Chapter one

1.1INTRODUCTION

The heart is a muscular organ that pumps blood throughout the various parts of the body. Anatomically, the heart lies within the mediastinum and rests on the diaphragm. Cardiac tissue differs from other muscle tissues of the body in its construction and is termed myocardium. The left side of the heart is responsible for the extensive systemic circulation; thus the left muscle wall is about three times as thick as the right side (Bontrager,2001).

The heart itself is divided into four chambers: the right and left atria and the right and left ventricles. Each chamber functions either to receive and/or pump blood. The blood circulation is a closed system, by which un oxygenated blood enters the right atrium from all parts of the body, is re oxygenated in the lungs, and returned to the body by the left ventricle (Bontrager, 2001).

A central issue in the management of patients undergoing to nuclear cardiology is the assessment of the adequacy of Characterization of heart disease using SPECT scintigraphy. Simply following the Noninvasive assessment of myocardial perfusion is important in the diagnosis and risk stratification of patients with known or suspected heart artery disease. A mathematical technique known as the quantitative analysis was used to provide a critical and previously missing element in the computations required to quantitate heart function scintigraphically. This computer-assisted technique allowed for the determination of the blood perfusion, of a labeled sestamibi analog (99mTc-sestamibi) to enter the heart via blood. The critical process provided for by quantitative is the mathematical simulation of a calculation blood perfusion after injection of tracer directly into the blood supply of the heart to estimate the stress and rest myocardial perfusion.The adequacy of SPECT reduce medical problems from the diagnosis of heart disease

in intermediate-risk patients and to improve the effectiveness of treatment and decreasing the risk of premature death.

Myocardial perfusion scan (also referred to as MPI) is a nuclear medicine procedure that illustrates the function of the heart muscle (myocardium).(U.S. National Library of Medicine 2017)

It evaluates many heart conditions, such as coronary artery disease (CAD), hypertrophic cardiomyopathy and heart wall motion abnormalities. The function of the myocardium is also evaluated by calculating the left ventricular ejection fraction (LVEF) of the heart. This scan is done in conjunction with a cardiac stress test.Myocardial perfusion scans (MPS) are obtained after the intravenous (IV) injection of a small dose of radiotracer at rest and following physiological (exercise) or pharmacologic (adenosine, dipyridamole or dobutamine) stress. Originally, thallium was used but it has been superseded by the technetium-labelled synthetic compounds, sestamibi and tetrofosmin, which yield better quality images at a lower radiation dose. Planar techniques, such as conventional scintigraphy, are rarely used. Rather, Single-photon emission computed tomography (SPECT) is more common. With multihued SPECT systems, imaging can often be completed in less than 10 minutes. With SPECT, interior and posterior abnormalities and small areas of infarction can be identified, as well as the occluded blood vessels and the mass of infarcted and viable myocardium(Lee, J. C et al 2013).

1.2 JUSTIFICATION

The adequacy of characterization of heart disease using SPECT scintigraphy reduce medical problems from the diagnosis of coronary disease in intermediate-risk patients and to improve the effectiveness of treatment and decreasing the risk of premature death.

The researcher suggests that the use of SPECT scintigraphyquantitative analysis as an indicator for the characterization of heart disease in intermediate-risk patients is feasible because estimation of rest and stress myocardial perfusion using quantitative analysis from patients information, and the estimation of heart function done by 99mTc sestamibi heart scintigraphy ranging from simple methods such blood perfusion (End-diastolic volume ,End-systolic volume and Ejection fraction) Therefor in this study from the patients information such as age and body mass index and mode of blood perfusion can estimate the stress and rest myocardial perfusion from patients information without need it to stress by 99mTc sestamibi.

1.3 OBJECTIVES

1.3.1 GENERAL OBJECTIVE

The main objective of this study is to Estimation of myocardial perfusion in SPECT Scintigraphy using quantitative analysis.

1.3.2 SPECIFIC OBJECTIVES

- To correlate between the patients information and patients histories.
- To correlate blood mod perfusion of rest with blood mode perfusion of stress.
- To estimate of rest myocardial perfusion from patient information.
- To estimate the stress myocardial perfusion from rest study.

1.4 Overviews of the study:

This study will fall into five chapters with 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 table and finally chapter five which include the discussion, conclusion and recommendation

Chapter two

Theoretical background

2.1 Anatomy of the heart

The heart is a hollow muscular organ that is somewhat pyramid shaped and lies within the pericardium in the mediastinum. It is connected at its base to the great blood vessels but otherwise lies free within the pericardium (Snell,2012).

2.1.1 Location and Size of the heart

For all its might, the heart is relatively small, roughly the same size (but not the same shape) as your closed fist. It is about 12 cm (5 in.) long, 9 cm (3.5 in.) wide at its broadest point, and 6 cm (2.5 in.) thick, with an average mass of 250 g (8 oz) in adult females and 300 g (10 oz) in adult males. The heart rests on the diaphragm, near the midline of the thoracic cavity. It lies in the mediastinum, an anatomical region that extends from the sternum to the vertebral column, the first rib to the diaphragm, and between the lungs (Figure 2.1). About two-thirds of the mass of the heart lies to the left of the body's midline (Figure2.1). (Gerard J et al 2014)

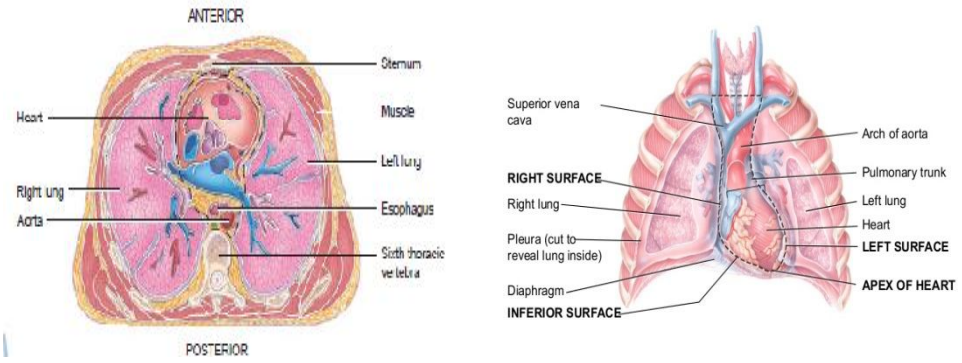


Figure 2.1.: Position of the heart and associated structures in the mediastinum

2.1.2 Pericardium and heart wall.

The membrane that surrounds and protects the heart is the pericardium. It confines the heart to its position in the mediastinum, while allowing sufficient freedom of movement for vigorous and rapid contraction. The pericardium consists of two main parts: (1) the fibrous pericardium and (2) the serous pericardium (Figure 2.2). The superficial fibrous pericardium is composed of tough, inelastic, dense irregular connective tissue. It resembles a bag that rests on and attaches to the diaphragm; its open end is fused to the connective tissues of the blood vessels entering and leaving the heart. The fibrous pericardium prevents overstretching of the heart, provides protection, and anchors the heart in the mediastinum. (Gerard J et al 2014)

The deeper serous pericardium is a thinner, more delicate membrane that forms a double layer around the heart (Figure 2.2). The outer parietal layer of the serous pericardium is fused to the fibrous pericardium. The inner visceral layer of the serous pericardium, also called the epicardium, is one of the layers of the heart wall and adheres tightly to the surface of the heart. Between the parietal and visceral layers of the serous pericardium is a thin film of lubricating serous fluid. This slippery secretion of the pericardial cells, known as pericardial fluid, reduces friction between the layers of the serous pericardium as the heart moves. The space that contains the few milliliters of pericardial fluid is called the pericardial cavity. (Gerard J.et al 2014)

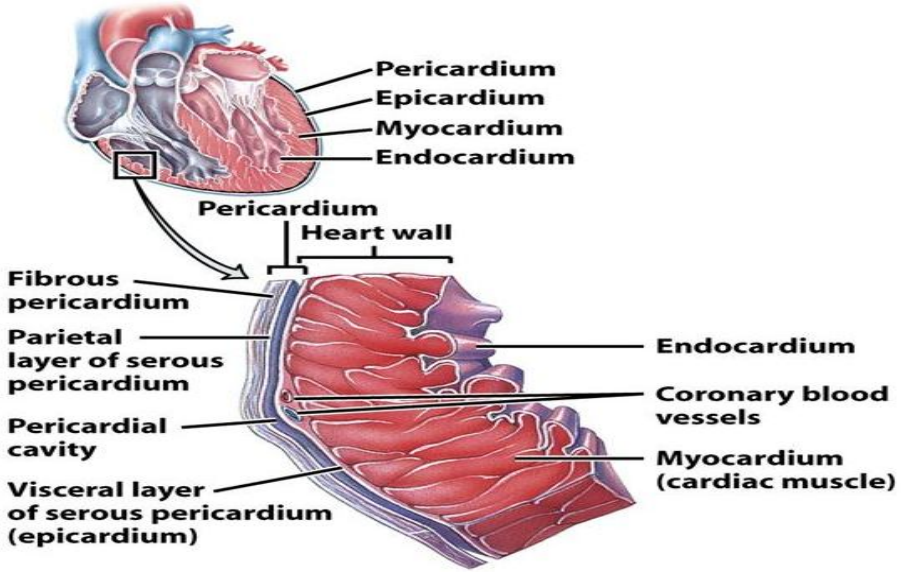


Figure 2.2:Portion of pericardium and right ventricular heart wall showing the divisions of the pericardium and layers of the heart wall

The wall of the heart consists of three layers (Figure 2.2): the epicardium (external layer), the myocardium (middle layer), and the endocardium (inner layer). As noted earlier, the outermost epicardium, the thin, transparent outer layer of the heart wall, is also called the visceral layer of the serous pericardium. It is composed of mesothelium and delicate connective tissue that imparts a smooth, slippery texture to the outermost surface of the heart. The middle myocardium, which is cardiac muscle tissue, makes up about 95% of the heart and is responsible for its pumping action. Although it is striated like skeletal muscle, cardiac muscle is involuntary like smooth muscle. The cardiac muscle fibers swirl diagonally around the heart in bundles (Figure 2.3). The innermost endocardium (endo- _ within) is a thin layer of endothelium overlying a thin layer of connective tissue. It provides a smooth lining for the chambers of the heart and covers the valves of the heart. The endocardium is continuous with the endothelial lining of the large blood vessels attached to the heart, and it minimizes surface friction as blood passes through the heart and blood vessels. (Gerard J.et al 2014)

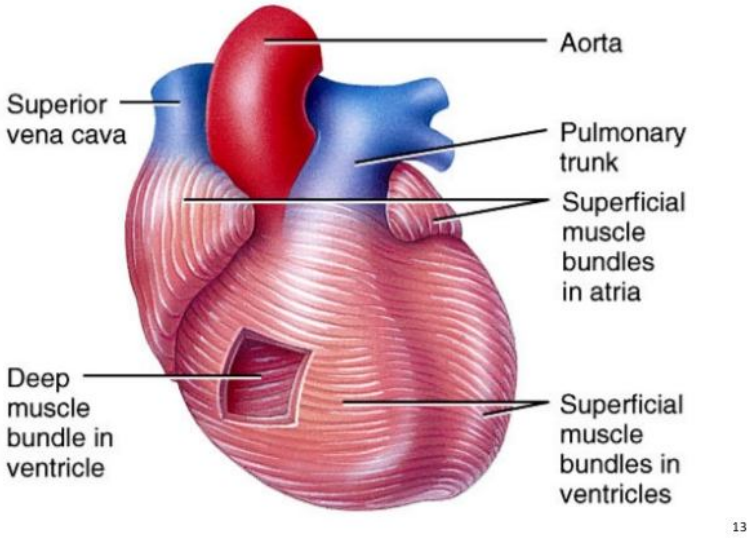


Figure 2.3:Cardiac muscle bundles of the myocardium

2.1.3 Chambers of the Heart

The heart has four chambers. The two superior receiving chambers are the atria, and the two inferior pumping chambers are the ventricles. On the anterior surface of each atrium is a wrinkled pouch like structure called an auricle, so named because of its resemblance to a dog's ear. Each auricle slightly increases the capacity of an atrium so that it can hold a greater volume of blood. (Gerard J.et al 2014)

2.1.3.1 Right atrium

The right atrium receives the superior vena cava in its upper and posterior part, the inferior vena cava and coronary sinus in its lower part, and the anterior cardiac vein (draining much of the front of the heart) anteriorly. Running more or less vertically downwards between the vena cava is a distinct muscular ridge, the crista terminalis (indicated on the outer surface of the atrium by a shallow groove—the sulcus terminalis) (Harold ,2006).

The openings of the inferior vena cava and the coronary sinus are guarded by rudimentary valves; that of the inferior vena cava being continuous with the annulus ovalis around the shallow depression on the atrial septum, the fossa ovalis, which marks the site of the fetal foramen ovale (Harold ,2006).

2.1.3.2 Right ventricle

This chamber projects to the left of the right atrium. The atrioventricular groove between the two is vertical over the front of the heart and anteroposterior on the inferior surface. It lodges the right coronary artery and is usually filled with fat.

The right ventricle narrows as it passes upwards towards the commencement of the pulmonary trunk. The interior of the cavity, whose walls are much thicker than those of the atrium, is thrown into a series of muscular ridges and bundles, the trabeculaecarneae.

One of these ridges has broken free and lies in the cavity attached by its two ends to the interventricular septum and the anterior papillary muscle. This is the septomarginaltrabecula (formerly the moderator band; it contains part of the right branch of the conducting bundle. Other bundles or bridges of muscle break free from the ventricular wall to form the papillary muscles which are attached to the cusps of the tricuspid valve (McMinn,2009).

The tricuspid valve guards the right atrioventricular orifice. It has three cusps and admits the tips of three fingers (the mitral valve has two cusps and admits two fingers). The three cusps, called anterior, posterior and septal, are attached by their bases to the fibrous atrioventricu1ar ring and are arranged to lie against the three walls of the ventricle - anterior, inferior and septal. The edges and ventricular surfaces of the cusps receive the attachments of the chordae tendineae, inelastic cords which diverge from the papillary muscles and prevent the cusps from being

everted when the ventricle contracts. Usually the large anterior papillary muscle is connected by chordae to the anterior and posterior cusps, a smaller posterior papillary muscle is attached to the posterior and septal cusps, and several small papillary muscles join the septal and anterior cusps (McMinn,2009).

The cavity of the right ventricle is flattened by the forward bulge of the intervenoicular septum. Thus the anterior wall and septum are of equal area, while the inferior wall (floor) is much narrower. The posterior cusp of the tricuspid valve is correspondingly smaller than the other two (Figure 2.4). (McMinn,2009).

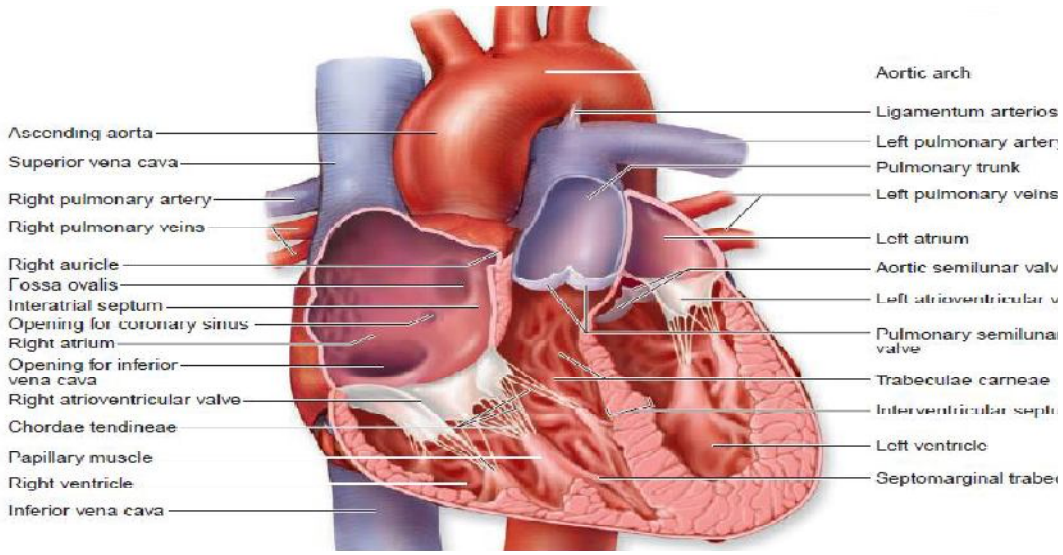


Figure 2.4:Internal Anatomy of the Heart. An illustration reveals the internal structure of the heart, including the valves and the musculature of the heart wall.

2.1.3.3 Left Atrium

Once gas exchange occurs in the lungs, the oxygenated blood travels through the pulmonary veins to the left atrium .The smooth posterior wall of the left atrium contains openings for

approximately four pulmonary veins. Sometimes two of these vessels fuse prior to reaching the left atrium, thus decreasing the number of openings through the atrial wall. Like the right atrium, the left atrium also has pectinate muscles along its anterior wall as well as an auricle (McKinley.et al 2008).

Separating the left atrium from the left ventricle is the **left atrioventricularopening**. This opening is covered by the **left atrioventricular (AV) valve** (alsocalled the bicuspid valve, since it has two triangular cusps). This valve is also sometimes called the mitral valve, because the two triangular cusps resemble a miter (the headpiece worn by a bishop). Oxygenated blood flows from the left atrium, through the left atrioventricular opening when the valve is open, into the left ventricle. The left AV valve is forced closed when the left ventricle begins to contract, preventing blood backflow into the left atrium (McKinley.et al 2008).

2.1.3.4 Left Ventricle

The left ventricle is the thickest chamber of the heart, averaging 10–15 mm (0.4– 0.6 in.) and forms the apex of the Like the right ventricle, the left ventricle contains trabeculaecarneae and has chordae tendineae that anchor the cusps of the bicuspid valve to papillary muscles (Tortora. et al 2011).

The walls of this cavity are three times as thick as those of the right ventricle . The interventricular septum bulges into the cavity of the right ventricle, so that in cross section the left ventricle is circular, the right crescentic. Trabeculaecarneae are well developed. There are two papillary muscles, anterior and posterior, the anterior being the: larger. Both are connected by chordae tendineae to each valve cusp. The posterior cusp receives the chordae on both its margin and its ventricular surface, but since blood is squirted across both surfaces of the anterior

cusp (down through the mitral orifice and up to the aortic) the chordae are attached to it only along its margins. The upper and right end of the septal wall is smooth; between the smooth part and the anterior cusp of the mitral valve is the aortic vestibule, which leads up to the aortic orifice (McMinn,2009).

The left intraventricular blood pressure is six times higher than that inside the right ventricle (Snell,2012).

The interventricular septum lies vertically from side to side across the body: the cavity of the right ventricle lies in front of it and that of the left ventricle behind it. It is marked on the surface of the heart by the interventricular branches of right and left coronary arteries. Its muscle wall, equal in thickness to that of the left ventricle, bulges forward into the cavity of the right ventricle. At its attachment to the fibrous skeleton (conjoined atrioventricular rings) it is thinner and more fibrous. This is the membranous part of the septum, and the aortic vestibule lies between it and the anterior cusp of the mitral valve (McMinn,2009).

The aortic orifice is guarded by the aortic valve, at the: entrance to the ascending aorta. It lies at a lower level than the pulmonary orifice, rather to its right side , and is more obliquely placed. Its three semilunar cusps are named right, left and posterior (in contrast to the anterior, right and left cusps of the pulmonary valve (McMinn,2009).

During fetal life, a temporary blood vessel, called the ductusarteriosus, shunts blood from the pulmonary trunk into the aorta. Hence, only a small amount of blood enters the nonfunctioning fetal lungs . The ductusarteriosus normally closes shortly after birth, leaving a remnant known as the ligamentumarteriosum , which connects the arch of the aorta and pulmonary trunk (Tortora. et al).

2.1.4 BLOOD SUPPLY

2.1.4.1 The Arterial Supply of the Heart:

The arterial supply of the heart is provided by the right and left coronary arteries, which arise from the ascending aorta immediately above the aortic valve. The coronary arteries and their major branches are distributed over the surface of the heart, lying within subepicardial connective tissue (Snell,2012).

The right coronary artery arises from the anterior aortic sinus of the ascending aorta and runs forward between the pulmonary trunk and the right auricle. It descends almost vertically in the right atrioventricular groove, and at the inferior border of the heart it continues posteriorly along the atrioventricular groove to anastomose with the left coronary artery in the posterior interventricular groove. The following branches from the right coronary artery supply the right atrium and right ventricle and parts of the left atrium and left ventricle and the atrioventricular septum (Snell,2012).

Branches

1. The right conus artery supplies the anterior surface of the pulmonary conus (infundibulum of the right ventricle) and the upper part of the anterior wall of the right ventricle (Snell,2012).

2. The anterior ventricular branches are two or three in number and supply the anterior surface of the right ventricle. The marginal branch is the largest and runs along the lower margin of the costal surface to reach the apex (Snell,2012).

3. The posterior ventricular branches are usually two in number and supply the diaphragmatic surface of the right ventricle (Snell,2012).

4. The posterior interventricular (descending) artery .It supplies branches to the posterior part of the ventricular septum but not to the apical part, which receives its supply from the anterior interventricular branch of the left coronary artery. A large septal branch supplies the atrioventricular node. In 10% of individuals, the posterior interventricular artery is replaced by a branch from the left coronary artery (Snell,2012).

