بسم الله الرحمن الرحيم

Sudan University of sciences and Technology

College of Graduate studies

Measurement of the Main Pulmonary Artery Diameter in Sudanese Patients by Using Computed Tomography

قياس قطر الشريان الرئوي الرئيسي لدي السودانيين باستخدام الأشعة المقطعية المحوسبه

A thesis submitted for partial fulfillment for the requirements of M.Sc degree in diagnostic radiological technology

By

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الآية

بِسْمِ اللهِّ الرَّحْمَنِ الرِّحيمِ

قال تعالي:(وَلَوْ أَنَّما فِي الأَرْضِ مِنْ شَجَرَةٍ أَقْلاَمٌ وَالْبَحْرُ يَمُدُّه مِنْ بَعْدِهِ سَبْعَةُ أَبْحُرٍ مَا نَفِدَتْ كَلِمَاتُ اللهِّ إِنَّ اللهَّ عَزِيزٌ حَكِيمٌ)

سورة لقمان الآية (27)

Dedication

To

My Family

My Teachers

My Friends

.....

Acknowledgement

This work was carried out under the Will of Allah.

I am extremely grateful to many people who supported me during the preparation of this study.

I would like to express my deep gratitude to my supervisor Ustaz Abd alkareem Altayb

My thanks also go to the staff of radiology department of Al Gazeira Traumatology center

Finally, I would like to sincerely thank my family for their support.

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List of abbreviations:-

CT	Computed Tomography
CAT	Computed axial tomography
SVC	Superior vena cava
IVC	Inferior vena cava
AV	Atrio ventricular
CHF	Congestive heart failure
PH	Pulmonary hypertension
PAP	Pulmonary artery pressure
MPAD	Main pulmonary artery diameter
Fig	Figure
PE	Pulmonary embolism
SA	Sino a trial
ATP	Adenosine tri phosphate
Bpm	Beat per minute

Contents:-

Abstract

The study was done to measure the main pulmonary artery diameter by Computed Tomography in normal subjects which were not suffer from the cardiac or pulmonary pathology .

Fifty patients (20 males and 30 females) were selected with normal pulmonary artery pressure .

This study was done in ALgazeira Traumatology center during the period from October 2015 to August 2016 .

The widest diameter perpendicular to the long axis of the main pulmonary artery was measure with computer caliper at the level of the bifurcation of the it to the right and left pulmonary arteries .

This study showed the mean of the MPAD is 26.30± 4.29mm.

The results showed there was well correlation between MPAD and age , gender and body mass index (BMI) .

In other word , this study found the mean of the main pulmonary artery diameter in males is larger than females (27.99mm in males and 25.11mm in females) .

Also this study found well correlation between the main pulmonary artery diameter and body mass index (BMI) . That observed the over weight patients had body mass index ≥30mm .

ملخص البحث

هده الدراسة أجريت لقياس قطر الشريان الرئوي الرئيس باستخدام الاشعه المقطعية المحوسبه في الأشخاص الدين لا يعانون من أمراض القلب أو أمراض الشرايين الرئوية .

أجريت هده الدراسة في مركز الجزيرة للإصابات في الفترة من أكتوبر 2015 إلي أغسطس 2016

وقد تم قياس قطر الشريان الرئوي الرئيس في مستوي قبل انقسامه إلي الشريان الرئوي الأيمن والأيسر

وجدت هده الدراسة إن قطر الشريان الرئوي الرئيس في الأشخاص الدين لا يعانون من أمراض القلب او الشرايين هو 26.30±4.29ملم

أيضا وجدت إن هنالك علاقة قويه بين قطر الشريان الرئوي الرئيسي مع العمر، الجنس و مؤشر كتله الجسم .

فقد وجدت هده الدراسة إن متوسط قطر الشريان الرئوي في الذكور اكبر من الإناث (27.99 ملم في الذكور ، 25.11ملم في الإناث) .

أيضا وجدت هده الدراسة إن هنالك ارتباط وثيق بين قطر الشريان الرئوي الرئيس و مؤشر كتله الجسم ، معظم الأشخاص أصحاب الوزن الزائد قطر الشريان الرئوي اكبر من أو يساوي 30 ملم .

Chapter One

Introduction

1-1 Introduction:-

Heart and lungs together form the circulatory and cardiovascular systems of our body due to which the oxygen rich blood is efficiently reached to every part of the body and the oxygen poor blood or the deoxygenated blood is returned back to the lungs for purification. Pulmonary artery is one of the most important parts of the whole heart anatomy and performs a unique function in the whole blood transportation system.

Pulmonary artery is one of the two vessels branching from the pulmonary trunk is an integral part of the whole heart anatomy and is the vessel that transports de-oxygenated blood to the lungs coming from the right ventricles. The right pulmonary artery is longer of the two arteries. It passes transversely midline across the upper chest below the aortic arch and enters the Hilum in the right lung. The left pulmonary artery is the shorter one and enters the Hilum of the left lung. Although almost all arteries carry oxygen rich blood, pulmonary artery is an exception and instead of carrying blood to the body parts it transports the oxygen poor blood into the lungs for purification. Most of the arteries divide from aorta but the pulmonary artery goes further from the heart's right ventricle into the left and the right pulmonary arteries.(Snell 1995).

Right cardiac catheterization is considered to be a gold standard for measuring pulmonary artery pressure (PAP). However, this is an invasive procedure and carries a risk of mortality and morbidity. Therefore, researchers have carried out several studies seeking a reliable and reproducible diagnostic imaging method for the assessment of the pulmonary artery diameter in order to predict the PAP. Some investigators have found reasonable correlations with pulmonary arterial size and PAP in studies with chest radiography . In addition, it has been reported that measurement of the pulmonary artery size by chest

radiography is poorly reliable as a method for the examination of pulmonary artery diameter. Several factors contribute to the problem: superposition of the mediastinal and hilar structures; concurrent parenchymal diseases; architectural distortion; and magnification differences .

After the introduction of helical CT, several studies have been performed to measure the pulmonary artery diameter, and have shown that the increase in the main pulmonary artery diameter (MPAD) is a reliable indicator of pulmonary hypertension (PH) . However, there areonly a few studies with small series that measure the MPAD in normal individuals by CT to determine the normal range of the pulmonary artery diameter . (Edward et al 1998)

1-2 problem of the study:-

The range of the diameter of the pulmonary artery is not fully shown in the current literature such as chest radiography and right cardiac catheterization .

1-3 Objectives:-

1-3-1 General objective:-

To measure the main pulmonary artery diameter by using computed tomography .

1-3-2 Specific objectives:-

To determine the normal range of the main pulmonary artery in person with normal pulmonary artery pressure .

To evaluate the relationship of the diameter with age ,gender and body mass index (BMI).

1-4 Significance of the study:

Contrast enhanced computed tomography of the thorax might be useful for measuring diameter of the main pulmonary artery .

1-5 The Overview of the research:-

Chapter one deals with introduction, problem, objectives, significance and overview of the research. Chapter two deals with literature review including theoretical background (anatomy, physiology and pathology) and previous studies. Chapter three deals with research Materials and Methods. Chapter four deals with results and finally chapter five deals with discussion, conclusion and recommendations.

Chapter two

Literature review

Literature Review

2-1 Theoretical background:-

2-1-1 Anatomy:-

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

An electrical system regulates the heart and uses electrical signals to contract the heart's walls. When the walls contract, blood is pumped into the circulatory system. A system of inlet and outlet valves in the heart chambers work to ensure that blood flows in the right direction. The heart is vital to your health and nearly everything that goes on in the body. Without the heart's pumping action, blood can't circulate within the body.

Blood carries the oxygen and nutrients that your organs need to work normally. Blood also carries carbon dioxide, a waste product, to your lungs to be passed out of the body and into the air. A healthy heart supplies the areas of the body with the right amount of blood at the rate needed to work normally. If disease or injury weakens the heart, the body's organs won't receive enough blood to work normally.

2-1-1-1 Location, Size and Shape of the Heart:-

The heart is located underneath the sternum in a thoracic compartment called the mediastinum, which occupies the space between the lungs. It is approximately the size of a man’s fist (250-350grams) and is shaped like an inverted cone. The narrow end of the heart is called the apex. It is directed downward and to the left and lie just above the arch of the

diaphragm at the approximate level of the fifth or sixth rib. The broad end of the heart is called the base and gives rise to the major blood vessels, which is directed upwards and to the right and lies at the approximate level of the second rib.

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

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

At the roots of the major blood vessels, the parietal pericardium reflects back over the surface of the heart to form the inner wall of the pericardium, the visceral pericardium. Because it is the outer layer of the heart wall, the visceral pericardium is referred to as the epicardium. Together, the parietal and visceral pericardial layers are also called the serous pericardium.

2-1-1-2 Chambers-Vessels and Valves:-

The walls of the four chambers of the heart are made of cardiac muscle called the myocardium. The chamber sare lined with endocardium, simple squamous epithelium that also covers the valves of the heart and continues into the vessels as their lining (endothelium).The important physical characteristic of the endocardium is not its thinness, but rather its smoothness. This very smooth tissue prevents abnormal blood clotting, because clotting would be initiated by contact of blood with a rough surface.

The upper chambers of the heart are the right and left atria (singular: atrium), which have relatively thin walls and are separated by a common wall of myocardium called the interatrial septum. The lower chambers are the right and left ventricles, which have thicker walls and are separated by the interventricular septum . As you will see, the atria receive blood, either from the body or the lungs, and the ventricles pump blood to either the lungs or the body. (Valerie and Tina 2007).

2-1-1-2-1 Right Atrium:-

The two large caval veins return blood from the body to the right atrium The superior vena cava carries blood from the upper body, and the inferior vena cava carries blood from the lower body. From the right atrium, blood will flow through the right atrioventricular (AV) valve, or tricuspid valve, into the right ventricle. The tricuspid valve is made of three flaps (or cusps) of endocardium reinforced with connective tissue. The general purpose of all valves in the circulatory system is to prevent backflow of blood. The specific purpose of the tricuspid valve is to prevent backflow of blood from the right ventricle to the right atrium when the right ventricle contracts. As the ventricle contracts, blood is

forced behind the three valve flaps, forcing them upward and together to close the valve. (Valerie and Tina 2007)

2-1-1-2-2 Left Atrium:-

The left atrium receives blood from the lungs, by way of four pulmonary veins. This blood will then flow into the left ventricle through the left atrioventricular (AV) valve, also called the mitral valve or bicuspid (two flaps) valve. The mitral valve prevents back flow of blood from the left ventricle to the left atrium when the left ventricle contracts.

Another function of the atria is the production of a hormone involved in blood pressure maintenance. When the walls of the atria are stretched by increased blood volume or blood pressure, the cells produce atrial natriuretic peptide (ANP), also called atrialnatriuretic hormone (ANH). (The ventricles of the heart produce a similar hormone called B-type natriureticpeptide, or BNP, but we will use ANP as there presentative cardiac hormone.) ANP decreases there absorption of sodium ions by the kidneys, so that more sodium ions are excreted in urine, which in turn increases the elimination of water. The loss of water lowers blood volume and blood pressure. You may have noticed that ANP is an antagonist to the hormone aldosterone, which raises blood pressure. (Valerie and Tina 2007).

2-1-1-2-3 Right Ventricle:-

When the right ventricle contracts, the tricuspid valve closes and the blood is pumped to the lungs through the pulmonary artery (or trunk). At the junction of this large artery and the right ventricle is the pulmonary

semilunar valve (or more simply, pulmonary valve).Its three flaps are forced open when the right ventricle contracts and pumps blood into the pulmonary artery. When the right ventricle relaxes, blood tends to come back, but this fills the valve flaps and closes the pulmonary valve to prevent backflow of blood into the right ventricle.

