Venerable Professor Samuel Obiajulu Ike

"the matter of the heart *assumes that every attention, concern, deep interest, or simply put, all that matters for deeper knowledge, research, inquiry in science, art and medicine as fields of vast study, is the heart."*

THE MATTER OF THE HEART

- The Vice Chancellor
- Distinguished Academics
- My Lords Spiritual and Temporal
- Distinguished Ladies and Gentlemen
- Lions and Lionesses

1.0 PREAMBLE

1.1. Welcome

It is my unique, singular and humble privilege and pleasure to welcome you to this Inaugural Lecture, on the day that the Lord has made. We shall rejoice and be glad in it.

The application to be permitted to deliver an Inaugural lecture, was penned the very next day after the very first lecturer from the Department of Medicine of this great University – a worthy mentor, teacher pacesetter, and not surprisingly, a Cardiologist too, Prof BJC Onwubere, had delivered his Inaugural Lecture, on 26th March, 2013 – after over 45 years of existence as a department. Prof Onwubere, had *inter alia* prayed in his preamble, that "the first Inaugural Lecture from my Department of Medicine….. would open up the flood gate for more lecturers from the department…."[1]. And here, today, is his answered prayer!

I most heartily give all the glory to God, in whom we live and move and have our being, the Paragon Omniscient Cardiologist.

I sincerely thank our most ebullient, pragmatic and visionary Vice Chancellor, Prof B.C. Ozumba, for the authorization and goodwill of the University of Nigeria to apportion this day for my lecture.

I, certainly am thankful, to the busy Senate Ceremonies Committee, under the chairmanship (of Prof Obi Njoku then, and)

now Prof Uchenna Nzewi, for according me the opportunity and necessary assistance.

And to all of you, who have made out time to come, to be present in this occasion, I cannot thank you enough. May the Good Lord bless you indeed.

1.2 Locating the Topic: Play of Words/Meanings or Expressions?

An Inaugural Lecture, in essence, should capture the active works, research, publications and general contributions which the potential lecturer, the professor, in his academic life, has passionately pursued and still envisions to be found in - albeit not for the sake of re- inventing the wheel – for the interest of the academia and the general public.

When, therefore, it became obvious that the epicentre of my life work, interest and pursuits, predating, and even beyond the four walls of the university and the altar of research has been **the heart** (perhaps my middle name, **Obiajulu**- "the heart is at peace" – prophetically, it now seems, christened by my parents, had been the pointer), it was a daunting but interesting exercise to choose the topic. A topic that would be of general – and not exclusively scientific or medical conclave - interest.

In my house is a brilliant team of passionate and incisive analysts – made up of the wife of my youth, Ruby, and my beloved children. We debated variously, at great length, which of the phrases, **the heart of the matter** or **the matter of the heart** best represented the essence of this lecture. Either of them seemed apt, each in its own angle, to very well justify why it should be the commanding topic.

For wordsmiths, **the heart of the matter** in a manner of speaking – refers to the vital or essential part, the core of a matter; and for good measure, is **the human heart** – physiologically, anatomically located, in the circulatory system – not the centre of the body/matter? Metaphorically, in daily

language expressions, is the heart not just regarded, in figure of speech, as the centre of the total personality – especially with regards to intuition, feeling or emotion but also the innermost or central part of anything or matter, chief of which is the human body and being?

And yet profoundly going for it, **the matter of the heart** assumes that every attention, concern, deep interest, or simply put, all that matters for deeper knowledge, research, inquiry in science, art and medicine as fields of vast study, is the heart. In the same vein, **all that matters** for meaningful existence in mundane emotional dealings of intimate proportions, rational decision making processes, as well as in sacred devotion and discourse, **is the heart**.

And so opens the door*, just opens*, into the deep, multifaceted and multidimensional angles, meanings, and for its measure, the reality of the heart.

Permit me, therefore highly esteemed listener/reader, to only make an attempt, in practical terms, in all modesty- knowing already our limitations as not possessing all knowledge about the heart from the foregoing, and our limited space and time frame – a peep, an overview, into **the matter of the heart**. This we will do etymologically, figuratively, in sacerdotal homily, and also medically, albeit from the physician's stand point (*a' la* the watch post of Habakkuk in the Holy Book).

Do, then please welcome to the matter of the heart!

1.3. Justification (Background)

What work, out of the quanta of knowledge of inestimable depth and range, available now, have we been able to do, with regards to the matter of the heart? I must also quickly settle your mind on the course this lecture is taking to explain the deposition inter alia, that the epicentre of my lifework and walk has been the heart, and justifiably so too.

The question has come often times of our work; *"You are a Cardiologist and also a priest, how do you reconcile, cope with, marry the two (vocations)?"* Our sincere humble answer has been, "Challenging, but easily so. As a **Cardiologist**, we deal with the matter of the heart physiologically and pathologically; as a **Priest,** we are enabled to deal with the heart spiritually and holistically, with ultimate eternity in view. They are both **matters of the heart**." This lecture serves, aptly, therefore, to take on works in these perspectives.

Suffice it to say that our heart beat is to pursue the heart beat of God. This matter of the heart thus entails winning souls to God's cause, for right living, imparting knowledge for purposeful existence, peace – filled and fulfilling destiny – in good cardiovascular, overall health (according to the World Health Organization's prescribing definition of health)-for men and women of all races, creed, stations in life and background.

Of course, needless to say that ***God***, Himself, is the Paragon Cardiologist-who not only searches the heart but examines the mind (*Jer. 17:10*). ***Jesus Christ***, the Saviour of our Souls, is the Quintessential Cardiologist – Interventional, and Cardiothoracic Surgeon – all rolled into one - for while men can only attempt the palliation, mending or repair of the physical and physiological structure of the heart, He constantly and consistently has been doing Closed and Open Heart Surgeries as well as Heart Transplant procedures - not with donor, but completely new (naïve) hearts of priceless salvific, spiritual and physical value, AT NO CHARGE, with never a consultation fee!

2.0 INTRODUCTION

In the course of this lecture, we will be addressing the matter of the heart, with focus on:

i) Definitions

ii) Figures of Speech

iii) Biblical Perspective

iv) Medically

2.1 Definitions

2.1.1: Origin – The origin of the word 'heart' came from the old English word (before 900 AD) – *heorte*, and later evolved from the Middle English word, *herte.* It has a bearing from the Latin word – *cor*, and the Greek expression of same, *kardia* (cardio)[2].

2.1.2: In usage, metaphorically, as a figure of speech, the heart refers to the centre of the personality, especially with reference to intuition, feeling or emotion[3].

2.1.3: From the Biblical perspective, the heart is the centre not only of spiritual activity but of all the operations of human life, the home of personal life, and hence a man is designated according to his heart[4].

2.1.4: Medically, the heart is the hollow muscular, chambered organ that is the centre of the circulatory system[5].

3.0 THE HEART IN FIGURE OF SPEECH

Among all the many and varied figurative expressions involving the heart, these one-third ($^1/_3$) of a century (34) forms[3] come to fore:

3.1. Heart as a Noun

(i) The *centre of the total personality* especially with reference to intuition, feeling or emotion (In your heart you know that he was speaking the truth).

(ii) The *centre of emotion*, especially as contrasted to the head as the centre of the intellect (His head told him to argue away eternity, but his heart told him it was real).

(iii) *Capacity for sympathy*, feeling, affection (His heart moved him to help that sick man).

(iv) *Spirit*, courage, or enthusiasm (The mother's heart picked up when she saw her daughter's confident expression).

(v) The *innermost* or central part of anything (The Nnamdi Azikiwe Stadium stands in the very heart of Enugu city).

(vi) The *vital* or essential part/core: the heart of the matter.

(vii) A *conventional shape* with rounded sides meeting in a point at the bottom and arriving inward to a cusp at the tip ()[6].

(viii) Symbolically, as a *logograph*, for the English verb "to love", which derived from the use in " I  NY " (I love New York), introduced in 1977[7].

3.2 The Heart in Idioms

(ix) *After one's heart* –In keeping with one's taste, tendencies or preference (There's a man after my heart).

(x) *At heart* – In reality / Fundamentally / Basically (At heart he is a workaholic)

(xi) *Break someone's heart* – Cause someone great disappointment or sorrow/as to disappoint in love (The breaking of their engagement broke her heart).

(xii) *By heart* – By memory/ word-for-word (They knew the song by heart).

(xiii) *Cross one's heart* – To maintain the truth of one's statement /affirm one's integrity (That is the exact truth, I cross my heart).

(xiv) *Do someone's heart good* – Give happiness or pleasure to/ delight (It does my heart good to hear from you).

(xv) *Eat someone's heart out* – Have sorrow or longing dominating one's emotions/grieve inconsolably (The children are eating their hearts out over their lost toy).

(xvi) *From the bottom of one's heart* – With complete sincerity (He took to teaching from the bottom of his heart).

(xvii) *Have a heart* – To be compassionate or merciful (Please have a heart and give the erring student another chance).

(xviii) *Have at heart* – To have as an object, aim or desire (To have another's best interest at heart).

(xix) *Set one's heart on* – To have as one's ambition to obtain/ covet (The student set his heart on passing that subject with distinction).

(xx) *Set one's heart against* – To be unalterably opposed to (The physician had set her heart against prescribing experimental drugs for that patient).

(xxi) *Set one's heart at rest*- To dismiss one's anxiety (The husband could not set his heart at rest until the wife had delivered safely).

(xxii) *Have one's heart in one's mouth* – To be very anxious, apprehensive or fearful (The recent Ebola outbreak has put many a people's heart in their mouth).

(xxiii) *Have one's heart in the right place* – To be fundamentally kind, generous or well – intentioned (That lecturer may have a stern look, but his heart is in the right place).

(xxiv) *Heart and soul* – Enthusiastically, absolutely, fervently, completely (They entered heart and soul into the church activity).

(xxv) *In one's heart of hearts* – In one's private thoughts or feelings deep within one (He knew, in his heart of hearts that the health condition was critical).

(xxvi) *Lose one's heart* – To fall in love with (He lost his heart to his university course-mate).

(xxvii) *To lose heart* – To become despondent or disillusioned over something (That set back in business due to the fire accident made him lose heart).

(xxviii) *Near one's heart* – Of great interest or concern to one (To read medicine is very near to that young lady's heart).

(xxix) *Not have the heart* – To lack the necessary courage to do something (The HOD did not have the heart to tell the student that he had failed for the 6th time).

(xxx) *Take heart*- To regain one's courage (His daughter's death was a great blow, but he eventually took heart, resigning in God's will).

(xxxi) *To one's heart content* – Until one is satisfied / as much or as long as one wishes (The students prayed on the matter to their heart's content).

(xxxii) *Wear one's heart on one's sleeve* – to make one's intimate feelings or personal affairs known to al l / openly (That troubled patient was not the kind who would wear her heart on her sleeve).

(xxxiii) *Take/lay to heart* – To think seriously about/concern oneself with (The professor took to heart the issue of where to spend eternity).

(xxxiv) *With all one's heart* – With earnestness or zeal (The most fulfilling pursuit in life and eternity is to love the Lord your God with all one's heart).