5. The atrial branches supply the anterior and lateral surfaces of the right atrium. One branch supplies the posterior surface of both the right and left atria. The artery of the sinuatrial node supplies the node and the right and left atria; in 35% of individuals it arises from the left coronary artery (Snell,2012).

The left coronary artery, which is usually larger than the right coronary artery, supplies the major part of the heart, including the greater part of the left atrium, left ventricle, and ventricular septum. It arises from the left posterior aortic sinus of the ascending aorta and passes forward between the pulmonary trunk and theleft auricle. It then enters the atrioventricular groove and divides into an anterior interventricular branch and a circumflex branch (Snell,2012). Branches

1.The anterior interventricular (descending) branch. Its branch supplies the right and left ventricles with numerous branches that also supply the anterior part of the ventricular septum. One of these ventricular branches (left diagonal artery) may arise directly from the trunk of the left coronary artery. A small left conus artery supplies the pulmonary conus (Snell,2012).

2. The circumflex artery is the same size as the anteriorinterventricularartery . It winds around the left margin of the heart in the atrioventricular groove. A left marginal artery is a large branch that supplies the left margin of the left ventricle down to the apex. Anterior ventricular and posterior ventricular branches supply the left ventricle. Atrial branches supply the left atrium (Snell,2012).

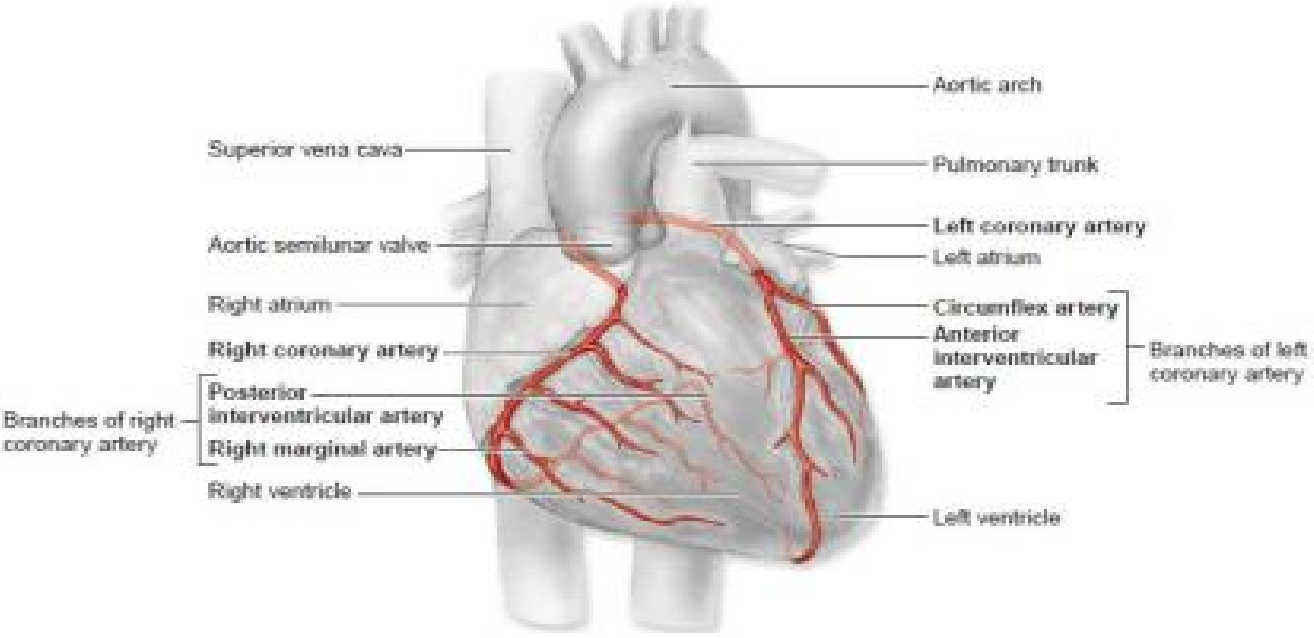


Figure2.5: Coronary Circulation. Anterior view of (a) coronary arteries.

2.1.4.2 Venous Drainage:

The bulk of the venous drainage of the heart is achieved by veins which accompany the coronary arteries and which open into the right atrium. The rest of the blood drains by means of small veins (venae cordisminimae) directly into the cardiac cavity (Harold ,2006).

The coronary sinus lies in the posterior atrioventricular groove and opens into the right atrium just to the left of the mouth of the inferior vena cava. It receives:

1-the great cardiac vein in the anterior interventricular groove;

2-the middle cardiac vein the inferior interventricular groove;

3-the small cardiac vein — accompanying the marginal artery along the lower border of the heart;

4-the oblique vein— descends obliquely on the posterior aspect of the left atrium. The anterior cardiac veins (up to three or four in number) cross the anterior atrioventricular groove, drain much of the anterior surface of the heart and open directly into the right atrium (Harold ,2006).

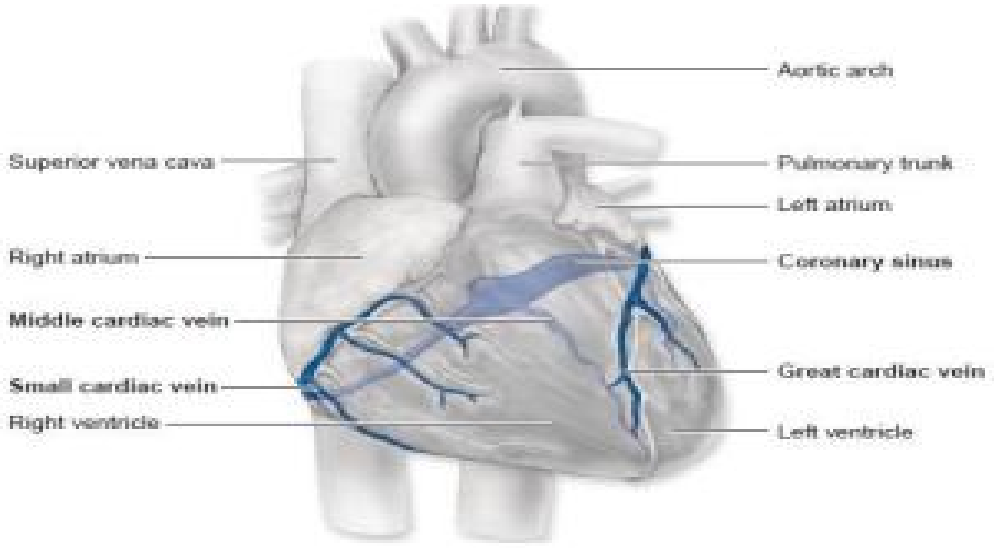


Figure2.6: Coronary Circulation. Anterior view of (a) coronary veins.

2.1.5 LYMPH DRAINAGE:

The lymphatics of the heart drain back along the coronary arteries, emerge from the fibrous pericardium along with the aorta and pulmonary trunk, and empty into the tracheobronchial lymph nodes and mediastinal lymph trunks (McMinn,2009).

2.1.6 NERVES SUPPLY

The nerve supply of the heart is derived from the vagus (cardio-inhibitor) and the cervical and upper 5 thoracic sympathetic ganglia (cardioaccelerator) by way of superficial and deep cardiac plexuses (Harold ,2006).

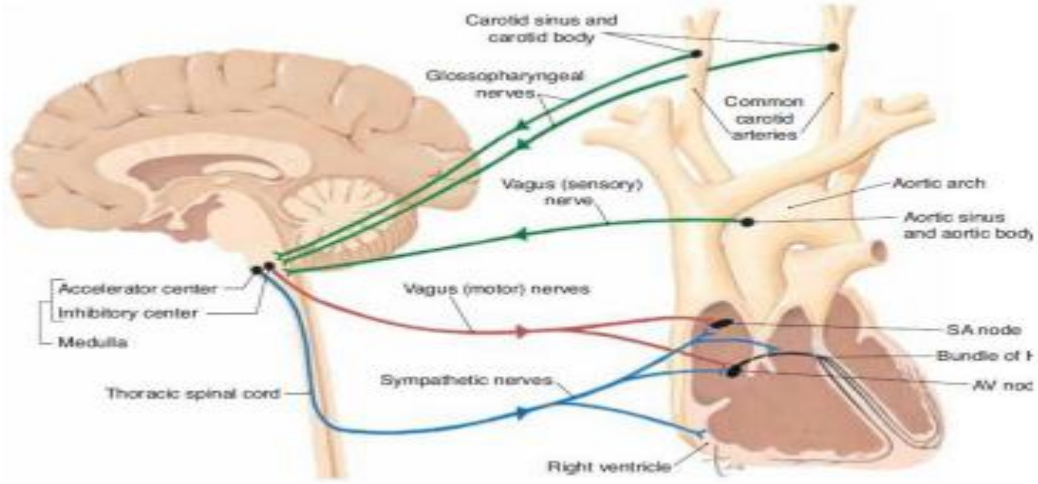


Figure 2.7: Nervous regulation of the heart. The brain and spinal cord are shown on the left. The heart and major blood vessels are shown on the right.

2.2 Physiology of The Heart

The parts of the heart normally beat in orderly sequence: Contraction of the atria (atrial systole) is followed by contraction of the ventricles (ventricular systole), and during diastole all four chambers are relaxed. The heartbeat originates in a specialized cardiac conduction system and spreads via this system to all parts of the myocardium. The structures that make up the conduction system are the sinoatrial node (SA node), the internodal atrial pathways, the atrioventricular node (AV node), the bundle of His and its branches, and the Purkinje system. The various parts of the conduction system and, under abnormal conditions, parts of the myocardium are capable of spontaneous discharge. However, the SA node normally discharges

most rapidly, depolarization spreading from it to the other regions before they discharge spontaneously. The SA node is therefore the normal cardiac pacemaker, its rate of discharge determining the rate at which the heart beats. Impulses generated in the SA node pass through the atrial pathways to the AV node, through this node to the bundle of His, and through the branches of the bundle of His via the Purkinje system to the ventricular muscle (Ganong,2005).

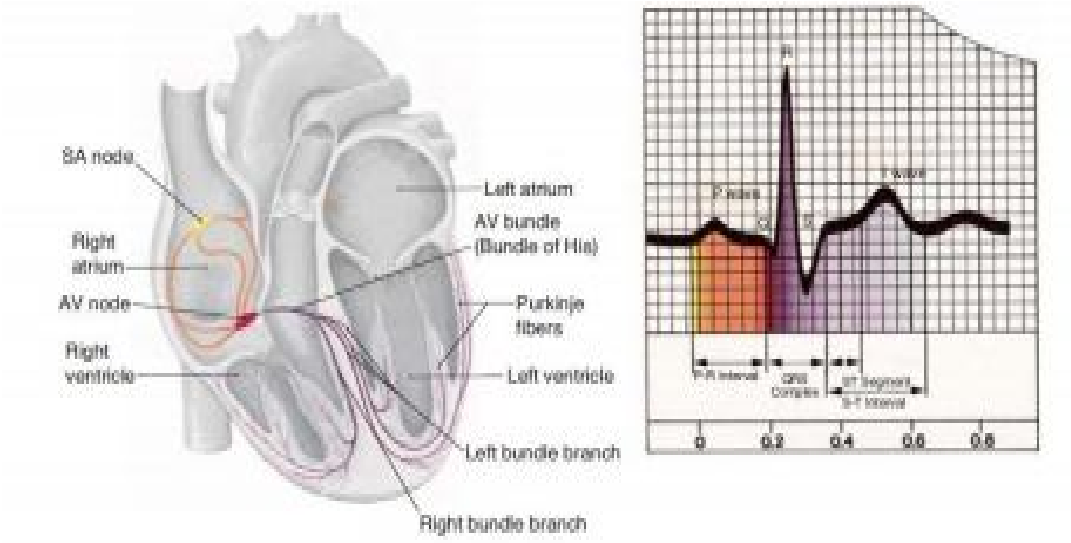


Figure2.8:Conduction pathway of the heart. Anterior view of the interior of the heart. The electrocardiogram tracing is of one normal heartbeat.

2.2.1 Anatomic Considerations

In the human heart, the SA node is located at the junction of the superior vena cava with the right atrium. The AV node is located in the right posterior portion of the interatrialseptum . There are three bundles of atrial fibers that contain Purkinje type fibers and connect the SA node to the AV node: the anterior internodal tract of Bachman, the middle internodal tract of Wenckebach, and

the posterior internodal tract of Thorel. Conduction also occurs through atrial myocytes, but it is more rapid in these bundles. The AV node is normally the only conducting pathway between the atria and ventricles. It is continuous with the bundle of His, which gives off a left bundle branch at the top of the interventricular septum and continues as the right bundle branch. The left bundle branch divides into an anterior fascicle and a posterior fascicle. The branches and fascicles run subendocardially down either side of the septum and come into contact with the Purkinje system, whose fibers spread to all parts of the ventricular myocardium (Ganong,2005).

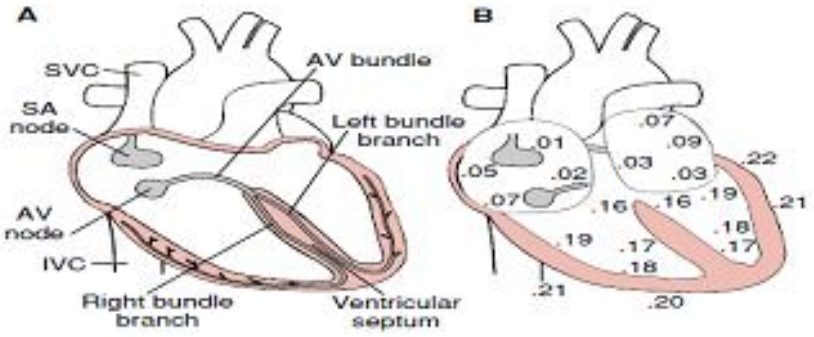


Figure 2.9:The timing of excitation of various areas of the heart (in fractions of a second).

The conduction system is composed for the most part of modified cardiac muscle that has fewer striations and indistinct boundaries. The SA node and, to a lesser extent, the AV node, also contain small round cells with few organelles, which are connected by gap junctions. These are probably the actual pacemaker cells, and therefore they are called P cells. The atrial muscle fibers are separated from those of the ventricles by a fibrous tissue ring, and normally the only conducting tissue between the atria and ventricles is the bundle of His (Ganong,2005). The heart actually is composed of two syncytiums: the atrial syncytium that constitutes the walls

of the two atria, and the ventricular syncytium that constitutes the walls of the two ventricles. Normally, potentials are not conducted from the atrial syncytium into the ventricular syncytium directly through this fibrous tissue. Instead, they are conducted only by way of a specialized conductive system called the A-V bundle (Guyton.et al 2006).

2.2.2 Action Potentials in Cardiac Muscle

The action potential recorded in a ventricular muscle Fiber, averages about 105 millivolts, which means that the intracellular potential rises from a very negative value, about -85 millivolts, between beats to a slightly positive value, about +20 millivolts, during each beat. After the initial spike, the membrane remains depolarized for about 0.2 second, exhibiting a plateau as shown in the figure, followed at the end of the plateau by abrupt repolarization. The presence of this plateau in the action potential causes ventricular contraction to last as much as 15 times as long in cardiac muscle as in skeletal muscle (Guyton.et al 2006).

2.2.3 The Cardiac Cycle

The cardiac events that occur from the beginning of one heartbeat to the beginning of the next are called the cardiac cycle. Each cycle is initiated by spontaneous generation of an action potential in the sinus node. This node islocated in the superior lateral wall of the right atrium near the opening of the superior vena cava, and the action potential travels from here rapidly through both atria and then through the A-V bundle into the ventricles. Because of this special arrangement of the conducting system from the atria into the ventricles, there is a delay of more than 0.1 second during passage of the cardiac impulse from the atria into the ventricles. This allows the atria to contract ahead of ventricular contraction, thereby pumping blood into the ventricles before the strong ventricular contraction begins. Thus, the atria act as primer pumps

for the ventricles, and the ventricles in turn provide the major source of power for moving blood through the body's vascular system (Guyton.et al 2006).

2.2.4 MECHANICAL EVENTS OF THE CARDIAC CYCLE

2.2.4.1 Diastole and Systole

The cardiac cycle consists of a period of relaxation called diastole, during which the heart fills with blood, followed by a period of contraction called systole. The top three curves show the pressure changes in the aorta, left ventricle, and left atrium, respectively. The fourth curve depicts the changes in left ventricular volume, the fifth the electrocardiogram, and the sixth a phonocardiogram, which is a recording of the sounds produced by the heart—mainly by the heart valves— as it pumps (Guyton.et al 2006).

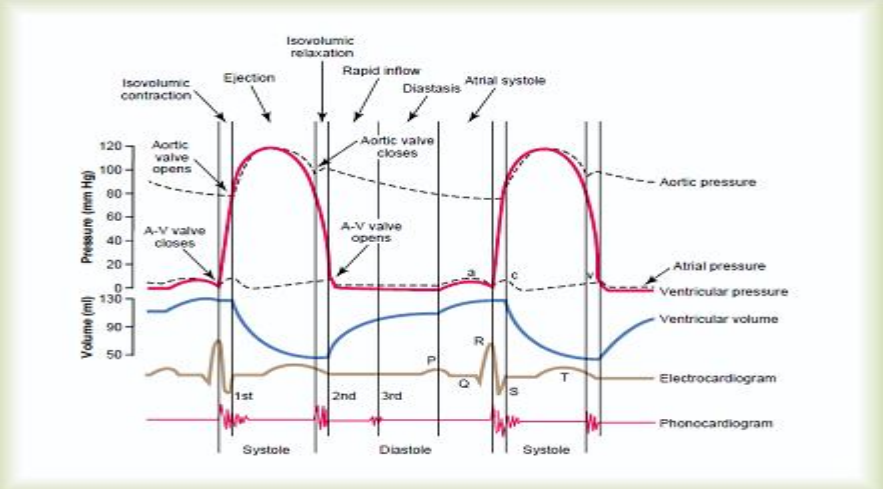


Figure 2.10:Events of the cardiac cycle for left ventricular function, showing changes in left atrial pressure, left ventricular pressure, aortic pressure, ventricular volume, the electrocardiogram, and the phonocardiogram.

2.2.4.2 Events in Late Diastole

Late in diastole, the mitral and tricuspid valves between the atria and ventricles are open and the aortic and pulmonary valves are closed. Blood flows into the heart throughout diastole, filling the atria and ventricles. The rate of filling declines as the ventricles become distended, and—especially when the heart rate is low—the cusps of the atrioventricular (AV) valves drift toward the closed position . The pressure in the ventricles remains low (Ganong,2005). Atrial Systole and Diastole. The P wave of the electrocardiogram (ECG) reflects atrial depolarization, which initiates atrial systole. Contraction of the atria “tops off” ventricular filling with a final, small volume of blood from the atria, producing the a wave. Under resting conditions, atrial systole is not essential for ventricular filling and, in its absence, ventricular filling is only slightly reduced. However, when increased cardiac output is required, as during exercise, the absence of atrial systole can limit ventricular filling and stroke volume. This happens in patients with atrial fibrillation, whose atria do not contract synchronously. The P wave is followed by an electrically quiet period, during which atrioventricular (AV) node transmission occurs (the PR segment). During this electrical pause, the mechanical events of atrial systole and ventricular filling are concluded before excitation and contraction of the ventricles begin. Atrial diastole follows atrial systole and occurs during ventricular systole. As the left atrium relaxes, blood enters the atrium from the pulmonary veins. Simultaneously, blood enters the right atrium from the superior and inferior vena cava. The gradual rise in left atrial pressure during atrial diastole produces the v wave and reflects its filling. The small pressure oscillation early in atrial diastole, called the c wave, is caused by bulging of the mitral valve and movements of the heart associated with ventricular contraction (Rhoades.et al 2003).

2.2.4.3 Ventricular Systole

At the start of ventricular systole, the mitral and tricuspid (AV) valves close. Ventricular muscle initially shortens relatively little, but intraventricular pressure rises sharply as the myocardium presses on the blood in the ventricle . This period of isovolumetric (isovolumic, isometric) ventricular contraction lasts about 0.05 s, until the pressures in the left and right ventricles exceed the pressures in the aorta (80 mm Hg; 10.6 kPa) and pulmonary artery (10 mm Hg) and the aortic and pulmonary valves open. During isovolumetric contraction, the AV valves bulge into the atria, causing a small but sharp rise in atrial pressure (Ganong,2005).

When the aortic and pulmonary valves open, the phase of ventricular ejection begins. Ejection is rapid at first, slowing down as systole progresses. The intraventricular pressure rises to a maximum and then declines somewhat before ventricular systole ends. Peak left ventricular pressure is about 120 mm Hg, and peak right ventricular pressure is 25 mm Hg or less. Late in systole, the aortic pressure actually exceeds the ventricular, but for a short period momentum keeps the blood moving forward. The AV valves are pulled down by the contractions of the ventricular muscle, and atrial pressure drops. The amount of blood ejected by each ventricle per stroke at rest is 70-90 mL. The end-diastolic ventricular volume is about 130 mL. Thus, about 50 mL of blood remains in each ventricle at the end of systole (end-systolic ventricular volume), and the ejection fraction, the percent of the end-diastolic ventricular volume that is ejected with each stroke, is about 65%. The ejection fraction is a valuable index of ventricular function. It can be measured by injecting radionuclide-labeled red blood cells, imaging the cardiac blood pool at the end of diastole and the end of systole (equilibrium radionuclide angiocardiography), and then calculating the ejection fraction(Ganong, 2005).