Projecting into the lower part of the right ventricle are columns of myocardium called papillary muscles. Strands of fibrous connective tissue, the chordate tendineae, extend from the papillary muscles to the flaps of the tricuspid valve. When the right ventricle contracts, the papillary muscles also contract and pull on the chordae tendineae to prevent inversion of the tricuspid valve. If you have ever had your umbrella blown inside out by a strong wind, you can see what would happen if the flaps of the tricuspid valve were not anchored by the chordae tendineae and papillary muscles.

2-1-1-2-4 Left Ventricle:-

The walls of the left ventricle are thicker than those of the right ventricle, which enables the left ventricle to contract more forcefully. The left ventricle pumps blood to the body through the aorta, the largest artery of the body. At the junction of the aorta and the left ventricle is the aortic semilunar valve (or aortic valve). This valve is opened by the force of contraction of the left ventricle, which also closes the mitral valve. The aortic valve closes when the left ventricle relaxes, to prevent backflow of blood from the aorta to the left ventricle. When the mitral(left AV) valve closes, it prevents backflow of blood to the left atrium; the flaps of the mitral valve are also anchored by chordae tendineae and papillary muscles.

As you can see from this description of the chambers and their vessels, the heart is really a double, or two-sided, pump. The right side of the

heart receives deoxygenated blood from the body and pumps it to the lungs to pick up oxygen and release carbon dioxide. The left side of the heart receives oxygenated blood from the lungs and pumps it to the body. Both pumps work simultaneously; that is, both atria contract together, followed by the contraction of both ventricles. (Valerie and Tina, 2007).

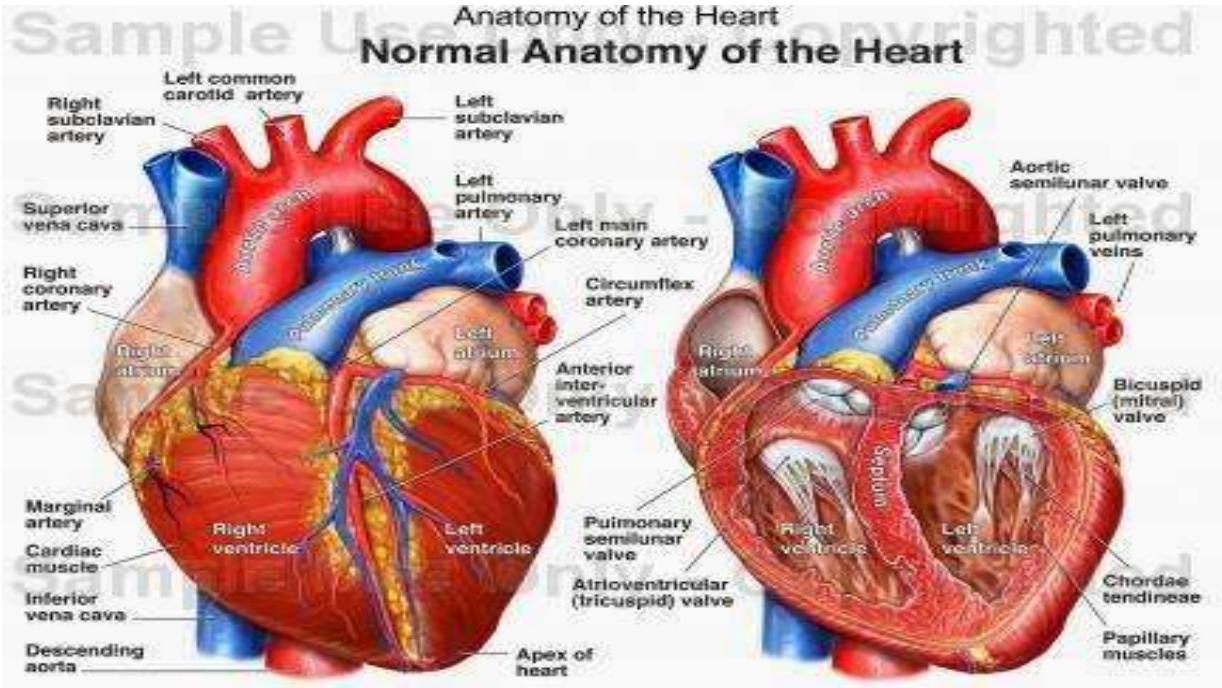

Fig (2-1) The heart Anatomy

2-1-1-3 Arterial supply of the heart:-

The arterial supply of the heart is provided by the right and left coronary arteries, which arise from the aorta immediately above the aortic valve.

The coronary arteries, the first branches of the aorta, the right and left coronary artery arise from the corresponding aortic sinuses at the

proximal part of the ascending aorta. The coronary arteries supply both the atria and ventricles (Keith and Arthur 2006).

Table (2-1): blood supply of the anatomical regions of the heart (Richard .E, Klabunde 2004)

Anatomic region of the heart	Coronary artery(most likely associated)
Inferior	Right coronary
Antero septal	Left anterior descending
Antero apical	Left anterior descending (distal)
Antero lateral	Circumflex
Posterior	Right coronary artery

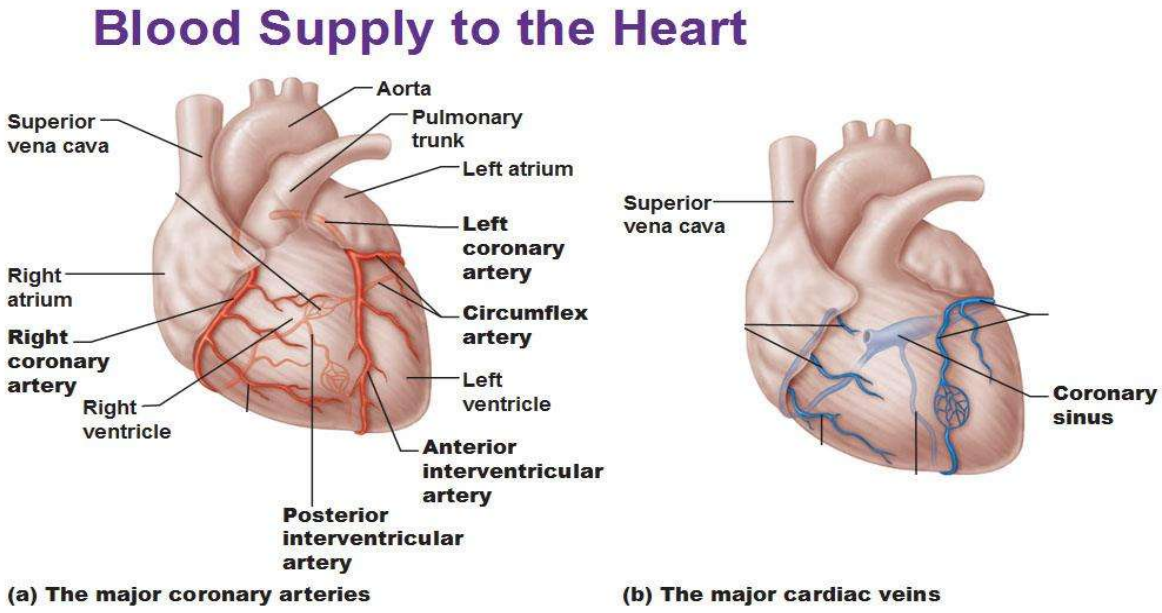

Fig (2-2) The blood supply of the heart

2-1-1-4 Venous drainage of the heart:-

Most of the blood from the heart wall drains into the right atrium through the coronary sinus. This lies in the posterior part of the atrio-ventricular 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 cardiac vein and the middle cardiac vein are tributaries of the coronary sinus. The remainder of the blood is returned to the right atrium by the anterior cardiac vein and also by small veins that open directly into the heart chambers. (Richard 2005).

2-1-1-5 Great Vessels:-

Blood travels to and from the heart through the great vessels, which include the aorta, pulmonary arteries and veins, and superior and inferior venae cavae .The aorta is the largest artery of the body and can be divided into the ascending aorta, aortic arch, and descending aorta. The ascending aorta begins at the base of the left ventricle at the level of the sternal angle, then curves superiorly and posteriorly as the aortic arch over the root of the left lung. The top of the aortic arch is approximately at T3 The arch continues as the descending aorta posterior to the left bronchus and pulmonary trunk, on the left side of the vertebral body of T4 .The descending aorta passes slightly anterior and to the left of the vertebral column as it descends through the thoracic and abdominal cavities. While in the thoracic cavity, the descending aorta is commonly called the thoracic aorta, and while in the abdominal cavity, it is called the abdominal aorta. The pulmonary trunk is the origin of the right and left pulmonary arteries and lies entirely within the pericardial sac. It arises from the right ventricle and ascends in front of the ascending aorta, courses posteriorly and to the left, where it bifurcates at the level of the sternal angle (T4) into the right and left pulmonary arteries .The

pulmonary trunk is attached to the aortic arch by a fibrous cord called the ligamentum arteriosum, the remnant of an important fetal blood vessel (ductus arteriosus) that links the pulmonary an systemic circuits during fetal development.

The right pulmonary artery courses laterally, posterior to the ascending aorta and superior vena cava, and anterior to the esophagus and right mainstem bronchus, to the hilum of the right lung. At the root of the right lung, the right pulmonary artery divides into two branches, with the lower branch supplying the middle and inferior lobes and the upper branch supplying the superior lobe .

The left pulmonary artery, shorter and smaller than the right, is also the most superior of the pulmonary vessels. It travels horizontally, arching over the left mainstem bronchus, and enters the hilum of the left lung just superior to the left mainstem bronchus .Within the lungs, each pulmonary artery descends posterolateral to the main bronchus and divides into lobar and segmental arteries, continuing to branch out and to follow along with the smallest divisions of the bronchial tree Located inferior to the pulmonary arteries are the four pulmonary veins, two each (superior and inferior) extending from each lung to enter the left atrium .(Jean Wilke and Rebecca Swisher 2007).

