

Introduction

-.Introduction 1-1

The heart is a hollow muscular organ that together with the root of the great vessels, is enclosed in a fibroserous sac, the pericardium. It is situated mainly left of the midline in the lower anterior part of the chest and attached to the central tendon of the diaphragm

..(Whithely, et al 2005

Blood is returned to the heart from body through the superior and inferior venae cavae which open into a thin-walled chamber called the right atrium, the right atrium pumps the blood through a valve called tricuspid valve into a thicker-walled chamber called right ventricle .The right ventricle in its turn pumps the blood through the pulmonary artery into the lungs .In the capillaries of the lungs the blood take up oxygen and releases carbon dioxide , the freshly oxygenated blood then passes through pulmonary veins into a thin-walled chamber called the left atrium, the left atrium pumps the blood through a valve called mitral valve into the a thickest-walled of the heart, the left ventricle. The left ventricle in turn pumps the blood into the aorta from whence it is distributed to

(the body. (Dean, west 1987

Hypertension is a major risk factor for cardiovascular morbidity and mortality. The presence of hypertension more than doubles the risk for coronary heart disease, including acute myocardial infarction and sudden death, and more than triples the risk of congestive heart failure as well as strokes. Patients with high blood pressure frequently have abnormalities of cardiac structure or function, including left ventricular hypertrophy, systolic and diastolic dysfunction and in extreme cases, overt heart failure. There may also be concomitant or related coronary heart disease and an increased risk of .(arrhythmias and sudden death (Kannel and Cobb. 1992

Cardio thoracic ratio (CRT) is the ratio use to evaluate heart size and chamber enlargement on standard chest projection, the ratio of the cardiac diameter of the chest should be no greater than 50% of a full inspiratory film. ((Danzar,1919

The heart can be expressed as the cardio thoracic ratio normal ratio is less than 0.50 .The ratio in adult is .influenced mainly by left ventricle enlargement

The CRT is the fraction derived by measuring the distance from the midline to the most lateral aspect of the left and right heart border and dividing that sum by the maximum horizontal measurement of the thorax, from left

pleural surface to right pleural surface on a postero-anterior chest radiograph.(Fraser 1988

-:problem of the study 1-2

Many misdiagnosis occurs when evaluating heart size on plain chest x-ray, and hypertension disease is nowadays widely spreading in Sudan, to the best of the researcher's knowledge. So, this study tries to assess the heart size in hypertensive Sudanese patients in order to address this problem.

-:Objectives 1-3

:General objective 1-3-1

To measure the cardio thoracic ratio in hypertensive patients using chest x-ray

:Specific objectives 1-3-2

To detect any relation between heart size and
.hypertension

To study the effect of age and gender on hypertensive
.patients

.To find out the relation between heart size and age

:Significance of the study 1-4

It is hopeful that all hospitals in Sudan can benefit from the results of this study to improve the chest imaging protocols and hence to acquire a chest x-ray of good
.quality

-.The Overview of the research 1-5

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

Literature Review

-.Theoretical background 2-1

-.Anatomy 2-1-1

The heart, slightly larger than a clenched fist, is a double, self-adjusting, suction and pressure pump is part of which work in union to propel blood to all parts of the body. Right side of the heart receives poorly oxygenated (venous) blood from the body through the SVC and IVC and pumps it through the pulmonary trunk to the lungs for
.oxygenation (Keith and Arthur 2006

The heart is located in the thoracic cavity between the lungs. This area is called the mediastinum. The base of the cone-shaped heart is uppermost, behind the sternum, and the great vessels enter or leave here. The apex (tip) of the heart points downward and is just above the diaphragm to the left of the midline. This is why we may think of the heart as being on the left side, because the strongest beat
.can be heard or felt here

The heart is enclosed in the pericardial membranes, of which there are three layers. The outermost is the fibrous pericardium, a loose fitting sac of strong fibrous connective tissue that extends inferiorly over the diaphragm and superiorly over the bases of the large vessels that enter and leave the heart. The serous pericardium is a folded membrane; the fold gives it two layers, parietal and visceral. Lining the fibrous pericardium is the parietal pericardium. On the surface of the heart muscle is the visceral pericardium, often called the epicardium. Between the parietal and visceral pericardial membranes is serous fluid, which prevents friction as the