2.2.4.4 Early Diastole

Once the ventricular muscle is fully contracted, the already falling ventricular pressures drop more rapidly. This is the period of protodiastole. It lasts about 0.04 s. It ends when the momentum of the ejected blood is overcome and the aortic and pulmonary valves close, setting up transient vibrations in the blood and blood vessel walls. After the valves are closed, pressure continues to drop rapidly during the period of isovolumetric ventricular relaxation. Isovolumetric relaxation ends when the ventricular pressure falls below the atrial pressure and the AV valves open, permitting the ventricles to fill. Filling is rapid at first, then slows as the next cardiac contraction approaches. Atrial pressure continues to rise after the end of ventricular systole until the AV valves open, then drops and slowly rises again until the next atrial systole. (Ganong,2005).

2.2.5 Function of the Atria as Primer Pumps

Blood normally flows continually from the great veins into the atria; about 80 per cent of the blood flows directly through the atria into the ventricles even before the atria contract. Then, atrial contraction usually causes an additional 20 per cent filling of the ventricles. Therefore, the atria simply function as primer pumps that increase the ventricular pumping effectiveness as much as 20 per cent. However, the heart can continue to operate under most 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.et al 2006). The a wave is caused by atrial contraction. Ordinarily, the right atrial pressure increases 4 to 6

mm Hg during atrial contraction, and the left atrial pressure increases about 7 to 8 mm Hg. The c wave occurs when the ventricles begin to contract; it is caused partly by slight backflow of blood into the atria at the onset of ventricular contraction but mainly by bulging of the A-V valves backward toward the atria because of increasing pressure in the ventricles (Guyton.et al 2006). The v wave occurs toward the end of ventricular contraction; it results from slow flow of blood into the atria from the veins while the A-V valves are closed during ventricular contraction. Then, when ventricular contraction is over, the A-V valves open, allowing this stored atrial blood to flow rapidly into the ventricles and causing the v wave to disappear (Guyton.et al 2006).

2.2.6 Function of the Ventricles as Pumps

Filling of the Ventricles**.**During ventricular systole, large amounts of blood accumulate in the right and left atria because of the closed A-V valves. Therefore, as soon as systole is over and the ventricular pressures fall again to their low diastolic values, the moderately increased pressures that have developed in the atria during ventricular systole immediately push the A-V valves open and allow blood to flow rapidly into the ventricles, as shown by the rise of the left ventricular volume curve.This is called the period of rapid filling of the ventricles (Guyton.et al 2006). Period of Ejection.When the left ventricular pressure rises slightly above 80 mm Hg (and the right ventricular pressure slightly above 8 mm Hg), the ventricular pressures push the semilunar valves open. Immediately, blood begins to pour out of the ventricles, with about 70 per cent of the blood emptying occurring during the first third of the period of ejection and the remaining 30 per cent emptying during the next two thirds. Therefore, the first third is called the period of rapid ejection, and the last two thirds, the period of slow ejection (Guyton& Hall,2006). Period of Isovolumic (Isometric) Relaxation.At the end of systole, ventricular relaxation begins suddenly, allowing both the right and left intraventricular pressures to decrease rapidly. The

elevated pressures in the distended large arteries that have just been filled with blood from the contracted ventricles immediately push blood back toward the ventricles, which snaps the aortic and pulmonary valves closed. For another 0.03 to 0.06 second, the ventricular muscle continues to relax, even though the ventricular volume does not change, giving rise to the period of isovolumic or isometric relaxation. During this period, the intraventricular pressures decrease rapidly back to their low diastolic levels. Then the A-V valves open to begin a new cycle of ventricular pumping. (Guyton.et al 2006)

2.2.7 End-Diastolic Volume, End-Systolic Volume, and Stroke Volume

During diastole, normal filling of the ventricles increases the volume of each ventricle to about 110 to 120 milliliters. This volume is called the enddiastolicvolume. Then, as the ventricles empty during systole, the volume decreases about 70 milliliters, which is called the stroke volume output. The remaining volume in each ventricle, about 40 to 50 milliliters, is called the endsystolic volume. The fraction of the end-diastolic volume that is ejected is called the ejection fraction—usually equal to about 60 per cent. When the heart contracts strongly, the end-systolic volume can be decreased to as little as 10 to 20 milliliters. Conversely, when large amounts of blood flow into the ventricles during diastole, the ventricular end diastolic volumes can become as great as 150 to 180 milliliters in the healthy heart. By both increasing the end-diastolic volume and decreasing the end-systolic volume, the stroke volume output can be increased to more than double normal (Guyton.et al 2006).

2.2.8 Function of the Valves

Atrioventricular Valves**.**The A-V valves (the tricuspid and mitral valves) prevent backflow of blood from the ventricles to the atria during systole, and the semilunar valves (the aortic and

pulmonary artery valves) prevent backflow from the aorta and pulmonary arteries into the ventricles during diastole. These valves,for the left ventricle, close and open passively. That is, they close when a backward pressure gradient pushes blood backward, and they open when a forward pressure gradient forces blood in the forward direction. For anatomical reasons, the thin, filmy A-V valves require almost no backflow to cause closure, whereas the much heavier semilunar valves require rather rapid backflow for a few milliseconds (Guyton.et al 2006).

Function of the Papillary Muscles**:**Papillary muscles that attach to the vanes of the A-V valves by the chordae tendineae. The papillary muscles contract when the ventricular walls contract, but contrary to what might be expected, they do not help the valves to close. Instead, they pull the vanes of the valves inward toward the ventricles to prevent their bulging too far backward toward the atria during ventricular contraction. If a chorda tendinea becomes ruptured or if one of the papillary muscles becomes paralyzed, the valve bulges far backward during ventricular contraction, sometimes so far that it leaks severely and results in severe or even lethal cardiac incapacity (Guyton.et al 2006).

Aortic and Pulmonary Artery Valves:The aortic and pulmonary artery semilunar valves function quite differently from the A-V valves. First, the high pressures in the arteries at the end of systole cause the semilunar valves to snap tothe 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.et al 2006).

2.2.9 CARDIAC OUTPUT

Ejection fraction is normally more than 55%. It is dependent on heart rate, preload, afterload, and contractility and provides a nonspecific index of ventricular function. Still, it has proved to be valuable in predicting the severity of heart disease in individual patients (Rhoades.et al2003).

2.2.10 THE ELECTROCARDIOGRAM

The electrocardiogram (ECG) is a continuous record of cardiac electrical activity obtained by placing sensing electrodes on the surface of the body and recording the voltage differences generated by the heart. The equipment amplifies these voltages and causes a pen to deflect proportionally on a paper moving under it. This gives a plot of voltage as a function of time (Rhoades.et al2003).

2.3 PATHOLOGY OF THE HEART

Cardiovascular disease (CVD) is the leading cause of death in men and women in the United States. Because of economic advances, social structures, and demographics, it is predicted that CVD will become the leading cause of death worldwide by 2020, surpassing infectious diseases. It is estimated that the direct and indirect costs of CVD in the United States alone were $448.5 billion for 2008. To reduce this increase in morbidity, mortality, and cost, strategies such as population-based public health measures, preventative programs for high-risk subgroups, and the allocation of resources for treatments for CVD can be useful (Porth.et al 2009).

Although many diseases can involve the heart and blood vessels, cardiovascular dysfunction results from one or more of five principal mechanisms (Kumar, et al, 2005).

• Failure of the pump. In the most common circumstance, the cardiac muscle contracts weakly or inadequately, and the chambers cannot empty properly. In some conditions, however, the muscle cannot relax sufficiently to permit ventricular filling (Kumar, et al,2005).

• An obstruction to flow, owing to a lesion preventing valve opening or otherwise causing increased ventricular chamber pressure (e.g., aortic valvular stenosis, systemic hypertension, or aortic coarctation). The increased pressure overworks the chamber that pumps against the obstruction (Kumar, et al,2005).

• Regurgitant flow causes some of the output from each contraction to flow backward, adding a volume workload to each of the chambers, which must pump the extra blood (e.g., left ventricle in aortic regurgitation; left atrium and left ventricle in mitral regurgitation) (Kumar, et al,2005).

• Disorders of cardiac conduction. Heart block or arrhythmias owing to uncoordinated generation of impulses (e.g., atrial or ventricular fibrillation) lead to nonuniform and inefficient contractions of the muscular walls.

• Disruption of the continuity of the circulatory system that permits blood to escape (e.g., gunshot wound through the thoracic aorta) (Kumar, et al,2005).

2.3.1 CORONARY ARTERY DISEASE

The term coronary artery disease (CAD) describes heart disease caused by impaired coronary blood flow. In most cases, CAD is caused by atherosclerosis, which affects not only the coronary arteries but arteries in other areas of the body. Diseases of the coronary arteries can cause

myocardial ischemia and angina, myocardial infarction or heart attack, cardiac arrhythmias, conduction defects,heart failure, and sudden death. Heart attack is the single largest killer of men and women in the United States, Canada, and other industrialized countries. Each year, more than 1.6 million Americans have new or recurrent myocardial infarctions; one third of those die within the first 24 hours, and many of those who survive suffer significant morbidity. In spite of these numbers, the overall death rate from CAD has declined over the past several decades (Porth, et al 2009).

Major risk factors for CAD include cigarette smoking, elevated blood pressure, elevated serum total and low-density lipoprotein (LDL) cholesterol, low serum high-density lipoprotein (HDL) cholesterol, diabetes, advancing age, abdominal obesity, and physical inactivity. Individuals with diabetes and the metabolic syndrome are at particularly increased risk for development of cardiovascular disease (Porth,et al 2009).

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

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

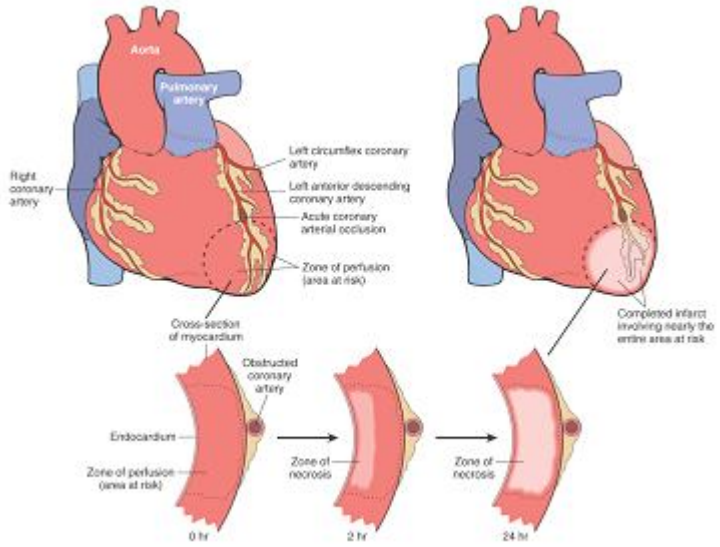


Figure2.11:Schematic representation of the progression of myocardial necrosis after coronary artery occlusion. Necrosis begins in a small zone of the myocardium beneath the endocardial surface in the center of the ischemic zone.

2.3.2 MYOCARDIAL INFARCTION (MI)

MI, also known as "heart attack," is the death of cardiac muscle resulting from ischemia. It is by far the most important form of IHD and alone is the leading cause of death in the United States and industrialized nations. About 1.5 million individuals in the United States suffer an acute MI annually and approximately one third of them die. At least 250,000 people a year die of a heart attack before they reach the hospital (Kumar, et al,2005).

2.3.2.1 Transmural versus Sub endocardial Infarction

Incidence and Risk Factors.Suffice it to say that MI may occur at virtually any age, but the frequency rises progressively with increasing age and when predispositions to atherosclerosis are present, such as hypertension, cigarette smoking, diabetes mellitus, genetic hypercholesterolemia, and other causes of hyperlipoproteinemia. Nearly 10% ofmyocardial infarcts occur in people under age 40, and 45% occur in people under age 65. Blacks and whites are equally affected. Throughout life, men are at significantly greater risk of MI than women; the differential progressively declines with advancing age. Except for those having some predisposing atherogenic condition, women are remarkably protected against MI during the reproductive years. Nevertheless, the decrease of estrogen following menopause can permit rapid development of coronary artery disease (CAD), and IHD is the overwhelming cause of death in elderly women. Moreover, recent epidemiologic evidence suggests that postmenopausal hormone replacement therapy does not protect women against MI (Kumar, et al,2005).

Pathogenesis.

We now consider the basis for and subsequent consequences of myocardial ischemia, particularly as they relate to the typicaltransmural myocardial infarct. Coronary Arterial Occlusion. transmuralacute MI results from a dynamic interaction among several or all of the following-coronary atherosclerosis, acute atheromatous plaque change (such as rupture), superimposed platelet activation, thrombosis, and vasospasm—resulting in an occlusive intracoronary thrombus overlying a disrupted plaque. In addition, either increased myocardial demand (as with hypertrophy or tachycardia) or hemodynamic compromise (as with a drop in blood pressure) can worsen the situation. Recall also that collateral circulation may provide perfusion to ischemic zones from a relatively unobstructed branch of the coronary tree,

bypassing the point of obstruction and protecting against the effects of an acute coronary occlusion (Kumar, et al,2005).

In the typical case of MI, the following sequence of events can be proposed:

• The initial event is a sudden change in the morphology of an atheromatous plaque, that is, disruption—manifest as intraplaque hemorrhage, erosion or ulceration, or rupture or fissuring (Kumar, et al,2005).

• Exposed to subendothelial collagen and necrotic plaque contents, platelets undergo adhesion, aggregation, activation, and release of potent aggregators including thromboxane A2 , serotonin, and platelet factors 3 and 4 (Kumar, et al,2005).

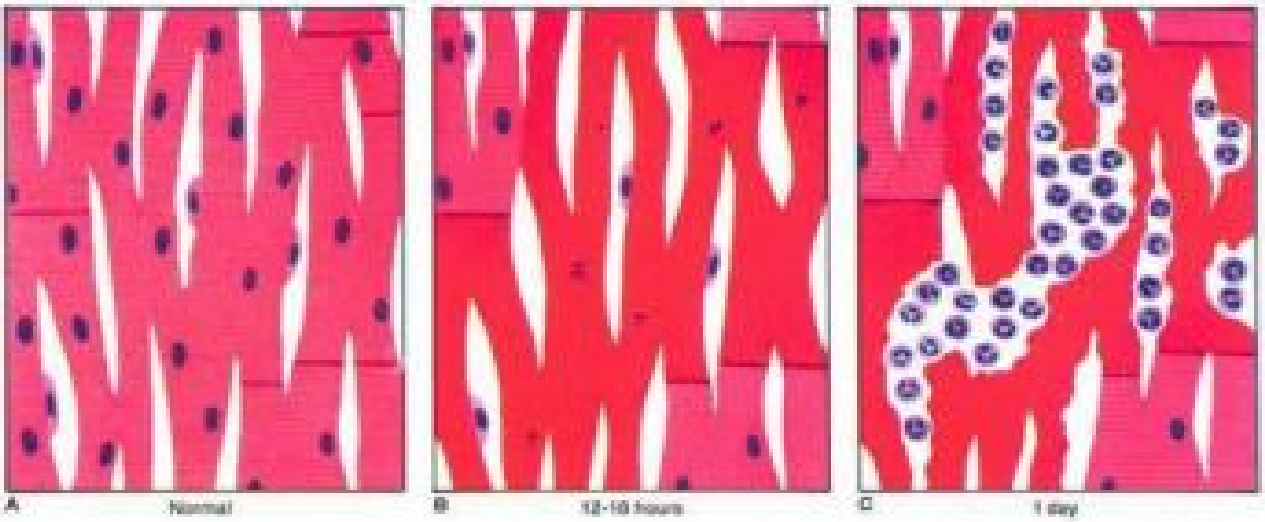
• Vasospasm is stimulated by platelet aggregation and the release of mediators (Kumar, et al,2005).

• Other mediators activate the extrinsic pathway of coagulation, adding to the bulk of the thrombus (Kumar, et al,2005).

• Frequently within minutes, the thrombus evolves to completely occlude the lumen of the coronary vessel (Kumar, et al,2005).

The evidence for this sequence is compelling and derives from (1) autopsy studies of patients dying with acute MI, (2) angiographic studies demonstrating a high frequency of thrombotic occlusion early after MI, (3) the high success rate of therapeutic thrombolysis and primary angioplasty, and (4) the demonstration of residual disrupted atherosclerotic lesions by angiography after thrombolysis. Although coronary angiography performed within 4 hours of the onset of apparent MI shows a thrombosed coronary artery in almost 90% of cases, the

observation of occlusion is seen in only about 60% when angiography is delayed until 12 to 24 hours after onset. Thus with the passage of time, at least some occlusions appear to clear



spontaneously owing to lysis of the thrombus or relaxation of spasm or both (Kumar, et al,2005).

Figure2.12: Development of a myocardial infarct. A. Normal myocardium. B. After about 12 to 18 hours, the infarcted myocardium shows eosinophilia (red staining) in sections of the heart stained with hematoxylin and eosin. C. About 24 hours after the onset of infarction, polymorphonuclear neutrophils infiltrate necrotic myocytes at the periphery of the infarct.

In approximately 10% of cases, transmuralacute MI is not associated with atherosclerotic plaque thrombosis stimulated by disruption. In such situations, other mechanisms may be involved:

• Vasospasm: isolated, intense, and relatively prolonged, with or without coronary atherosclerosis, perhaps in association with platelet aggregation (sometimes related to cocaine abuse) (Kumar, et al,2005).

• Emboli: from the left atrium in association with atrial fibrillation, a left-sided mural thrombus or vegetative endocarditis; or paradoxical emboli from the right side of the heart or the peripheral veins which cross to the systemic circulation, through a patent foramen ovale, causing coronary occlusion (Kumar, et al,2005).

• Unexplained: cases without detectable coronary atherosclerosis and thrombosis may be caused by diseases of small intramural coronary vessels such as vasculitis, hematologic abnormalities such as hemoglobinopathies, amyloid deposition in vascular walls, or other unusual disorders, such as vascular dissection and inadequate protection during cardiac surgery (Kumar, et al,2005).

2.4 Principle of nuclear medicine

Nuclear medicine imaging procedures differ from ordinary x-rays in that the gamma rays are emitted from the body rather than transmitted across the body, as in the case of x-rays. As in most modern imaging, the principle of tomography is used, that is, the person is viewed by radiation detectors surrounding the body, or by rotation of a gamma camera around the body. Small amount of radiopharmaceuticals is injected into vein, and its distribution at specific time periods afterward is imaged in certain organs of the body or in the entire body. The images are created by measuring the gamma-ray photons emitted from the organs or regions of interest within the body.

2.4.1 Myocardial perfusion study

Myocardial perfusion Scintigraphy is currently by far the most commonly performed cardiac nuclear study, constituting approximately one third of all nuclear medicine procedures done annually in the United States, with substantial growth annually. Gated myocardial perfusion Scintigraphy plays an important role in the diagnosis, prognosis, risk assessment, and management of heart disease. Myocardial perfusion scintigraphy depicts two sequential physiological events. First, the radiopharmaceutical must be delivered to the myocardium. Second, a viable metabolically active myocardial cell must be present to extract the radiotracer.

The scintigraphic images are a map of regional myocardial perfusion to viable myocardial tissue. If a patient has a decrease in regional perfusion due to hemodynamically significant coronary artery disease or a loss of cell viability as a result of myocardial infarction, a photon-deficient or cold region is seen on the perfusion images. All diagnostic patterns in the many diverse applications follow from these observations (Harvey A. Ziessman, et al 2014)

2.4.2 Radiopharmaceuticals

Radiolabeled potassium (K+) was first considered for myocardial perfusion imaging because it is the major intracellular cation. Sodium (Na+)–K+ homeostasis is maintained as an energy-dependent process involving the Na+–K+ATPase (adenosine triphosphatase) pump in the myocardial cell membrane. However, neither K+ nor its analogs, cesium and rubidium,were found suitable for single-photon imaging because of their high energy photons. Rubidium (Rb-82) has found use in dual-photon imaging and positron emission tomography (PET).

The first radiopharmaceutical used on a clinical basis for perfusion imaging was thallium-201 chloride (Tl-201). Tl-201 behaves physiologically much like K+, although it is not a true K+ analog in a chemical sense. First used for myocardial scintigraphy in the mid-1970s,it remained the only perfusion agent available until 1990. Two Tc-99mlabeled cardiac perfusion radiopharmaceuticals, sestamibi and tetrofosmin,were introduced. All three are now routinely used clinically for myocardial perfusion scintigraphy (Harvey A. Ziessman, et al 2014).

