing percentage in low EF (decreasing in EF) in case of LVH in all studied group controlled and uncontrolled.

The percentage was equalized in both group sample in case of nil LVH was (33.33%) and in in case of LVH was (66.7%). That may be means the LVH is affecting in ejection fraction of patient. Also this study showed that the normal EF was increase in case of nil LVH (54.4%) more than in case LVH 45.6% in uncontrolled group but decreased in case of nil LVH (39.1%) in controlled more than in case of LVH (60.9%) . That means may be the LVH had effect on EF, so for uncontrolled group sample the normal was decreased in case of LVH more than in case of nil LVH that may be because the studied sample were took medication but not regularly or this group sample was included new discovered patients so they were not affected by LVH although they were uncontrolled. In addition may be also there suffering from HFpEF . While for controlled was decreased in case of nil LVH more than LVH that may be due to good controlling and life style. Also this study showed there is no directly relationship between LVH and high EF but there is probability of HCM developed by HTN that touched through the increasing percentage of high EF in patients with nil LVH (57.1%) of uncontrolled more than in case of LVH (20%) in case of controlling while in case of LVH of uncontrolled was (28.6%) less than in controlled group 80% that means the uncontrolled pats had probability of HCM due to their uncontrolled group of HTN more than controlled group even though in case of nil LVH . While in controlled group the probability of HCM increased in case of LVH more than in case of nil LVH but was considered less numbering of patents than uncontrolled that may be their is correlation of HCM , HTN and HCM, high EF (*figure 4.7, 4.8*). This result in line with studies from (*Gregory , 2001*) , (*Myo clinic, 2015*) and (*Jeffrey, 2004*).

This study showed the relationship between the LVH and advancing age in uncontrolled HTN and controlled and especially in middle age ranging (50-65) years. In uncontrolled the percentage increase in nil LVH 69.1% more than others

ages (≤ 50) (5.4%) and (65-95) (25.5%) that means a lot of study sample in this range were normal LVH while decrease the percentage in age ranging (66-95) years due to relationship of LVH with age and also in age >50 the percentage was decreased may be due to few number of patients in this group . or may be short duration of HTN with unregularly treatment or new discovered HTN.

Also the percentage in uncontrolled in case LVH was increased more age ranging (50-65) years more than others ages group (≤ 50) 25.8% and (66-95) years (22.6%) may be that this group age ranging (50-65) years were more affected group with LVH . Or may be that due to the relationship of LVH with aging specially they were not well controlled. While in controlled group there was increasing percentage of nil LVH in age ranging (50-65) years more than others age ranging (<50) years (5.3%) and (65-95) years (44.7%) although there is relationship between LVH and age but may be that due to they were took the treatment correctly and they were good controlling and good exercise more than age ranging (65-95) years or the group were more in this ranging group comparing with numbers of patients in other groups according to table(1) and figure (2). While for age >50 either because this group were few numbers or they were small age. And in controlled in case of LVH the percentage was increasing with age because there is relationship of LVH with age except in age ranging (65-95) years may be that either due to the number of patients in this slice were less than age ranging (50-65) years or the duration of HTN was long term without good controlling of treatment .This study confirmed with study of (*figure 4.9 and 4.10*). This result is agree with study done by (*Cuspidi , 2012*), (*Richard et al , 1987*), (*Thrainsdottir1 et al ,2003*) , (*Nikitin et al 2003*) and (*Cleveland clinic ,1995*) .

The study showed the LVH had relationship with gender through the increasing percentage of female more than male in both studied group controlled and uncontrolled may be because the women were not controlled good in

uncontrolled and they were not do exercises in controlled HTN and may be they suffering from obesity more than men(*figure 4.11 and 4.12*). This result match with the study of (*AHA , 2015*),(*Stojanov et al , 2012*),(*Avdić et al , 2007*)and (*East et al 2003*). and (*Jakovljevic B et al , 2010*).

The study showed that the low and high EF were increased more in case of LVDD more than nil in uncontrolled patients and controlled may be the LVDD had effect in EF rate of heart (*figure 4.11 and 4.12*). This result was match with study of (*Pasquale Palmiero, 2014*).

The study also showed increased normal EF in patients with LVDD more than without LVDD (nil) in both studied group controlled and uncontrolled and more in uncontrolled than the control .In case of controlled may be due to they were good controlled while in case uncontrolled may be two probabilities either there were really normal due to short duration of HTN in order to they included new discovered patients and they were took medication but not regularly nor they were bad controlling and they may be their the heart was not working well according to (HFpEF) that discuss in second part of discussion(*figure 4.13 and 4.14*).This result was match with study of (*Mooi et al , 2012*)and (*Brunotteet al , 2003*).

The study showed there is relationship between LVDD and age. That touched through the percentage f LVDD was increased with age in both group sampled. Except in age ranging (66-95) years may be that due to the numbers of this group was less than age group ranging (50-95) years as showed in table 1 and figure 1.And in case nil LVDD the uncontrolled the percentage must be decreased but it was increased may be that due to difference number of that age groups because the age ranging (50-65) years represented high percentage than others and it was more numbers than other age group.While in controlled in case of nil LVDD the percentage must be decreased with aging but was increasing in age (50-

65)years that may be due to numbers of patients in this group aging (*figure 4.15 and 4.16*) .This result in line with study of (*Palmiero et al ,2014*) .