(heart beats (Valerie and Tina 2007

:-The surface of the heart 2-1-1-1

The four surface of the heart are the anterior (sternocostal) surface, formed mainly by the right ventricle, diaphragmatic (inferior) surface, formed mainly by the left ventricle and partly by the right ventricle, it is related mainly to the central tendon of the diaphragm, right pulmonary surface, formed mainly by the right atrium, left pulmonary surface, formed mainly by the left

.(ventricle (Keith and Arthur 2006

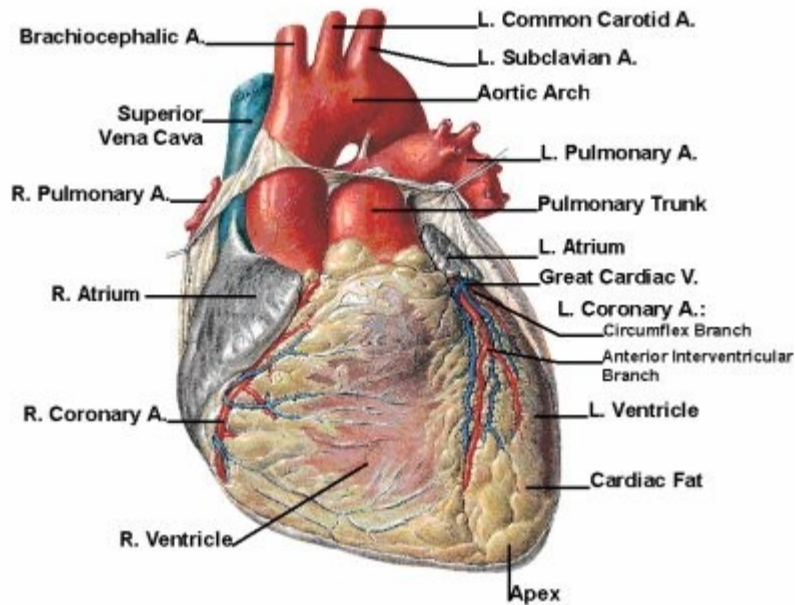


Fig (2-1) The heart and blood vessels

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

The walls of the four chambers of the heart are made of cardiac muscle called the myocardium. The chambers are 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

-.Left Atrium 2-1-1-2-2

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

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

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 chordae 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