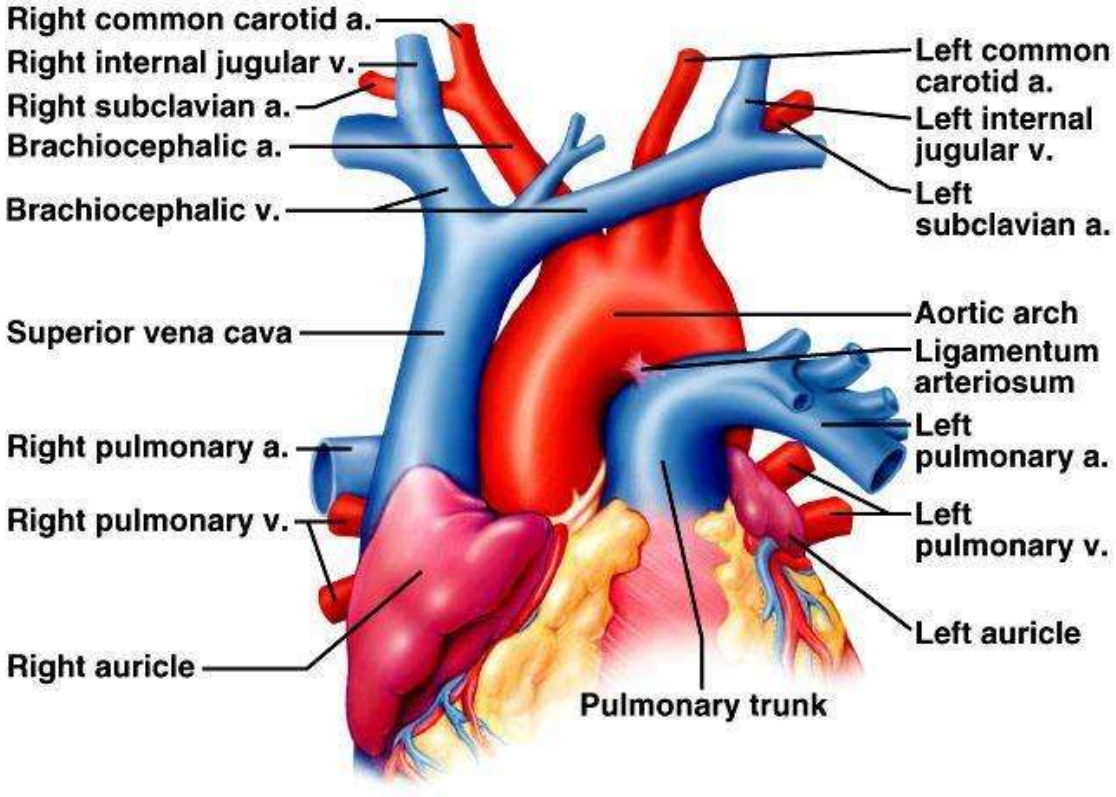

fig (2-3) Great vessels of the heart

2-1-2 Physiology:-

The heart is muscular organ of the circularity system that constantly pumps blood throughout the body. The human heart is actually two pumps in one. The right side receives oxygen poor blood from the various region of the body and delivers it to the lungs. In the lungs, oxygen is absorbed in the blood. The left side of the heart receives the oxygen rich blood from the lungs and delivers it to the rest of the body (William2003).

The heart has four separate compartment or chambers, the upper chamber on each side of the heart, which is called an atrium, receives and collects the blood coming to the heart. The atrium then delivers blood to the powerful lower chamber, called a ventricle, which pumps blood away from the heart through powerful and rhythmic contractions.

2-1-2-1 Cardiac Cycle:-

Cardiac cycle is the term used to describe the relaxation and contraction that occur, as a heart works to pump blood through the body. Heart rate is a term used to describe the frequency of the cardiac cycle. It is considered one of the four vital signs. Usually it is calculated as the number of contractions (heart beats) of the heart in one minute and expressed as "beats per minute" (bpm). When resting, the adult human heart beats at about 70 bpm (males) and 75 bpm (females), but this rate varies between people. However, the reference range is nominally between 60 bpm (if less termed bradycardia) and 100 bpm (if greater, termed tachycardia). Resting heart rates can be significantly lower in athletes, and significantly higher in the obese.

The pulse is the most straightforward way of measuring the heart rate, but it can be deceptive when some strokes do not lead to much cardiac output. In these cases (as happens in some arrhythmias), the heart rate may be considerably higher than the pulse. Every single 'beat' of the heart involves three major stages: atrial systole, ventricular systole and complete cardiac diastole. Throughout the cardiac cycle, the blood pressure increases and decreases. As ventricles contract the pressure rise, causing the AV valve to slam shut (Provophys et al 2006)

2-1-2-1-1 Systole:-

Systole, or contraction, of the heart is initiated by the electrical cells of the sinoatrial node, which is the heart's natural pacemaker. These cells are activated spontaneously by depolarization of their membranes beyond a certain threshold for excitation. At this point, voltage gated calcium channels on the cell membrane open and allow calcium ions to pass through, into the sarcoplasm, or interior, of the muscle cell. Some calcium ions bind to receptors on the sarcoplasmic reticulum causing an influx of calcium ions into the sarcoplasm. The calcium ions bind to the troponin, causing a conformation change, breaking the bond between the protein tropomyosin, to which the troponin is attached, and the myosin binding sites. This allows the myosin heads to bind to the myosin binding sites on the actin protein filament and contraction results as the myosin heads draw the actin filaments along, are bound by ATP, causing them to release the actin, and return to their original position, breaking down the ATP into ADP and a phosphate group. The action potential spreads via the passage of sodium ions through the gap junctions that connect the sarcoplasm of adjacent myocardial cells (Provophys et al 2006).

2-1-2-1-2 Diastole:-

The heart in the diastole phase, Cardiac Diastole is the period of time when the heart relaxes after contraction in preparation for refilling with circulating blood. Ventricular diastole is when the ventricles are relaxing, while atrial diastole is when the atria are relaxing. Together they are known as complete cardiac diastole. During ventricular diastole, the pressure in the (left and right) ventricles drops from the peak that it reaches in systole. When the pressure in the left ventricle drops to below the pressure in the left atrium, the mitral valve opens, and the left ventricle fills with blood that was accumulating in the left atrium. Likewise, when the pressure in the right ventricle drops below that in the right atrium, the tricuspid valve opens and the right ventricle fills with blood that was in the right atrium (Provophys et al 2006)

2-1-2-2 Heart Sounds:-

Two sounds are normally heard through a stethoscope during each cardiac cycle. The first is a low, slightly prolonged “lub (first sound), caused by vibrations set up by the sudden closure of the AV valves at the start of ventricular systole. The second is a shorter, high-pitched “dup” (second sound), caused by vibrations associated with closure of the aortic and pulmonary valves just after the end of ventricular systole. A soft, low-pitched third sound is heard about one third of the way through diastole in many normal young individuals. It coincides with the period of rapid ventricular filling and is probably due to vibrations set up by the inrush of blood. A fourth sound can sometimes be heard immediately before the first sound when atrial pressure is high or the ventricle is stiff in conditions such as ventricular hypertrophy. It is due to ventricular filling and is rarely heard in normal adults.

The first sound has a duration of about 0.15 s and a frequency of 25 to 45 Hz. It is soft when the heart rate is low, because the ventricles are well filled with blood and the leaflets of the AV valves float together before systole. The second sound lasts about 0.12 s, with a frequency of 50 Hz. It is loud and sharp when the diastolic pressure in the aorta or pulmonary artery is elevated, causing the respective valves to shut briskly at the end of systole. The interval between aortic and pulmonary valve closure during inspiration is frequently long enough for the second sound to be reduplicated (physiologic splitting of the second sound). Splitting also occurs in various diseases. The third sound, when present, has a duration of 0.1s (Barrett et al, 2010).

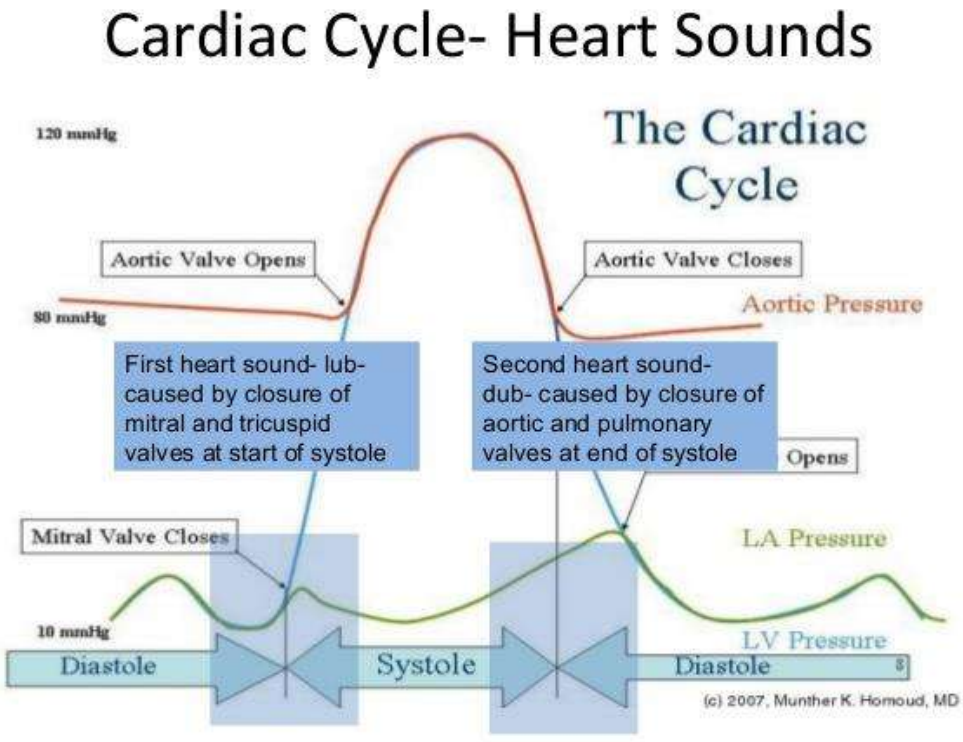

Fig (2-4): cardiac cycle and heart sound

2-1-2-3 Function of the Atria:-

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 conditions 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 and Hall, 2006).

2-1-2-4 Function 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 .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 and Hall, 2006)

2-1-2-5 Function of the Valves:-

2-1-2-5-1 Atrioventricular Valves:-

The A-V valves (the tricuspid and mitral valves) prevent backflow of blood from the ventricles to the atria during systole, and the semilunar valves (the aortic and pulmonary artery valves) prevent backflow from the aorta and pulmonary arteries into the ventricles during diastole. These valves, shown in Figure 9–6 for the left ventricle, close and open passively. That is, they close when a backward pressure gradient pushes blood backward, and they open when a forward pressure gradient forces blood in the forward direction. For anatomical reasons, the thin, filmy A-V valves require almost no backflow to cause closure, whereas the much heavier semilunar valves require rather rapid backflow for a few milliseconds.

2-1-2-5-2 Aortic and Pulmonary Artery Valves:-

The aortic and pulmonary artery semilunar valves function quite differently from the A-V valves. First, the high pressures in the arteries at the end of systole cause the semilunar valves to snap to the closed position, in contrast to the much softer closure of the A-V valves. Second, because of smaller openings, the velocity of blood ejection through the aortic and pulmonary valves is far greater than that through the much larger A-V valves. Also, because of the rapid closure and rapid ejection, the edges of the aortic and pulmonary valves are subjected to much greater mechanical abrasion than are the A-V valves. Finally, the A-V

valves are supported by the chordae tendineae, which is not true for the semilunar valves. It is obvious from the anatomy of the aortic and pulmonary valves that they must be constructed with an especially strong yet very pliable fibrous tissue base to withstand the extra physical stresses (Guyton and Hall, 2006).

2-1-2-6 The Heart's Electrical Conduction System:-

The heart is primarily made up of muscle tissue. A network of nerve fibers coordinates the contraction and relaxation of the cardiac muscle tissue to obtain an efficient, wave-like pumping action of the heart.

The structures that make up the conduction system are the sinoatrial node (SA node), the internodal atrial pathways, the atrioventricular node (AV node), the bundle of His and its branches, and the Purkinje system. The various parts of the conduction system and, under abnormal conditions, parts of the myocardium, are capable of spontaneous discharge. However, the SA node normally discharges most rapidly, with depolarization spreading from it to the other regions before they discharge spontaneously. The SA node is therefore the normal cardiac pacemaker, with its rate of discharge determining the rate at which the heart beats. Impulses generated in the SA node pass through the atrial pathways to the AV node, through this node to the bundle of His, and through the branches of the bundle of His via the Purkinje system to the ventricular muscle (Barrett et al, 2010).

