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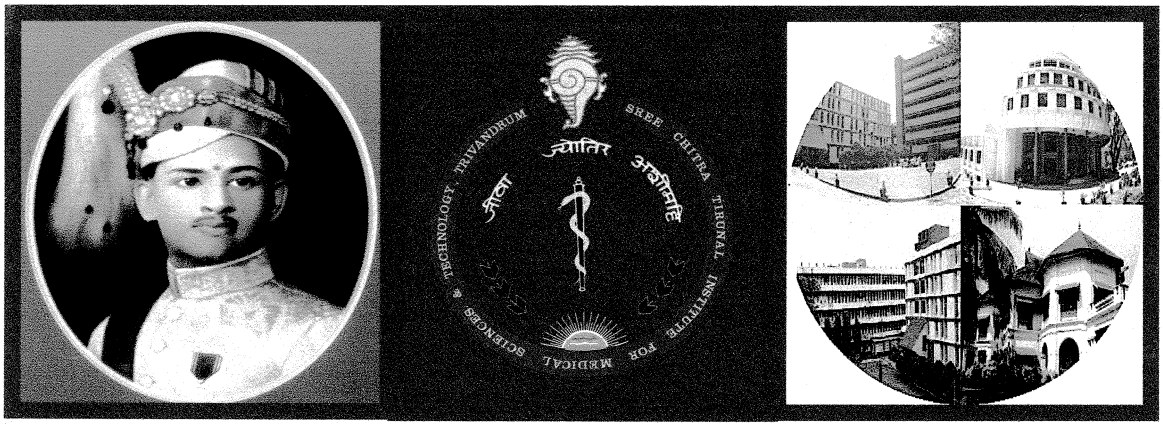

DIPLOMA IN CARDIAC LABORATORY TECHNOLOGY

Workbook submitted by

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DEPARTMENT OF CARDIOLOGY,
SCTIMST,
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2005-2006

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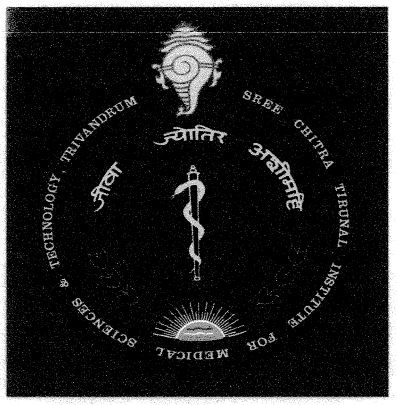


CERTIFICATE

I Mr. ANEESH..... here by declare that I have actually performed all the procedures listed under the course of Diploma in Cardiac Laboratory Technology (DCLT)

Signature .....

Name ..ANEESH.S....

Place: THIRUVANANTHAPURAM

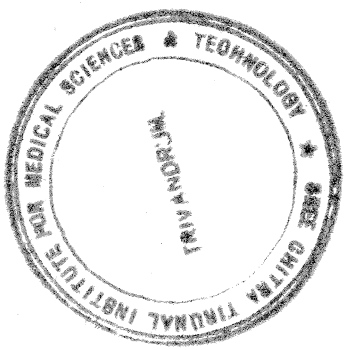
Date: 01/12/06

Forwarded he had carried out the minimum requirements of procedure for the completion of the course.

Signature.....

Head of the Department of Cardiology,

SCTIMST, Thiruvananthapuram.



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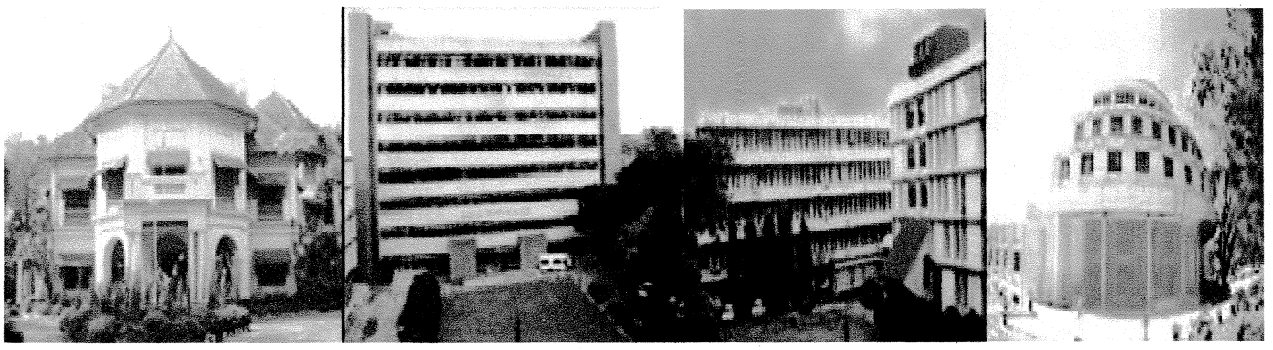

The Sree Chitra Tirunal Institute for Medical Sciences &Technology (SCTIMST), Thiruvananthapuram is an Institute of National Importance established by an Act of the Indian Parliament. It is an autonomous Institute under the administrative control of the Department of Science and Technology, Government of India.

The Institute signifies the convergence of medical sciences and technology and its mission is to enable the indigenous growth of biomedical technology, besides demonstrating high standards of patient care in medical specialties and evolving postgraduate training programs in advanced medical specialties, biomedical engineering and technology, as well as in public health.

It has a 239-bedded hospital for tertiary care of cardiovascular and neurological diseases, a biomedical technology wing with facilities for developing medical devices from a conceptual stage to commercialization, and a center of excellence for training and research in public health.

The Institute has the status of a University and offers postdoctoral, doctoral and postgraduate courses in medical specialties, public health, nursing, basic sciences and health care technology. It is a member of the Association of Indian Universities and the Association of Commonwealth Universities.

THE COURSE

Diploma in Cardiac Laboratory Technology (DCLT) is the first Diplom course started by the institute in the year of 1991. This course enable students to manage an invasive as well as a non-invasive lab of cardiology. I trains the students to use all the machines in the ECG, Echo, TMT, Holter an cardiac catheterization laboratory.

The first year of the course is in non-invasive labs such as ECG, TM Echo and Holter labs. Students are trained to independently take ECG'S an assist in the treadmill test and in connecting and analyzing the Holte recordings. He is assisting the cardiologist in the Echo room for all Ech procedure such as Trans Thoracic Echo, Trans Esophageal Echo, Stress Echo Contrast Echo and Tissue Doppler Echocardiography.

The second year of the course is fully in cath lab which gives the studen to get a deep knowledge in the hardware's used in the field of interventiona cardiology, and to know about the clinical, pathological and pathophysiologica conditions in cardiology their diagnosis and therapy. As an institute, whic carries out all the new modalities in intervention, we are able to know mor about the Electrophysiological ideas and pacemaker implantation and th pediatric interventions.

A whole year experience in the cardiac cath lab makes the student able to handle all the emergencies in the cath lab.

Educational Qualification: BSc in Physics from a recognized university with a minimum of 60% mark and good academic performance.

Duration: Two calendar years.

Admission: Through a common Entrance Test and followed by an interview.

THE WORKING PLACE

During the commencement of the course the students have to work in the following place in weekdays except Sunday.

NON-INVASIVE LABS:

ECG lab: We had a well equipped ECG lab with three machines BPL CARDIART 108T/K, Hewlett packards page writer, and GE's MAC500 portable ECG machine. The working time is from morning 8.00 to evening 4.00 and then with a call duty for emergency cases at the night. Daily we have an average of 80 to 100 cases of ECG.

TMT lab: We had a TMT machine of Marquette. The working hours are from morning 8.00 to evening 4.00. Daily we had a minimum of 10 TMT cases.

Holter Lab: We had three Holter machines of Delmar. Usually we have three connections and analyses three cases also.

Echo Lab: We had four echo rooms with a sonos 1000, CFM 500, System Five from GE and a VIVID 7 from GE. The working hour is from morning 9.00 to evening 6.00. We daily have a case of minimum 40 to 50 in a single lab.

INVASIVE LABS:

The Cardiac Catheterization Lab: We had three-catheterization lab of Philips integris, Philips BV 300 plus, and a Siemens Elema. The Philips Integris lab is exclusively for coronary intervention and pediatric interventions, the Philips BV plus is a portable machine in this lab we are doing pacemakers and Electrophysiological studies.

The working hour of cardiac cath lab is from morning 7.00 to evening 7.00 or it extends till the last case ends. We usually had an average of 15 cases per day.

ACKNOWLEDGEMENT

First and foremost I would like to thank the Head of the Department c Cardiology **Prof. Jagan Mohan Tharakan** who guided me through th different phases of studies and encouraged and helped me in all the aspect c my training.

I thank the Director of this Institute **Dr K. Mohandas**, Dean **Dr K Radhakrishnan** and Registrar **Dr A.V. George** for their valuable advice, hel and attention towards me.

I express my gratitude to Professor **Dr Thomas Titus** and Professo **Dr Ajith Kumar** for their invaluable advice, which helped me for th successful completion of the course. I also thank all other faculty members c the Department who helped and encouraged me in technical studies.

I extend my heartful thanks to all the members of the technical staf who are my teachers, for their timely guidance and ideas that helped me t learn more. I am grateful to all the P.G students of the Department c Cardiology. I am also thankful to all my friends who helped me during th study in the Institute and the patients who were the core medium of th study.

Last but not least I would like to acknowledge my sincere thanks to m senior and juniors and my colleague for their co-operation in the work place and in studies. I also thank my well wishers who helped me in all the wa during the last two years of my study.

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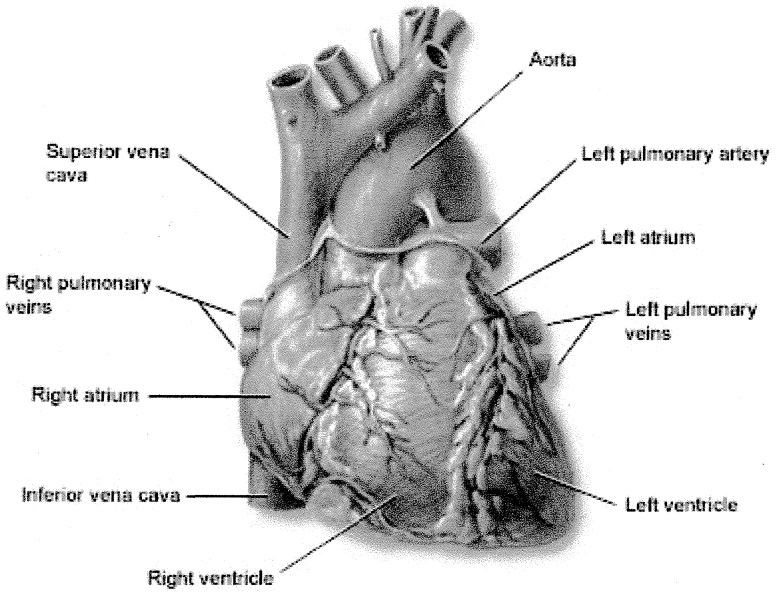
HEART ANATOMY



Introduction

The **Heart** is a hollow, muscular organ in vertebrates, responsible for pumping blood through the blood vessels by repeated, rhythmic contractions, or a similar structure in annelids, mollusks, and arthropods. The term *cardiac* (as in cardiology) means "related to the heart" and comes from the Greek *kardia*, for "heart." The heart is composed of cardiac muscle, an involuntary muscle tissue which is found only within this organ.

The heart is the organ responsible for pumping blood throughout the body. The normal heart has a right side and left side, separated by a wall (septum). Each side is further divided into an upper collecting chamber (atrium) and lower pumping chamber (ventricle). A series of valves located in the heart help regulate blood flow in the right direction. Arteries are the large blood vessels, which take blood away from the heart; veins are blood vessels returning blood to the heart.

The heart weighs between 7 and 15 ounces (200 to 425 grams) and is a little larger than the size of your fist. By the end of a long life, a person's heart may have beat

(expanded and contracted) more than 3.5 billion times. In fact, each day, the average heart beats 100,000 times, pumping about 2,000 gallons (7,571 liters) of blood.

The heart is located between the lungs in the middle of our chest, behind and slightly to the left of our breastbone (sternum). The heart is usually felt to be on the left side. The left lung is smaller than the right lung because the heart occupies more of the left hemithorax. A double-layered membrane called the pericardium surrounds our heart like a sac. The outer layer of the pericardium surrounds the roots of our heart's major blood vessels and is attached by ligaments to our spinal column, diaphragm, and other parts of our body. The inner layer of the pericardium is attached to the heart muscle. A coating of fluid separates the two layers of membrane, letting the heart move as it beats, yet still be attached to our body.

Our heart has 4 chambers. The upper chambers are called the left and right atria, and the lower chambers are called the left and right ventricles. A wall of muscle called the septum separates the left and right atria and the left and right ventricles. The left ventricle is the largest and strongest chamber in our heart. The left ventricle's chamber walls are only about a half-inch thick, but they have enough force to push blood through the aortic valve and into our body.

Pericardium

The **Pericardium** is a double-walled sac that contains the heart and the roots of the great vessels.

There are two layers to this sac: the fibrous pericardium and the serous pericardium. The serous pericardium, in turn, is divided into two layers; in between these two layers there is a space called the pericardial cavity.

The **Fibrous pericardium** is the most superficial layer. It is a dense connective tissue, protecting the heart, anchoring it to the surrounding walls, and preventing it from overfilling with blood. It is continuous with the outer adventitial layer of the neighboring great blood vessels.

The **Serous pericardium** is deep to the fibrous pericardium. It contains two layers, both of which function in lubricating the heart to prevent friction from occurring during heart activity.

- The layer next to the fibrous pericardium is the parietal layer.
- The layer next to the heart is the visceral layer, also known as the epicardium.

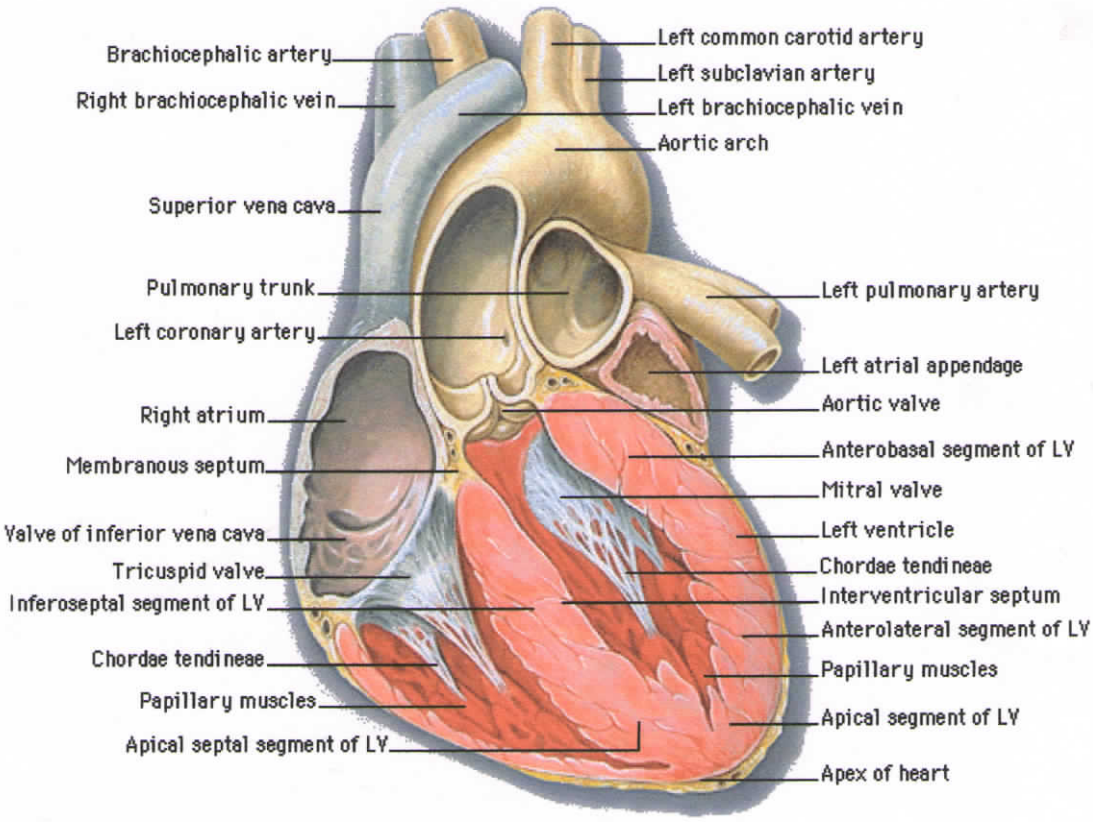
Together these two layers form a continuous uninterrupted membrane. Between these two layers exists a small cavity called the pericardial cavity, which contains a

supply of serous fluid.The serous fluid that is found in this space is known as th pericardial fluid.

Pericardial fluid is secreted by the serous membrane on the pericardious sac c the outside of the heart. The pericardial cavity contains between 15 and 50ml pericardial fluid. It is similar to the serous fluid that is found in the brain for cushionir and ability to move semi-freely.

Cardiac Chambers

The heart consists of four chambers: the right atrium, right ventricle, left atriur and left ventricle.



Right Atrium

The **Right Atrium** is a right posterolateral chamber. The Right Atrium is largo than the Left Atrium but has thinner walls. The Right Atrium has two major veins th returns blood to the heart from all parts of the body. Two major veins returning th blood to the heart are the Superior Vena Cava and the Inferior Vena Cava. These tw

veins are sometimes called the "Great Veins". The Superior Vena Cava returns the deoxygenated blood from the upper part of the body and the Inferior Vena Cava returns the deoxygenated blood from the lower part of the body. The Right Atrium also receives blood back from the heart muscle itself (coronary sinus) and numerous small Thebasian veins. After the blood is collected in the Right Atrium it is pumped into the Right Ventricle through the Tricuspid Valve (three leaf valve). The sinoatrial node sends an impulse that causes the cardiac muscle tissue of the atrium to contract in a coordinated, wave-like manner.

