



## Sudan University of Science and Technology College of Graduate Studies



#### The Study of Pulmonary Vessels in Patients with Pulmonary Embolism using Computed Tomography

دراسة الاوعية الدموية للرئتين لدى المرضى المصابين بمرض الإنسداد الرئوى بإستخدام التصوير الإشعاعي الطبقي

A Thesis submitted Partial Fulfillment of Requirements of the Msc Degree in Medical Diagnostic Radiological Technology

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## الآية



#### قال الله تعالى:

(قَالَ رَبِّ اشْرَحْ لِي صَدْرِي (25) وَيَسِرَّ لِي أَمْرِي (26) وَيَسِرَّ لِي أَمْرِي (26) وَالله وَالله عُقْدَةً مِنْ لِسِنَانِي (27) يَفْقَهُوا قَوْلِي )(28)صدق الله العظيم

سورةطه ( 25-28 )

### **Dedication**

TO SOULS OF MY PARENTS

MY BROTHERS & SISTERS

AND MY FRIENDS

#### Acknowledgment

First of all thanks to **ALLAH** for helping me to achieve this work. I would like also to express my deep gratitude to **Dr. Mona Ahmed Mohamed** for her keen supervision, help, and support during my completion of thesis, and gave me much of her effort, experience, cooperative and magnificent help, and close supervision throughout the work. Without her support and help this thesis would not have come to an end. Having completed this research, I would like to thanks all those who directly or indirectly extend support to the research. And my great appreciation is extended to all those who shared either practically or morally in the accomplishment of this work.

Finally My appreciation and deepest thanks to the staff of Radiology Department in **Mubarak Alkabir Hospital** (**Kuwait city**) for their great assistance for providing all practical facilities for the study and for their great efforts in helping me in data collection. I want to thanks the staff of college and doctors of Medical Radiological Sciences, for there scientific help and valuable advices. I would like to extend my sincerest thanks and appreciation to those patients who helped me accomplish this study.

#### **Abstract**

The aim of the study was to study the pulmonary vessels in patients with pulmonary embolism PE using Computed Tomography scan (CT). this study was conducted at Mubarak Alkabir Hospital (Kuwait city); in the period between (2016 – 2017) a sample of (54) patients with clinically diagnosed as PE was enrolled, their mean ages were (52.20  $\pm$  17.08) years, the sample included both genders, (23) patients were females while (31) were males and their ages were ranged from (23 - 86) years old. (CTPA) scans were acquired, all patients were examined by using multislice (CT) scanner, the data collected by special designed sheet from findings and analyzed statistically by using an (IBM) (SPSS) Statistics software package. The PE sample was directed to study the relationship of PE with the presence of pulmonary vessels measurements, also to know the plural-parenchymal findings and abnormalities and the patients complains correlation associated with the PE were investigated. The results showed that the right and left main pulmonary arteries diameters changes were found to be significantly related to the presence of PE at (p  $\leq$  0.05), while there were no significant difference in the main pulmonary artery, right distal pulmonary artery, left distal pulmonary artery diameters measurements at (p  $\geq$  0.05). pleural effusion, Consolidation, ground glass opacifications, Atelectasis, were present in the majority of patients. The common complains from PE patients were tachycardia, shortness of breathing, chest pain, lower limb swelling, and cough. (CTA) plays an important role in the diagnostic evaluation of patients with PE this due to it is easily accessible and excellent non-invasive method for the clot visualization and pleuroparenchyma evaluation.

#### المستخلص

هدفت الدراسة إلي دراسة الأوعية الدموية للرئتين عند المرضى المصابين بمرض الإنسداد الرئوي باستخدام التصوير الإشعاعي الطبقي.

أجريت هذه الدراسة في مستشفى مبارك الكبير (مدينة الكويت). في الفترة ما بين2016-2017 تمثلت في 54 مريضا مع تشخيص سريري الإنسداد الرئوي، وكان متوسط أعمار هم  $52.20\pm 17.08$  سنة، وشملت العينة كلا الجنسين. 23 مريض كانو من النساء و 31 كانو من الرجال و كانت اعمار هم تتراوح مابين 23-86 سنة. تم إجراء صور التصوير المقطعي للأوعية الدموية للرئتين، تم فحص جميع المرضى بإستخدام جهاز التصوير الطبقى متعدد المقاطع، البيانات تم جمعها عن طريق ورقة مصممة خصيصا من النتائج و تحليلها إحصائيا بإستخدام (SPSS) (IBM) . تم توجية العينة لدراسة علاقة الانسداد الرئوى بوجود تغييرات في قياسات الأوعية الدموية الرئوية ، ايضا لمعرفة إذا كان وجود النتائج الخاصة بالنسيج اللبي الخاص بغشاء البلورا و أمراضة و شكوي المرضى له علاقة ارتباط بالانسداد الرئوى. أظهرت النتائج أن هنالك علاقة ارتباط بين الانسداد الرئوى و التغييرات في اقطار الشريانين الرئويين الايمن و الايسر متمثلة في علاقة ارتباط  $P \le 0.05$  ، في حين لم يكن هناك فرق كبير في قياسات الشريان الرئوي الرئيسي و الشريانين الرئويين الأيمن والأيسر البعيدين بعلاقة ارتباط  $p \ge 0.05$ . الإنسكاب البلوري، التصلب الرئوي، التعتيمات، وكذلك الإنخماص فقد كانت موجودة عند معظم المرضى كانت شكوى مرضى الانسداد الرئوى تتمثل في زيادة خفقان القلب، ضيق في التنفس، ألم في الصدر، تورم في الأطراف السفلي، والكحة . ألتصوير المقطعي للأوعية يلعب دورا هاما في التقييم ألتشخيصي للمرضى الذين يعانون من الإنسداد الرئوى هذا يرجع إلى أنه وسيلة من الوسائل من السهل الوصول إليها وممتازة بطريقة غير تدخلية في تصوير الجلطة و تقييم النسيج اللبي للبلور ا

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#### **Abbreviations**

Abbreviation	Phrase
CHD	Congenital Heart Disease
CM	Contrast Media
СО	Cardiac Out put
COPD	Chronic Obstructive Pulmonary Disease
CT	Computed Tomography
CTA	Computed Tomography Angiography
СТРА	Computed Tomography of Pulmonary
	Arteries Angiography
CXR	Chest X-Ray
DAS	Data Acquisition System
DVT	Deep Venous Thrombosis
ECG	Electrocardiography
FOV	Field OF View
HU	Hounsfield Unit
IPAH	Idiopathic Pulmonary Arterial Hypertension
KVP	Kilo Voltage Peak
MA	Milliampere
MAS	Milliampere / Second
MDCT	Multi-Detector Computed Tomography
MDCTPA	Multidetector Computed Tomography
	Pulmonary Angiography
MPAD	Main Pulmonary Artery Diameter
MPR	Multi-Planar Reconstruction
MRI	Magnetic Resonance Imaging
MSCT	Multiple Slice Computed Tomography
MIP	Maximum Intensity Projection

PA	Pulmonary Arteriography
PAP	Pulmonary Artery Pressure
PE	Pulmonary Embolism
PVR	Pulmonary Vascular Resistance
RMPA	Right Main Pulmonary Artery
SOB	Short OF Breath
VTE	Venous Thromboembolism
VR	Virtual Reality
2D	Two Dimension
3D	Three Dimension

## **CHAPTER ONE Introduction**

#### Chapter one

#### 1.1Introduction:

Pulmonary vascular disease span a variety of disease entities including pulmonary arterial hypertension (PAH), pulmonary venous hypertension, pulmonary embolism, pulmonary arteriovenous malformation, pulmonary arterial stenosis, pulmonary arterial aneurysm, pulmonary veno-occlusive disease (PVOD)and pulmonary capillary hemangiomatosis.(Budoff and Shinbane. 2016).

#### 1.1.1Pulmonary embolism (PE).

Is a sudden blockage in a lung artery. The blockage is usually caused by a blood clot that travels to the lung from a peripheral vein. The pulmonary embolism is a common disease with potentially high morbidity and mortality because many patients with PE are initially asymptomatic, most patients who have symptoms often have a typical and / or nonspecific symptom, such as dyspnea, tachypnea, and chest pain.PE is a serious condition that can lead to damage part of the lung, low oxygen levels in the blood and if a blood clots is large PE can cause death. Over the past decade, computed tomography angiography (CTA) has become the primary tool for the diagnosis of PE because it is non invasive, takes minutes to perform, and has very high sensitivity and specifity. It is therefore not surprising that (CTA) utilization has grown rapidly.(Andrew J.Schissler2013 ). The most important advantages of (CT) are the both mediastinal and parenchymal structures can be evaluated, and thrombus can be directly visualized, 16 detector row (CT) scanners now allow for coverage of the entire chest with 1-mm or submillimeter resolution within a short single breath-hold now less than 10 sec in the case of 16 detector row (CT). (Schoepf and Castello 2004). The near isotropic nature of high-spatial-resolution multi-detector row (CT) data lends it self into two and three dimentional visualization.

This may in some instances improve diagnosis of PE. (Schoepf and Castello 2004). This study was designed to answer two basic questions based on (CTPA) findings that done for clinical suspicion of PE: firstly, what are the pleural parenchymal abnormalities associated with PE, secondly, correlation of PE with the presence of pulmonary vessels measurements.

#### 1.2Problem of the study:

The pulmonary embolism is a common disease with potentially high morbidity and mortality due to clinical and conventional misdiagnosis. Non invasive method is needed for study and evaluate patients with PE.

#### **1.3Objectives of the study:**

#### 1.3.1General objectives:

To study the pulmonary vessels in patients with pulmonary embolism PE using Computed Tomography scan CT.

#### 1.3.2Specific objectives:

- To study the pulmonary trunk diameter.
- To study the main right and left pulmonary arteries diameter.
- To study the distal pulmonary artery diameter.
- To investigate the presence of the pleural-parenchymal findings correlates with the PE.
- To investigate the presence of the complains findings correlates with the PE.

#### 1.4Overviews of the study:

This study was fall into five chapters, chapter one is an introduction, problem of the study, objectives and overview. Chapter two include literature review while chapter three include material used and the method of data collection and analysis. Chapter four presents the result

of the study in a line graphs and tables and finally chapter five which include the discussion, conclusion and recommendations.

# CHAPTER TWO Theoretical Background and Literature Review

#### **Chapter Two**

#### Theoretical Background and literature review

#### 2.1Anatomy:

#### 2.1.1The Heart

The heart is a roughly cone-shaped hollow muscular organ. It is about 10 cm long and is about the size of the owner's fist. It weighs about 225 g in women and is heavier in men (about 310 g). The heart lies in the thoracic cavity in the mediastinum between the lungs. It lies obliquely, a little more to the left than the right, and presents abase above, and an apex below. The apex is about 9 cm to the left of the midline at the level of the 5th intercostals space, i.e. a little below the nipple and slightly nearer the midline. The base extends to the level of the 2nd rib. (Waugh and Grant, 2014).

