"Characterization of the major risk factors of Cardiovascular diseases and analysis of hypertension as a complication among the relatively newly diagnosed patients all over Bangladesh: A reflective study"

A dissertation submitted to BRAC University in partial fulfillment of the requirements for the degree, Bachelor (honours) of Science in Biotechnology

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December, 2018

Declaration

I hereby state that, this thesis, entitled **"Characterization of the major risk factors Cardiovascular diseases and analysis of hypertension as a complication among the relatively newly diagnosed patients all over Bangladesh: A reflective study"**

is based on my own work and it contains no material previously published or written by another person and not accepted for the award of any other degree of university or other institute of higher education.

This research work was carried out at the institute **"Ibrahim Cardiac Hospital & Research Institute",** Shahbagh, Dhaka under the supervision of Dr. Mohammad Rafiqul Islam, Associate Professor, Department of Mathematics and Natural Sciences, BRAC University, Dhaka.

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Acknowledgement

I would like to express my sincere gratefulness to **Professor A F M Yusuf Haider**, Chairperson, Department of Mathematics and Natural Sciences for allowing me to carry out my studies in this department. I also express my sincere gratitude to **Late Prof. A. A. Ziauddin Ahmad**, former Chairperson, Department of Mathematics and Natural Sciences, BRAC University for blessing me with his inspirations and supports.

I wish to convey my gratitude to my respected supervisor **Dr. Mohammad Rafiqul Islam**, Associate Professor, Department of Mathematics and Natural Sciences, who has guided me by his valuable advices throughout the study.

My special thanks to **Mr. MHM Mubassir**, Lecturer, Department of Mathematics and Natural sciences, for providing me with valuable insights regarding the technical aspects of this work.

I wish to express my gratitude to **Professor Dr. M. A. Rashid**, Chief Executive Officer, Ibrahim Cardiac Hospital & Research Institute, for allowing me to carry out my study at his esteemed institution.

Finally, I convey my humble gratitude to my mom and dad - not only for their support, their inspiration - and their humour, but also for their genes!

Asif Jahan Shuvro

December, 2018

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Abbreviations

Cardiovascular Disease (s) (CVDs)

Coronary Heart Disease (CHD)

Coronary Arterial Disase (CAD)

Coronary Pulmonary Disease (CPD)

Bangladesh Demographic and Health Survey (BDHS)

World Health Organisation (WHO)

Non Communicable Disease (s) (NCDs)

Sino Atrial Node (SAN)

Atrio Ventricular (AV)

Driving Pressure (DP)

Cardiac Output (CO)

Pulmonary Vascular Resistance (PVR)

Pulmonary Blood Flow (PBF)

Functional Residual Capacity (FRC)

Residual Volume (RV)

Total Lung Capacity (TLC)

Alveolar Pressure (Pa)

Positive End Expiratory Pressure (PEEP)

Positive Pressure Ventilation (PPV)

Non – Adrenergic Non – Cholinergic (NANC)

Pulmonary Hypertension (PHTN)

Angiotensin Converting Enzyme (ACE)

National Heart, Lung and Blood Institute (NHLBI)

Hypertrophic Cardio – Myopathy (HCM)

Dilated Cardio – Myopathy (DCM)

Restrictive Cardio – Myopathy (RCM)

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

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Ambulatory Blood Pressure Measurements (ABPM)

Body Mass Index (BMI)

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Combined Military Hospital (CMH)

Atrial Septal Defect (ASD)

Ventricular Septal Defect (VSD)

Patent Ductus Arteriosus (PDA)

Tetralogy of Fallot (TOF)

Ibrahim Cardiac Hospital & Research Institute (ICHRI)

Abstract

Heart related illness is a worldwide issue as of late. A large number of individuals everywhere throughout the world are experiencing some type of this sort of medical problem. The example of cardiovascular illness may vary in various parts of the world. Also, a substantial number of heart patients experience the ill effects of interminable issue. The target of this study is to distinguish the significant risk factors, decide a basic age range for beginning of coronary illness and portraying the heart patients of Bangladesh by dissecting distinctive cofactors on a reflective study. The study was completed in Ibrahim Cardiac Hospital and Research Institute (ICHRI) which have facilities in research, analysis and treatment of heart patients. An aggregate of around 502 subjects information were taken under this investigation for examining their cases who were enlisted at the institute in 2017. From the patients record, separate information were gathered for further investigation. The predominance of coronary illness was observed in a higher rate in men (55.6%) than women (44.4%). A large portion of the subjects were of the age scope of 51-60 or more than 60 years (~27% each). This explicit age range was found likewise inside both male and female subjects. Subsequent to figuring the Weight List of the subjects, it was discovered that, in spite of the fact that the biggest level of it exists in the typical weight limit (61.4%), there was a concerning rate exists in as far as possible (24.1%). From the aggregate number of subjects, 57.57% were experiencing Hypertension. In addition, a solid relationship of hypertension with Incessant Coronary illness was additionally watched. Other than these, the vast majority of the patients (27.3%) blood group was AB+(ve).

The proof from the investigation introduced that, the basic age range for beginning of coronary illness is over 50 years for both male and female individual and there is a solid relationship of hypertension with Chronic Coronary illness.

1. Introduction

Cardiovascular diseases (CVDs) are caused by disorders of the heart and blood vessels and include coronary heart disease (heart attacks), cerebro - vascular disease (stroke), raised blood pressure (hypertension), peripheral artery disease, rheumatic heart disease, congenital heart disease and heart failure. Cardiovascular diseases are the leading cause of death globally. Approximately 17.3 million people died from CVDs in 2008 which represents 30% of all global deaths. Among them an estimated 7.3 million deaths were due to coronary heart disease and 6.2 million were due to stroke. Among all the CVD deaths 80% take place in low- and middle-income countries and occur almost equally among men and women. It is estimated that by 2030, almost 23.6 million people will die from CVDs, mainly heart disease and stroke (WHO, 2011).Unlike other developing countries, Bangladesh is also experiencing an epidemiological transition from infectious, communicable diseases to chronic, non-communicable diseases (NCDs) like cardiovascular diseases, diabetes, cancer, chronic respiratory diseases, and injury (BDHS, 2011).According to WHO (2011) in Bangladesh, NCDs were estimated to account for 52% of all deaths and among them 27% were due to CVDs. In a recent study it was projected that death rate from CVDs would be 4 times higher in 2010 and 21 times higher by 2025 (Karar et Al.2009). According to World Health Rankings (2011), coronary artery disease was ranked as the first and stroke was the third leading cause of death which accounts for 17.11% and 8.57% respectively.

According to World Health Organization the most important behavioral risk factors of CVDs are unhealthy diet, physical inactivity, tobacco use and harmful use of alcohol. These risk factors are responsible for about 80% of coronary heart disease and cerebrovascular disease (WHO, 2011). People in low- and middle-income countries are more exposed to risk factors such as tobacco, leading to CVDs and other non - communicable diseases. At the same time they often do not have the benefit of prevention programs compared to people in high-income

countries. People in low- and middle-income countries who suffer from CVDs and other non -communicable diseases have less access to effective and equitable health care services which respond to their needs (including detection of diseases at early stage). As a result, many people in low and middle-income countries die younger from CVDs and other non -communicable diseases, often in their most productive years.

Unlike other developing countries, Bangladesh is also experiencing an epidemiological transition from infectious, communicable diseases to chronic, non communicable diseases (NCDs) like cardiovascular diseases, diabetes, cancer, chronic respiratory diseases, and injury (BDHS, 2011).According to WHO (2011) in Bangladesh, NCDs were estimated to account for 52% of all deaths and among them 27% were due to CVDs. In a recent study it was projected that death rate from CVDs would be 4 times higher in 2010 and 21 times higher by 2025 (Karar et Al.2009). According to World Health Rankings (2011), coronary artery disease was ranked as the first and stroke was the third leading cause of death which accounts for 17.11% and 8.57% respectively.

CVDs are emerging as an epidemic in a poor country like Bangladesh. So it will be better if we can prevent the risk factors of CVDs. So risk factor identification is the only way to achieve this goal. In this regard my study will find out the risk factors and prevention strategies of CVDs that are needed to develop effective national health policy to prevent and manage them. This way, we can reduce morbidity and mortality among CVD patients and alleviate the burden of CVDs. This paper explored the availability of literature on risk factors and prevention strategies of CVDs in Bangladesh through a scoping review which, unlike a systematic review, offers a much broader perspective in the respective field which makes it more appropriate method to assess the risk factors and prevention strategies of CVDs in Bangladesh.

1.1 A Brief Introduction to Human Heart

The heart is a muscular pump that serves two functions: (1) to collect blood from the tissues of the body and pump it to the lungs and (2) to collect blood from the lungs and pump it to all tissues of the body. The human heart lies in the protective thorax, posterior to the sternum and costal cartilages, and rests on the superior surface of the diaphragm. The heart assumes an oblique position in the thorax, with two-thirds to the left of midline. It occupies a space between the pleural cavities called the *middle mediastinum,* defined as the space inside the pericardium, the covering around the heart. This serous membrane has inner and outer layers, with a lubricating fluid in between. The fluid allows the inner visceral pericardium to "glide" against the outer parietal pericardium.

The internal anatomy of the heart reveals four chambers composed of cardiac muscle or myocardium. The two upper chambers (or atria) function mainly as collecting chambers; the two lower chambers (ventricles) are much stronger and function to pump blood. The role of the right atrium and ventricle is to collect blood from the body and pump it to the lungs. The role of the left atrium and ventricle is to collect blood from the lungs and pump it throughout the body. There is a one-way flow of blood through the heart; this flow is maintained by a set of four valves. The atrioventricular valves (tricuspid and bicuspid) allow blood to flow only from atria to ventricles. The semilunar valves (pulmonary and semilunar) allow blood to flow only from the ventricles out of the heart and through the great arteries. A number of structures that can be observed in the adult heart are remnants of fetal circulation. In the fetus, the lungs do not function as a site for the exchange of oxygen and carbon dioxide, and the fetus receives all of its oxygen from the mother. In the fetal heart, blood arriving to the right side of the heart is passed through specialized structures to the left side. Shortly after birth, these specialized fetal structures normally collapse, and the heart takes on the "adult" pattern of circulation. However, in rare cases, some fetal remnants and defects can occur.

Although the heart is filled with blood, it provides very little nourishment and oxygen to the tissues of the heart. Instead, the tissues of the heart are supplied by a separate vascular supply committed only to the heart. The arterial supply to the heart arises from the base of the aorta as the right and left coronary arteries (running in the coronary sulcus). The venous drainage is via cardiac veins that return deoxygenated blood to the right atrium.

It is important to note that, besides pumping oxygen rich blood to the tissues of the body for exchange of oxygen for carbon dioxide, the blood also circulates many other important substances. Nutrients from digestion are collected from the small intestine and pumped through the circulatory system to be delivered to all cells of the body. Hormones are produced from one type of tissues and distributed to all cells of the body. The circulatory system carries waste materials (salts, nitrogenous wastes, and excess water) from cells to the hearts, where they are extracted and passed to the bladder. The pumping of interstitial fluid from the blood into the extracellular space is an important function of the heart. Excess interstitial fluid is then returned to the circulatory system via the lymphatic system.

1.1.1 External Anatomy of the Heart

The heart lies in the protective thorax, posterior to the sternum and costal cartilages, and rests on the superior surface of the diaphragm. The thorax is often referred to as the thoracic cage because of its protective function of the delicate structures within. The heart is located between the two lungs, which occupy the lateral spaces, called *thepleural cavities.* The space between these two cavities is referred to as the *mediastinum* ("in the middle"; Figure. 1).

The mediastinum is divided first into the superior and inferior mediastinum by a midsagittal imaginary line called the *transverse thoracic plane.* This plane passes through the sternalangle (junction of the manubrium and body of the sternum) and the space between

thoracic vertebrae T4 and T5. This plane acts as a convenient landmark because it also passes through

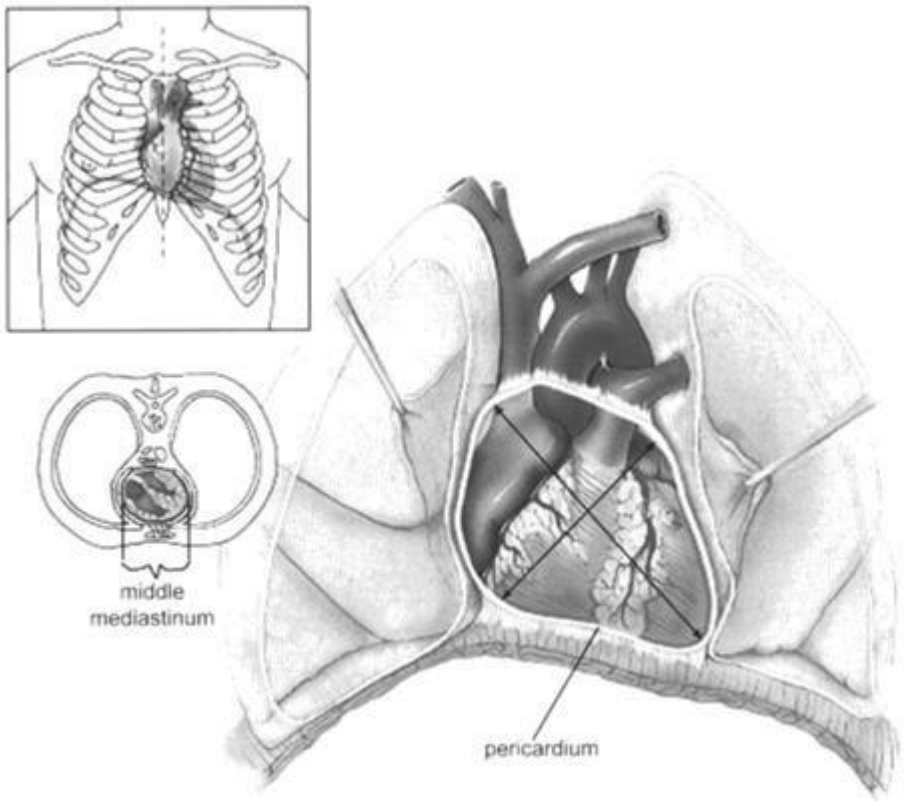

Figure.1: Position of the heart in the thorax. The heart lies in the protective thorax, posterior to the sternum and costal cartilages, and rests on the superior surface of the diaphragm. The heart assumes an oblique position in the thorax, with two-thirds to the left of midline. It is located between the two lungs, which occupy the lateral spaces called the *pleural cavities.* The space between these two cavities is referred to as the *mediastinum.* The heart lies obliquely in a division of this space, the middle mediastinum, surrounded by the pericardium. (*Human Anatomy,*3rd Ed. by Elaine N. Marieb and Jon Mallatt. © 2001 by Benjamin Cummings. Reprinted by permission of Pearson Education, Inc.)

the following structures: the bifurcation of the trachea, the superior border of the pericardium, the base of the aorta, and the bifurcation of the pulmonary trunk.