Structurally, the Right Atrium consists of free wall and septal components. The free wall has a smooth posterior region and a more muscular anterior region. The posterior aspect receives the two caval veins and has a veinlike appearance. The anterior aspect exhibits a muscular wall and a large pyramidal atrial appendage. A prominent C-shaped ridge of muscle, the Crista Terminalis serve to separate the two regions and also froms one of the tracts for internodal conduction .Numerous Pectinate muscles arise from the crista terminalis and travel as parellel ridges along the anterior aspect of the free wall.When viewed from the right, the septum has an interatrial component and an atrioventricular component. The interatrial portion is relatively small, and its most prominent feature is fossa ovalis. This consist of a horseshoe shaped muscular rim-Limbus, which forms a pathway for internodal conduction –and central sheet of thin fiberous tissue-the valve of Fossa Ovalis. The atrioventricular septum corresponds to the triangle of Koch, an important anatomic surgical landmark because it contains the atrioventricular node and the proximal portion of the His bundle.

Right Ventricle

The **Right Ventricle** is triangular in form, and extends from the right atrium to near the apex of the heart. Its anterosuperior surface is rounded and convex, and forms the larger part of the sternocostal surface of the heart. Its under surface is flattened, rests upon the diaphragm, and forms a small part of the diaphragmatic surface of the heart. Its posterior wall is formed by the ventricular septum, which bulges into the right ventricle, so that a transverse section of the cavity presents a semilunar outline. Its upper and left angle forms a conical pouch, the conus arteriosus, from which the pulmonary artery arises. The right ventricle receives de-oxygenated blood as the right atrium contracts. The pulmonary valve leading into the pulmonary artery is closed, allowing the ventricle to fill with blood. Once the ventricles are full, they contract. As the right ventricle contracts, the tricuspid valve closes and the pulmonary valve opens. The closure of the tricuspid valve prevents blood from backing into the right atrium and the opening of the pulmonary valve allows the blood to flow into the pulmonary artery toward the lungs. The walls of the Right Ventricle are a little thicker than the Right Atrium.

The right atrioventricular orifice (tricuspid orifice) is the large oval aperture of communication between the right atrium and ventricle. Situated at the base of the ventricle.There are two papillary muscles, anterior and posterior: of these, the anterior is the larger, and its chordae tendineae are connected with the anterior and posterior cusps of the valve: the posterior papillary muscle sometimes consists of two or three parts; its

chordae tendineae are connected with the posterior and medial cusps. A muscular band, well-marked in sheep and some other animals, frequently extends from the base of the anterior papillary muscle to the ventricular septum. From its attachments it may assist in preventing overdistension of the ventricle, and so has been named the moderator band.

Left Atrium

The **Left Atrium** is a midline posterior chamber. The left atrium receives oxygenated blood from the lungs through the pulmonary veins. As the contraction triggered by the sinoatrial node progresses through the atria, the blood passes through the mitral valve into the left ventricle.

Structurally, the Light Atrium consists of free wall and septal components.The free wall includes a dom-shaped body, which receives the pulmonary veins, and a fingerlike appandage. These two regions are separated externally by the left atrial coronary vein and ligament of marshell, and internally by the ostium of the appandage. The appandage contains numerous small pectinate muscles. Outside the appandage, the body of the left atrium contains no pectinate muscles, and there is no terminal crest. When viewed from the left, the septum is entirely interatrail. Along its anterosuperior border, the valve of fossa ovalis contains one or more fenestrations. Neither the limb of the oval fossa nor the atrioventicular septum is visible from the left atrium. Several small thebesian veins drain directly into the left atrial cavity, particularly along the septum.

Left Ventricle

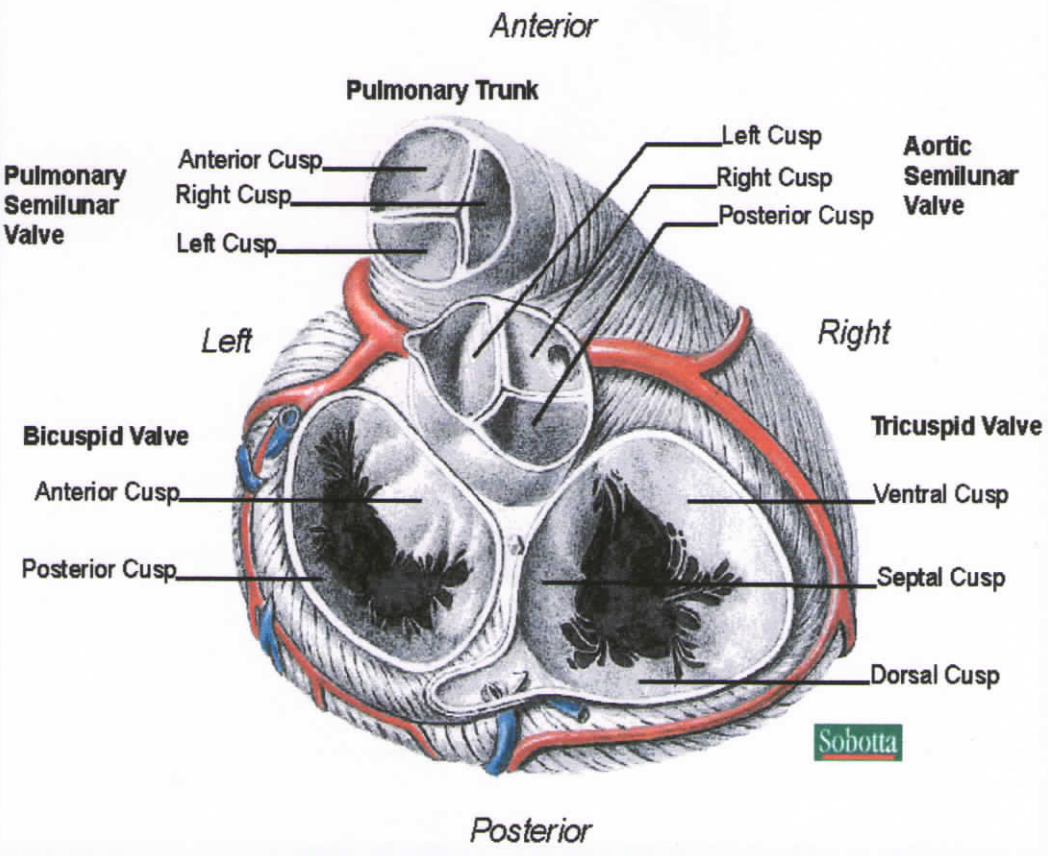
The **Left Ventricle** is longer and more conical in shape than the right, and on transverse section its concavity presents an oval or nearly circular outline. It forms a small part of the sternocostal surface and a considerable part of the diaphragmatic surface of the heart; it also forms the apex of the heart. Its walls are about three times as thick as those of the right ventricle. This is important because the oxygenated blood that it receives from the Left Atrium has to be pump throughout the body. The Bicuspid Valve closes and the blood is collected in the Left Ventricle. The closing of the Bicuspid Valve stops the backflow of blood. When the Heart muscle contracts the blood is forced through the Aortic Semilunar Valve which has the same features as the Pulmonary Valve. The blood then passes through the Aortic Semilunar Valve into the Aorta.

The left atrioventricular opening (mitral orifice) is placed below and to the left of the aortic orifice. It is a little smaller than the corresponding aperture of the opposite side, admitting only two fingers. It is surrounded by a dense fibrous ring, covered by the lining membrane of the heart, and is guarded by the bicuspid or mitral valve. The aortic opening is a circular aperture, in front and to the right of the atrioventricular, from which it is separated by the anterior cusp of the bicuspid valve. Its orifice is guarded by the

aortic semilunar valves. The ventricular septum is directed obliquely backward and to th right, and is curved with the convexity toward the right ventricle: its margins correspon with the anterior and posterior longitudinal sulci. The greater portion of it is thick an muscular and constitutes the muscular ventricular septum, but its upper and posteric part, which separates the aortic vestibule from the lower part of the right atrium an upper part of the right ventricle, is thin and fibrous, and is termed the membranou ventricular septum. An abnormal communication may exist between the ventricles at thi part owing to defective development of the membranous septum. The musculi papillare are two in number, one being connected to the anterior, the other to the posterior wal they are of large size, and end in rounded extremities from which the chordae tendinea arise. The chordae tendineae from each papillary muscle are connected to both cusps c the bicuspid valve.

Cardiac Valves

The heart consists of mainly four valves : Mitral valve, Tricuspid valve,Pulmonar valve & Aortic valve



Tricuspid valve

The **Tricuspid Valve** is on the right side of the heart, between the right atrium and the right ventricle. The normal tricuspid valve usually has three leaflets and three papillary muscles. The tricuspid valve consists of three somewhat triangular cusps or segments. The largest cusp is interposed between the atrioventricular orifice and the conus arteriosus and is termed the anterior or infundibular cusp. A second, the posterior or marginal cusp, is in relation to the right margin of the ventricle, and a third, the medial or septal cusp, to the ventricular septum. They are formed by duplicatures of the lining membrane of the heart, strengthened by intervening layers of fibrous tissue: their central parts are thick and strong, their marginal portions thin and translucent Tricuspid valves may also occur with two or four leaflets, and the number may change during life.

Pulmonary valve

The **Pulmonary Valve** (or **pulmonic valve**) is the semilunar valve of the heart that lies between the right ventricle and the pulmonary artery and has three cusps. Two in front and one behind, formed by duplicatures of the lining membrane, strengthened by fibrous tissue. They are attached, by their convex margins, to the wall of the artery, at its junction with the ventricle, their free borders being directed upward into the lumen of the vessel. The free and attached margins of each are strengthened by tendinous fibers, and the former presents, at its middle, a thickened nodule. Similar to the aortic valve, the pulmonic valve opens in ventricular systole, when the pressure in the right ventricle rises above the pressure in the pulmonary artery. At the end of ventricular systole, when the pressure in the right ventricle falls rapidly, the pressure in the pulmonary artery will close the pulmonic valve.

Mitral valve

The mitral valve (also known as the bicuspid valve or left atrioventricular valve), is a dual flap (bi = 2) valve in the heart that lies between the left atrium (LA) and the left ventricle (LV). In Latin, the term mitral means shaped like a miter, or bishop's cap. The bicuspid or mitral valve is attached to the circumference of the left atrioventricular orifice in the same way that the tricuspid valve is on the opposite side. It consists of two triangular cusps, formed by duplicatures of the lining membrane, strengthened by fibrous tissue, and containing a few muscular fibers. The mitral valve and the tricuspid valve are known collectivly as the atrioventricular valves because they lie between the atria and the ventricles of the heart and control flow. The inelastic chordae tendineae are attached at one end to the papillary muscles and the other to the valve cusps. Papillary muscles are finger like projections from the wall of the left ventricle. Chordae tendinae from each muscle are attached to both leaflets of the mitral valve. Thus when the ventricle contracts, the intraventricular pressure forces the valve to close, while the tendons prevent the valve from opening in the wrong direction. The cusps are of unequal size, and are larger, thicker, and stronger than those of the tricuspid valve. The larger cusp is placed in front and to the right between the atrioventricular and aortic orifices, and is

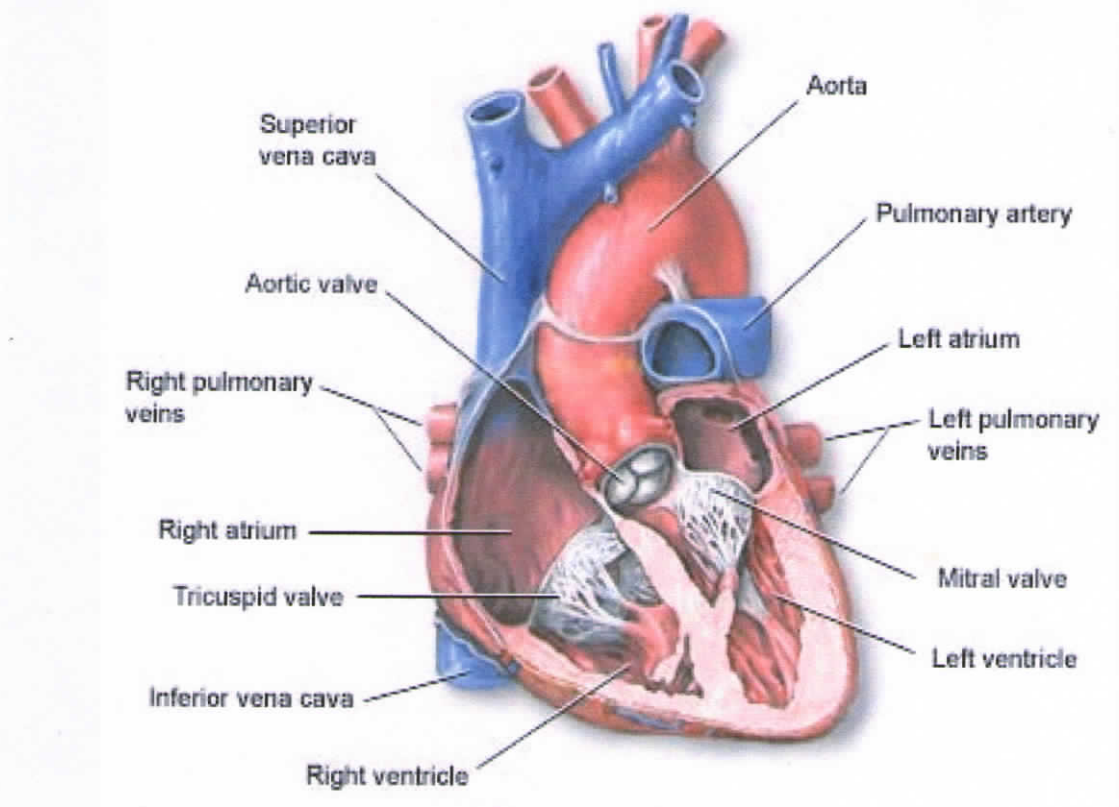
known as the anterior or aortic cusp; the smaller or posterior cusp is placed behind ar to the left of the opening.

Aortic valve

The **Aortic Valve** is one of the valves of the heart. It lies between the le ventricle and the aorta. The aortic valve has three cusps. Two are anterior (right and lef and one posterior. These cusps are half moon shaped hence also called aortic semiluna valve. Each cusp has a small swelling in the center called the nodule. Dilatation of th wall of the aorta opposite these cusps is called aortic sinus. When the aortic valve open, the normal size of the orifice is 3-4 cm^2. During ventricular systole, pressure rise in the left ventricle. When the pressure in the left ventricle rises above the pressure i the aorta, the aortic valve opens, allowing blood to exit the left ventricle into the aorta When ventricular systole ends, pressure in the left ventricle rapidly drops. When th pressure in the left ventricle decreases, the aortic pressure forces the aortic valve t close. The aortic sinuses help in closure of the valve by creating backflow eddies.

Cardiac vessels

The heart consists of mainly five great vessels. Inferior and superior vena cav (great veins), 4 pulmonary veins, aorta and pulmonary artery (great arteries).



Inferior vena cava

The **inferior vena cava** (IVC) is the large vein that carries deoxygenated blood from the lower half of the body into the heart. It is formed by the joining of the left and right common iliac veins and brings blood into the right atrium of the heart. It also anastomoses with the azygos vein system (which runs on the right side of the vertebral column) and the venous plexuses next to the spinal cord. The IVC is posterior to the abdominal cavity and runs along side of the vertebral column on its right side (i.e. it is a retroperitoneal structure).

Superior vena cava

The **superior vena cava** (SVC) is a large but short vein that carries de-oxygenated blood from the upper half of the body to the heart's right atrium. It is formed by the left and right brachiocephalic veins (also referred to as the innominate veins) which receive blood from the upper limbs and the head and neck. The azygous vein (which receives blood from the ribcage) joins it just before it enters the right atrium.

Aorta

The **aorta** is the largest artery in the human body, originating from the left ventricle of the heart and bringing oxygenated blood to all parts of the body in the systemic circulation. The aorta is usually divided into three segments/section.

- Ascending aorta — the section between the heart and the arch of aorta
- Arch of aorta — the peak part that looks like an arch
- Descending aorta — the section between the arch of aorta to the point where it bifurcates into the common iliac arteries
    - Thoracic aorta — the half of the descending aorta above the diaphragm
    - Abdominal aorta — the half ot the descending aorta below the diaphragm

The aorta is an elastic artery, and as such is quite distensible. When the left ventricle contracts to force blood into the aorta, the aorta expands. This stretching gives the potential energy that will help maintain blood pressure during diastole, as during this time the aorta contracts passively.

Pulmonary artery

The **pulmonary arteries** carry blood from the heart to the lungs. They are the only arteries (other than umbilical arteries in the fetus) that carry deoxygenated blood.

In the human heart, the **pulmonary trunk** (**pulmonary artery** or **main pulmonary artery**) begins at the base of the right ventricle. It is short and wide - approximately 5 cm (2 inches) in length and 3 cm (1.2 inches) in diameter. It then branches into two pulmonary arteries (left and right), which deliver deoxygenated blood to the corresponding lung.