4.0 THE HEART OF MAN FROM A BIBLICAL PERSPECTIVE

The Bible has much to say about the heart of man. The word "heart" in the New King James Version (NKJV) of the Bible, is found 835 times in 775 verses (according to E-sword)[8] . The study of/ **the matter of the heart** of man is thus a massive one, and need I add – from the human point of view – a complex one. God alone knows all of our hearts, "for you alone know the hearts of all the sons of men" (*1 Kings 8:39 NKJV*). And herein is the POSER: Do you and I know our own heart?

The essence of the Biblical perspective of the heart is this: when we read passages in the Bible about men deceiving themselves *(Galatians 6:3; James 1:26*) it causes us all the more concern for the question becomes am I being honest with myself? We definitely need to learn all we can about the human heart with the hope, expectation and intention that we can come to know ourselves better, and improve our heart, and by this matter of the heart, IMPROVE OUR LIVES, reasons for existence and our destiny or don't we have one?

Such questions as, "Who is the real me? Who am I? What is the true state/matter of my heart (not what I think about it) but what is its true state in reality?"

When the scripture speaks of the human heart it is addressing the thinking of a man, the will, a man's emotions or feelings, a man's conscience, or any given combinations of these. This matter of the heart may also have reference to the whole inner being of man combing all these elements/dimensions into the one whole that makes up the man. A look at each of these aspects of the matter of the human heart:

4.1 The heart is the thinking aspect of man

"For as he thinks in his heart, so is he!"

- Jesus asks, "Why do you think evil in your heart?"
- "For out of the heart proceeds evil thoughts."
- "But Mary kept all these things and pondered them in her heart."

Thus the heart here typifies the place of thought, reasoning, and understanding within man. (Proverb 23:7; Matt. 9:4; Matt 15:19, Luke 2:19).

So, the fallout from this aspect of the human heart becomes "Am I responsible for the way I think?" The answer is in the affirmative. "For to be carnally minded is death, but to be spiritually minded is life and peace. Because the carnal mind is enmity against God; for it is not subject to the law of God, nor indeed can be" *(Rom. 8: 6*). " Whatever things are true, whatever things are noble, whatever things are just, whatever things are pure, whatever things are lovely,…of good report, if there is any virtue and if there is anything praiseworthy-mediate on these things" *(Phil 4:8).*

We readily admit that it is a tough task to change the way we think (by ourselves) – but that is the very thing we do when we repent (turn to God, through Jesus, who is the author of our life). Repentance is certainly a change of the will but it is also a change of mind, a change of our thinking. When one accepts the gospel, and Christ into his life, he is availing himself to his thinking being guided by the precepts of the scriptures, and particularly of Christ, and does not go on thinking about things the way he used to. We can thus change our attitudes and the way we think.

4.2 The heart is the will of man

The will provides us with the determination to carry through with our thinking. Of Daniel it was said, "from the very first day that

you set your heart to understand, and to humble yourself before your God, your words were heard" (*Dan 10:12*) – Daniel willed or determined to understand and to humble himself. "Set your heart" means to "will in your heart".

There were rulers who believed in Jesus, but were unwilling to confess him lest they be put out of the Synagogue (*John 12:42*). No one obeys the gospel, or God's demands on his life, who does not first find it in his heart to do so, will it in his heart to do it.

To be a strong – willed person is good provided the will has been directed properly by correct thinking and proper knowledge. Without such guidance, the will can become a man's worst enemy for oftentimes it can be said of strong willed people that they are only stubborn people (e.g. Pharaoh). "Stubbornness is as iniquity and idolatry" *(1 Sam 15:23*). We must have the will to do God's will but we must make certain it is His will, and not our version of what we would like his will to be. For therein is true life.

4.3. The heart is a man's feelings or emotions

"Your heart should not be grieved" *(Deut 15:10*). The heart sorrows, "Even in laughter the heart may sorrow" (*Prov 14:13*). Joy resides in the heart, "my servants shall sing for joy of heart, but you shall cry for sorrow of heart" (*Isa. 65:14*). Rejoicing takes place in the heart. "Your heart will rejoice" *(John 16:22*).

In actual fact, all of the following expressions of emotion can be found in the heart: gladness[1], desires[2], is troubled[3], trusts[4], is broken[5], suffers turmoil[6], fails one[7], is pained[8], is proud[9], hates[10],

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1 **Psalm 4:7**
2 **Ps 20:4**
3 **Ps 25:17**
4 **Ps 28:7**
5 **Ps 34:18**
6 **Ps 38:8**
7 **Ps 40:12**
8 **Ps 55:4**
9 **Ps 101:5**
10 **Ps 105:25**

is wounded[11], is distressed[12], despises[13], is anxious[14], is bitter[15], is merry[16], is haughty[17], is proud[18], envies[19], delights[20].

What about "you shall love the Lord your God with all your heart, with all your soul, and with all your mind" *(Matt. 22:37*)? "Christian love (agape). is not an impulse from the feelings, it does not always run with the natural inclinations, nor does it spend itself only upon those for whom some affinity is discovered. Christian love has God for its primary object, and expresses itself first of all in implicit obedience to his commandments" ."For this is the love of God, that we keep his commandments" (*1 John 5:3).*

4.4. The heart is a man's conscience

On the day of Pentecost, the men who heard Peter preach, "*were cut to the heart" (Acts 2:37*). They felt guilty. Their conscience was bothering them. As a result, they bowed over to the superior demands of the Master Cardiologist, Jesus Christ, yielding their lives, will emotions and entire being unto Him *(Acts 2:40,41*).

What is the conclusion of the matter of the heart, from the Biblical perspective?

Our heart reflects all we are in our thinking, our will, our emotions, our conscience – the totality of our being wrapped up into one package.

The heart of the unregenerate man- man in his raw nature (with all the polish of education, accomplishment, acquisitions) "is

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11 **Ps 109:22**
12 **Ps143:4**
13 **Prov 5:12**
14 **Prov 12:25**
15 **Prov 14:10**
16 **Prov 15:13**
17 **Prov. 18:12**
18 **Prov 21:4**
19 **Prov 23:17**
20 **Prov 27:9**

desperately wicked and beyond human cure, who can know it (fathom) it?" (*Jer 17:9*). It needs an "exchange blood transfusion" of the sort of which only the Chief Consulting Heart Surgeon can operate –"Behold, I have been standing at the door **(of your heart**), and I am constantly knocking. If anyone hears me calling him and opens the door, I will come in and fellowship with him, and he with me" "For where your treasure is, there your heart will be also, for the preparations of the heart belong to man" *(Rev 3:20, Mat. 6:21, Prov 16:1*). YOU can heed the call now. YOU can prepare your heart, if YOU are willing.

Let the **'Charge d' Affaires' of the matter of the heart, the Lord Jesus Christ**, into your heart!

5.0 THE HEART: MEDICALLY

WHAT IS THE HEART? – DESCRIPTION

The heart is the centre of the cardiovascular system[9].

The heart, while only a single organ, works as a double pump. It propels blood though the lungs (pulmonary circulation) and the rest of the body (systemic circulation) simultaneously.

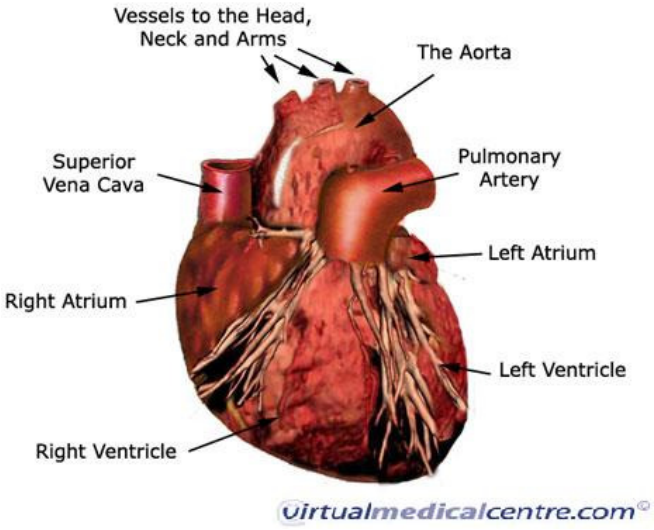


Fig. 1: The Heart: Anatomy – Anterior View

IN HUMANS, Anatomically, the heart is a hollow, pump-like organ of blood circulation, composed mainly of rhythmically contractile smooth muscle, located in the chest between the lungs, and slightly to the left, and consisting of four chambers: a right atrium that receives blood returning from the body via the superior and inferior vena cavae, a right ventricle that pumps the blood through the pulmonary artery to the lungs for oxygenation, a left atrium that receives the oxygenated blood via the pulmonary veins and passes it through the mitral valve, and a left ventricle that pumps the oxygenated blood, via the aorta, throughout the body.

THE MAGNITUDE OF THE WORK / IMPORTANCE OF THE HEART

At rest, the heart pumps about 7,200 litres of blood per day [equivalent of 100x of a 70-litre tank vehicle], through about 96,000 kilometres of blood vessels. This is equivalent to travelling from Enugu to Maiduguri (a distance of about 1179km), over 80x; or Lagos to Maiduguri (about 1606km), over 60x; 160x travelling from Enugu to Abuja (595km); or 16x on flight from Lagos to London (of about 6000km), or even still, 100x the speed of a Boeing 747 jumbo jet, cruising at the speed of 920km/hour.

A. LOCATION AND SIZE OF THE HEART

SIZE: The heart is a hollow cone-shaped organ, weighing about **283.5gm** (1/250th of the average 70kg man), in the adult. It consists of two upper chambers, the atria, and the two lower chambers, the ventricles.

LOCATION: The heart rests on the muscular diaphragm separating the thoracic and abdominal cavities. The thoracic space in which it sits is known as the mediastinum.

The apex -- About 2/3 of the heart mass lies to the left of the midline, with its apex, formed by the tip of the left ventricle, lying 9cm from the midline, deep to the 5th intercostal space.

The base -- Opposite to the apex is the base of the heart. It lies superior and posterior in the mediastinum and is formed mostly by the left atrium.

B. THE PERICARDIUM

The pericardium -- The pericardium is a triple-layered bag that surrounds and protects the heart, confining it to its position within the mediastinum, yet allowing it freedom of movement for contraction.

The pericardium consists of two main portions:

Fibrous -- The outer fibrous pericardium is a tough, inelastic, fibrous connective tissue attached to the great vessels associated with the heart, the diaphragm, and at the roots of the lungs. It serves to anchor the heart within the mediastinum, prevent over-stretching of the heart during exercise, and offers some degree of protection.

Serous (parietal vs. visceral) -- The inner serous pericardium is a thinner and more delicate membrane that forms a double layer around the heart. It is subdivided into two layers.

Parietal -- The outer portion of the serous pericardium, the parietal layer, which is fused to the inside surface of the fibrous pericardium, the sternum, vertebral column and diaphragm.

Visceral -- The inner visceral layer of the serous pericardium, also known as the epicardium or the outer wall of the heart itself, adheres tightly to the surface of the heart muscle (the myocardium).

The pericardial cavity -- Between the parietal and visceral layers is a small space called the pericardial cavity. It contains a small amount of pericardial fluid,(usually about 50mls), secreted by

the serous pericardium that is used for lubrication to reduce friction as the heart moves.