The human heart assumes an oblique position in the thorax, with two-thirds to the left of midline (Figure. 2 and 3). The heart is roughly in a plane that runs from the right shoulder

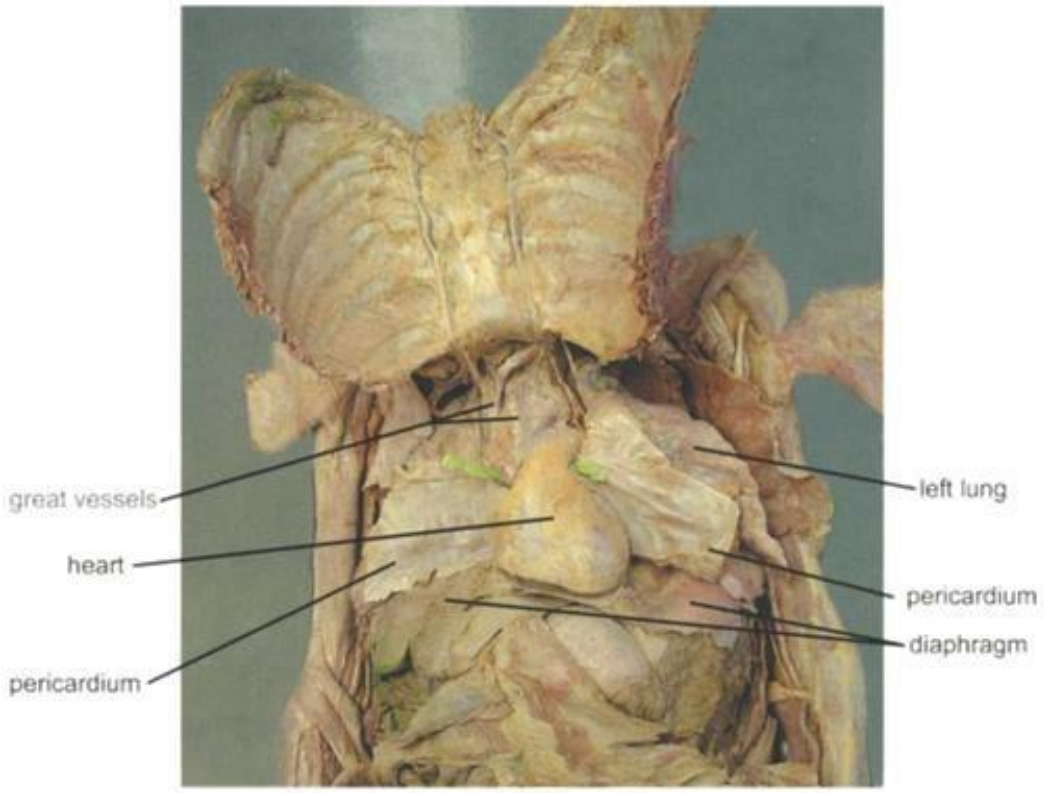

Figure.2: Cadaveric dissection. Human cadaver dissection in which the ribs were cut laterally, and the sternum and ribs were reflected superiorly. This dissection exposes the contents of the thorax (heart, great vessels, lungs, and diaphragm).

to the left nipple. The base is located below the third rib as it approaches the sternum (note that the sternal angle occurs at the level of the second rib). The base is directed superiorly to the right of mid-line and posterior. The pointed apex projects to the left of mid-line and anterior. Thus, the heartbeat can be most easily palpated between the fifth and sixth ribs (just inferior to the left nipple) from the apex of the heart where it comes into contact with the thoracic wall. Importantly, the heart lies in such an oblique plane that it is often referred to as

horizontal. Thus, the anterior side is often referred to as superior and the posterior side as inferior.

Again, the heart is composed of four distinct chambers. There are two atria (left and right) responsible for collecting blood and two ventricles (left and right) responsible for pumping blood. The atria are positioned superior to (posterior to) and to the right of their respective ventricles (Figure. 3). From superior to inferior, down the anterior (superior) surface of the heart runs the anterior interventricular sulcus ("a groove"). This sulcus separates the left and right ventricles. The groove continues around the apex as the posterior interventricular sulcus on the posterior (inferior) surface. Between these sulci, located within the heart, is the interventricular septum ("wall between the ventricles"). The base of the heart is defined by a plane that separates the atria from the ventricles, called the *atrioventricular groove* or *sulcus.* This groove appears like a belt cinched around the heart. Because this groove appears as though it might also be formed by placing a crown atop the heart, the groove is also called the coronary sulcus. The plane of this sulcus also contains the atrioventricular valves (and the semilunar valves) and a structure that surrounds the valves called the *cardiac skeleton.* The interatrial ("between the atria") septum is represented on the posterior surface of the heart as the atrial sulcus.It is noteworthy that the great arteries, aorta and pulmonary trunk, arise from the base of the heart. The right and left atrial auricles, so named because they look like ‘dog ears’ - appear as extensions hanging off each atria.

The anterior or superior surface of the heart is formed primarily by the right ventricle. The right lateral border is formed by the right atrium and the left lateral border by the left ventricle. The posterior surface is formed by the left ventricle and the left atrium, which is centered equally on the midline.

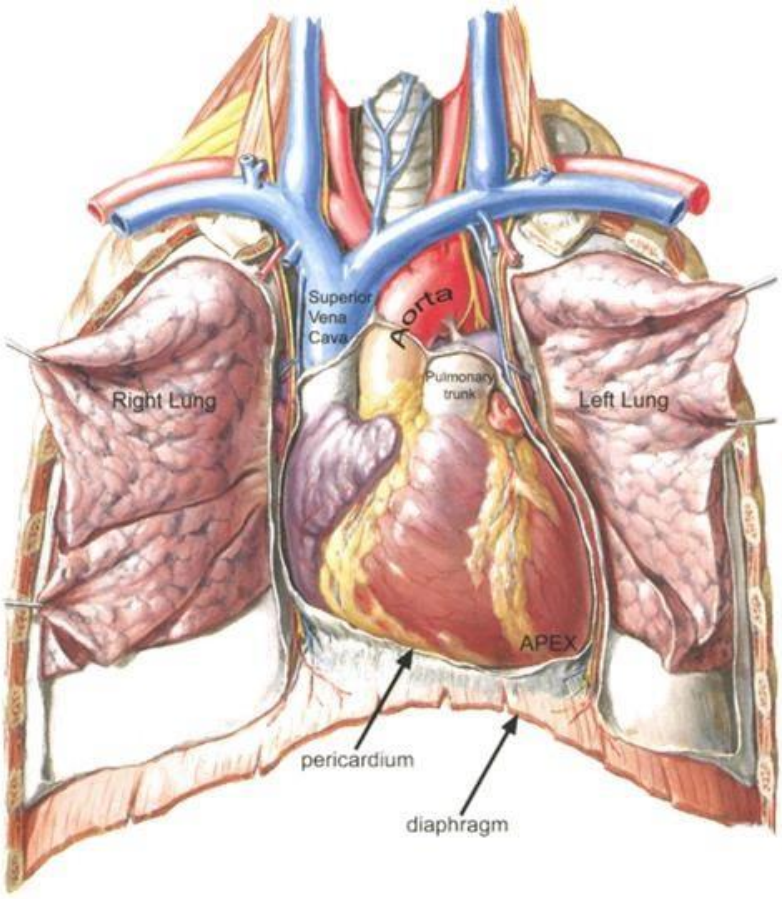

Figure.3: The anterior surface of the heart. The atria are positioned superior to (posterior to) and to the right of their respective ventricles. Sulcus separates the left and right ventricles. The base of the heart is defined by a plane, called the atrioventricular groove or sulcus, that separates the atria from the ventricles. Note that the great arteries, aorta, and pulmonary trunk arise from the base of the heart. The right and left atrial appendages appear as extensions hanging off each atria. The anterior (superior) surface of the heart is formed primarily by the right ventricle. The right lateral border is formed by the right atrium, and the left lateral border by the left ventricle. The posterior surface is formed by the left ventricle and the left atrium, which is centered equally on the midline.

More so, the acute angle found on the right anterior side of the heart is referred to as the *acute* margin of the heart and continues toward the diaphragmatic surface.

1.1.2 The Internal anatomy of Human Heart

The tissue of the heart – wall, and the layer that actually contracts is called myocardium. The myocardium consists of cardiac muscles in a spiral arrangement of myocardium that squeeze blood through the heart in the proper directions: inferiorly through the atria and superiorly through the ventricles. A cross section cut through the heart reveals three layers (Figure. 4): A superficial visceral pericardium or epicardium; a middle myocardium; and a deep lining called the endocardium.

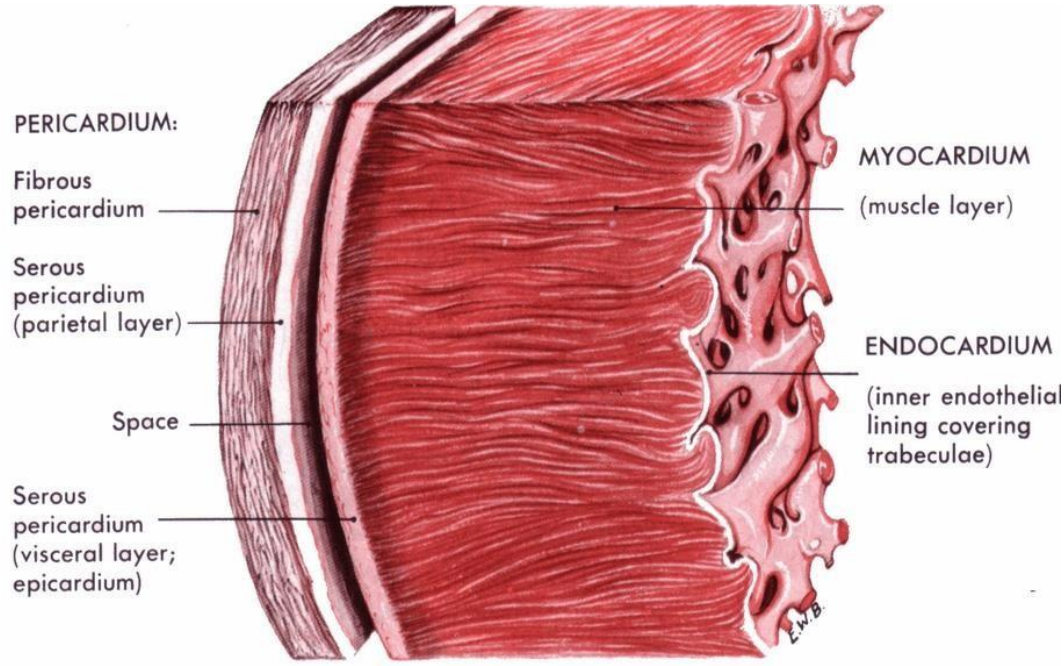

Figure .4: Section of the heart wall showing the components of the outer pericardium (heart sac), muscle layer(myocardium), and inner lining (endocardium).

Cardiac muscle cells are triggered to contract by the flow of Ca^{2+} ions into the cell. Cardiac muscle cells are joined by complex junctions called intercalated discs which contain adherents to hold the cells together. Certain gap junctions allow ions to pass easily between the cells – hence allow the free movement of ions between cells for the direct transmission of an electrical impulse through a complex network of cardiac muscle cells. This impulse, again, signals all the muscle cells to contract simultaneously.

1.1.2.1 Major Internal Chambers Inside Heart

The inside of the heart is divided into four hollow chambers, with two on the left and two on the right. The upper chambers are called atria and receive blood returning to the heart. They have auricles, which are small projections that extend anteriorly. The lower chambers are called ventricles and receive blood from the atria, which they pump out into the arteries (Figure.5). The left atria and ventricle are separated from the right atria and ventricle by a solid wall like structure called septum. This keeps blood from one side of the heart from mixing with blood from the other side (except in a developing fetus). The atrioventricular valve (AV valve), which consists of the mitral valve on the left and the tricuspid valve on the right, ensures one-way blood flow between the atria and ventricles.