This study also showed the LVDD relationship with gender . There was increased in female more than male in uncontrolled group while vice versa in controlled the male was increased affect with LVDD more than female that may be the female had good controlling of HTN than male in controlling group while the controlling of female in uncontrolled group was bad than men (*figure 4.17 and 4.18*). This result was match with studies done by (*Zhao et al , 2014*),(*Baloch et al , 2010*) ,(*Avdić et al, 2007*)and (*Ogah , 2012*).

The study showed the involving of LVDD with LVH in uncontrolled and controlled HTN that touched through the in uncontrolled HTN the percentage of nil LVDD was 16.7% in nil LVH while (83.3%) in LVH and LVDD percentage was (67.1%) in nil LVH while 32.9% in LVH that may be due to the LVDD can occur after LVH as result of HTN duration without well treatment. That means also those pts were represented 83.3% in case of LVH was not complicated to LVDD due to the duration of HTN or they were took medication but not regularly. This result agree with studiesfrom(*Cheng et al ,2014*)and(*Sharief et al , 2016*).

In case of LVDD may be occur without LVH that may be the duration of HTN was long and they were bad controlled which let the diastolic function affected directly and others factors like (Type of medication , duration of HTN ,life style, etc)so they affected with LVDD in nil LVH .While in controlled HTN the percentage of nil LVDD was 10.3% in nil LVH while (89.7%) and LVDD percentage was represented 49.3% in nil LVH while 50.7% in LVH that means the LVDD occur after LVH and affected with LVH may be that due to its degree because group sample were included concentric and eccentric LVH the percentage increased in case of LVH and may be due to the controlling and may be were eccentric LVH in percentage of nil LVDD with nil LVH which was decreased than in LVH and in LVDD with or without LVH(*figure 4.19 and 4.20*) .This result agree with studies from (*Adamu et al , 2010*),(*Masugata,2011*) and (*Avdić et al , 2007*) .

5.2 Conclusion

It conclude in that:

- The effectiveness of modality (Echocardiography with transthoracic technique) which can be detect the more common complications of hypertension in the heart champers specially left ventricular (Hypertrophy and diastolic dysfunction) that considered the first part of heart can be affected by hypertension .
- The middle age (50-65) years were more effected age group than others ages (≤ 50 and (66-95).
- The relationship of hypertension with age, gender and ejection fraction. Also showed the ejection fraction relationship with age, gender and the correlation with the common complications of hypertension(left ventricular hypertrophy and left ventricular diastolic dysfunction).
- The Indicator of heart failure in hypertension patients in cases that recorded as low and normal of ejection fraction by echocardiography in both group sample and increased in uncontrolled group.
- The probability of hypertrophic cardiomyopathy in hypertension in cases that recorded with high ejection fraction.
- The female was more affected than male in low ejection fraction and left ventricular hypertrophy in both group samples. While in left ventricle diastolic dysfunction the female more than male in uncontrolled and vice versa in controlled.
- The heart failure is common complications of hypertension if not treated or managed well.

5.3 Recommendations

The study recommended that :

- It is recommended that using echocardiography in investigation of the heart.
- It is recommended that make studies to evaluate the complications of hypertension in valves by echo to confirm the effectiveness of echo in all hypertension complications in the heart.
- Hypertension and its complications are needed more and more focusing from all worlds because they will be lead to heart failure that can be threatening the life.
- It is necessary to make call for all technologist whom related to health for applying the care for those patients and help them because the hypertension is considered very dangerous disease (silent) on more organs if not at first diagnosed early and controlled well which that can apply with continuous assessment .
- Awareness all hypertension patients specially uncontrolled of hypertension about the complications of hypertension when they are not regulate their medications , life style and visits their doctors at least monthly will lost their life easily because there are more factors can be contribute in increase the complications of hypertension .
- It is recommended that make studies to determine the levels or stages of left ventricular hypertrophy (eccentric or concentric) and accurate duration of hypertension because is required in diagnosis of hypertension complications effectively.
- It is recommended that make studies in Sudan .