#### 2.1.1.1Organs associated with the heart.

**Inferiorly**, the apex rests on the central tendon of the diaphragm

**Superiorly**, the great blood vessels, i.e. the aorta, superior venacava, pulmonary artery and pulmonary veins

**Posteriorly**, the oesophagus, trachea, left and right bronchus, descending aorta, inferior venacava and thoracic vertebrae

**Laterally**, the lungs, the left lung overlaps the left side of the heart **Anteriorly**, the sternum, ribs and intercostals smuscles. (Waugh and Grant, 2014).

#### 2.1.1.2Layers of the heart

The heart is composed of three layers of tissue. Pericardium, myocardium and endocardium. The Pericardium is made up of two sacs. The outer sac consists of fibrous tissue and the inner of a continuous double layer of serous membrane. The myocardium is composed of specialized cardiac muscle found only in the heart. It is not under voluntary control but, like

skeletal muscle, cross-stripes are seen on microscopic examination. (Waugh and Grant, 2014).

The myocardium is thickest at the apex and thins out towards the base. This reflects the amount of work each chamber contributes to the pumping of blood. It is thickest in the left ventricle. (Waugh and Grant, 2014).

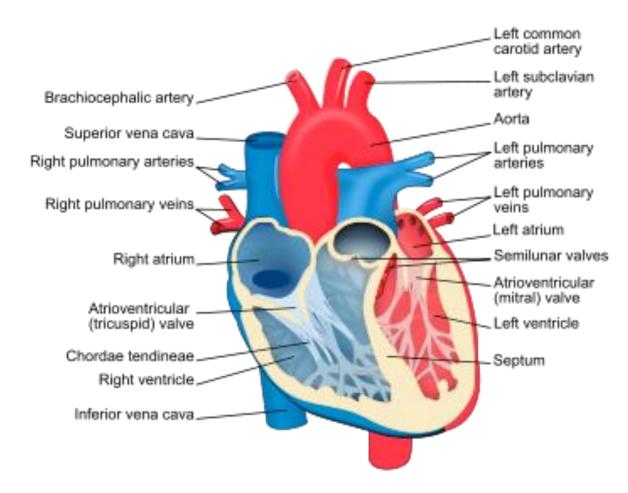
The endocardium this forms the lining of the myocardium and the heart valves. It is a thin, smooth, glistening membrane which permits smooth flow of blood inside the heart. It consists of flattened epithelial cells, continuous with the endothelium that lines the blood vessels. (Waugh and Grant, 2014).

#### 2.1.1.3Interior of the heart

The heart is divided into a right and left side by the septum, a partition consisting of myocardium covered by endocardium. After birth blood cannot cross the septum from one side to the other. Each side is divided by an atrio-ventricular valve into an upper chamber, the atrium, and a lower chamber, the ventricle. (Waugh and Grant, 2014).

The atriov-entricular valves are formed by double folds of endocardium strengthened by a little fibrous tissue. The right atrio-ventricular valve (tricuspid valve) has three flaps or cusps and the left atrioventricular valve (mitral valve) has two cusps. The valves between the atria and ventricles open and close passively according to changes in pressure in the chambers. They open when the pressure in the atria is greater than that in the ventricles. During ventricular rsystole (contraction) the pressure in the ventricles rises above that in the atria and the valves snap shut preventing backward flow of blood. The valves are prevented from opening upwards into the atria by tendinous cords, called chordate tendineae, which extend from the inferior surface of the cusps to little

projections of myocardium covered with endothelium, called papillary muscles.(Waugh and Grant, 2014).



**Figure 2.1** Internal Anatomy of the Heart. (wikipedia.org, 2016).

#### 2.1.2Arteries and arterioles

These are the blood vessels that transport the oxygenated blood away from the heart. They vary considerably in size and their walls consist of three layers of tissue. Outer layer of fibrous tissue, middle layer of smooth muscle and elastic tissue, inner lining of squamous epithelium called endothelium. (Waugh and Grant, 2014).

#### 2.1.3Veins and venules

The veins are the blood vessels that return the deoxygenated blood at low pressure to the heart. The walls of the veins are thinner than those of

arteries but have the same three layers of tissues. They are thinner because there is less muscle and elastic tissue in the middle layer. When cut, the veins collapse while the thicker-walled arteries remain open. Some veins possess valves, which prevent back flow of blood, ensuring that it flows towards the heart. Valves are abundant in the veins of the limbs, especially the lower limbs where blood must travel a considerable distance against gravity when the individual is standing. Valves are absent in very small and very large veins in the thorax and abdomen. They are formed by a fold of tunica intima strengthened by connective tissue. The cusps are semilunar in shape with the concavity towards the heart. The smallest veins are called venules. (Waugh and Grant, 2014).

#### 2.1.4The Thoracic cavity

The cavity of the thorax can be divided into a median partition, called the mediastinum, and the laterally placed pleurae and lungs. The lungs are covered by a thin membrane called the visceral pleura, which passes from each lung at its root to the inner surface of the chest wall, where it is called the parietal pleura. In this manner, two membranous sacs called the pleural cavities are formed, one on each side of the thorax, between the lungs and the thoracic walls. (Snell, 2011)

#### 2.1.5Mediastinum

The mediastinum, though thick, is a movable partition that extends superiorly to the thoracic outlet and the root of the neck and inferiorly to the diaphragm. It extends anteriorly to the sternum and posteriorly to the vertebral column. It contains the remains of the thymus, the heart and large blood vessels, the trachea and esophagus, the thoracic duct and lymph nodes, the vagus and phrenic nerves, and the sympathetic trunks. For purposes of description, the mediastinum is divided into superior and inferior mediastina. The inferior is further subdivided into the middle

mediastinum which consist of the pericardium and heart. The anterior mediastinum which is space between the pericardium and the sternum. The posterior mediastinum which lies between the pericardium and the vertebral column. (Snell, 2011).

#### 2.1.6Pleurae

The pleurae and lungs lie on either side of the mediastinum within the chest cavity. Each pleura has two parts: a parietal layer, which lines the thoracic wall, covers the thoracic surface of the diaphragm and the lateral aspect of the mediastinum, and a visceral layer, which completely covers the outer surfaces of the lungs. The parietal and visceral layers of pleura are separated from one another by a slit like space, the (pleural cavity). The pleural cavity normally contains a small amount of tissue fluid, the pleural fluid, which covers the surfaces of the pleura as a thin film and permits the two layers to move on each other with the minimum of friction. (Snell, 2011)

#### **2.1.7Lungs**

The lungs are situated so that one lies on each side of the mediastinum. There are soft and spongy and very elastic. They are therefore separated from each other by the heart and great vessels and other structures in the mediastinum. Each lung is conical, covered with visceral pleura, and suspended free in its own pleural cavity, being attached to the mediastinum only by its root. Each lung has a blunt apex, which projects upward into the neck for about 1 inch. (2.5 cm) above the clavicle; a concave base that sits on the diaphragm; a convex costal surface, which corresponds to the concave chest wall; and a concave mediastinal surface, which is molded to the pericardium and other mediastinal structures. At about the middle of this surface is the hilum, a depression in which the bronchi, vessels, and nerves that form the root enter and leave the lung.

The anterior border is thin and overlaps the heart; it is here on the left lung that the cardiac notch is found. The posterior border is thick and lies beside the vertebral column. The right lung is slightly larger than the left and is divided by the oblique and horizontal fissures into three lobes: the upper, middle, and lower lobes. The left lung is divided by a similar oblique fissure into two lobes: the upper and lower lobes. There is no horizontal fissure in the left lung. (Snell, 2011).

#### 2.1.8Pulmonary trunk

The pulmonary trunk carries deoxygenated blood from the right ventricle of the heart to the lung circulation. It is about 5 cm in length and 3 cm in diameter, and arises from the right ventricle base It is a short vessel, arising from the pulmonary conus of the right ventricle at the pulmonary semilunar valves. The pulmonary trunk lies totally within the pericardium. It divides into the left and right pulmonary arteries The diameter of the right pulmonary artery ranges between 17 and 30 mm (mean, 23.4 mm). The caliber of the main pulmonary artery is between 20 and 30 mm (mean, 26.4 mm). The sum of the diameters of the left and right main branches is greater than the diameter of the main pulmonary artery (Kaufman, 1997).

#### 2.1.8.1Right Pulmonary artery

The right pulmonary artery is only slightly smaller in caliber than the main artery, as seen in angiograms. It runs a horizontal, sometimes slightly downward. Where it divides into superior and inferior branches. It lies behind the ascending aorta and the superior vena cava and in front of the tracheal bifurcation and esophagus. The right pulmonary artery divides at the right hilum into two main branches: the ascending branch, to the right upper lobe, and the descending branch, to the right middle lobe and the right lower lobe. The ascending branch of the right

pulmonary artery supplies the right upper lobe. The descending branch of the right pulmonary artery supplies the right middle and lower lobes.(Kaufman, 1997).

#### 2.1.8.2Left Pulmonary artery

The left pulmonary artery is a short continuation of the main pulmonary artery. It lies in front of the descending aorta, beneath the curve of the aortic arch the left pulmonary artery is short and bifurcates in the left hilum into ascending and descending branches, which supply the left upper and lower lobes, respectively. (Kaufman, 1997).

#### 2.1.8.3Pulmonary arteries, veins and capillaries

The pulmonary arteries and veins have three distinct vascular layers. The tunica intima is the innermost layer of the vessel and is composed of a homogenous population of endothelial cells. The medial layer, the tunica media, is comprised of smooth muscle cells. The tunica externa is the outermost layer that consists primarily of collagen and fibroblasts. Each respective layer is separated by an extracellular matrix known as the basement membrane. Within the distal component of the lung, an extensive capillary network separates arteries from veins. The structural composition of pulmonary arteries is functionally altered as it extends down the length of the vessel towards the base of the lung. Pulmonary arteries can be defined according to their structural composition as elastic, muscular or partially muscular pulmonary arteries. Elastic vessels contain numerous elastic laminae bound by external and internal elastic laminae and extend peripherally into transitional vessels which contain fewer elastic laminae. Once only an internal and external elastic lamina exists around the smooth muscle cell layer, these vessels are termed muscular. As the artery extends into the more distal portions of the lungs, the smooth muscle cell layer becomes sparse (partially-muscular) or absent (non-muscular) prior to extension into the capillary bed. (DeMello & Reid, 1991; Jones & Capen, 2011). Pre-acinar arteries (including the main pulmonary artery) are associated with bronchi, bronchioles or terminal bronchioles and contain numerous elastic laminae between smooth muscle layers and are more than 3200µm in diameter. As it extends beyond the ninth airway generation, the vessels become muscular and are generally more than 150µm in diameter. (Elliot & Reid, 1964; Jones & Capen, 2011). Partially muscular pulmonary arteries are typically 75µm to 90µm in diameter. (Jones & Capen, 2011). Pulmonary arteries are much less muscular than systemic arteries as the pressure and resistance is much lower within these arteries. Pulmonary arteries follow a branching pattern closely associated with the branching pattern of the bronchial tree, although there are many more pulmonary arterial branches than there are bronchial branches and these increase within the periphery. (Elliot & Reid, 1964). The pulmonary artery will eventually extend into the alveolar capillaries, which form a dense anastomosing hexagonal network for which gas exchange can occur. (Weibel, 1963) Oxygenated blood is then drained into the pulmonary vein, which carries the blood to the left atria where it is retained until diastole, allowing it to enter the left ventricle for subsequent pumping around the body.