The right atrium receives blood from two large veins called the superior vena cava and the inferior vena cava as well as a smaller vein (the coronary sinus), which drains blood in to the right atrium from the heart's myocardium. The tricuspid valve has projections (cusps) and lies between the right atrium and ventricle. This valve allows blood to move from the right atrium into the right ventricle while preventing backflow. The cusps of the tricuspid valve are attached to strong fibers called chordae tendineae, which originate from small papillary muscles that project inward from the ventricle walls. These muscles contract as the ventricle contracts. When the tricuspid valve closes, they pull on the chordae tendineae to prevent the cusps from swinging back into the atrium. The right ventricle's muscular wall is thinner than that of the left ventricle, as it only pumps blood to the lungs with a low resistance to blood flow. The left ventricle is thicker because it must force blood to all body parts, with a much higher resistance to blood flow. As the right ventricle contracts, its blood increases in pressure to passively close the tricuspid valve. Therefore, this blood can only exit through the pulmonary trunk, which divides into the left and right pulmonary arteries that supply the

lungs. At the trunk's base, there is a pulmonary valve with three cusps that allow blood to leave the right ventricle while preventing backflow into the ventricular chamber (Figure.6).

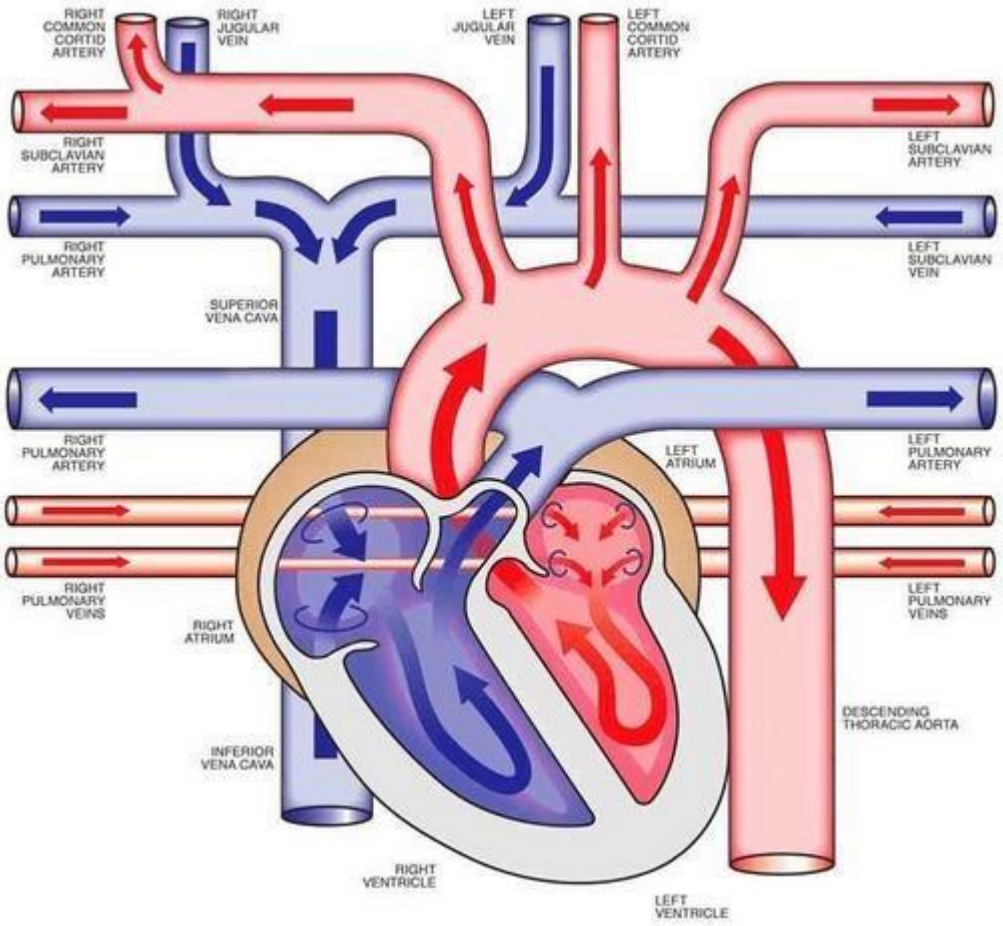

Figure.5: Blood flow through the heart. Deoxygenated (carbon-dioxide-enriched) blood (blue arrows) flows into the right atrium from the systemic circulation and is pumped first into the right ventricle and then into the pulmonary artery which delivers it to the lungs. In the lungs, the blood releases its carbon dioxide and absorbs oxygen. Re oxygenated blood (red arrows) is returned to the left atrium, then flows into the left ventricle, which pumps it to the rest of the body through the systemic circuit.

Four pulmonary veins (two from each of the lungs) supply the left atrium with blood. Blood passes from the left atrium into the left ventricle through the mitral valve (bicuspid valve), preventing blood from flowing back into the left atrium from the ventricle. Like the tricuspid

valve, the papillary muscles and chordae tendineae (Figure.7) prevent the mitral valve's cusps from swinging back into the left atrium when the ventricle contracts. The mitral valve closes passively, directing blood through the large artery known as the aorta.

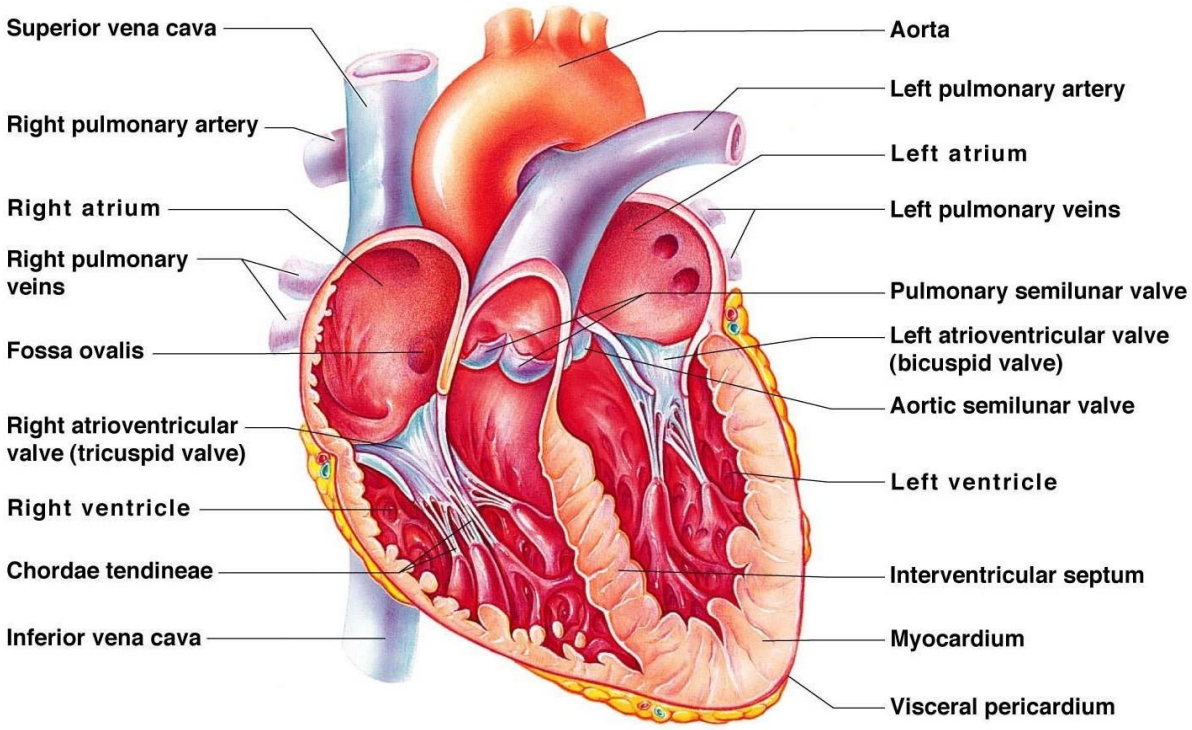

Figure.6: Heart valves. A cross-section of the heart showing the four chambers and the location of the major vessels and valves.

The right atrium receives low-oxygen blood through the vena cava and coronary sinus. As the right atrium contracts, the blood passes through the tricuspid valve into the right ventricle (Figure.6). As the right ventricle contracts, the tricuspid valve closes. Blood moves through the pulmonary valve into the capillaries of the alveoli of the lungs, where gas exchanges occur. This freshly oxygenated blood then returns to the heart through the pulmonary veins, into the left atrium. As the left atrium contracts, blood moves through the mitral valve into the left ventricle. When the left ventricle contracts, the mitral valve closes. Blood moves through the aortic valve into the aorta and its branches. The first two aortic branches are

called the right and left coronary arteries. They supply blood to the heart tissues, with openings lying just beyond the aortic valve.

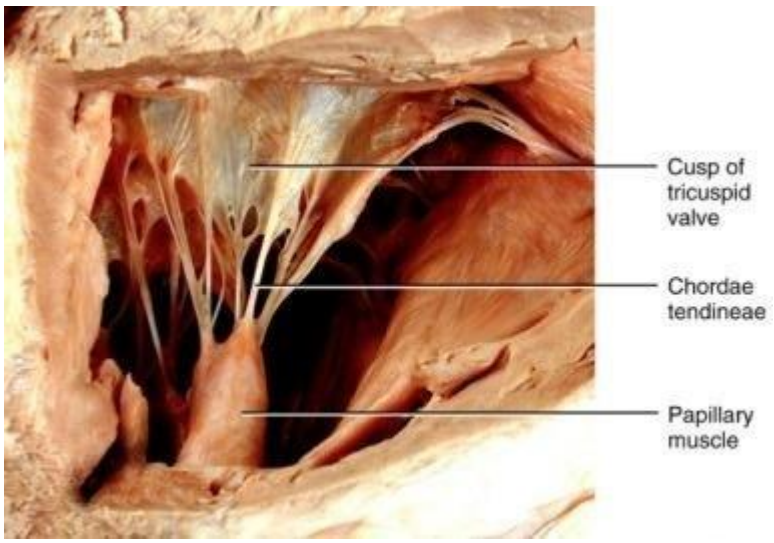

Figure.7: Primary cross section of heart showing Chordae tendineae

1.1.3 Function of the Human Heart: The Cardiac Cycle

The cardiac cycle is the sequence of events in one heartbeat. In its simplest form, the cardiac cycle is the simultaneous contraction of both atria, followed a fraction of a second later by the simultaneous contraction of both ventricles. The heart consists of cardiac muscle cells that connect with each other – they are branched – and so when one contracts, they stimulate their neighbors and they all contract. The heart is an 'all-or-nothing' muscle, getting its rest between beats. It can only respire aerobically.

A heartbeat has two phases: Phase 1 or Systole is the term for contraction. This occurs when the ventricles contract, closing the A-V valves and opening the Semi-Lunar valves to pump blood into the two major vessels leaving the heart. Phase 2 or Diastole is the term for relaxation. This occurs when the ventricles relax, allowing the back pressure of the blood to close the semi-lunar valves and opening the A-V valves.

The cardiac cycle also creates the heart sounds: each heartbeat produces two sounds, often called lub-dup, that can be heard with a stethoscope. The first sound is caused by the contraction of the ventricles (ventricular systole) closing the A-V valves. The second sound is caused by the snapping shut of the Aortic and Pulmonary Valves (Semi-lunar valves). If any of the valves do not close properly, an extra sound called a 'heart murmur' may be heard. Although the heart is a single muscle, it does not contract all at once. The contraction spreads over the heart like a wave, beginning in a small region of specialized cells in the right atrium called the Sino-Atrial Node (SAN). This is the hearts natural pacemaker, and it initiates each beat The impulse spreads from the SAN through the cardiac muscle of the right and left atrium, causing both atria to contract almost simultaneously. When the impulse reaches another special area of the heart, right in the centre of the septum, known as the Atrio-Ventricular (or AV) Node, the impulse is delayed for approximately 0.2 s. This allows time for the ventricles to fill completely. The AV Node relays the electrical impulse down the septum, along the Bundle of His, to the base of the ventricles. The ventricles then contract simultaneously, from the bottom upwards, thus allowing them to empty completely with each beat.

The heartbeat is initiated by the Sino-Atrial Node and passes through the Atrio-Ventricular Node, remaining at the same rhythm until nerve impulses cause it to speed up or to slow down. Unlike other muscles, it does not require a new nerve impulse for each contraction. The autonomic nervous system controls heart rate. The accelerator nerve of the sympathetic nervous system increases heart rate and the vagus nerve of the parasympathetic nervous system decreases heart rate. For most people, their resting heart rate is between 60 and 80 b.p.m. During exercise that can increase to as many as 200 beats per minute for an athlete; for the rest of us, we can safely manage about 150 b.p.m.

1.1.4 Physiological features of the pulmonary versus systemic circulation

Blood flow to the pulmonary circulation is roughly equal to that of the systemic circulation (6 litres at rest, up to 25 litres at full exercise). The pressure however is greatly reduced and as a consequence resistance ($R=\Delta P/\text{Blood Flow}$). As shown in the diagram (Figure.8), the mean pressure for the pulmonary circulation is 15 mmHg compared to 100mmHg for the systemic circulation. The pressure decreases by roughly the same amount in the arterioles, capillaries and venules as opposed to the systemic circulation where the majority of decrease is in the arterioles. A consequence of the reduced pressures is also a right ventricle which has only half the muscle mass of the left ventricle.

Whilst the high pressures and resistance of the systemic circulation enable changes to perfusion of specific organs and regions, the control in the pulmonary system is significantly reduced and subject to much greater variation with respect to gravity resulting in the under and over perfusion of parts of the lung. The benefit of this system however is that it is well designed to perform its primary function of gas exchange, providing pressures which are just enough to perfuse the top of the lungs (and therefore in disease states this may be the first impaired). The reduced pressures also reduce the likelihood of transudation into the aveoli (pulmonary oedema) which causes significant impairment of gas exchange.

The pulmonary arterioles contain little smooth muscle (although vasoconstriction is still possible) and the venules and veins are almost devoid of smooth muscle and are therefore very distensible. These features ensure that the pulmonary system is also able to act as a blood reserve and increase its volume up to five times when systemic return is increased from 0.5-1.0 litres to 2.5-3.0 litres.