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

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

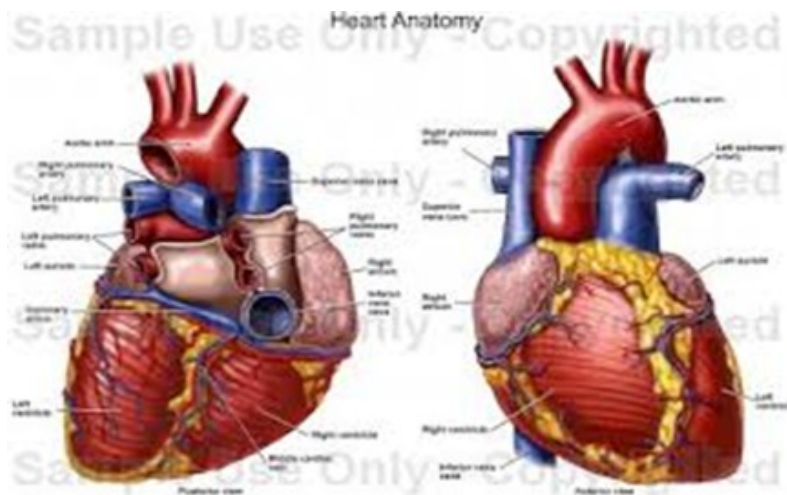


Fig (2-2) The heart Anatomy

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

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

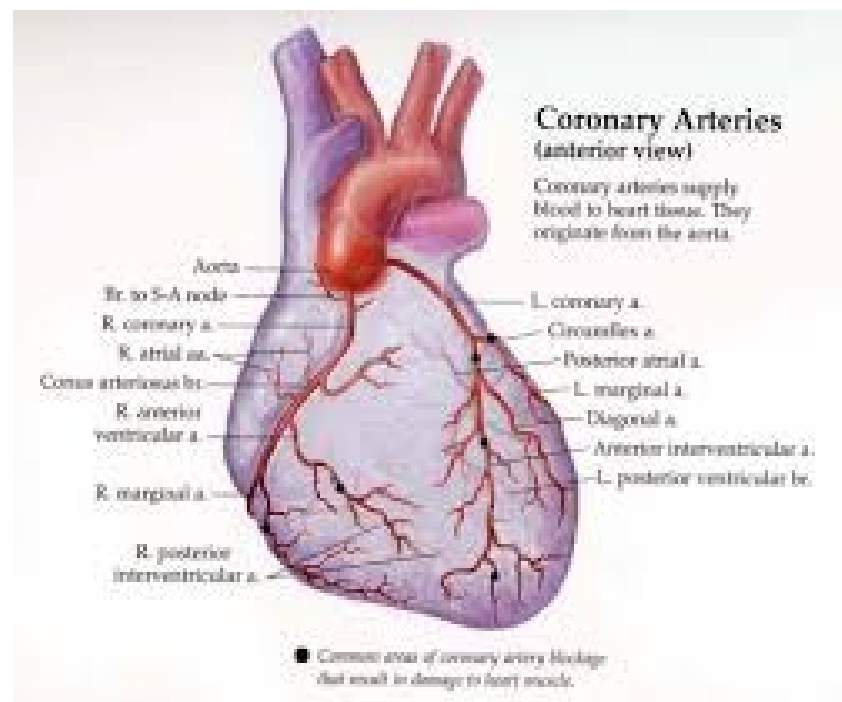


Fig (2-3) The blood supply of the heart

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

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

-.:Physiology 2-1-2

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

-.:Cardiac Cycle 2-1-2-1

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 (Provopphys et al 2006

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

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

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

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 etal 2006

-.Heart Sounds 2-1-2-2

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

Cardiac Cycle- Heart Sounds

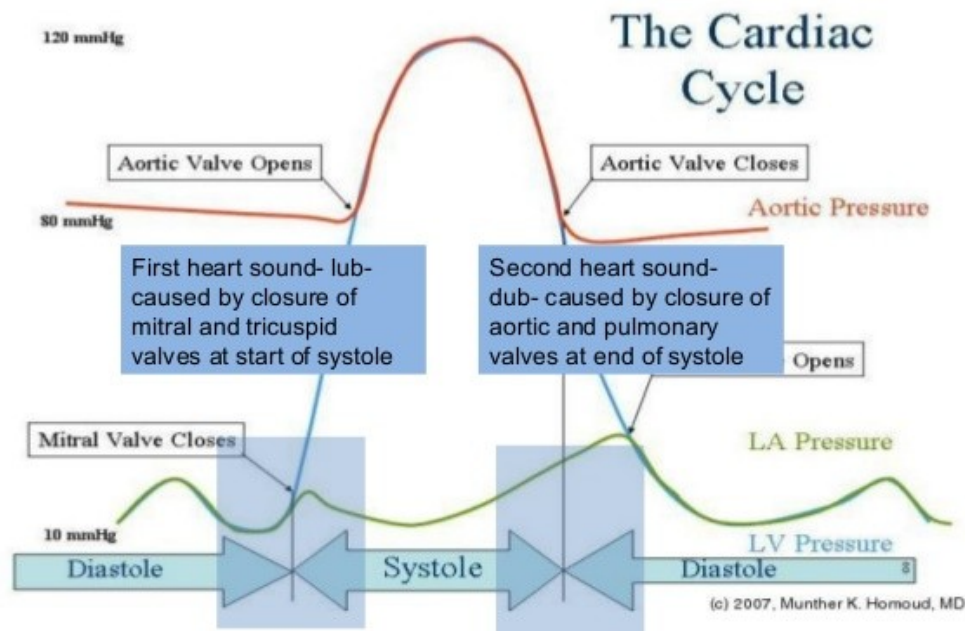


Fig (2-4): cardiac cycle and heart sound

Function of the Atria 2-1-2-3

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

-.Function of the Ventricles 2-1-2-4

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

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

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

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 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

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

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

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

-.: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).