2-1-2-7 Control of Heartbeat:-

The heart contains two cardiac pacemakers that spontaneously cause the heart to beat. These can be controlled by the autonomic nervous system and circulating adrenaline. If the cardiac muscles just contracted and relaxed randomly at a natural rhythm the cycle would become disordered and the heart would become unable to carry on its function of being a pump. Sometimes when the heart undergoes great damage to one part of the cardiac muscle or the person incurs an electric shock, the cardiac cycle can become uncoordinated and chaotic. Some parts of the heart will contract whilst others will relax so that instead of contracting and relaxing as a whole, the heart will flutter abnormally. This is called fibrillation and can be fatal if not treated within 1 minute.

2-1-2-8 Cardiac Muscle Contraction:-

After an action potential excites the plasma membrane of the cardiac muscle cell the contraction is due to an increase in the cytoplasmic concentration of Calcium ions. Similar to skeletal muscle, the release of Ca+ ions from the sarcoplasmic reticulum binds to troponin which allows actin to bind with myosin. The difference between skeletal muscle and cardiac muscle is that when the action potential opens voltage gated calcium ion channels in the T-tubules. The increase in cytosolic calcium causes calcium ions to bind to receptors on the surface of the sarcoplasmic reticulum. The binding of calcium ions to these receptors causes the opening of more calcium ion channels in the SR membrane. Calcium ions then rush out of the SR and bind to troponin and allow the myosin and actin to bind together which causes contraction. This sequence is called calcium-induced calcium release. Contraction ends

when the level of cytosolic calcium returns to normal resting levels (Barrett et al 2010).

2-1-2-9 Blood Pressure:-

Blood pressure is the pressure exerted by the blood on the walls of the blood vessels. Unless indicated otherwise, blood pressure refers to systemic arterial blood pressure, i.e., the pressure in the large arteries delivering blood to body parts other than the lungs, such as the brachial artery (in the arm). The pressure of the blood in other vessels is lower than the arterial pressure. Blood pressure values are universally stated in millimeters of mercury (mm Hg). The systolic pressure is defined as the peak pressure in the arteries during the cardiac cycle; the diastolic pressure is the lowest pressure (at the resting phase of the cardiac cycle). The mean arterial pressure and pulse pressure are other important quantities. Typical values for a resting, healthy adult are approximately 120 mm Hg systolic and 80 mm Hg diastolic (written as 120/80 mm Hg), with large individual variations. These measures of blood pressure are not static, but undergo natural variations from one heartbeat to another or throughout the day (in a circadian rhythm); they also change in response to stress, nutritional factors, drugs, or disease (Provophys et al, 2006).

2-1-2-9-1 Systolic Pressure:-

Systolic Pressure is the highest when the blood is being pumped out of the left ventricle into the aorta during ventricular systole. The average high during systole is 120 mm Hg.

2-1-2-9-2 Diastolic Pressure:-

Diastolic blood pressure lowers steadily lowers to a low of 80 mm Hg during ventricular diastole (Provophys et al, 2006)

2-1-3 Pathology of the heart:-

2-1-3-1 Heart Failure:-

Heart failure (also called congestive heart failure or CHF) is a frequent end point of many of the conditions mentioned above. In the United States alone, CHF affects nearly 5 million individuals annually, necessitating >1million hospitalizations, and contributes to death of 300,000 patients a year. Most heart failure is the consequence of systolic dysfunction, the progressive deterioration of myocardial contractile function; this is most commonly due to ischemic heart disease or hypertension. However, in 20% to 50% of patients the heart contracts normally but relaxation is abnormal. These patients with "diastolic" failure are generally older and more likely to be female with hypertension or diabetes mellitus. Heart failure may be caused by valve failure (e.g., endocarditis) or can also occur in normal hearts suddenly burdened with an abnormal load (e.g., fluid or pressure overload).

In heart failure, the heart is unable to pump blood at rate that meets the requirements of the metabolizing tissues, or can only do so only with filling pressures that are higher than normal. Onset may be insidious or acute. In most cases of CHF the heart cannot keep pace with basic peripheral demands; in a minority of cases, heart failure results from greatly increased tissue demands for blood (high-output failure). Excluded from the definition are conditions in which inadequate cardiac output occurs because of blood loss or some other process that impairs blood return to the heart.

In a mechanical sense, the failing heart in CHF can no longer pump the blood delivered to it by the venous circulation. Inadequate cardiac output—called forward failure—is almost always accompanied by increased congestion of the venous circulation (backward failure),

because the failing ventricle is unable to eject the venous blood delivered to it. This results in an increased end diastolic ventricular volume, leading to increased end diastolic pressures and, finally, elevated venous pressures.

Although the root problem in CHF is typically abnormal cardiac function, virtually every other organ is eventually affected by some combination of forward and backward failure.

The cardiovascular system can adapt to reduced myocardial contractility or increased hemodynamic burden by a few different pathways. The most important are

• Activation of neurohumoral systems, especially

(1) release of the nuro transmitter norepinephrine by the sympathetic nervous system (increases heart rate and augments myocardial contractility and vascular resistance),

(2) activation of the renin-angiotensinaldosterone system.

(3) release of atrial natriureticpeptide (ANP).

This is a polypeptide hormone secreted by the atria in the setting of atrial distension. It causes vasodilation, naturiuresis, and diuresis that help alleviate volume or pressure overload states.

• The Frank-Starling mechanism. As cardiac failure progresses, end-diastolic pressures increase, causing individual cardiac muscle fibers to stretch; this ultimately increases the volume of the cardiac chamber. In accordance with the Frank-Starling relationship, these length ened fibers initially contract more forcibly, thereby increasing cardiac output. If the dilated ventricleis 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. However, increasing dilation increases ventricular wall tension, which increases 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 entera phase termed decompensated heart failure.

• Myocardial structural changes, including augmentedmuscle mass (hypertrophy), to increase the mass of contractile tissue. Because adult cardiac myocytes cannot proliferate, adaptation to a chronically increased workload involves hypertrophy of individual muscle cells. In pressure overload states (e.g., hypertension,valvular stenosis), the hypertrophy is characterized by increased diameter of individual muscle fibers. This yields concentric hypertrophy, in which the thickness of the ventricular wall increases without an increase in the size of the chamber. In volume overload states (e.g., valvular regurgitation or abnormal shunts),it is the length of individual muscle fibers that increases. This results in eccentric hypertrophy, characterized by an increase in heart size as well as an increase in wall thickness.

Initially, these adaptive mechanisms may be adequate to maintain cardiac output in the face of declining cardiac performance. However, with sustained orworsening heart function, pathologic changes may eventually supervene, resulting in structural and functional disturbances; such degenerative changes include myocyte apoptosis, cyto skeletal alterations, and altered extracellular matrix synthesis and remodeling. Even hypertrophy comes at a significant cost to the cell. Oxygen requirements of the hypertrophic myocardium are increased as a result of increased myocardial cell mass and increased tension of the ventricular wall .Because the myocardial capillary bed does not always increase in step with the increased oxygen demands of the hypertrophic muscle fibers, the myocardium becomes vulnerable to ischemic injury.

Heart failure can affect predominantly the left side or the right side, or both sides of the heart. The most common causes of left-sided cardiac failure are (1) IHD,(2) systemic hypertension, (3) mitral or aortic valve

disease, and (4) 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 can 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 primary pulmonic or tricuspid valve disease. It sometimes follows congenital heart diseases, i.e., in the setting of left-to-right shunts with chronic volume and pressure overloads.(Kumar etal 2007).

2-1-3-1-1 Left-Sided Heart Failure:-

The morphologic and clinical effects of left-sided CHF primarily result from progressive damming of blood within the pulmonary circulation and the consequences of diminished peripheral blood pressure and flow. Other manifestations of left ventricular failure include an enlarged heart (cardiomegaly), tachycardia, a third heart sound (S3), and fine rales at the lung bases, produced by respirations through edematous pulmonary alveoli. With progressive ventricular dilation, the papillary muscles are displaced laterally, causing mitral regurgitation and a systolic murmur. Subsequent chronic dilation of the left atrium is often associated with atrial fibrillation, manifested by an “irregularly irregular” heartbeat (Kumar 2007).

2-1-3-1-2 Right-Sided Heart Failure:-

Right-sided heart failure is usually the consequence of left-sided heart failure; any pressure increase in the pulmonary circulation inevitably produces an increased burden on the right side of the heart. Isolated right-sided heart failure is less common and it occurs in patients with intrinsic disease of lung parenchyma and/or pulmonary vasculature that result in chronic pulmonary hypertension (corpulmonale). It can also occur in patients with pulmonic or tricuspid valve disease. Congenital heart diseases with right-to-left shunt can cause isolated right sided heart failure, as well. Hypertrophy and dilation are generally confined to the right ventricle and atrium, although bulging of the ventricular septum to the left can cause dysfunction of the left ventricle.

The major morphologic and clinical effects of pure right-sided heart failure differ from those of left-sided heart failure in that pulmonary congestion is minimal, whereas engorgement of the systemic and portal venous systems is typically pronounced.

Clinical Features. Dyspnea (breathlessness) is usually the earliest and most significant complaint of patients in left sided heart failure; cough is also a common accompaniment of left heart failure due to fluid transudation into airspaces. With further cardiac impairment, patients develop dyspnea when recumbent (so-called orthopnea); this occurs because of increased venous return from the lower extremities and by elevation of the diaphragm when in the supine position. Orthopnea is typically relieved by sitting or standing, so that such patients usually sleep while sitting upright. Paroxysmal nocturnal dyspnea is a particularly dramatic form of breathlessness awakening patients from sleep with attacks of extreme dyspnea bordering on suffocation.

Clinical Features. While the symptoms of left-sided heart failure are largely due to pulmonary congestion and edema, pure right-sided heart failure typically causes very few respiratory symptoms. Instead, there is systemic and portal venous congestion, with hepatic and splenic enlargement, peripheral edema, pleural effusion, and ascites. It is worth emphasizing, however, that in most cases of chronic cardiac decompensation, patients present with biventricular CHF, encompassing the clinical syndromes of both right-sided and left-sided heart failure. As CHF progresses, patients can become frankly cyanotic and acidotic, as a result of decreased tissue perfusion (Kumar 2007).

2-1-3-2 Hypertensive Vascular Disease:-

Systemic and local blood pressure must be tightly regulated .Low pressures result in inadequate organ perfusion, leading to dysfunction and/or tissue death. Conversely, high pressures that drive blood flow in excess of metabolic demands provide no additional benefit but result in blood vessel and end-organ damage. Elevated blood pressure is called hypertension; as we saw previously, it is one of the major risk factors for atherosclerosis. Here we will first discuss the mechanisms of normal blood pressure control, followed by pathways that may under lie hypertension, and finally the pathologic changes in vessels associated with hypertension.

Although hypertension is a common health problem with occasionally devastating outcomes, it typically remains asymptomatic until late in its course. Besides contributing to the pathogenesis of coronary heart disease and cerebro vascular accidents, hypertension can also cause cardiac hypertrophy and heart failure (hypertensive heart disease), aortic dissection, and renal failure. Although we have an improving understanding of them molecular pathways that regulate normal blood

pressure, the mechanisms of hypertension in the vast majority of people remain unknown; consequently, we refer to most of these as “essential hypertension.