Pulmonary veins

The **pulmonary veins** carry oxygen rich blood from the lungs to the left atrium of the heart. They are the only veins in the post-fetal human body that carry oxygenated blood.The pulmonary veins return the arterialized blood from the lungs to the left atrium of the heart. They are four in number, two from each lung, and are destitute of valves. They are:

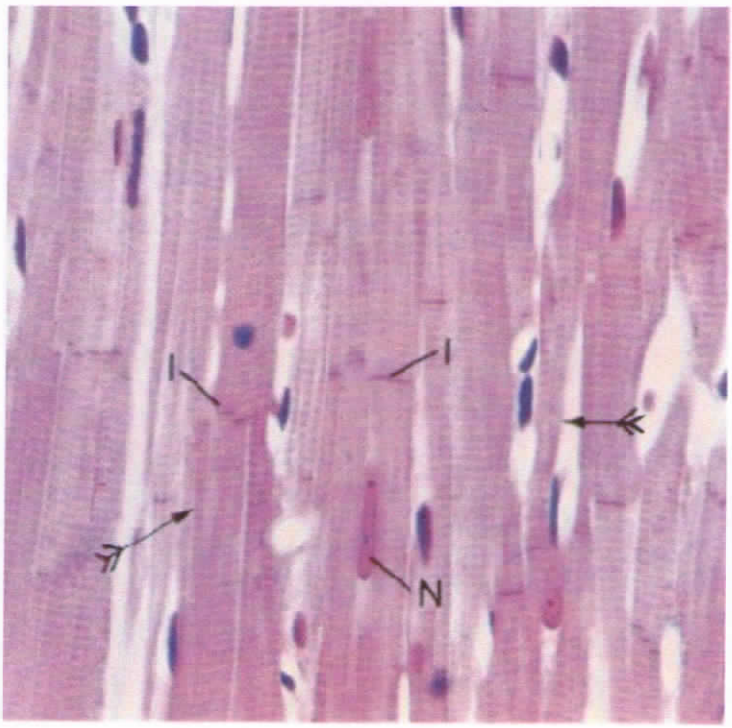
- right inferior
- right superior
- left inferior
- left superior

They commence in a capillary net-work upon the walls of the air sacs, where they are continuous with the capillary ramifications of the pulmonary artery, and, joining together, form one vessel for each lobule.These vessels uniting successively, form a single trunk for each lobe, three for the right, and two for the left lung.The vein from the middle lobe of the right lung generally unites with that from the upper lobe, so that ultimately two trunks from each lung are formed; they perforate the fibrous layer of the pericardium and open separately into the upper and back part of the left atrium.Occasionally the three veins on the right side remain separate.Not infrequently the two left pulmonary veins end by a common opening.At the root of the lung, the superior pulmonary vein lies in front of and a little below the pulmonary artery; the inferior is situated at the lowest part of the hilus of the lung and on a plane posterior to the upper vein. Behind the pulmonary artery is the bronchus.Within the pericardium, their anterior surfaces are invested by the serous layer of this membrane.The right pulmonary veins pass behind the right atrium and superior vena cava; the left in front of the descending thoracic aorta.

Cardiac muscle

Cardiac muscle is a type of involuntary mononucleated, or uninucleated, striated muscle found exclusivly within the heart. Its function is to "pump" blood through the circulatory system by contracting.Unlike skeletal muscle, which contracts in response to nerve stimulation, and like smooth muscle, cardiac muscle is myogenic, meaning that it stimulates its own contraction without a requisite electrical impulse coming from the central nervous system. A single cardiac muscle cell, if left without input, will contract rhythmically at a steady rate; if two cardiac muscle cells are in contact, whichever one

contracts first will stimulate the other to contract, and so on. This transmission impulses makes cardiac muscle tissue similar to nerve tissue, although the cells a connected by intercalated discs, which conduct electrical potentials directly, rather th the chemical synapses used by neurons.Specialized pacemaker cells in the sinoatr node normally determine the overall rate of contractions, with an average resting pu of 72 beats per minute. The central nervous system does not directly create the impuls to contact the heart, but only sends signals to speed up or slow down the heart ra through the autonomic nervous system using two modes: (1) sympathetic nervo system (stress) and (2) parasympathetic nervous system (restful) modulation, rath than controlling each beat. Since cardiac muscle is myogenic, the pacemaker serves o to modulate the cells; the cardiac muscles would still fire in the absence of a pacemak albeit randomly, and the heart would go into fibrillation.Cardiac muscle exhibits cro striations formed by alternation segments of thick and thin protein filaments which a anchored by segments called Z-lines. The primary structural proteins are actin a myosin. The actin filaments are thin causing the lighter band appearance in musc while myosin is thicker and darker lending a darker appearance to the alternating ban in cardiac muscle as observed by a light enhanced microscope. A unique aspect cardiac muscle is the number of nuclei found inside the cell. Skeletal muscle cells a multinucleated from the fusion of muscle cells and smooth muscle cells are stric mononucleated, and cardiac muscle cells are mononucleated in humans.



Cardiac Muscle
I = Intercalated discs
N = Nuclei
Arrows = Cross trabeculae

Epicardium

Epicardium describes the outer layer of heart tissue (from Greek; *epi-* outer, *cardium* heart). When considered as a part of the pericardium, it is the inner layer, or *visceral pericardium*.

Its largest constituent is connective tissue and functions as a protective layer. The visceral pericardium apparently produces the pericardial fluid, which lubricates motion between the inner and outer layers of the pericardium.During ventricular contraction, the wave of depolarization moves from endocardial to epicardial surface.

Endocardium

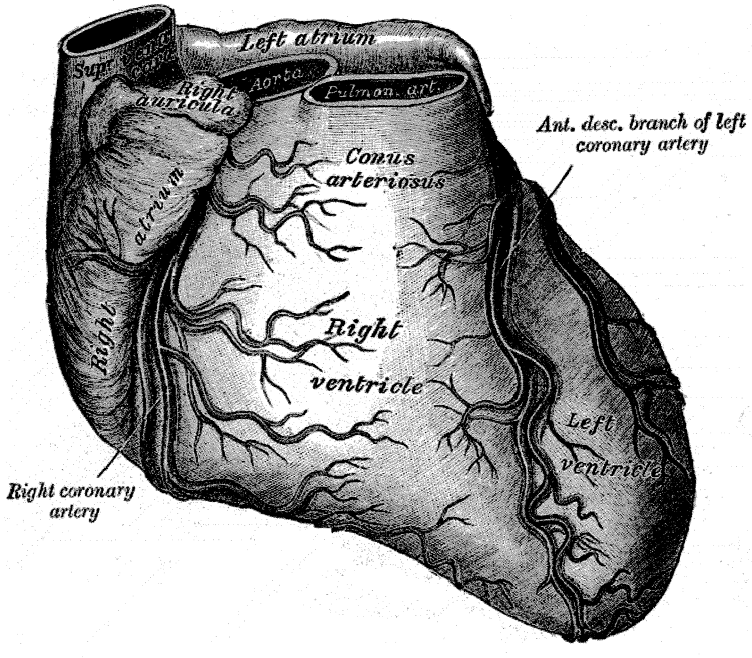
In the heart, the **endocardium** is the innermost layer of cells, embryologically and biologically similar to the endothelium that lines blood vessels.The *endocardium* overlies the much more voluminous myocardium, the muscular tissue responsible for the contraction of the heart. The outer layer of the heart is termed epicardium and the heart is surrounded by a small amount of fluid enclosed by a fibrous sac called the pericardium.Recently, it has become evident that the endocardium, which is primarily made up of endothelial cells, controls myocardial function. This modulating role is separate from the homeometric and heterometric regulatory mechanisms that control myocardial contractility. Moreover, the endothelium of the myocardial capillaries, which is also closely appositioned to the cardiomyocytes is involved in this modulatory role. Thus, the cardiac endothelium (endocardial endothelium and the endothelium of the myocardial capillaries) controls the development of the heart in the embryo, as well as in the adult, i.e. during hypertrophy. Additionally, the contractility and electrophysiological environment of the cardiomyocyte are regulated by the cardiac endothelium. The endocardial endothelium may also act as a kind of blood-heart barrier (analogous to the blood-brain barrier), thus controlling the ionic composition of the extracellular fluid in which the cardiomyocytes bathe.

Myocardium

Myocardium is the muscular tissue of the heart. Other tissues are the endocardium (inner lining, effectively a specialised endothelium) and the pericardium (a connective tissue layer around the heart).The myocardium is composed of specialized cardiac muscle cells with an ability not possessed by muscle tissue elsewhere in the body. Cardiac muscle, like other muscles, can contract, but it can also conduct electricity, like nerves.The blood supply of the myocardium is by the coronary arteries. If these arteries are occluded by atherosclerosis and/or thrombosis, this can lead to angina pectoris or myocardial infarction.Certain viruses lead to inflammation of the myocardium, or myocarditis.Failure of the heart to contract properly (for various reasons) is termed heart failure, generally leading to fluid retention, edema, pulmonary oedema, renal insufficiency, hepatomegaly, a shortened life expectancy and decreased quality of life.

CORONARY ARTERIES

The oxygen and nutrients for myocardium are supplied by the right and left coronary arteries, originating from the aortic root.



Left main coronary artery (LCA)

The left main coronary artery passes between the main pulmonary artery and the left atrial appendage. The left main coronary artery is a short trunk arising from the left aortic sinus running from its ostium through the aortic wall to left and anteriorly in the anterior atrio ventricular sulcus . The Left main trunk varies considerably in size considerably ranging from a few millimeters to 4 cm. As a rule the Left main coronary artery bifurcates into the left anterior descending and the left circumflex. On occasion it may trifurcates into an LAD an LCX and a Ramus inter mediuns.

Left Anterior Descending Artery (LAD)

The left anterior descending is usually a direct continuation of the left main coronary artery. It runs in the anterior interventricular groove and thus varies in position depending upon the presence of right or left ventricular hypertrophy. At sometimes it may leave the interventricular sulcus and cross over the ventricle. The LAD is usually

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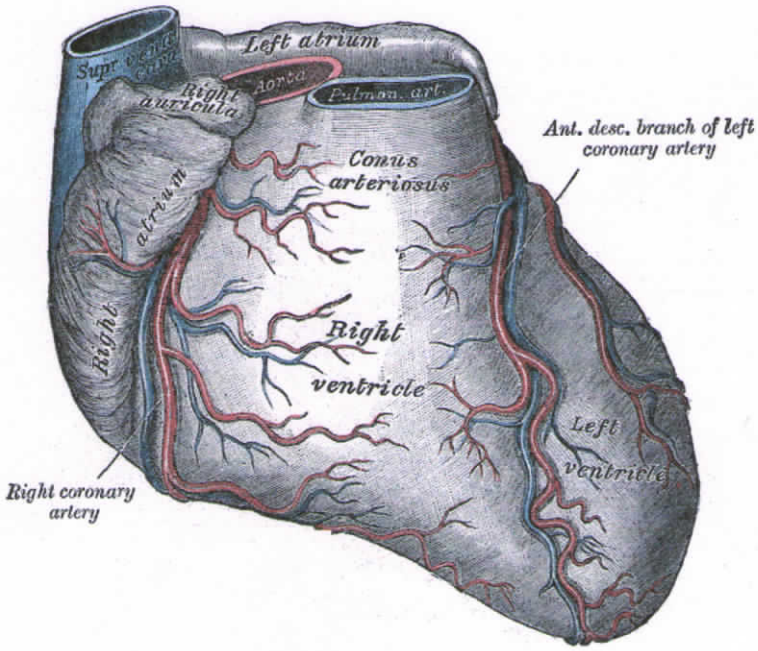

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classified into three types in accordance with its approach towards the apex. If the LAD is not reaching up to the apex then it is called a Type I LAD .If it is reaching up to the apex it is then called a Type II LAD, and if it reaches the apex and continues as the posterior ascending artery it is called Type III LAD.

The most important branches of the left anterior descending arteries are branches to the inter ventricular septum, which are called the septal branches. These anterior septal branches anastamose with the posterior septal branches arising from the posterior descending artery. The diagonal branches arise at oblique angles to the LAD to descend diagonally towards the obtuse margin of the heart. The diagonal branches vary in number from 1 to 5 then we name them as D1,D2....D5. They are supplying the anterior wall of the LV.

Left Circumflex Artery (LCX)

The vessel is a branch of Left main coronary artery. It may vary considerably in size depending upon whether the posterior descending artery is a branch of the right coronary or the circumflex coronary itself. In some individuals with strongly dominant right coronary artery the circumflex will be too small and may be absent. The circumflex coronary artery measures generally 2mm to 4mm in external diameter. It runs through the left atrioventricular groove then turns down and goes to the postero-lateral part of the LV. Running parallel to it and superficial or anterior to it is the great cardiac vein and it unites with the large atrial vein to the origin of the coronary sinus. If we are running the venous phase of the angio the coronary sinus just fills exactly over where the circumflex is.

The first branch arising from the circumflex artery is in 40% of the people the sinus node artery, which supplies the SA node –the pacemaker of the heart. Kugel's artery may also arise from the proximal portion of the circumflex, this vessel forms an important collateral pathway when the right or left coronary arteries are occluded and supply of blood to the posterior left ventricle. Kugel's artery is a vessel which runs in the inter atrial septum.

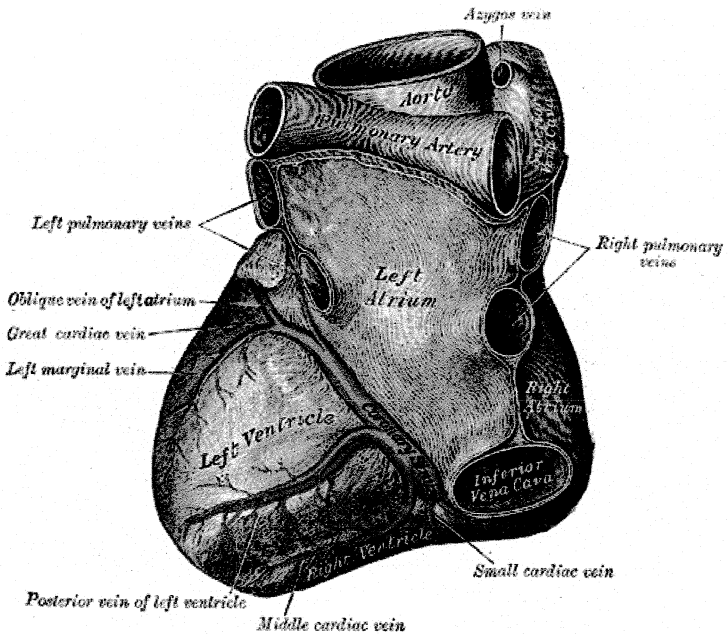
The next major artery arising from the circumflex is the left atria circumflex artery. In case of dominant right coronary artery the LA circumflex may be the only artery arising from the LCX. It supplies the lower portion of the left atrium. Marginal branches are important branches arising from the circumflex, they course towards the obtuse margin of the heart and then curve towards the apex, the so called artery is the Obtuse marginal artery which supplies the lateral wall of the left ventricle. Obtuse margin may be one or more so we name them as OM1, OM2...

Right Coronary Artery (RCA)

The Right coronary artery arises from the right aortic sinus. It runs through the right atrioventricular groove turns posteriorly and continues through the posterior interventricular branches. Its main branches are in 60% SA nodal artery arises form RCA, which supplies the sinus node. Next is a Conus artery arising from RCA but it is only in 35% in 65% of people the onus artery has separate origin. The Conus artery supplies the RVOT. Next the RCA turns in the acute margin of the heart from there the next artery called the Acute margin arises, which supplies the Right ventricular free wall. On the posterior side the RCA continues through posterior interventricular groove, at the crux of the heart it gives the next branch called the AV nodal branch, which supplies the AV node. Now the RCA continues as the posterior descending artery (PDA) and give several posterior septal branches, which supplies the posterior part of the septum. Next branches are the posterior left ventricular branches (PLVB) which supplies the posterior part of the left ventricle. RCA is said to be a Dominant vessel if it continues as PDA and gives rise to posterior left ventricular branches.

CARDIAC VEINS

The principal cardiac veins are greater and middle cardiac veins; posterior left ventricular vein enters the right atrium via the coronary sinus.



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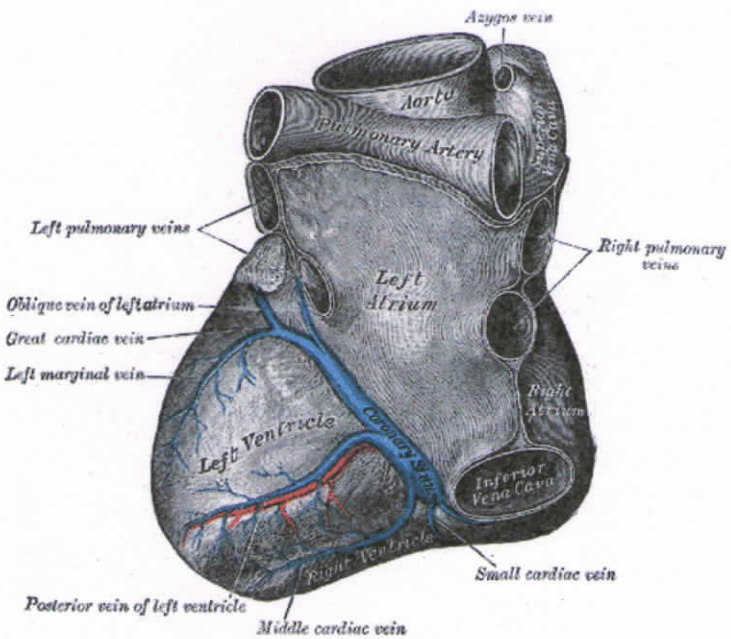


Great Cardiac Vein

The **Great Cardiac Vein** (left coronary vein) begins at the apex of the heart and ascends along the anterior longitudinal sulcus to the base of the ventricles. It then curves to the left in the coronary sulcus, and reaching the back of the heart, opens into the left extremity of the coronary sinus. It receives tributaries from the left atrium and from both ventricles: one, the **left marginal vein**, is of considerable size, and ascends along the left margin of the heart.