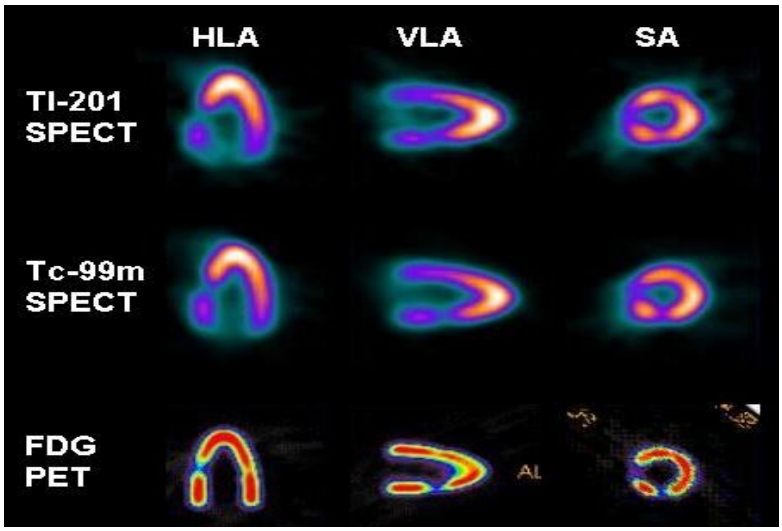


Figure 2.13:realistic heart phantom with defects of varying sizes, encased in a nonuniform attenuation medium, was imaged in SPECT mode when filled with either Tl-201 or Tc-99m, and in PET mode when filled with F-18 FDG.

2.4.3 Myocardial perfusion technique

Ensuring an optimal scintigraphic result starts with proper patient preparation. Place the patient supine Position with heart in center field of view and left arm up over head if possible. If arm is down at side because of problems with shoulder joint or recent surgery, both the rest and stress images should be taken the same way Figure 1.4.3. Table 1.4.3 shows the commonly used protocols. When thallium alone is used, stress injection is given first and redistribution imaging at 3 to 4 hours later. Also, 24-hour imaging may be done for additional information about ischemic viable myocardium. Imaging with technetium-labeled tracers can be done with either a 1- or 2-day protocol. In the 2-day protocol, the rest and stress examinations are done on separate days, each with a dose of 20 to 30 mCi (typically 30 mCi). For the 1-day study, patients can have either the rest or stress study performed first, typically with a dose of 10 mCi, followed about 4 to 6 hours later (to permit both physical decay and biological loss of radiopharmaceutical from the myocardium) by the other examination, with a dose of 30 mCi. Typically, the rest study is performed with the low dose first, followed by the stress study several hours later.

A dual isotope technique, using thallium for the rest imaging and a technetium agent (sestamibi or tetrofosmin) for the stress-imaging is an alternative approach for a one day imaging protocol. There is a minimal amount of activity in the technetium window from the rest injected thallium (caused by a 12% incidence of gamma photons from 201Tl). The advantage of this approach is that activity in the bowel is minimized, and the stress test can be performed immediately after the rest thallium images are obtained, eliminating the waiting time between the rest and stress studies when a technetium labeled tracer is used for both the rest and stress images.

Before the stress procedure, a history and cardiac directed physical examination should be completed to evaluate the patient's overall status and determine whether the patient is likely to perform sufficient exercise to achieve the target heart rate. If it appears the patient is too frail or has a medical problem that would preclude exercise, pharmacological stress should be used (table 1.4.3.1) .A complete medication history is obtained to determine if any medication would alter the sensitivity of the stress procedure. If the test is being done to determine if medical therapy reduces the incidence of ischemia, it is helpful to perform the procedure while the patient is taking the medication. If the test is done to make the diagnosis of coronary disease, β-blocking medication and calcium channel–blocking medication should be stopped for at least 24 hours before the stress. During the stress test, patients must be continuously observed, a 12-lead ECG must be recorded each minute, and blood pressure must record at least every 3 minutes. Indications to stop the stress test are summarized in (table 1.4.3.1) Contraindications to performing a stress test are summarized in (table 1.4.3.2).(Paul E. & Kristen M. 2012).

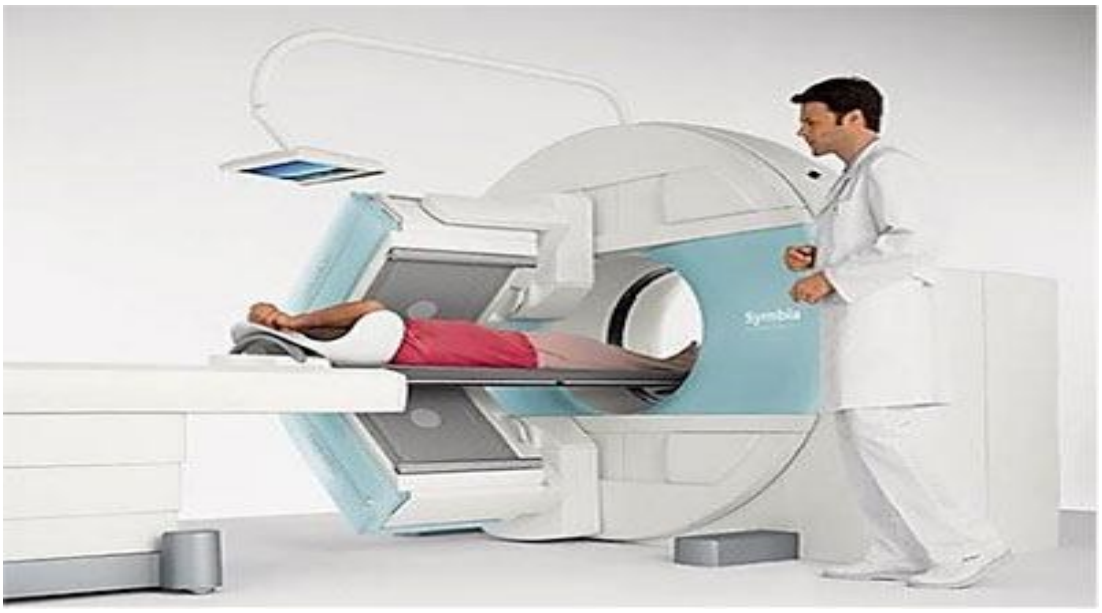


Figure 2.14 shows the patient position for the myocardial perfusion scan

Table 2.1: shows imaging protocols for MPS

Protocol	Comments
Technetium Agents: 99mTc-sestamibi or 99mTc-tetrofosmin **Two-day stress-rest:** Inject 20 to 30 mCi at peak exercise. Begin imaging at 15 to 30 minutes after injection during exercise. On day 2, give same dose at rest. Begin imaging at 45 to 60 minutes after injection at rest. **One-day rest-stress**: This is a same-day imaging protocol using a low-dose (8 to 10 mCi) resting study followed by a high-dose (20 to 30 mCi) stress study. This protocol is preferred over the stress-rest Technique. **One-day stress-rest**: Give stress injection of 10 to 15 mCi, followed by an interval of 2 to 4 hours; then give rest injection of 20 to 30 mCi. **Thallium-201 (201Tl)** **Stress and delay (standard):** Inject 2.5 to 4.0 mCi at peak stress. Record images within 20 minutes of injection. Wait 2 to 4 hours (3 hours is preferred) and record "redistribution" images. After recording the stress images, an additional 1.5 mCi of 201Tl may be injected, before recording the rest images at 3 to 4 hours. **Rest redistribution**: Inject 3 mCi at rest and obtain initial image. Delayed	Observing a waiting period after injection allows adequate hepatobiliary clearance. For sestamibi, the recommended wait after injection is at least 15 to 20 minutes for exercise stress, 45 minutes for pharmacological stress, and 45 to 60 minutes for rest. For tetrofosmin, the recommended wait after injection is at least 10 to 15 minutes after exercise stress, 45 minutes after pharmacological stress, and 30 to 45 minutes after rest.

image is acquired at 3 hours. Further imaging may be done at 24 hours without reinjection. **Dual Isotope** 201Tl rest and 99mTc-sestamibi/tetrofosmin stress studies are performed the same day. Inject 3 mCi of thallium and image. Later inject 20 to 30 mCi of technetium agent at stress. Image at appropriate times.	

Table 2.2: shows the indications to Stop Exercise

Indications to Stop Exercise
1. Marked arrhythmia induced by exercise
2. Decrease in BP > 20 mm Hg below the starting BP or heart
rate as exercise progresses
3. Extreme elevation in blood pressure (systolic pressure
>250 mm Hg or diastolic pressure >120 mm Hg)
4. Severe chest pain, marked dyspnea, and dizziness
5. Achievement of greater than 85% (preferably 100%) of predicted
heart rate for age, computed as 220 − age (yr)
6. Severe ST depression (>3 mm) or >1 mm ST elevation in leads
without pathological Q waves
7. Onset of advanced atrioventricular block
8. Onset of bundle branch block
9. Failure of monitoring system
10. Severe fatigue, leg pain, or breathlessness

Table 2.3: shows the contraindications for Stress Test

contraindications for Stress Test
Uncontrolled unstable angina; however, patients with suspected unstable angina at presentation who are otherwise stable and pain free can undergo stress testing
2. Patient with decompensated or inadequately controlled congestive heart failure
3. Uncontrolled hypertension (BP > 200/115 mm Hg)
4. Acute myocardial infarction within last 2 to 3 days
5. Severe pulmonary hypertension

2.4.4 SPECT Imaging

SPECT is the standard method for myocardial perfusion scintigraphy. The cross-sectional images have high-contrast resolution and are displayed three-dimensionally along the short and long axis of the heart (Figs2.15 and 2.16), providing good delineation of the various regional myocardial perfusion beds supplied by their individual Coronary arteries (Harvey A. Ziessman, et al 2014)

SPECT data are collected using a 180-degree orbit, with the left arm extended above the head to be out of the field of view. Right arm can be at the side. Data acquisition or the 180-degree orbit typically begins at 45 degrees RAO and ends at 45 degrees LPO. Image acquisition time is about 30 minutes using a dual detector camera with the detectors at 90 degrees following injection of 30 mCi (the dose typically used at stress) and a 3-degree step angle. A typical SPECT protocol requires 64 stops for 180-degree acquisition.

Two types of artifacts occur fairly frequently on SPECT images: attenuation (caused by breast or stomach tissue) and motion (caused by slight patient movement or differences in the phase of

respiration as a projection is being recorded). Both of these artifacts tend to cause areas of decreased counts in the reconstructed data that can be mistaken for zones of decreased perfusion. These artifacts can be readily appreciated if the projection data are reviewed in a cinematic display. (Paul E. & Kristen M. 2012).

The SPECT cross-sectional images depict the regional perfusion of the myocardium as it relates to the coronary artery supplying blood to that region and permits visual estimation of the degree and extent of the perfusion abnormality (Figs2.17). (Harvey A. Ziessman, et al 2014)

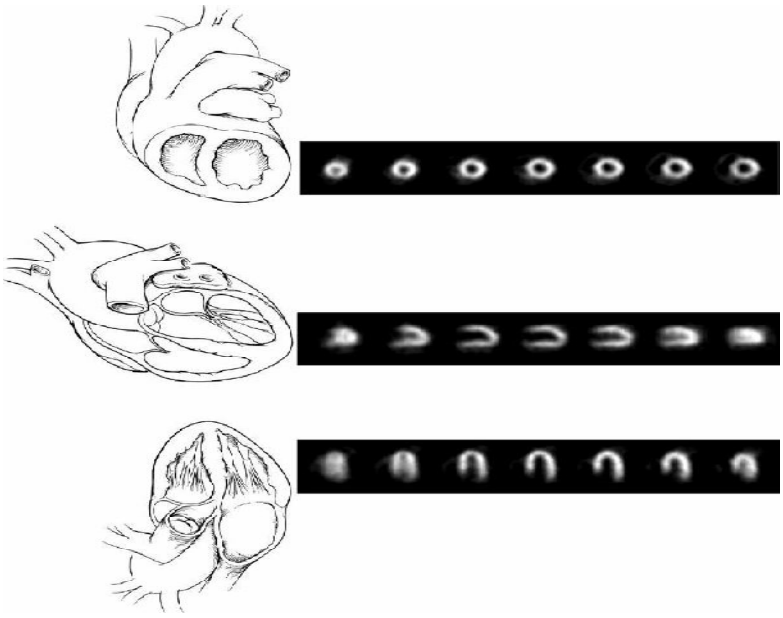


Figure 2.15: shows Cardiac SPECT cross-sectional images with corresponding anatomy. The slices are cut along the short and long axis of the heart: top (short-axis), middle (vertical long axis), bottom (horizontal long axis)

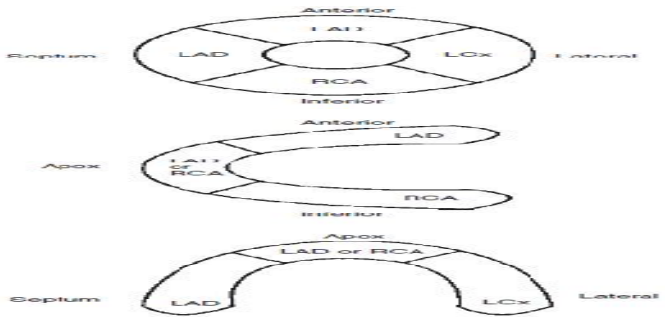


Figure 2.16: shows SPECT processed to obtain cross-sectional slices cut along the short and long axis of the heart.

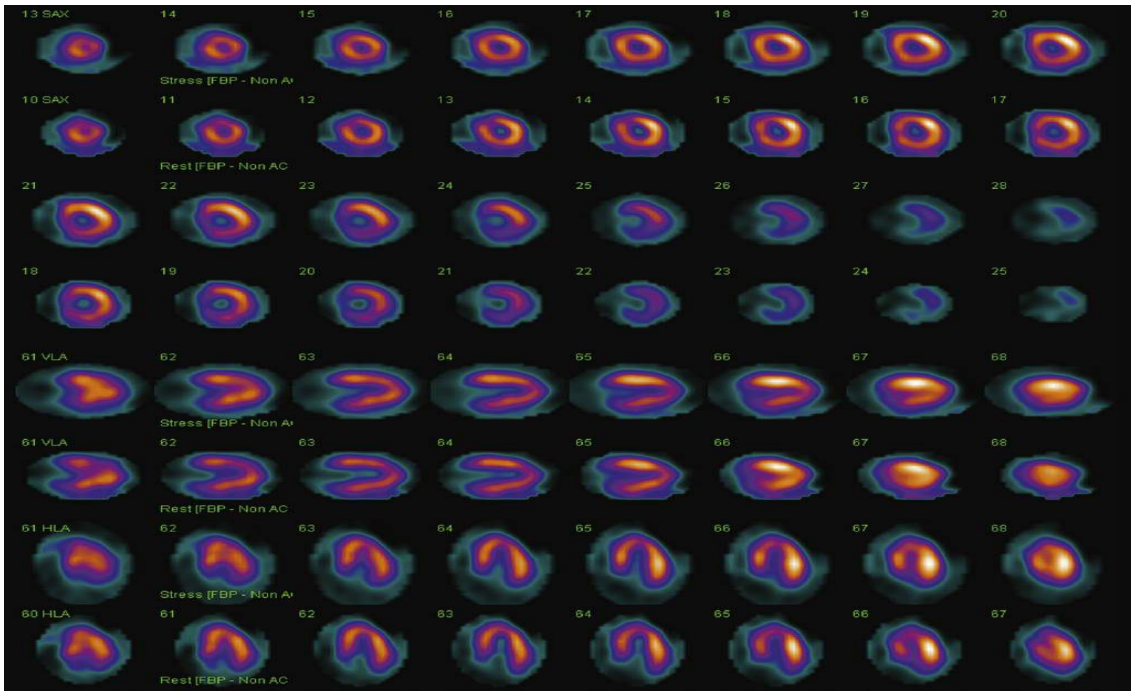


Figure 2.17: shows Normal myocardial SPECT perfusion study. Top four rows display the short-axis transaxial images (stress above, rest below), fifth and sixth rows display the horizontal long-axis (sagittal) stress and rest views, and the seventh and eighth rows display the vertical long-axis (coronal) views.

2.4.5 Gated perfusion study

Gated perfusion SPECT is typically recorded using 8 frames per cardiac cycle. When 64 angles are recorded with 8 frames/cycle at each location, a total of 512 images are obtained. Although end-systole is not correctly identified under this circumstance, the apparent loss of ejection fraction is typically on the order of 5% and is most apparent in patients with high ejection

fractions at rest. This type of study has sufficient precision to differentiate patients with normal ejection fractions from those with depressed function.

2.4.6 Data Analysis.

Myocardial perfusion images are initially interpreted subjectively to detect zones of diminished tracer localization in the left and right ventricular myocardium. The images are also evaluated using quantitative techniques to determine the relative activity in each segment of myocardium. On the tomographic images the quantitative data compares the relative distribution in each segment to a database of normal subjects. Regions with diminished activity are highlighted. Qualitative interpretation reviews the images for the factors listed in. SPECT images are usually quantified by stacking the reconstructed/ reoriented short-axis slices onto a bull's-eye display, where the apex is at the center and the basal slice is at the periphery (Figure 2.18). (Paul E. & Kristen M. 2012).

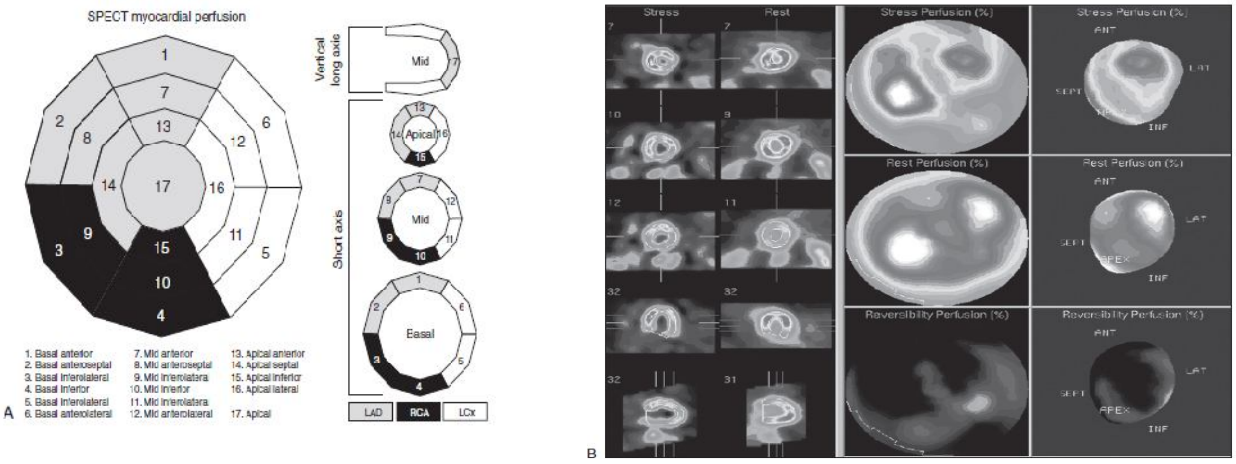


Figure 2.18: shows A, Diagram of 17-segment model. Left, Bull's-eye format of concentric short-axis views; right, the location of the segments in the short and vertical long axes. The gray segments are in the left anterior descending (LAD) territory, the black segments are right coronary artery (RCA) territory, and the blank segments are left circumflex (LCx). B, The AutoQuant display of the Bull's-eye (ADAC) showing the ischemic lesion in the LAD and LCx territories (middle column) and selected short-axis and vertical long-axis views (stress left and rest right), and the bullet depiction (right column). Several manufacturers offer variations of this display.

2.5 Previous study

Boiten HJ et al 2015studied the Eleven-year prognostic value of dobutamine stress (99m)Tc-sestamibi myocardial perfusion imaging in patients with limited exercise capacity.The aim of their study was to assess the long-term prognostic value of dobutamine stress technetium-99m ((99m)Tc)-sestamibi MPI in these patients. The methodology used wereconsisted of a high-risk cohort of 531 consecutive patients with limited exercise capacity who underwent dobutamine stress (99m)Tc-sestamibi MPI for the assessment of known or suspected coronary artery disease.Follow-up was successful in 528 patients. Because of early revascularization, 55 patients were excluded. The present data are based on 473 patients. The end points were all-cause mortality, cardiac death, nonfatal myocardial infarction, and late (>60 days) coronary revascularization. Kaplan-Meier survival curves were performed and univariate and multivariate analyses were performed to identify predictors of very long term outcome. the result which found was that The mean age of the patients was 61 ± 12 years, and 58% were men. Abnormal results (defined as the presence of reversible or fixed defects) were observed in 312 patients (66%). During a mean follow-up period of 11.3 ± 6.7 years, 287 patients (61%) died (all-cause mortality), of whom 125 (26%) died due to cardiac causes. Nonfatal myocardial infarction occurred in 59 patients (12%). Late coronary revascularization was performed in 61 patients (13%). Univariate predictors of major cardiac events included age, male gender, previous infarction, diabetes mellitus, history of angina, heart failure, ST-segment changes, abnormal results on (99m)Tc-sestamibi scan, reversible defect, fixed defect, summed rest score, and summed stress score. Multivariate analysis identified abnormal results on MPI as a strong independent predictor of major adverse cardiac events.The conclusion of this study was the patients with limited exercise capacity, dobutamine stress (99m)Tc-sestamibi single-photon

emission computed tomography provides incremental prognostic information in addition to clinical and stress test parameters for the prediction of very long term outcomes.