#### 2.2Physiology:

#### 2.2.1Flow of blood through the heart.

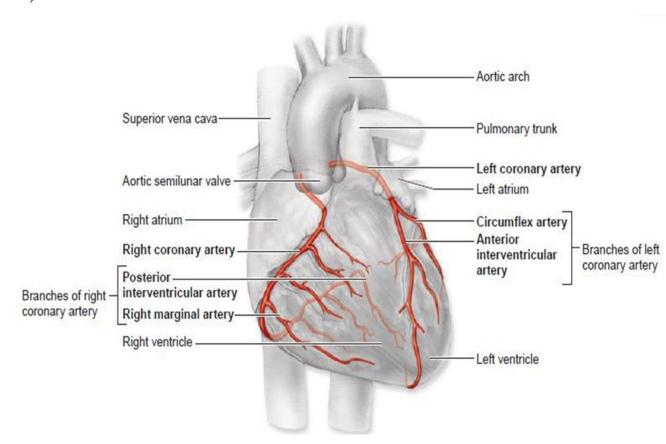
The two largest veins of the body, the superior and inferior venae cavae, empty their contents into the right atrium. This blood passes via the right atrioventricular valve into the right ventricle, and from there it is pumped into the pulmonary artery or trunk (the only artery in the body which carries deoxygenated blood). The opening of the pulmonary artery is guarded by the pulmonary valve, formed by three semilunar cusps. This valve prevents the back flow of blood into the right ventricle when the

ventricular muscle relaxes. After leaving the heart the pulmonary artery divides into left and right pulmonary arteries, which carry the venous blood to the lungs where exchange of gases takes place: carbon dioxide is excreted and oxygen is absorbed. Two pulmonary veins from each lung carry oxygenated blood back to the left atrium. Blood then passes through the left atrioventricular valve into the left ventricle, and from there it is pumped into the aorta, the first artery of the general circulation. The opening of the aorta is guarded by the aortic valve, formed by three semilunar cusps. From this sequence of events it can be seen that the blood passes from the right to the left side of the heart via the lungs, or pulmonary circulation. However, it should be noted that both atria contract at the same time and this is followed by the simultaneous contraction of both ventricles. The muscle layer of the walls of the atria is very thin in comparison with that of the ventricles. This is consistent with the amount of work it does. The atria, usually assisted by gravity, only propel the blood through the atrioventricular valves into the ventricles, whereas the ventricles actively pump the blood to the lungs and round the whole body. The muscle layer is thickest in the wall of the left ventricle. The pulmonary trunk leaves the heart from the upper part of the right ventricle, and the aortaleaves from the upper part of the left ventricle. (Waugh and Grant, 2014).

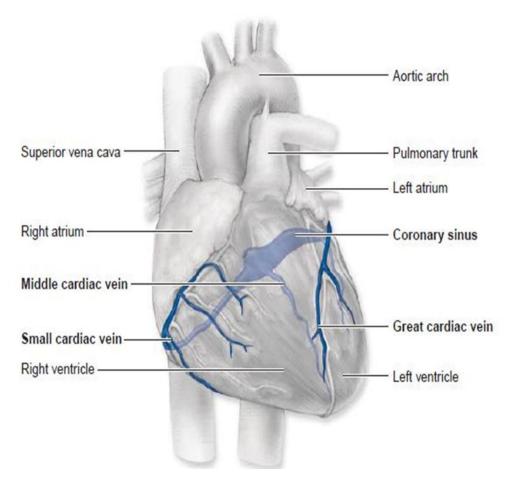
#### 2.2.2Blood supply to the heart

Arterial supply: The heart is supplied with arterial blood by the right and left coronary arteries which branch from the aorta immediately distal to the aortic valve. The coronary arteries receive about 5% of the blood pumped from the heart, although the heart comprises a small proportion of body weight. This large blood supply, especially to the left ventricle, highlights the importance of the heart to body function. The coronary arteries traverse the heart, eventually forming a vast network of

capillaries. (Waugh and Grant, 2014). Venous drainage: Most of the venous blood is collected into several small veins that join to form the coronary sinus which opens into the right atrium. (Waugh and Grant, 2014).



**Figure 2.2**Coronary Circulation. Anterior view of (a) coronary arteries.(Tortora and Derrickson, 2012).



**Figure 2.3** Coronary Circulation. Anterior view of (a) coronary veins. (Tortora and Derrickson, 2012)

#### 2.2.3The cardiac cycle

The function of the heart is to maintain a constant circulation of blood throughout the body. The heart acts as a pump and its action consists of a series of events known as the cardiac cycle. During each heartbeat, or cardiac cycle, the heart contracts and then relaxes. The period of contraction is called systole and that of relaxation, diastole. (Waugh and Grant, 2014).

#### 2.2.4Cardiac output

The cardiac output is the amount of blood ejected from the heart. The amount expelled by each contraction of the ventricles is the stroke volume. Cardiac output is expressed in liters per minute (1/min) and is

calculated by multiplying the stroke volume by the heart rate (measured in beats per minute).

Cardiac output = Stroke volume x Heart rate. In a healthy adult at rest, the stroke volume is approximately 70 ml and if the heart rate is 72 per minute, the cardiac output is 51/minute. This can be greatly increased to meet the demands of exercise to around 251/minute, and in athletes up to 351/minute. This increase during exercise is called the cardiac reserve. When increased blood supply is needed to meet increased tissue requirements of oxygen and nutrients, heart rate and/or stroke volume can be increased. (Waugh and Grant, 2014)

#### 2.2.5Stroke volume

The stroke volume is determined by the volume of blood in the ventricles immediately before they contract, i.e. the ventricular end-diastolic volume (VEDV), sometimes called preload. This depends on the amount of blood returning to the heart through the superior and inferior venae cavae (the venous return). Increased VEDV leads to stronger myocardial contraction, and more blood is expelled. In turn the stroke volume and cardiac output rise. This capacity to increase the stroke volume with increasing VEDV is finite, and when the limit is reached, i.e. the cardiac output cannot match the venous return, the cardiac output decreases and the heart begins to fail. Other factors that increase myocardial contraction include, increased stimulation of the sympathetic nerves innervating the heart and hormones, e.g. adrenaline, noradrenaline, thyroxine.

(Waugh and Grant, 2014).

#### 2.2.6 Circulations of the blood

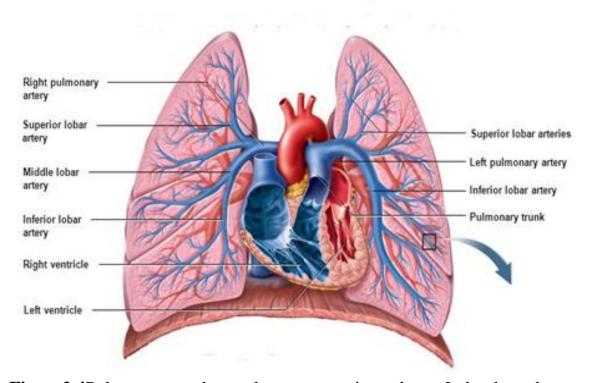
#### 2.2.6.1Systemic circulation

Systemic circulation is the movement of blood from the heart through the body to provide oxygen and nutrients, and bringing deoxygenated blood back to the heart. Oxygen-rich blood from the lungs leaves the pulmonary circulation when it enters the left atrium through the pulmonary veins. The blood is then pumped through the mitral valve into the left ventricle. From the left ventricle, blood is pumped through the aortic valve and into theaorta, the body's largest artery. The aorta arches and branches into major arteries to the upper body before passing through the diaphragm, where it branches further into arteries which supply the lower parts of the body. The arteries branch into smaller arteries, arterioles, and finally capillaries. Waste and carbon dioxide diffuse out of the cell into the blood, while oxygen in the blood diffuses out of the blood and into the cell. The deoxygenated blood continues through the capillaries which merge into venules, then veins, and finally the venae cavae, which drain into the right atrium of the heart. From the right atrium, the blood will travel through the pulmonary circulation to be oxygenated before (wikipedia.org, 2016).

#### 2.2.6.2 Pulmonary circulation

At birth we are primed to take our first breath. Instantly there is a dramatic reduction in pulmonary vascular resistance (PVR) accompanied by closure of the ductus arteriosus, which is used in fetal life to bypass the pulmonary circulation. Subsequently the pulmonary arterial pressures (PAPs) rise and the lungs assume their prime function of gaseous exchange. (wikipedia.org, 2016)Pulmonary circulation is the movement of blood from the heart to the lungs for oxygenation, then back to the heart again. Oxygen-depleted blood from the body leaves the systemic circulation when it enters the right atrium through the superior and inferior venae cavae. The blood is then pumped through the tricuspid valve into the right ventricle. From the right ventricle, blood is pumped through the pulmonary valve and into the pulmonary artery. The pulmonary artery splits into the right and left pulmonary arteries and

travel to each lung. At the lungs, the blood travels through capillary beds on the alveoli where respiration occurs, removing carbon dioxide and adding oxygen to the blood. The alveoli are air sacs in the lungs that provide the surface for gas exchange during respiration. The oxygenated blood then leaves the lungs through pulmonary veins, which returns it to the left atrium, completing the pulmonary circuit. Once entering the left heart, the blood flows through the bicuspid valve into the left ventricle. From the left ventricle, the blood is pumped through the aortic valve into the aorta to travel through systemic circulation, delivering oxygenated blood to the body before returning again to the pulmonary circulation. (wikipedia.org, 2016).