Small Cardiac Vein

The **Small Cardiac Vein** (right coronary vein) runs in the coronary sulcus between the right atrium and ventricle, and opens into the right extremity of the coronary sinus. It receives blood from the back of the right atrium and ventricle; the **right marginal vein** ascends along the right margin of the heart and joins it in the coronary sulcus, or opens directly into the right atrium.

Middle Cardiac Vein

The **Middle Cardiac Vein** commences at the apex of the heart, ascends in the posterior longitudinal sulcus, and ends in the coronary sinus near its right extremity

The **Posterior Vein of the Left Ventricle** runs on the diaphragmatic surface of the left ventricle to the coronary sinus, but may end in the great cardiac vein

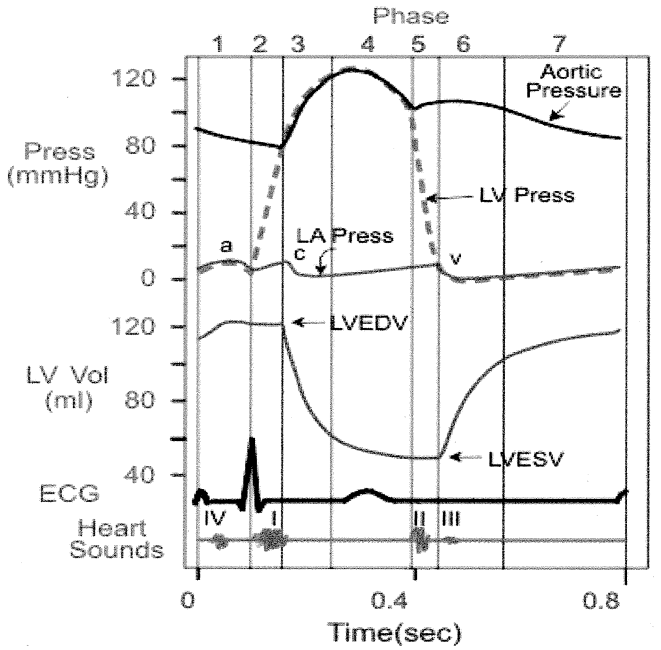
The **Oblique Vein of the Left Atrium** (oblique vein of Marshall) is a small vessel which descends obliquely on the back of the left atrium and ends in the coronary sinus near its left extremity; it is continuous above with the **ligament of the left vena cava** (lig. venae cavae sinistrae vestigial fold of Marshall), and the two structures form the remnant of the left Cuvierian duct.

The Cardiac Cycle

The proper functioning of the cardiac conduction system, with the consequent coordination of contraction and valve opening and closing in each region of the heart, is critical for efficient pumping of blood. Each phase of the cardiac cycle is described below:

- An impulse arising from the SA node results in depolarization and contraction of the atria (the right atrium contracts slightly before the left atrium)
- The atrioventricular valves open and the ventricles are filled with blood
- There is a short delay of the electrical impulse in the AV junction that allows the ventricles to fill completely

- The electrical impulse is then propagated through the His bundle and Purkinje system to allow the ventricles to contract from the apex of the heart towards the base
- As the ventricles contract and the pressure in the ventricles exceeds that in the atria, the atrioventricular valves close and the atria begin to relax and refill with blood
- When the pressure in the ventricles exceed the pressure in the pulmonary artery and aorta, the pulmonic and aortic valves open, and blood is pumped into the pulmonary and systemic circulations, respectively
- As the ventricles begin to relax after systole, the pulmonic and aortic valves close and diastole begins



Abbreviations:
LV Press, left ventricular pressure
a, a-wave; c, c-wave; v, v-wave
ECG, electrocardiogram
LVEDV, left ventricular end-diastolic volume
LVESV, left ventricular end-systolic volume

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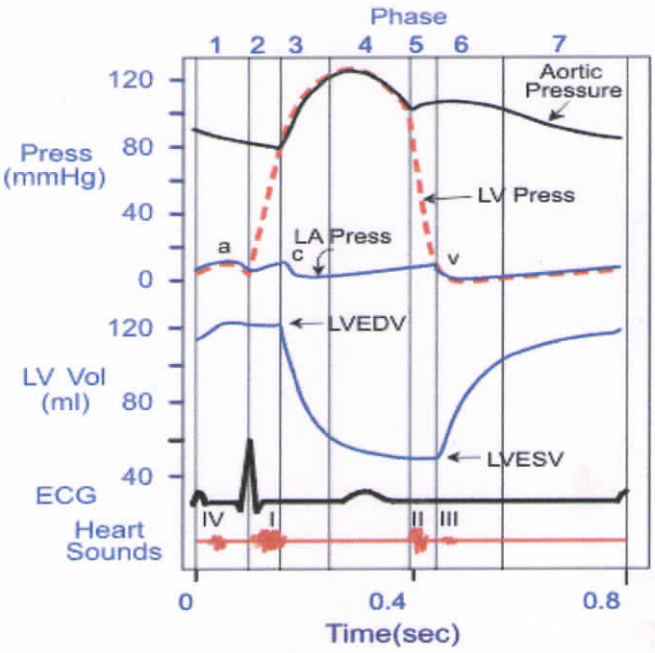

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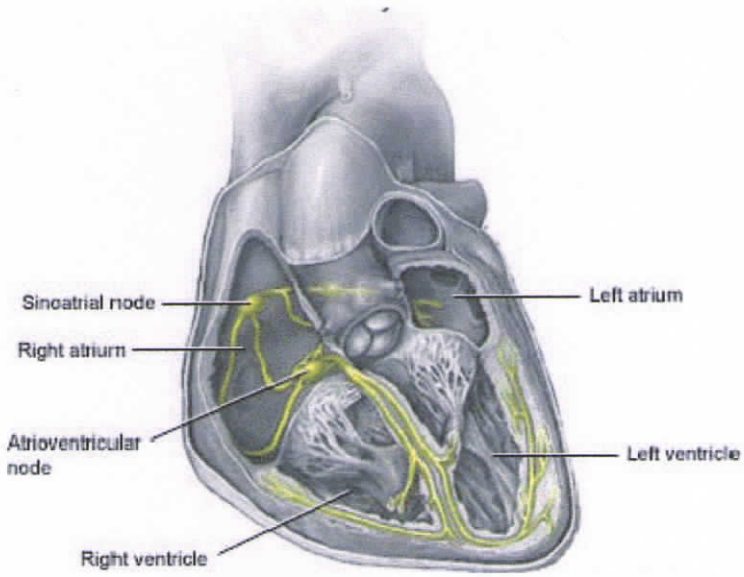
Cardiac Conduction System



Normally the signal for cardiac stimulation starts in the sinus node, also called t sinoatrial (SA) node. The node is located in the right atrium near the opening of t superior vena cava. It is a small collection of specialized cells capable of automatica generating an electrical signal. From the sinus node this stimulus spreads first throu the right atrium and then into left atrium. Thus sinus node functions as the pacemaker the heart.

The first phase of cardiac muscle activation is electrical stimulation of right a left atria. This in turn signals the atria to contract and pump blood simultaneou through tricuspid and mitral valves into the right and left ventricles. The electri stimulus then spreads to specialized conduction tissues in the atrioventricular A junction, which includes the AV node and bundle of His, and then into left and rig bundle branches, which transmits the stimulus to the ventricular muscle cells.

The AV junction, which acts as an electrical bridge connecting atria and ventricle is located at the base of the inter atrial septum and extends into the inter ventricu septum. The upper part of the AV junction is the AV node. The lower part of the A junction is called the bundle of His after the physiologist who described it. The bundle His the divides into two main branches: the right bundle branch, which distributes t stimulus to the right ventricle and the left bundle branch, which distributes the stimul to the left ventricle. The electrical signals spreads simultaneously down the left and rig bundle branches into the ventricular myocardium by way of specialized conducting ce

called Purkinje fibers, which are located in the ventricular myocardium. The spread of stimuli through the ventricles leads to ventricular contraction, with pumping of blood to the lungs and into the general circulation.

Cardiac Output

Cardiac output is the volume of blood pumped by the heart per minute. It is determined by multiplying the stroke volume (the amount of blood ejected in each contraction) by heart rate. If 70 ml of blood is the stroke volume and 80 is the heart rate 5.6 L/min is the cardiac output.

Determinants of Cardiac output

Depending on the needs of the body, cardiac output is increased or decreased by four interrelated factors: heart rate, preload, after load, and contractility.

Heart Rate

Heart rate refers to the frequency of the contraction, reflected by the pulse rate. Although sinus node usually determines the heart rate, neural and factors also play in determining the heart rate. A change in the heart rate will alter the following three factors

Preload

Preload is the amount of blood in the ventricle before it contracts. It is also known as the left ventricular end diastolic volume (LVEDV). The greater the preload within the physiologic limits, the more the myocardial fibers stretch and subsequently the more forcefully the fibres shorten to produce an increase in stroke volume. This relation is known as Frank-Starling law.

Afterload

Afterload is the resistance that ventricles must overcome during systole. Aortic distensibility, the systemic vascular bed, the blood volume and vascular bed create resistance. Left or right ventricular outflow obstructions also increase the workload of the heart.

Contractility

Contractility refers to the inotropic state of ventricle, independent of changes in the heart rate, preload, or afterload. Sympathetic stimulation or inotropic agents such as digitalis, calcium etc increases contractility and cardiac output.

Heart Sounds

The first heart tone, or **S_1**, is caused by the closure of the atrioventricular valves, mitral and tricuspid, at the beginning of ventricular contraction, or systole. When the pressure in the ventricles rises above the pressure in the atria, these valves close to prevent regurgitation of blood from the ventricles into the atria.

The second heart tone, or **S_2**, is caused by the closure of the aortic and pulmonic valves at the end of ventricular systole. As the left ventricle empties, its pressure falls below the pressure in the aorta, and the aortic valve closes. Similarly, as the pressure in the right ventricle falls below the pressure in the pulmonary artery, the pulmonic valve closes.

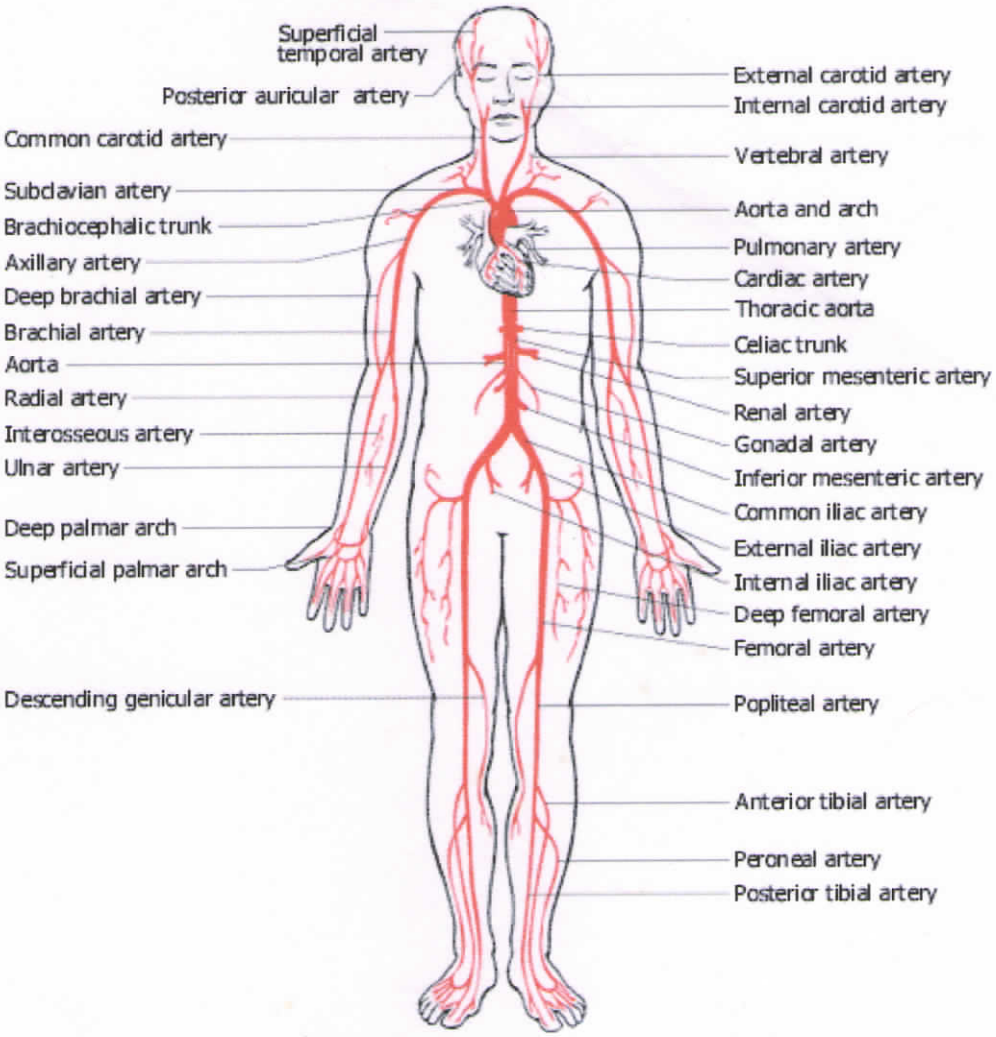
Due to the higher pressure in the aorta compared to the pulmonary artery, the aortic valve normally closes before the pulmonic valve, so the second heart tone may have an audible split. A split S_2 is made up of an A_2 and a P_2 components, caused by the closure of the aortic and pulmonic valves, respectively.

The splitting of the second heart tone varies with the phases of respiration. During inspiration, negative intrathoracic pressure causes increased blood return into the right side of the heart. The increased blood volume in the right ventricle causes the pulmonic valve to stay open longer during ventricular systole. This causes an increased delay in the P_2 component of S_2. During expiration, the positive intrathoracic pressure causes decreased blood return to the right side of the heart. The reduced volume in the right ventricle allows the pulmonic valve to close earlier at the end of ventricular systole, causing P_2 to occur earlier, and "closer" to A_2. It is physiological to hear the splitting of the second heart tone by younger people and during inspiration. During expiration normally the interval between the two components shortens and the tone becomes merged.

Heart Murmurs

Murmurs are produced by any condition which produces noisy, or turbulent, flow of blood. This most commonly results from narrowing or leaking of valves or the presence of abnormal passages through which blood flows in or near the heart. Without their cause being known, murmurs can still be designated according to the part of the cardiac cycle in which they are heard, as systolic or diastolic. More accurately we can divide phases of the cardiac cycle into three parts: proto-, meso- and tele- systole or diastole. For telediastole we usually use the term presystole. Murmurs are also classified by their shape (crescendo, decrescendo, crescendo-decrescendo), loudness and frequency range (high-pitched, low-pitched), point of maximum volume on the chest wall and spreading in different directions (how does the murmur loudness change in different directions).

THE ARTERIAL SYSTEM



THE AORTA

The aorta is the main trunk of a series of vessels which convey the oxygenat blood to the tissues of the body for their nutrition. It commences at the upper part of t left ventricle, where it is about 3 cm. in diameter, and after ascending for a sh distance, arches backward and to the left side, over the root of the left lung; it th descends within the thorax on the left side of the vertebral column, passes into t abdominal cavity through the aortic hiatus in the diaphragm, and ends, considera diminished in size (about 1.75 cm. in diameter), opposite the lower border of the fou lumbar vertebra, by dividing into the right and left common iliac arteries. Hence it described in several portions, viz., the ascending aorta, the arch of the aorta, and t descending aorta, which last is again divided into the thoracic and abdominal aorta.

The Ascending Aorta

The ascending aorta is about 5 cm. in length. It commences at the upper part of the base of the left ventricle, on a level with the lower border of the third costal cartilage behind the left half of the sternum; it passes obliquely upward, forward, and to the right, in the direction of the heart's axis, as high as the upper border of the second right costal cartilage, describing a slight curve in its course, and being situated, about 6 cm. behind the posterior surface of the sternum. At its origin it presents, opposite the segments of the aortic valve, three small dilatations called the aortic sinuses.

The only branches of the ascending aorta are the two coronary arteries which supply the heart; they arise near the commencement of the aorta immediately above the attached margins of the semilunar valves.

The Arch of the Aorta

The arch of the aorta begins at the level of the upper border of the second sternocostal articulation of the right side, and runs at first upward, backward, and to the left in front of the trachea; it is then directed backward on the left side of the trachea and finally passes downward on the left side of the body of the fourth thoracic vertebra, at the lower border of which it becomes continuous with the descending aorta. It thus forms two curvatures: one with its convexity upward, the other with its convexity forward and to the left. Its upper border is usually about 2.5 cm.

The branches given off from the arch of the aorta are three in number: the innominate, the left common carotid, and the left subclavian.

The Innominate Artery (Brachiocephalic Artery)

The innominate artery is the largest branch of the arch of the aorta, and is from 4 to 5 cm. in length. It arises, on a level with the upper border of the second right costal cartilage, from the commencement of the arch of the aorta, on a plane anterior to the origin of the left carotid; it ascends obliquely upward, backward, and to the right to the level of the upper border of the right sternoclavicular articulation, where it divides into the right common carotid and right subclavian arteries.

The Common Carotid Artery

The principal arteries of supply to the head and neck are the two common carotids; they ascend in the neck and each divides into two branches, viz., (1) the external carotid, supplying the exterior of the head, the face, and the greater part of the neck; (2) the internal carotid, supplying to a great extent the parts within the cranial and orbital cavities.