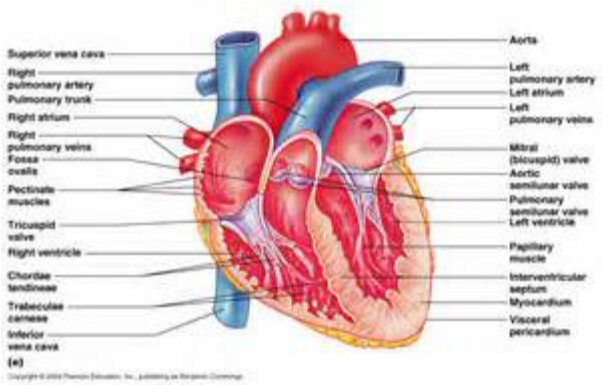


Fig. 2: The Heart – Pericardium, Wall and Chambers

C. THE HEART WALL

The epicardium -- The wall of the heart itself is subdivided into three layers. The epicardium, the outermost layer, is also known as the visceral layer of the serous pericardium. It is a thin, transparent membrane that imparts a slippery texture to the outer surface of the heart.

The myocardium -- The myocardium, the middle layer, consists of cardiac muscle cells and is responsible for the pumping action of the heart. The cardiac muscle fibres are involuntary, striated, and branched, swirling diagonally around the heart in interlacing bundles to form two large networks, the atria and the ventricles.

The intercalated discs -- Each cardiac muscle cell contacts neighbouring cells by transverse thickenings of the sarcolemma called intercalated discs, within which are gap junctions that electrically couple the cells so that they work as a unit (functional syncytium).

The endocardium -- The innermost layer of the heart wall is the endocardium, a simple squamous epithelium overlying a thin connective tissue. The endocardium becomes continuous with the endothelium of the blood vessels.

D. CHAMBERS OF THE HEART

The heart chambers -- The interior of the heart is divided into four compartments that receive the circulating blood.

The two superior chambers are the right atrium and the left atrium, each of which has an appendage called the auricle that increases the volume of the atrium and is used during exercise.

The two lower chambers are the right ventricle and the left ventricle.

The coronary sulcus and interventricular sulci -- Externally, the heart chambers are delineated from one another by a series of grooves within which lie the coronary arteries and coronary veins.

The coronary sulcus separates the atria from the ventricles.

The anterior and posterior interventricular sulci separate the two ventricles front and back.

The interatrial septum -- Internally the chambers of the heart are separated by muscular walls called septa.

The interventricular septum -- The interventricular septum separates the ventricles and is divided into two portions: the superior membranous and inferior muscular interventricular septum.

Atrial wall thickness -- The myocardium of the atria is relatively thin since it has only to move blood into the ventricles, and therefore needs to generate only a small amount of pressure.

Ventricular wall thickness **--** The myocardium of the ventricles is considerably thicker since it must move blood to the lungs (right ventricle) or to the rest of the body (left ventricle). The left

ventricle wall is thickest since it must generate the largest amount of pressure.

E. CIRCULATORY PATHWAY

BLOOD FLOW THROUGH THE HEART

The basic pattern of blood flow from the body to the heart is through three veins: superior vena cava, inferior vena cava, and the coronary sinus.

The superior vena cava brings blood from most of the upper body (head, neck, upper extremity, and thorax), to the heart.

The inferior vena cava brings blood from all parts of the body inferior to the diaphragm.

The coronary sinus receives blood from the coronary veins draining the heart itself and delivers it to the right atrium.

From the right atrium blood moves into the right ventricle, is pumped into the pulmonary trunk, which divides into right and left pulmonary arteries, each of which carries deoxygenated blood to its respective lung.

Oxygenated blood from the lungs passes to the left atrium via 4 pulmonary veins.

Blood then passes from the left atrium into the left ventricle, from which it is pumped into the aorta for distribution throughout the systemic circulation.

THE CORONARY CIRCULATION

The myocardium has its own blood supply and does not rely upon diffusion of nutrients from the blood circulating through the chambers to meet its needs. Two coronary arteries branching from the ascending aorta, right and left, are responsible for the total blood flow to the myocardium.

1. CORONARY ARTERIES

Left coronary artery -- The left coronary artery emerges from the aorta to the left of the pulmonary trunk and almost immediately divides into two branches: anterior interventricular artery (left anterior descending or LAD) circumflex artery. The left coronary artery, via its branches is responsible for most of the blood supply to the anterior myocardium of both ventricles and to the left atrium.

Right coronary artery -- The right coronary artery emerges from the aorta to the right of the pulmonary trunk and passes in the groove between the right atrium and right ventricle. It gives rise to two arteries: marginal artery and posterior interventricular artery. The right coronary supplies blood to the right atrium and the posterior myocardium of the ventricles.

Anastomoses of the heart -- There are a great many interconnections (anastomoses) between the branches of the coronary arteries, particularly where the anterior and posterior interventricular arteries meet.They are collateral arteries. They are largely nonfunctional in the normal heart because no pressure gradient develops across them.

Anastomoses provide a number of alternate routes for blood flow should one path become blocked.

2. CORONARY VEINS

These include the great cardiac veins, middle and small cardiac veins.

The coronary sinus lies in the groove between the left atrium and the ventricles. It receives venous blood from the great, middle, and small cardiac veins, and then opens into the right atrium.

F. VALVES OF THE HEART

As each chamber of the heart contracts, it pushes a portion of its blood into a ventricle or into a great artery. To prevent backflow of blood, the heart is equipped with valves, formed from the connective tissue of the cardiac skeleton and covered with endocardium. They open and close by pressure changes.

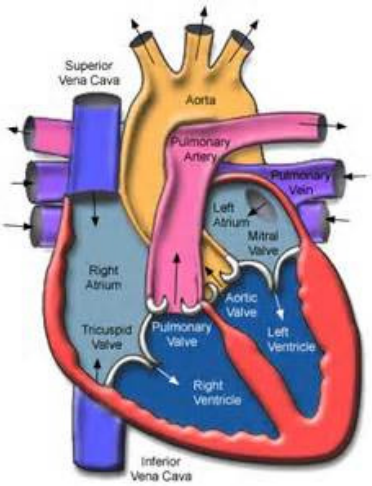
There are two types of heart valves:

1. Atrioventricular (AV) valves 2. Semilunar (SL) valves

1. ATRIOVENTRICULAR VALVES

Fig. 3: The Heart – The Chambers, Great Vessels and Valves

Atrioventricular (AV) valves lie between the atria and the



ventricles. The tricuspid valve is on the right side, and the bicuspid (mitral) valve is on the left. Each cusp of an AV valve is roughly shaped like a triangle; the base is attached to the heart wall and the apex is pointed down into the ventricle.

Attached to the apices of the cusps are tendon-like cords of connective tissue called the chordae tendineae, which anchor

the valves down inside the ventricle wall by attaching to papillary muscles.

2. SEMILUNAR VALVES

Locations -- The semilunar (SL) valves, the second type of heart valves, are located in the pulmonary trunk and aorta just as each vessel emerges from its respective ventricle. Each of these valves consists of three half-moon shaped cusps that are attached to the artery wall like a pocket is attached to a shirt, with a free upper margin.

G. CONDUCTION SYSTEM AND PACEMAKER

The heart stimulates itself to beat by using an inherent and rhythmic electrical activity.The autonomic nervous system and the endocrine system can only modify the heartbeat, but they do not establish the fundamental rhythm.

Certain cardiac muscle cells repeatedly create spontaneous action potentials that then trigger contractions. These are called autorhythmic cells.

THE AUTOARRHYTHMIC CELLS

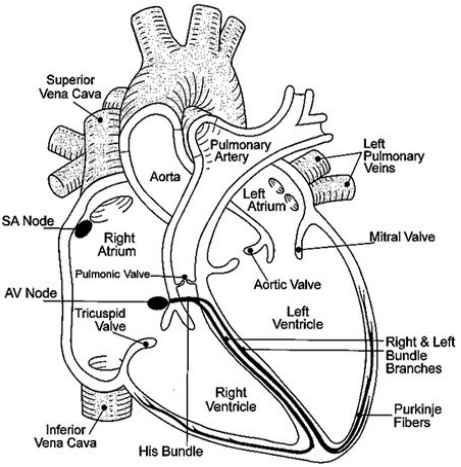
During heart development about 1 % of the forming cardiac muscle cells lose the ability to contract and become instead autorhythmic (self-excitable).

1. They act as a pacemaker, setting the basic rhythm for the entire heart.

2. They form the conduction system, the route for conducting the impulses throughout the heart muscle. The conduction system assures that the cardiac chambers contract in a coordinated and timely fashion, thus making the heart an effective pump.

Fig. 4: The Heart with the Conduction System

1. The sinoatrial (SA) node is a collection of autorhythmic cells in the posterior right atrial wall, just inferior to the opening of the superior vena cava. It is the primary pacemaker, setting the basic rhythm of the heart at about 90-100 beats/minute.



2. The atrioventricular (AV) node is a collection of autorhythmic cells at the junction of the four chambers, within the cardiac skeleton. It is the secondary pacemaker, setting the basic rhythm of the heart at about 40-50 beats/minute.

3. The atrioventricular (AV) bundle extends from the AV node into the membranous interventricular septum.

4. At the muscular septum the AV bundle divides into the right and left bundle branches, each travelling through its respective ventricle.

5. Extending from the bundle branches are Purkinje fibers, which pass the action potentials to the ventricular myocardium and cause the muscle cells to contract.

H. CIRCULATORY PHYSIOLOGY AND THE CARDIAC CYCLE

The events that control blood flow through the heart are:

1. Contraction and relaxation of the myocardium, controlled by the conduction system
2. Opening and closing of the AV and SL valves, controlled by pressure changes in the heart chambers and the great arteries. Blood flows from an area of high pressure to an area of low pressure. The pressure developed within a heart chamber is related to*:*
    1. The volume of blood within the chamber exerts a fluid pressure.
    2. The size of the chamber: as a chamber contracts its size gets smaller and therefore the pressure within increases.

PHASES OF THE CARDIAC CYCLE

The cardiac cycle refers to all the events associated with blood flow through the heart.

One complete cardiac cycle consists of a systole and a diastole of both atria, plus a systole and diastole of both ventricles.

The Cardiac cycle of a normal resting adult is in three main phases:

Relaxation phase -- at the end of a heart beat when the ventricles start to relax, all four chambers are in diastole. This is known as the quiescent period.

The Relaxation Phase:

As the ventricles relax, ventricular pressure drops and blood begins to flow from the great arteries back toward their respective chamber. This results in closure of the SV valves, giving the second heart sound. At this point, the volume of blood within the ventricles does not change because the AV valves are also closed. This period is called isovolumetric relaxation, at the early diastolic phase.

As the ventricles continue to relax, their chamber size continues increasing until ventricular pressure drops below atrial pressure. As a result the AV valves open. Blood from the atria begins to move into the ventricles, passing through the open AV valves, following the pressure gradient, and the ventricles begin to fill.

The second phase of the cardiac cycle: Ventricular Filling.

This is during mid - to-- late diastole.

Ventricular filling -- 70% of ventricular filling occurs just after the AV valves open because of blood that had been filling the atria from the venous circulation while they were in diastole and the AV valves were closed.

The first third of ventricular filling is thus known as the period of rapid ventricular filling, and occurs without the benefit of atrial systole.