Like height and weight, blood pressure is a continuously distributed variable, with essential hypertension at one end of the distribution rather than a distinct entity. The detrimental effects of blood pressure increase continuously as the pressure rises; no rigidly defined threshold level of blood pressure distinguishes risk from safety .Nevertheless, a sustained diastolic pressure greater than90mmHg, or a sustained systolic pressure in excess of 140 mmHg, constitutes hypertension; systolic blood pressure is more important than diastolic blood pressure in determining cardiovascular risk. By either criteria, some 25% of individuals in the general population are hypertensive. The prevalence and vulnerability to complications increase with age; they are also higher in African Americans. Reduction of blood pressure dramatically reduces the incidence and death rates from IHD, heart failure, and stroke (Kumar, 2007).

2-1-3-3 **Pulmonary Vascular Disease:**

The definition of pulmonary vascular disease is simple: any condition that affects the blood vessels along the route between the heart and lungs Blood travels from the heart, to the lungs, and back to the heart. This process continually refills the blood with oxygen, and lets carbon dioxide be exhaled. Here's how the process works:

- Oxygen-poor blood returns from the body's tissues through the veins back to the right side of the heart.
- The right heart pumps oxygen-poor blood through the pulmonary arteries into the lungs. This blood becomes filled with oxygen.

- The oxygen-rich blood returns from the lungs back to the left side of the heart. The left heart pumps the oxygen-rich blood into the body through the aorta and many other arteries.

Any part of the heart-lung blood circuit can become damaged or blocked, leading to pulmonary vascular disease.

Causes of Pulmonary Vascular Disease:-

The causes of pulmonary vascular disease vary according to which of the lungs' blood vessels are affected. Pulmonary vascular disease is divided into several categories:

Pulmonary Arterial Hypertension: Increased blood pressure in the pulmonary arteries (carrying blood away from the heart to the lungs). Pulmonary arterial hypertension can be caused by lung disease, autoimmune disease, or heart failure. When there is no apparent cause, it's called idiopathic pulmonary arterial hypertension.

Pulmonary Venous Hypertension: Increased blood pressure in the pulmonary veins (carrying blood away from the lungs, to the heart). Pulmonary venous hypertension is most often caused by congestive heart failure. A damaged mitral valve in the heart (mitral stenosis or mitral regurgitation) may contribute to pulmonary venous hypertension.

Symptoms of Pulmonary Vascular Disease: The symptoms of pulmonary vascular disease vary according to several factors:

- The suddenness of the process affecting the pulmonary blood vessels
- Which pulmonary blood vessels are affected (where the pulmonary vascular disease is)

- How much of the pulmonary vascular system is affected

For example, a sudden, large pulmonary embolism blocking a large pulmonary artery can cause severe shortness of breath and chest pain. But a very small pulmonary embolism (blocking only a small blood vessel) may cause no noticeable symptoms.

Although symptoms of pulmonary vascular disease can vary widely, each of the causes of pulmonary vascular disease has a set of usual symptoms:

Pulmonary arterial hypertension: This most often causes slowly progressive shortness of breath. As the condition worsens, chest pain or fainting (syncope) with exertion can occur.

Pulmonary embolism: A blood clot to the lungs typically occurs suddenly. Shortness of breath, chest pain (often worse with deep breaths), and a rapid heart rate are common symptoms. Pulmonary embolism symptoms range from barely noticeable to severe, based on the size of the blood clot(s).

Pulmonary venous hypertension: This form of pulmonary vascular disease also causes shortness of breath, due to the congestive heart failure that's usually present. Shortness of breath may be worse while lying flat, when blood pressure is uncontrolled, or when extra fluid is present (edema).

Tests for Pulmonary Vascular Disease:-

Based on a person's symptoms, signs, and history, a doctor may begin to suspect the presence of pulmonary vascular disease. The diagnosis of pulmonary vascular disease is usually made using one or more of the following tests:

Computed tomography (CT scan): A CT scanner takes multiple X-rays, and a computer constructs detailed images of the lungs and chest. CT scanning can usually detect a pulmonary embolism in a pulmonary artery. CT scans can also uncover problems affecting the lungs themselves.

Ventilation/perfusion scan (V/Q scan): This nuclear medicine test takes images of how well the lungs fill with air. Those images are compared to pictures of how well blood flows through the pulmonary blood vessels. Unmatched areas may suggest a pulmonary embolism (blood clot) is present.

Pulmonary Embolism: A blood clot breaks off from a deep vein (usually in the leg), travels into the right heart, and is pumped into the lungs. Rarely, the embolism can be a large bubble of air, or ball of fat, rather than a blood clot.

Chronic Thromboembolic Disease: In rare cases, a blood clot to the lungs (pulmonary embolism) is never reabsorbed by the body. Instead, a reaction occurs in which multiple small blood vessels in the lungs also become diseased. The process occurs slowly, and gradually affects a large part of the pulmonary arterial system.

Echocardiography (echocardiogram): An ultrasound video of the beating heart. Congestive heart failure, heart valve disease, and other conditions contributing to pulmonary vascular disease can be discovered with echocardiogram.

Right heart catheterization: A pressure sensor is inserted through a needle into a vein in the neck or groin. A doctor advances the sensor through the veins, into the right heart, then into the pulmonary artery.

Right heart catheterization is the best test to diagnose pulmonary arterial hypertension.

Chest X-ray film: A simple chest X-ray can't diagnose pulmonary vascular disease. However, it may identify contributing lung disease, or show enlarged pulmonary arteries that suggest pulmonary arterial hypertension.

Pulmonary angiography (angiogram): Contrast dye is injected into the blood, and X-ray images of the chest show detailed images of the pulmonary arterial system. Angiography is very good at diagnosing pulmonary embolism but is rarely performed anymore because CT scans are easier, less invasive, and have lower risk (www.wiki.com).

2-1-4 Computed Tomography :-

A CT scan, also called X-ray computed tomography (X-ray CT) and computerized axial tomography scan (CAT scan),makes use of computer-processed combinations of many X-ray images taken from different angles to produce cross-sectional (tomographic) images (virtual "slices") of specific areas of a scanned object, allowing the user to see inside the object without cutting. Digital geometry processing is used to generate a three-dimensional image of the inside of the object from a large series of two-dimensional radiographic images taken around a single axis of rotation .

Medical use :

Since its introduction in the 1970s, CT has become an important tool in medical imaging to supplement X-rays and medical ultrasonography. It has more recently been used for preventive medicine or screening for disease, for example CT colonography for people with a high risk of colon cancer, or full-motion heart scans for people with high risk of heart disease. A number of institutions offer full-body scans for the general population although this practice goes against the advice and official position of many professional organizations in the field.

Major component of the computed Tomography :

There are three major system :

Imaging System
Computer System
Image Display/Recording/Storage System
Each system in a separate room

Major Components

Scanner room :

Imaging system

gantry assembly

Computer and electronic room :

Power

Computer

Generator

Operator’s Area Display / recording / storage

Automatic injector ;

contrast medium is introduced into a patient’s blood vessel via a power injector. Contrast power injectors are typically flow-rate controlled with user-adjustable pressure-limiting capability. The flow rate is dependent on solution viscosity, solution volume, pressure, and the cross-sectional area of the tubing .

Basic components :

Control panel

Syringe

Heating device

Injector power head and high pressure mechanism

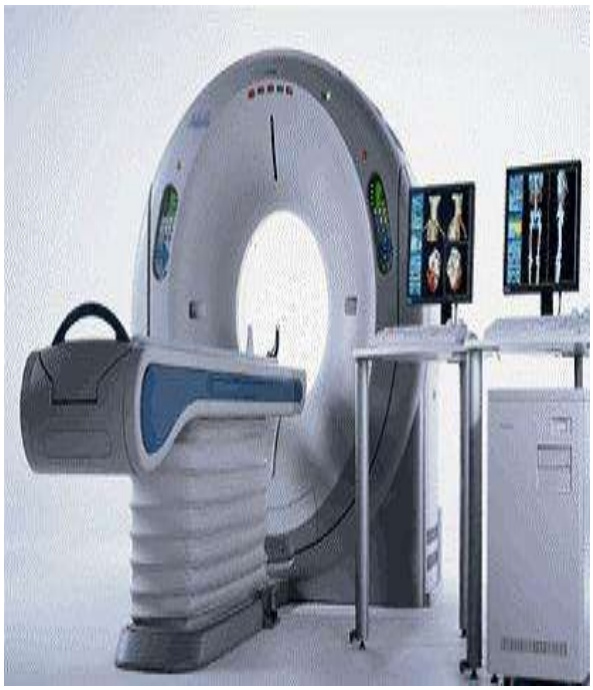

Fig (2-5): computed tomography units

Fig(2-6): automatic injector

2-2 Previous Studies:-

Edward et al 1998 , determine the upper limit of the main pulmonary artery diameter using a modern CT system . this study aimed to measure the normal diameter of the main pulmonary artery in alarge number of subjects without using contrast media , using conventional mdiastinum window ,these values were then compared with those obtained from patients with pulmonary artery hypertension .

Patients group consisted of 100 subjects (61 males , 39 females) , age group from 18 -90years .

The result of this study , mean pulmonary artery diameter in patients with pulmonary artery hypertension was significantly greater (v- value less than 0.01) than in normal patients . Values for pulmonary artery diameter greater than 3.32 cm (mean normal pulmonary artery diameter + 2SD) .

Also this study attempted to define the relationship between main pulmonary artery diameter and age and sex . No significant correlation was found between pulmonary artery diameter and age . However , this study did show that mean PA diameter is greater in men than women (males, 27.7mm; females,26.4 mm) .

Kuriyama et al 2007 , Pulmonary artery diameters measured by multidetector-row computed tomography in healthy adults. Worked to determined To determine the diameters of PAs in subjects with normal PA pressure by using thoracic CT.

Patients group consist of 26 subjects aged between 19 and 46 years, having normal thoracic CTs (5-mm slice thickness) and normal PA pressures ((25 mmHg, determined by echocardiography), were included in the study. The diameters of the main, right, and left PAs were measured by using multidetector CT .

The result of this study ; The main PA diameters of all the subjects showed a normal distribution, and the mean was 24.0 ± 2.8 mm. The main PA diameters in male and female subjects also showed a normal distribution. The difference between the sexes for the main PA diameters was not significant (P=0.08). There were correlations between main PA diameter and body-mass index (BMI) (P=0.001) and weight (P=0.001). However, there was no significant correlation between main PA diameter and height (P=0.6).

Chapter three

Material and Methods

Material and Methods

3-1 Materials:-

3-1-1 Place and Time of study:-

This study was performed at the departments of radiology in Algazira Traumatology Center in Algazira states during the period of October 2015 to August 2016

3-1-2 Population:-

This study included 50 subjects (20 males and 30 females).

3-1-3 Inclusion Criteria:-

Subjects with normal pulmonary artery pressure

3-1-4 Exclusion Criteria:-

Patient with cardiac or pulmonary diseases .

High pulmonary artery pressure which was determined by echocardiography .

3-1-5 Study Variables:-

The variables that were collected from each subject included gender, patient age and body mass index (BMI) .

3-1-6 Equipment:-

CT Toshiba 16 slices (Toshiba medical systems , Nasu , Japan 2003) were used .

3-2 Methods:-

3-2-1 CT Chest with contrast Technique:-

A CT or CAT scan is a shortened name for computerized tomography. A CT scan takes pictures of the inside of the body. The pictures are more detailed than a typical x-ray. During a CT scan of the chest, pictures are taken of cross sections or slices of the thoracic structures in your body. The thoracic structures include your lungs, heart and the bones around these areas. When contrast is used during a CT scan of the chest thoracic structures are highlighted even more.