The common carotid arteries differ in length and in their mode of origin. The right begins at the bifurcation of the innominate artery behind the sternoclavicular joint and is confined to the neck. The left springs from the highest part of the arch of the aorta to the left of, and on a plane posterior to the innominate artery, and therefore consists of a thoracic and a cervical portion. The thoracic portion of the left common carotid artery ascends from the arch of the aorta through the superior mediastinum to the level of the left sternoclavicular joint, where it is continuous with the cervical portion.

The external carotid artery begins opposite the upper border of the thyroid cartilage, and, taking a slightly curved course, passes upward and forward, and then inclines backward to the space behind the neck of the mandible, where it divides into the superficial temporal and internal maxillary arteries. It rapidly diminishes in size in its course up the neck, owing to the number and large size of the branches given off from it. In the child, it is somewhat smaller than the internal carotid; but in the adult, the two vessels are of nearly equal size. At its origin, this artery is more superficial, and placed nearer the middle line than the internal carotid, and is contained within the carotid triangle.

The internal carotid artery supplies the anterior part of the brain, the eye and its appendages, and sends branches to the forehead and nose. Its size, in the adult, is equal to that of the external carotid, though, in the child, it is larger than that vessel. It is remarkable for the number of curvatures that it presents in different parts of its course. It occasionally has one or two flexures near the base of the skull, while in its passage through the carotid canal and along the side of the body of the sphenoid bone it describes a double curvature and resembles the italic letter *S*.

The Subclavian Artery

The artery which supplies the upper extremity continues as a single trunk from its commencement down to the elbow; but different portions of it have received different names, according to the regions through which they pass. That part of the vessel which extends from its origin to the outer border of the first rib is termed the subclavian; beyond this point to the lower border of the axilla it is named the axillary; and from the lower margin of the axillary space to the bend of the elbow it is termed brachial; here the trunk ends by dividing into two branches the radial and ulnar.

On the right side the subclavian artery arises from the innominate artery behind the right sternoclavicular articulation; on the left side it springs from the arch of the aorta. The two vessels, therefore, in the first part of their course, differ in length, direction, and relation with neighboring structures. The first part of the right subclavian artery arises from the innominate artery, behind the upper part of the right sternoclavicular articulation, and passes upward and lateralward to the medial margin of the Scalenus anterior. It ascends a little above the clavicle, the extent to which it does so varying in different cases.

The first part of the left subclavian artery arises from the arch of the aorta, behind the left common carotid, and at the level of the fourth thoracic vertebra; it ascends in the superior mediastinal cavity to the root of the neck and then arches lateralward to the medial border of the Scalenus anterior. The second portion of the subclavian artery lies behind the Scalenus anterior; it is very short, and forms the highest part of the arch described by the vessel. The branches of the subclavian artery are: Vertebral, Internal mammary artery.

The axillary artery the continuation of the subclavian, commences at the outer border of the first rib, and ends at the lower border of the tendon of the Teres major, where it takes the name of brachial. Its direction varies with the position of the limb; thus the vessel is nearly straight when the arm is directed at right angles with the trunk, concave upward when the arm is elevated above this, and convex upward and lateralward when the arm lies by the side. At its origin the artery is very deeply situated, but near its termination is superficial, being covered only by the skin and fascia. To facilitate the description of the vessel it is divided into three portions; the first part lies above, the second behind, and the third below the Pectoralis minor.

The brachial artery commences at the lower margin of the tendon of the Teres major, and, passing down the arm, ends about 1 cm. below the bend of the elbow, where it divides into the radial and ulnar arteries. At first the brachial artery lies medial to the humerus; but as it runs down the arm it gradually gets in front of the bone, and at the bend of the elbow it lies midway between its two epicondyles.

The radial artery appears, from its direction, to be the continuation of the brachial, but it is smaller in caliber than the ulnar. It commences at the bifurcation of the brachial, just below the bend of the elbow, and passes along the radial side of the forearm to the wrist. It then winds backward, around the lateral side of the carpus, beneath the tendons of the Abductor pollicis longus and Extensores pollicis longus and brevis to the upper end of the space between the metacarpal bones of the thumb and index finger. Finally it passes forward between the two heads of the first Interosseous dorsalis, into the palm of the hand, where it crosses the metacarpal bones and at the ulnar side of the hand unites with the deep volar branch of the ulnar artery to form the deep volar arch. The radial artery therefore consists of three portions, one in the forearm, a second at the back of the wrist, and a third in the hand.

The ulnar artery the larger of the two terminal branches of the brachial, begins a little below the bend of the elbow, and, passing obliquely downward, reaches the ulnar side of the forearm at a point about midway between the elbow and the wrist. It then runs along the ulnar border to the wrist, crosses the transverse carpal ligament on the radial side of the pisiform bone, and immediately beyond this bone divides into two branches, which enter into the formation of the superficial and deep volar arches.

The Descending Aorta

The descending aorta is divided into two portions, the thoracic and abdominal, in correspondence with the two great cavities of the trunk in which it is situated

The Thoracic Aorta

The thoracic aorta is contained in the posterior mediastinal cavity. It begins at the lower border of the fourth thoracic vertebra where it is continuous with the aortic arch, and ends in front of the lower border of the twelfth at the aortic hiatus in the diaphragm. At its commencement, it is situated on the left of the vertebral column; it approaches the median line as it descends; and, at its termination, lies directly in front of the column. The vessel describes a curve which is concave forward, and as the branches given off from it are small, its diminution in size is inconsiderable. Branches of the Thoracic Aorta: (1) Visceral: Pericardial, Bronchial, Esophageal, Mediastinal. (2) Parietal: Intercostal, Subcostal, Superior Phrenic.

The Abdominal Aorta

The abdominal aorta begins at the aortic hiatus of the diaphragm, in front of the lower border of the body of the last thoracic vertebra, and, descending in front of the vertebral column, ends on the body of the fourth lumbar vertebra, commonly a little to the left of the middle line, by dividing into the two common iliac arteries. It diminishes rapidly in size, in consequence of the many large branches which it gives off. As it lies upon the bodies of the vertebræ, the curve which it describes is convex forward, the summit of the convexity corresponding to the third lumbar vertebra. The branches of the abdominal aorta may be divided into three sets: visceral, parietal, and terminal. Visceral Branches: Celiac, Superior Mesenteric, Inferior Mesenteric, Middle Suprarenals, Renals, Internal Spermatics, Ovarian (in the female). Parietal Branches: Inferior Phrenics, Lumbars, Middle Sacral. Terminal Branches: Common Iliacs.

The Common Iliac Arteries

The abdominal aorta divides, on the left side of the body of the fourth lumbar vertebra, into the two common iliac arteries. Each is about 5 cm. in length. They diverge from the termination of the aorta, pass downward and lateralward, and divide, opposite the intervertebral fibrocartilage between the last lumbar vertebra and the sacrum, into two branches, the external iliac and hypogastric arteries; the former supplies the lower extremity; the latter, the viscera and parietes of the pelvis.

The right common iliac artery is somewhat longer than the left, and passes more obliquely across the body of the last lumbar vertebra. The left common iliac artery is in relation, crossed at its point of bifurcation by the ureter. It rests on the bodies of the fourth and fifth lumbar vertebrae, and the intervening fibrocartilage.

The External Iliac Artery

The external iliac artery is larger than the hypogastric, and passes obliquely downward and lateralward along the medial border of the Psoas major, from the bifurcation of the common iliac to a point beneath the inguinal ligament, midway between the anterior superior spine of the ilium and the symphysis pubis, where it enters the thigh and becomes the femoral artery.

The Femoral Artery

The artery which supplies the greater part of the lower extremity is the direct continuation of the external iliac. It runs as a single trunk from the inguinal ligament to the lower border of the Popliteus, where it divides into two branches, the anterior and posterior tibial. The upper part of the main trunk is named the femoral, the lower part the popliteal.

The femoral artery begins immediately behind the inguinal ligament, midway between the anterior superior spine of the ilium and the symphysis pubis, and passes down the front and medial side of the thigh. It ends at the junction of the middle with the lower third of the thigh, where it passes through an opening in the Adductor magnus to become the popliteal artery. The vessel, at the upper part of the thigh, lies in front of the hip-joint; in the lower part of its course it lies to the medial side of the body of the femur, and between these two parts, where it crosses the angle between the head and body, the vessel is some distance from the bone.

The Popliteal Artery

The popliteal artery is the continuation of the femoral, and courses through the popliteal fossa. It extends from the opening in the Adductor magnus, at the junction of the middle and lower thirds of the thigh, downward and lateralward to the intercondyloid fossa of the femur, and then vertically downward to the lower border of the Popliteus, where it divides into anterior and posterior tibial arteries.

The Anterior Tibial Artery

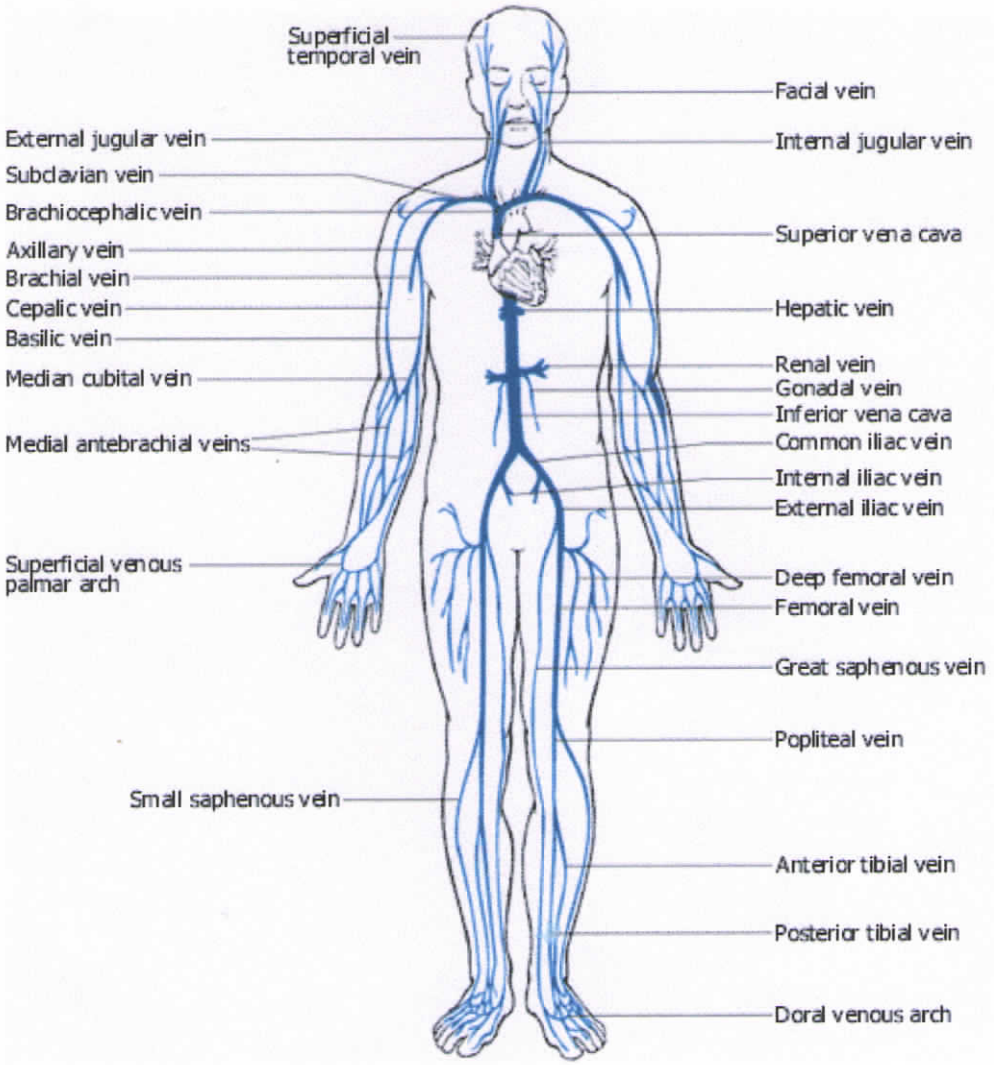
The anterior tibial artery commences at the bifurcation of the popliteal, at the lower border of the Popliteus, passes forward between the two heads of the Tibialis posterior, and through the aperture above the upper border of the interosseous membrane, to the deep part of the front of the leg: it here lies close to the medial side of the neck of the fibula. It then descends on the anterior surface of the interosseous membrane, gradually approaching the tibia; at the lower part of the leg it lies on this bone, and then on the front of the ankle-joint, where it is more superficial, and becomes the dorsalis pedis.

The arteria dorsalis pedis the continuation of the anterior tibial, passes forward from the ankle-joint along the tibial side of the dorsum of the foot to the proximal part of the first intermetatarsal space, where it divides into two branches, the first dorsal metatarsal and the deep plantar.

The Posterior Tibial Artery

The posterior tibial artery begins at the lower border of the Popliteus, opposite the interval between the tibia and fibula; it extends obliquely downward, and, as it descends, it approaches the tibial side of the leg, lying behind the tibia, and in the lower part of its course is situated midway between the medial malleolus and the medial process of the calcaneal tuberosity. Here it divides beneath the origin of the Adductor hallucis into the medial and lateral plantar arteries.

THE VENOUS SYSTEM



The Veins of the Neck

The external jugular vein receives the greater part of the blood from the exter of the cranium and the deep parts of the face, being formed by the junction of t posterior division of the posterior facial with the posterior auricular vein. The exter jugular vein varies in size, bearing an inverse proportion to the other veins of the neck is occasionally double. It is provided with two pairs of valves, the lower pair being plac at its entrance into the subclavian vein, the upper in most cases about 4 cm. above t clavicle. The portion of vein between the two sets of valves is often dilated, and is term the sinus. These valves do not prevent the regurgitation of the blood, or the passage injection from below upward.

The internal jugular vein collects the blood from the brain, from the superficial parts of the face, and from the neck. At its origin it is somewhat dilated, and this dilatation is called the superior bulb. It runs down the side of the neck in a vertical direction, lying at first lateral to the internal carotid artery, and then lateral to the common carotid, and at the root of the neck unites with the subclavian vein to form the innominate vein; a little above its termination is a second dilatation, the inferior bulb. At the root of the neck the right internal jugular vein is placed at a little distance from the common carotid artery, and crosses the first part of the subclavian artery, while the left internal jugular vein usually overlaps the common carotid artery. The left vein is generally smaller than the right, and each contains a pair of valves, which are placed about 2.5 cm. above the termination of the vessel.

The Veins of the Brain

The veins of the brain possess no valves, and their walls, owing to the absence of muscular tissue, are extremely thin. They pierce the arachnoid membrane and the inner or meningeal layer of the dura mater, and open into the cranial venous sinuses. They may be divided into two sets, cerebral and cerebellar. The cerebral veins are divisible into external and internal groups according as they drain the outer surfaces or the inner parts of the hemispheres. The external veins are the superior, inferior, and middle cerebral. The cerebellar veins are placed on the surface of the cerebellum, and are disposed in two sets, superior and inferior.

The Veins of the Upper Extremity

The veins of the upper extremity are divided into two sets, superficial and deep; the two sets anastomose frequently with each other. The superficial veins are placed immediately beneath the integument between the two layers of superficial fascia. The deep veins accompany the arteries, and constitute the venae comitantes of those vessels.

The superficial veins of the upper extremity are the digital, metacarpal, cephalic, basilic, median. The dorsal digital veins pass along the sides of the fingers and are joined to one another by oblique communicating branches. Those from the adjacent sides of the fingers unite to form three dorsal metacarpal veins The cephalic vein begins in the radial part of the dorsal venous net-work and winds upward around the radial border of the forearm, receiving tributaries from both surfaces. Below the front of the elbow it gives off the vena mediana cubiti, which receives a communicating branch from the deep veins of the forearm and passes across to join the basilic vein. The basilic vein begins in the ulnar part of the dorsal venous network. It runs up the posterior surface of the ulnar side of the forearm and inclines forward to the anterior surface below the elbow, where it is joined by the vena mediana cubiti. It then runs upward along the medial border of the Biceps brachii, perforates the deep fascia a little below the middle of the arm, and, ascending on the medial side of the brachial artery to the lower border of the Teres major, is continued onward as the axillary vein.

The deep veins follow the course of the arteries, forming their venae comitantes. They are generally arranged in pairs, and are situated one on either side of the corresponding artery, and connected at intervals by short transverse branches.

The brachial veins are placed one on either side of the brachial artery, receiving tributaries corresponding with the branches given off from that vessel; near the lower margin of the Subscapularis, they join the axillary vein; the medial one frequently joins the basilic vein. These deep veins have numerous anastomoses, not only with each other, but also with the superficial veins.

The axillary vein begins at the lower border of the Teres major, as the continuation of the basilic vein, increases in size as it ascends, and ends at the outer border of the first rib as the subclavian vein. Near the lower border of the Subscapularis it receives the brachial veins and, close to its termination, the cephalic vein; its other tributaries correspond with the branches of the axillary artery. It is provided with a pair of valves opposite the lower border of the Subscapularis; valves are also found at the ends of the cephalic and subscapular veins.

The subclavian vein the continuation of the axillary, extends from the outer border of the first rib to the sternal end of the clavicle, where it unites with the internal jugular to form the innominate vein. It is in relation, in front, with the clavicle and Subclavius; behind and above, with the subclavian artery. It is usually provided with a pair of valves, which are situated about 2.5 cm. from its termination.

The Veins of the Thorax

The innominate veins are two large trunks, placed one on either side of the root of the neck, and formed by the union of the internal jugular and subclavian veins of the corresponding side; they are devoid of valves.

The Right Innominate Vein is a short vessel, about 2.5 cm. in length, which begins behind the sternal end of the clavicle, and, passing almost vertically downward, joins with the left innominate vein just below the cartilage of the first rib, close to the right border of the sternum, to form the superior vena cava. It lies in front and to the right of the innominate artery. This vein, at its commencement, receives the right vertebral vein; and, lower down, the right internal mammary and right inferior thyroid veins, and sometimes the vein from the first intercostal space.