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

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

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

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

-.:Blood Pressure 2-1-2-9

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

-.Systolic Pressure 2-1-2-9-1

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

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

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

-:Pathology of the heart 2-1-3

-:Heart Failure 2-1-3-1

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 •

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

.activation of the renin-angiotensinaldosterone system (2)

.(release of atrial natriureticpeptide (ANP (3)

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 ended 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. 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 augmented • muscle 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 or worsening heart function, pathologic changes may eventually supervene, resulting in structural and functional disturbances; such degenerative changes include myocyte apoptosis, cytoskeletal 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 (cor pulmonale) 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 et al 2007).

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

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

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

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 (cor pulmonale). 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 underlie 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 cerebrovascular 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 the 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 than 90mmHg, or a sustained systolic pressure in excess of 140mmHg, 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

-.:Hypertension 2-1-3-3

Hypertension or high blood pressure is a medical condition wherein the blood pressure is chronically elevated. Persistent hypertension is one of the risk factors for strokes, heart attacks, heart failure and arterial aneurysm, and is a leading cause of chronic renal failure (Provophys .(et al 2006

-:Regulation of Blood Pressure 2-1-3-4

Blood pressure is a complex trait involving the interaction of multiple genetic and environmental factors that influence two hemodynamic variables: cardiac output and peripheral vascular resistance. Cardiac output is affected by blood volume, itself strongly dependent on sodium concentrations. Peripheral resistance is regulated predominantly at the level of the arterioles and influenced by neural and hormonal inputs. Normal vascular tone reflects an interplay between circulating factors that induce vasoconstriction (e.g. angiotensin II and catecholamines) and vasodilation (e.g., kinins, prostaglandins, and nitric oxide). Resistance vessels also exhibit autoregulation, whereby increased blood flow induces vasoconstriction to protect tissues against hyperperfusion. Other local factors such as pH and hypoxia, as well as neural interactions are also involved. The integrated function of these systems ensures adequate systemic perfusion

Despite regional demand differences. The kidneys (primarily) and adrenals (secondarily) are central players in blood pressure regulation; they interact with each other to modify vessel tone and blood volume

The kidney influences peripheral resistance and sodium homeostasis primarily through the renin-angiotensin system. Renin is a proteolytic enzyme produced in the

kidney by the juxtaglomerular cells modified myoepithelial cells that surround the glomerular afferent arterioles. When blood volume or pressure is reduced, the kidney senses this as a decreased pressure in the afferent arterioles. Moreover, lower volumes or pressures result in a reduced glomerular filtration rate in the kidney with increased reabsorption of sodium by proximal tubules; these latter two effects putatively conserve sodium and expand the blood volume.

The juxtaglomerular cells respond to reduced intraluminal pressures in the afferent arterioles by releasing renin; they also produce renin when the cells of the macula densa sense decreased sodium concentration

in the distal convoluted tubule.

Renin catabolizes plasma angiotensinogen to angiotensin I, which in turn is converted to angiotensin II by angiotensin-converting enzyme in the periphery. Angiotensin II raises blood pressure by: increasing peripheral resistance by inducing vascular SMC contraction; increasing blood volume by stimulating aldosterone secretion in the adrenals; increasing distal tubular reabsorption of sodium.