**Figure 2.4** Pulmonary trunk to pulmonary arteries to lungs Lobar branches for each lobe. (wikipedia.org, 2016)

#### **2.2.6.2.1**The function of the pulmonary circulation

The primary function of the pulmonary circulation is to facilitate gas exchange unloading of carbon dioxide and loading of oxygen. Oxygen is obtained from inspired air and binds with haemoglobin within red blood

cells and is essential to sustain metabolic processes throughout the entire body. Re-oxygenation is exquisitely facilitated by the gas-blood interface between the alveoli and the extensive alveolar capillary network. In addition to facilitating gas exchange, the pulmonary circulation functions as a filtration system to remove fine particles and potentially lethal thromboemboli from the mixed venous blood before it returns to the systemic circulation. (Comroe, 1966) This process is chiefly regulated by the pulmonary endothelium which releases mediators that promotes fibrinolysis. The location of the lungs together with the vast surface area of the pulmonary vasculature allows for this unique filtration function. Additionally the pulmonary circulation also serves as a blood reservoir for the left ventricle. (Comroe, 1966).

#### 2.2.6.2.2 Regulation of blood flow in the pulmonary circulation

Pulmonary blood flow is most profoundly regulated by gravity and increases about 9 fold from the apex to the base of the lung. (West et al., 1964) Arterial, venous and alveolar pressures affect the distribution of blood flow within the lungs (West et al., 1964). Blood flow and ventilation is regulated by ventilation-perfusion matching (West &Dollery, 1960). In 1946, Von Euler and Liljestrand characterised a vital difference in response to hypoxia between the systemic and pulmonary circulations (Euler &Liljestrand, 1946). In the systemic circulation hypoxia caused vasodilatation. In contrast, they observed that the pulmonary arteries uniquely constricted in response to hypoxia. This is perhaps not surprising considering the functional role of the two separate systems. In the pulmonary circulation the vasoconstrictor response to hypoxia aids in moving the passage of blood to well aerated areas mediating ventilation-perfusion matching. In the systemic circulation, hypoxia results in vasodilation in order to increase perfusion to meet the energy requirements of tissues and organs. In the lungs, ventilationperfusion matching is also achieved by airway constriction in response to pulmonary arterial occlusion. This serves to direct inspired air towards alveoli with a denser blood flow. The matching of blood to a sufficient oxygen supply is therefore a critical regulator of blood flow in the lungs. (Zilmer Johansen, 2014).

#### 2.3Pathology of Pulmonary vascular system:

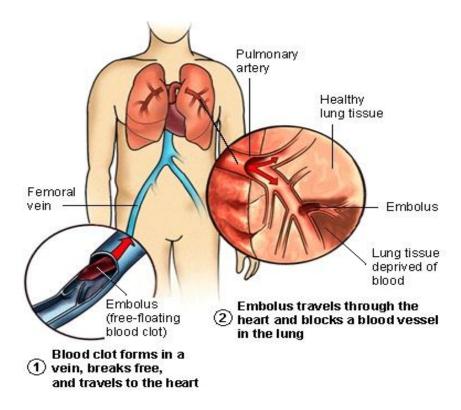
#### 2.3.1Pulmonary embolism

Pulmonary embolism is a blockage in one or more pulmonary arteries in the lungs. In most cases, pulmonary embolism is caused by blood clots that travel to the lungs from the legs or, rarely, other parts of the body (deep vein thrombosis). Because pulmonary embolism almost always occurs in conjunction with deep vein thrombosis, most doctors refer to the two conditions together as venous thromboembolism. Although anyone can develop deep vein thrombosis (DVT) and pulmonary embolism, factors such as immobility, cancer and surgery increase the risk. Pulmonary embolism can be life-threatening, but prompt treatment can greatly reduce the risk of death. Taking measures to prevent blood clots in the legs will help protect against pulmonary embolism. (Swanson et al., 2008).

Acute pulmonary embolism is an under-diagnosed but potentially fatal condition. This condition presents with a wide clinical spectrum, from asymptomatic small PE to life-threatening one causing cardiogenic shock.(Çobanoğlu, 2012).Deep venous thrombosis (DVT) and pulmonary embolism constitute clinical presentations of the same vascular disease, known as venous thromboembolism (VTE). VTE It is associated with high morbidity and mortality and represents a primary cause of preventable death. There is strong evidence that obesity is an independent risk factor for (DVT) and PE. (Çobanoğlu, 2012).

PE is an obstruction of a pulmonary artery caused by a blood clot, air, fat, or tumor tissue. The most common cause of the obstruction is a blood clot (thrombus) usually from a peripheral vein. Most patients with deep vein thrombosis (DVT) develop PE. Left untreated, PE has a high mortality rate and accounts for 5–10% of all in-hospital deaths. The classic triad of signs and symptoms of PE (hemoptysis, dyspnea, SOB and chest pain) are neither sensitive nor specific, and many patients with PE are initially asymptomatic; most patients who have symptoms often have atypical and/or nonspecific symptoms, such as dyspnea, tachypnea, and chest pain. (Budoff and Shinbane, 2016).

PE is a sudden occurence of a blood clot in a pulmonary artery with obstruction of the blood supply to the lung circulation. Embolization occurs when a venous thrombus is dislodged from the endothelial wall of a vein and passes through to the lung circulation. Depending on its size and length, the embolus may occlude different parts of the arterial branch, from the main pulmonary artery, through the bifurcation (saddle embolus), to the left or right pulmonary artery along to the smaller branching pulmonary arteries. The clots arise to a large extent from thrombi within the large deep veins in the legs, mainly the iliac, femoral, and popliteal veins, and less commonly from more distal veins or veins from other locations, such as the heart. (Marten soderberg, 2008).



**Figure 2.5** Pulmonary Embolism: Blood clot from the leg vein travels to the heart and lodged inside a blood vessel in the lungs, blocking blood supply.(wikipedia.org, 2016).

# 2.3.1.1 Classifications of pulmonary embolism

Acute PE: PE developed over a short period of time and Chronic PE: PE with recurring embolization despite treatment. The disease develops over several years. Clinical classification of PE: Idiopathic PE: No known risk factor, Primary PE: Thrombophilia as risk factor, Secondary PE: Identifiable risk factor(s) such as pregnancy, cancer, surgery or trauma. Anatomically massive PE: More than 50% obstruction of the vascular bed or two or more of the lobar arteries. Clinically massive PE:PE with signs of shock or hypotension (blood pressure <90 mmHg or a pressure drop >40 mmHg for >15 min) Non massive PE: all other PE. Sub massive PE: Nonmassive PE with signs of right ventricular dysfunction on echocardiography but without hemodynamic instability. Massive and Submassive PE can be divided into Type A: PE with highly mobile emboli, arising from peripheral veins (poorer prognosis). And Type B: PE

with immobile emboli, originating in the RV (better prognosis (Palla, A and Perrser, A. 2000, Wood, K.E. 2002).

#### 2.3.1.2Signs and symptoms of pulmonary embolism

Many pulmonary emboli occur silently, but there are three typical clinical presentations. A clinical deep venous thrombosis not commonly observed, although detailed investigation of the lower limb and pelvic veins will reveal thrombosis in more of the cases. (A.V. Hoffbrand et al, 2001).

# 2.3.1.2.1Small/ medium pulmonary embolism:

In this situation an embolus has impacted in a terminal pulmonary vessel. Symptoms are pleuritic chest pain and breathlessness. Haemoptysis occurs in 30% often three or more days after the initial event. (A.V. Hoffbrand et al, 2001).

# 2.3.1.2.2 Massive pulmonary embolism:

This is a much rare condition where sudden collapse occur due to an acute obstruction of the right ventricular outflow tract. The patient has sever central chest pain (cardiac ischaemia due to lack of coronary blood flow) and becomes shocked, pale and sweaty. Syncope may result if the cardiac output is transiently but dramatically reduced, and death may occur. (A.V. Hoffbrand et al, 2001)

# 2.3.1.2.3 Multiple recurrent pulmonary emboli:

This leads to increased breathlessness often over weeks or months. It is accompanied by weakness, syncope on exertion and occasionally angina. The physical signs are due to pulmonary hypertension that has developed from multiple occlusions of the pulmonary vasculature. (A.V. Hoffbrand et al, 2001)Pulmonary embolism symptoms can vary greatly, depending on how much of the lung is involved, the size of the clots and the overall

health especially the presence or absence of underlying lung disease or heart disease. (Swanson et al., 2008).

#### 2.3.1.3Common signs and symptoms of pulmonary embolism:

**Shortness of breath**: This symptom typically appears suddenly and always gets worse with exertion.

**Chest pain**: The patient feel like he having a heart attack. The pain may become worse when breathe deeply (pleurisy), cough, eat, bend or stoop. The pain will get worse with exertion but won't go away during the rest.

**Cough**: The cough may produce bloody or blood-streaked sputum.

Other signs and symptoms that can occur with pulmonary embolism include: Leg pain or swelling, or both, usually in the calf, Clammy or discolored skin (cyanosis), Fever, Excessive sweating, Rapid or irregular heartbeat, Lightheadedness or dizziness.(Swanson et al., 2008).

PE is a disease with different clinical expressions in different patients. The presentation can vary from a clinically silent disease to an acute lifethreatening condition requiring intensive care treatment thrombolysis. PE is potentially fatal. The clinical accuracy and recognition of signs of PE are often inaccurate, and objective diagnostic tests are mandatory in patients with clinical suspicion of PE. Concomitant medical conditions, especially cardiovascular and pulmonary disease, as well as malignancy, along with findings of ongoing medication, hereditary anamnesis, body constitution, and time from onset of the first symptom (patient's delay), can significantly affect the attending physician's ability to diagnose PE. The degree and extent of the vessel occlusion also influences the presentation. Both symptoms and signs of suspected PE can be found in patients with other diseases when PE is excluded (Miniati, M., & Monti, S 2003; Stein, P.D. & Henry, J.W. 1997).

# 2.3.1.4Causes of pulmonary embolism:

Pulmonary embolism occurs when a clump of material, most often a blood clot, gets wedged into an artery in the lungs. These blood clots most commonly originate in the deep veins of the legs, but they can also come from other parts of the body. This condition is known as deep vein thrombosis (DVT). Not all DVT blood clots result in pulmonary embolism. Occasionally, substances other than blood clots can form blockages within the blood vessels inside the lungs. Examples include: Fat from within the marrow of a broken long bone, Part of a tumor, Air bubbles. It's rare to have a single pulmonary embolism. In most cases, multiple clots are involved but not necessarily all at once. The portions of lung tissue served by each blocked artery are robbed of blood and may die. This is known as pulmonary infarction. This makes it more difficult for the lungs to provide oxygen to the rest of the body. (Swanson et al., 2008).

# 2.3.1.5Risk factors of pulmonary embolism:

Although anyone can develop blood clots and subsequent pulmonary embolism, certain factors can increase your risk.