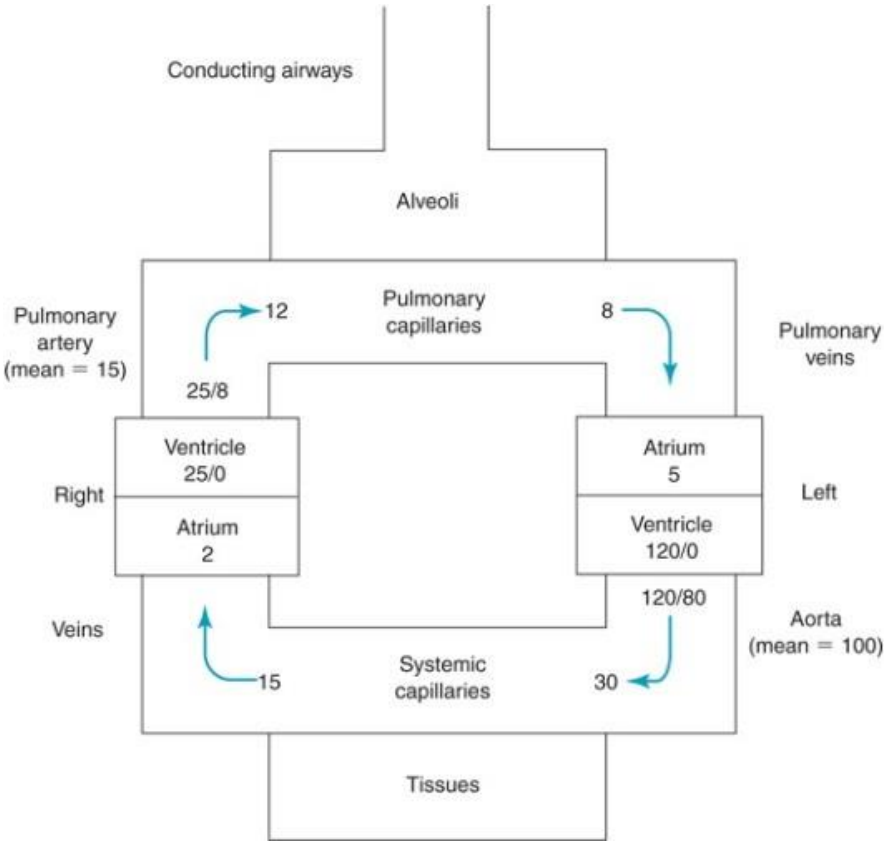

Figure.8: Comparison of Pressures, expressed in mm Hg, in the systemic and pulmonary circulations.

Pulmonary blood volume is influenced by both posture (changing from supine to erect decreases volume by a third due to pooling in dependent regions of the systemic circ.) and systemic vascular tone (endogenous or exogenous pressers, g-suits or diving).Venous Admixture refers to the degree of mixed venous blood with pulmonary end capillary PO_2 that would be required to produce the observed difference in the end cap PO_2 and the arterial PO_2. Not all blood that passes through the pulmonary circulation is oxygenated. This constitutes the intrapulmonary shunt. There is also the bronchial circulation which usually arises from the aorta or intercostal arteries. This supplies the pulmonary system down to the respiratory bronchioles therefore a majority does not tke part in gas exchange. It returns via the pulmonary veins (unoxygenated to the left heart) or the normal venous return (via the right heart therefore not shunted).

1.1.4.1 Pulmonary vascular pressures

As stated previously the pulmonary artery pressure is only about a sixth of the systemic system. There is only a small pressure drop in the pulmonary arterioles and therefore unlike the systemic system the control is reduced. In order to correctly interpret the physiological parameters there are three different pressures that are measured and used in clinical practice. The most common in the intravascular pressure. This is calculated by measuring the pressure in the pulmonary system and comparing to atmospheric pressure. The same method is used in the systemic circulation. The second method is the transmural pressure, which is the difference in the vessel compared to that in the tissue surrounding the vessel. It is measured by an oesophageal balloon (which equates to pleural pressures) and is helpful when you need to exclude the effect of raised intra thoracic pressures (which may suggest a much higher intravascular pressure). The final pressure is the driving pressure. This is the difference between one point in the system and another and is usually used for the calculation of system vascular resistance by comparing the pressures in pulmonary artery and left atrium (ΔP).

1.1.4.2 Pulmonary Vascular Resistance

This is an expression of the relationship between driving pressure (DP) and flow (often simplified to the cardiac output CO), as in the case of resistance to gas flow. As previously stated it may be expressed as;

$$PVR = DP/CO \quad \text{..... (1)}$$

It is important to note however that this relationship is non linear, which is mainly due to passive changes in the pulmonary circulatory system although active changes may play some part.

1.1.4.3 Passive changes in pulmonary blood flow and resistance

Three main mechanisms contribute to passive changes in PBF/PVR. Recruitment and distension due to increases in cardiac output, changes due to the opposing influences of different lung volumes have on intra and extra alveolar blood vessels and the effect of gravity and the so called west's zones of perfusion. Each are considered separately below.

Adaption: There are 2 different mechanisms that can explain this decrease in PVR in response to elevated blood flow and perfusion pressure: recruitment and distention (Figure. 9).

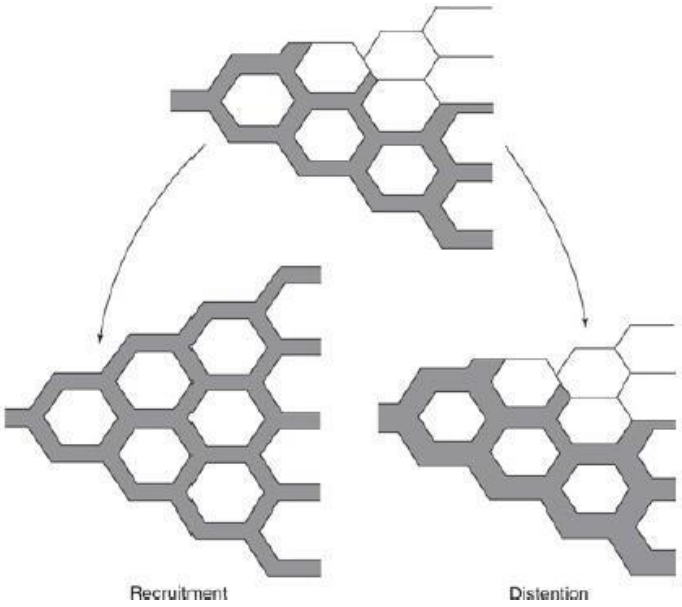

Figure.9: Illustration of the mechanisms by which increased mean pulmonary artery pressure may decrease pulmonary vascular resistance. The upper figure shows a group of pulmonary capillaries, some of which are perfused. At left, the previously unperfused capillaries are recruited (opened) by the increased perfusion pressure. At right, the increased perfusion pressure has distended those vessels already open.

The pulmonary circulation can adapt to large changes in cardiac output with only small increases in the pulmonary pressures. Therefore by using the equation shown above there must be corresponding decrease in vascular resistance with increase in CO (PVR × CO=DP).

The answer to the question of whether it is recruitment or distention that causes the decreased PVR seen with elevated perfusion pressure is probably *both*. Perhaps *recruitment* of pulmonary capillaries occurs with small increases in pulmonary vascular pressures and *distention* at higher pressures. Note that recruitment increases the surface area for gas exchange and may decrease alveolar dead space. Derecruitment caused by low right ventricular output or high alveolar pressures decreases the surface area for gas exchange and may increase alveolar dead space.

Lung Inflation due to Pulmonary Vascular Resistance: Two different groups of pulmonary vessels must be considered when the effects of changes in lung volume on PVR are analyzed: those vessels that are exposed to the mechanical influences of the alveoli and the larger vessels that are not the alveolar and extra-alveolar vessels (Figure .10). As lung volume increases during a normal negative-pressure inspiration, the alveoli increase in volume. While the alveoli expand, the vessels found between them, mainly pulmonary capillaries, are elongated. As these vessels are stretched, their diameters decrease, just as stretching a rubber tube causes its diameter to narrow. Resistance to blood flow through the alveolar vessels increases as the alveoli expand because the alveolar vessels are longer (resistance is directly proportional to length) and because their radii are smaller (resistance is inversely proportional to radius to the fourth power).

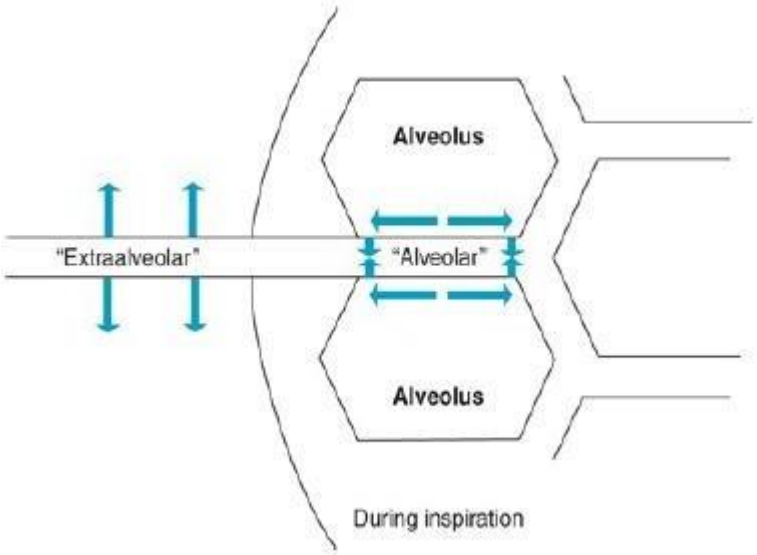

Figure.10: Illustration of alveolar and extraalveolar pulmonary vessels during an inspiration. The alveolar vessels (pulmonary capillaries) are exposed to the expanding alveoli and elongated. The extraalveolar vessels, here shown exposed to the intrapleural pressure, expand as the intrapleural pressure becomes more negative and as radial traction increases during the inspiration.

At high lung volumes, then, the resistance to blood flow offered by the alveolar vessels increases greatly; at low lung volumes, the resistance to blood flow offered by the alveolar vessels decreases. This can be seen in the 'alveolar' curve in Figure.11.

One group of the extra alveolar vessels, the larger arteries and veins, is exposed to the intrapleural pressure. As lung volume is increased by making the intrapleural pressure more negative, the transmural pressure difference of the larger arteries and veins increases and they distend. Another factor tending to decrease the resistance to blood flow offered by the extraalveolar vessels at higher lung volumes is 'radial traction' by the connective tissue and alveolar septa holding the larger vessels in place in the lung. Thus, at high lung volumes(attained by normal negative-pressure breathing), the resistance to blood flow offered by the extraalveolar vessels decreases (Figure.11). During a forced expiration to low

lung volumes, however, intrapleural pressure becomes very positive. Extra alveolar vessels are compressed, and as the alveoli decrease in size, they exert less radial traction on the extraalveolar vessels. The resistance to blood flow offered by the extraalveolar vessels increases greatly (see left side of Figure.11).

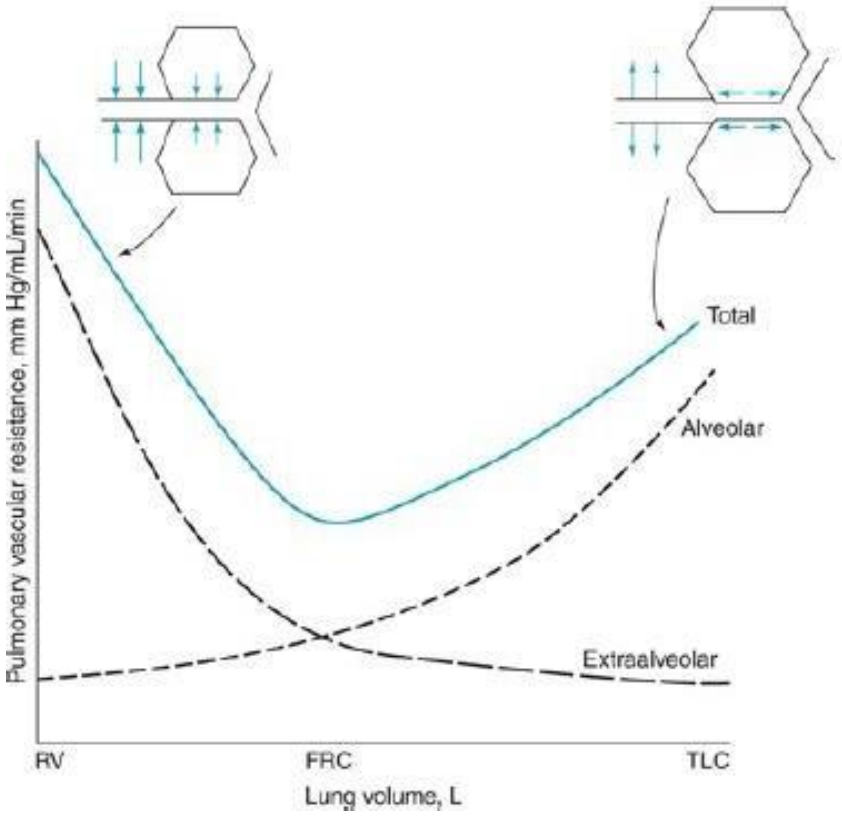

Figure.11: The effects of lung volume on pulmonary vascular resistance (PVR). PVR is lowest near the functional residual capacity (FRC) and increases at both high and low lung volumes because of the combined effects on the alveolar and extraalveolar vessels. To achieve low lung volumes, one must generate positive intrapleural pressures so that the extraalveolar vessels are compressed, as seen at left in the figure. RV = residual volume; TLC = total lung capacity. (Reproduced with permission from Graph after Murray, 1976, 1986.)