References

References

- "ACCF/ASE/AHA/ASNC/HFSA/HRS/SCAI/SCCM/SCCT/SCMR,(2011) Appropriate Use Criteria for Echocardiography". Journal of the American College of Cardiology 57 (9): 1126–1166.
- American Heart Association (AHA) updated August (2015),<http://www.heart.org/HEARTORG/Conditions18> .
- Andrew R Houghton, (2014) Making Sense of Echocardiography, Taylor and Francis Group, 2nd edition, Boca Raton.
- Anne K. Chung,(2006), Reynolds Cardiovascular Clinical Research Center, Women Have Higher Left Ventricular Ejection Fractions Than Men Independent of Differences in Left Ventricular Volume The Dallas Heart Study ,113:1597-1604.
- Anne Waugh & Allison Grant .(2010), Ross and Wilson Anatomy And Physiology In Health and illness, 11th edition , Churchill LivingStone Elsevier, China.
- Aronow WS, Ahn C, Kronzon I. Normal left ventricular ejection fraction in older persons with congestive heart failure. Chest,(1998); 113:867– 869
- Arthur C. Guyton, & John E. Hall (2006),Textbook Of Medical Physiology , 11th edn, Elsevier , China.
- Brent M Egan ,(2016), Treatment of hypertension in the elderly patient, particularly isolated systolic hypertension ,Wolters KluwerHealth.
- Bruce M. Koeppen, & Bruce A. Stanton, ,(2010),Berne & Levy Physiology, 6thedn, Mosby Elsevier,Canada P.p.
- Cardiomyopathy/HypertrophicCardiomyopathy_UCM_444317_Article.jsp#.VojLQrYrLcs.2015.)
- Cheng S et al ,(2014), US National Library of MedicineNational Institutes of Health ,Age and the effectiveness of anti-hypertensive therapy on improvement in diastolic function, 32(1):174-80.
- ChingSiewMooi et al,(2012),Life Science Journal, Prevalence of Left Ventricular Diastolic Dysfunction among Hypertensive Adults in Klang Valley, Malaysia, 9(3):713-719.
- Chummy S. Sinnatamby,Last`s Anatomy Regional and Applied , Elsevier, China.
- Cleveland Clinic , (2016) , Heart Failure in Women, 216.444.0261.

- Cleveland Clinic ,Left Ventricular Hypertrophy (LVH) ,(2016),<http://my.clevelandclinic.org/services/heart/disorders/left-ventricular-hypertrophy-lvh>. Access on 22/01/2016.
- Cunningham's J.C.D,(1987),Text book of anatomy, Twelvthedn ,Oxford University, New York,.
- Cuspidic,(2012),PUBMED, Age related prevalence of severe left ventricular hypertrophy in essential hypertension: echocardiographic findings from the ETODH study. ;21(3):139-45.:2012.668662.
- DAD Muhammed .Baloch et al ,(2010),Pakistan Heart Journal , Prevalence of left ventricular diastolic dysfunction in patient with left ventricular hypertrophy,Vol.43.No.1-2.
- Dennisk. Burn, S and VinayKumer,(2007),Robins Basic Pathology , 7th edit, Saunders, an imprint of Elsevier Inc.
- Douglas, P. S.; Garcia, M. J.; Haines, D. E.; Lai, W. W.; Manning, W. J.; Patel, A. R.; Picard, M. H.; Polk, D. M.; Ragosta, M.; Ward, R. P.; Douglas, R. B.; Weiner, R. B.; Society for Cardiovascular Angiography Interventions; Society of Critical Care Medicine; American Society of Echocardiography; American Society of Nuclear Cardiology; Heart Failure Society of America; Society for Cardiovascular Magnetic Resonance; Society of Cardiovascular Computed Tomography; American Heart Association; Heart Rhythm Society (2011).
- East MA et al ,(2003), PUPMED US National Library of Medicine National Institutes of Health , The influence of left ventricular hypertrophy on survival in patients with coronary artery disease: do race and gender matter?, 41(6):949-54.
- Ellis H, (1987), Clinical Anatomy, 7thedn, Year book medical ,Great Britain.
- Gregory Y.H. Lip, MD ,(2001), Circulation Journal Of American Heart Association,Regression of Left Ventricular Hypertrophy and Improved Prognosis,104:1582-1584.
- GU Adamu et al ,(2010),AFRICAN HEALTH SCIENCES , Prevalence of left ventricular diastolic dysfunction in newly diagnosed Nigerians with systemic hypertension: a pulsed wave Doppler echocardiographic study, 10(2): 177–182.
- Hamilton W.J,(1976),Text book of Human anatomy , 2ndedn,Hardcover,London,.
- HosseinBahrami,(2014), Journal of The American College Cardiology,Vol.63(12_S), (14)60732-4.