The Left Innominate Vein about 6 cm. in length, begins behind the sternal end of the clavicle and runs obliquely downward and to the right behind the upper half of the manubrium sterni to the sternal end of the first right costal cartilage, where it unites with the right innominate vein to form the superior vena cava.

The Veins of the Lower Extremity, Abdomen, and Pelvis

The veins of the lower extremity are subdivided, like those of the upper, into two sets, superficial and deep; the superficial veins are placed beneath the integument between the two layers of superficial fascia; the deep veins accompany the arteries. Both sets of veins are provided with valves, which are more numerous in the deep than in the superficial set. Valves are also more numerous in the veins of the lower than in those of the upper limb.

The superficial veins of the lower extremity are the great and small saphenous veins and their tributaries. The great saphenous vein the longest vein in the body, *begins* in the medial marginal vein of the dorsum of the foot and ends in the femoral vein about 3 cm. below the inguinal ligament. It ascends in front of the tibial malleolus and along the medial side of the leg in relation with the saphenous nerve. It runs upward behind the medial condyles of the tibia and femur and along the medial side of the thigh and, passing through the fossa ovalis, ends in the femoral vein. The small saphenous vein *begins* behind the lateral malleolus as a continuation of the lateral marginal vein; it first ascends along the lateral margin of the tendocalcaneus, and then crosses it to reach the middle of the back of the leg. Running directly upward, it perforates the deep fascia in the lower part of the popliteal fossa, and ends in the popliteal vein, between the heads of the Gastrocnemius. It communicates with the deep veins on the dorsum of the foot, and receives numerous large tributaries from the back of the leg. Before it pierces the deep fascia, it gives off a branch which runs upward and forward to join the great saphenous vein. The small saphenous vein possesses from nine to twelve valves, one of which is always found near its termination in the popliteal vein. In the lower third of the leg the small saphenous vein is in close relation with the sural nerve, in the upper two-thirds with the medial sural cutaneous nerve.

The deep veins of the lower extremity accompany the arteries and their branches; they possess numerous valves.

The plantar digital veins arise from plexuses on the plantar surfaces of the digits, and, after sending intercapitular veins to join the dorsal digital veins, unite to form four metatarsal veins; these run backward in the metatarsal spaces, communicate, by means of perforating veins, with the veins on the dorsum of the foot, and unite to form the deep plantar venous arch which lies alongside the plantar arterial arch. From the deep plantar venous arch the medial and lateral plantar veins run backward close to the corresponding arteries and, after communicating with the great and small saphenous veins, unite behind the medial malleolus to form the posterior tibial veins. The posterior tibial veins accompany the posterior tibial artery, and are joined by the peroneal veins. The anterior tibial veins are the upward continuation of the venae comitantes of the dorsalis pedis artery. They leave the front of the leg by passing between the tibia and fibula, over the interosseous membrane, and unite with the posterior tibial, to form the popliteal vein.

The Popliteal Vein is formed by the junction of the anterior and posterior tibial veins at the lower border of the Popliteus; it ascends through the popliteal fossa to the aperture in the Adductor magnus, where it becomes the femoral vein. In the lower part of its course it is placed medial to the artery; between the heads of the Gastrocnemius it is superficial to that vessel; but above the knee-joint, it is close to its lateral side. It receives tributaries corresponding to the branches of the popliteal artery, and it also receives the small saphenous vein. The valves in the popliteal vein are usually four in number. The femoral vein accompanies the femoral artery through the upper two-thirds of the thigh. In the lower part of its course it lies lateral to the artery; higher up, it is behind it; and at the inguinal ligament, it lies on its medial side, and on the same plane. It receives numerous muscular tributaries, and about 4 cm. below the inguinal ligament is joined by the v. profunda femoris; near its termination it is joined by the great saphenous vein. The valves in the femoral vein are three in number. The Deep Femoral Vein receives tributaries corresponding to the perforating branches of the profunda artery, and through these

establishes communications with the popliteal vein below and the inferior gluteal vein above. It also receives the medial and lateral femoral circumflex veins.

The external iliac vein the upward continuation of the femoral vein, begins behind the inguinal ligament, and, passing upward along the brim of the lesser pelvis, ends opposite the sacroiliac articulation, by uniting with the hypogastric vein to form the common iliac vein. The common iliac veins are formed by the union of the external iliac and hypogastric veins, in front of the sacroiliac articulation; passing obliquely upward toward the right side, they end upon the fifth lumbar vertebra, by uniting with each other at an acute angle to form the inferior vena cava. The right common iliac is shorter than the left, nearly vertical in its direction, and ascends behind and then lateral to its corresponding artery. The left common iliac, longer than the right and more oblique in its course, is at first situated on the medial side of the corresponding artery, and then behind the right common iliac. Each common iliac receives the iliolumbar, and sometimes the lateral sacral veins. The left receives, in addition, the middle sacral vein. No valves are found in these veins.

The inferior vena cava returns to the heart the blood from the parts below the diaphragm. It is formed by the junction of the two common iliac veins, on the right side of the fifth lumbar vertebra. It ascends along the front of the vertebral column, on the right side of the aorta. It then perforates the diaphragm between the median and right portions of its central tendon; it subsequently inclines forward and medialward for about 2.5 cm and open into the lower and back part of the right atrium.

ELECTROCARDIOGRAM

Introduction

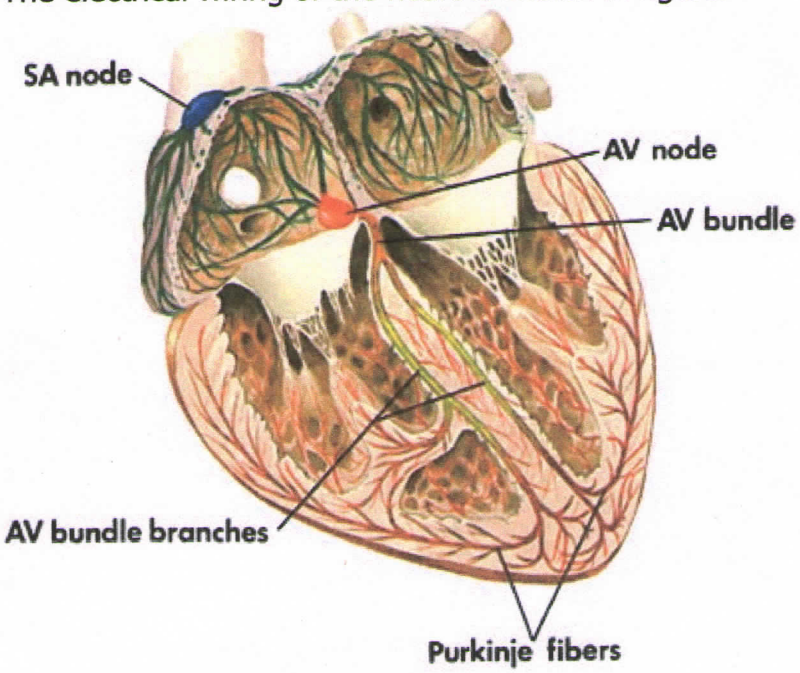
Electrocardiogram is the graphical representation of the electrical activity of the heart, with respect to time. ECG records cardiac electrical potentials by means of electrodes placed on the surface of the body. These electrodes are placed on the arms, legs and chest wall.

Basic Cardiac Electrophysiology

The function of the heart is to contract rhythmically and pump blood to the lungs for oxygenation and then to pump this oxygenated blood into the systemic circulation. The signal for cardiac contraction is the spread of electrical currents through the heart muscle. These currents are produced both by pacemaker cells and specialized conducting tissue within the heart and by the heart muscle itself.

Electrical Stimulation Of The Heart

The electrical wiring of the heart is shown in figure.



Normally the signal for cardiac stimulation starts in the sinus node, also called the sinoatrial (SA) node. The node is located in the right atrium near the opening of the superior vena cava. It is a small collection of specialized cells capable of automatically generating an electrical signal. From the sinus node this stimulus spreads first through

the right atrium and then into left atrium. Thus sinus node functions as the pacemaker of the heart.

The first phase of cardiac muscle activation is electrical stimulation of right and left atria. This in turn signals the atria to contract and pump blood simultaneously through tricuspid and mitral valves into the right and left ventricles. The electrical stimulus then spreads to specialized conduction tissues in the atrioventricular (AV) junction, which includes the AV node and bundle of His, and then into left and right bundle branches, which transmits the stimulus to the ventricular muscle cells.

The AV junction, which acts as an electrical bridge connecting atria and ventricles, is located at the base of the inter atrial septum and extends into the inter ventricular septum. The upper part of the AV junction is the AV node. The lower part of the AV junction is called the bundle of His after the physiologist who described it. The bundle of His the divides into two main branches: the right bundle branch, which distributes the stimulus to the right ventricle and the left bundle branch, which distributes the stimulus to the left ventricle. The electrical signals spreads simultaneously down the left and right bundle branches into the ventricular myocardium by way of specialized conducting cells called Purkinje fibers, which are located in the ventricular myocardium. The spread of stimuli through the ventricles leads to ventricular contraction, with pumping of blood to the lungs and into the general circulation.

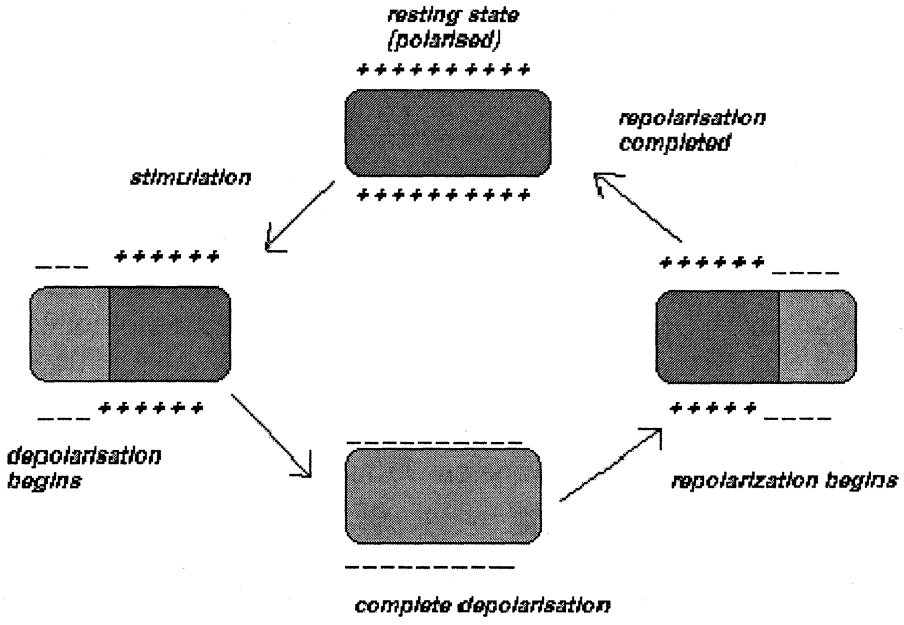
Cardiac Conductivity And Automaticity

The speed of electrical conduction through different part of the heart varies. For example conduction is slowest through the AV node and fastest through the Purkinje fibers. The relatively slow conduction through the AV node is of functional importance because it allows the ventricles time to fill with blood before the signals for cardiac contraction arrives. In addition to conductivity, a major electrical activity of the heart is the automaticity. Automaticity refers to the capacity of certain cardiac cells to function as pacemakers by spontaneously generating electrical impulses that spread throughout the heart. As mentioned earlier the sinus node is the primary pacemaker of the heart because of its inherent automaticity. Under special conditions, however other cells outside the sinus node (in atria, AV junction, or ventricles) can also act as independent pacemakers. For example if the automaticity of the sinus node is depressed, the AV junction can act as a secondary or escape pacemaker.

Depolarization And Repolarization

Normally all myocardial cells are polarized, carries an electrical charge in their surface. The outside of a resting cell is positive and the inside is negative (about 90mv).

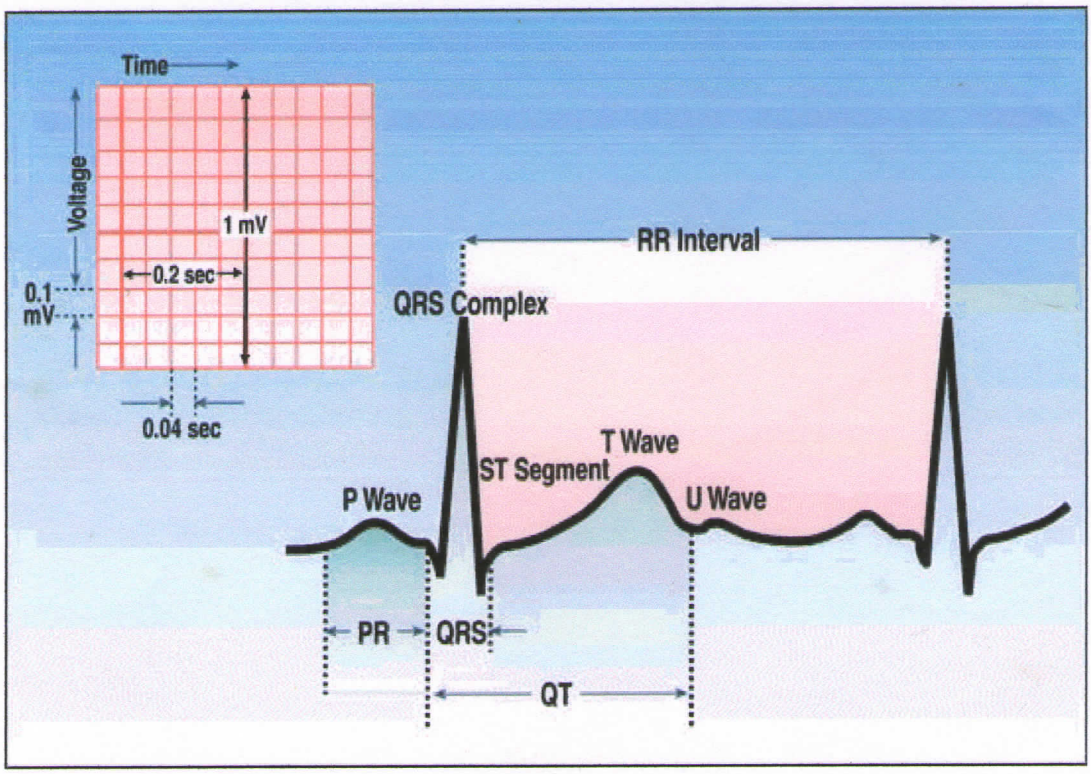
When a heart muscle is stimulated, it depolarizes. As a result the outside of the cell, where the stimulation has occurred becomes negative and the inside of the cell becomes positive. This produces a difference in voltage on the outside surface of the cell between the stimulated depolarized area and the unstimulated polarized area. Consequently a small electric current is formed that spread along the length of the cell, and the whole length of the cell gets depolarized. An arrow as shown in the figure can represent the path of depolarization.



For individual myocardial cells depolarization and repolarization proceed in the same direction. However for the entire myocardium depolarization proceed from innermost layer (endocardium) to outermost layer (epicardium), where as repolarization proceeds in the opposite direction.

The depolarizing electrical current is recorded by the ECG as a P wave when the atria are stimulated, and as a QRS complex when the ventricles are stimulated. After a time fully stimulated and depolarized cells begins to return to the resting state known as repolarization.

Basic ECG Complexes , QRS , T And U Waves



ECG Intervals and Waves

The P wave represents atrial activation; the PR interval is the time from onset of atrial activation to onset of ventricular activation. The QRS complex represents ventricular activation; the QRS duration is the duration of ventricular activation. The ST-T wave represents ventricular repolarization. The QT interval is the duration of ventricular activation and recovery. The U wave probably represents "afterdepolarizations" in the ventricles.

The basic ECG waves are labeled alphabetically and begin with the P wave.

P wave – atrial depolarization (stimulation)

QRS complex – ventricular depolarization

ST segment wave, and U wave – ventricular repolarization

The atrial ST segments and T wave sere generally not observed on the normal ECG because of their low amplitudes and they are buried in the QRS complex.

ECG Paper

The P-QRS-T segments are recorded on special ECG graph paper. Each of the small boxes is 1millimetre square. The paper usually moves sat a speed of 25mm/second.therefore horizontally each unit represents 0.04 sec. The lines between every five boxes are heavier, so that each 5mm unit horizontally represents to 0.2 second. Vertically the ECG graph measures the voltages or amplitudes of the ECG wave. The exact voltage can be measured because the electrocardiograph is standardized so that 1mv signal produces a deflection of 10mm amplitude.

By convention an upward deflection or wave is called positive. A downward deflection is called negative. A deflection that rests on the baseline is called to be bioelectric. A deflection that is partly positive and partly negative is called biphasic.

Standardization Mark

The electrocardiograph must be properly calibrated so that a 1-mv deflection produces a 10 mm deflection. The standardization mark is produced when the machine is correctly calibrated is a square wave 10mm tall. If the machine is not standardized properly, the 1mv signal produces a deflection either more or less than 10mm and the amplitudes of the waveforms will be larger or smaller of the actual value.

P Wave

The P wave, which represents atrial depolarization, is a small positive or negative deflection before the QRS complex. The normal P wave is usually no more than 2.5mm in height and 0.12 second in width.

PR Interval

The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex. PR interval may vary slightly in different leads, and the shortest PR interval should be noted. PR interval represents the time it takes for the stimulus to spared through the atria and pass through the AV junction. In adults the PR interval is between 0.12 and 0.2 seconds. Prolongation of the PR interval above 0.2 second is called first degree AV block.