# 2.3.1.5.1 Medical history

The patient at higher risk if he had venous blood clots or pulmonary embolism in the past. This may be due to inherited disorders that affect blood, making it more prone to clot. In addition, certain medical conditions can put the patient at risk, such as:

**Heart disease**: Cardiovascular disease, specifically heart failure, makes clot formation more likely. (Swanson et al., 2008)

**Cancer**: Certain cancers especially pancreatic, ovarian and lung cancers, and many cancers with metastasis can increase levels of substances that help blood clot, and chemotherapy further increases the risk. Women with

a personal or family history of breast cancer who are taking tamoxifen or raloxifene also are at higher risk of blood clots. (Swanson et al., 2008)

# 2.3.1.5.2Prolonged immobility

Blood clots are more likely to form in the legs during periods of inactivity, such as:

**Bed rest**: Being confined to bed for an extended period after surgery, a heart attack, leg fracture, trauma or any serious illness makes far more vulnerable to blood clots. When the lower extremities are horizontal for long periods of time, the flow of venous blood slows and blood pools in the legs. (Swanson et al., 2008).

**Long journeys**: Sitting in a cramped position during lengthy plane or car trips slows blood flow, which contributes to the formation of clots in legs. (Swanson et al., 2008).

#### **2.3.1.5.3** Surgery

Surgery is one of the leading causes of problem blood clots, especially seen after joint replacements of the hip and knee. During the preparation of the bones for the artificial joints, tissue debris may enter the bloodstream and contribute to causing a clot. Simply being immobile during any type of surgery can lead to the formation of clots. The risk increases with the length of time under general anesthesia. For this reason, most people undergoing a type of surgery predisposing them to DVT will receive medication before and after surgery to prevent clot formation. (Swanson et al., 2008).

# 2.3.1.5.4Other risk factors of pulmonary embolism

**Smoking**:For reasons that aren't well-understood, tobacco use predisposes some people to blood clot formation, especially when combined with other risk factors. (Swanson et al., 2008)

**Being overweight**: Excess weight increases the risk of blood clots particularly in women who smoke or have high blood pressure. (Swanson et al., 2008)

Supplemental estrogen: The estrogen in birth control pills and in hormone replacement therapy can increase clotting factors in the blood, especially in smoking or overweight. (Swanson et al., 2008)

**Pregnancy**: The weight of the baby pressing on veins in the pelvis can slow blood return from the legs. Clots are more likely to form when blood slows or pools. (Swanson et al., 2008)

**Age**: Older people are at higher risk of developing clots. Factors include: Valve malformation: Tiny valves located every few inches within the larger vein keep the blood moving in the right direction. However, these valves tend to degrade with age. When there don't work properly' blood pools and sometimes form clots. (Swanson et al., 2008)

**Dehydration**: Older people are at higher risk of dehydration, which may thicken the blood and make clots more likely. (Swanson et al., 2008)

**Medical problems:** Older people are also more likely to have medical problems that expose them to independent risk factors for clot-such as joint replacement surgery, cancer or heart disease. It is rare for children to develop DVT or VTE. (Swanson et al., 2008).

# 2.3.1.6Complications of Pulmonary embolism

Pulmonary embolism can be life-threatening. About one-third of people with undiagnosed and untreated pulmonary embolism don't survive. When the condition is diagnosed and treated promptly, however, that number drops dramatically. Pulmonary embolism can also lead to pulmonary hypertension, a condition in which the blood pressure in the lungs and in the right side of the heart is too high. When obstructions in the arteries inside the lungs, the heart must work harder to push blood through those vessels. This increases the blood pressure within these

vessels and the right side of the heart, which can weaken the heart. In rare cases, small emboli occur frequently and develop over time, resulting in chronic pulmonary hypertension, also known as chronic thromboembolic pulmonary hypertension (CTEPH). (Swanson et al., 2008).

# **2.4**Computed tomography (CT scan)

#### 2.4.1Helical Scanning

in the late 1980s, helical CT has revolutionized clinical imaging. Also called spiral (or continuous acquisition) scanning, helical scanning brought dramatic improvement in scanning speed by eliminating the interscan delay. There are three basic ingredients that define a helical scan process: a continually rotating x-ray tube, constant x-ray output, and uninterrupted table movement. Increasing the scan speed results in improved image resolution owing to the ability to obtain images with improved iodinated contrast concentration, decreased respiratory and cardiac motion artifact, and superior multiplanar and three dimensional (3-D) reformation capabilities. In addition to improved diagnostic accuracy, the speed associated with helical scanning is also beneficial in regards to patient comfort and department productivity.(Lois E.2011).

Helical scanners were constructed with a single row of detectors. Since then, MDCT systems with as many as 64 detector rows have been introduced. By further improving scan speed, these systems have made clinical applications, such as CT angiography (CTA) .(Lois E.2011).

# 2.4.2CT Machine Equipment

The rotating part of the system consists of the X-ray tube, High voltage generator, Detectors and Data acquisition system (DAS). The stationary part consists of the front-end memory and computer and the first stage high voltage component. The X-ray tube and detectors rotate continuously during data collection because the cable wraparound

problem has been eliminated by slip ring technology. Because large amounts of projection data are collected very quickly, increased storage is needed. This accommodated by the front-end memory fast solid state, and magnetic disk storage. In spiral CT scanners, the X-ray tube is energized for longer periods of time compared with conventional CT tubes. This character requires X- ray tubes that are physically larger than conventional X-ray tubes and have heat unit's capacities greater than 3 million heat units (MHU) and anode cooling rates of (1 MHU) per minute. X-ray detectors for single slice spiral CT scanning are one dimensional (ID) array and should be solid state because their overall efficiency is greater than gas ionization detectors.

The high voltage generator for spiral CT scanner is a high frequency generator with high power output. The high voltage generator is mounted on the rotating frame of the CT gantry and positioned close to the X-ray tube. X- ray tubes operate are high voltages (about 80 to 140 kVp) to produce X- rays with the intensity needed for CT scanning. At such high voltages, arcing between the brushes and rings of the gantry may occur during scanning. To solve this problem, one approach (high voltage SR) is to divide the power supply into a first stage on the stationary part of the scanner, where the voltage is increased to an intermediate level and a second stage on the rotating part of the scanner, where the voltage is increased to the requirement high voltages needed for X-ray production and finally rectified to direct current potential. Another approach passes a low voltage across the brushes to the slip rings, the high voltage generator and then the X-ray tube. In both designs, only a low to intermediate voltage is applied to the brush / slip ring interface, thus decreasing the chances of arcing.

# 2.4.3Slip Ring Technology

One of major technical factors that contribute to the success of spiral CT scanning is slip ring technology. The purpose of the slip ring is to allow the X-ray tube and detectors (in third generation CT systems),to rotate continuously so that a volume of slices, rather than one slice, can be scanned very quickly in a single breath hold. The slip rings also eliminate the long, high tension cables to the X-ray tube used in conventional start stop CT scanners. As the X-ray tube rotates continuously, the patient also moves continuously through the aperture of the gantry so that data can be acquired from a volume of tissue. Regular CT scans take X-rays from many different angles and then combine them to form images showing 2-D "slices" of the internal structures. In a spiral (helical) CT scan, the scanner rotates around the body in a spiral like the stripe on a candy cane to create 3-D images. This type of CT can detect abnormalities within the arteries in the lungs with much greater precision, and it's also much faster than are conventional CT scans. In some cases, contrast material is given intravenously during the CT scan to outline the pulmonary arteries. (Swanson et al., 2008).

Computed tomography is now able to enhance radiology in general. It is helpful in the investigation of non-malignant diseases, abnormalities of lung parenchyma for example. Pulmonary angiography fine slices of the lung are scanned by spiral CT so that filling defects in the pulmonary arteries is visualized. (A.V Hoff brand et al, 2001) It can show good sensitivity and specificity for medium-sized pulmonary emboli. They do not exclude pulmonary emboli in small arteries. (Kumar and Clark, 1998).

# 2.4.4Computed Tomography of the Pulmonary Arteries (CTPA)

CTPA has become more widely used as a diagnostic tool in PE because of the development of the computed tomography CT technique. A CTPA can be used both to exclude and to diagnose PE (Musset, D. et al 2002) and it can confirm alternative diagnoses with high sensitivity and specificity (Rathbun, S. W. et al 2000). Indirect signs of PE, such as atelectasis, (i.e. pleural-based densities), dilatations of pulmonary arteries and pleural effusions can also be visualized on (CTPA). (Coche, E E. et al 1998).

(CTPA) is available at almost all hospitals and is considered as the firstline investigation in many recommendations. (Torbicki, A et al 2014).(CTPA) is fast, and relatively inexpensive, and requires less contrast media. Newer CT techniques with multi-detector row CT (MDCT) and multiple slices CT (MSCT) are thought to increase the diagnostic safety of sub segmental PE. (CTPA) can safely diagnose PE down to the sub segmental part of the pulmonary arteries, although the investigation is less specific more distally. Recent studies show that (MDCT) and conventional single-detector row CT have similar accuracy in detecting sub segmental PE (Nijkeuter, et al 2008).

#### 2.5Other Tests and Diagnosis Methods of Pulmonary Embolism

Pulmonary embolism can be difficult to diagnose, especially in people who have underlying heart or lung disease. For that reason, ordering a series of tests to help find the cause of the symptoms. (Marten soderberg, 2008).

The clinical presentation combined with a history and physical examination can help in the diagnostic work-up in patients with suspected PE. Arterial blood gases, ECG, and CXR can help identify alternative diagnoses, and echocardiography can help assess the severity of PE. These investigations are nonspecific and patients "with PE can have

pathological findings, although normal findings do not exclude PE (Goldhaber, A. Z. & Elliott, C.G. (2003).

#### 2.5.1Blood Tests

The blood test is order for the clot-dissolving substance D dimer in the blood. High levels may suggest an increased likelihood of blood clots, although D dimer levels may be elevated by many other factors, including recent surgery. In addition, blood tests may be done to determine an inherited clotting disorder. (Swanson et al., 2008)

# 2.5.2Echocardiography (ECG)

The ECG can show sinus tachycardia, atrial fibrillation and signs of right ventricular strain with S1Q3T3-syndrome, right bundle-branch block, and T-wave inversion in lead V1-3. These signs are nonspecific and the most common finding in PE is normal ECG, although patients with massive PE often show dramatic changes in ECG. (Grifoni, S. et al. 2000).

This is performed to determine whether there is right heart strain which occurs only in relatively sever cases. In small/medium pulmonary emboli is usually normal, in multiple recurrent pulmonary emboli can be normal or show signs of pulmonary hypertension. (Kumar and Clark, 1998)

The echocardiography is well studied in PE, (Goldhaber, S.Z. 2002; Riberio, A., 1997) Echocardiography is of value at the time of diagnosis and in the follow-up of patients with PE. Signs of right –sided systolic dysfunction are associated with increased mortality and echocardiography is mandatory in the work-up of patients with signs of massive PE requiring thrombolytic therapy, More than 50% of patients with PE have a normal echocardiography pattern.(Kearon C et al 2008, Toosi, M. S. et al 2008).