The kidneys filter 170 liters of plasma containing 23 moles of salt daily! Moreover, 99.5% of the filtered salt must be reabsorbed to maintain homeostasis (assuming daily ingestion of only 100 mEq). As it turns out, the absorption of the last 2% of sodium is the key to normal sodium homeostasis; this is regulated by the renin-

angiotensin system acting on the epithelial Na^+ channel
(ENaC)

The kidney also produces a variety of vasorelaxant or •
anti hypertensive substances (including prostaglandins
and nitric oxide) that presumably counterbalance the
.vasopressor effects of angiotensin

When renal excretory function is impaired, increased •
arterial pressure is a compensatory mechanism that can
.help restore fluid and electrolyte balance

Other tissues can also influence blood pressure and •
volume. Thus, atrial natriuretic peptide, secreted by heart
atria in response to volume expansion (e.g., in heart
failure) inhibits sodium reabsorption in distal tubules and
(causes global vasodilation (Kumar 2007

:-Computed radiography 2-1-4

Use very similar equipment to conventional radiography
except that in place of a film to create the image, an
imaging plate (IP) made of photo stimulable phosphor is

used. The imaging plate housed in a special cassette and placed under the body part or object to be examined and the x-ray exposure is made. Hence, instead of taking an exposed film into a darkroom for developing in chemical tanks or automatic film processor, the imaging plate is run through a special laser scanner or CR reader, that reads and digitizes the image can then be viewed and enhanced using software that has functions very similar to other conventional digital image processing software, such as .(contrast, brightness, filtration and zoom (Wikipedia.org



Fig (2-5): computed radiography units

-.:Previous Studies 2-2

Mohamed Yousef et al (2014), Aortic and Heart Dimensions in Adult Sudanese Population Using Chest x-ray (Taibah University K.S.A). The main finding of this study showed that the cardio thoracic ratio decreased with age, the mean values for males and females were 44 and 42 respectively. This research also set upper and lower limits for normal aortic arch diameters within the Sudanese populations. The mean value of aortic arch diameters was 5.3 and there was no significant correlation between age and CTR, as $p\text{-value}=0.379$ and the correlation value is weak ($r=0.127$) there was no significant correlation between BMI (body mass index) and CTR as $p\text{-value}=0.424$ and the correlation value was weak $((r=0.116$

Rayan Altayeb, (2015), Assessment of cardio thoracic ratio for hypertensive patients using computed tomography. The result of this research showed a significant relation between CTR hypertensive and high blood pressure $p\text{-value}=0.05$. The result also showed that there was a significant relationship between the cardio thoracic ratio value with age, weight and medication duration of hypertensive patients

Sinha.U et al, (2013), Comparative Study of Cardiac Size Using Chest X-ray and Echocardiography (Journal of anatomical society of India). The result of the study showed that chest x-ray is useful for the diagnosis of cardiomegaly due to various types of cardiac disease. On chest x-ray, the CTR and transverse diameter showed a strong positive correlation with total ventricular dimensions on echocardiography. Other parameters such as transverse diameter and transverse left diameter on chest x-ray also showed a positive correlation with right ventricular dimensions on echocardiography. Therefore, chest x-ray is reliable for the diagnosis of cardiomegaly in .the absence of echocardiography according to that study

Material and Method

-:Materials 3-1

-:Population 3-1-1

This study included 50 subjects (9 males and 41 females), Patients who were clinically diagnosed as having hypertension disease and patients who had done chest x-ray in PA projections were included. Normal patients and patients who had done chest x-ray in AP projections were .excluded

-:Study Variables 3-1-2

The variables that were collected from each subject included gender, patient age, hypertension age and cardio .(thoracic ratio (CTR

-:Equipment 3-1-3

X-ray machine SHIMADZU (Mobile Art eco) made in Japan,
.Max KV=125, Max MAS=100

X-ray machine SHIMADZU (FLEXAVISION) made in Japan,
.Max KV=150, Max MAS=80

-.Methods 3-2

-.Technique 3-2-1

The patient was positioned facing the cassette, with the chin extended and centered to the middle of the top of the cassette the feet were paced slightly apart so that the patient was able to remain steady. The median sagittal plane was adjusted at right angles to the middle of the cassette, the shoulders were rotated forward and pressed downward in contact with the cassette. This was achieved by placing the dorsal aspect of the hands behind and below the hips with the elbows brought forward or by allowing the arms to encircle the cassette