CT scans can help determine a diagnosis early. Your doctor will use this information to determine the best treatment for you.

Preparing for the Test

- For children under 18 years - Do not eat 4 hours before the test is scheduled. You/your child may drink clear fluids only. It is important that you drink enough clear liquids (like water) to be well hydrated prior to the test.
- For adults –There is no food restriction for this scan. It is important that you drink enough clear liquids (like water) to be well hydrated prior to the test.
- Wear clothing you can remove from the waist up. You will be given a gown to wear.
- Avoid having any barium studies done 2 to 3 days before the CT scan.
- Talk with your doctor before the test if you have a history of reactions to contrast in the past.

- If you take metformin or metformin-containing drugs for diabetes (Glucophage, Glucovance, Janumet, etc.), discuss this with your doctor prior to your CT appointment. There are specific requirements for you following your scan.
- If you have not had a recent blood test for creatinine, a finger-poke blood test will be done just prior to your scan.
- Please arrive 20 minutes before the test is scheduled. The radiology technologist will ask you questions and have you complete a questionnaire.

Patient position : Supine and arm elevated above the head .

Contiguous 10mm slices were obtained using Toshiba 16 slices machine and 15 breath hold spiral technique (pitch 1.25- 1.5 , 0.75 s rotation time , 120 kv , 10mm reconstruction interval) .

Using 18-gauge syringe, 80-100 cc nonionic contrast medium was administered manually into the antecubital vein. The contrast media was introduced into the patient by the power injector(flow rate 3-4ml\sec) .
Toshiba sure start may be used for optimized contrast media monitoring in CTA .When the contrast reached a set value (Threshold) , the monitoring scan end and the main scan (helical scan) start to provide images when the contrast flow in the vessel is optimum .

3-2-2 Method of measuring MPA diameter by using CT Scan :-

The region of the ascending aorta and pulmonary artery was magnified to full screen size . The widest diameter perpendicular to the long axis of the main pulmonary artery was measure with computer caliper at the level of the pulmonary artery bifurcation fig(3-1) .

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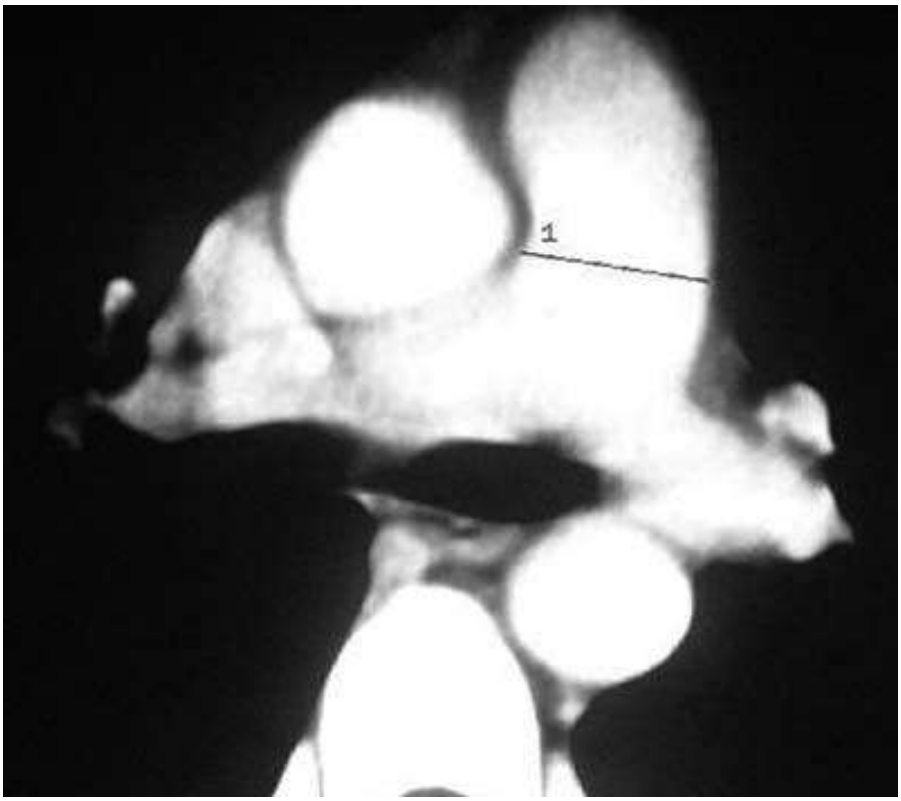

Fig(3-1). Axial CT image of main pulmonary artery and measurement point.

3-2-3 Data collection:-

Data collection sheet used and other information were collected directly from patients in addition to references, websites and previous studies.

3-2-4 Data analysis:-

The results were scheduled for analysis by using statistical package for social studies (SPSS) and Excel to obtain the results related to correlation between variables.

Chapter four

Results

Results

[A] Frequency distribution

Table (4-1): Distribution of patients according to gender

Gender	Frequency	%
Females	30	60
Males	20	40
Total	**50**	**100%**

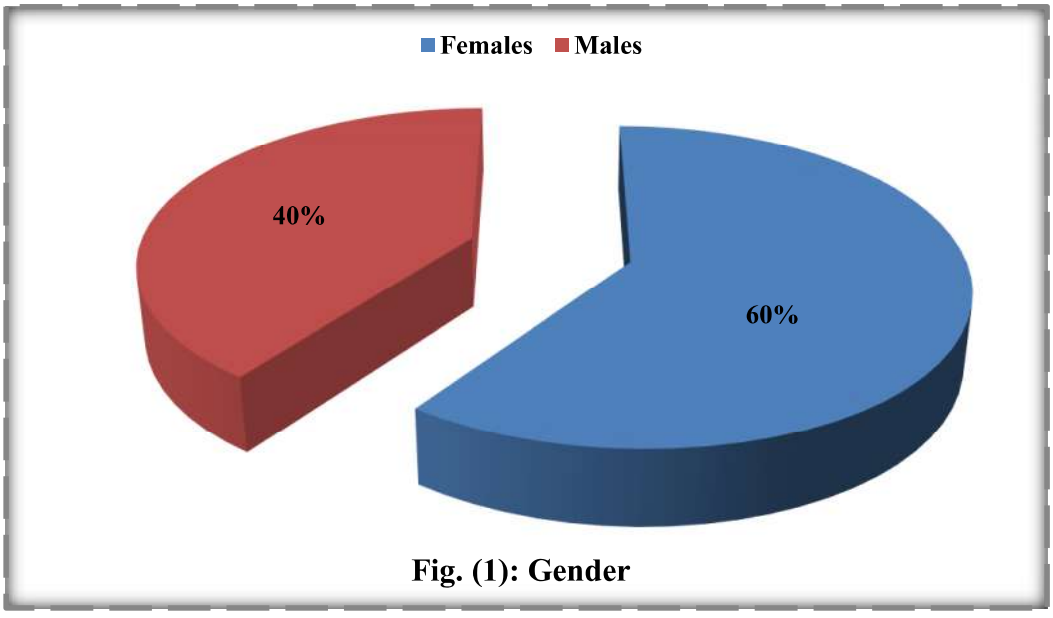

Fig. (1): Gender

As shown in Table (4-1) and Fig. (4-1), females represent (60%) of study population and (40%) are males.

Table (4-2): Distribution of patients according to age

Age group (years)	Frequency	%
<20	3	6
20-39	12	24
40-59	17	34
≥60	18	36
Total	**50**	**100%**
Mean±SD	**48.34±17.02**	
Range	**18-80**	

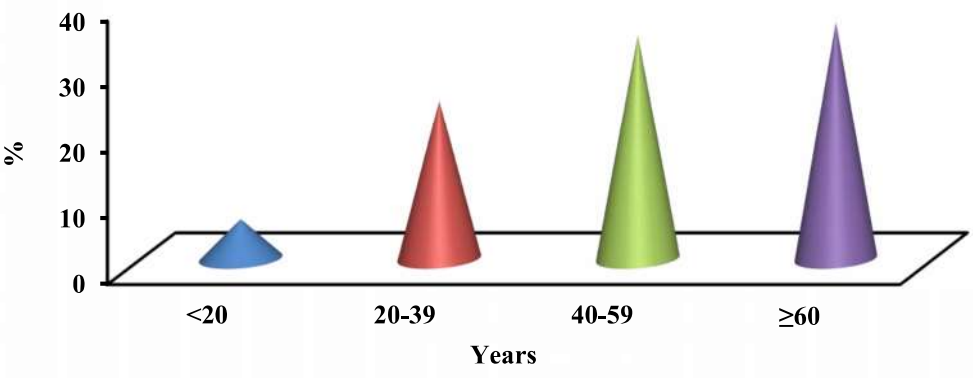

Fig. (4-2): Age

Age group of patients understudy was illustrated in Table (4-2) and Fig. (4-2). 3 out of 50 (6%) their ages less than 20 years, followed by 12 out of 50 (24%) between 20-39 years, 17 out of 50 (34%) between 40-59 years and the rest 18 out of 50 (36%) are 60 years of age or above.

Table (4-3): Distribution of patients according to body mass index

BMI	Frequency	%
<18.5	4	8
18.5-24.9	42	84
25.0-29.9	3	6
≥30	1	2
Total	**50**	**100%**
Mean±SD	**22.62±2.43**	
Range	**18-30**	

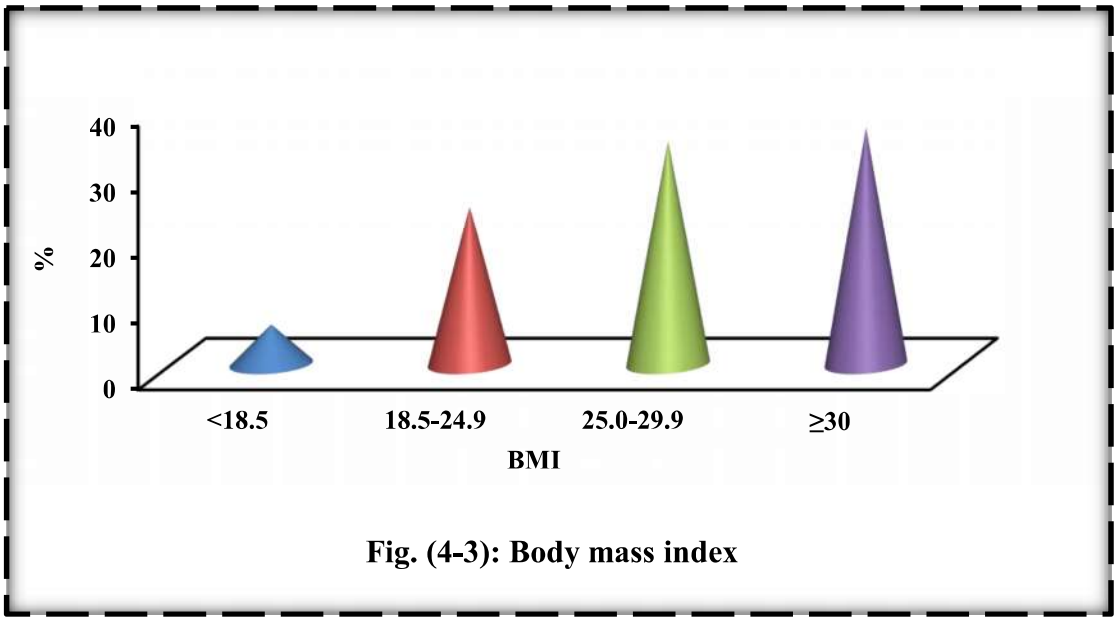

Fig. (4-3): Body mass index

Distribution of patients according to body mass index was illustrated in Table (4-3) and Fig. (4-3). 4 out of 50 (8%) are under weight (<18.5), followed by 42 out of 50 (84%) are normal (18.5-24.9), 3 out of 50 (6%) are overweight (25.0-29.9) and 1 out of 50 (2%) are obese.