Romero-Farina G et al 2013 Analyzed of the diastolic function by myocardial perfusion gated SPECT after coronary revascularization in acute myocardial infarction, the aim of their study was to evaluate the evaluative changes in diastolic function after percutaneous coronary revascularization (PCR) in acute myocardial infarction (AMI), using myocardial perfusion gated SPECT. The methodology used were thirty-two patients (mean 61.9±9.7 years, 7 women) were studied by two at rest gated SPECT: the first gated-SPECT-1 was performed with an injection of a dose of (99m)Tc-tetrofosmin prior to PCR and the second gated-SPECT-2 between the fourth and fifth weeks after AMI. Changes of peak filling rate (PFR) and the time to peak filling rate (TTPF) were assessed between both studies, and were related to the extent of salvaged myocardium (SM), end-diastolic (EDV) and end-systolic (ESV) volumes, and left ventricular ejection fraction (LVEF) changes. The result which they found were An improvement was observed in diastolic function parameters Gated-SPECT-2: PFR increased significantly (P=0.011) while the TTPF decreased without reaching statistical significance (P=0.288). In multivariate analysis, adjusted by clinical and coronary variables, improvement of PFR was significantly associated with percentage of SM (P=0.030), increase in LVEF (P=0.004) and with ESV volume reduction (P=0.005). Improvement of TTPF was only related significantly to the percentage of SM (P=0.046). PFR increased 0.01 EDV/sec. and TTPF decreased 1.14ms for each cm(2) increase of the area of SM. the conclusion was after PCR in AMI, the myocardial perfusion gated SPECT makes it possible to assess the significant improvement in diastolic function mainly related to the amount of MS.

Cuberas-Borrós et al 2013 describe the Gated-SPECT Myocardial Perfusion Imaging as a Complementary Technique to Magnetic Resonance Imaging in Chronic Myocardial Infarction Patients. The aim of this study was to compare magnetic resonance and gated-SPECT myocardial perfusion imaging in patients with chronic myocardial infarction. The methodology used was magnetic resonance imaging and gated-SPECT were performed in 104 patients (mean age, 61 [12] years; 87.5% male) with a previous infarction. Left ventricular volumes and ejection fraction and classic late gadolinium enhancement viability criteria (<75% transmurality) were correlated with those of SPECT (uptake >50%) in the 17 segments of the left ventricle. Motion, thickening, and ischemia on gated-SPECT were analyzed in segments showing nonviable tissue or equivocal enhancement features (50%-75% transmurality).the result which found was a good correlation was observed between the 2 techniques for volumes, ejection fraction (P<.05), and estimated necrotic mass (P<.01). In total, 82 of 264 segments (31%) with >75% enhancement had >50% SPECT uptake. Of the 106 equivocal segments (50%-75% enhancement) on magnetic resonance imaging, 68 (64%) had >50% uptake, 41 (38.7%) had normal motion, 46 (43.4%) had normal thickening, and 17 (16%) had ischemic criteria on SPECT. the conclusion of this study was a third of nonviable segments on magnetic resonance imaging showed >50% uptake on SPECT. Gated-SPECT can be useful in the analysis of motion, thickening, and ischemic criteria in segments with questionable viability on magnetic resonance imaging.

Magdalena K and Wojciech S 2012the prognostic value of normal myocardial perfusion SPECT with positive coronary angiography; the aim of their study was to show the prognostic value of normal myocardial perfusion spect with positive coronary angiography, the methodology used were 45 patients were included into the study. All patients had normal SPECT study and positive angiography in the ≤ 6 months after SPECT. 20 of them were

women.Sixpatients had diabetes mellitus, 8 was smokers. Two patients had had left main coronary artery and 12 had multivessel disease. Baseline clinical risk factors were recorded for each patients and compared to outcomes. And the result which their found were 45 patients, 20 were women, 40 (88,8%) had at least one risk factor (12 — hypertension; 29 — dyslipidemia, 6 — type 2 diabetes, 8 — smoking and 40 — family history of CAD). Before exercise testing, 10 women and 1 man were not taking anti-angina medication; 11 patients (7 women) were taking beta-blockers; 11 patients (10 women), calcium antagonists; 11 patients (10 women), nitrates, and 16 patients (10 women), antiplatelet drugs. All patients had positive exercise test and normal 99mTc MIBI SPECT perfusion images. All 45 patients underwent coronary angiography using Seldinger's technique at ≤ 6 months after the SPECT study, assuming no previous complications. One of the 20 women had 40% stenosis of the left main coronary artery, 6 had 1 and 14 had 2-vessel disease. One of the 25 men had 50% stenosis of the left main coronary artery, 8 had multivessel disease and 16 had one vessel disease. Finally they observed that the normal SPECT with positive ECG pattern is infrequent and has a very good prognostic value However, the long-term survival among a patient cohort with a normal exercise SPECT study is influenced by the number of concomitant CAD risk factors. they conclude that there was is an importance of modifying CAD risk factors among patients with a normal SPECT.

Kazuya et al 2010 describe the Clinical significance of ischemic electrocardiographic changes during stress myocardial perfusion imaging. The present study was designed to determine the clinical significance and prediction for cardiac events in patients who showed ischemic ECG changes during stress myocardial perfusion SPECT. the data was collected among 4,670 registered patients for Japanese-assessment of cardiac event and survival (J-ACCESS) study, patients with conduction abnormality on baseline were excluded and revascularization within

60 days of SPECT study were censored from the prognostic portion of analysis. Stress and rest myocardial perfusion SPECT imaging with 99mTc-tetrofosmin were performed and occurrence and nature of cardiac events were investigated at 1, 2 and 3 years after registration. PCI and CABG, as well as recurrent angina and non-severe heart failure were classified as soft events. Cardiac death, non-fatal MI and heart failure requiring hospitalization were classified as major cardiac events, and hard events comprised cardiac death and non-fatal MI. the result showed that a total of 3,125 patients performed exercise (n = 2,383) or vasodilator (n = 742) stress MPI and significant ischemic ECG changes were obtained in 538 during exercise and 35 during vasodilator stress. Kaplan–Meier analysis revealed that the patients with both ischemic ECG changes and reversible perfusion defect on MPI had significantly higher incidence for major cardiac events, such as cardiac death, non-fatal MI and severe heart failure (P = 0.0038), and for cardiac hard events, such as cardiac death and non-fatal MI (P = 0.0028), in exercise stress. Interestingly, patients without reversible perfusion defect showed significantly fewer events despite presence of ischemic ECG changes. In the conclusion founded ischemic ECG changes during exercise stress are well associated with higher incidence of cardiac events in patients demonstrated reversible perfusion defect on MPI.

Rosemarie G. Ramos, and Kenneth Olden 2008studied the Prevalence of Metabolic Syndrome among US Women of Childbearing Age. The present study was designed to determine whether the prevalence of metabolic syndrome among US women of childbearing age (18–44 years) has increased since 1988 and to estimate its current prevalence by race/ethnicity and risk that a maternal history of select metabolic syndrome characteristics imposes on offspring.The data was collected amongsurvey-specific data analysis methods to examine data from the National Health and Nutrition Examination Surveys conducted from 1988 to 2004.for this study, “women of

childbearing age" were defined as those aged between 18 and 44 years at the time of their participation in the NHANES and who reported being non-Hispanic White, non-Hispanic Black, or Hispanic. Because were interested in characterizing the prevalence of metabolic syndrome within an otherwise healthy population, they excluded women who reported having been diagnosed with diabetes. And they also excluded those who were pregnant at the time of their participation in NHANES and those who reported fasting for less than 8 hours before blood collection during the laboratory assessment phase of the survey. The result showed thatthe prevalence of the metabolic syndrome phenotype and 2 of its clinical correlates significantly increased between 1988 and 2004 (increase for metabolic syndrome phenotype=7.6%, for obesity=13.3%, and for elevated C-reactive protein=10.6%; $P<.001$ for all 3). Hispanic women were more likely than were White women to possess the phenotype ($P= .004$). Women who reported that their mothers had been diagnosed with diabetes were more likely to possess the phenotype than those whose mothers had not been so diagnosed (odds ratio=1.9; 95% confidence interval=1.3, 2.8). In the conclusion foundedthat the metabolic syndrome consider the relationship between the increasing prevalence of the metabolic syndrome phenotype among women of child-bearing age and increasing morbidity and mortality from CVD among girls and women of all ages.

Sciagrà R 2007 describe the expanding role of left ventricular functional assessment using gated myocardial perfusion SPECT, the aim of his study was to describe this evolution of gated SPECT functional assessment from a supporting rank with respect to perfusion, to a main actor position in the field of cardiac imaging. He found that the indications for myocardial perfusion imaging and strengthened its position among the different imaging modalities. Moreover, several

studies show that the evaluation of ventricular function may have a leading part in justifying the execution of perfusion scintigraphy in various clinical conditions.

Booth GLet al 2006 describe theRelation between age and cardiovascular disease in men and women with diabetes compared with non-diabetic people. The aim of their study was to find out the age at which people with diabetes develop a high risk of CVD, as defined by: an event rate equivalent to a 10-year risk of 20% or more; or an event rate equivalent to that associated with previous myocardial infarction. The methodology used wasa population-based retrospective cohort study using provincial health claims to identify all adults with (n=379,003) and (n=9,018,082) without diabetes mellitus living in Ontario, Canada, on April 1, 1994. Individuals were followed up to record CVD events until March 31, 2000.The result which they found was the transition to a high-risk category occurred at a younger age for men and women with diabetes than for those without diabetes (mean difference 14.6 years). For the outcome of acute myocardial infarction (AMI), stroke, or death from any cause, diabetic men and women entered the high-risk category at ages 47.9 and 54.3 years respectively. When they used a broader definition of CVD that also included coronary or carotid revascularization, the ages were 41.3 and 47.7 years for men and women with diabetes respectively. The conclusion was found that the diabetes confers an equivalent risk to ageing 15 years. However, in general, younger people with diabetes (age 40 or younger) do not seem to be at high risk of CVD. Age should be taken into account in targeting of risk reduction in people with diabetes.

Yusuf et al 2005 describe the obesity and the risk of myocardial infarction in 27 000 participants from 52 countries. The aim of their study was to assess whether markers of obesity, especially waist-to-hip ratio, would be stronger indicators of myocardial infarction than body-mass index (BMI), the conventional measure. The methodology used was a standardized case-control study

of acute myocardial infarction with 27 098 participants in 52 countries (12 461 cases and 14 637 controls) representing several major ethnic groups. They assessed the relation between BMI, waist and hip circumferences, and waist-to-hip ratio to myocardial infarction overall and for each group. The result which they found was BMI showed a modest and graded association with myocardial infarction (OR 1·44, 95% CI 1·32–1·57 top quintile vs bottom quintile before adjustment), which was substantially reduced after adjustment for waist-to-hip ratio (1·12, 1·03–1·22), and non-significant after adjustment for other risk factors (0·98, 0·88–1·09). For waist-to-hip ratio, the odds ratios for every successive quintile were significantly greater than that of the previous one (2nd quintile: 1·15, 1·05–1·26; 3rd quintile: 1·39; 1·28–1·52; 4th quintile: 1·90, 1·74–2·07; and 5th quintiles: 2·52, 2·31–2·74 [adjusted for age, sex, region, and smoking]). Waist (adjusted OR 1·77; 1·59–1·97) and hip (0·73; 0·66–0·80) circumferences were both highly significant after adjustment for BMI ($p<0·0001$ top vs bottom quintiles). Waist-to-hip ratio and waist and hip circumferences were closely ($p<0·0001$) associated with risk of myocardial infarction even after adjustment for other risk factors (ORs for top quintile vs lowest quintiles were 1·75, 1·33, and 0·76, respectively). The population-attributable risks of myocardial infarction for increased waist-to-hip ratio in the top two quintiles was 24·3% (95% CI 22·5–26·2) compared with only 7·7% (6·0–10·0) for the top two quintiles of BMI. The conclusion was found the Waist-to-hip ratio shows a graded and highly significant association with myocardial infarction risk worldwide. Redefinition of obesity based on waist-to-hip ratio instead of BMI increases the estimate of myocardial infarction attributable to obesity in most ethnic groups.

Bialostozky D.2004 described the patient with chest pain syndrome in normal or non-diagnostic conventional ECG at the emergency service, assessment with myocardial perfusion (SPECT) and

ventricular function (Gated-SPECT).the aim of the study was to assessment of paint with chest pain syndrome in normal or non-diagnostic conventional ECG at the emergency service by using myocardial perfusion (SPECT) and ventricular function (Gated-SPECT). The methodology was around 6 million persons are seen each year at the Emergency units in the USA, More than half of the patients are admitted for their cardiac evaluation, cardiac origin is confirmed in 10 to 15%, and about 15% of them develop myocardial infarction. However, 5 to 10% of patients are dismissed and develop myocardial infarction during the next 48 h, the diagnosis of the infarct is inadvertent and/or patients is not hospitalized in 2 to 8%. A conservative observation conduct and/or diagnostic expectation is taken, with the consequent saturation of the intensive care unit that loses its critical character and avoids quick mobilization of the patient with an increase in costs. The result whish was found that the clinical judgment, a meticulous clinical history, and careful physical examination play a key role in the differential diagnosis of the precordial pain syndrome; however, pain can be atypical, absent or manifest as an equivalent of pain, which does not exclude the diagnosis of myocardial infarction or ischemia. Likewise, chest pain in the presence of a normal conventional ECG at rest, non-diagnostic or with minimal variations, does not rule out the possibility of a coronary obstruction and does not mean that the pain is not of coronary origin. Other characteristics of the ECG, such as T wave and ST segment alterations, bundle branch block (BBB), LV hypertrophy, interpretation discrepancies, can pose doubts or mistakes in the diagnosis. Although its diagnostic information is essential, other non-invasive laboratory tests are needed, such as the treadmill stress ECG, serial bioenzymatic markers, and myocardial perfusion scintigraphy (SPECT and Gated-SPECT) at rest or under physical or pharmacologic stress. The conclusion which was found it was necessary to modified the clinical educational patterns and to revaluate the advantages and limitations of the clinical history,

physical exploration, as well as of the conventional ECG at rest and other diagnostic methods used specifically in relation to the chest pain syndrome with a normal or non diagnostic conventional ECG. SPECT and Gated-SPECT scintigraphy is considered as the best individual and isolated non-invasive test for the diagnostic solution of the precordial syndrome at the Emergency Unit.

Candell-Riera J et al 2004 studied the early myocardial perfusion gated-SPECT in patients with chest pain and non-diagnostic ECG in the emergency department. The aim of their study was to analyze the value of early resting myocardial perfusion gated-SPECT in patients with chest pain and non-diagnostic ECG in the emergency department. The methodology were used 222 patients (49% women, mean age 61 [13] years) with atypical chest pain and with non-diagnostic ECG were randomized into two groups. Group A comprised 111 patients in whom early resting myocardial perfusion gated-SPECT (<6 hours since the end of chest pain) was performed and CK-MB mass and troponin I were determined at 0, 4 and 8 hours. Group B comprised 111 patients with conventional management in the emergency department without gated-SPECT. Results which were found that myocardial perfusion gated-SPECT was positive in all 8 patients with increased levels of CK-MB mass and troponin I. This corresponded to a sensitivity and a negative predictive value of 100% for the diagnosis of AMI. Specificity was 84% and positive predictive value was 33% when doubtful results were considered as negative. The number of patients admitted (18.4% vs 32.7%, P<.027) and length of stay (13 [6] hours vs 15.9 [8.6] hours, P<.009) in the emergency department were lower in group A. they conclude that in patients with atypical chest pain and non-diagnostic ECG in the emergency department, early resting gated-SPECT was highly sensitive and showed good negative predictive value for the diagnosis of

AMI, but positive predictive value was low. This technique may reduce the number of hospitalized patients and length of stay in the emergency department.

Elhendy A1, 2004 describe the Incidence and predictors of heart failure during long-term follow-up after stress Tc-99m sestamibi tomography in patients with suspected coronary artery disease. The aim of their study was to define the incidence and predictors of heart failure during long-term follow-up in patients with suspected CAD referred for stress myocardial perfusion imaging. The methodology used was 787 patients (mean age, 57 +/- 12 years; 470 men) with suspected CAD who had no history of previous myocardial infarction or heart failure with exercise (n = 508) or dobutamine (n = 279) stress technetium 99m sestamibi single photon emission computed tomography. Patients were followed up for the occurrence of heart failure, nonfatal myocardial infarction, and death. The result which found was an abnormal perfusion scan (reversible or fixed perfusion defect) was detected in 341 patients (43%). During a mean follow-up of 6.7 +/- 2.3 years, heart failure occurred in 46 patients (6%), 170 patients (22%) died, and 52 patients (7%) had nonfatal myocardial infarction. Patients in whom heart failure developed were older (mean age, 60 +/- 12 years vs 56 +/- 12 years; P = .01) and were more likely to be men (34 [74%] vs 436 [59%], P = .01) and to have an abnormal scan (32 [70%] vs 309 [42%], P = .0002) compared with patients without heart failure. Nonfatal myocardial infarction occurred before the onset of heart failure in only 3 patients (7%). By multivariate analysis, predictors of heart failure were age (risk ratio [RR], 1.04 [95% CI, 1.01-1.08]), male gender (RR, 2 [95% CI, 1.3-4.5]), resting heart rate (RR, 1.1 [95% CI, 1.05-1.2]), and abnormal scan (RR, 2.3 [95% CI, 1.4-3.9]). The annual mortality rate was 15% after the diagnosis of heart failure. The conclusion was In patients with suspected CAD and no history of myocardial infarction, late heart failure is predicted by age, gender, resting heart rate, and abnormal

perfusion, and it is associated with a substantial mortality rate. The majority of heart failure events are heralded by perfusion abnormalities on sestamibi single photon emission computed tomography but not by an earlier myocardial infarction

Jeanine E et al 2002 describe the Risk factors for coronary heart disease: implications of gender,the aim of the study was toshow the role of cardiovascular risk factors focusing on the differential impact they might have on men and women. They found that the role of a number of risk factors with emphasis on possible differences between men and women. Except for female hormonal status, no risk factor has been recognized as acting on one gender but not on the other. This finding indicates that the pathogenesis of CHD is very similar for men and women. Yet, diabetes, HDL and triglycerides levels have been found to have a greater impact on CHD risk in women compared to men. In addition, there are indications that risk factors such as smoking, family history and inflammation characterized as C-reactive protein, have a more negative influence on CHD in women than in men. On the other hand the evidence showing that lipoprotein(a) is a cardiovascular risk factor seems to be stronger in men than in women. The majority of cardiovascular risk factors show no important differences between the genders. For optimal treatment and prevention of CHD it is necessary to acknowledge that it is not self-evident that women and men show a similar response to risk factors or to treatment. Therefore, it is essential that studies present results according to gender, in order to comprehend to what extent CHD prevention measures are similar for men and women.

TaliSharir et al 1999 describe the Incremental Prognostic Value of Post-Stress Left Ventricular Ejection Fraction and Volume by Gated Myocardial Perfusion Single Photon Emission Computed Tomography. The aim of the study was to evaluate whether post-stress EF and left

ventricular volume, measured by gated myocardial perfusion SPECT, had incremental prognostic value over clinical exercise, and perfusion data in predicting cardiac death in a large, unselected population of patients referred for nuclear testing. The methodology used was 1924 consecutive patients who underwent separate acquisition dual-isotope myocardial-perfusion gated SPECT (rest Tl-201/stress Tc-99m sestamibi gated SPECT) at Cedars-Sinai Medical Center and who were followed-up $1 year for cardiac events. Patients with nonischemic cardiomyopathy (EF,45% and prescan likelihood of CAD,0.40) were excluded (n515), and patients revascularized within 60 days after nuclear testing (n5229) were censored from the prognostic portion of the analysis which left 1680 patients (1029 underwent treadmill exercise and 651 adenosine stress testing). Normal limits of post-stress left ventricular volume and EF were determined in 44 patients (22 men and 22 women aged 53610 years) with a low prescan likelihood (,5%) of CAD. Reslts which their found were Receiveroperator characteristics analysis defined an EF,45%, an end-systolic volume (ESV) .70 mL, and an end-diastolic volume .120 mL as optimal thresholds, yielding moderate sensitivity and high specificity in the prediction of cardiac death. Patients with an EF$45% had mortality rates ,1%/year, despite severe perfusion abnormalities, whereas patients with an EF,45% had high mortality rates, even with only mild/moderate perfusion abnormalities (9.2%/year; P,0.00001). Similarly, an ESV#70 mL was related to a low cardiac death rate (,1.2%/year), even for patients with severe perfusion abnormalities, whereas patients with an ESV.70 mL and only mild/moderate perfusion abnormalities had high death rates (8.2%/year; P,0.00001). Patients with an EF,45% and an ESV#70 mL had low cardiac death rates (1.7%/year); those with an EF,45% but an ESV.70 mL had high death rates (7.9%/year; P,0.02). Multivariate Cox proportional hazards regression showed that perfusion variables and ESV were independent predictors of overall coronary events, whereas EF and ESV demonstrated

incremental prognostic values over prescan and perfusion information in predicting cardiac death and cardiac death or myocardial infarction. The conclusion was Post-stress Tc-99m sestamibi gated SPECT provides incremental prognostic information in patients with known or suspected CAD that is better than perfusion data alone. Although perfusion variables are powerful in predicting worsening of coronary disease, post-stress EF and ESV provide incremental value in the prediction of cardiac death. Therefore, the information provided by gated SPECT should be considered in the referral of patients for coronary angiography and revascularization.