The horizontal central beam was directed at right angles to the cassette at the level of the eighth thoracic vertebra, which was coincident with the lung midpoint. Exposure was made in full normal arrested inspiration, the central beam was centered automatically to the middle of the .film

Method of measuring CTR from PA 3-2-2

-.Chest

In the PA chest x-ray the measurement was made from the middle of the spine horizontally to the most lateral aspect of the cardiac apex. This procedure was repeated in a similar fashion from the midline of the spine to the most lateral aspect of the right atrium. These two measurements were added and divided by the largest horizontal width of the chest, from left to right pleural .surface, derived at the level of the left hemi diaphragm

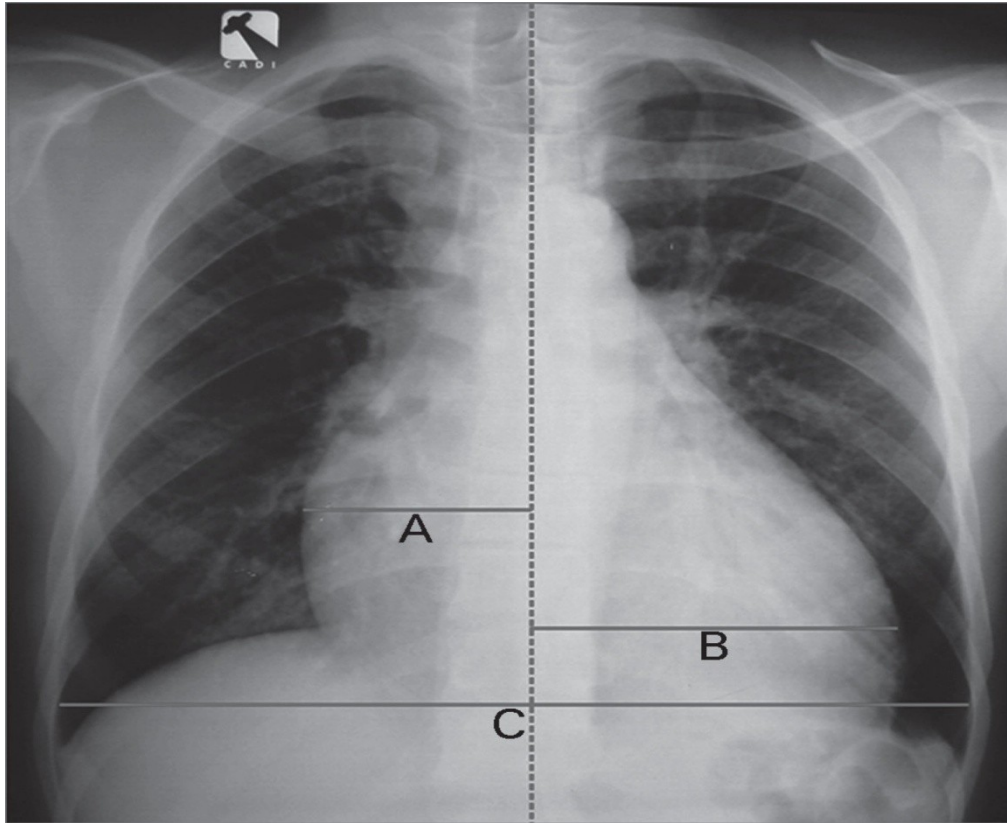


Fig (3-1): measurement of CTR from chest x-ray

-.Data collection 3-2-3

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

-.Data analysis 3-2-4

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

Results

The following tables and figures present the data obtained from 50 hypertensive patients who were examined for plain Chest x-ray, at Radiology Departments of Alshab Teaching Hospital and Alfaisal .Specialized Hospital