- <http://edemainformation.blogspot.ca/2005/11/edema-pathophysiology-and-treatment.htm>, 30 January 2015, at 13:32.
- Jae K. Oh , James B. Seward, M.D and A. Jamil Tajik,(2006),The Echo Manual,Lippincott Williams and Wilkins(Wolter Kluwer),3rd edn, China.
- Jakovljevic, B et al ,(2010), Journal of Hypertension, Gender Differences In Left Ventricular Hypertrophy Among Hypertensive Patients, PP.22.406, Vol. 28 - p e366
- Jeffrey , 2004, Left ventricular hypertrophy in hypertrophic cardiomyopathy
- Johannesson M,(1994),Scandinavian Journal of Primary Health Care (Scand J PrimHealth), The Costs of Treating Hypertension in Sweden, 07/2009; 9(3):155-160.
- K.M.Sharief et al , 2016,Medico Research Chronicles,Probability of Heart Failure in Uncontrolled Hypertensive Patients, Vol. 6, No.2; 264-273.
- K.M.Sharief,Osman.H. Hussein.AHassan,ElSafi Ahmed , (2015),Medico Research Chronicles,Ejection Fraction Rate For Hypertensive Patients Using Echocardiography,2 (3), 322-329.
- Libhaber et al ,(2013),Journal of Hypertension, Relationship between inappropriate left ventricular hypertrophy and ejection fraction independent of absolute or indexed mass in a community sample of black African ancestry, Vol. 31 - Issue 1: p 169–176.
- Masugata. H ,(2011), PUPMED US National Library of Medicine National Institutes of Health , Differences in left ventricular diastolic dysfunction between eccentric and concentric left ventricular hypertrophy in hypertensive patients with preserved systolic function.
- Medical Imaging radiology ,(2016), Echocardiography, V3 87–88, ICD-10-PCS B, CPT 70010–79999)
- Micha T. Maeder, MD &David M. Kaye, ,(2009),Journal of the American college of Cardiology, 53(11):905-918. doi:10.1016/j.jacc.2008.12.007.
- Michael Donnelly ,(2013), Gent An Exercise in Health , High Blood Pressure – The Silent Killer.
- Myo clinic , (2015), Left ventricular hypertrophy,Mayo Clinic Healthy Heart for LifeNewsletter: Mayo Clinic Health Letter
- National Heart, Lung, and Blood Institute,(2013), www.nhlbi.nih.gov/guidelines/hypertension/.

- Nikitin NP1 et al ,(2003), PUPMED US National Library of Medicine National Institutes of Health, Effect of age and sex on left atrial morphology and function, 4(1):36-42.
- O.Giovanni De Simone et al,(2011), PUPMED US National Library of Medicine National Institutes of Health, Sex differences in obesity-related changes in left ventricular morphology: the Strong Heart Study, 29(7): 1431–1438.
- Okechukwu S Ogah, (2012) ,World Journal of Cardiology , Blood pressure, prevalence of hypertension and hypertension related complications in Nigerian Africans: A review, 4(12): 327–340.
- Pasquale Palmiero et al, (2014) ,Journal of Clinical Medical Research , Left Ventricular Diastolic Function in Hypertension: Methodological Considerations and Clinical Implications, 7(3): 137–144.
- Paul Lesson, Andrew Mitchell and HaraldBecher,(2012),Oxford Specialist Handbooks in Cardiology, Oxfod University press, 2nd edn, U.S.A.
- Rani Gera,(2015),Step by Step Pediatric Echocardiography, Jaypee Brothers Medical Publishers, 3rdedn,India.
- Ricahard N. et al , (20012), pocket companion to Robbins and Cotran Pathologic Basis of Disease, Eieghthedn, Saunders, an imprint of Elsevier Inc.
- Richard B. et Al , (1987), Left Ventricular Hypertrophy in Hypertension Prevalence and Relationship to Pathophysiologic Variables, From the Department of Medicine, The New York Hospital-CornellMedical Center; the Department of Epidemiology and Social Medicine,Albert Einstein College of Medicine; and the Department of Physiology,Columbia University College of Physicians and Surgeons, New York, NewYork.
- Richard S. Snell,MD,phD,(1992),Clinical Anatomy for student, 7th edition, Wolters Kluwer business, USA.
- Robert O.Bonow, Douglas L, Mann, Douglas P.Zipes, and Peter Libby, 2012 ,Braunwald`S HEART DISEASE , Ninth edn, Saunders, an imprint of Elsevier Inc.
- SaraswathyManickavasagam et al ,(2009),Med Scape multispecialty, Management of Hypertension in Chronic Heart Failure, 2009;7(4):423-433.
- SevletaAvdić et al ,(2007),Bosnian Journal of Medical Sceinces, Left Venticular Diastolic Dysfunction in Essential Hypertension, 7 (1): 15-20.
- Susan StandringphD,DSc, FKC,(2008),Gray`s Anatomy ,Fortieth edn, Elsevier ,China.