Soman P et al 1999 described the prognostic value of a normal Tc-99m sestamibi SPECT study in suspected coronary artery disease. The aim of their study was to assess the incidence of cardiac death and non-fatal myocardial infarction in patients with an intermediate probability of coronary artery disease (CAD). The methodology was used for 473 patients with normal stress Tc-99m-sestamibi SPECT were monitored for 30+/-16 (6 to 56) months to assess serious cardiac events. There were 272 men and 201 women, with a mean age of 56+/-2 years, of whom 89% had symptoms suggestive of CAD, 65% had an abnormal exercise electrocardiography, 6% had known CAD, and 5% had a high risk of CAD. The average workload was 9.14 metabolic equivalents, peak exercise heart rate was 93%+/-13% of the age predicted target. The result found was the annualized mortality rate was 0.2% (95%CI 0.02% to 0.7%) and no infarctions occurred in this group. The conclusion was a normal stress Tc-99m-sestamibi is highly predictive of a benign outcome, even in patients with intermediate probability of CAD.

Lloyd-Jones DM et al 1999 described the Lifetime risk of developing coronary heart disease. The aim of the study was to show the lifetime risks of initial coronary events at different ages. The methodology was assessed data for 7733 participants in the Framingham Heart Study, who

had been examined at least once at age 40-94 years between 1971 and 1975, found to be free of coronary heart disease, and then followed up. They estimated the lifetime risks of coronary heart disease (angina pectoris, coronary insufficiency, myocardial infarction, or death from coronary heart disease) by multiple-decrement life-table methods.The result found wasthe 7733 patients were followed up for a total of 109,948 person-years. Overall, 1157 participants developed coronary heart disease. 1312 died from non-coronary heart disease causes. Lifetime risk of coronary heart disease at age 40 years was 48.6% (95% CI 45.8-51.3) for men and 31.7% (29.2-34.2) for women. At age 70 years, lifetime risk was 34.9% (31.2-38.7) for men and 24.2% (21.4-27.0) for women. After we excluded isolated angina pectoris as an initial event, the lifetime risk of coronary artery disease events at age 40 years was 42.4% for men and 24.9% for women.The conclusion was that the Lifetime risk at age 40 years is one in two for men and one in three for women. Even at age 70 years it is one in three for men and one in four for women. This knowledge may promote efforts in education, screening, and treatment for prevention of coronary heart disease in younger and older patients.

Taillefer R et al 1997described the Comparative diagnostic accuracy of Tl-201 and Tc-99m sestamibi SPECT imaging (perfusion and ECG-gated SPECT) in detecting coronary artery disease in women. The aim of the study was to compare the sensitivity and specificity of thallium-201 (Tl-201), technetium-99m (Tc-99m) sestamibi perfusion and Tc-99m sestamibi electrocardiographic (ECG)-gated single-photon emission computed tomographic (SPECT) studies for detection of coronary artery disease (CAD). The methodology was Eighty-five patients with suspected CAD, scheduled for coronary angiography, and 30 volunteers with a pretest likelihood of < or = 5% for CAD were evaluated. Within 1 week, each patient underwent Tl-201 and Tc-99m sestamibi SPECT imaging procedures (both perfusion and gated SPECT

imaging). Treadmill stress testing was used in 78 patients and dipyridamole in the remaining 37 patients. All images were interpreted by three observers in a blinded manner (consensus reading). Technetium-99m sestamibi SPECT studies were read without and then with ECG gating. Technetium-99m sestamibi gated SPECT studies were used to differentiate scar tissue from soft tissue attenuation artifact. The result which their found were the overall sensitivities for detecting > or = 50% and > or = 70% stenoses were 75.0% and 84.3%, respectively, for Tl-201, and 71.9% and 80.4%, respectively, for Tc-99m sestamibi perfusion studies (p = 0.48). The specificity for lesions > or = 50% was 61.9% for Tl-201 and 85.7% for Tc-99m sestamibi perfusion (p = 0.07), whereas for lesions > or = 70% it was 58.8% for Tl-201 and 82.4% for Tc-99m sestamibi perfusion (p = 0.01). When the 34 patients with a normal coronary angiogram were added to the group of 30 normal volunteers, the "specificity" for lesions > or = 70% was 67.2% for Tl-201, 84.4% for Tc-99m sestamibi SPECT perfusion (p = 0.02) and 92.2% for Tc-99m sestamibi gated SPECT (p = 0.0004). They concluded to Both Tl-201 SPECT and Tc-99m sestamibi SPECT perfusion studies had a similar sensitivity for the detection of CAD in women. However, Tc-99m sestamibi SPECT perfusion imaging shows a significantly better specificity, which is further enhanced by the use of ECG gating.

Rory Hachamovitch et al 1996 studied the exercise Myocardial Perfusion SPECT in Patients without known Coronary Artery Disease. The goals of this study were to define the statistical incremental prognostic value of exercise stress myocardial perfusion SPECT in a patient population without previously defined CAD and to define the clinical role of the test in risk stratification of this population. The methodology was identified 2268 consecutive patients who underwent exercise dual-isotope SPECT between January 1, 1991, and December 1, 1993, who at the time of their nuclear tests had not undergone cardiac catheterization, coronary artery

bypass surgery, or percutaneous transluminal coronary angioplasty and had no known history of previous myocardial infarction. Patients who underwent pharmacological dual-isotope SPECT or who were known to have valvular heart disease or primary cardiomyopathy were not included in this study. Of the initial population, 68 patients were lost to follow-up, and the remaining 2200 patients with successful follow-up (97%) were included in this study. At the time of nuclear study, all patients completed a questionnaire detailing the characteristics of their presenting symptomology. All patients were then interviewed by a physician who reviewed their responses prior to the initiation of the treadmill portion of their tests. The result which their found were Examination of clinical, exercise, and nuclear models by use of pre–exercise tolerance test (ETT), post-ETT, and nuclear information using a stepwise Cox proportional hazards model and receiver-operating characteristic curve analysis revealed that nuclear testing added incremental prognostic value after inclusion of the most predictive clinical and exercise variables (global χ^2=12 for clinical variables; 31 for clinical+exercise variables; 169 for nuclear variables; gain in χ^2, P<.0001 for all; receiver-operating characteristic areas: 0.66±0.04 for clinical, 0.73±0.04 for clinical+ exercise variables, 0.87±0.03 for nuclear variables, P=.03 for gain in area with exercise variables; P<.001 for increase with nuclear variables). Multiple logistic regression analysis revealed that scan information contributed 95% of the information regarding referral to catheterization with further additional information provided by presenting symptoms and exercise-induced ischemia. Referral rates to early catheterization and revascularization paralleled the hard event rates in all scan categories−very low referral rates in patients with normal scans and significant increases in referral rates as a function of worsening scan results. Even after stratification by clinical and exercise variables such as the Duke treadmill score, pre- and post-ETT likelihood of coronary artery disease, presenting symptoms, sex, and age, the nuclear scan

results further risk-stratified the patient subgroups, thus demonstrating clinical incremental value. The conclusion of this study was reveal that exercise sestamibi myocardial perfusion SPECT adds incremental prognostic information when used in patients who have not undergone previous catheterization or revascularization and have not had previous myocardial infarction and who are at overall low-intermediate risk (1.8% hard event rate, 1.2% per year of follow-up). Further, physicians referred patients to catheterization and revascularization in proportion to the extent and severity of their scan results and, thus, to their risk of cardiac events. In light of this, the effect of testing on patient management appears to be both powerful and appropriate.

Chapter three

Material and Methods

The researcher following the methodology of experimental and practical studies over specific 193 patients suffering from different types of heart diseases referring from different clinics to nuclear medicine department to characterization cardiovascular system function.

All patients who came to the nuclear medicine department to do radionuclide myocardial perfusion scan requested by physician for follow up.

3.1Instrument of data collocation

Gamma camera (dual head SPECT), type digital**,**ADAC leberatryies**,** System, forte,js, AZ SPECT, ¾**,** Gauntry ring motion 26% **,** CE0086

Radiopharmaceutical:

Technetium-99m tracers ,Sestamibi

Technetium has six hours physical half-life with its 140 kev gamma emission.

3.2 study duration

Study was done from August 2013 to August 2017

3.3 Place of Study

Study was conducted at NMDC, Fedial hospital and Riyadh care hospital (KSA).

3.4 Sample size

Sample selection of the study was non unstable patient. 193 patients (104 females and 89 males) with the average age 59.3 year under went to get Tc99m Sestamibi Scintigraphy.

3.5 Method of data collocation

Primary data were collected patients files and interview through question.

MPS with Tc^{99m} Sestamibi which were requested by doctor.

3.5 methodologies

3.5.1Patient preparation

When the patients were referred for myocardial perfusion study. The patients asked for the medication intake if found (stopped for 24hr before the study, as well as to ensure patient NPO 2–14 hours before exam (usually 4–6 hours, 2 hours for infants). And explain the procedures (usually runs ~1 hour but baseline). Inject 20 to 30 mCi at peak exercise. Begin imaging at 15 to 30 minutes after injection during exercise. On day 2, give same dose at rest. Begin imaging at 45 to 60 minutes after injection at rest.

3.5.2 Procedure

Place the patient in supine with heart in center field of view and left arm up over head if possible. If arm is down at side because of problems with shoulder joint or recent surgery, both the rest and stress images should be taken the same way.

Protocol stress: Stress testing the heart organ in myocardial perfusion imaging techniques are through physical exercise and pharmacologic. When stress testing was done, it is necessary to observe the heart rate on the computer check the condition of the patient. Radiopharmaceutical

injection can perform after the heart rate has reached 85 percent. Radiopharmaceutical was injected with the range of dose between 20–30 mCi.

Protocol rest: Patient waits 45–60 minutes after injection of 99mTc-sestamibi, 8–30 mCi before imaging and giving the patient a glass of cold water before imaging to clear thyroid, liver, and bowel. Patient was in the supine position with heart in center field of view and left arm up over head. If arm is down at side because of problems with shoulder joint or recent surgery, both the rest and stress images should be taken the same way. Images may include a static anterior picture first (300 seconds). Start SPECT images at appropriate time with camera right anterior oblique to left posterior oblique (Except for patients with dextrocardia).

Scanning and acquisition SPECT: Scanning was done with dual head SPECT gamma camera parameters Cardiac Gated SPECT. This study were applied parameters zoom 1.46 x (40.9) cm, matrix size 64 x 64, the number of angles 64, time per angle 20 seconds, the saturation level of 32.767, the relative angle detector 900 , the initial angle of 450 , and the direction of rotation clockwise. SPECT gamma camera detector was positioned on the patient's body left chest. The detector was rotated 180 degrees from the position of 45 degrees right anterior oblique (RAO) to 45 degrees left posterior oblique (LPO).

3.6 Variable of data collocation

The data would be taken practically and from reports of the patient, Social background about patient (age, gender, BMI), Disease background (symptoms and signs, and type of disease), and the (end-diastolic, end-systolic and ejection fraction of both rest and stress study).

3.7 Method of data analysis

Data was entered and analyzed by using Microsoft Excel and, statistical professional for social science program (SPSS)

Chapter four

Results

This chapter presents the result which consisted of tables and figures showed the statistical parameters for all patients at age, BMI, End-Diastolic Volume, End-Systolic Volume and Ejection fraction, the frequency and percentage of the patients, the frequency and percentage of the history for all patientsand the correlation between the rest and stress at EDV, ESV and EJ.

Table 4.1: show statistical parameters for all patients

	Mean	***Median***	***SD***	***Min***	***Max***
Age	**60.31**	**62**	**11.59**	**39**	**85**
BMI	**26.85**	**25**	**7.04**	**18**	**45**
Stress EDV	**94.58**	**85**	**39.59**	**33**	**236**
Stress ESV	**40.32**	**31**	**27.35**	**12**	**128**
Stress EF	**59.59**	**65**	**15.41**	**27**	**88**
Rest EDV	**98.81**	**68**	**37.55**	**50**	**206**
Rest ESV	**51.17**	**35**	**49.24**	**16**	**348**
Rest EF	**55.93**	**59**	**13.78**	**31**	**87**

Table 4.2: show the frequency and percentage of the patients

Gender	Frequency	Percent
Male	89	46
Female	104	54

Table 4.3: show the frequency and percentage of the history for all patients

History	Frequency	Percent
None	3	2.3
Chest Pain	19	14.6
IHD	10	7.7
DM & Hypertension	6	4.6
Inferior MI	7	5.4
Shortness of breath	2	1.5
CABG	7	5.4
CKD	2	1.5
Angina	3	2.3

Table 4.4: show statistical parameters for all patients at End-Diastolic Volume, End-Systolic Volume and Ejection fraction:

Rest	*Mean*	*SD*	*Stress*	*Mean*	*SD*
EDV	*98.81*	*37.55*	*EDV*	*94.58*	*27.35*
ESV	*51.17*	*49.24*	*ESV*	*40.32*	*13.78*
EJ	*55.93*	*13.78*	*EJ*	*59.59*	*15.41*

Table 4.5 show correlation between the rest and stress at EDV, ESV and EJ

***Paired* Samples Correlations**			
		Correlation	***P.value***
Pair 1	***Rest EDV & Stress EDV***	***0.795***	***.000***
Pair 2	***Rest ESV & Stress ESV***	***0.513***	***.000***
Pair 3	***Rest EJ & Stress EJ***	***0.716***	***.000***