#### 2.5.3Chest X-ray

This noninvasive test shows images of the heart and lungs on film. Although X-rays can't diagnose pulmonary embolism and may even appear normal when pulmonary embolism exists, they can rule out conditions that mimic the disease. The chest x-ray is often abnormal in PE(Investigators, 1990, Stein, P.D. & henary 1997) and a variety of findings has been described, such as atelectasis, parenchymal consolidations (infiltrates), pulmonary oedema elevated hemidiaphragm, focal oligemia (an area with diminished blood supply; westermark's sign), enlarged right pulmonary artery (palla's sign), pleuric fluid, and a wedged- shaped density indicating an early lung infarction (Hampton's hump) etc. The main value of CXR is to find an alternative diagnosis. A current chest X-ray is required at the time lung scintigraphy is performed, mainly as an aid in categorizing in the lung scintigram, and to exclude other abnormalities, such as cardiac failure, chest infection or pulmonary hypertension, which may a count for the patient's symptoms. (Sharp et al; 1989)

#### 2.5.4Ultrasound

A noninvasive "sonar" test known as duplex ultrasonography (sometimes called duplex scan, or compression ultrasonography) uses high-frequency sound waves to check for blood clots in the thigh veins. In this test, we use a wand-shaped device called a transducer to direct the sound waves to the veins being tested. These waves are then reflected back to the transducer and translated into a moving image by a computer. The absence of the presence of clots reduces the likelihood of DVT. If the upper thigh vessels are clear, the ultrasonography will also scan the veins

behind the knee looking for residual clots. If clots are present, treatment likely started immediately. (Swanson et al., 2008)

#### 2.5.5Pulmonary angiogram

This test provides a clear picture of the blood flow in the arteries of the lungs. It's the most accurate way to diagnose pulmonary embolism, but because it requires a high degree of skill to administer and has potentially serious risks, it's usually performed when other tests fail to provide a definitive diagnosis. In a pulmonary angiogram, a flexible tube (catheter) is inserted into a large vein usually in the groin and threaded through into the heart and on into the pulmonary arteries. A special dye is then injected into the catheter, and X-rays are taken as the dye travels along the arteries in the lungs. One risk of this procedure is a temporary change in the heart rhythm. In addition, the dye may cause kidney damage in people with decreased kidney function. (Swanson et al., 2008).PA is considered the most specific test for PE and is used as the reference method in many studies. Although the performances cannot be calculated, studies have shown that it is safe to withhold anticoagulants in patients with a normal (PA) (Nilsson, T. et al 1998). PA is an invasive investigation- contrast medium is injected (IV) directly into the pulmonary arteries through a catheter introduced in the femoral vein. (PA) has several advantages because it can be used to visualize the emboli, directly and because it provides hemodynamic data and opportunities for direct treatment through the insertion of thrombotytic agents and vena caval filters. PA also has several disadvantages: for example, the high cost, limited availability. Invasive technique with risk for bleeding and arrhythmias. and complications related to the contrast media, the use of (PA) has diminished during the past decade. in favor of (CTPA), mainly because of the disadvantages, but there are still clinical

conditions when PA should be used for example in patients with contraindications for or, inconclusive (CTPA). (Stein, P. D. et al, 1992).

# **2.5.6Magnetic Resonance Imaging (MRI scan)**

MRI scans use radio waves and a powerful magnetic field to produce detailed images of internal structures. Because MRI is expensive, it's usually reserved for pregnant women (to avoid radiation to the fetus) and people whose kidneys may be harmed by dyes used in other tests. This study can be performed with or without respiratory and (ECG)-gated studies. The mediastinum and hilar structure are well shown, more peripheral lesion is less clearly identified. Gadolinium-enhanced MRI is a relatively new expensive but accurate technique. (Glenda J. Bryan, 1987)

# 2.6Treatments and drugs of pulmonary embolism

#### 2.6.1 Medications

Blood thinners (anticoagulants): These drugs prevent new clots from forming while the body works to break up the clots. Heparin is a frequently used anticoagulant that can be given through the vein or injected under the skin. It acts quickly and is often overlapped for several days with an oral anticoagulant, such as warfarin, until it becomes effective, which can take days. A newer class of anticoagulants has been tested and approved for treatment of venous thromboembolism, including pulmonary embolism. These medications have the advantage of being given by mouth, without the need for overlap with heparin. Also, they work quickly and have fewer interactions with other medications. All blood thinners have side effects, with bleeding being the most common. (Swanson et al., 2008).

Clot dissolvers (thrombolytics): While clots usually dissolve on their own, there are medications given through the vein that can dissolve clots quickly. Because these clot-busting drugs can cause sudden and severe

bleeding, they usually are reserved for life-threatening situations. (Swanson et al., 2008).

#### 2.6.2Surgical and other procedures

Clot removal: can be removing large, life-threatening clot in the lung, via a thin, flexible tube (catheter) threaded through the blood vessels. (Swanson et al., 2008).

Vein filter: A catheter can also be used to position a filter into the body's main vein called the inferior vena cava that leads from the legs to the right side of the heart. This filter can help keep clots from being carried into the lungs. This procedure is typically reserved for people who can't take anticoagulant drugs or when anticoagulant drugs don't work well enough or fast enough. The catheter with the filter in the tip is usually inserted in a vein in the neck, and then into the vena cava. (Swanson et al., 2008).

#### 2.7 Previous studies:

Study done by (Bešlić, et al 2005), which include (Multislice computed tomography of pulmonary embolism). The purpose of this study is to analyze the contribution of multislice computed tomography (MSCT) as a diagnostic method in the diagnosis of pulmonary embolism (PE) and spectrum of findings in our material. During the period of one and a half year, they found PE in 25 patients (15 males and 10 females). The average age of the patients was 54.4 years (25 - 74). The examination was performed by »Somatom Volume Zoom« Siemens CT machine with four row detectors, with retrospective ECG gating, collimation 4 x 2.5 mm and reconstructed section with 0.8 mm. Contrast medium (130 ml) and 10 ml of saline was applied, administered with a flow rate of 3.5 ml/s and with time delay of 22 seconds. During the examination, they found embolism of the main branches of pulmonary artery in 14(56%) patients, at the right branch in 10 (40%), at the left one in 4 (16%), and bilateral pulmonary embolism in 11 (44%) patients. Subsegmental pulmonary emboli were noticed in 8 (32%) patients. contrast enhanced consolidation of pulmonary parenchyma in 10 (40%), rag zones of ground glass attenuation in 15 (60%), haemorrhage in 21 (84%), striped and reticular pulmonary drawing in 11(44%), and mosaic olighemy in 3 (12%)cases. They concluded that MSCT is an excellent non-invasive method for visualization of thrombus in the pulmonary artery. In their study, they have more often found embolism of the right branch of pulmonary artery, and pleural effusion, infarct contrast enhanced consolidation of pulmonary parenchyma, ground glass attenuation zone.

Study done by (Barry Donald Hutchinson 2015), which about (Overdiagnosis of Pulmonary Embolism by Pulmonary CT Angiography) The purpose of this study is to evaluate the rate of overdiagnosis of pulmonary embolism (PE) by pulmonary CT angiography (CTA) in a

tertiary-care university hospital. A total of 937 pulmonary CTA studies were performed over the study period. PE was diagnosed in the initial report in 174 of these cases (18.6%). There was discordance between the chest radiologists and the original radiologist in 45 of 174 (25.9%) cases. Discordance occurred more often where the original reported PE was solitary (46.2% of reported solitary PEs were considered negative on retrospective review) and located in a segmental or subsegmental pulmonary artery (26.8% of segmental and 59.4% of subsegmental PE diagnoses were considered negative on retrospective review). The conclusion is In routine clinical practice, PEs diagnosed by pulmonary CTA are frequently over diagnosed, when compared with the consensus opinion of a panel of expert chest radiologists. Improvements in the quality of pulmonary CTA examination and increased familiarity with potential diagnostic pitfalls in pulmonary CTA are recommended to minimize misdiagnosis of PE.

Study done by (Schoepf and Costello 2004) which include ( CT angiography for diagnosis of pulmonary embolism ). In daily clinical routine, computed tomography (CT) has practically become the first-line modality for imaging of pulmonary circulation in patients suspected of having pulmonary embolism (PE). However, limitations regarding accurate diagnosis of small peripheral emboli have so far prevented unanimous acceptance of CT as the reference standard for imaging of PE. The development of multi– detector row CT has led to improved visualization of peripheral pulmonary arteries and detection of small emboli. The finding of a small isolated clot at pulmonary CT angiography, however, may be increasingly difficult to correlate with results of other imaging modalities, and the clinical importance of such findings is uncertain. Therefore, the most realistic scenario to measure efficacy of pulmonary CT angiography when PE is suspected may be

assessment of patient outcome. Meanwhile, the high negative predictive value of a normal pulmonary CT angiographic study and its association with beneficial patient outcome has been demonstrated. While the introduction of multi-detector row technology has improved CT diagnosis of PE, it has also challenged its users to develop strategies for optimized contrast material delivery, reduction of radiation dose, and management of large-volume data sets created at those examinations. They conclude CT has become the first-line modality for imaging in patients suspected of having PE.

Study done by (Kim et al 2010) in detection of Pulmonary Embolism in the Postoperative Orthopedic Patient Using Spiral CT Scans to compare the clinical presentations of a suspected versus a documented PE/DVT and to determine the actual incidence of PE/DVT in the postoperative orthopedic patient in whom CT was ordered. All 695 patients at our institution who had a postoperative spiral CT to rule out PE/DVT from March 2004 to February 2006 were evaluated and information regarding their surgical procedure, risk factors, presenting symptoms, location of PE/DVT, and anticoagulation were assessed. The most common presenting symptoms were tachycardia (56%, 393/695), low oxygen saturation (48%,336/695),and shortness of breath (19.6%, 136/695). Symptoms significantly associated with DVT were syncope and chest pain.

Study done by (Storto et al 2005), studied the Incidental Detection of Pulmonary Emboli on Routine MDCT of the Chest his aim to assess the prevalence of pulmonary embolism incidentally detected on routine MDCT of the chest and to determine whether the use of wide window settings can improve detection of unsuspected pulmonary embolism, they studied 589 patients with CT angiograms obtained for suspected pulmonary embolism or thoracic aorta disease were not considered.