Table (4-1): Shows the population Percentage of male and female

Gender	Frequency	Percent
Male	9	18 %
Female	41	82 %
Total	50	100%

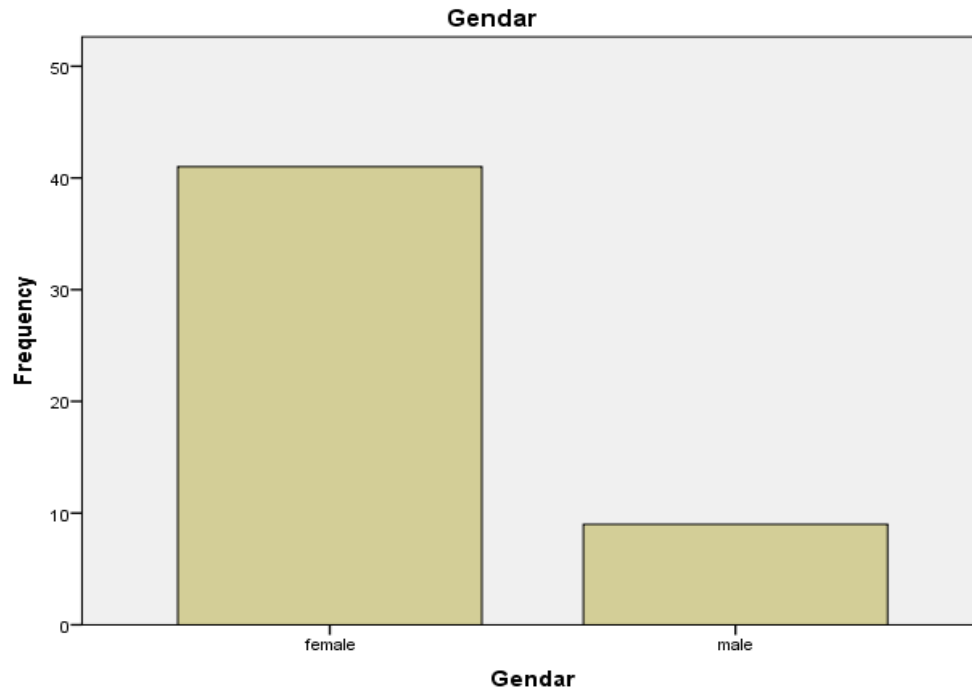


Fig (4-1): Bar graph showing the distribution of sample to gender

Table (4-2): Shows the distribution of sample according to the age categories

Age group	Frequency	Percent
19-29	1	2%
30-39	2	4%
40-49	8	16%
50-59	7	14%
60-69	12	24%
70-79	14	28%

80-89	5	10%
90-100	1	2%
Total	50	100%

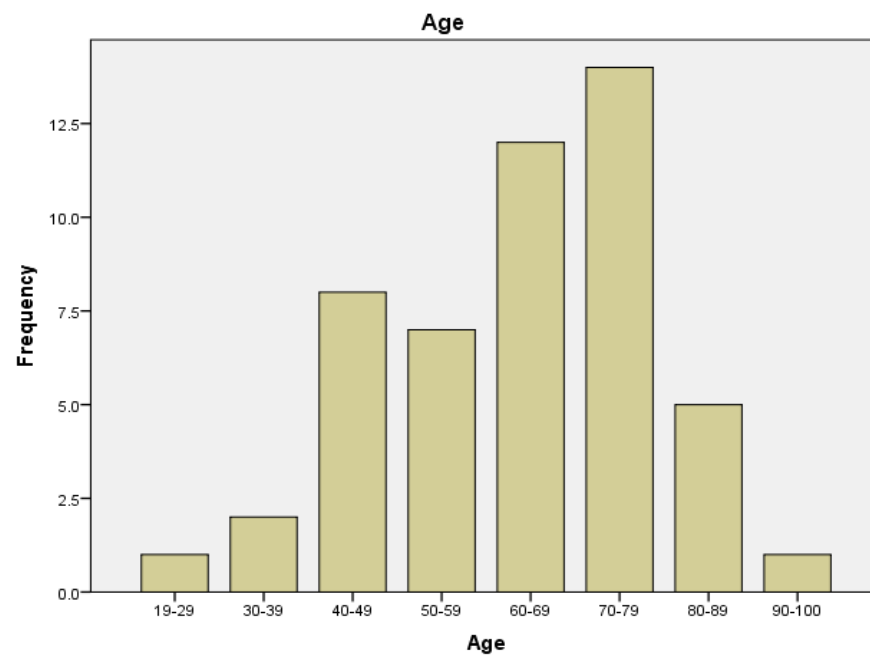


Fig (4-2): Bar graph demonstrating the age categories

Table (4-3): Shows variable statistics age, CTR and age of hypertension

Std. Deviation	Mean	Maximum	Minimum	Age
15.117	61.62	90	19	Age
6.873	9.32	30	1	Years
0.052262	0.53560	0.680	0.420	CTR