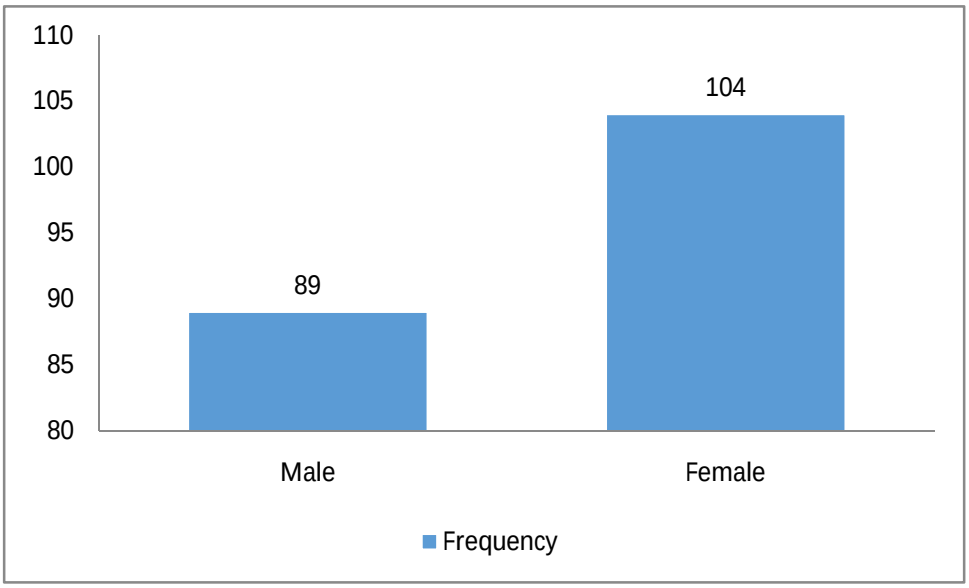


Figure 4.1: show the frequency and percentage of gender to the patients

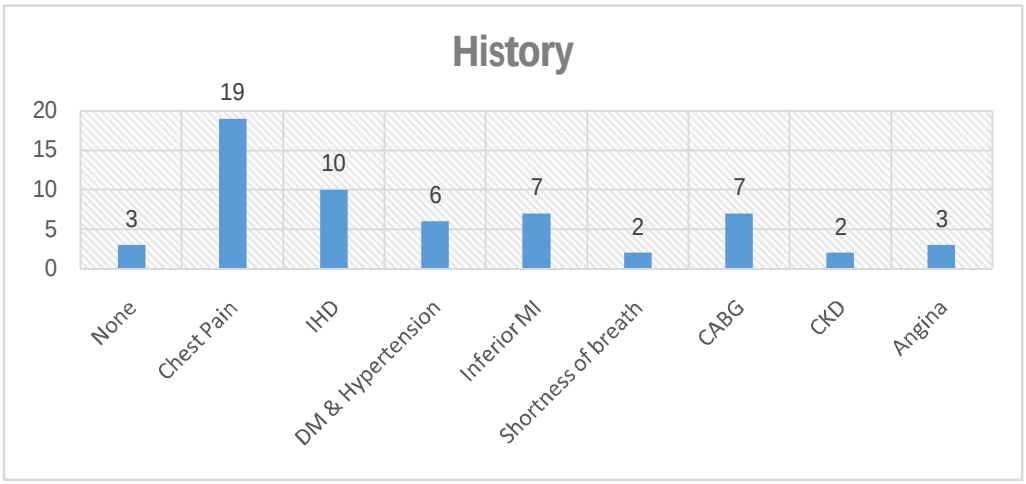


Figure 4.2: show the frequency and percentage of the histories for all patients

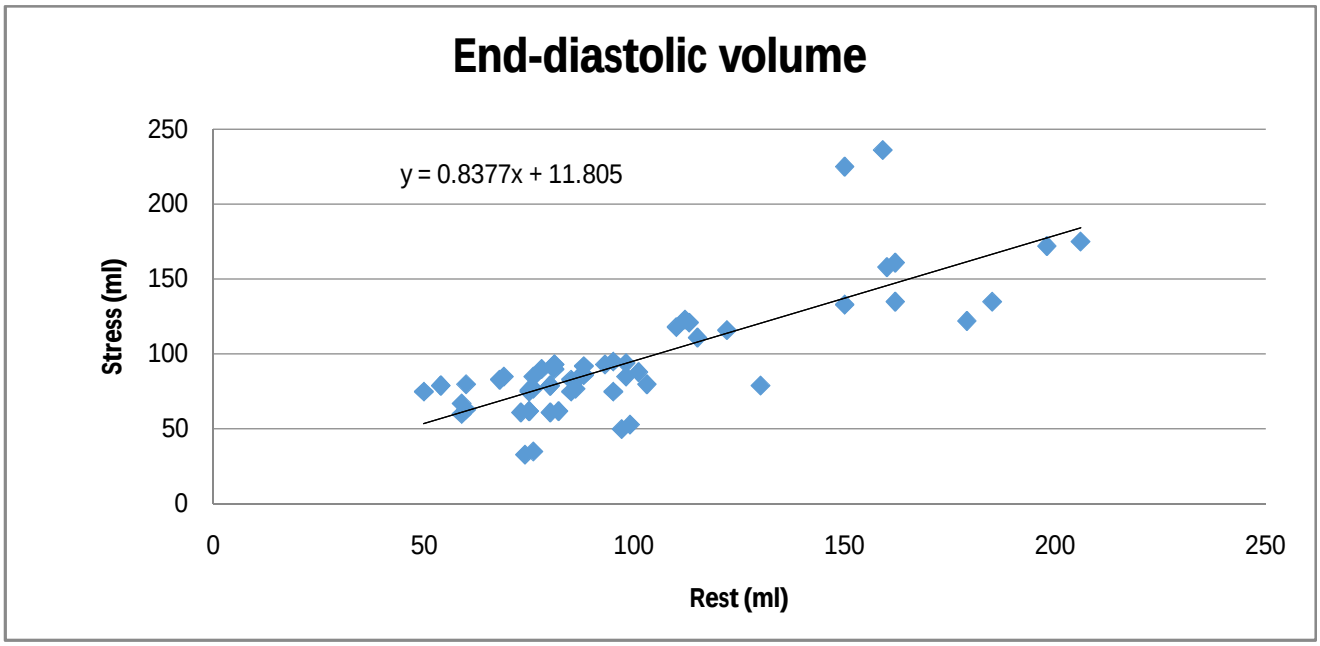


Figure 4.3: end-diastolic curve rest and stress

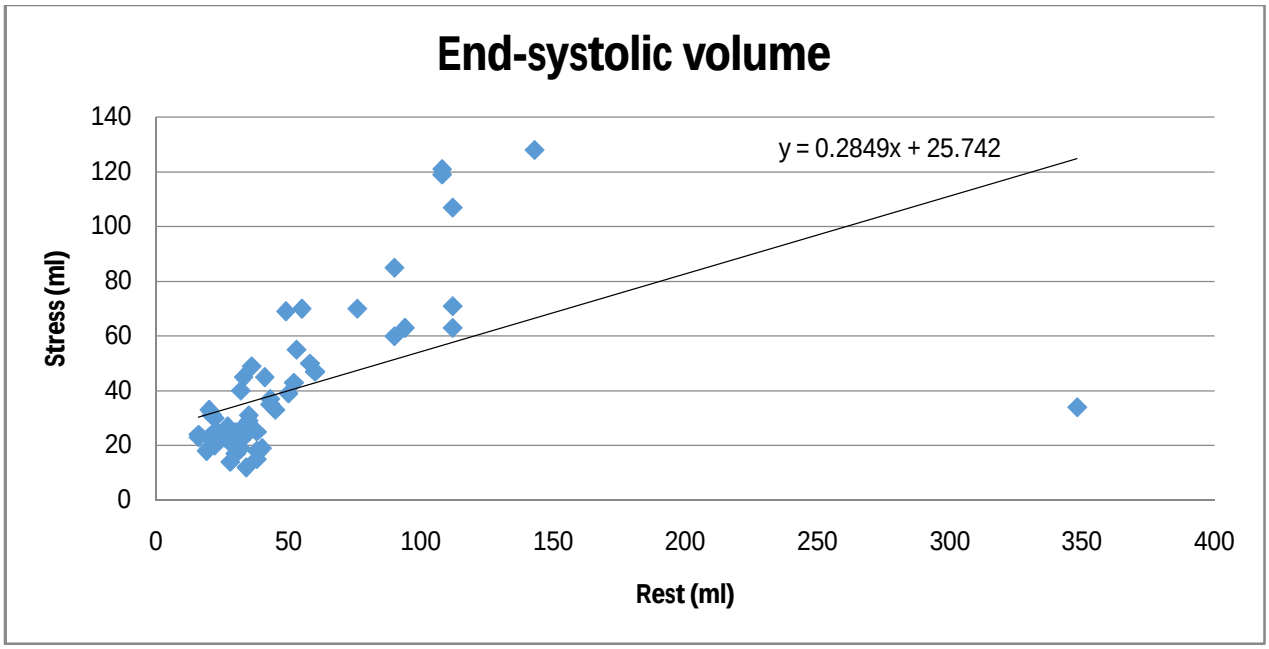


Figure 4.4: end-systolic curve rest and stress

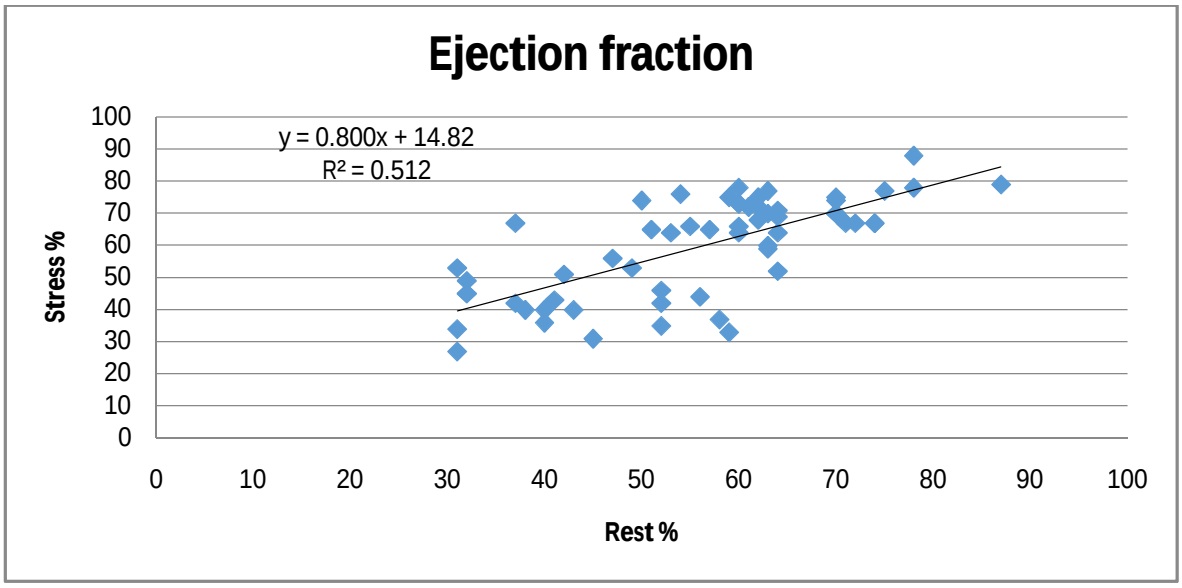


Figure 4.5: ejection fraction curve rest and stress

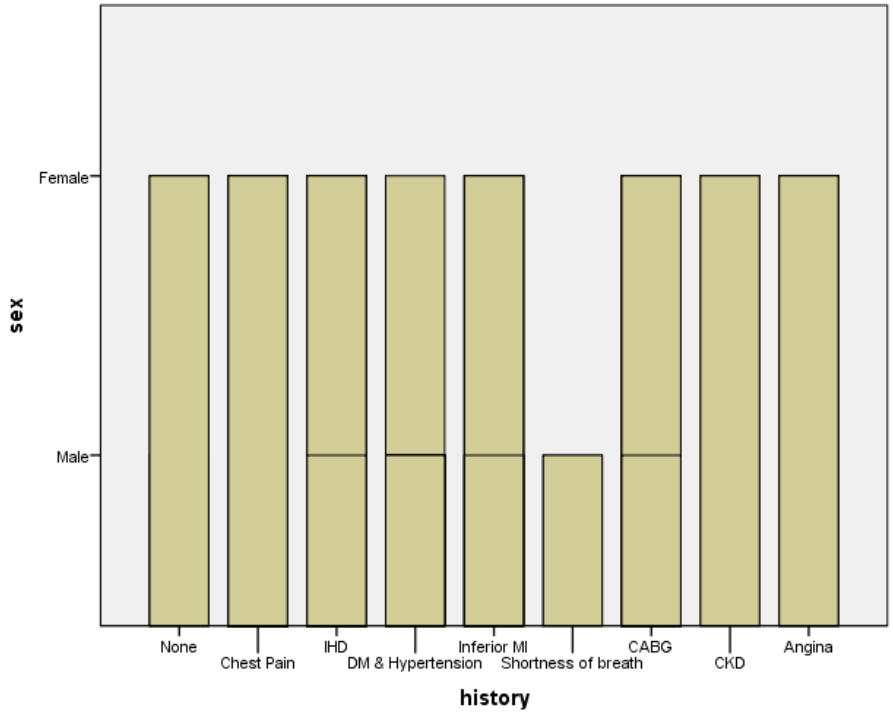


Figure 4.6: show the correlation between histories and sex

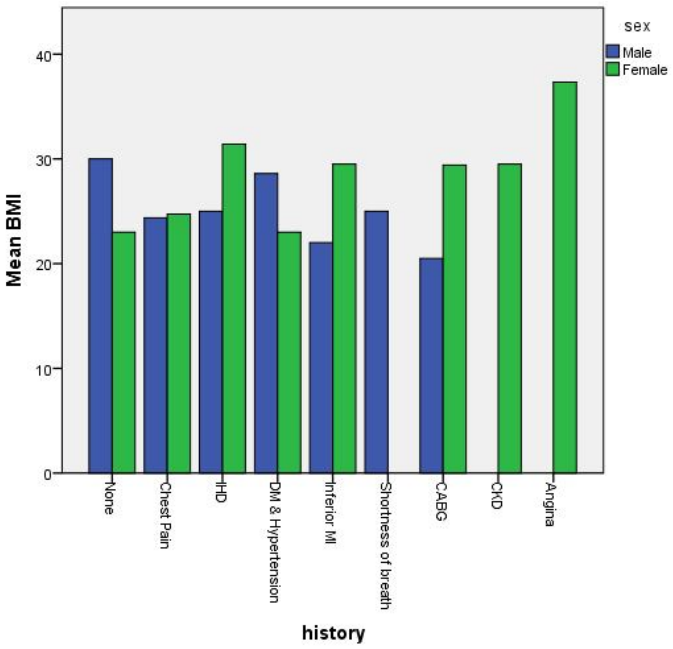


Figure 4.7: show the correlation between histories and mean BMI

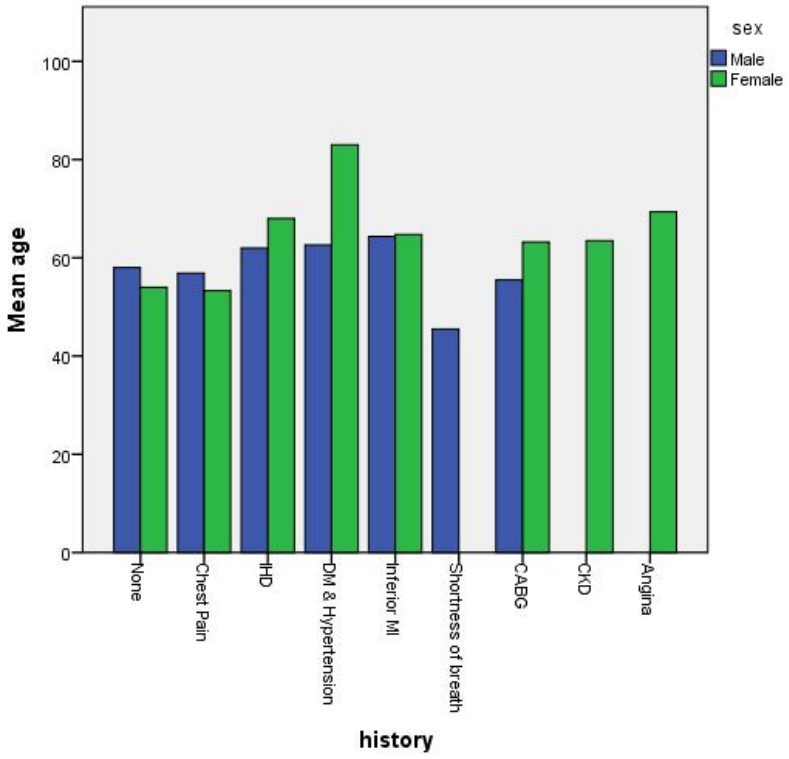


Figure 4.8: show the correlation between histories and mean ages

Chapter five

Discussion and Recommendation

5-1 Discussion

This study carried out to this study is to Estimation of myocardial perfusion in SPECT Scintigraphy using quantitative analysis.

In table 4.1 show statistical parameters for all patients as mean ±SD, median, in the age was 60.31±11.59 years and at the body mass index was 26.85±7.04 kg/cm^2 and at the stress end-diastolic volume was 94.58±39.59ml and at stress end-systolic volume was 40.32±27.35ml and the percentage of stress ejection fraction was 59.59±15.41 and at rest end-diastolic volume was 98.81±37.55 ml and at rest end-systolic volume was 51.17±49.24 ml and the percentage of rest ejection fraction was 55.93±13.78

In table 4.2 show the frequency and the percentage of the gender, male frequency was 26 with percent 20% and female was frequency 33 with percent 25%. Same as figure 4.1.

In table 4.3 show the frequency and percentage of the histories for all patients , patients without history was frequency 3 with percent 2.3% and chest pain was frequency 19 with percent 14.6% and IHD was frequency 10 with percent 7.7% and DM& hypertension was frequency 6 with percent 4.6% and inferior MI was frequency 7 with percent 5.4% and shortness of breath was frequency 2 with percent 1.5% and CABG was frequency 7 with percent 5.4% and CKD was frequency 2 with percent 1.5% and angina was frequency 3 with percent 2.3%.Same as figure 4.2

In table 4.4 show statistical parameter for all patients at the three mode of blood perfusion as mean ±SD in the end-diastolic volume at rest was 98.81±37.55 and at stress was 94.58±27.35, in

end-systolic volume at rest was 51.17±49.24 and at stress was 40.32±13.78 and in ejection fraction at rest was 55.93±13.78 and at stress was 59.59±15.41.

In table 4.5 show paired sample (t,test) show the correlation between the rest and stress at three mode of blood perfusion.in paired one the correlation vale between rest and stress at end-diastolic volume was 0.795, in paired two the correlation vale between rest and stress at end-systolic volume was 0.513 and in paired three the correlation vale between rest and stress at ejection fraction was 0.716, and at the three blood perfusion mode the p.value show p.value 0.00 which mean there is no significant difference at end-diastolic volume, end-systolic volume and ejection fraction.

In figure 4.3 shows there was a linear regression between the rest and stress at end-diastolic volume, the rate of change in stress was 0.8377 at each ml from rest.

In figure 4.4 shows there was a linear regression between the rest and stress at end-systolic volume, the rate of change in stress was 0.2849 at each ml from rest.

In figure 4.5 shows there was a linear regression between the rest and stress at ejection fraction percent, the rate of change in stress was 0.8004 at each percent from rest.

In figure 4.6 shows correlation between the history of patients disease with the patients gender were the number of history was 9 disease contributed as the all normal patients was a female same as the patients with chest pain, for ischemic heart disease was for both gender similar to diabetic maleates and hypertension and as same with inferior myocardia infraction and CABG, for the patients with shortness of breath was just a male, and the patients with chronic kidney disease and angina just with females patient.

In figure 4.7 show correlations between the history of patient's disease and BMI according to gender, in normal patients, diabetes maleates and hypertension the males was higher in BMI than

females, and at ischemic heart disease and inferior myocardial infarction the females was higher in BMI than males, and in chest pain the BMI consider the same in males and females. But for the patient with shortness of breath the BMI represented only in males patients and for the patient with chronic kidney disease and angina, the BMI represented only females' patient.

In figure 4.8 show correlation between history and mean age according to gender, in normal patient and chest pain the males was higher in mean age than females and at ischemic heart disease, diabetes maleates and CABG the females were higher in mean age than males and at inferior myocardial infarction the mean age in males and females were the same. But in patients with shortness of breath the mean age represented only in males, and for the chronic kidney disease the and angina the mean age only represented in females

5.2 Conclusion

Heart disease is one of the cause's numbers of death in the world, If the heart could not pump blood and distribute it to all parts of the body, pain in the chest will happen while heavy work or walk a rush. Myocardial perfusion imaging (MPI) techniques is an ideal approach to doing the assessments of myocardial perfusion as non-invasive imaging modalities; it can examine the functions of the various segments of the heart muscle to reflect whether there is a malfunction of the heart.

the result shows that Estimation of stress myocardial perfusion characterize to three mode of blood perfusion (end-diastolic, end-systolic volumes and ejection fraction), and estimated well by the linear equation for the three mode of blood perfusion, and from the patients information such as age and body mass index can estimate the rest myocardial perfusion from patient's information without need it to rest scan by 99mTc sestamibi.

And the estimation of rest from patient information and stress form rest done by regression equation:

Rest EDV= Age × (-0.090) +BMI × (1.570) – 62.105

Rest ESV = Age × (0.844) + BMI × (1.642) – (-43.819)

Rest EF = Age × (-0.227) + BMI × (-1) – 96.452

Stress End-diastolic Volume = 0.8377 Rest + 11.805

Stress End-systolic Volume = 0.2849 × Rest + 25.742

Stress Ejection Fraction = 0.8004 × Rest + 14.823

5.3 Recommendation

- It is worth recommended to widen the sample size to include different types of heart disease.
- To improve the validity of the study and ensure that the findings can be determined rich data, a full previously validated questionnaire could be used to collect both Coronary Heart Disease knowledge and historical background.
- Knowing the prevalence of various modifiable risk factors may help in planning appropriate secondary preventive programs to target the different age groups. Emphasis for the elderly population should be more targeted at better control of hypertension.
- Patients with suspected or confirmed myocardial disease should be cared for by staff trained and experienced in modern care.
- Patients and their associates should be informed of how to recognize and respond to a further heart attack.
- Single photon emission computed tomography (SPECT) could be acquired with special modifications to provide improved visualization of regional heart function.