Image evaluation was performed' on a. dedicated workstation during two separate review sessions using different window settings: standard, with a width of 400 H and level of 40 H; and wide, with a width of 600 H and level of 100-150 H. The quality of vascular enhancement was recorded. Eight examinations were excluded because of poor quality. Unsuspected pulmonary embolism was present hi 20 (3.4%) of 581 patients with an inpatient prevalence of 4.0% (19/474) and outpatient prevalence of 0.9% (1/107). Fourteen patients (70.0%) with unsuspected pulmonary embolism had cancer. The proximal extent of pulmonary embolism involved the main pulmonary artery in five patients, a lobar artery in five, and a segmental artery in 10; isolated subsegmental thrombi were never found. The use of wide window settings allowed detection of pulmonary embolism in two more patients than did the standard settings.

Study done by (Salah Eldein Hassan Aloub 2016) which about Characterization of Heart and Pulmonary Vessels in Patients with Pulmonary Hypertension and Embolism using Computed Tomography his aim to characterize the heart and pulmonary vessels in patients with pulmonary embolism PE and pulmonary arterial hypertension PAH using Computed Tomography scan CT. Prospective study of 150 Sudanese patients with clinically suspected PE, PAH, and CTEPH were enrolled. Included random samples of 150 patients in different genders and ages. (92) patient were female while the (58) were males and their ages are ranged from (21-95) years old. his sample demonstrate that the patients affected with PH after affected with PE were 25 patient with the most affected ages were between 51-60 years constituting 11(44.0%), where those who were affected with PE only were 18(32.7%) for the same age group. his study include the clinical and radiological findings the main clinical finding was chest pain in PE patients, tachycardia and syncope were present with least frequencies, consolidation scored the high

frequency in PE patients. Pulmonary trunk diameter in PH patients and CTEPH was found to be larger than patients with PE such enlargement of pulmonary trunk is a common finding in patient with chronic thromboembolic pulmonary hypertension.

Study done by (Edwards, P.D, et al, 1998) purpose of this study was to determine the normal range of the main pulmonary artery diameter (MPAD) by computed tomography (CT) in persons with normal pulmonary artery pressure, and then to evaluate the relationship of the diameter with age, gender, and body surface area (BSA). Between October 2005 and June 2007, among patients who had previously undergone a contrast enhanced thorax CT scan, 112 persons (47 females, 65 males). All patients had normal mean pulmonary artery pressure. The widest diameter perpendicular to the long axis of the main pulmonary artery was measured. The mean (MPAD) was  $2.7 \pm 2.8$  cm they conclude The present study demonstrated that in individuals with normal pulmonary artery pressure, the upper limit of the (MPAD) is 3.2 cm.

# CHAPTER THREE Materials and Methods

# **Chapter Three**

# **Materials and Methods**

# 3.1Materials

# 3.1.1Study design

This is prospective study, included samples of 54 patients in different genders and ages.

# 3.1.2Instrumentation

Multislice CT scanner GE machine : (64 multi-slice detector) at radiology department in Mubarak Alkabir Hospital ( Kuwait city ).



**Figure 3.1**CT GE Evolution 64 Slice

#### 3.1.3Patient's Population

This study was done in Mubarak Alkabir Hospital (Kuwait city); in the period between (2016 - 2017). Study of54adult patients with clinically suspected PE. All patients were examined by using multislice CT scanner. The study included patients in different genders and ages, (23) patients were females while the (31) were males and their ages were ranged from (23 - 86) years old.

#### 3.2Methods

#### 3.2.1Data collection

Data collected from radiologist reports with special designed sheet from findings which appear in different CT cuts.

#### 3.2.2Statistical analysis

The data obtained were analyzed statistically by computing descriptive statistics like mean  $\pm$  SD values and percentages, with an independent T-test, ANOVA test, and by correlation analysis using an IBM SPSS Statistics software package.

# 3.2.3Technique

# 3.2.3.1Patient preparation

A successful CTPA examination depends on careful preparation of the patient before the examination. Such preparation requires that both the technologist and radiologist work together to obtain the appropriate and correct information from the patient and to ensure that the patient understands the procedure. Preceding the CTPA, a patient history was obtained to identify patients with histories of iodine allergy, renal dysfunction, cardiac disease and asthma. Steroid Pre-medication was administrated to those patients with a history of iodine allergy or previous reaction to iodinated contrast agents. Patients with a history of renal dysfunction were further evaluated with creatinine level and blood urea

nitrogen level assessed before the procedure. In the other hand we should consider the size of the needle and the site of injection. various size intravenous angio-catheters such as 18 or 20 gauge, and the injection better to be at the elbow.

# **3.2.3.2** protocol

CT angiography (CTA) is defined as "any CT image of a blood vessel that has been opacified by a contrast medium". During spiral data acquisition, the entire area of interest can be scanned during the injection of contrast. Images can be captured when vessels are fully opacified to demonstrate arterial phase enhancement through the acquisition of data sets (arterial).

1-Volume of contrast medium 70-90 ml followed by saline chase 20-30 ml.

2-rate of injection 4-5 mls.

3-delay: in this study it was better to use (smart-prep) because there was variety in the patients heart rate, a smart-prep is positioned over the pulmonary artery at the level of the carina. After starting the injection the scan is triggered when you can see the contrast at the superior vena cava. or we can use bolus tracking using software supplied with most multi detector scanner.

- 4-Detector width- reconstruction (mm)-(0.625-1.25).
- 5- Scan direction and extension-caudocranial direction helps reduce artifact from contrast in the superior vena cava, less important with faster multislice scanner, scan from hemi diaphragm to the lung apex. Imaging review and post-processing, imaging should be reviewed at three setting:
  - Mediastinal window (window width-window level) (400-40 HU)
  - Pulmonary embolism -specific window (700-100 HU)
  - Lung window (1500-600 HU)

Multiplanar reformatted images through the longitudinal axis of a vessel can be helpful to overcome difficulties encountered on axial section of obliquely oriented arteries, adding confidence in diagnosis or" exclusion of thrombus.

# 3.2.4Post Processing Technique

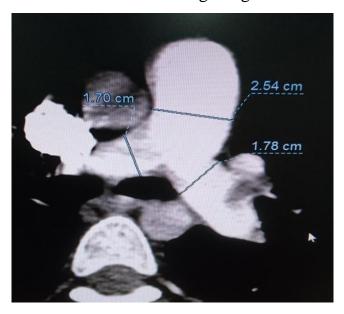
#### 3.2.4.1 Visualization Tools:

# 3.2.4.1.1Multiplanar reconstruction (MPR)

MPR was the first visualization tool used in CTPA. The reformat was done at the workstation as sagittal, coronal, left and right oblique including curved planes for better diagnose and interpretation of the embolism.

# 3.2.4.1.2 Interpretation

All the CT images were diagnosed by two professional radiologists and the measurements of the variables were done by one technologist. The measurements were done as the following image:



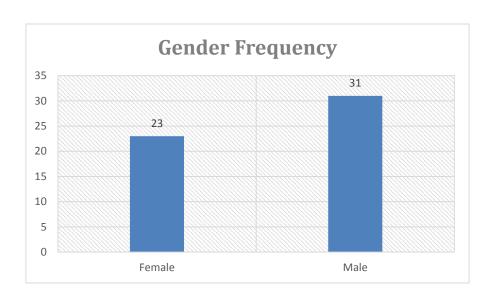
**Figure 3.2**Showed the method of measurement of main pulmonary diameters

# CHAPTER FOUR Results

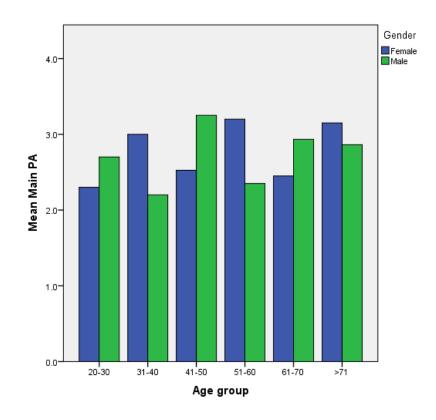
# CHAPTER FOUR Results

Table 4.1Frequency distribution of the gender

Gender	Frequency	Percent
Female	23	42.6%
Male	31	57.4%
Total	54	100.0%



**Figure 4.1**Frequency distribution of the gender



**Figure 4.2**Show the correlation between the age group with mean main Pulmonary artery according to the gender

Table 4.2Frequency distribution of the age

Age group	Frequency	Percent
20-30	7	13.0
31-40	7	13.0
41-50 51-60	12 11	22.2 20.4
61-70	5	9.3
>71	12	22.2
Total	54	100.0