The middle third of ventricular filling is called diastasis. It occurs as blood flow from the atria slows and a much smaller volume of blood enters the ventricles. Excitation of the SA node initiates atrial systole, as the AV valves open, marking the end of the quiescent period, and causing the last third of ventricular filling to occur (remaining 30%) as the atria completely empty themselves.

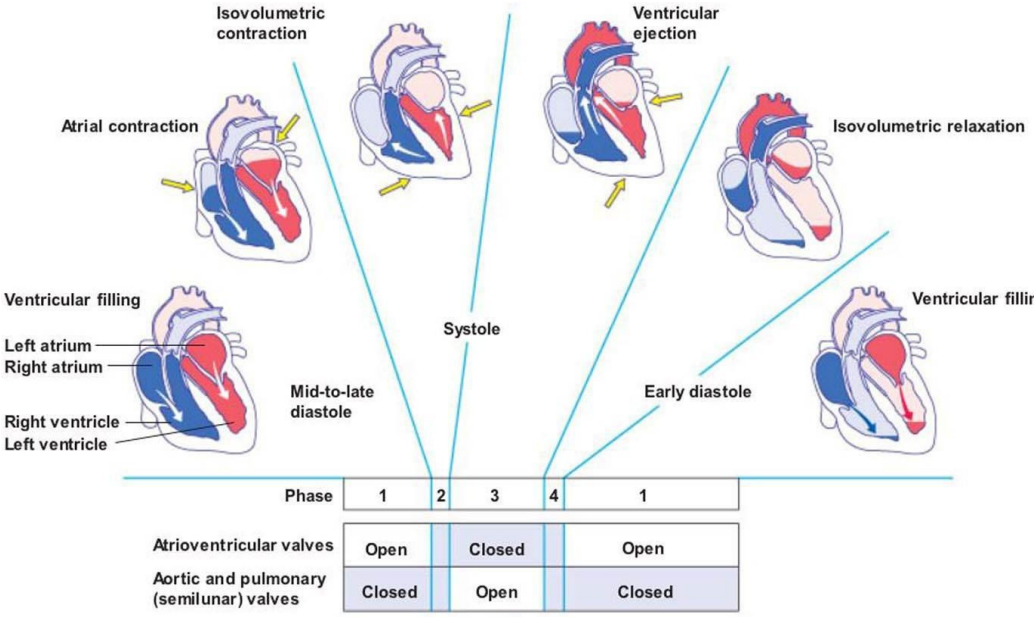


Fig. 5: Phases of Cardiac Cycle[10]

At the end of ventricular diastole, each ventricle contains about 130 ml of blood, the end-diastolic volume (EDV). Throughout this entire phase the AV valves are open and the SL valves are closed.

The third phase of the cardiac cycle: Ventricular Systole.

Ventricular systole -- As the excitation passes into the AV node then throughout the rest of the conduction system, the ventricular myocardium enters systole. Ventricular pressure, which was already increased by ventricular filling, now begins to rise even higher. As a result, when the ventri-cular pressure exceeds the atrial pressure, the AV valves close, as the atria relax. This is known as the period of isovolumetric contraction because the volume of blood within the ventricles remains the same (130ml).

When ventricular pressure finally exceeds arterial pressure, the SL valves open and blood is ejected from the ventricles into the

appropriate artery. This period is known as ventricular ejection and continues until the ventricles begin to relax.

Once the relaxation phase has begun again, arterial pressure exceeds ventricular pressure and the SL valves close again. At this time each ventricle contains about 60ml of blood, the end systolic volume (ESV).

The volume of blood moved from each ventricle during ventricular systole is known as the stroke volume (70 ml).

(SV = EDV – ESV) or (SV = 130 ml - 60 ml)

I. THE ELECTROCARDIOGRAM AND THE HEART

Impulse conduction through the heart generates electrical currents that can be detected at the body surface. A recording of the electrical changes that accompany each cardiac cycle is called an electrocardiogram (ECG). The ECG is a composite of action potentials produced by all the heart muscle fibres during each heartbeat. In a typical recording, there are several clearly recognizable and named parts.

P wave -- The P wave is the first small upward deflection that represents atrial depolarization as it spreads from the SA node and across both atria.

QRS complex -- The QRS complex, the second wave, begins as a small deflection down, followed by a large deflection upward, and ends with a small deflection down. It represents ventricular depolarization, the spread of excitation through the ventricles.

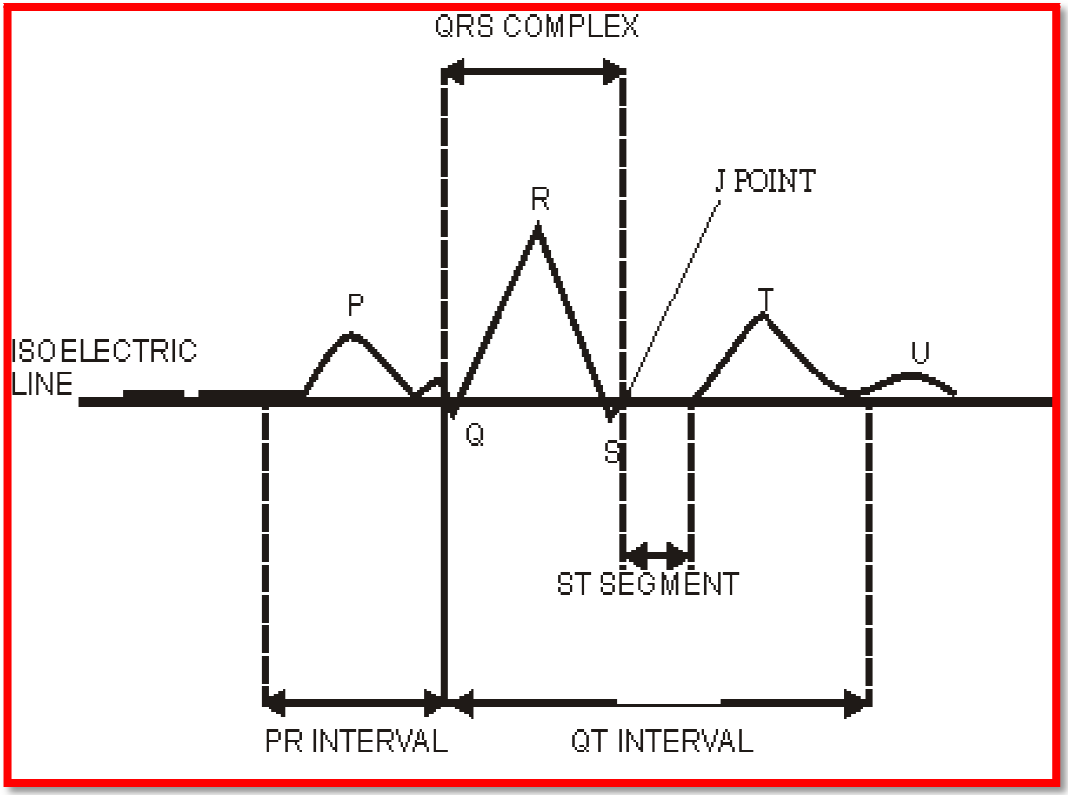


Fig. 6: Electrocardiogram Tracing

T wave -- The T wave, the third wave, is a small dome-shaped upward deflection that represents ventricular repolarization. The T wave appears just before the ventricles begin to relax.

P-R interval -- The P-R interval is measured from the beginning of the P wave to the beginning of the QRS complex. It

represents the conduction time from the beginning of the atrial excitation to the beginning of ventricular depolarization.

S-T segment -- The S-T segment begins at the end of the S wave and ends at the beginning of the T wave. It represents the time when ventricular contractile fibres are fully depolarized.

Quiescent period -- The quiescent period is the time period between the end of the T wave and the beginning of the next P wave. It represents that time when all heart muscle is at rest.

J. CARDIAC OUTPUT

Cardiac output (CO) is defined as the amount of blood ejected from the left ventricle into the aorta per minute. It would be the same for the right ventricle.

Stroke volume -- Stroke volume (SV) is the amount of blood ejected from the ventricle per beat.

Heart rate -- Heart rate (HR) is the number of heart beats per minute.

CO = SV x HR

CO = (70 ml/beat) x (75 beats/minute)

CO = 5,250 ml (5L)/minute

A cardiac output of 5.25 L/minute, at rest, is virtually the entire volume of blood in the body flowing through the entire circulation once per minute. If body demand increases cardiac output increases to meet the challenge.

Cardiac reserve is the ratio between the maximum CO a person can achieve and the CO at rest. A normal adult has a cardiac reserve of 4-5 times the resting value (21 L/min or about 300% of resting value).

The two basic factors that alter cardiac output are Stroke volume and Heart rate. Therefore, factors that alter either will alter cardiac output.

1. REGULATION OF STROKE VOLUME (SV). A healthy heart pumps out all of the blood that was moved into its chamber during the previous diastole. At rest this is 50-60% of the total volume because 40-50% remains in the ventricles.

End-diastolic volume (EDV) = the volume of blood in a ventricle at the end of a diastole.

End-systolic volume (ESV) = the volume of blood remaining in the ventricle after systole.

SV = EDV - ESV

Anything that alters either EDV or ESV will alter stroke volume and therefore alter cardiac output.

. EJECTION FRACTION: The overall systolic function is frequently quantified by the ejection fraction. This is the ratio of the SV to the EDV (SV/EDV X100%) – the fraction of blood in the ventricle ejected with each ventricular contraction.

The normal Ejection Fraction (EF) is about 60%.

The Ventricular diastolic function **(lusitropy**) also plays an important role in overall cardiac performance.

The three (3) **factors that regulate stroke volume** and ensure that the left and right ventricles pump equal volumes of blood are:

a) Preload -- Preload is the stretch on the heart muscle before it contracts.

b) Contractility -- Contractility is the forcefulness of contraction of individual cardiac muscle cells

c) Afterload -- Afterload is the pressure that must be exceeded by the ventricle before blood can be ejected from the ventricle into the artery.

a. Preload: Effect of Stretching. A greater stretching on cardiac muscle cells just before they contract increases their force of contraction.

Within physiological limits, the more the heart is filled during diastole, the greater the force of contraction. This is known as the Frank-Starling law of the heart.

The preload depends on the volume of blood that fills the ventricles at the end of diastole (EDV) and is determined by two factors: (1) length of diastole and (2) venous pressure.

b. Contractility. Myocardial contractility is the strength of contraction at any given preload. Inotropic agents are believed to affect cardiac contractility by altering the flow of calcium ions during impulse conduction through the cardiac muscle fibers and thus altering the strength of contraction.

c. Afterload. Afterload is the arterial pressure that must be exceeded before ventricular ejection can occur. Right ventricular pressure must exceed arterial pressure in the pulmonary trunk and left ventricular pressure must exceed arterial pressure in the aorta.

With an increased afterload (hypertension or high blood pressure), stroke volume decreases and more blood remains in the ventricle at the end of systole (increased ESV). As blood pools in the ventricle there is increased contractility and the ventricle has to work harder to pump.

2. REGULATION OF HEART RATE

a. Autonomic Control of Heart Rate

The second major controller of cardiac output heart rate is.

Regulation of heart rate -- Cardiac output depends upon

heart rate as well as stroke volume; therefore, anything that affects heart rate ultimately affects cardiac output.