Because the alveolar and extraalveolar vessels may be thought of as 2 groups of resistances in series with each other, the resistances of the alveolar and extraalveolar vessels are additive at any lung volume. Thus, the effect of changes in lung volume on the total PVR gives the U-shaped curve seen in Figure.11. PVR is lowest near the functional residual capacity and increases at both high and low lung volumes.

Also note that during mechanical positive-pressure ventilation, alveolar pressure (Pa) and intrapleural pressure are positive during inspiration. In this case, both the alveolar and extraalveolar vessels are compressed as lung volume increases, and the resistance to blood flow offered by both alveolar and extraalveolar vessels increases during lung inflation. This is especially a problem during mechanical positive-pressure ventilation with positive end-expiratory pressure (PEEP). During PEEP, airway pressure (and thus alveolar pressure) is kept positive at end expiration to help prevent atelectasis. In this situation, alveolar pressure and intrapleural pressure are positive during both inspiration and expiration. PVR is elevated in both alveolar and extraalveolar vessels throughout the respiratory cycle. In addition, because intrapleural pressure is always positive, the other intrathoracic blood vessels are subjected to decreased transmural pressure differences; the venae cavae, which have low intra vascular pressure, are also compressed. If cardiovascular reflexes are unable to adjust to this situation, cardiac output may fall precipitously because of decreased venous return and high PVR.

Gravity and West's Zones of the Lung: The interplay between of alveolar pressure, flow rate and vascular resistance is best considered by dividing the lung field into three zones (Figure.12). This was first described by West and colleagues using a starling resistor model. It is best explained using a weir model.

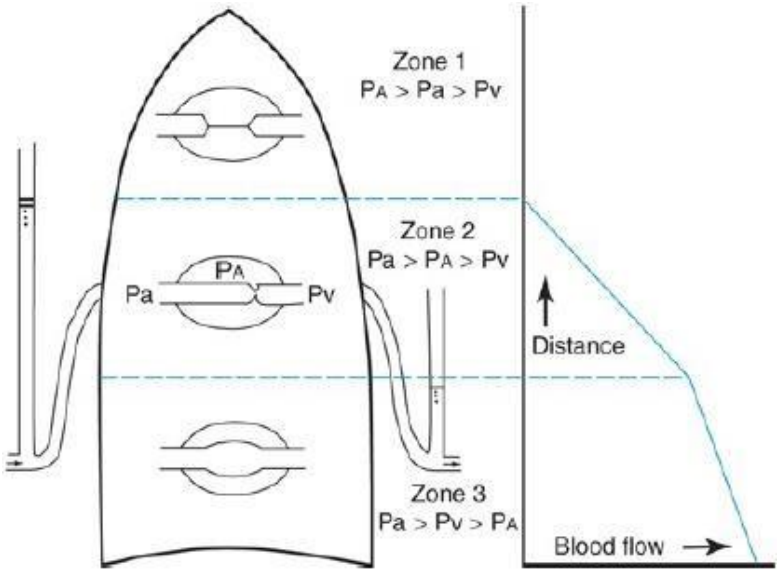

Figure.12: The zones of the lung. The effects of gravity and alveolar pressure on the perfusion of the lung. Described in text.(Redrawn from West, 1964, with permission.)

In zone 1 the alveolar pressure exceeds the precapillary pressure therefore there is no flow. This does not occur normally although maybe present if the pulmonary artery pressure is decreased due to shock or iatrogenically due to PPV.

Zone 2 is a situation where the alveolar pressure is less than the precap but more then the post cap. In this setting, the flow is determined by the pressure difference between the precap and the alveolar.

In the third zone the alveolar pressure is less than both the precap and post cap, therefore it is these two parameters which determine flow.

A Zone 4 is sometimes referred to where an increase in extralveolar pressures due to gravity increase resistance and thus flow as described above. Therefore, at the very base there is actually a reduction in flow compared to zone 3.

1.1.4.3Active changes in pulmonary blood flow and resistance

In addition to the three mechanisms described above the pulmonary vasculature is able to control resistance by both active vasoconstriction and vasodilation. It is believed that the lung is held in a state of active vasodilation. Cellular mechanisms which include receptors and their associated second messengers do influence vascular tone but many of these are poorly understood. There is good evidence that the basal production of NO occurs in normal lungs and contributes to maintenance of low pulmonary vascular resistance. There are three autonomic systems which influence active control of vascular tone, adrenergic, cholinergic and non-adrenergic non-cholinergic (NANC). Adrenergic control is the most important, alpha 1 receptors mediate vasoconstriction and predominate, beta 2 receptors will cause vasodilation to a lesser extent. Humoral control actioned by catecholamines, Eicosanoids (which may mediate thedevelopment of sepsis associated PHTN) amines and peptides play a role. Drugs are also important, especially inhaled NO which causes localised vasodilation, prostacyclin,ACE inhibitors and calcium channel antagonists reduce pulomnary pressures. Phosphodiesterase inhibitors such as sildenafil have significant effects as do endothelin receptor antagonists such as bosanten.

1.2 Types of Cardiovascular Disease

The term “heart disease” conforms to several types of heart conditions. The most common type is coronary artery disease, which can cause heart attack. Other kinds of heart disease may involve the valves in the heart, or the heart may not pump well and cause heart failure. Some people are born with heart disease.

Cardiovascular disease refers to a class of diseases that involve the heart and/or blood vessels (i.e arteries). It is commonly related to artherosclerosis, a process whereby fatty deposits (‘plaques’) form in your arteries, causing them to narrow and possibly block completely.

When atherosclerosis affects the major arteries in the body it can cause a heart attack, stroke or peripheral arterial disease. By recognising the warning signs and and symptoms and seeking medical care promptly, one may be able to avert or reduce the severity of a critical lack of blood supply to their heart (heart attack), brain (stroke) or hands and feet (peripheral arterial disease).

1.2.1 Coronary heart disease (CHD)

Coronary heart disease (CHD), also known as coronary artery disease (CAD), is caused by the build up of plaque in the arteries that supply oxygen-rich blood to the heart. Plaque, a mixture of fat, cholesterol, and calcium deposits, can build up in the arteries over many years. Over time, this plaque can cause the narrowing and hardening of the coronary arteries, a condition called atherosclerosis (Figure. 13).

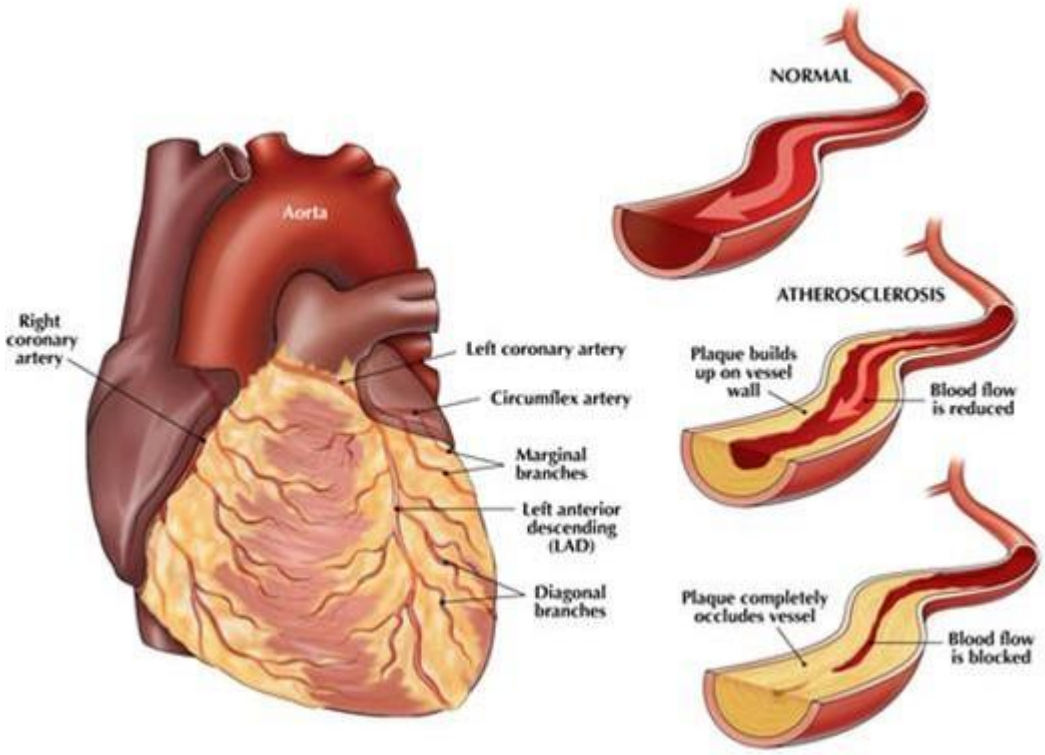

Figure.13: Coronary artery anatomy and degrees of atherosclerosis

Coronary heart disease can often be symptom-free but people with CHD have an increased risk of angina (chest pain or discomfort), heart attack, heart failure, and cardiac arrhythmias. Angina and heart attacks are caused by reduced or blocked blood flow to the heart. Stable angina will typically intensify with physical exertion and subside with rest but a heart attack can cause heart muscle death and requires emergency attention. Information about family history, medical history, and certain tests can aid in diagnosing CHD.

1.2.2 Valvular Heart Disease

Valvular involvement by disease causes stenosis, insufficiency (regurgitation or incompetence), or both. Stenosis is the failure of a valve to open completely, thereby impeding forward flow. Insufficiency, in contrast, results from failure of a valve to close completely, thereby allowing reversed flow. These abnormalities can be either pure, when only stenosis or regurgitation is present, or mixed, when both stenosis and regurgitation coexist in the same valve, but one of these defects usually predominates. Isolated disease refers to disease affecting one valve, and combined disease implies that more than one valve may be dysfunctional. Functional regurgitation results when a valve becomes incompetent owing to either (1) dilation of the ventricle, which causes the right or left ventricular papillary muscles to be pulled down and outward, thereby preventing cooptation of otherwise intact mitral or tricuspid leaflets during systole, or (2) dilation of the aortic or pulmonary artery, pulling the valve commissures apart and preventing full closure of the aortic or pulmonary valve cusps. Abnormalities of flow often produce abnormal heart sounds known as murmurs.

The most frequent calcific valvular diseases, illustrated in Figure. 14, are calcific aortic stenosis, calcificat ion of a congenitally bicuspid aortic valve, and mitral annular calcification.

Each comprises primarily dystrophic calcification without significant lipid deposition or cellular proliferation, a process distinct from but with some features of atherosclerosis.

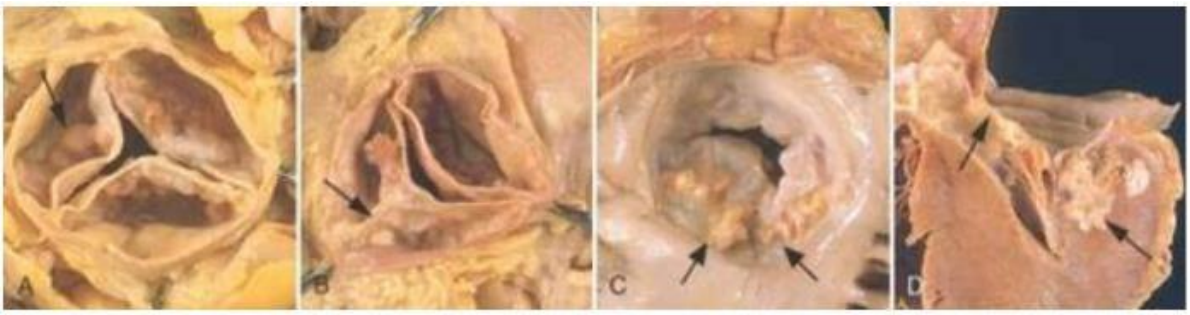

Figure.14: Calcific valvular degeneration. A, Calcific aortic stenosis of a previously normal valve having three cusps (viewed from aortic aspect). Nodular masses of calcium are heaped-up within the sinuses of Valsalva (arrow). Note that the commissures are not fused, as in postrheumatic aortic valve stenosis. B, Calcific aortic stenosis occurring on a congenitally bicuspid valve. One cusp has a partial fusion at its center, called a raphe (arrow). C and D, Mitral annular calcification, with calcific nodules at the base (attachment margin) of the anterior mitral leaflet (arrows).C, Left atrial view.D, Cut section of myocardium.