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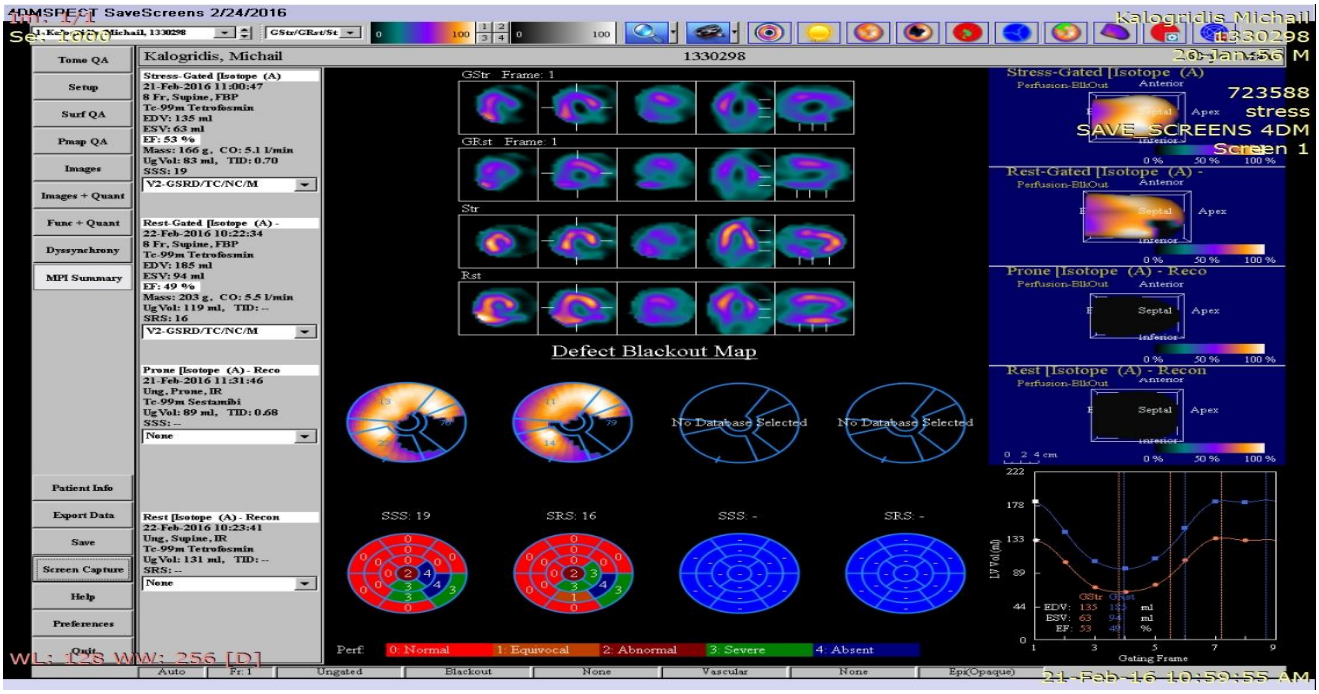
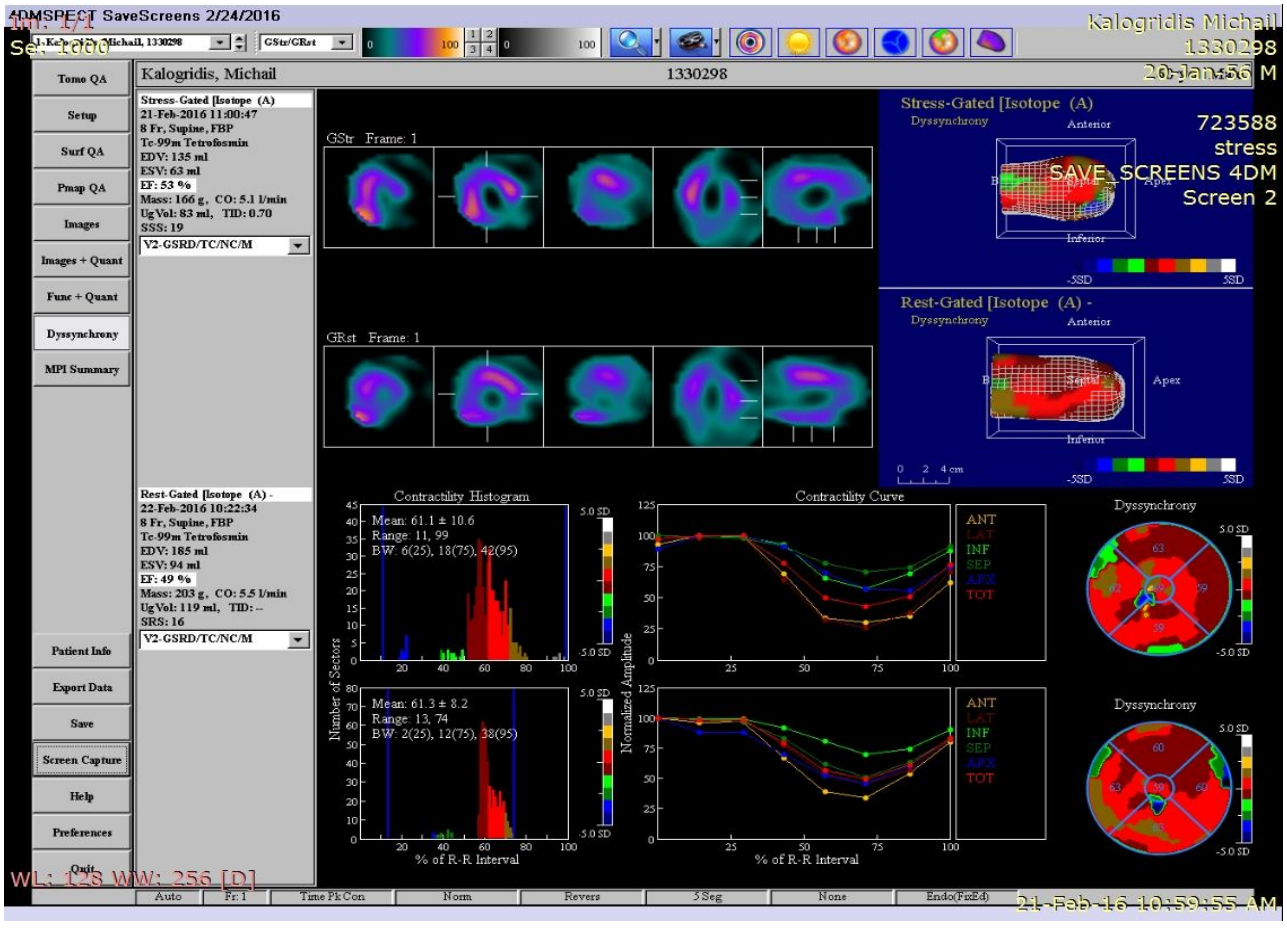
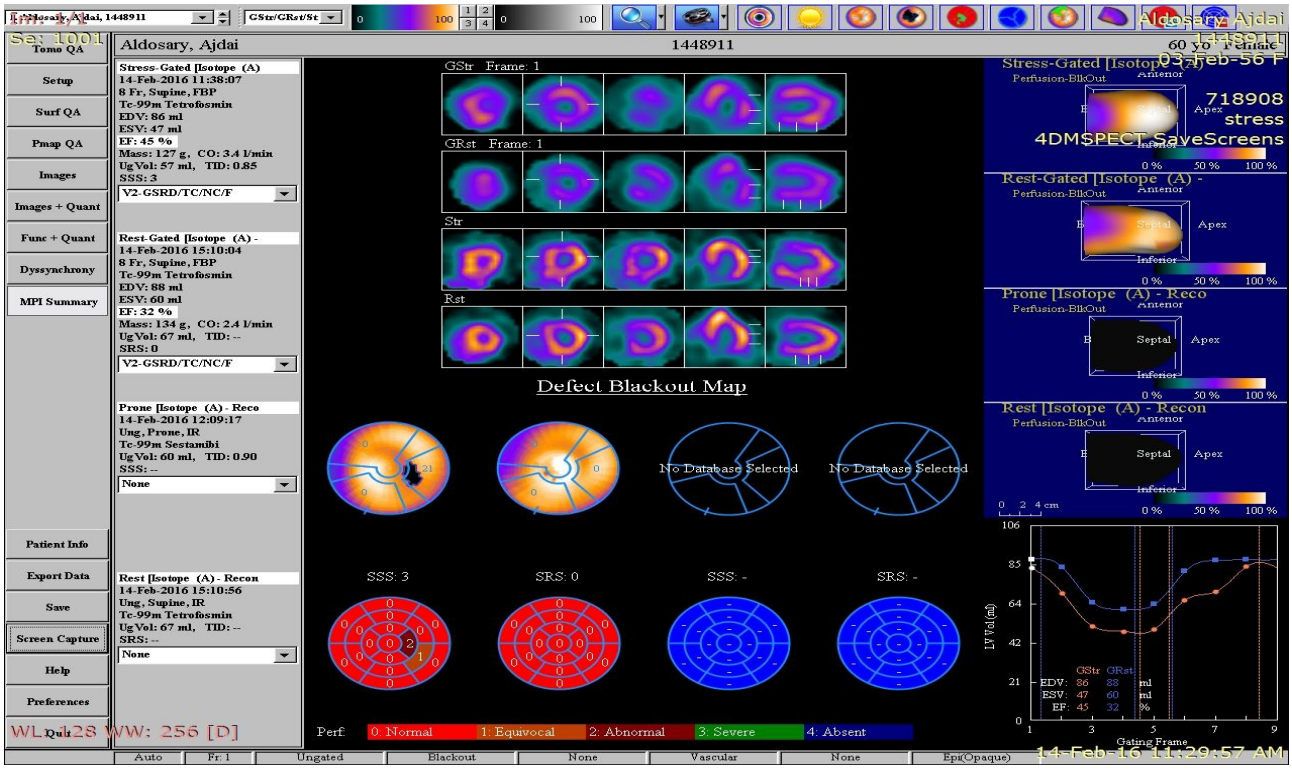
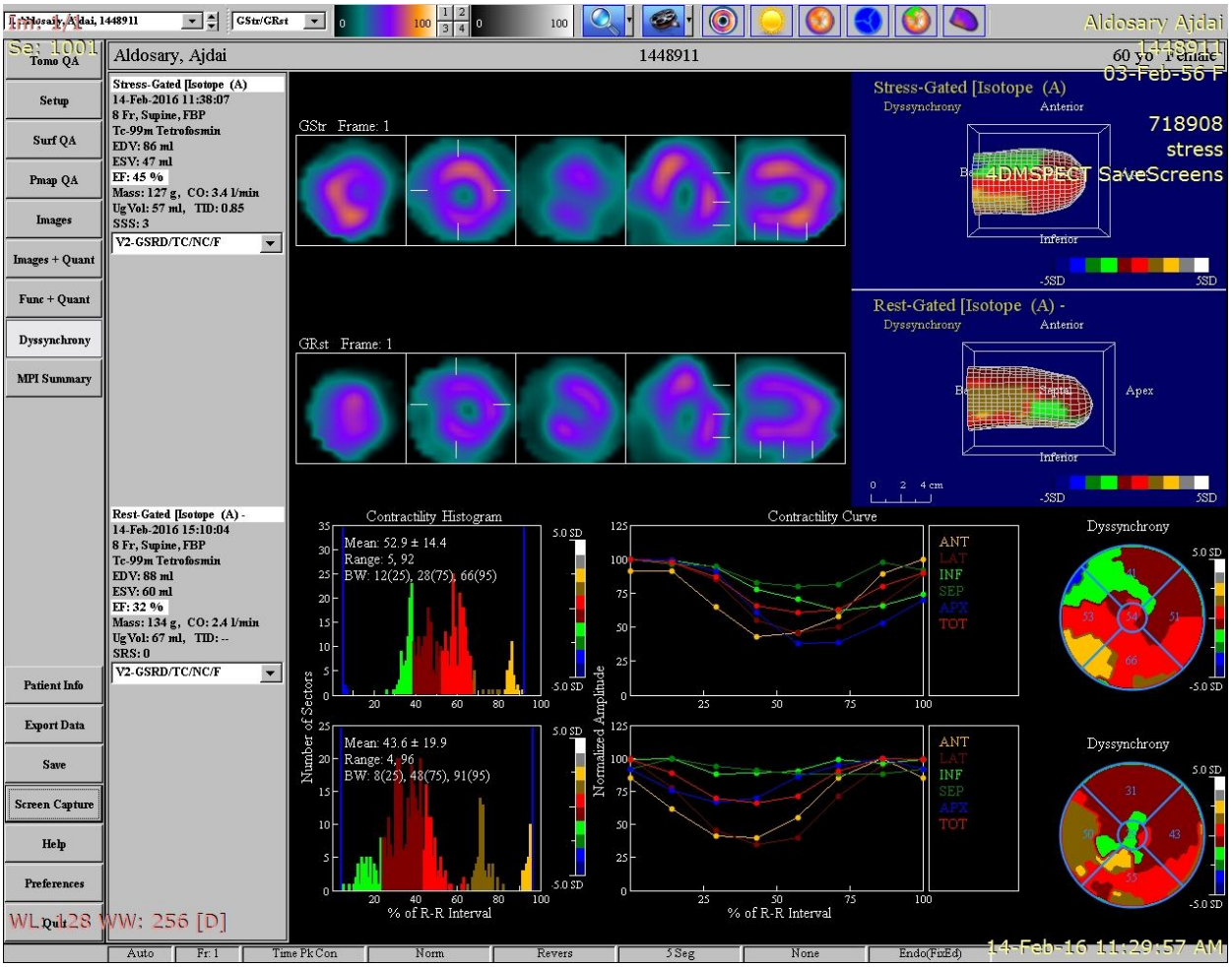
Appendix

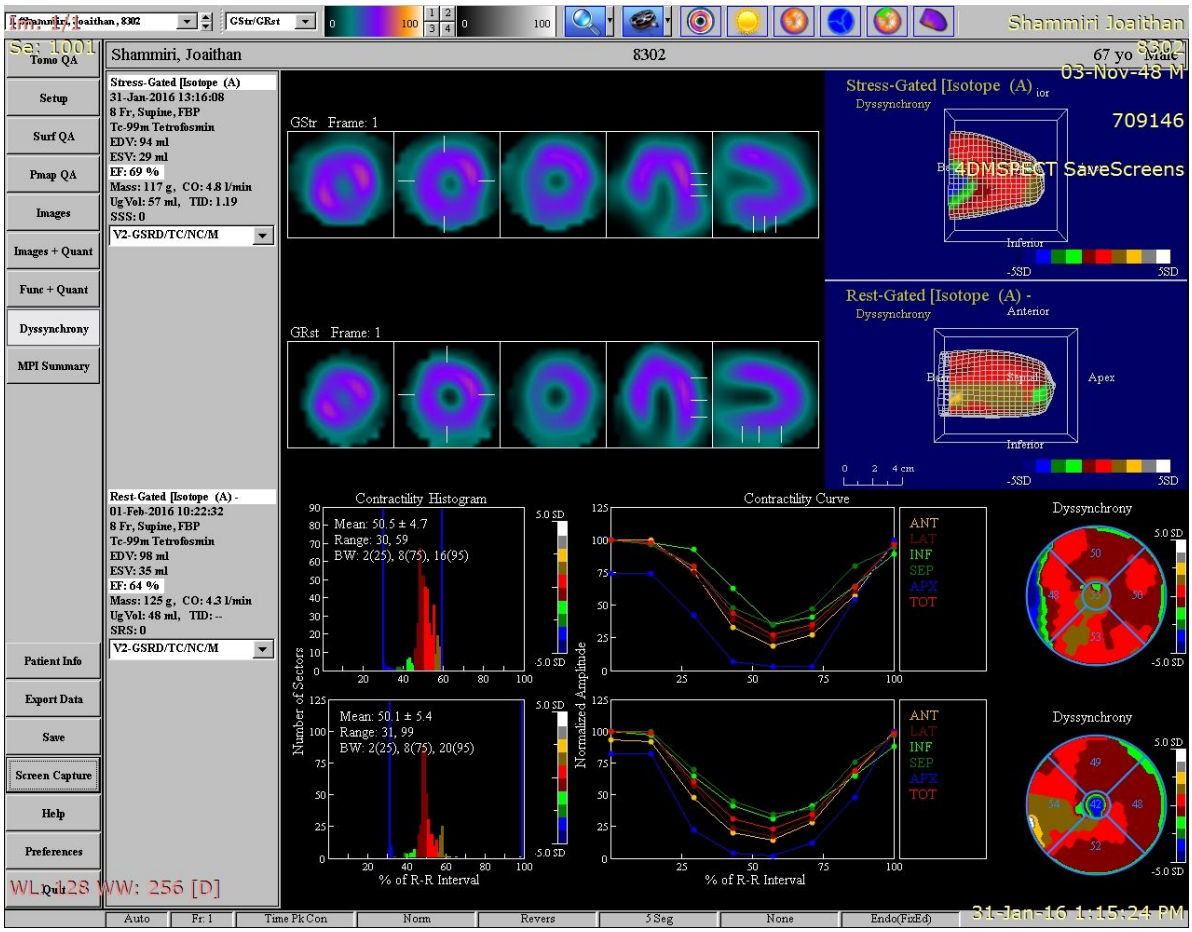
Images of myocardial perfusion scan (MPI)

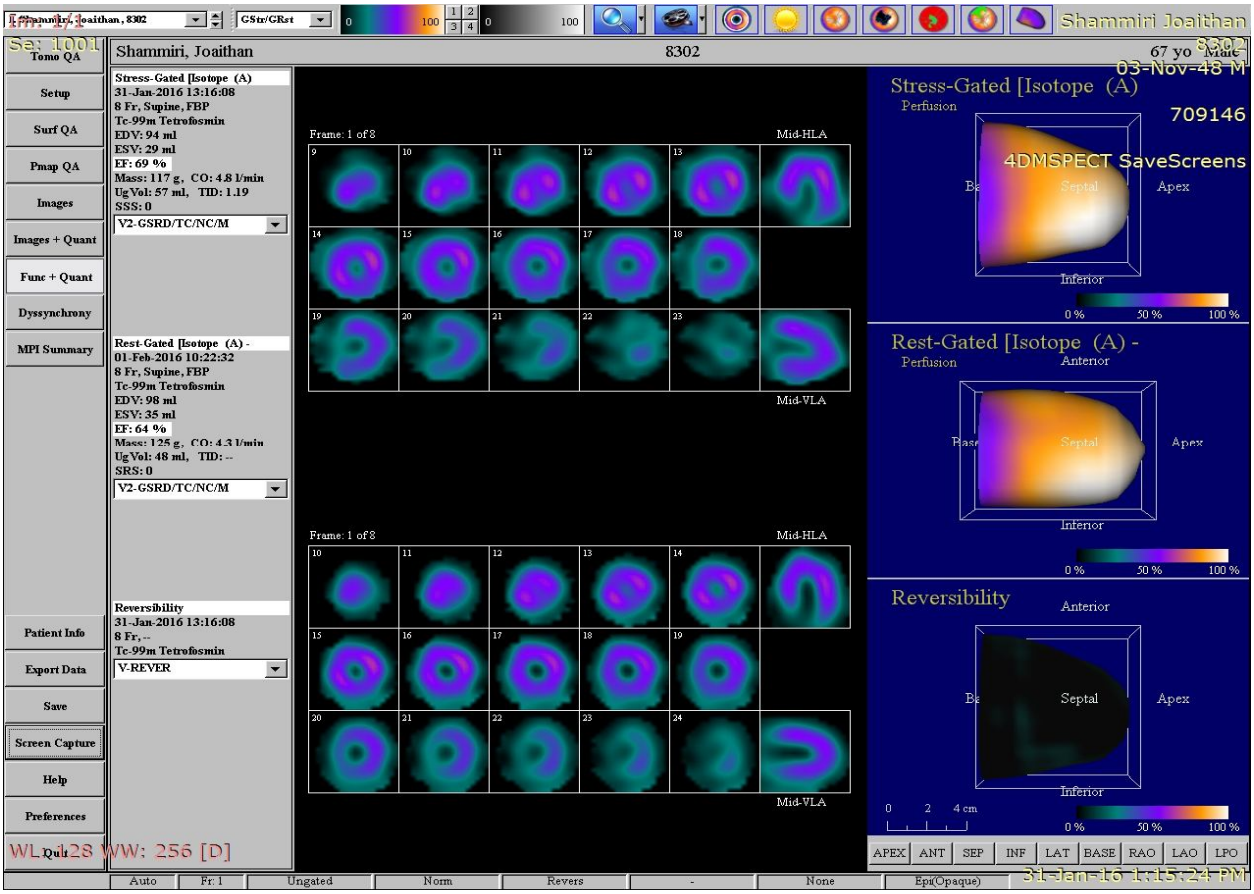





Aldosary, Ajdai
1448911
60 yo Female
03-Feb-56 F
Stress-Gated [Isotope (A)
718908
stress
4DMSPECT SaveScreens
Rest-Gated [Isotope (A) -
Prone [Isotope (A) - Reco
Rest [Isotope (A) - Recon
Defect Blackout Map
No Database Selected
No Database Selected
SSS: 3
SRS: 0
SSS: -
SRS: -
Perf: 0: Normal 1: Equivocal 2: Abnormal 3: Severe 4: Absent
Stress-Gated [Isotope (A)
14-Feb-2016 11:38:07
8 Fr, Supine, FBP
Tc-99m Tetrofosmin
EDV: 86 ml
ESV: 47 ml
EF: 45 %
Mass: 127 g, CO: 3.4 l/min
UgVol: 57 ml, TID: 0.85
SSS: 3
Rest-Gated [Isotope (A) -
14-Feb-2016 15:10:04
8 Fr, Supine, FBP
Tc-99m Tetrofosmin
EDV: 88 ml
ESV: 60 ml
EF: 32 %
Mass: 134 g, CO: 2.4 l/min
UgVol: 67 ml, TID: --
SRS: 0
Prone [Isotope (A) - Reco
14-Feb-2016 12:09:17
Ung, Prone, IR
Tc-99m Sestamibi
UgVol: 60 ml, TID: 0.90
Rest [Isotope (A) - Recon
14-Feb-2016 15:10:56
Ung, Supine, IR
Tc-99m Tetrofosmin
UgVol: 67 ml, TID: --
Tomo QA
Setup
Surf QA
Pmap QA
Images
Images + Quant
Func + Quant
Dyssynchrony
MPI Summary
Patient Info
Export Data
Save
Screen Capture
Help
Preferences
Quit
WL: 128 WW: 256 [D]
Auto
Fr: 1
Ungated
Blackout
None
Vascular
None
Epi(Opaque)
14-Feb-16 11:29:57 AM


Aldosary, Ajdai
1448911
60 yo Female
03-Feb-56 F
Stress-Gated [Isotope (A)
Dyssynchrony
718908
stress
4DMSPECT SaveScreens
Rest-Gated [Isotope (A) -
Contractility Histogram
Mean: 52.9 ± 14.4
Range: 5, 92
BW: 12(25), 28(75), 66(95)
Mean: 43.6 ± 19.9
Range: 4, 96
BW: 8(25), 48(75), 91(95)
Contractility Curve
ANT
LAT
INF
SEP
APX
TOT
Dyssynchrony
Number of Sectors
Normalized Amplitude
% of R-R Interval
% of R-R Interval
Stress-Gated [Isotope (A)
14-Feb-2016 11:38:07
8 Fr, Supine, FBP
Tc-99m Tetrofosmin
EDV: 86 ml
ESV: 47 ml
EF: 45 %
Mass: 127 g, CO: 3.4 l/min
UgVol: 57 ml, TID: 0.85
SSS: 3
Rest-Gated [Isotope (A) -
14-Feb-2016 15:10:04
8 Fr, Supine, FBP
Tc-99m Tetrofosmin
EDV: 88 ml
ESV: 60 ml
EF: 32 %
Mass: 134 g, CO: 2.4 l/min
UgVol: 67 ml, TID: --
SRS: 0
Tomo QA
Setup
Surf QA
Pmap QA
Images
Images + Quant
Func + Quant
Dyssynchrony
MPI Summary
Patient Info
Export Data
Save
Screen Capture
Help
Preferences
Quit
WL: 128 WW: 256 [D]
Auto
Fr: 1
Time Pk Con
Norm
Revers
3 Seg
None
Endo(FixEd)
14-Feb-16 11:29:57 AM


Shammiri Joaithan
Shammiri, Joaithan
8302
67 yo Male
03-Nov-48 M
Stress-Gated [Isotope (A)
31-Jan-2016 13:16:08
8 Fr, Supine, FBP
Tc-99m Tetrofosmin
EDV: 94 ml
ESV: 29 ml
EF: 69 %
Mass: 117 g, CO: 4.8 l/min
UgVol: 57 ml, TID: 1.19
SSS: 0
V2-GSRD/TC/NC/M
Rest-Gated [Isotope (A) -
01-Feb-2016 10:22:32
8 Fr, Supine, FBP
Tc-99m Tetrofosmin
EDV: 98 ml
ESV: 35 ml
EF: 64 %
Mass: 125 g, CO: 4.3 l/min
UgVol: 48 ml, TID: --
SRS: 0
V2-GSRD/TC/NC/M
GStr Frame: 1
GRst Frame: 1
Contractility Histogram
Mean: 50.5 ± 4.7
Range: 30, 59
BW: 2(25), 8(75), 16(95)
Mean: 50.1 ± 5.4
Range: 31, 99
BW: 2(25), 8(75), 20(95)
Number of Sectors
% of R-R Interval
Contractility Curve
Normalized Amplitude
ANT
LAT
INF
SEP
APX
TOT
Dyssynchrony
Stress-Gated [Isotope (A) ior
Rest-Gated [Isotope (A) -
709146
4DMSPECT SaveScreens
Anterior
Apex
Inferior
-5SD
5SD
5.0 SD
-5.0 SD
Tomo QA
Setup
Surf QA
Pmap QA
Images
Images + Quant
Func + Quant
Dyssynchrony
MPI Summary
Patient Info
Export Data
Save
Screen Capture
Help
Preferences
Quit
WL: 128 WW: 256 [D]
Auto
Fr 1
Time Pk Con
Norm
Revers
5 Seg
None
Endo(FixEd)
31-Jan-16 1:15:24 PM


Shammiri Joaithan
Shammiri, Joaithan
8302
67 yo Male
03-Nov-48 M
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31-Jan-2016 13:16:08
8 Fr, Supine, FBP
Tc-99m Tetrofosmin
EDV: 94 ml
ESV: 29 ml
EF: 69 %
Mass: 117 g, CO: 4.8 l/min
UgVol: 57 ml, TID: 1.19
SSS: 0
V2-GSRD/TC/NC/M
Rest-Gated [Isotope (A) -
01-Feb-2016 10:22:32
8 Fr, Supine, FBP
Tc-99m Tetrofosmin
EDV: 98 ml
ESV: 35 ml
EF: 64 %
Mass: 125 g, CO: 4.3 l/min
UgVol: 48 ml, TID: --
SRS: 0
V2-GSRD/TC/NC/M
Reversibility
31-Jan-2016 13:16:08
8 Fr, --
Tc-99m Tetrofosmin
V-REVER
Frame: 1 of 8
Mid-HLA
Mid-VLA
Perfusion
Septal
Apex
Inferior
Anterior
709146
4DMSPECT SaveScreens
0 %
50 %
100 %
Tomo QA
Setup
Surf QA
Pmap QA
Images
Images + Quant
Func + Quant
Dyssynchrony
MPI Summary
Patient Info
Export Data
Save
Screen Capture
Help
Preferences
Quit
WL: 128 WW: 256 [D]
APEX
ANT
SEP
INF
LAT
BASE
RAO
LAO
LPO
Auto
Fr 1
Ungated
Norm
Revers
None
Epi(Opaque)
31-Jan-16 1:15:24 PM