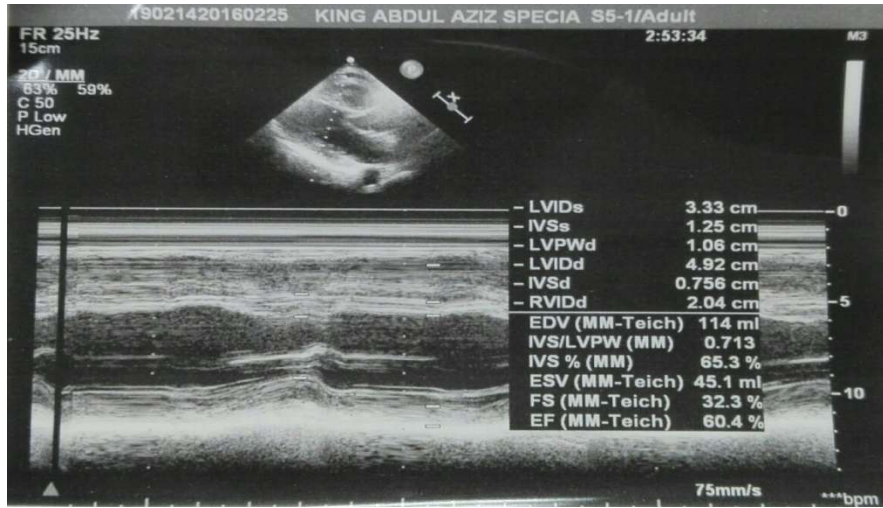
- Suzanne Oparil et al ,(2003), Annals of Internal Medicine , Pathogenesis of Hypertension, Vol 139, No. 9> , 0003-4819 139(9):761-776.
- Thrainsdottir1. L .S et al ,(2003), Journal Of Founded 1963 International Medicine, Survival and trends of occurrence of left ventricular hypertrophy, gender differences, 1967–92: The Reykjavik Study,DOI: 10.1046/j.1365-2796.2003.01111.x.
- VesnaStojanov et al , (2012), Cardiolog y Society of Serbia,The influence of obesity on left ventricular hypertrophy. Relation to gender, 31(4): 269-274
- VesnaStojanov et al ,(2012), Cardiolog y Society of Serbia,The influence of obesity on left ventricular hypertrophy. Relation to gender, 31(4): 269-274
- William C. Little, ,(2008), American Heart Association , Hypertension, Heart Failure, and Ejection Fraction, NC 27157–1045.
- William F. Armstrong and Thomas Ryan, (2010), Feigenbaum`s Echocardiography,, Wolter Kluwer health , 7thedn,Philadelphia U.S.A.
- Zhuo Zhao et al in,(2014),American Journal of Physiology , in Sex and Gender Differences Cardiovascular Physiology–Back to the BasicsRole of estrogen in diastolic dysfunction, 306(5): 628–640.

Appendixes

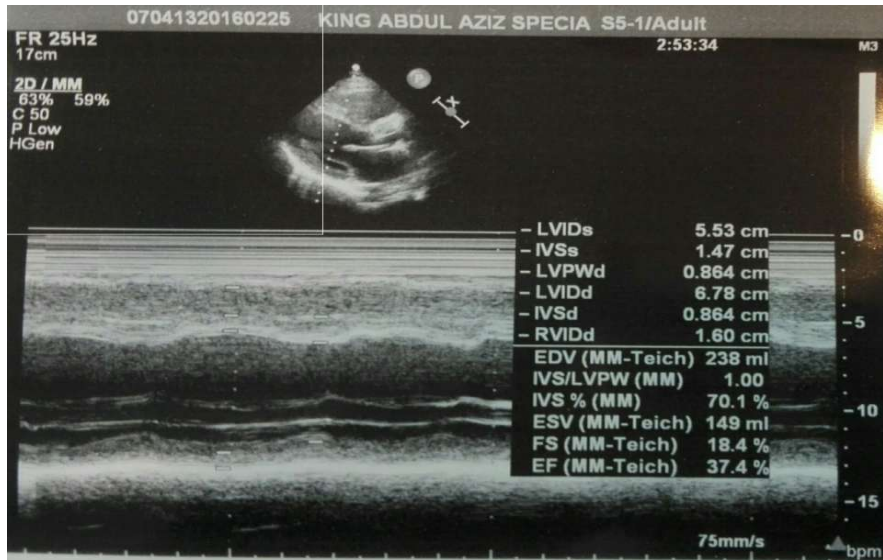
Appendix 1:Data Collection sheet

Cases	Gender	Age	LVH	LVDD	LVEDD mm	LVESD	EF %	FS %	Systolic function	LV internal dimension

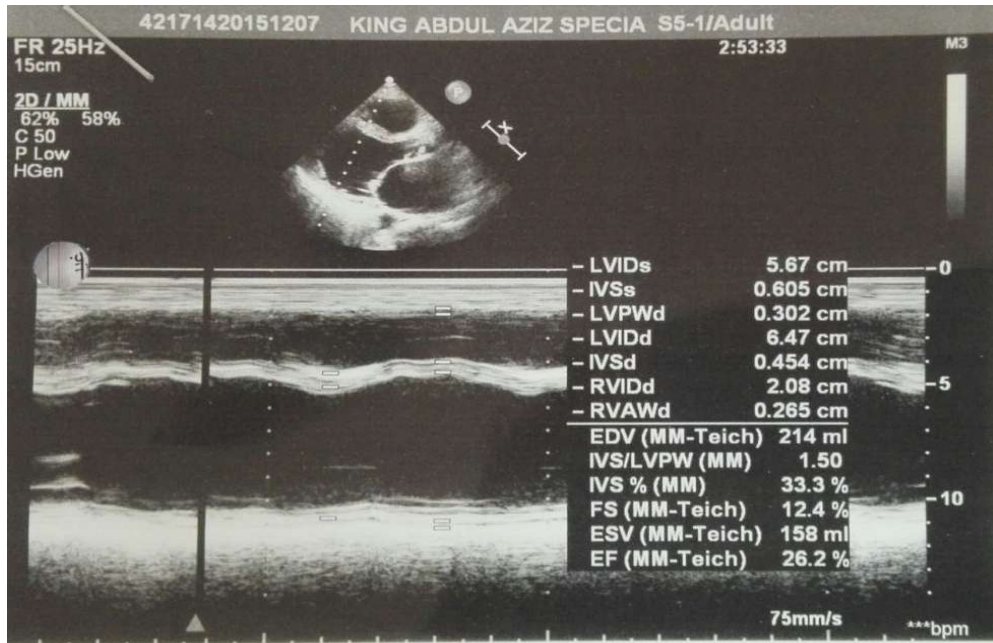
Appendix 1: Echocardiography images and pictures:



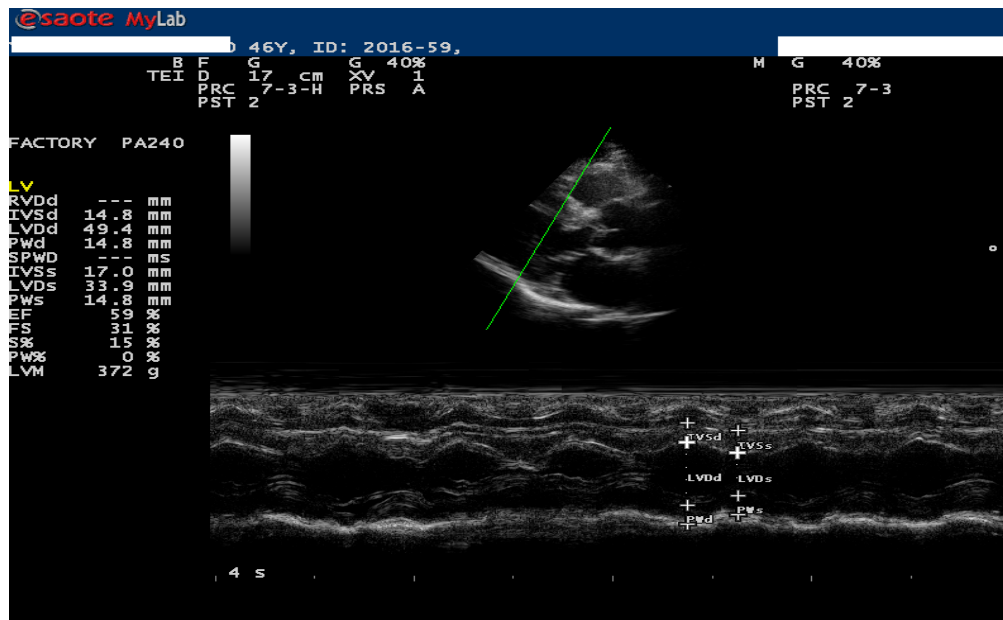
(image.1) Apical two-chamber view from a 55-year-old hypertensive patient(male) ,LVDd 4.92cm; LVDs 576 cm; IVS 1.25cm; EF 60%.



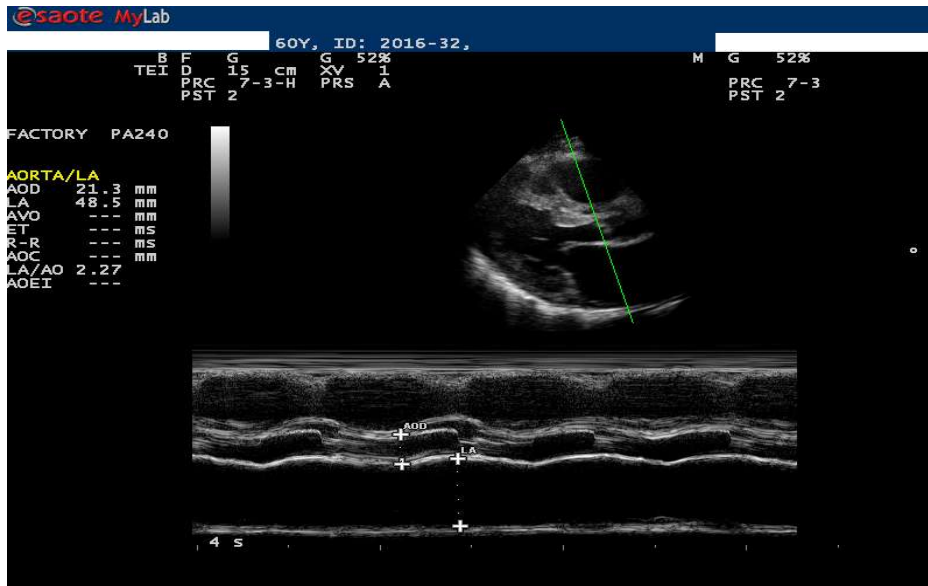
(image.2)A63-year-old hypertensive patient (female)concentric left ventricular hypertrophy LVH.LVDd 56 mm; LVDs 5.53 cm; IVS 1.47 cm ; EF 34.4%.