QRS Complex

QRS complex represents the spread of a stimulus through the ventricles. The initial deflection of the QRS complex is negative; it is called a Q wave. The first positive deflection of the QRS complex is called an R wave. A negative deflection following the r

wave is called an S wave. If the entire complex positive it is called an R wave. If the entire complex is negative, it is termed as a QS wave. Sometimes the QRS complex contains more than two or three deflections. Such extra waves are called R' or R prime waves if they are positive and S' or S prime if they are negative.The QRS width or interval, represents the time required for a stimulus to spread through the ventricles and is normally 0.1 second or less.

ST Segment

The ST segment of an ECG starts from the end of the QRS complex to the beginning of the T wave. It represents the beginning of the ventricular repolarization. The normal ST segment is usually isoelectric. Some pathologic conditions such as myocardial infarction produce abnormal deviation of the ST segment. The beginning of the ST segment, the junction between the end of the QRS complex and the beginning of ST segment is called the J point.

T Wave

The T wave represents the part of ventricular repolarization. It has an asymmetric shape; its peak closer to the end of the wave.

QT Interval

The QT interval is measured from the beginning of the QRS complex to the end of the T wave. It represents the return of the stimulated ventricles to their resting state. The QT interval shortens with the increase in heart rate and vice versa.

U Wave

The U wave is a small rounded deflection sometimes seen after T wave. Its exact significance is not known; functionally u wave represents the last phase of the ventricular repolarization. Prominent U waves are the characteristic of hypokalemia.

Calculation Of Heart Rate

Two methods to measure the heart rate form the ECG

1. When the heart rate is regular , count the number of large boxes (0.2s) between two successive QRS complexes and divide a constant 300 by this (because 300x0.2=60 and the heart rate is being calculated in beats per minute)
2. If the heart rate is irregular, one can determine an average rate by counting the number of cardiac cycles every 6seconds and multiply this number by 10

ECG Leads

The usual way of recording the voltages from the heart is the 12 standard ECG leads. These leads actually show the differences in voltages between electrodes placed on the surface of the body. The ECG leads can be subdivided into two groups: the six extremity leads (limb leads) and six chest leads (precordial leads). The six limb leads – II, III, aVR, aVL, aVF and I records the voltage differences by means of electrodes placed on the limb leads. They are further classified into two – the bipolar extremity leads (I, II, III) and the unipolar extremity leads (aV_R, aV_L, aV_F).

The six chest leads V1, V2, V3, V4, V5, and V6 record voltage differences by means of electrodes placed at various positions on the chest wall. The 12 channels or leads of ECG record the same event, with each lead from a different angle.

Extremity Leads(Limb Leads)

Bipolar Leads (I, II and III)

The electrical voltages of the heart are conducted through the torso to the extremities. There for an electrode placed on the right wrist detects electrical voltages equivalent to those recorded below the right shoulder. Similarly the voltages detected at the left wrist or are equivalent to those recorded below the left shoulder. Voltages detected by the left leg electrode are comparable to those at left thigh or near groin. In practice the electrodes are attached to the wrists and ankles simply for convenience. In connecting a patient to an electrocardiograph first place metal electrodes on the arm and legs. The right leg electrode functions as an electrical ground. The arm electrodes are attached just above the ankles.The bipolar leads are so named so because they record the differences in electrical voltages between two extremities.

Lead I record the difference in voltage between left arm (LA) and right arm (RA) electrodes.

$$\text{Lead I} = \text{LA} - \text{RA}$$

Lead II records the differences in voltage between the left leg (LL) and right arm (RA) electrodes.

$$\text{Lead II} = \text{LL} - \text{RA}$$

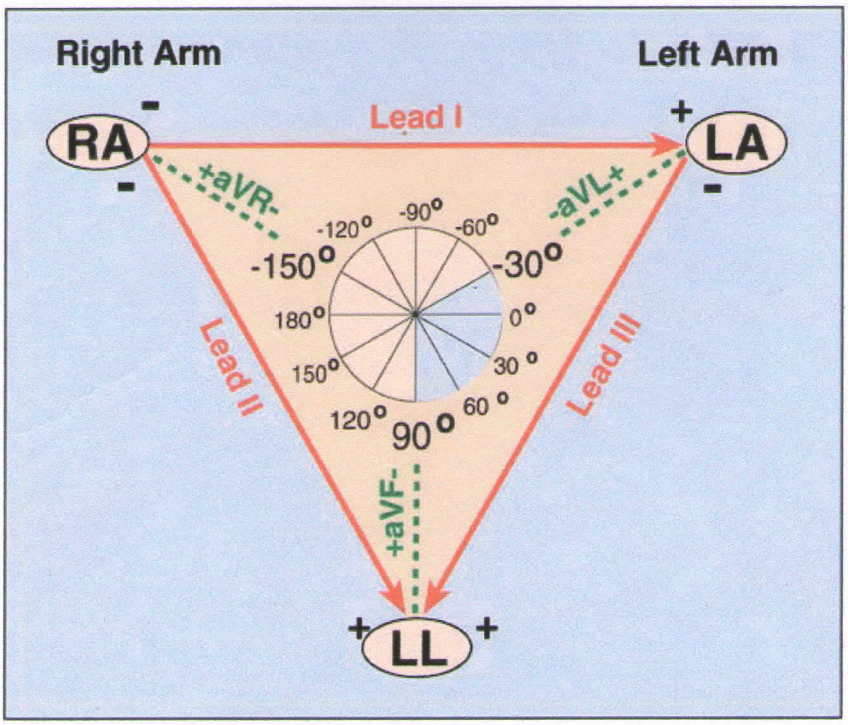
Lead III records the voltage difference between the left leg(LL) and left arm(LA) electrodes.

$$\text{Lead III} = \text{LL} - \text{LA}$$

Leads I, II, and III can be represented schematically in terms of a triangle, called Einthoven's triangle after the Dutch physician who invented the electrocardiograph in the early 1900s. Einthoven's triangle shows the spatial orientation of the three bipolar extremity leads. Lead I points horizontally. Its left pole (LA) is positive and its right pole (RA) is negative. Lead II points diagonally downwards. Its lower end positive and upper pole is negative. Lead III also points diagonally downward. Its lower pole (LA) is positive and the upper pole (LA) is negative.

Einthoven related the bipolar leads by the equation:

Lead I + Lead III = Lead II

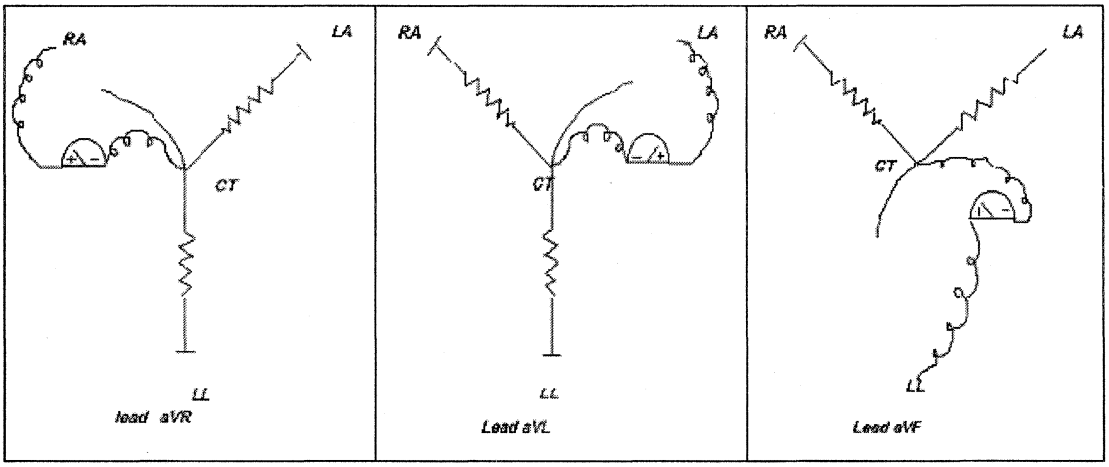


In other words the sum of voltage in lead I and lead III gives the voltage in lead II. In the figure Einthoven's triangle has been redrawn so that leads I, II and III will intersect at a common point. This is done by sliding lead I downward, lead II rightward and lead III leftward and is known as triaxial diagram.

Unipolar Leads (aV_R, aV_L and aV_F)

In the 1930s Dr Frank Wilson and his colleagues at the university of Michigan invented the unipolar extremity leads and introduced the six unipolar chest leads, V_1 through V_6. Few years later Dr. Emmanuel Gold Berger invented the three augmented unipolar extremity leads: aV_R, aV_L and aV_F. The abbreviation "a" refers to augmented; V to voltage; R, L, and F to right arm, left arm and left foot respectively. A unipolar lead records the electrical voltages at one location relative to zero potential rather than

relative to voltages at another extremity, as in the case of bipolar leads. The zero potential is obtained by joining the three extremity leads to a central terminal. Because the sum of voltages of lead I, II and lead III equals zero, the central terminal has a zero voltage.



By connecting all six limb leads, a hexaxial reference system is produced by arranging all the limb leads to a to have a common center point. Einthoven's triangle and hexaxial diagram is essential for determination of electrical axis.

Precordial Leads (Chest Leads)

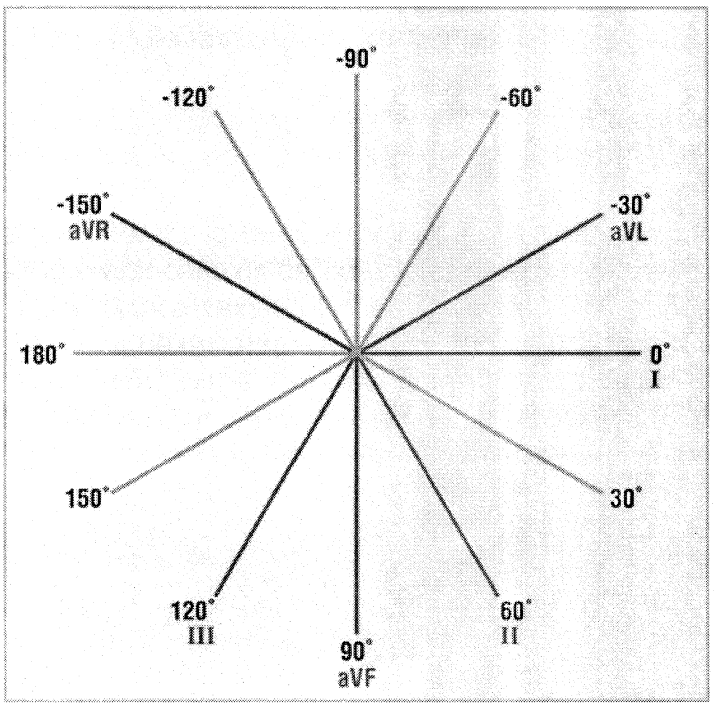
Precordial leads consists of six unipolar chest leads, which include leads V1-6. Lead V1 is placed in the fourth intercostal space at the right sternal border, and lead V2 is in the fourth intercostal space at the left sternal border. Lead V4 is in the fifth intercostals space along the left midclavicular line and lead V3 is halfway between V1 and V2. Leads V1 andV6 are in the fifth intercostals space along the anterior and midaxillary lines respectively.

For special purposes additional precordial leads are necessary. Additional right precordial leads; V3R-5R corresponds to the locations of their opposite numbers on the left side of the chest. These leads are valuable when dealing with various congenital heart diseases, particularly dextrocardia. Sometimes further left and posterior leads may be required. LeadV7 is in the fifth intercostal space along the posterior axillary line. Leads V8 and V9 are at the angle of the scapula and over the spine at the same level as leads V4-6. Occasionally leads V4-6 are taken one intercostal space higher or lower than the usual level. These leads are helpful in diagnosing posterior and lateral myocardial infarction more accurately.

In summary precordial leads corresponds to the horizontal or transverse plane of the electrocardiogram. Many abnormalities like anterior myocardial infarction, posterior infarction, left or right bundle branch block, left or right ventricular hypertrophy can be diagnosed from precordial leads. Apart from that leadV1 is extremely valuable in identifying P waves

Electrical Axis

The electrical axis refers to the vector of the impulses generated by the heart. In conventional ECGs the electrical axis can be determined from the frontal plane and in the horizontal plane. The electrical axes of the P waves, QRS and T waves should be determined separately. The hexaxial reference system is used for the determination of the electrical axis as in the figure. In the hex axial diagram positive pole of lead I is said to be 0°. All points below the lead I axis are positive and all points above that axis are negative. The hex axial diagram used to measure the QRS axis is shown in the figure.



By conventional an electrical axis that points towards lead aVL is termed as leftward or horizontal. An axis that points toward leads II, III and aVF is rightward or vertical.

Mean QRS Axis Calculation

If a wave of depolarization spreads towards the positive pole of any lead, an upward deflection occurs and if the wave moves away from a lead or it spreads towards the negative pole of any lead a downward deflection occurs.

As a general rule, the mean QRS axis lies midway between any two leads that show tall R waves of equal height.

If a wave is oriented right angles to any lead, shows a biphasic RS complex. In this situation, the mean QRS axis is right angle to the lead showing biphasic complex and towards the other lead that show tall R waves.

Axis Deviation

The normal mean QRS axis lies between –30° and +100°. An axis more than +100 or more positive is termed as right axis deviation (RAD). An axis of more negative than – 30 is termed as left axis deviation (LAD)

Right Axis Deviation

RAD is present if the R wave in lead III is taller than the R wave in lead II. In addition lead I show rS complex. RAD can be seen in several conditions including RVH, left posterior hemi block, lateral wall myocardial infraction and chronic lung disease. RAD is some time seen in the ECG of normal people.

Left Axis Deviation

If lead II shows an RS complex in which the S wave is deeper than the R wave. Lead I show a tall R wave and lead III shows a deep S wave. LAD is commonly seen in the ECGs of patients with LVH, left anterior hemi block. Its is also sometimes seen in normal ECGs

More rarely the QRS complex is biphasic in all six-extremity leads. This makes the mean electrical axis indeterminate.

ECG IN CHAMBER ENLARGEMENT AND HYPERTROPHY

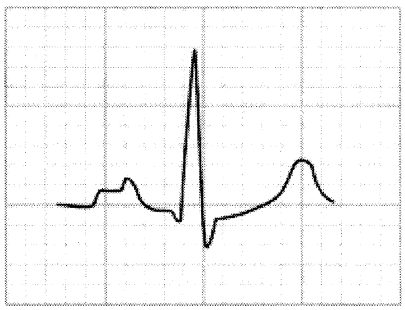
ATRIAL HYPERTROPHY

The atrial activity is reflected in ECG as the P wave. The P wave has largest amplitude in lead II in most adults, because P axis is similar to the axis of lead II. A normal P wave has 2.5mm of width and depth and round upright deflection in lead II and

an inverted configuration in lead aVR. In lead V1 a P wave has two components, positive and negative since it involves left and right atrium. When right or left atrial hypertrophy occurs, the configuration of the P wave is altered with increased amplitude or width of the corresponding component of the hypertrophied atrium.

LEFT ATRIAL HYPERTROPHY

A typical left atrial hypertrophy is manifested by wide (0.12s or more) and notched P waves, especially in leads I, II, and aVL and a wide deep (1mm or more) negative component of the P wave in lead V1 or V2. The P axis of the left atrial hypertrophy is commonly between 0 and +45 in the frontal plane. Since the left atrial hypertrophy is most commonly due to mitral stenosis, the term "P mitrale" is used to designate the finding. When the cardiac mechanism changes to atrial fibrillation, P mitrale pattern is not shown. Here left atrial hypertrophy produces large atrial fibrillation waves, termed as atrial fibrillation. Atrial fibrillation is diagnosed when the fibrillation wave shows an amplitude 1mm or more in lead V1 or V2.



DIAGNOSTIC CRITERIA

Wide (3mm or more) and notched P waves in lead I, II, and aVL

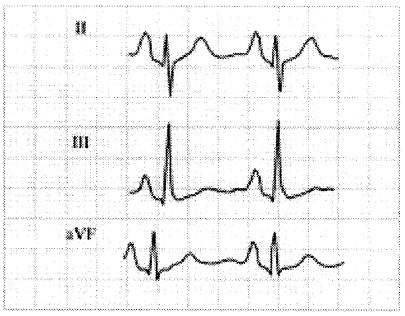
Negative component of P waves in leads V1-2 with depth and width 1mm or more

Coarse atrial fibrillation ("f " waves in lead V1or V2, 1mm or more amplitude)

RIGHT ATRIAL HYPERTROPHY

In right atrial hypertrophy the amplitude of the right atrial component during atrial depolarization is increased. As a result the P waves are tall and tent shaped in leads II, III, and aVF. The P waves are also often tall and peaked in leads V1-2. The most common disease responsible for right atrial hypertrophy is chronic lung disease; the term "P pulmonale" is used to designate right atrial hypertrophy. The P axis ranges from +60 to +90 degrees in the frontal plane. The width of the P wave is not usually prolonged in right

atrial hypertrophy. P pulmonale pattern may be found in marked hypokalemia without right atrial hypertrophy. Inn addition transient P pulmonale pattern is the important sign of pulmonary embolism infarction. When right atrial hypertrophy is caused by congenital heart diseases, the term "P congenitale" is used.



DIAGNOTIC CRITERIA

Tent shaped and tall P waves (3mm or more) in leads I, II and aVF

Positive component of P wave sin leads V1-2 with amplitude 2mm or more.

BIATRIAL HYPERTROPHY

Biatrial hypertrophy is commonly caused by various congenital heart disease and cardiomyopathies

DIAGNOSTIC CRITERIA

Wide and tall P waves in limb leads.

Positive component of P waves in leads V1-2 with amplitude 2mm or more.