Sympathetic fibers, called cardiac acceleratory nerves, synapse in the SA node, AV node, and most of the myo-cardium,

releasing norepinephrine and causing increased HR and force of contraction. As a result, CO increases.

Parasympathetic fibers reach the heart via cranial nerve X (10 - right and left vagus), synapse in the SA node and AV node, release acetylcholine, and slow the heart rate. As a result CO decreases. This is the dominant control of the heart at rest.

b. Chemical Regulation of Heart Rate

The hormones epinephrine, norepinephrine, and thyroxine increase HR and force of contraction while glucagon increases HR only. Ions, in particular the relative concentrations of Na+, K+, and Ca++ in the extracellular fluids have a large impact on heart rate.

Increased K+ blocks impulse formation by the SA and AV nodes, increased Na+ causes decreased force of contraction, and moderate increase in Ca++ speeds and strengthens the heart.

6.0 THE HEART AND RELATED DISEASES - HISTORICAL PERSPECTIVES AND CURRENT TRENDS

6.1 William Harvey

Until the 17th century, two separate systems were thought to be involved in blood circulation: the *natural* system, containing venous blood which had its origin in the liver, and the *vital* system, containing arterial blood and the 'spirits' which flowed from the heart, distributing heat and life to all parts. Like bellows, the lungs fanned and cooled this vital blood[11].

Independently from **Ibn Al-Nafis**, Michael Servetus identified **pulmonary circulation**, but this discovery did not reach the public because it was written down for the first time in the Manuscript of Paris in **1546[12].**

William Harvey (1 April 1578 – 3 June 1657) was an English physician. He was the first to describe completely and in detail the systemic circulation and properties of blood being pumped to the brain and body by the heart, though earlier writers had provided precursors of the theory[13].

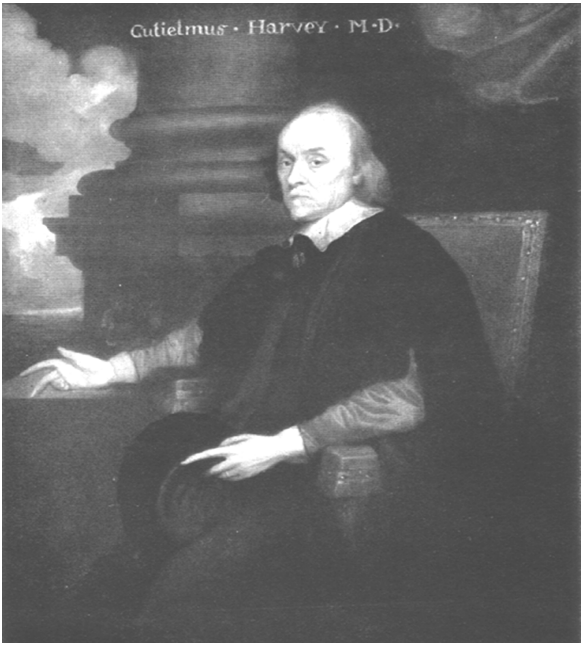


Fig. 7: Portrait of William Harvey

*In **1628** he published in Latin, in **Frankfurt**, his completed treatise on the circulation of the blood, "**Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus"** (otherwise known as "**On the Motion of the Heart and Blood"),** stating that it was extremely important to study the heart when it was active in order to truly comprehend its true movement; a task which even he "found of great difficulty, as he says:*

"...I found the task so truly arduous... that I was almost tempted to think... that the movement of the heart was only to be comprehended by God. For I could neither rightly perceive at first when the systole and when the diastole took place by reason of the rapidity of the movement..."[14]

6.2 For the Case of Heart Failure

It is possible to trace a remarkable history that, for Western medicine, extends back to clinical descriptions collected in works attributed to **Hippocrates** in ancient Greece[13] .

The Hippocratic corpus stated thus “Should [phlegm coming from the brain] make its way to the heart, palpitation and difficulty breathing supervene … and the heart palpitates, so that under this compulsion difficulty of breathing and orthopnea result”[15].

Galen, a Greek physician who lived in the Roman Empire during the **second century CE**, viewed the heart as the source of heat, but believed that the pulse is transmitted along the walls of the arteries rather than by blood flowing through their lumen. He described what almost certainly represents atrial fibrillation when he noted “complete irregularity or unevenness [of the pulse], both in the single beat and in the succession of beats”[16].

Galen’s view that the heart’s primary function is to distribute heat by an ebb and flow was to dominate Western thinking for more than 1500 years.

There was no way to relate the clinical findings to heart disease until 1628, when William Harvey clearly described the circulation: “I am obliged to conclude that in animals the blood is driven round a circuit with an unceasing, circular sort of movement, that this is an activity or function of the heart which it carries out by virtue of its pulsation, and that in sum it constitutes the sole reason for the heart’s pulsatile movement”[17]. Harvey’s discovery provided a basis for understanding the hemodynamic abnormalities in heart failure.

Forty years later, **Richard Lower** noted that ejection was impaired by compression of the heart in pericardial tamponade[18], and in **1715, Raymond Vieussens** published a remarkably clear description of the physiological basis for the signs and symptoms in a patient with mitral stenosis, then the most common cause of heart failure[19].

At the beginning of the 18th century, physicians began to focus on the abnormal structure of failing hearts. **Giovanni Maria Lancisi**, in a text published in **1745**, noted that **valvular regurgitation** leads to ventricular dilatation but that the left ventricular cavity does not enlarge in **aortic stenosis**[20]. A century later**, Nicolas Corvisart**[21] and **John Bell**[22], both of whom observed that eccentric hypertrophy (dilatation) has a worse prognosis than **concentric hypertrophy**. Corvisart also noted that patients with heart failure can die in 2 ways**: progressive heart failure,** which "advances slowly, [until] life is insensibly extinguished," and **sudden death**, which can occur at any time in the course of this syndrome.

Distinctions between **dilatation**, with and without ventricular wall thickening, and hypertrophy, with and without reduction in cavity volume, were made (and confirmed at autopsy) in the 19th century, when there was no way to image the heart; **Röntgen** did not discover **x-rays** until **1895. Cavity size** and wall thickness were evaluated at the bedside by palpation, percussion, and the characteristics of **heart sounds** and **murmurs.** Distinctions between various forms of cardiac enlargement continued into the 20th century, but the focus of efforts to understand the **pathophysiology of heart failure** returned to **haemodynamics** after **1918**, when **Ernest H. Starling** described his "**Law of the Heart**"[23].

Hemodynamic abnormalities were of enormous importance in heart failure during the first half of the 20th century, when almost three fourths of patients hospitalized for heart disease in England had structural abnormalities (51% rheumatic, 11% bacterial endocarditis, 9% cardiovascular syphilis, and 2% congenital). In the United States at the same time, **rheumatic valvular disease** accounted for 60% to 80% of adult heart disease[24,25]. Today, in developed countries, rheumatic heart disease has become a rarity, which makes it difficult to appreciate the impact of this cause of heart failure, which had been a scourge since antiquity

It was not until the early **1940s** that introduction of **cardiac catheterization** by **André Cournand** and **Dickinson W. Richards** brought more than a half century of hemodynamic research to the bedside[26].

Treatment of cardiac injury during **World War II** demonstrated the **feasibility of operating on human hearts.** Their wartime experience led **Charles Bailey** and **Dwight Harken** in the United States and **Russell Brock** in Great Britain to develop operations to open the narrowed valve in patients with rheumatic mitral stenosis. Harken told how thoracic surgeons overcame the fear of operating on the heart, once thought to be impossibly dangerous, when they began to remove shrapnel from the hearts of wounded soldiers

*Development of **open heart surgery** and **prosthetic valves**, which began in the **1960s**, allowed cardiac surgeons to palliate many forms of **structural heart disease,** both **rheumatic** and **congenital.** However, these advances did not solve the challenges posed by heart failure because **ischaemic heart disease** and **dilated cardiomyopathies** were emerging as the major causes of systolic heart failure, and systemic arterial hypertension and reduced aortic compliance led to an epidemic of **diastolic heart failure** in today's aging population.*

***Biochemical Abnormalities:** Beginning in the **1950s**, 3 areas of biochemistry came to have a major impact on cardiology. The initial focus was on energetics, which had influenced thinking in muscle physiology since the beginning of the 19th century. The second began to unfold in the 1960s, when elucidation of the mechanisms responsible for muscle contraction, relaxation, and excitation–contraction coupling helped in understanding how hearts failed and initiated a search for **new inotropic drugs** that were initially viewed as able to cure, or at least significantly palliate, this syndrome. At the same time, rapid advances in the third area, the biochemical basis for the neurohumoral response to reduced cardiac output, led to the first major advances in*

treating this syndrome since the introduction of ***effective diuretics****.*

Eugene Braunwald's group was able to show convincingly in the late **1960**s that contractility is reduced in patients with **chronic heart failure**[27].

These discoveries provided clues to mechanisms that depress contractility in failing hearts and stimulated efforts to develop inotropic drugs more powerful than **digoxin**, the benefits of which in heart failure were then viewed as resulting from increased contractility

The Neurohumoral Response: Peter Harris, in **1983**, provided a clear explanation of the adverse role played by the body's responses to **lowered cardiac output**, the most important of which are vasoconstriction, salt and water retention, and adrenergic stimulation[28].Harris pointed out that these responses, which had evolved to maintain cardiac output during exercise and support the circulation when cardiac output falls after haemorrhage, become harmful when they are sustained and therefore are deleterious in chronic heart failure.

The ability of reduced afterload to increase both cardiac efficiency and cardiac output provided a rationale for the introduction of **vasodilators** to treat heart failure[29]. These considerations stimulated **Jay N. Cohn** and others to organize the Vasodilator Heart Failure Trial **(VHeFT**) I to examine the effects of vasodilators on long-term prognosis in these patients[30]. This randomized double-blind trial, which was the first of the large heart failure trials that now represent the gold standard in evaluating therapy. Cooperative North Scandinavian Enalapril Survival Study (**CONSENSUS** I), which documented a dramatic benefit of angiotensin II–converting enzyme **(ACE) inhibitors**[31]. The implications of CONSENSUS I became apparent when the results of this trial were first presented in **1986,** investigators had begun to explore the possibility that angiotensin II is not only a vasodilator but also a regulator of proliferative signalling.

*A critical clue to the mechanisms responsible for the survival benefits of ACE inhibitors was published in **1985** when **Janis Pfeffer, Mark Pfeffer**, and **Eugene Braunwald** found that these drugs slow the progressive cavity enlargement, which they called* **remodeling,** *that follows experimental myocardial infarction[32].*

6.3 Current Classification of Heart Failure

The approach most commonly used to classify heart failure now is based on the degree of functional limitation imposed by the condition. These are in 4 classes, depending on degree of effort needed to elicit symptoms.

Table 1: NYHA Functional Classification of HF Based on Severity of Symptoms and Physical Activity[33].

Class 1	No limitation of physical activity. Ordinary physical activity does not cause undue breathlessness, fatigue or palpitations.
Class II	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in undue breathlessness, fatigue or palpitations.
Class III	Marked limitation of physical activity. Comfortable at rest, but less than ordinary physical activity results in undue breathlessness, fatigue or palpitations.
Class IV	Unable to carry on any physical activity without discomfort. Symptoms at rest can be present. If any physical activity is undertaken, discomfort is increased.