1.2.3 Cardiomyopathy

Cardiomyopathy is a pathologic disorder of the heart muscle that affects millions of individuals and accounts for approximately 23,000 deaths annually in the United States (NHLBI 2012). It is characterised by defects in the mechanical function of the heart, which often lead to heart failure syndrome or sudden cardiac death due to increased predisposition to lethal arrhythmias. Several classes of cardiomyopathy have been defined, mainly distinguished by their pathologic findings and clinical course, which can be attributed to inherited or acquired causes. It is well documented that acquired cardiomyopathy leads to myocyte injury via ischemia, increased loading conditions or systemic disorders affecting the

heart, but the pathophysiology of inherited cardiomyopathy remains poorly understood. As illustrated in Figure. 15, hypertrophic cardio-myopathy (HCM) is notable for

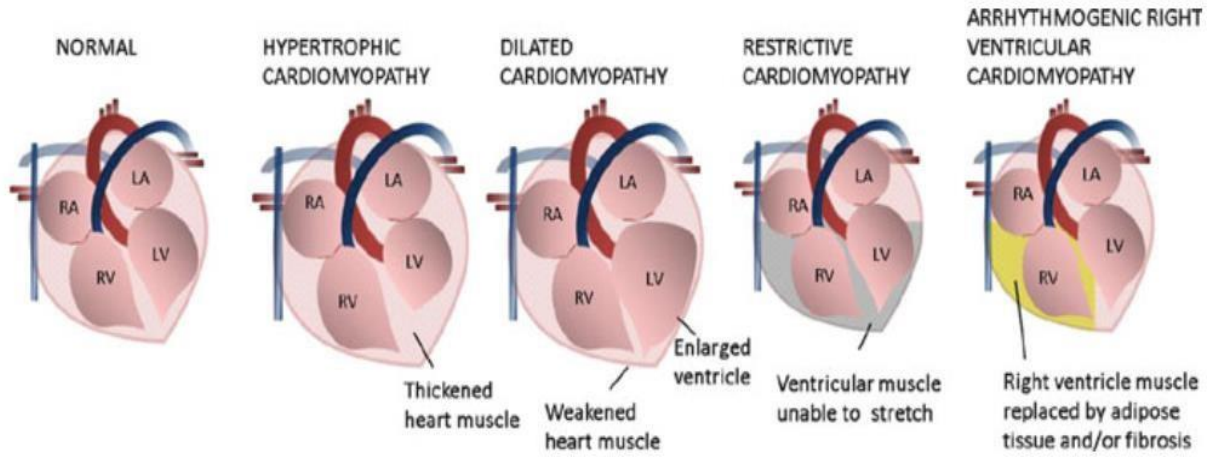

Figure.15: Classification of cardiomyopathies. Schematic diagram depicting structural changes that occur in cardiac muscle during cardiomyopathy. In dilated cardiomyopathy, progressive weakness in ventricular muscle is observed, accompanied by enlargement of one or both ventricular cavities, whereas in hypertrophic cardiomyopathy, enlargement is mostly in the left ventricular muscle, constricting the ventricle(s). Restrictive cardiomyopathy is characterised by impaired diastolic filling of one or both ventricles. Finally, arrhythmogenic right ventricular cardiomyopathy is typically characterised by fibro fatty deposition in the right ventricle

Increased relative thickness of (mostly) the left ventricle, which is often asymmetric and associated with hemodynamic significance. Dilated cardiomyopathy (DCM) is characterised by progressive dilation of one or both ventricular cavities, whereas restrictive cardiomyopathy (RCM) is characterised by impaired diastolic filling of one or both ventricles. Arrhythmogenic right ventricular cardiomyopathy (ARVC) is recognised most commonly by fibrofatty infiltration of the right ventricle leading to arrhythmic disorders and sudden death.

1.2.4 Cardiac arrhythmias

Cardiac arrhythmias are disturbances in the rhythm of the heart, manifested by irregularity or by abnormally fast rates (tachycardias) or abnormally slow rates (bradycardias). Patients who perceive these abnormalities most frequently observe palpitations,which some describe as the sensation of ‘my heart turning over in my chest’, or awareness that their hearts are beating rapidly or slowly. Other symptoms include weakness, shortness of breath, lightheadedness, dizziness, fainting (syncope) and, occasionally, chest pain. The symptoms tend to be more severe when the rate is faster, the ventricular function is worse, or the arrhythmia is associated with abnormalities of autonomic tone. However, many patients with arrhythmias report no symptoms, and the condition might first be discovered during a routine examination. A tachyarrhythmia that is rapid enough and lasts long enough can produce cardiomyopathy and congestive heart failure. In these cases, treatment of the arrhythmia can often return normal function to the ventricles.

1.3 Symptoms of Heart Disease

Coronary heart disease (CHD), also called coronary artery disease, occurs when plaque builds up inside the coronary arteries. These arteries supply your heart muscle with oxygen-rich blood. Plaque is made up of fat, cholesterol, calcium, and other substances found in the blood. Over time, plaque hardens and narrows the arteries, reducing blood flow to your heart muscle.

Eventually, an area of plaque can rupture, causing a blood clot to form on the surface of the plaque. If the clot becomes large enough, it can mostly or completely block the flow of oxygen-rich blood to the part of the heart muscle fed by the artery. This can lead to angina or a heart attack.

1.3.1 Angina

Angina pectoris is a symptom that occurs when blood supply to an area of the heart muscle doesn't meet its needs. Angina may be felt as heaviness below the breast bone which may spread to either arm, to the neck or the back. On occasion, angina can be an indigestion-like discomfort in the upper stomach or a burning or heartburn-like feeling below the breastbone. Angina may occur during physical activity, at rest or it may awaken you when you're asleep. Angina that becomes more frequent or severe, or that occurs at rest and lasts for longer periods of time, is of greater concern. This change in pattern of angina is known as *Unstable Angina* and maybe an early warning sign of a heart attack. When angina lasts for longer than 20 minutes, there is a risk that heart damage has occurred. If this occurs, you should call your doctor or, if there is any delay, have someone take you to the nearest hospital's Emergency Department.

1.3.2 Myocardial infarction

Myocardial infarction is the medical synonym for the term heart attack. It has also been known as coronary thrombosis or simply as "coronary". The pain of myocardial infarction lasts longer than that of angina pectoris. Generally there is prolonged, sudden crushing chest pain accompanied by shortness of breath, sweating, nausea, vomiting and perhaps lightheadedness. This pain may spread to the arms, neck, jaw, shoulders and back. For some people symptoms of angina and heart attack may be felt only as shortness of breath. For others, heart attacks may occur silently without any symptoms of chest pain or they may be passed off as mild indigestion.

1.4 Causes and Risk Factors of Cardiovascular Disease

Research suggests that CHD starts when certain factors damage the inner layers of the coronary arteries. These factors include smoking, high amounts of certain fats and

cholesterol in the blood, high blood pressure, and high amounts of sugar in the blood due to insulin resistance or diabetes.

When damage occurs, your body starts a healing process. This process causes plaque to build up where the arteries are damaged. The buildup of plaque in the coronary arteries may start in childhood.

Certain traits, conditions, or habits raise your risk for CHD. These conditions are known as risk factors. The major risk factors for CHD include:

• Unhealthy blood cholesterol levels

• High blood pressure

• Smoking

• Insulin resistance

• Diabetes

• Overweight or obesity

• Metabolic syndrome

• Lack of physical activity

• Age (as you get older, your risk for CHD increases)

• Family history of early heart disease

1.5 Relationship of Cardiovascular Diseases with Blood Group

ABO blood groups have been associated with various disease phenotypes, particularly cardiovascular diseases (H.Wang, M. K. Ohman et al., 2012). Recent GWASs have identified ABO as a locus for thrombosis, myocardial infarction, and multiple cardiovascular risk biomarkers, refocusing attention on mechanisms and potential for clinical advances (A.L'Abbate., 2010). The widespread use of genome-wide association studies (GWASs) over the last 5 years has spurred an enormous acceleration in discoveries across the entire

spectrum of CVD (T. Zeller, S. Blankenberg, and P. Diemert,. 2012). Recent GWASs have confirmed *ABO* as a locus for venous thrombo embolism (VTE), myocardial infarction (MI), and multiple cardiovascular biomarkers (R. C. Figueiredo et al., 2015).

1.6 Relationship of Cardiovascular disease with the size of the heart

Aetiological studies of myocardial ischaemia and myocardial infarction have tended to concentrate on factors which influence athero thrombotic processes in the coronary arteries, rather than directly on myocardial patho physiology (Chambers J., 2000). Left ventricular mass and left ventricular systolic dysfunction are among the more important measures of myocardial patho physiology (Levy D. et al., 2005). Although hypertrophy of the left ventricular myocardium has long been recognized as a strong predictor of coronary heart disease events, only recently has evidence of its independence as a coronary risk factor been available.

1.7 Relationship of Cardiovascular Diseases with Hypertension

Hypertension (HT) is a major risk factor for coronary heart disease (CHD). Among the numerous risk factors associated with CHD, HT plays a major role given its high frequency and its physio pathogenesis. Thus, roughly 15% of the general adult population manifest HT with a net male predominance, and 25% of patients with CHD have HT (Collins R, McMahon S. et al. 1994) . CHD is the first cause of morbidity and mortality in hypertensive patients. Numerous epidemiological studies have shown that the presence of HT increases the risk of CHD, not only in at risk populations but also in the general population. The prevalence of CHD is closely related to the BP level, especially systolic BP. This has been shown in studies of clinical BP and also in studies using ambulatory BP measurements(ABPM) (Kannel WB., 1996).

1.8 Relationship between Cardiovascular Diseases and Body Mass Index (BMI)

Obesity is considered an independent cardiovascular risk factor that is associated with poor clinical outcomes (Berghofer A., 2008). In the general population, a higher BMI is associated with an increased risk of coronary artery disease (CAD) (Flegal KM., 2007). Epidemiological and clinical studies in the general population have demonstrated that overweight and obesity increase the risk of developing chronic conditions such as diabetes, dyslipidaemia, hypertension, and cancers, and are associated with all cause and cardiovascular (CV) morbidity and mortality, independently of gender, age, and ethnicity (Schatzkin A., Jacobson A., 1998). However, a growing body of literature, including large meta-analyses, has recently revealed a phenomenon called the 'obesity paradox'(Lavie CJ.,2009).Indeed, obesity has been associated with better survival in some groups of patients, such as individuals with heart failure (Padwal R.,2008),diabetes (Palaniappan L.,2014), chronic heart disease (Oreopoulos A., 2007) and in patients with a history of coronary heart disease (CHD) (Karason K., 2013). Despite the well-known limitations of observational studies, Dixonet al.,in a commentary on the obesity paradox, considered that a body mass index (BMI) in the obese range may provide a survival advantage compared with that in the normal range. It may therefore be preferable to focus on good quality nutrition and physical activity rather than intentional weight loss, which has uncertain effects (Dixon JB., 2014).

There are, however, several limitations to previous studies. For example the relationship between obesity and mortality in patients with CAD and HF has been examined using analyses of retrospectively collected cohort data or post hoc analyses of randomized controlled trials originally designed to evaluate a specific drug or device (Mercado N., 2005) many of which have been limited by short follow-up times (Ghali WA., 2000).

1.9 Global Situation of Cardiovascular Diseases

At the beginning of this century, cardiovascular diseases (CVD) showed an epidemiological behavior very similar to those of the great endemics of past centuries and were responsible for high mortality rates worldwide (Buttler D., 2011). This current epidemiological profile of CVD is evident in data from the World Health Organization (WHO), which show that of the 56.9 million total deaths reported worldwide, approximately 30.5 % or 17 million people had CVD listed as the cause of death. Moreover, data released by the WHO in 2008 indicate that of the total number of CVD deaths worldwide, approximately 80.1 % occurred in low- and middle-income countries, and only 19.9 % occurred in high-income countries (WHO Statistical Information Center. World Health Statistics 2009 – En-WHS 2009.).If this global scenario is already alarming at the beginning of this new century, the expectations for the future are even more troubling; it is estimated that if concrete actions are not implemented, by the year 2030, seven out of ten deaths will be due to non-communicable diseases (NCDs), and CVD will account for the highest percentage of these deaths (Beaglehole R., 1999). However, although the CVD mortality rates in developed countries have been declining in recent decades, as previously mentioned, the rates in most developing countries are still increasing. This is due, among other factors, to the increased economic power of developing countries. Rising incomes *per capita* have led to improvement in the health and basic living conditions of these populations, resulting in a significant reduction in the incidence of and mortality from infectious and parasitic diseases, with a proportional increase in the number of deaths caused by NCDs. In addition, the lifestyle adopted by urban populations in developing countries has significantly increased the prevalence of risk factors for cardiovascular diseases such as obesity, physical inactivity, tobacco use, high blood pressure, excessive salt intake, dyslipidemia, and diabetes (World Health Organization, World Heart

Federation, World Stroke Organization. Global atlas on cardio-vascular disease prevention and control: policies, strategies, and interventions. 2011.)

More recent studies, however, reveal a balance between preventive and therapeutic actions in the fight against CVD. In 2007, an epidemiological analysis was published that used the validated IMPACT mortality model, and it showed a significant decrease in mortality rates due to coronary heart disease in both men and women in the USA between1980 and 2000 (Figure. 16).

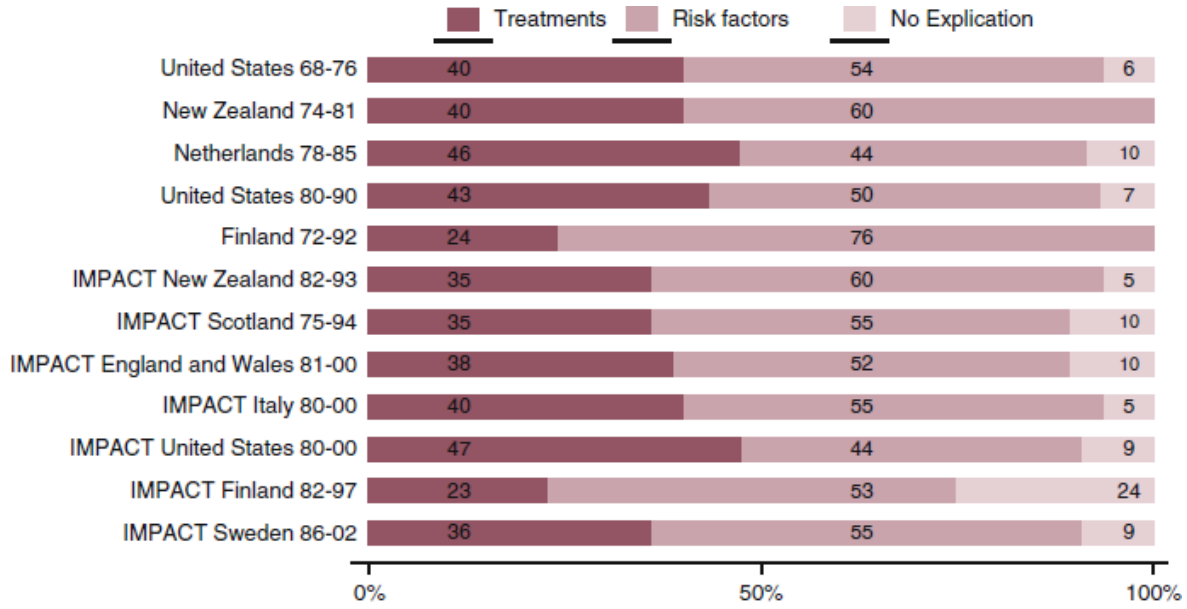

Figure.16: Percent decrease in the number of deaths from coronary heart disease attributed to changes in treatment and risk factors in different populations. (Adapted from Di Chiara and Vanuzzo)

Furthermore, it was concluded that approximately 44 % of this decrease was due to the control of several cardiovascular risk factors, while 47 %resulted from therapeutic actions. Preventive actions that contributed to this result included reductions in total cholesterol (24 %), systolic blood pressure (24 %), the prevalence of tobacco use (12 %) and physical inactivity (5 %). This result was counterbalanced by the significant increase in the prevalence of obesity and diabetes in this population (Di Chiara A, Vanuzzo D., 2009).