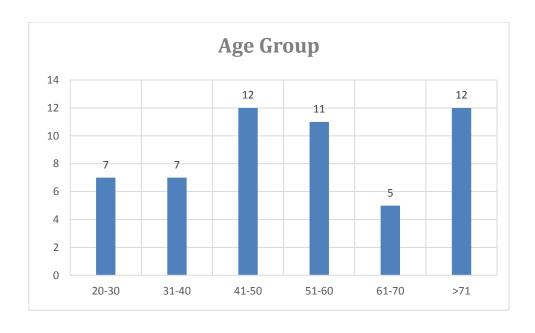


Figure 4.3Frequency distribution of the age

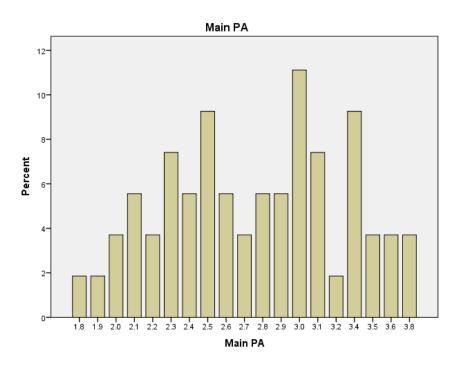


Figure 4.4Frequency distribution of the main pulmonary artery

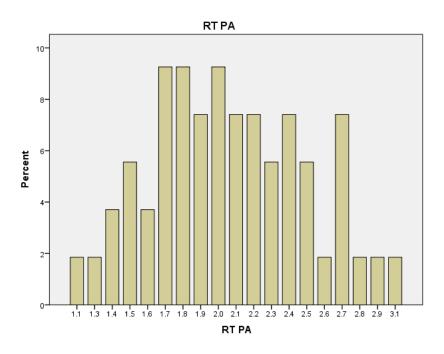


Figure 4.5Frequency distribution of the right main pulmonary artery

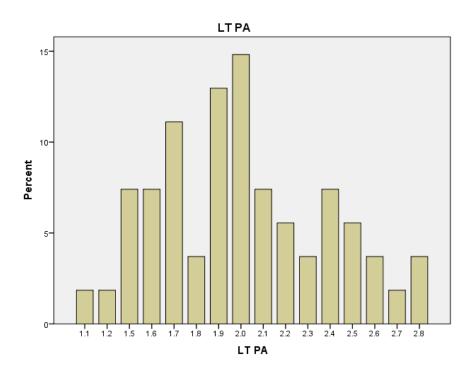


Figure 4.6Frequency distribution of the left main pulmonary artery

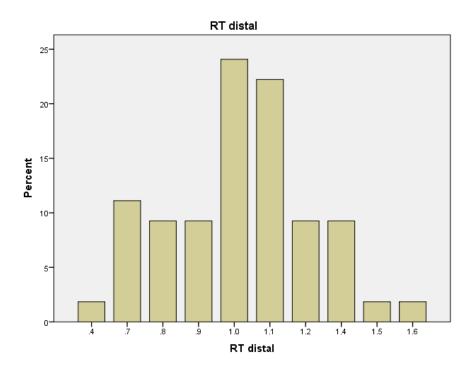


Figure 4.7Frequency distribution of the right distal pulmonary artery

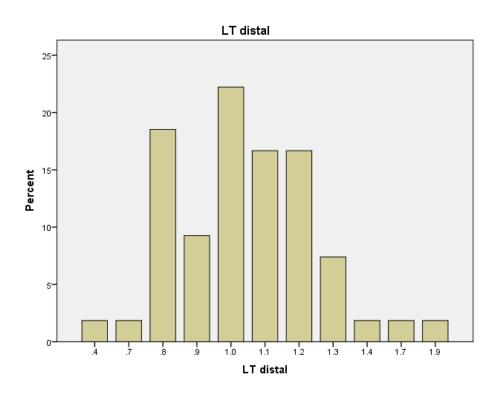


Figure 4.8Frequency distribution of the left distal pulmonary artery

**Table 4.3**Show statistical parameters of all patients as mean  $\pm$  SD

	Age	Main	RT PA	LT PA	RT	LT
		PA			distal	distal
					PA	PA
Mean	52.20	2.778	2.063	2.000	1.026	1.046
Std.						
Deviation	17.081	.5171	.4431	.3885	.2316	.2401
Minimum	23	1.8	1.1	1.1	.4	.4
Maximum	86	3.8	3.1	2.8	1.6	1.9

**Table 4.4**IndependentSamples Test for means between and within measured variables in patients with PE

# ANOVA

		Sum of Squares	Mean Square	Sig.
	Between Groups	2.384	.477	.105
Main PA	Within Groups	11.790	.246	
	Total	14.173		
	Between Groups	3.816	.763	.000
RT PA	Within Groups	6.590	.137	
	Total	10.406		
	Between Groups	1.943	.389	.017
LT PA	Within Groups	6.057	.126	
	Total	8.000		
	Between Groups	.409	.082	.175
RT distal	Within Groups	2.435	.051	
	Total	2.844		
	Between Groups	.312	.062	.377
LT distal	Within Groups	2.742	.057	
	Total	3.054		

**Table 4.5**The frequency of the complains findings in patients with pulmonary embolism.

Complain	Positive/Negative	Patients with (PE)
Complain	1 Oshtive/ Negative	N=54
Tachycardia	+Ve	38
		70%
	-Ve	16
		29.6%
Shortness of	+Ve	29
breath	+ve	53.7%
	–Ve	25
	-ve	46.2%
Chest pain	+Ve	25
		46.2%
	-Ve	29
		53.7%
L.L swelling	+Ve	9
		16.6%
	-Ve	45
		83.3%
Cough	+Ve	8
		14.8%
	-Ve	46
		85.1%

**Table 4.6**The frequency of pleural-parenchymal findings in patients with pulmonary embolism.

PleuralParenchymal Findings	Condition	Patients with(PE)N=54
Pleural effusion	+Ve	24
		44.4%
	-Ve	30
		55.5%
Consolidation	+Ve	17
		31.4%
	-Ve	37
		68.5%
Ground	+Ve	5
Glass Opacity		9.2%
	-Ve	49
		90.7%
Atelectasis	+Ve	6
		11.1%
	-Ve	48
		88.8%

# CHAPTER FIVE Discussion, Conclusion and Recommendations

#### **Chapter Five**

#### **Discussion, Conclusion and Recommendations**

#### 5.1Discussion

in this study I found the frequency of males was (31) with percentage (57.4%) and the females(23) with percentage (42.6%) as shown in table (4.1) and figure (4.1). this mean the percentage of PE in males more than the females this was similar to the findings of study done by (Bešlić, et al 2005).

About the correlation between the age group with mean main pulmonary artery according to the gender as shown in figure (4.2), resulted that the age group (20-30) years showed the males was higher in mean than the females and the age ranged from (31-40) years showed the mean of females was higher than males when the age group (41-50) years showed the mean of males higher than females, in the age ranged between (51-60) years represented the mean of the females higher than males also the age group (61-70) years the mean of the males was higher than females when the last age group which was bigger than (71) years the mean of the females was higher than males. From the previous figure I concluded the age group ranged from (41-50) years and bigger than (71) years were higher frequency with 12(22.2%) while lower frequency was (61-70) years with 5(9.3%) as shown in table (4.2) and figure (4.2). Several published studies provide different data. (Bešlić, et al 2005) and (Salah Eldein Hassan Aloub 2016). This discrepancy may be due to the small size of the data.

Figure (4.4) showed frequency distribution of main pulmonary artery the frequency (3.0) showed the highest value when (3.2) represented the lowest frequency.

Figure (4.5) showed the frequency distribution of the right main pulmonary artery were the values (1.7), (1.8) and (2.0) scored the highest

frequencies when (1.1), (1.3), (2.6), (2.8), (2.9) and (3.1) showed the lowest frequencies.

Figure (4.6) the frequency distribution of left main pulmonary artery were the value (2.0) showed the highest frequency when (1.1), (1.2) and (2.7) represented the lowest frequencies.

Figure (4.7) represented the frequency distribution of the right distal pulmonary artery were the value (1.0) scored the highest frequency when the values (0.4), (1.5), (1.6) were the lowest frequencies.

Figure (4.8) showed the frequency distribution of the left distal pulmonary artery were the value (1.0) scored the highest frequency while the values (0.4), (0.7), (1.4), (1.7) and (1.9) represented the lowest frequencies.

The statistical parameters for all patients have been analyzed as (mean  $\pm$  SD) for age the (mean  $\pm$  SD) was (52.20  $\pm$  17), for the main pulmonary artery was (2.77  $\pm$  0.5)which the same finding of (Edwards, P.D, et al, 1998). For right main pulmonary artery was (2.06  $\pm$ 0.4), for left main pulmonary artery was (2.00  $\pm$ 0.3), for right distal pulmonary artery was (1.02  $\pm$ 0.2), for left distal pulmonary artery was (1.04  $\pm$ 0.2), as shown in table (4.3).

The significant difference between the variables which have been evaluated in patients with PE, right pulmonary artery, left pulmonary artery measurements diameters were changed significantly in patients with PE at (p=0.000), (p=0.017) this support previous study done by (Salah Eldein Hassan Aloub 2016).while there were no significant difference in the main pulmonary artery, right distal pulmonary artery, left distal pulmonary artery measurements diameters at (p=0.105), (p=0.175), (p=0.377) which the similar findings of (Moore, N.R, et al, 1998). this have been demonstrated in table (4.4).

The patients with PE presented with symptoms such as shortness of breath (SOB), atypical chest pain, tachycardia and cough. The main clinical findings was tachycardia 38(70%), shortness of breath (SOB) 29(53%) and chest pain 25(46%) this was similar to study done by (Kim et al 2010).lower limbs swelling and cough were also presented with the least frequencies as shown in table (4.5).

The most common pleural parenchyma findings in patients with PE was pleural effusion constituting 24(44.4%). This method and findings was similar to the study done by Eva Castañer, et al 2009. (EvaCastaner, et al 2009) . In second place was consolidation which scored 17(31.4%) when the ground glass opacity and atelectasis represented the least frequencies this was in table (4.6) .

#### 5.2 Conclusion

This study concluded that the incidence rate of PE in males was more than the females.

The common complain of patients suspected of having PE was tachycardia.

Regarding the study results findings of (CTPA) performed for clinical suspicion of PE lead to consolidation, ground glass opacifications, Atelectasis and pleural effusion were found to be presented in the majority of patients undergoing (CTPA) for the clinical suspicion of PE. The presence of pulmonary vessels measurements were found to have correlation with the presence of PE,(CT) measurements of right main pulmonary artery and left main pulmonary artery dimensions shown a strong correlation with the presence of PE, while there were no correlation with the main pulmonary trunk, right and left distal pulmonary artery measurements.

#### **5.3Recommendations**

- Clinical assessment should be made for patient suspected of having PE before the CT examination.
- Other investigations must be done before CTPA, as CXR, ECG, and echocardiography.
- Other quantification methods must be used to diagnose PE including blood gas values and lung attenuation.
- More care for patients complaining of tachycardia because it may represent as a marker of severe PE.
- Future research is recommended for measuring the pulmonary vessels in patients with PE associating another modalities with CT.

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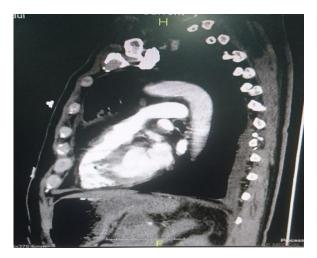
# **Appendices**

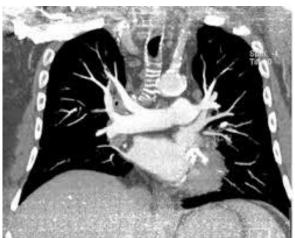
# Appendices(i)

### Data sheet

No	Patient information				Pulmonary Arteries					<b>6</b> 7
	Age	Gender		tory	pulmonary trunk	Rt mainpulmo nary artery	Lt mainpulmo nary artery	Rt Distalpulm onary artery	Lt Distalpulmo nary artery	CT Diagnostic
1										
2										
3										
4										
5										
6										
7										
8										
9										
10										
11										
12										

# Appendices(ii)





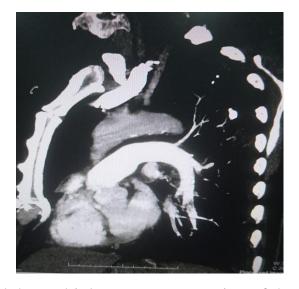


Figure 3.3 showed the multiplanner reconstruction of the pulmonary artery



Figure 3.4 maximum intensity projection (MIP)

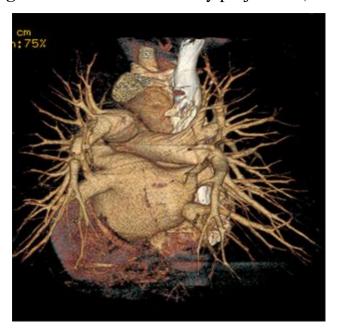


Figure 3.5Three dimensional VR (3D-VR)