Genomics:** A new approach to understanding the mechanisms responsible for the progressive deterioration of failing heart came into focus in **1987**, when molecular biology moved to centre stage in cardiology[34]. Three years later, in **1990**, the **Seidman

*laboratory reported the first molecular cause of a **familial cardiomyopathy**, a missense mutation in the cardiac β-myosin heavy chain gene[35].This discovery identified one of a growing number of mutations involving additional proteins that cause both **hypertrophic and dilated cardiomyopathies**[36]. The possibility of modifying the signal pathways controlled by these mutations raises the possibility that transcriptional therapy can be developed to help some of these patients. Similarly, efforts to modify signaling pathways that could allow activation of adaptive cardiac myocyte growth and inhibition of maladaptive hypertrophy are under way[37].*

***Epigenetics:** A newly discovered type of regulation, referred to as* epigenetics[38]*, has recently been identified as operating in heart failure. Epigenetic mechanisms modify later steps in proliferative signaling pathways, including methylation of cytosine in genomic DNA, histone acetylation, and inhibition of RNA translation by small RNA sequences, called* microRNAs*. Cytosine methylation has been implicated in some familial cardiomyopathies, and histone acetylation can modify overload-induced cardiac hypertrophy. Evidence that microRNAs regulate cardiac hypertrophy is of potential therapeutic importance because short RNA segments, called small-interfering (si)RNAs, can silence specific genes. The ability of siRNAs, which are readily synthesized commercially, to block specific proliferative pathways promises additional approaches to inhibiting maladaptive hypertrophy to **slow deterioration of failing hearts**[39].*

7.0 DIASTOLIC DYSFUNCTION

7.1 Definition

Left Ventricular Diastolic Dysfunction has been defined as a functional abnormality of diastolic relaxation, filling or distensibility of the left ventricle, and when this is associated with

symptoms of heart failure, in the presence of normal Ejection Fraction (EF), Diastolic Heart Failure is diagnosed[40].

7.2 Early Marker

It is now widely regarded that left ventricular diastolic dysfunction (LVDD) is the earliest manifestation of heart disease in hypertension, and is a precursor for heart failure. DD is the earliest discernible cardiac manifestation of Hypertension, Diabetes Mellitus, as well as Obesity[41-43].

7.3 With Heart Failure

For years after William Harvey described the heart as a pump, this pumping / systolic function of the heart was emphasized, and most cardiac dysfunctions were ascribed to it. However, it was noted that almost half of the patients with Heart Failure (HF) had normal or near normal Systolic Function. This realization/fact has thus generated a lot of interest and research into Diastolic dysfunction in the past 2 decades. It has resulted in placing the filling or diastolic functions of the heart on equal level with systolic function[11,44].

Diastolic dysfunction has thus been shown to be associated with the future occurrence of HF, and is a predictor of cardiovascular morbidity and mortality, in the general population.

7.4 Pathophysiology

Diastolic dysfunction is caused by conditions that impair cardiomyocyte bioenergetics – like Diabetes Mellitus and Ischaemic Heart Diseases – because of the energy dependent nature of diastole. Equally, conditions that result in cardiomyocyte and extracellular matrix remodelling also lead to Diastolic dysfunction – like Hypertension, Hypertrophic Cardiomyopathy and Diabetes Mellitus.

Some of the currently held concepts on the Pathophysiological basis include. The extent of the Left Ventricular Hypertrophy (LVH) is associated with regional LV relaxation abnormalities,

with LVH being correlated with severity of diastolic dysfunction. Inappropriate LV Mass can slightly predict severity of diastolic dysfunction in uncomplicated Hypertension.

Cardiac interstitial and perivascular fibrosis; Incresed levels of serologic markers of myocardial collagen synthesis turnover, such as Serum Carboxy Terminal Propeptide of Procollagen Type 1 have been found to correlate with myocardial fibrosis. Myocardial ischaemia and intrinsic myocardial impairment have also been implicated[45,46].

7.5 Prevalence

In the absence of risk factors for diastolic abnormalities, LVDD is rare. Diastolic dysfunction is thus highly prevalent in the setting of Hypertension, where usually, but not invariably, it is associated with LVH.

The prevalence of diastolic dysfunction in hypertensive Europeans ranges between 51-59%[47]; among Caucasians, about 46 – 48%[11]; In Asia, about 44%[48].

In Nigerians, it is about 11.3% in non-hypertensives, and 62-82% in newly diagnosed hypertensives[49,50].

7.6 Morbidity and Mortality

LVDD is an independent predictor of cardiovascular risk factors. Patients with Asymptomatic LVDD have a higher incidence of all-cause mortality adjusted for age, sex and Ejection Fraction. Mild diastolic dysfunction accounts for up to 8.3 fold increase in mortality. Moderate to severe diastolic dysfunction accounts for up to 10.2 fold increase in mortality. Diastolic dysfunction also causes decreased exercise performance[51].

7.7 Evaluation

Cardiac catheterization is the gold standard for determining Diastolic dysfunction – but it is expensive, requires sophisticated equipment, and is invasive.

Two- Dimensional echocardiography, with Tissue Doppler Imaging Techniques, have thus become more widely used for diastolic dysfunction evaluation.

7.8 Grading of Diastolic Dysfunction

Grade 1 – Mild LVDD or Impaired Relaxation

Grade II – Moderate LVDD or Pseudonormalized Pattern

Grade III – Severe LVDD or Restrictive pattern

Treating patients with Restrictive filling may lead to reversion to impaired relaxation (IIIa); patients who fail to respond (IIIb) may have increased morbidity and mortality[52].

7.9 Treatment

Though relatively much is known about diastolic dysfunction now, its optimal treatment strategy is still less clear as compared to systolic dysfunction[53].

Beta blockers (selective), by increasing diastolic filling blood flow, and reducing heart rate and myocardial oxygen demand have been found to have beneficial effects on DD.

Preliminary evidence has associated ACE Inhibitors with better diastolic function, from improved diastolic filling parameters, due to regression of LVH from prolonged ACEI use.

The lusitrophic (relaxation – enhancing) effect of dihydropyridine Calcium Channel Blockers has been found to improve systolic function, stress tolerance, as well as diastolic dysfunction.

More recent data from VALIDD (Valsartan In DD) study, has, however, shown that, in those with hypertension, a better blood pressure control resulted in improvement in diastolic function, regardless of the type of antihypertensive drug used, even in the absence of LVH.

8.0 HYPERTENSION AND CARDIOVASCULAR LIFESTYLE MODIFICATION FACTORS / HABITS

No overview, as this lecture, on the heart, will set sail or fly, much less be complete, without a courtesy call on Hypertension, the **"kingmaker**" and chief promoter of the **'matters'** of, and about**, the heart**. While we take a look at the most basic, but vital, rudiments of knowledge on Hypertension, on one hand; on the other hand, we use it as a template for discussing the **lifestyle modification factors / habits** – which form **the pivot for the non – drug treatment of most of the cardiovascular diseases**.

8.1 What is Blood Pressure?

When the heart contracts, blood is pumped out and pushes against the walls of the arteries causing them to expand. A measure of the force of the blood pushing against the walls is known as the systolic blood pressure. After the contraction, the heart relaxes and the blood vessels recoil. A measurement at this point demonstrates the diastolic blood pressure.

What is high blood pressure? "High Blood Pressure" occurs if the blood pressure is almost always persistently higher than the levels established as normal for both the systolic and diastolic values, which have been accepted to be 140/90mmHg in an adult at rest[54] .

What is hypertension? High blood pressure is also called hypertension by doctors. The "Hyper" part in word 'hypertension' means "too much" and the "tension" refers to the pressure on the artery walls.

What are the symptoms of high blood pressure (hypertension)? Hypertension usually produces no warning signs or symptoms. You usually cannot "feel" blood pressure even when it is unusually high. A few people may have symptoms, such as dizziness, nose-bleeds, chest pain,

palpitation or headaches that are symptoms of hypertension, but also could be due to other medical problems.

The vast majority of individuals never know that their blood pressure is elevated until it is checked by a health professional.

What are the possible risks? Even if you "feel fine," it is very important that your hypertension be treated and kept under control. If the high blood pressure is not controlled, it is likely to go higher, and the higher it goes, the more likely it is to develop serious complications such as heart attacks, heart failure, strokes, and kidney failure. Thus the most important reason for controlling blood pressure is to reduce the risk of developing more serious or even fatal complications.

What causes hypertension? Unfortunately, no one yet knows what causes hypertension in over 90% of the cases. However, it can be managed in almost all such cases.

In cases where the causes of hypertension is not known, the condition is called *primary* or *essential* hypertension. In less than 10% of the cases where the cause is known, the condition is called *secondary* hypertension.

Causes of secondary hypertension include certain kidney and blood vessel diseases, hormonal disorders, and birth defects. Some cases can be corrected by surgery or controlled by medication. Other causes may be due to certain medicines. In such cases the blood pressure will return to normal when doses of the medicines are reduced or stopped.

Who gets essential hypertension? Anybody can have it or develop it. However, statistics show that the development of essential hypertension is associated with the following factors:

Family History. Some families tend to be susceptible to high blood pressure. If both parents have hypertension, the risk of their child developing it is approximately 50%.

Age. Although it is more common in older age groups, it can occur at any age. Nearly half the people over the age of 64 have it, and it is usually first detected between ages 55 and 60.

Sex. Before the age of 50, it occurs more frequently in men than women. The rates are about equal at age 50, but by 55 to 60, more women have it.

Race. It has been found that hypertension is more common among black than white individuals for all age groups beyond adolescence. At a given level of high blood pressure more damage will occur in black individuals than in whites.

Weight. High blood pressure occurs much more frequently in overweight people, and weight loss may be accompanied by a reduction in blood pressure.

Salt. People who have a tendency to develop high blood pressure may be more likely to get it or worsen it if they eat highly salted foods.

Stress. Some medical studies have reported an association between certain stress factors and hypertension.

Other Factors. Other factors or conditions that may be associated with high blood pressure are physical inactivity, alcohol intake, diabetes, and cigarette smoking.

8.2 Why is Treatment so Important?

Although one may feel fine without treatment, the most important reason for treating hypertension is to prevent serious consequences. One may have hypertension for 15 to 20 years before symptoms appear, but some permanent damage may have already been done to vital organs of the body. According to statistics, even mild hypertension can shorten your life expectancy. People with hypertension are much more likely to develop heart attacks, strokes, kidney failure and blood vessels damage in the leg and eyes.

8.3 What are the Goals of Treatment?

The major goals of treatment are to control the blood pressure and reduce other risk factors that may contribute to the development of diseases of the heart and other body organs.

8.4 Risk Factors for Cardiovascular Diseases

There are two types of risk factors for Cardiovascular diseases (of which hypertension is a major player): those that are permanent and cannot be changed and others that can be modified or eliminated.

Permanent, or unmodifiable, risk factors are: An inherited susceptibility to hypertension and heart problem; Being a male; Being diabetic; Being over the age of 40.

Modifiable risk factors include: High blood pressure /Hypertension, Cigarette smoking; A high content of cholesterol in the blood; Being overweight; Excessive nervous tension and stress.

Hypertension: Hypertension can accelerate the process of atherosclerosis, or hardening of the arteries.