1.10 Perspective of Cardiovascular Diseases in Bangladesh:

The exact prevalence of CVD in Bangladesh is not known. Probably the first attempt to determine the prevalence of heart disease was made by Malik et al. in a survey amongst 7062 people of different age groups in Dhaka City and in a village; the surgery revealed the prevalence of 2.92%. 11 Self-reported prevalence of heart disease among the 25 to 64-year-old respondents were 5.3% to 66.3% in males and 7.8% to 77.7% in females in another study in 2005. 12 The wide range of prevalence is presumably due to differences in study design and methodology.

Like CAD, hypertension is an increasingly important medical and public health problem in Bangladesh. The reported prevalence varies widely from 1.21% to 32%.According to the Bangladesh NCD Risk Factor Survey 201027, the prevalence of hypertension is 17.9% in general, 18.5% in men and17.3% in women. According to a cross sectional study done by Bangladesh NCD Risk Factor Survey done in 2010 the prevalence rate was found to be 17.9%. Again, just two years later, another survey revealed this rate to be 18.5% (Cravedi et al.). One of the most recent meta - analysis on issue reveals the prevalence rate to be 15.1% (Fatema et al., 2016). On the other hand, overall, age standardized prevalence of prehypertension and hypertension were 27.1 and 24.4%, respectively, in a recently published analysis based on the nation wide population-based 2011 Bangladesh Demographic and Health Survey (BDHS). Even higher prevalence of hypertension of 40% (95% confidence interval (CI) 38-42%) was found in a population-based study involving 3096 adults aged >30 years from rural Bangladesh. A recently-published meta-analysis concerning risk factors for CVD in Bangladesh found the prevalence of hypertension to be 15.1%.CVDs are emerging as an epidemic in a resource poor country like Bangladesh. So it will be better if we can prevent the risk factors of CVDs.

Little is known regarding the incidence and prevalence of congenital heart disease (CHD) in Bangladesh. A proportion of CHD in children may remain undetected unless specific efforts are made to diagnose them. In a prospective, hospital based study conducted over January 2006 to December 2008 in the Pediatric Cardiology unit of Combined Military Hospital (CMH)Dhaka, 142 babies out of 5668 live birth had CHD, giving an incidence of 25/1000 live births. Most common CHDs were atrial septal defect (ASD, 26%), ventricular septal defect (VSD, 16.9%), patent ductus arteriosus (PDA,18%), tetralogy of Fallot (TOF, 14%),and pulmonarystenosis (PS, 7.75%).46 Another study conducted in Dhaka Shishu Hospital from January 2008 to December. So risk factor identification is the only way to achieve this goal. In this regard this study will find out the risk factors and prevention strategies of CVDs that are needed to develop effective national health policy to prevent and manage them. This way, we can reduce morbidity and mortality among CVD patients and alleviate the burden of CVDs. This paper explored the availability of literature on risk factors and prevention strategies of CVDs in Bangladesh through a scoping review which, unlike a systematic review, offers a much broader perspective in the respective field which makes it more appropriate method to assess the risk factors and prevention strategies of CVDs in Bangladesh.

2. Methods and Materials

2.1 Place of study

The study was carried out in an institution specialized in research, diagnosis and treatment of heart patients. The name of the institution is “Ibrahim Cardiac Hospital & Research Institute” situated in Shahbagh, Dhaka.

2.2 Duration of study

The study was carried out from January to November, 2018 for 11 (eleven) months.

2.3 Population of study

The population of the study was heart patients who were registered at the institute. A total of 502 patients’ data were taken under the study for investigating their cases.

2.4 Methodology

The patients who were with heart complications and registered at the institution were the subjects of the study. From the patients file, respective data were collected for further analysis. The statistical methods include, frequency tables, cross table analysis, graphs and charts. The software used for statistical analysis was SPSS, developed by the IBM.

3. Objective

3.1 Primary Objective

The objective of the study is to identify major risk factors, determining a critical age range for onset of heart disease and characterizing the occurrence of heart disease in Bangladesh by analyzing different cofactors on a retrospective study.

3.2 Secondary objective

Secondary objects of the study include:

1. To collect the information and knowledge about heart disease.
2. To verify whether heart problems depends on age and sex with the objectives.
3. To learn the specialties of heart patients.
4. To review the facts based on different variables.
5. To evaluate of the factors taking into consideration of different categories.

4. Results and Discussion

4.1 Distribution of the Heart Patients based on gender

Data of five hundred and two (502) patients were studied. Among them, two hundred and seventy nine (279) were male and the remaining two hundred and twenty three (223) were female.

Table 01: Frequency Table of distribution of Heart Patients based on gender

Gender	Frequency	Percentage (%)
Male	279	55.6
Female	223	44.4
Total	502	100.0

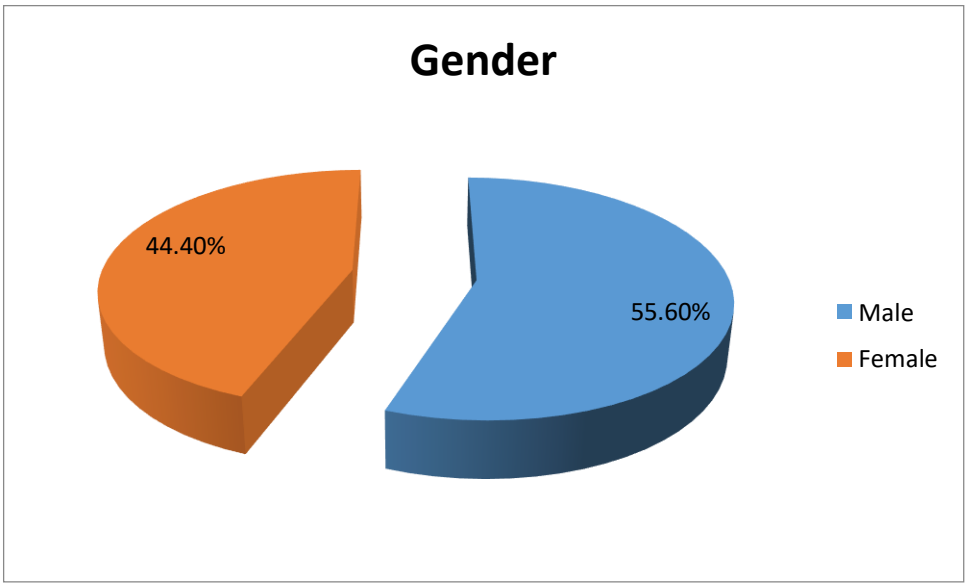

Figure 17: Distribution of heart patients based on gender

It was observed in the study that, the prevalence of heart disease is highest in males. From a total of 502 patients, 279 (55.6%) were males and 223 (44.40%) were females.

4.2 Distribution of Heart Patients based on Age range

All the patients fall in the age range of 10 to 85. Out of the total number of patients, age group of 10 to 30 years had 1 patient, 31 to 50 years had 5 patients, and 51 to 85 years had 496 patients.

Table 02: Frequency Table of Distribution of Heart patients based on age range

Age	Frequency	Percentage (%)
10 - 30	1	0.2
31 - 50	5	1.0
51 - 85	496	98.8
Total	502	100.0

It was observed from the study that the highest number of heart patients fall in the age group of 51 to 85 years (98.8%).

4.3 Distribution of Age Range of the Heart Patients based on Gender

It was identified earlier that, the most critical age range for the occurrence of Heart Disease is 51-85 years.

Table 03: Cross Table Analysis between Gender of the Heart Patients and their Age Range

Age	Gender				Total
	Male	Percentage (%)	Female	Percentage (%)	
10 - 30	1	0.4	0	0.0	1
31 - 50	3	1.1	2	0.9	5
51 - 85	275	98.6	221	99.1	496
Total	281	100	221	100	502

Among the 281 male patients, age group of 10 to 30 years had 0.4% patients, 31 to 50 years had 1.1% patients, and 51 to 85 years had 98.6% patients.

On the other hand, out of 221 female patients, age group of 10 to 30 years had 0% patients, 31 to 50 years had 0.9% patients, and 51 to 85 years had 99.1% patients.

It can be observed from the data assembly that, in both male and female patients, most of the patients are from age range of 51 to 85 years. The next critical age range is 31 to 50 years.

4.4 Distribution of Heart Patients based on Body Mass Index (BMI)

Body Mass Index (BMI) is a measurement of body fat based on weight and height of a person and that applies to both men and women. BMI is used to indicate if a person is underweight, normal, overweight or obese.

BMI value of less than 18.5 indicates underweight, 18.5 to 24.9 is normal, 25 to 29.9 is overweight and 30 and above is considered as obese. BMI of a person can be calculated by using the following formula:

BMI= Weight (Kilograms) / [Height (m)]^2

Table 04: Frequency Table of Distribution of Heart patients based on Body Mass Index (BMI)

BMI	Frequency	Percentage (%)
Less than 18.5	32	6.4
18.5 - 27	308	61.4
27.1 –29.9	121	24.1
30 and above	41	8.1
Total	502	100.0

In patients under the study, BMI value was found less than 18.5 for 6.4% patients, from 18.5 to 27 for 61.4% patients, from 27.1 to 29.9 for 24.1% patients and 30 and above for 8.1% patients.

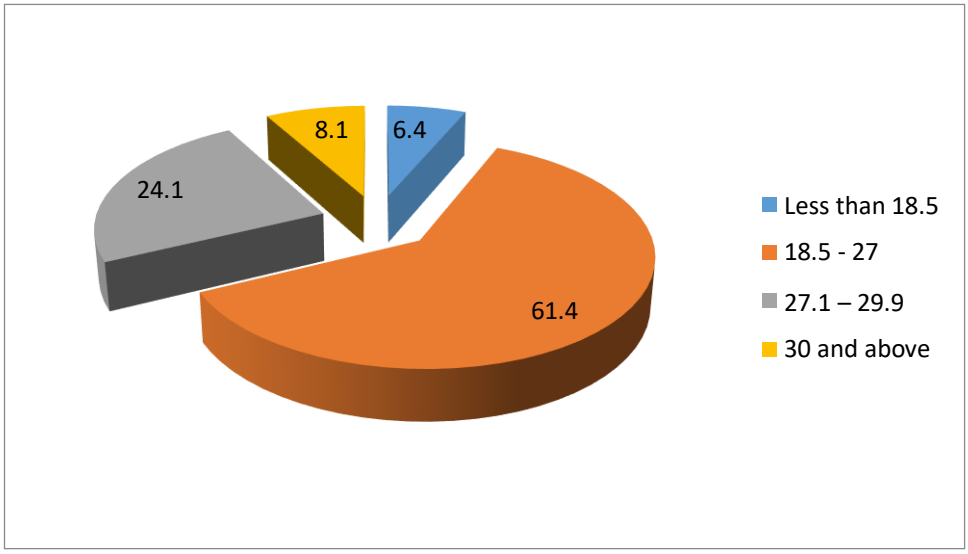

Figure 18: Distribution of Heart Patients based on Body Mass Index (BMI)

4.5 Distribution of BMI of the Heart Patients based on Gender

It was observed from the total patients that, most of the patients (61.3%) fall in the BMI value of 18.5 to 27. On the other hand, a high percentage of patients (25.89%) were overweight, 8.14% patients were obese and only 6.37 % of the patients were underweight.

Table 05: Cross Table Analysis between the Gender of the Heart Patients and their BMI

BMI	Gender				Total
	Male	Percentage (%)	Female	Percentage (%)	
Less than 18.5	16	5.7	16	7.2	32
18.5 - 27	176	62.4	132	60.1	310
27.1 – 29.9	67	24.0	54	24.2	121
30 and above	22	7.9	19	8.5	41
Total	281	100	221	100	502

Out of 279 male patients, only 5.7% was underweight, 60.93% patients were in normal weight, 25.08% patients were overweight and remaining 8.29% patients were obese. On the

other hand, out of 223 female patients, only 7.2% patients were underweight, 58.29% patients were in normal weight, 25.56% patients were overweight and remaining 8.95% patients were obese.