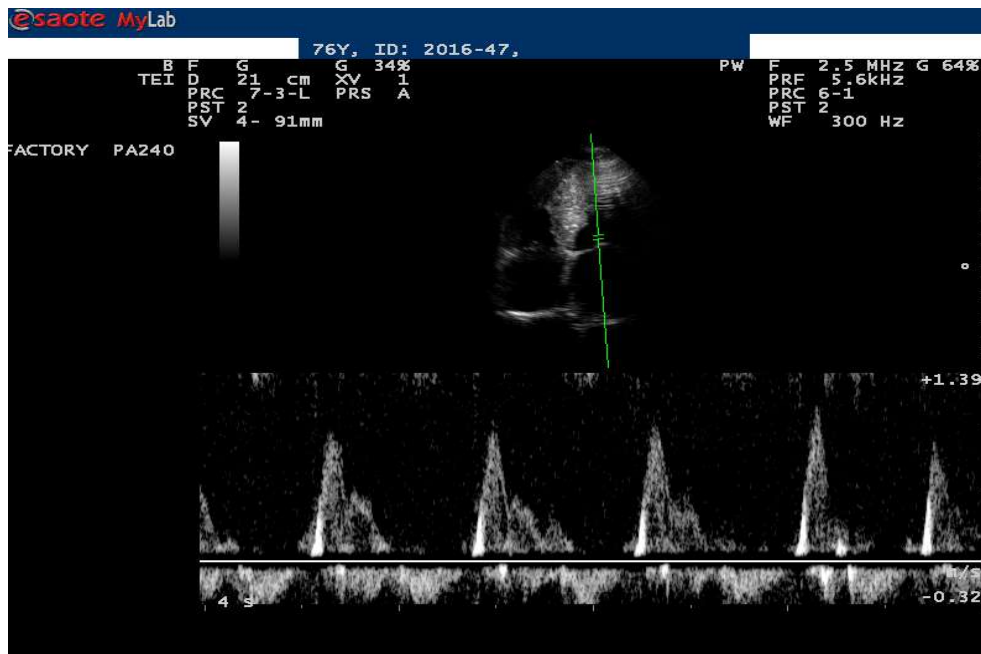
(image.3) A 70-year-old hypertensive patient (male) suffering from LVH and heart failure. He had EF 26.2%



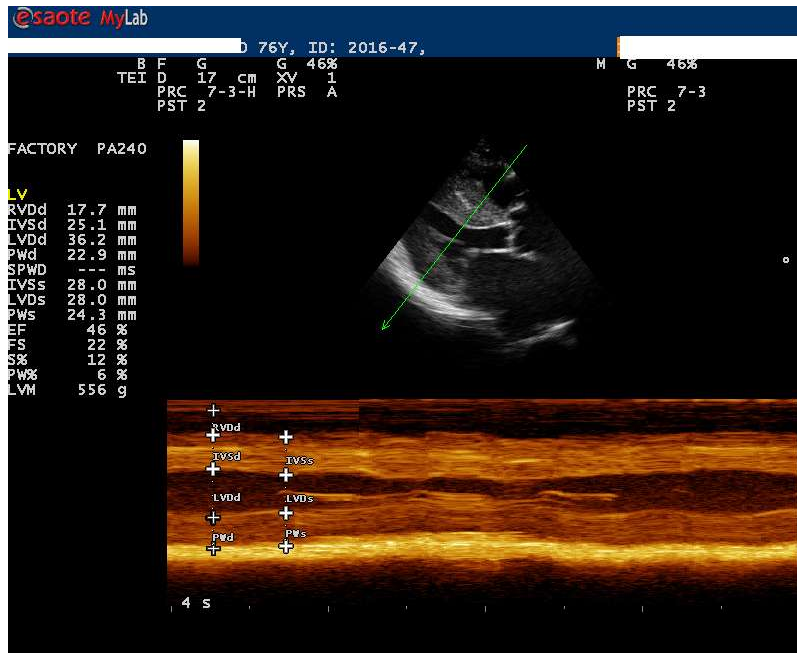
(image.4) TTE -4 chamber showed 46 years old hypertensive patient (male) with mild concentric LVH with grade I diastolic dysfunction, Normal LV systolic function, estimated EF 59%. No RWMA at rest.