When blood pressure is consistently high, it pushes against the arterial walls with extra force. The muscular layer of the artery walls gradually becomes hard and thick. They lose their elasticity, and the passages become smaller and the vessel wall, stiffer. Vessels also develop a buildup of fat or cholesterol. If one already has some atherosclerosis, high blood pressure worsens or accelerates the process. Thus, atherosclerosis makes the vessels make the blood pressure rise, and more fat accumulates in the vessel walls. As the cycle continues, it increases the danger of serious complications such as heart diseases, renal failure and strokes. People with high blood pressure are much more likely to have heart diseases. The majority of all stroke victims have high blood pressure.

High blood pressure also makes the heart pump harder to keep the blood moving, especially as the vessels narrow and thicken. Eventually the extra work can make the heart muscle lose its

pumping power until it is no longer enough to keep the blood circulating. Congestion or a backup fluid in the circulatory system can occur and result in a condition commonly called **Congestive Heart Failure (CHF**).

Cigarettes smoking: In addition to its damaging effect on the lungs, cigarette smoking is also damaging to the heart and blood vessels.

Cigarette smoking:

- Stimulates the heart, making it beat faster, and narrows the blood vessels making it hard to pump out blood; with tendency to increase blood pressure;
- Decreases the supply of oxygen to the heart and body tissues;
- Makes blood more likely to form clots; Causes “extra” or irregular heartbeats,
- Produces potentially harmful changes in the amount of lipids (fats) that circulate in the blood. Medical statistics show that **Coronary Heart Disease** (CHD) occurs more commonly and more severely among smokers than non-smokers; cigarette smokers are much more likely to have a heart attack; and they often have a more difficult recovery.

Cholesterol and other fats. Certain kinds of fatty substances are made in the body and are normally carried in the blood stream. They are called lipids, and one of them is cholesterol, which is also found in many foods. Eating foods high in cholesterol, can increase the amount of cholesterol in the body. Although the body needs a certain amount of cholesterol and other lipids, an excessive amount of them can cause problems. As they flow through the blood stream, they can stick to the artery walls. Statistics show that a high level of cholesterol in the blood is major cause of atherosclerosis and the higher the level

of cholesterol in your blood, the more the likelihood of developing cardiovascular disease.

What is atherosclerosis? As people get older, fats and other substances carried in the blood build up into deposits on the vessel wall called *plaque.* This is known as atherosclerosis or hardening of the arteries. As the deposits become larger, the passage inside the vessels becomes narrower or blocked. Atherosclerosis is a slow progressive disease that may begin in childhood. It often takes years for the plaque to build up inside any artery in the body. Atherosclerosis can lead to many types of circulatory problems.

Two of the major problems due to atherosclerosis are heart attacks and strokes.

Atherosclerosis is especially likely to be a major problem when it develops in the coronary arteries that carry blood deep into the heart muscle. If plaque builds up inside these arteries and blood can't get to the heart, the heart muscle may become "starved" for the oxygen carried by the blood. When atherosclerosis involves one or more of the coronary arteries, it is called **Coronary Artery Disease, CAD**.

Coronary artery disease is by far the leading cause of serious heart problems such as **angina pectoris** (chest pain due to heart disease) and **myocardial infarction (heart attack**). Angina pectoris, or angina, occurs when the heart temporarily does not get enough oxygen. A myocardial infarction can occur when the heart muscle gets very little or no oxygen for a period of time. The area of the heart muscle that loses its oxygen supply is permanently damaged, and the heart muscle cells in that area die.

Overweight. Being overweight puts an extra work load on the heart. Even when at rest, an overweight body must work harder to breathe because it needs more oxygen.

According to statistics: -

- The rate of heart attacks among people who are overweight is greater than that for people of normal weight,
- The risk of death or serious cardiac disease increases in direct relation to the degree of overweight for individuals whose weight is more than 30% greater than normal;
- Overweight people are much more likely to have hypertension, and they often have high levels of cholesterol and other fats associated with increased risk of heart problem.

Stress. Stress can result from outside pressure such as business and family problems or inside causes such as worry or emotional depression. During periods of stress, the body may react in a way that the heart beats more rapidly, and the blood pressure rises.

8.5 Lifestyle Modification

For most of the Cardiovascular diseases apart from hypertension, lifestyle modification is a very effective preventive and even treatment option.

Blood pressure can be lowered without drugs in many individuals by losing weight, restricting intake of salt, and learning to overcome stress if one has mild hypertension. Doing the above may be enough to lower the blood pressure into the normal range, and one may not need any drugs. For others, it may result in the need for fewer drugs or lower dosages of drugs.

Statistics show that a reduction of blood pressure often occurs with **weight loss** and if the extra weight is not restored, the blood pressure often remains lowered. Weight loss may be the only treatment required for some hypertensive people. As important, once lost, effort must be maintained to avoid regaining the extra weight.

Reducing the daily salt intake can be done by following a few simple guidelines, including:

- Eat meat, vegetables, and fruits that are fresh, rather than processed;
- Read the labels on canned or processed foods and beverages and avoid those that have a high salt or 'sodium' content;
- Add no extra salt at meal;
- Avoid salted foods such as bacon, sausages, pickles, mustard and some cheeses;

Reducing intake of cholesterol and saturated fats

An intake of some cholesterol and other fats found in food is essential for energy and general good health. However, the amount and kind of fat in the diet should be controlled because the amount affects the weight, and the kind of fat affects the cholesterol level. In addition to cholesterol, there are two other types of fat in food that can change the level of cholesterol in the blood. There are **saturated fats** (found in meat, butter, margarine, and even some plant, and oils) that may increase cholesterol levels in the blood, and there are **unsaturated fats** (for example, in corn oil, olive oil, and fish oils) that may help reduce blood cholesterol.

Some useful guidelines are:

- Avoid high cholesterol foods such egg yolks, fatty meals, and organ, meats (liver and kidneys):
- Avoid or reduce your intake of foods that are high in saturated fats, including animal and dairy products such as meats, cream, cheese, and butter;
- Have foods in your diet that contain unsaturated fats, including vegetable products such as corn oil, soyabean oil, and other vegetable oil.

Smoking. One year after cessation of smoking, the extra risk of a heart attack will decrease by almost 50%. The risk of death

from other diseases also decreases with time. No matter your age or how long you have been smoking, quitting now can improve your overall health and increase your life expectancy.

Stress. Knowing how to avoid or relieve stress directly or indirectly benefits people with high blood pressure. A direct benefit may be reducing blood pressure, and an indirect benefit may be making one 'feel better' physically and emotionally.

While it is virtually impossible for anyone to completely avoid stress, it can be reduced and controlled. Thus, *"whatever steals your peace is not worth your attention". [Be anxious for nothing, but in everything by prayer and supplication, with thanksgiving, let your requests be made known to God: and the peace of God, which passes all understanding, will guard your hearts and minds through Christ Jesus.* ***Phil. 4:6, 7****; NKJV].*

Exercise. People who get little or no exercise and are not physically fit may be more likely to have a heart attack and slower to recover after having one. However, by exercising regularly according to a program which should be suggested by your physician or other appropriate expert;

- Your heart will pump blood more efficiently, your circulation will improve and your blood pressure may be reduced;
- Your blood levels of cholesterol and other lipids may be improved and, thus may slow down the development of atherosclerosis;
- Your physical strength and muscle tone will improve and, thus, you will tire less easily and be able to do more,
- Your mental state may improve and allow you to cope better with stress, relax more easily and sleep better;
- Your weight can be controlled more easily with a reasonably proper diet.

When are drugs needed to control blood pressure?

Certain drugs may be prescribed to decrease the blood pressure if it is elevated and cannot be reduced to the normal range by dietary and/or other non drug treatment measures.

If the doctor prescribes medication to control your hypertension, it is important that you take it exactly as instructed, and comply to clinic appointments / check-ups, as directed.

9.0 OUR CONTRIBUTIONS TO ISSUES OF THE MATTER OF THE HEART

9.1 Pivotal Role on Diastolic Dysfunction

Our prompting into the *medical matter of the heart* had been particularly fired from June 1998, after passing the Part 1 Fellowship examination of the National Postgraduate Medical College of Nigeria the previous month. Having been inspired to settle for Cardiology as a Specialist study area – for the purpose of my Part II Qualifying Exams as a Specialist Physician, and subsequently, main area of work in life – the hurdle to cross towards this was to decide on a topic for the dissertation that would take 3 years of dedicated research to complete work on.

Then, followed an unrelenting search that took poring over countless literature, medical volumes, MEDLINE search and overseas E-library mailings. There were interspersing sessions of brain – storming with senior colleagues and mentors, particularly Prof Oli, (then Drs) VO Ikeh and BJC Onwubere. Out of a list of more than 15 possible topics, and the routine of delisting and enlisting – no thanks especially to the (thankfully now) unyielding stance of 'VO'- we arrived at a study on Diastolic dysfunction of the heart. This was to use adult hypertensive Nigerians as surrogates[55].

As at this point, **in the late 1990's**, **Diastolic dysfunction** as an area of study – and then – unfolding handle to many critical health outcomes concerning **the matters of the heart** (viz **H**ypertension, **C**ongestive Heart failure (CHF),

Cardiomyopathies, Ischaemic Heart Diseases, **V**alvular Heart Diseases, **D**iabetic [Endocrine] and **R**enal affectations of the heart, as well as **E**xercise/**E**lectrophysiological studies), had only come into the lexicon of researchers and the lenses of Cardiologists, for just about a decade. Information on it was limited.

As it turned out, this study proved to be a ***seminal work*** not only in the field of Hypertension and Cardiology, but also in the country, and among blacks. There had only been one other work, mentioning Diastolic dysfunction as an entity, in the country by this time, that by Balogun et al [56].

As a result of this and other related subsequent works, there is now an armada of published work and a gamut of ongoing researches on this pivotal area of Cardiovascular medicine in Nigeria. With more awareness globally on the subject of Diastolic dysfunction, Cardiovascular Medicine, Congestive Heart Failure, and especially Hypertension, have been better served in understanding of the pathophysiologic mechanisms, treatment and management options, as well as outcome.

9.2 Other Works on the Matter of the Heart

- Our odyssey with issues of the matter of the heart, medically, was further fuelled when our *hospital based studies* brought to the fore, the horrendous magnitude and burden of these disease conditions – far surpassing the prevailing comorbidity pattern, when compared with other medical illnesses.This had dated back to 1999, and has served to generate vital local data on cardiovascular rated diseases, from the early 90's till date[57-61].
- We have followed up with *epidemiological Community/population – based studies*, as well as on the Knowledge, Awareness, Application and Practice (KAAP) concerning Cardiovascular Diseases in different segments of the society – students[62], rural community[63], priests[64],

orthopaedic and plastic patients[65], as well as adult hypertensives[66].

- Works on *socioeconomic and psychological problems confronting the raising of* manpower to combat these scourges – with **the resident doctors training**[67], the syndrome of **brain drain** of hordes of qualified medical personnel[68]; to when the **economic meltdown** set in[69].;added to which was determining the factors affecting the **efficiency** and productivity of these health practitioners in their places of work[70,71].

- Tackling specific issues and challenges that had to deal with *the treatment and control of hypertension*[72,73].

- *Conducting drug trials* for antihypertensive drugs[74,75], in collaboration with other colleagues, as well as pointing to *specific treatment outlines*[76].