Fig (4-3): A scatter plot diagram shows the correlation between CTR and duration of hypertension

Fig (4-4): A scatter plot diagram shows the correlation between CTR and age and linear relationship

Discussion, Conclusion and Recommendations

-:Discussion 5-1

This study aimed to measure the heart size in hypertensive patients using chest x-ray. A number of 50 hypertensive subjects (82% were females and 18% were males) underwent chest x-ray during the period from September to December 2015.

The frequency distribution of the age group that was commonly affected by hypertension was calculated; the most affected age group was (70-79) (14) 28% , (60-69) (12) 24%, (40-49) (8) 16%, (50-59) (7) 14%, (80-89) (5) 10%, (30-39) (2) 4%, (19-29) (1) 2%, (90-100) (1) 2% as (shown in table (4-2) and fig (4-2

The results showed that the mean value of age, hypertension age (years) and CTR were (61.62 ± 15.117) (9.32 ± 6.873) (0.5356 ± 0.052262) respectively as shown in

table (4-3), as compared to Mohamed Yousef et al 2014 study which dealt with normal patients which showed mean values of CTR of males and females were 44 and 42 .respectively

A correlation was made between CTR and age of hypertension in order to investigate the effect of hypertension on the measurement of CTR, and it was found that there was the direct relation between CTR and age of hypertension, CTR increase by 0.001 starting from 0.526 for every one year increment in hypertension and $R^2=0.016$ which means that this correlation was weak

A correlation was made between CTR and age and it was found that there was a direct relation between CTR and age. CTR increase by 0.001 starting from 0.462 for every one year increment in age and $R^2=0.116$ which means a .weak correlation between CTR and age

-:Conclusion 5-2

This study was done on hypertensive patients to measure the CTR and detect the relation between age, age of hypertension, data collection from patients and analysis .this variables

From the study it could be concluded that there was a direct relation between CTR and age of hypertension but there was a weak correlation between this variables. In

other words, the effect of hypertension age on the
.measurement of CTR was very weak

Also the result showed the there was a direct relation
between CTR and age of patients, although the correlation
was weak. That means the effect of age to the
.measurement of CTR was weak

It can be concluded that the effect of age on the CTR was
.greater than on hypertension

-.Recommendations 5-3

Hypertensive patients should have regular measurements
.to blood pressure to control the disease

Future study should be done to study the effect of
.hypertension on the shape of the heart

In chest x-ray technique the FFD should be adjusted to
..(150-180cm

.The position of patients should be (PA) if possible

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Data collection sheet

CTR	Years	Gender	Age	No
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-:Sample of data

CTR	Years	Gender	Age	No
$0.55=3+6.5/17$	7	Female	60	1
$0.47=2.3+5.2/15.8$	7	Female	30	2
$0.54=3.5+4.5/14.7$	10	Male	70	3
$0.54=2+5.3/13.3$	8	Female	47	4
$0.63=3.5+6/15$	4	Male	90	5
$0.5=2.5+5/15$	5	Female	40	6
$0.53=5.4+12/33$	25	Male	75	7
$0.57=2.5+5/13$	9	Female	70	8

0.54=3+6/16.5	9	Female	41	9
0.56=2.5+6/15	12	Female	60	10

Chest x-rays

Male patient , 52 years old, 4 years of hypertension, CTR
0.52



Female patient, 65 years old, 15 years of hypertension,
CTR 0.62



Male patient , 65 years old, 10 years of hypertension, CTR
0.60



Female patient, 47 years old, 8 years of hypertension, CTR
0.54