Table (4-4): Distribution of patients according to main pulmonary artery diameter

Diameter (mm)	Frequency	%
<20	1	2
20-24	18	36
25-29	21	42
≥30	10	20
Total	**50**	**100%**
Mean±SD	**26.30±4.29**	
Range	**18-32**	

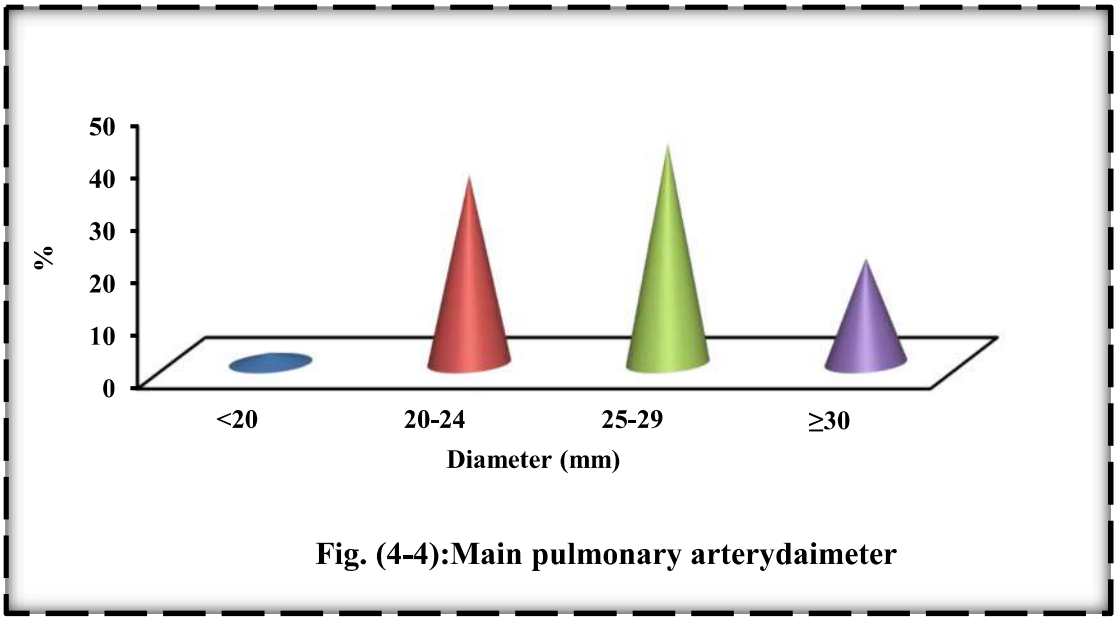

Fig. (4-4):Main pulmonary arterydaimeter

Main pulmonary artery diameter of investigated patients was demonstrated in Table (4-4) and Fig. (4-4). 1 out of 50 (2%) are below 20 mm, 18 out of 50 (36%) between 20-24 mm, 21 out of 50 (42%) , while 10 out of 50 (20%) their pulmonary artery diameter was 30 mm or above.

[B] Relationships

Table (4-5): Cross-tabulation of main pulmonary artery diameter and gender

Diameter (mm)	Females		Males	
	Frequency	%	Frequency	%
<20	1	2	-	-
20-24	15	30	3	6
25-29	10	20	11	22
≥30	4	8	6	12
P-value	**0.0014****			

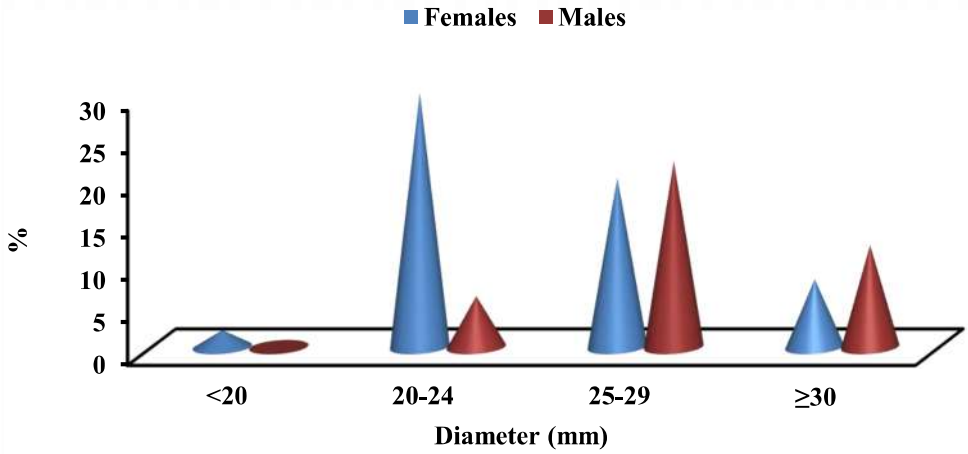

Fig. (4-5):Main pulmonary artery diameter vs gender

Cross-tabulation of main pulmonary artery diameter and gender of patients understudy was presented in Table (4-5) and Fig (4-5). 2, 30, 20 and 8% of female patients their pulmonary diameter are <20, 20-24, 25-29 and ≥30 mm, respectively; while 6, 22 and 22% of male patients, their volumes ranged between 20-24, 25-29 and ≥30 mm, respectively.

Main pulmonary artery diameter of patients compared with gender shows highly significant difference (P=0.0014).

Table (4-6): Relationship between main pulmonary artery diameter and age

Diameter (mm)	Age group (years)							
	<20		20-39		40-59		≥60	
	No.	%	No.	%	No.	%	No.	%
<20	1	2	-	-	-	-	-	-
20-24	1	2	5	10	8	16	4	8
25-29	1	2	5	10	6	12	9	18
≥30	-	-	2	4	3	6	5	10
P-value	0.0**							

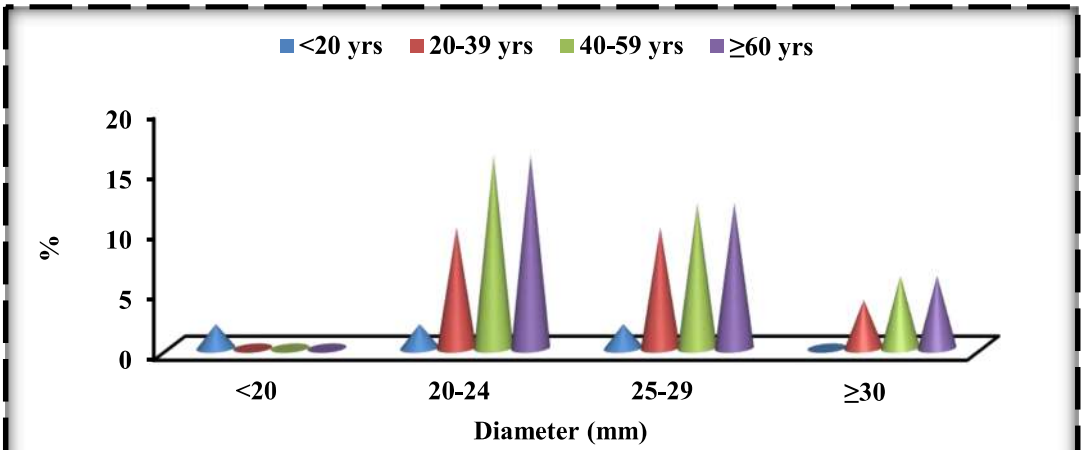

Fig. (4-6):Main pulmonary artery daimeter and age

Relationship between main pulmonary artery diameter and age was illustrated in Table (4-6) and Fig. (4-6). (2%) of patients aged less than 20 years, their diameters are <20, 20-24, (25-29 and 30 mm or above. For patients aged between 20-39 year, (10%), (10%) and (4%) their diameters are 20-24, 25-29 and ≥30 mm, respectively. (16%), (12%) and (6%) of patients aged 40-59 years, their diameters are 20-24, 25-29 and ≥30 mm; while (8%), (18%) and (10%) of patients of 60 years age or above, their diameters are 20-24, 25-29 and ≥30 mm.

Chi-square test show very highly significant difference (P=0.00) between main pulmonary artery diameter of patients under investigation and their ages.

Table (4-7): Relationship between main pulmonary artery diameter and body mass index

Diameter (mm)	Body mass index							
	<18.5		18.5-24.9		25.0-29.9		≥30	
	No.	%	No.	%	No.	%	No.	%
<20	-	-	1	2	-	-	-	-
20-24	2	4	16	32	-	-	-	-
25-29	2	4	19	38	-	-	-	-
≥30	-	-	6	12	3	6	1	2
P-value	0.0002							

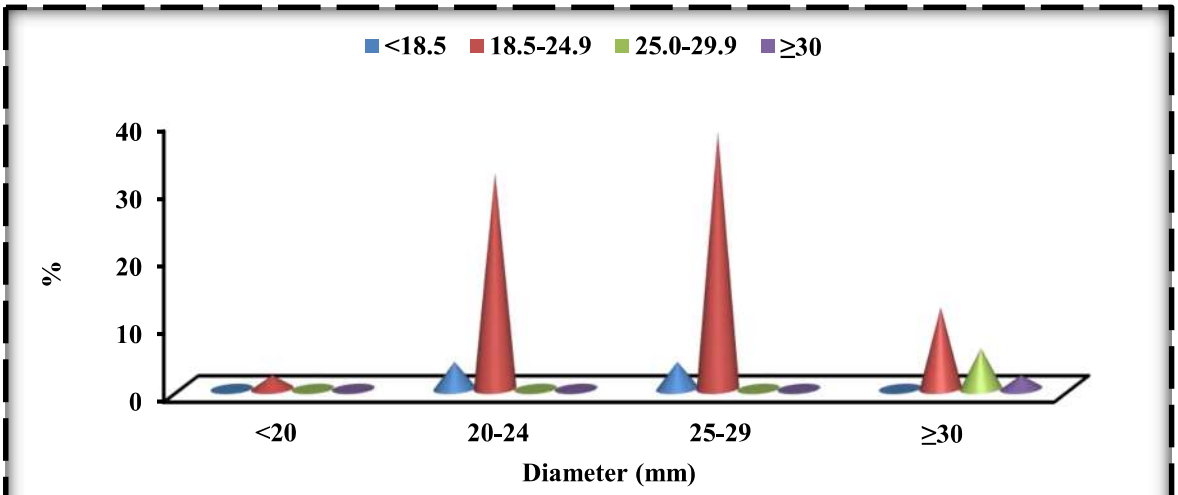

Fig. (4-7):Mian pulmonary artery diameter vs BMI

As shown in Table (4-7) and Fig. (4-7), (4%) of underweight patients their pulmonary artery diameters are and 25-29 mm, respectively, (2%), (32%), (38%) and (12%) of normal patients their diameters are <20, 20-24, 25-29 and ≥30 mm, respectively. (6%) and (2%), respectively of overweight patients their diameter is ≥30 mm.

Pulmonary artery volume compared with body mass index showed highly significant difference (P=0.0002).