- Specific forays into *evaluation* (both clinically and with tools) *of different aspects of Cardiovascular Diseases*, their *prevalence in our environment*, vis a vis their management – Endomyocardial fibrosis[77], Atrial fibrillation and Cardiac arrhythmias[78,79], Congenital Heart Diseases[80,81],Valvular Heart Diseases[82], Rheumatic Heart Diseases[83], Ventricular and Atrial Septal Defects[84,85], Pulmonary Artery Hypertension[86], Congestive Heart Failure[87], Chronic Kidney Diseases[88,89], Thyrotoxicosis[90], Sickle Cell Anaemia[91-95], Intracardiac masses[96], as well as Echocardiographic and Electrocardiography evaluation modalities[97-99].

- Work on *comorbidities and noted complications of these cardiovascular diseases*, as well as their roles in fast-tracking the denouement of these matter of the heart affectations[100-105].

- Particular engagement with the *mortality, case fatality rate and significant risk ratios of these cardiovascular disorders*, providing relevant data for strategic policy framework[106-108].

- Portrait of *emergency salvaging techniques* to prevent/delay the decimating scourge of these cardiovascular diseases[109].
- Particular concern with, and *work on,* ***diastolic dysfunction*** [50,110-114].

9.3. Paper Presentations

Apart from these engaging involvements in targeted research on the matter of the heart (cardiovascular diseases), we have been actively involved in local, national and international cardiovascular meetings and scientific conferences attendance, facilitating and planning. This has resulted in the delivery of **52 paper presentations specifically** in **areas of the matter of the heart**.

10.0 RECOMMENDATIONS/WAY FORWARD

10.1 What More Needs to be Focused on in the Matter of the Heart, in Our Context?

- We realize the challenges that still confront us as a behemoth in these diverse matters of the heart, but are encouraged to know that most of them are eminently preventable / can be palliated (including the spiritual matter of the heart that, unattended to, resultantly leads to a dislocated life, disenchanted existence, peace-less heart and a damned future, from sin).
- The way forward in medical matters of the heart revolve around ***3P's*** – The roles of the (i) General Public (ii) Practitioners/Practice (iii) Policy makers–Institutional and National.

10.2 The General Public

Everyone needs to leave this lecture venue with the clear awareness and understanding that, for all persons with Cardiovascular diseases, and especially the infamous queen of

them all, Hypertension), the potential benefits of lifestyle modification – i.e. healthy diet, weight control, salt restriction, cessation of cigarette smoking, regular exercise – which are cost-effective measures, need to be continually emphasized and advocated. These have the potential to improve blood pressure control, reduce the need for/dose of medications, improve the quality of life, and avert dire consequences.

Preventive campaigns that utilize the understanding of promoting heart – healthy behaviours and target risk behaviours are, therefore, paramount, and are required from all of us.

10.3 Practitioners/Practice

10.3.1 Since Heart Failure (HF) is an ocean in which most of the rivers of the other Cardiovascular Diseases must necessarily and dutifully empty into; and knowing that there is a truision in **Sir Thomas Lewis (1913**) saying that "the very essence of Cardiovascular medicine is the early detection of HF" [115]; and yet realizing the poor prognosis of HF (25% - men;38% women, 5 year post – diagnosis survival rate (Framingham), despite currently available, and recent innovation in, therapy; there is need, now, for:

(i) Large targeted placebo-controlled trials for DD (Diastolic dysfunction – the earliest marker of heart disease) – targeted at therapy: to elucidate further the optimal treatment options, and improve the prognosis of (Diastolic) Heart Failure.

(ii) Characterizing Diastolic dysfunction in different and special populations, eg blacks, as data on prevalence and import of Diastolic dysfunction on black African subjects are limited, and it is known that sex, racial/ethnic variations obtain in the heart response to adverse stimuli.

(iii) More vigorous application of assessment of the potentially modifiable newer parameters for Cardiovascular Risk Factors – such as Homocysteine, C- Reactive Protein,

Fibrinogen levels-in the bid to lessen the burden and scourge of Cardiovascular diseases.

10.3.2 Emphasis on management of HF is shifting towards early interventions, preservation of quality of life (QALY's) gained, and lowering mortality. The priority, therefore, is on Primary Prevention, Early Recognition and Prompt Diagnosis.

With the high prevalence of Hypertension in Nigeria (22.5-25%), and attendant significant morbidity and mortality of, especially resultant Hypertensive Heart Failure*; identification of high risk patients* before overt complications occur, is highly desirable, as this impacts positively on outcome.

The European Society of Cardiology (ESC) has recommended a diagnostic preventive tool to detect Asymptomatic Left Ventricular Systolic Dysfunction (ALVSD) – a precursor of HF in high risk patients – a serologic marker that is relatively inexpensive, for use in disadvantaged settings, such as is found in our environment, the cardiac neurohormone biomarker, B type Natriuretic Peptide (BNP).

There is the need, in our resource – poor setting to**: i**)Source for, and *employ assiduously the* cost – effective *BNP markers for prompt diagnosis*, cardiovascular risk monitoring and prognostication of HF, as it offers simple and objective measurement of cardiac function

Progressively *deploy* other serologic markers, which are noninvasive surrogates for histopathological diagnosis of Diastolic dysfunction- such as Serum Carboxy Terminal Peptide of Procollagen Type1, *in our transition quest to more preventive care of the matters of the heart.*

10.4 Policy Makers/Framework

10.4.1 Institutional

The *Cardiology unit* of the Department of Medicine, of the University of Nigeria, is long overdue for an *upgrade to sub*

departmental status. An *Institute of Cardiology/Cardiovascular Medicine* should even now be a more suitable designation/creation by the relevant University/National Universities Commission authorities. This is to accommodate the continually growing and emerging needs of the Cardiovascular Diseases as the most important cause of Non Communicable Diseases worldwide and yet ravaging not less than one – third of our adult population in Nigeria. There is thus the critical need to pool experts together who are actively engaged in research, teaching and dissemination of vital knowledge /statistics, to harness these together and help contend with the as–yet many unanswered and begging **questions of the matter of the heart.**

10.4.2 National

The National Health Policy needs to

(i) Evolve to more pragmatically *capture the treatment and even surgical attention* (as may be necessary) of these matters of the heart/cardiovascular diseases (**so dear to all our hearts**) in the *National Health Insurance Scheme*. Since some of these ailments are life long, and many of them capital intensive in evaluation and treatment, they need to be greatly funded/subsidized, to ease the burden of cost and enhance the productivity of their sufferers in different sectors of the economy, as well as their sense of well being..

(ii) Develop a Protocol, as a matter of urgent national importance, whereby many of the investigative tools and diagnostic agents – most of which we have not yet developed technology for – attract Import Duty waiver and prompt clearance. This will definitely boost our cardiovascular health service delivery and greatly *attenuate* the haemorrhaging capital flight that obtains currently, through *medical tourism* for proper diagnosis and adequate treatment abroad.

(iii) *Thoroughly equip our centres*, especially the National Cardiothoracic Centre of Excellence, here in the University of Nigeria Teaching Hospital (UNTH), Enugu (so designated since

the 1980's, but more in name, than in function), and even Regional Centres in the geopolitical zones, *with state of the art functional equipment* (a' la **Boris Yeltsin** of Moscow Cardiac Centre).

The **UNTH/University of Nigeria College of Medicine** currently harbours some of the best brains and most experienced hands in the field of Cardiology, nay Cardiovascular Medicine and Surgery, in the West Coast of Africa, with the **greatest number of the pool of specialists** in any one institution in Nigeria currently - brimming with such personages as Professors Anyanwu, Ikeh,Aghaji, Onwubere and Ike, among 9 (nine) other Cardiovascular / Cardiothoracic Specialists - who had shown early in the day a trail-blazing pathway (with the first Open Heart Surgery in Nigeria, done here, in 1974).

A **fully functional Cardiothoracic Centre** comparable with the best anywhere else in the world is the least we can ask for in this most populous black nation on earth, which now has the largest (rebased) growing economy in Africa!

11.0 CONCLUSION

In a lecture delivered on the occasion of the presentation of a copy of Harvey's *De Motu Cordis in* Chicago, in **1956,** *Dr Louis Katz, a* notable Cardiologist and a knowledgeable researcher in **matters of the heart** in his day, had stated: *"Research is a dignified profession, to be pursued only by the consecrated and inspired... It cannot be placed on a business footing where one new fact is to be returned for each quantum of dollars invested. Great discoveries are not produced on the assembly line. Only duplicates can be so manufactured. The original must come about through the deliberate activity of a creative mind. …. Great discoveries evolve—they are not delivered on call. This was the case with Harvey*[116].

And exactly so has been **the journey of the matter of the heart** till date. As the frontiers of development of issues of the heart evolve in better understanding of the mechanisms, more innovative evaluation and diagnostic methods/ facilities, and *hopefully*, better preventive approaches, potentiated curative applications, healthier prognosis, and enhanced quality of life, welcome to a holistic paradigm shift, in **matters of the heart**.

GRATITUDE

Once more, to the Paragon Omniscient Cardiologist, the Lord God Almighty, in whom we live and move and have our being, be all the glory, adoration and majesty for the gift of life, purposeful living and His innumerable blessings.

And to Jesus Christ, the Saviour and Redeemer of our souls, the Quintessential Cardiologist, Interventional and holistic Cardiothoracic Surgeon, be all praise, for the work of a new heart, new life (second birth), as the progenitor of inspiration.

I sincerely again thank our pragmatic, well-versed and tutored, principled and visionary Vice Chancellor, who understands the pulsating matter of the heart right from the womb, Prof B. C. Ozumba, for this opportunity to deliver this 87th Inaugural Lecture.

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My working life outside my family had literally revolved around a tripod of ***3C's*** – namely; the **C**lassroom (academics), the **C**linic (bedside, in medical practice) and the **C**hurch (the pulpit and spiritual concerns).

In each of these areas I owe so many people unqualified gratitude.

May I express a token of appreciation for what these personalities and mentors have been in my life: ***Most*** Rev. Dr. Emmanuel Chukwuma, the intelligent and bold Bishop and Archbishop, who despite a tough exterior, has a ***simple heart*** that looks out for others; ***Ven***. Professor Chinedu Nebo, the administrator with passionate preaching, the man with a ***heart of good*** for all; ***Ven***. Professor Ernest Ukaejiofo, the consummate academic–researcher, with a ***pastoral heart*** to the core; ***Professo***r Benjamin Osisioma, the brilliant academic who preaches with panache, has always been there for us, with a ***heart*** that feels at home with everyone; ***Ven***. Professor Ernest Onwasigwe, the current Provost of the College of Medicine, my first supervising Archdeacon, a man with a ***large heart***; and ***Elder*** Goddy Orjinta, a man God used on that Sunday morning early in January 1977, at FGC Enugu to open my ***heart*** to the saving knowledge of the *master cardiologist*, Jesus Christ; as well as ***Rev***. Professor Uzo Aniebue – my colleague in the shepherding ministry for years now, a man with a loyal and ***sincere heart***. .

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Your Lordships, Distinguished academics, Ladies and gentlemen, Lions and lionesses, once more, thank you for giving me the honour of your presence today, and your unparalleled attention.

May God bless you all abundantly and grant you safety in *matters of the heart* and as you journey home, in Jesus name. Amen!

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