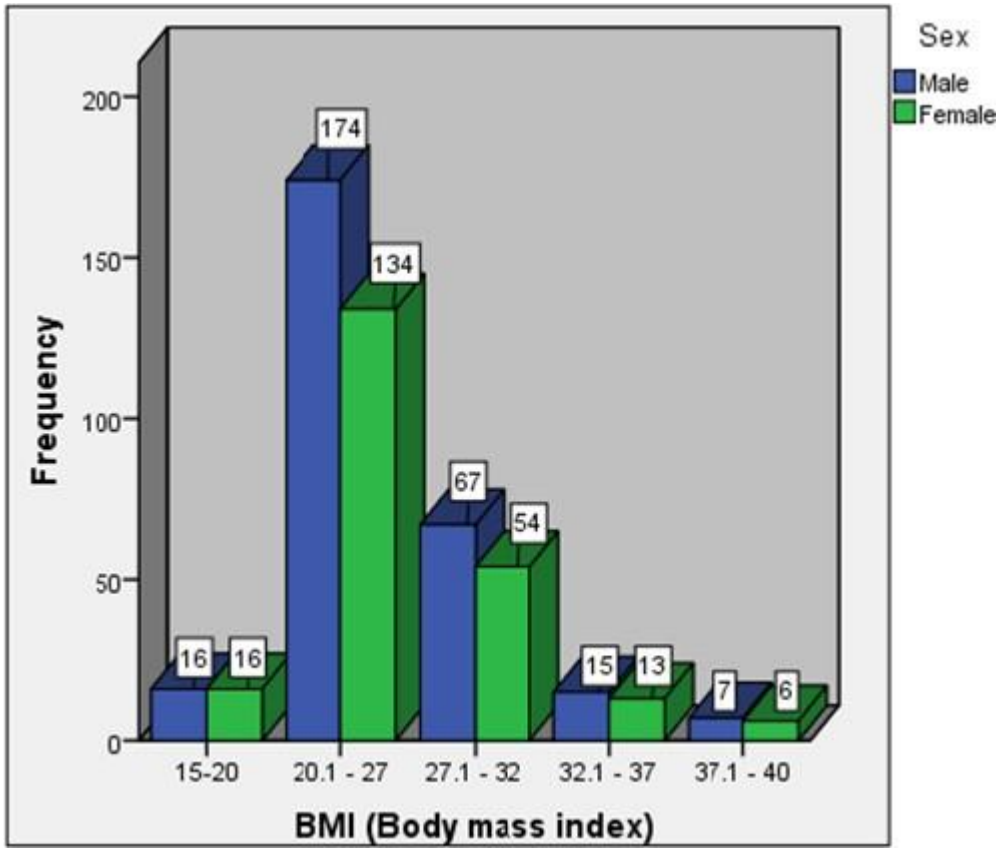

Figure 19: Distribution of male and female patients based on Body Mass Index (BMI)

4.6 Distribution of Total Cholesterol level of the HeartPatients

Less than 200 mg/dL is the desirable level that puts a person at lower risk for coronary heart disease. A cholesterol level of 200 mg/dL or higher raises the risk. 200 to 239 mg/dL is the Borderline high. 240 mg/dL and above indicated high blood cholesterol. A person with this level has more than twice the risk of coronary heart disease as someone whose cholesterol is below 200 mg/dL.

From the total 502 patients, 214 had a cholesterol level of less than 200. 151 of them had a cholesterol level between 200 and 239. 137 of them have a level above 24 which indicates high blood cholesterol.

Table 06: Frequency table of Distribution of Total Cholesterol level of the Heart Patients

Total Cholesterol level (mg/dL)	Frequency	Percentage (%)
Below 200	214	42.64
200-239	151	30.07
240 and above	137	27.29
Total	502	100

4.7 Distribution of Heart Patients based on their Triglyceride level

According to the guidelines set by The National Cholesterol Education Program for triglyceride levels, less than 150 (mg/dL) is considered to be the normal level. 150 to 199 is Borderline high. 200 and above is considered to be an indication of high level of Triglyceride.

Table 07: Frequency table of Distribution of Heart Patients based on their Triglyceride level

Triglyceride level (mg/dL)	Frequency	Percentage (%)
Below 150	51	10.15
150-199	82	16.33
200 and above	369	73.52
Total	502	100

From the data assembly, it can be observed that, most of the patients (73.52%) had a high triglyceride level. 16% of the patients triglyceride level was borderline high and 10% patients had normal triglyceride level.

4.8 Distribution of Triglyceride level of the Heart Patients based on Gender

It can be observed from the data assembly that, among 281 male patients, 9.96% had a normal value, 14.96% had a borderline value and the remaining 75% had a higher value.

Table 08: Frequency table of Distribution of Triglyceride level of the Heart Patients based on Gender

Triglyceride level (mg/dl)	Gender				Total
	Male	Percentage (%)	Female	Percentage (%)	
Below 150	28	9.96	23	10.41	51
150 - 199	42	14.96	40	18.09	82
200 and above	211	75.08	158	71.50	369
Total	281	100	221	100	502

On the other hand, from 221 female patients, only 10.41% patients had a normal value, 18.09% had a borderline value and the remaining 71.50% had a higher value.

4.9 Distribution of Heart Patients based on their LDL (low-density lipoproteins)Cholesterol level

LDL cholesterol levels should be less than 100 mg/dL. Levels of 100 to 129 mg/dL are acceptable for people with no health issues but may be of more concern for those with heart disease or heart disease risk factors. A reading of 130 to 159 mg/dL is borderline high and 160 to 189 mg/dL is high.

Table 09: Frequency table of Distribution of Heart Patients based on their LDL (low-density lipoproteins) Cholesterol level

LDLCholesterol level (mg/dL)	Frequency	Percentage (%)
Below 100	3	0.59
100-129	0	0
130-159	11	2.20
160 and above	488	97.21
Total	502	100

From the data assembly, it can be observed that, most of the patients (97.21%) had a high LDL level. 2.20% of the patients LDL level was borderline high and only 0.59% patients had normal triglyceride level.

4.10 Distribution of LDL Cholesterol level of the Heart Patients based on Gender

It can be observed from the data assembly that, among 281 male patients, 0.71% had a normal value, 2.85% had a borderline value and the remaining 96.44% had a higher value.

Table 10: Frequency table of Distribution of LDL Cholesterol level of the Heart Patients based on Gender

LDLCholesterol level (mg/dl)	Gender				Total
	Male	Percentage (%)	Female	Percentage (%)	
Below 100	2	0.71	1	0.45	3
100-129	0	0	0	0	0
130-159	8	2.85	3	1.36	11
160 and above	271	96.44	217	98.19	488
Total	281	100	221	100	502

Furthermore, from 221 female patients, only 0.45% patients had a normal value, 1.36% had a borderline value and the remaining 98.19% had a higher value.

4.11 Distribution of Chloride level of the Heart patients based on Gender

From 281 male patients, only 25 had a low chloride level, 52 patients had a level of (71-97) mmol/l, 117 patients had a level of (98-106) mmol/l, 87 patients had a level of (107-119) mmol/l and no patients had the level of more than 119.

From 221 female patients, only 1 had a low chloride level, 24 patients had a level of (71-97) mmol/l, 136 patients had a level of (98-106) mmol/l, 60 patients had a level of (107-119) mmol/l and no patients had the level of more than 119.

Table 11: Cross Table Analysis between Chloride level of the Heart Patients and their gender

Chloride (mmol/l)	Gender				Total
	Male	Percentage (%)	Female	Percentage (%)	
0-70	25	8.90	1	0.45	26
71-97	52	18.50	24	10.86	76
98-106	117	41.63	136	61.54	253
107-119	87	30.97	60	27.15	147
Above 119	0	0	0	0	0
Total	281	100	221	100	502

4.12 Distribution of Heart Patients based on their Blood group

From all the 502 patients, 7.17% patients had A+(ve), 7.17% had A-(ve), 12.75% had B+(ve), 8.17 had B-(ve), 27.3% had AB+(ve), 18.92% had AB-(ve), 8.76% had O+(ve), and 9.76% had O-(ve) .

Table 12: Frequency Table Analysis of Distribution of Heart Patients based on their Blood group

Blood group	Frequency	Percentage (%)
A+(ve)	36	7.17
A-(ve)	36	7.17
B+(ve)	64	12.75
B-(ve)	41	8.17
AB+(ve)	137	27.3
AB-(ve)	95	18.92
O+(ve)	44	8.76
O-(ve)	49	9.76
Total	502	100

4.13 Distribution of Blood groups of the Heart Patients based on Gender

From 281 male patients, majority of them had AB-+(ve) blood group.

On the other hand, from 221 female patients, majority of them had also AB+(ve) blood group.

Table 13: Cross Table of Analysis between the Blood group of the Heart Patients and their Gender

Blood group	Gender				Total
	Male	Percentage (%)	Female	Percentage (%)	
A+	20	7.12	16	7.24	36
A-	11	3.91	25	11.31	36
B+	2	0.71	62	28.05	64
B-	37	13.18	4	1.81	41
AB+	58	20.64	79	35.75	137
AB-	86	30.60	9	4.08	95
O+	22	7.83	22	9.95	44

O-	45	16.01	4	1.81	49
Total	281	100	221	100	502

4.14 Distribution of Heart Patients based on their Random Blood Sugar (RBS)

Normal Random Blood Sugar (RBS) range in adult is (4.4-7.8) mmol/l. From the data assembly, it can be observed that, RBS range is (0-4.3) mmol/l in 3.78 patients, (4.4-7.8) mmol/l in 25.70% and 7.9 mmol/l and above in 70.52% patients. Therefore, it can be stated that, most of the patients (70.52%) had a high Random Blood Sugar.

Table 14: Frequency Table Analysis of Distribution of Heart Patients based on their Random Blood Sugar (RBS)

RBS (mmol/l)	Frequency	Percentage (%)
0-4.3	19	3.78
4.4-7.8	129	25.70
7.9 and above	354	70.52
Total	502	100

4.15 Distribution of Heart Patients based on their Systolic Blood Pressure

Most of the heart patients had an irregular blood pressure and they were suffering from hypertension. Moreover, systolic blood pressure of the patients were randomly measured. The majority of the patients had a high systolic blood pressure.

Table 15: Frequency Table of Distribution of Heart Patients based on their Systolic Blood Pressure

Systolic BP (mmHg)	Frequency	Percentage (%)
80-90	6	1.20
91-100	32	6.37
101-120	72	14.34

121-140	148	29.48
Above 140	244	48.61
Total	502	100

From the data assembly, it can be observed that, 48.61% patients had a high systolic blood pressure. 29.48% of the patients had a systolic blood pressure of 121 to 140 mmHg and it can be considered that, they had a moderately high systolic BP.

4.16 Distribution of Heart Patients based on their Diastolic Blood Pressure

Diastolic blood pressure of the patients were randomly measured. The majority of the patients had a high diastolic blood pressure.

From the data assembly, it can be observed that, 25.90% patients had a high diastolic blood pressure. The maximum percentage (62.75%) of the patients had a diastolic blood pressure of 80 to 90 mmHg and it can be considered that, they had a moderately high diastolic BP.

Table 16: Frequency Table of Distribution of Heart Patients based on their Diastolic Blood Pressure

Diastolic BP (mmHg)	Frequency	Percentage (%)
50-79	57	11.35
80-90	315	62.75
Above 90	130	25.90
Total	502	100

4.17 Distribution of Hypertension in Heart Patients based on Gender

It has been found that, from all the patients, 289 patients had Hypertension. From them, 52% were male patients and 48% were female patients. This analysis shows that, the prevalence of hypertension in Heart patients is almost similar in both male and female patients.

Table 17: Frequency Table of Distribution of Hypertension in Heart Patients based on Gender

Gender	Frequency	Percentage (%)
Male	146	52
Female	143	48
Total	289	100

4.18 Distribution of duration of Hypertension in Heart Patients based on Gender

From 146 male patients with hypertension, 59.59% were suffering for 1 to 10 years, 31.51% were for 11 to 20 years and only 8.90% patients were suffering for more than 20 years.

On the other hand, from 143 female patients with hypertension, 55.94% were suffering for 1 to 10 years, 30.04% were for 11 to 20 years and 13.97% patients were suffering from hypertension for more than 20 years.

Table 18: Cross Table Analysis between the duration of Hypertension in Heart Patients and their Gender

Duration (Years)	Gender				Total
	Male	Percentage (%)	Female	Percentage (%)	
1-10	87	59.59	80	55.94	34
11-20	46	31.51	43	30.09	18
Above 20	13	8.90	20	13.97	10
Total	146	100	143	100	289

5. Conclusion

This is evident from the study that, there is a noticeable increasing trend in the prevalence of Heart Disease particularly Chronic Heart Disease in present Bangladesh.

An alarming prevalence rate of Heart disease is observed in the age group of 51-60 and above 60 years. This data was proved almost similarly predominant in both male and female subjects. This analysis also show the negative impacts of high blood pressure and uncontrolled blood sugar regulation on the Hearts since both of these diseases interrupt the functions of the Hearts.

A strong association of both hypertension Chronic Heart Disease (CHD) was also found where the disease lie in concerning percentage (57.57%) on the subjects and remaining 42.43% other heart diseases. Besides these, most of the patients' (27.3%) blood group was AB+(ve).

Lifestyle can be a major point of concern behind the rise in number of Heart patients in our country. As a higher percentage of overweight (61.4%) and obese (24.1%) patients found from the study, maintaining a regular body weight is necessary to fight against Heart problems. Moreover, extra body weight increases the risk of diabetes and hypertension where both of the diseases are considered as the major risk factors of Chronic Heart Disease.

Drinking impure water is also dangerous for the Hearts as it may contain several harmful chemicals or metals. Therefore, it is much important to drink pure water in an adequate amount to facilitate the proper functioning of the Hearts.

Therefore, along with further studies, we need to be more concerned about maintaining a healthy weight by eliminating high calorie foods from our diet as well as doing some physical exercise regularly to correlate lifestyle with onset of Heart Diseases in our country.

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