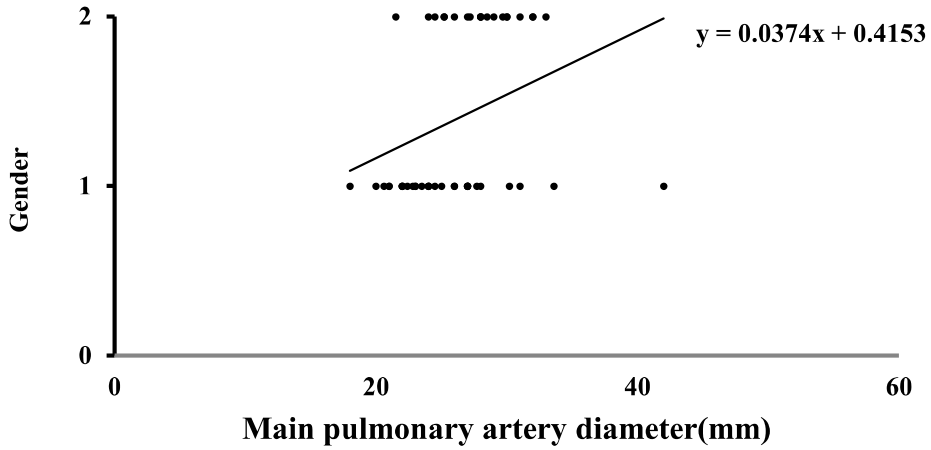

Fig. (4-8): pulmonary artery diameter versus gender

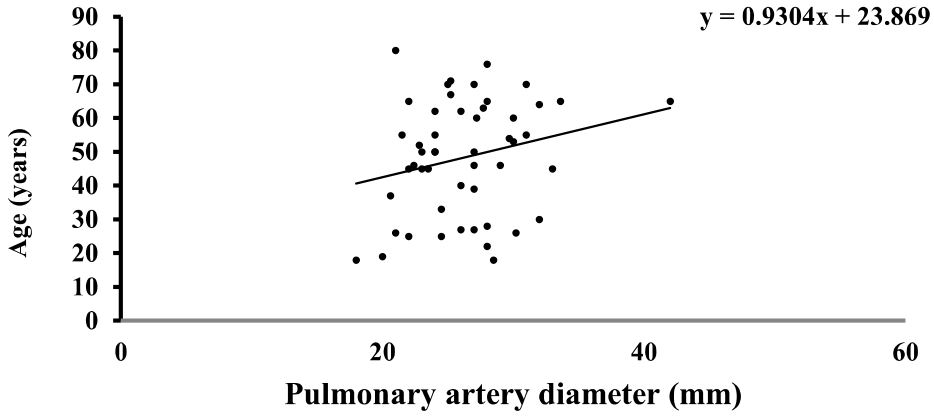

Fig. (4-9): Pulmonary artery diameter versus age

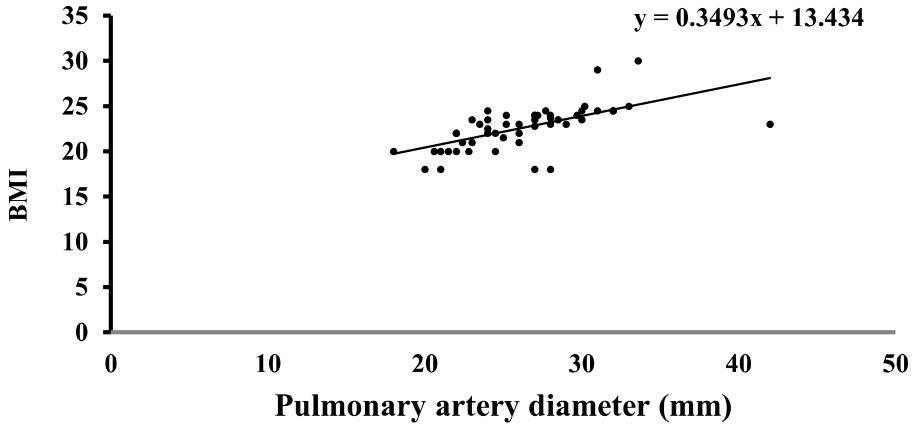

Fig. (4-10): Pulmonary artery diameter versus BMI

Chapter five

Discussion, conclusion and recommendations

Discussion, conclusion and recommendations

5-1 Discussion:-

These results aimed to measure the main pulmonary artery diameter using computed tomography. An underwent CT chest with contrast during the period from October 2015 to August 2016 , number of 50 subjects (60% were females and 40% were males) .

.On the most of the largest series that evaluated the MPAD in normal

CT was the study of Edwards et al., which included 100 normal individuals and 12 patients with PH. This study was performed without using an intravenous contrast medium and the mean MPAD was 27.2 ± 0.6 mm . Kuriyamaet al reported the mean MPAD as 24.2 ± 2.2 mm .

In the present study, mean MPAD was 26.30±4.29 mm (18-32mm) , in agreement with the results of the previous studies.

When the MPAD was ≥33.2 mm, Edwards et al. obtained 58% sensitivity and 95% specificity for the detection of PH with CT . According to Kuriyama et al., an MPAD ≥ 28.6 mm measures pulmonary hypertension. In the present study, our patients were without pulmonary pathology and we found the upper limit of the MPAD to be 32 mm.

Some investigators found a correlation between age and pulmonary artery diameter, but others did not. In this study, we found a statistically significant relationship between pulmonary artery diameter and age (P=0.00) .

According to Edward et al., the MPAD in males was larger than in females; that finding was attributed to the tendency of male subjects to have larger overall dimensions than female patients . In the study of Kuriyama et al., no significant difference was found between the pulmonary artery diameters of males and females . In the present study,

the MPAD in males was found to be larger than in females (mean MPAD was 27.99mm in males , 25.11 in females).

In this study also we found a positive linear correlation between MPAD and BMI ,that mean showed highly significant difference (P=0.0002) .

5-2 Conclusion:-

The present study demonstrated that in individuals with normal pulmonary artery pressure, the upper limit of the MPAD is 32 mm and the mean of the main pulmonary artery diameter was 26.6 ± 4.9 mm, in agreement with the results of the previous studies .

From this study it could be concluded there was direct relationship between the min pulmonary artery diameter and age , gender and body mass index . In other word , this study found statically significant relationship between the main pulmonary artery diameter and age . Also found the mean of the MPAD is larger in males than females (mean MPAD is 27.99mm in males and 25.11mm in females) this difference is statically significant , a difference is 2.88mm .

This study also was influenced by the body mass index ,that showed highly significant difference between them (P=0.0002) , also we observed the over weight patient had MPAD ≥30mm .

5-3 Recommendations:-

Measurement of the main pulmonary artery diameter may play an important role in aiding diagnosis of the pulmonary hypertension .

Using of Computed Tomography in the measurement of the main pulmonary artery diameter is valuable and non invasive procedure .

BMI is generally considered the best way to determine if an individual is at a healthy weight. Using BMI is popular because it is simple, quick, effective and applies to adult men and women .

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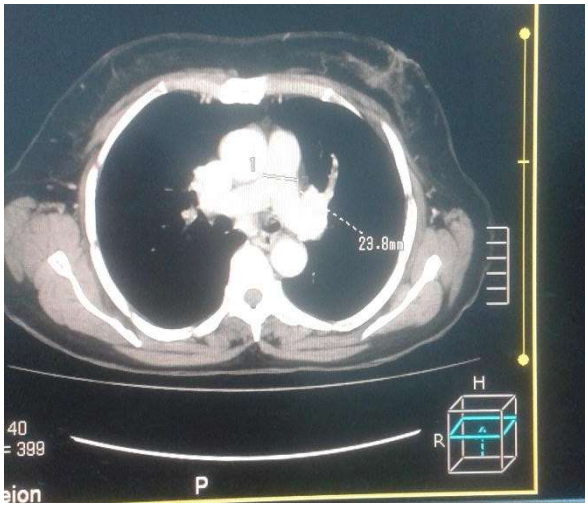
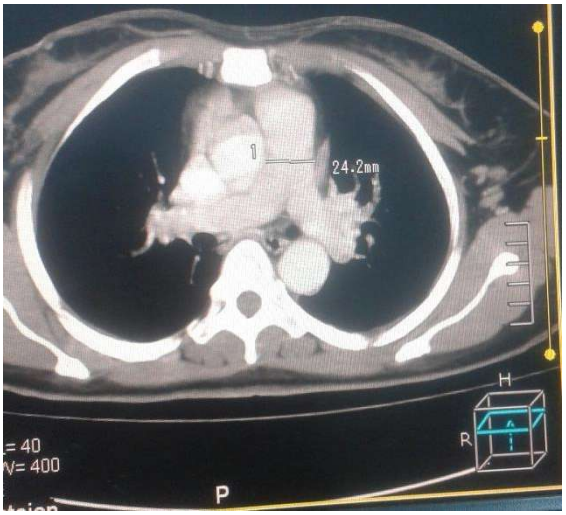
Appendices

Data collection sheet

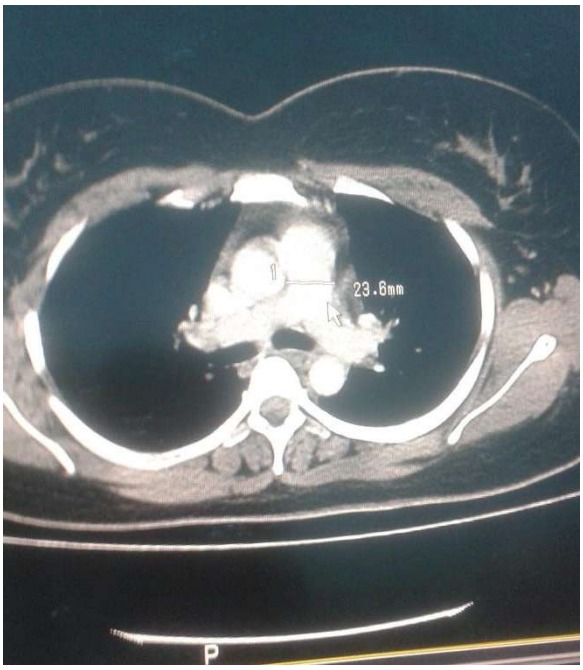
No	Age	Gender	BMI	MPAD



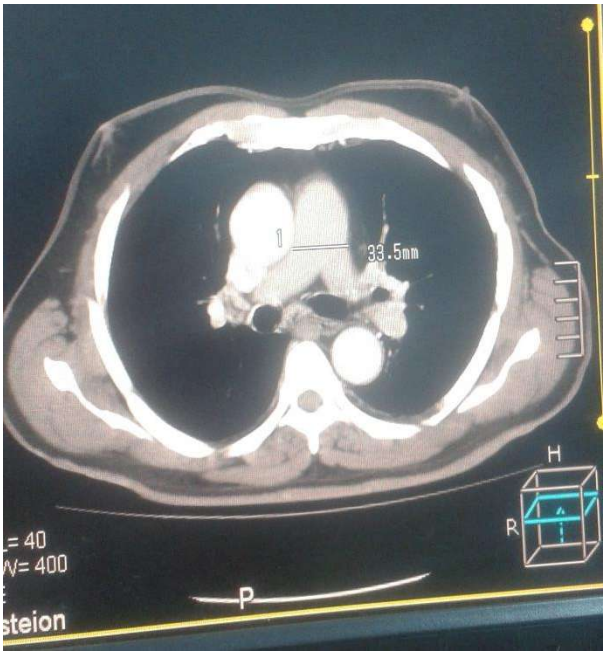
18years female BMI is 23.8



23years female BMI is 24.2



24years male BMI is 23.6



38years male BMI is 33.5