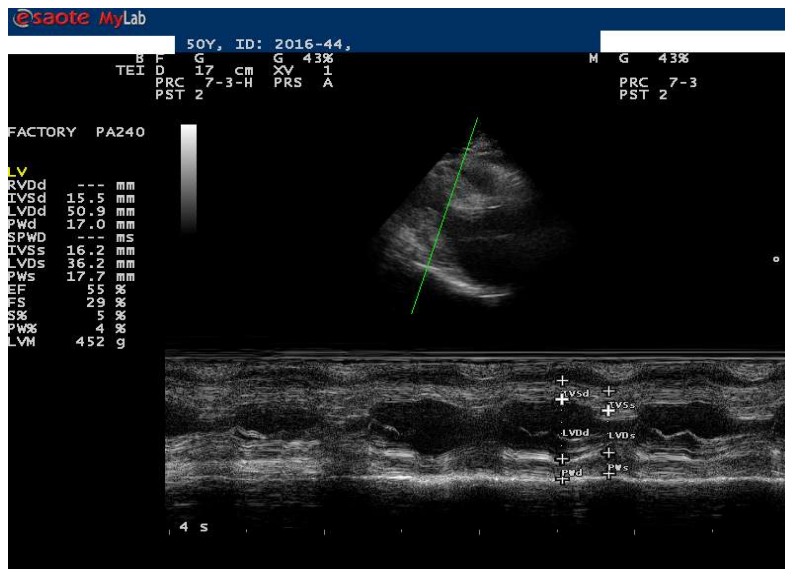
(image.5) A60-year-old hypertensive female showing concentric left ventricular hypertrophy (LVH)and left atrial enlargement .



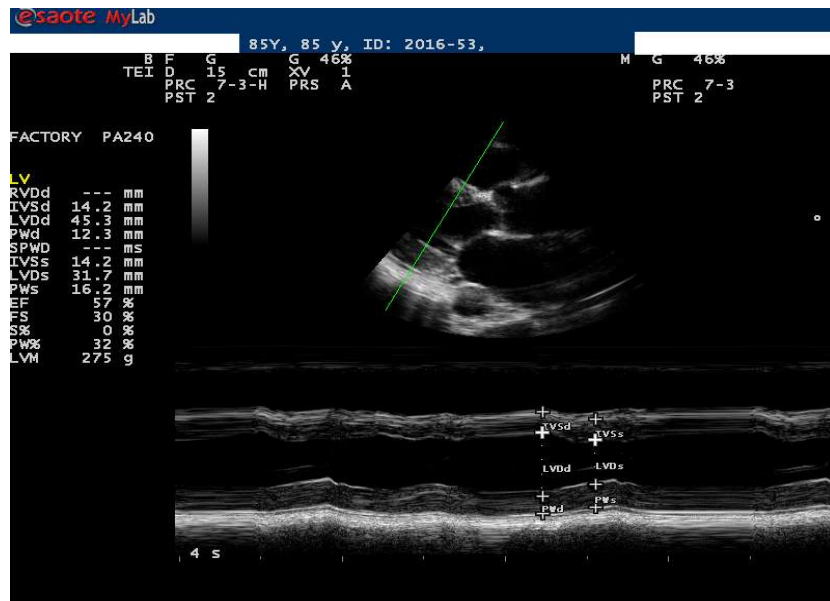
(image.6)M-mode scan 4-champers showed a76-year-old hypertensive patient (male) with normal morphology .



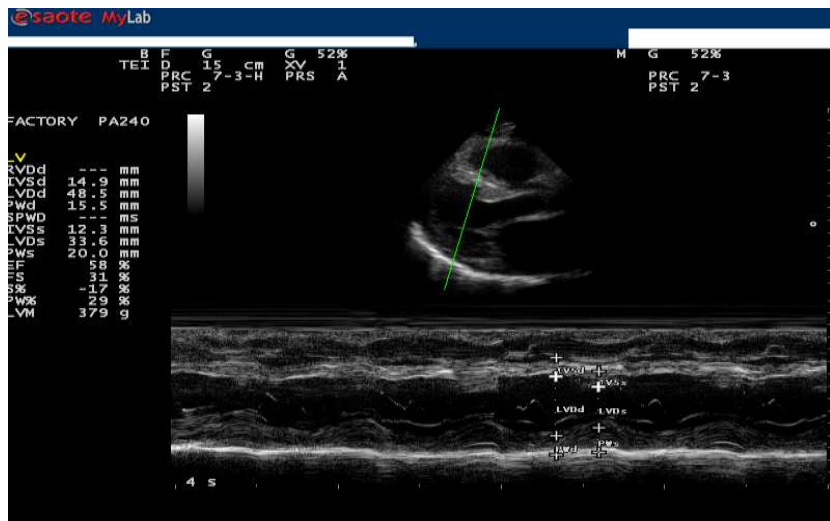
- (image.7)A 76-year-old hypertensive male patient with severe concentric LVH with mildly impaired LV systolic function and severe diastolic dysfunction;,LVEDD34 mm; LVESD 25.1 mm; EF 51%.



- (image.8)A 50-year-old hypertensive female patient with mild concentric LVH with grade I diastolic dysfunction, Normal LV systolic function, estimated EF 55 % ,LVEDD 50.9 mm; LVESD 15.5 mm .No RWMA at rest.



(image.9)A 85-year-old hypertensive male patient with moderate concentric LVH with preserved LV systolic function, estimated EF 57 % ,LVEDD 45.3 mm; LVESD 14.2 mm. No RWMA at rest.



(image.10)A 60-year-old hypertensive male patient with moderate concentric LVH with grade II diastolic dysfunction, Normal LV systolic function, estimated EF 58 % . ,LVEDD 48.5 mm; LVESD 14.9 mm . No RWMA at rest.