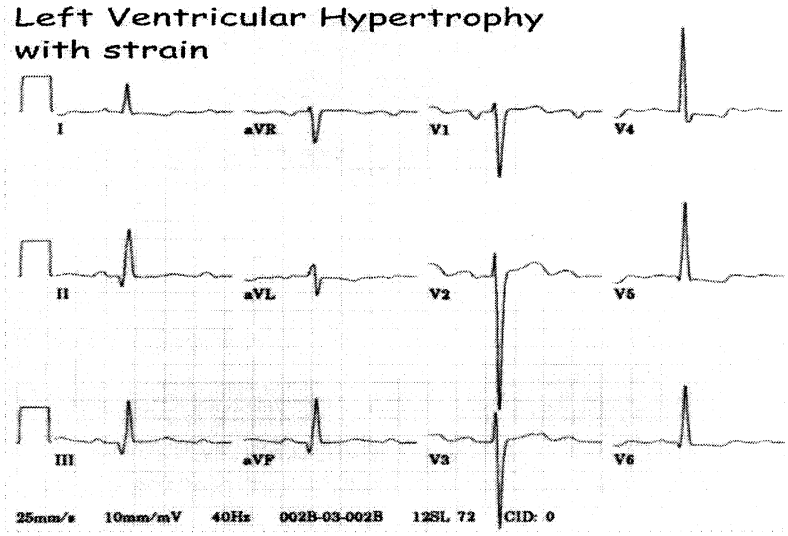
Negative component of P waves in leads V1-2 with depth and width 1mm or more.

VENTRICULAR HYPERTROPHY

The left ventricle is approximately three times thicker than right ventricle. In addition the electrical potential of the left ventricle is about ten times greater than that of right ventricle. So the configuration of the QRS complex during ventricular depolarization is mainly influenced by left ventricular activation. Normally a progressive increment of the R wave amplitude from leads V1 to V6 and a small septal q wave begins to appear from leads V4 to V6. Ventricular hypertrophy alters this normal sequence of electrical activation.

LEFT VENTRICULAR HYPERTROPHY

When left ventricular hypertrophy (LVH) is present left ventricle becomes much more dominant ventricle during ventricular depolarization. As a result abnormally tall positive R waves are usually seen in the left chest leads, and abnormally deep negative S waves are present in the right chest leads



R wave in V5 = 30mm S wave in V1 = 25mm R wave in V5+S wave in V1 = 55mm

Further more a typical LVH produces "strain pattern" in the left precordial leads. It is characterized by a convex upward S_T segment depression with an inverted T wave, most pronounced in leads V5-6, which ours due to secondary T wave change. When LVH is marked, there may be some degree of left axis deviation. But in some cases LVH marked posterior axis deviation is often found.

DIAGNOSTIC CRITERIA

R wave in lead V5 or V6$\geq$ 26mm

R wave in lead V5 or V6 plus S wave in lead V1 $\geq$ 35mm

R wave in lead I $\geq$ 15 mm

R wave in lead I plus S wave in lead III $\geq$ 25 mm

R wave in lead aVL $\geq$ 13 mm or R wave in lead II, III, or aVF $\geq$ 20mm

Secondary T wave change (strain pattern) in leads V5-6 (in systolic overload)

SYSTOLIC OVERLOAD LVH

Systolic overload in the left ventricle occurs when there is an abnormal resistance in the left ventricular outflow tract. In this case heart has to pump against an obstruction, and strain is produced during the systolic phase. Common causes of systolic overload LVH are hypertension. Aortic stenosis, and coarctation of aorta

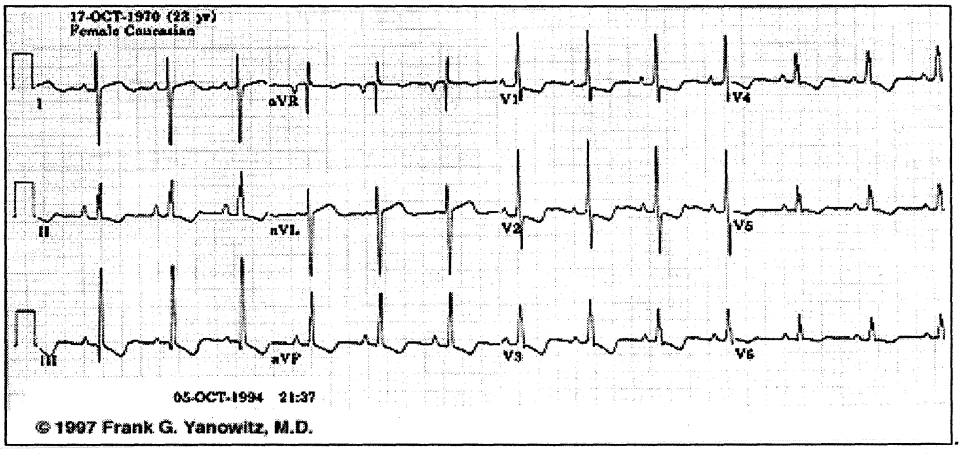
DIASTOLIC OVERLOAD LVH

Diastolic overload occurs in the left ventricle when there is an overflow into the left ventricle. In this case strain is produced primarily during the diastolic phase. Common causes of diastolic overload LVH are aortic insufficiency, mitral insufficiency, and ventricular septal defect. The difference in ECG finding is, the classic strain pattern in left chest leads found in systolic overload LVH is absent in diastolic overload LVH. Also Q waves in leads in left chest leads tend to deeper and R waves tend to be much taller in diastolic overload LVH

RIGHT VENTRICULAR HYPERTROPHY

Right ventricular hypertrophy occurs due to variety of diseases like pulmonary stenosis, atrial septal defect, teratology of Fallot, or Eisenmengers's syndrome. Patient with long standing pulmonary disease may have pulmonary artery hypertension and RVH. Mitral stenosis also can produce a combination of left atrial enlargement and RVH.

With RVH the right chest leads show tall R waves indicating the spread of positive voltages from the hypertrophied right ventricle towards the right. Instead of rS complex seen in lead V1, a tall positive (R) is seen. RVH produces right axis deviation right ventricular strain pattern (t wave inversion) on ECG



DIAGNOSTIC CRITERIA

Right axis deviation

Tall R wave in lead V1

Deep S waves in leads I, aVL, and V4-6

Secondary T wave changes in right hest leads (strain pattern)

BIVENTRICULAR HYPERTROPHY

The diagnosis of biventricular hypertrophy is difficult by ECG because, increased electrical forces in both the ventricles may cancel out each other. A left ventricular hypertrophy pattern in the precordial leads and right axis deviation in limb leads or vice versa indicates biventricular hypertrophy (BVH). Tall R waves in all precordial leads; particularly with large voltage of equiphasic QRS pattern complexes in the midleft precordial leads. Biventricular hypertrophy is caused by congenital heart diseases or cardiomyopathies

DIAGNOSTIC CRITERIA

Left ventricular hypertrophy pattern in the precordial leads and right axis deviation.

Right ventricular hypertrophy in the precordial leads and left axis deviation.

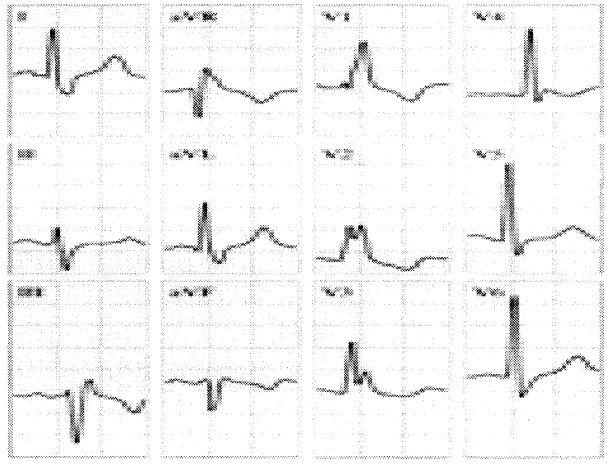
Tall R waves in all precordial leads.

VENTRICULAR CONDUCTION DISTUEBANCES

The most common form of ventricular conduction disturbances is right and left bundle branch blocks. In a bundle branch block, one of the conduction branches will be blocked. So activation will be conducted through an intact bundle. So it produces asynchronous activation of the two ventricles instead of simultaneous activation. So in ECG this is manifested as widened QRS complexes. Bundle branch block produces axis shift also. If the QRS widening is 0.12 s or more in limb leads it is said to be complete bundle branch block. When QRS interval is between 0.10 and 0.11 s it is called as incomplete bundle branch block.

RIGHT BUNDLE BRANH BLOCK (RBBB)

Right bundle branch block produces rSR' complex or M pattern in the right precordial leads and slurred deep S waves in left precordial leads (lead I. aVL and V4-6) due to the delayed activation of the right ventricle. Figure shows ECG with RBBB pattern



DIAGNOSTIC CRITERIA

QRS interval is $\geq$ 0.12s

rSR' or 'M' pattern in leads V1-3

Deep and slurred S wave in leads I, aVL and V4-6

Secondary ST, T wave changes in leads V1-3

RBBB is seen in diagnostic conditions like atrial septal defect with left to right shunt, pulmonary diseases with pulmonary artery hypertension, and pulmonary stenosis.

LEFT BUNDLE BRANCH BLOCK

First phase of ventricular stimulation- depolarization of the septum is from right to left instead of the normal direction- left to right. Thus LBBB shows a loss of r wave in leads V1-3 and absence of septal q wave in leads V4-6. Furthermore total time for ventricular activation is delayed in LBBB, so the QRS complex is abnormally wide. The right chest lead shows a QS pattern, which sometimes shows a notching at its point showing a characteristic W pattern. Similarly the R wave in lead V6 may show the notching at its peak, giving a distinctive M shape. The QRS axis may be normal, but it commonly shows left axis deviation.

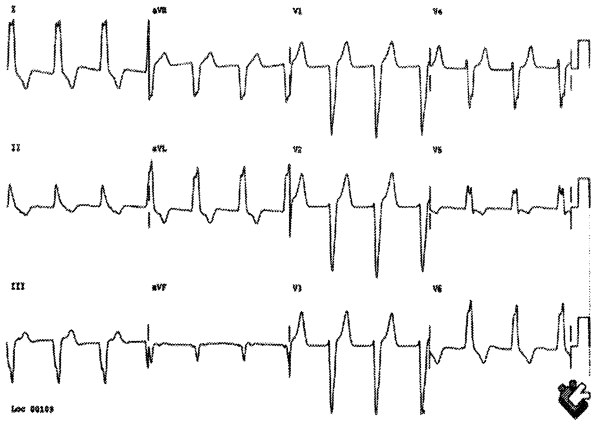


Figure shows an example of LBBB pattern

DIAGNOSTIC CRITERIA

QRS interval $\geq$ 0.12 s

Absence of septal q waves in leads I, aVL, and V4-6

M pattern of R waves in leads I, aVL and V4-6

Secondary T wave changes in leads I, aVL and V4-6

HEMIBLOCKS

Left bundle branch has two subdivisions, anterior (superior) and posterior (inferior) fascicles. When any of these branches is blocked, it is called as hemi block. Recognition of hemi blocks on ECG is related to the subject of axis deviation. Unlike LBBB or RBBB hemi blocks does not widen the QRS complex. The diagnosis of hemi blocks is made from the mean QRS axis in the extremity leads, not in the chest leads.

Left anterior fascicular block

It is characterized by a mean QRS axis of -45° or more. The height of the R wave in lead I is equal to the depth of the S wave in lead aVF when the axis is more than -45°, S wave in lead I becomes larger than R wave of lead I

Left posterior fascicular block

It is characterized by marked right axis deviation. Mean QRS axis is +120° or more. Usually rS complex is seen in lead I and a qR complex is seen in leads II, III, and aVF. However this kind of block is very rare and before making the diagnosis other common causes of RAD must be excluded.

MYOCARDIAL ISCHEMIA AND INFARCTION

The majority of cases myocardial ischemia, injury and infarction are caused by coronary artery disease. If severe narrowing or block in a coronary artery occurs, blood flow becomes inadequate and ischemia of the heart muscle develops. If ischemia is severe necrosis of that part occurs, referred as myocardial infarction (M I). Myocardial ischemia and injury are reversible but infarction or necrosis is irreversible. Myocardial ischemia is manifested by the alteration of T wave change in ECG. Myocardial infarction is the most advanced stage and is manifested by alteration of the QRS complex. The intermediate stage between these two is myocardial injury, which is manifested by the alteration of the ST segment.

Subendocardial Ischemia

Repolarization wave moves from epicardium to endocardium in a normal heart. In subendocardial ischemia a delayed recovery in that region takes place. But it does not reverse the repolarization process because of intact epicardial layer. So it produces prolongation of the QT interval. Magnitude of T wave is also increased. So characteristic feature of subendocardial ischemia is tall and upright T waves and prolonged QT interval.

Subepicardial Ischemia

In subepicardial ischemia the direction of repolarization is reversed from endocardium to epicardium, since the intact endocardium layers start to repolarize before the ischemic epicardial layer. This is reflected as inverted T wave in ECG, which are the primary T wave changes. Characteristically subepicardial ischemia is manifested by deeply and symmetrically inverted T waves.

Myocardial Injury

A horizontal depression or elevation of the ST segment usually indicates injury of the myocardium. A subendocardial injury produces ST segment depression where as subepicardial injury produces ST segment elevation. A transmural injury also gives the same pattern of subepicardial injury. During the early phase of acute MI (24-72hrs) there is always evidence of subepicardial injury.

Myocardial Infarction

Myocardial infarction is the end stage of ischemia-injury-necrosis sequence. Most of the myocardial infarctions are located in the left ventricular free wall and ventricular septum. Uncommon sites of infarction include the right ventricle and the atria. Electrocardiographically the location of the myocardial infarction is expressed according to the abnormality shown in specific leads. Lead II, III and aVF indicate inferior wall involvement. Infarction of anterior wall is shown in chest leads and lead I and aVL. In posterior wall infarction only indirect evidence or reciprocal changes will be shown in leads V1-3.

A transmural infarction generally produces changes in both myocardial depolarization (QRS complex) and myocardial repolarisation (ST-T complex). The earliest ECG changes seen with an acute myocardial infarction usually occurs in the ST-T complex in two sequential phases. The acute phase and evolving phase. Acute phase is marked by the appearance of ST segment elevations and sometimes-tall positive T waves in certain leads. The evolving phase occurs hours or days later and is characterized by deep T wave inversions in the leads that previously showed ST elevations. One of the important characteristics of the ST-T changes seen with MI is their reciprocity. Thus in an anterior wall infarction, with ST elevations in V1-6, ST segment depression is often seen in inferior leads II, III and aVF. In an acute inferior wall infarction leads II, III and aVF shows ST segment elevation and reciprocal ST depression is seen in leads V1-3, I and aVL. In posterior wall MI, only reciprocal changes is shown in the opposite facing leads V1-3.

The ST segment elevation seen in acute MI is called as current of injury. Normally ST segment is isoelectric because no net current flow occurs at this time. MI alters the electrical charge on the myocardial cell membranes and the current flow becomes abnormal producing ST segment deviation. The ST segment elevation seen with acute MI may have different shapes and appearances. It may be plateau shaped, dome shaped or some time obliquely elevated.

QRS Changes: Q wave of Infarction

MI produces characteristic changes in the QRS depolarization and mostly the appearance of new Q waves. A Q wave indicates electrical voltages are directed away from that facing lead. When MI occurs, that portion is electrically dead. So instead of R waves Q waves are recorded on that particular lead either as QS or QR complex. The new Q waves of an MI generally appear within the first day of the infarction. With an anterior wall infarction these Q waves are seen one or more of leads V1-6. with inferior wall MI the new Q waves appear in leads II, III, and aVF

Localization of infarction

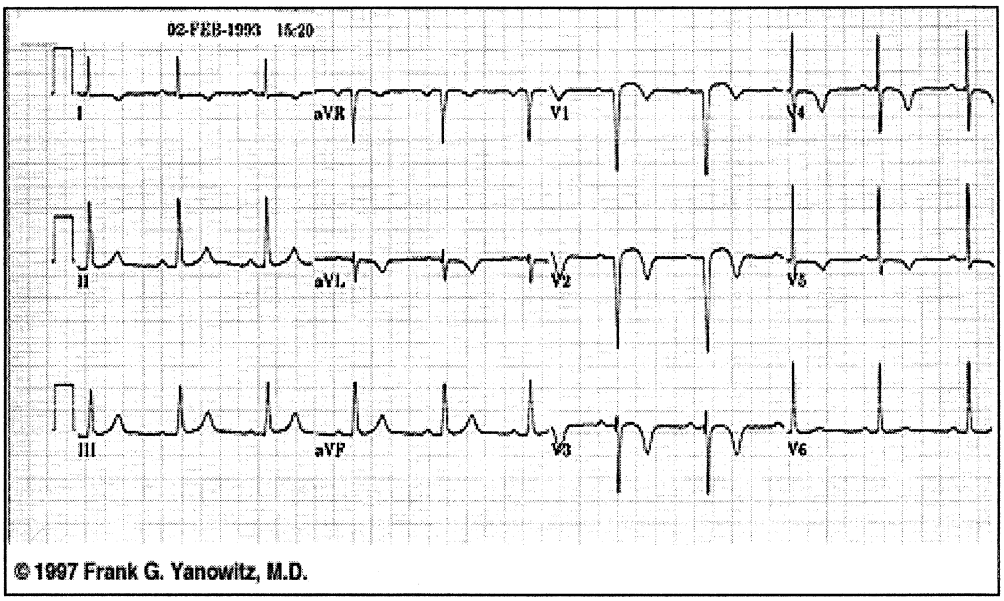
MIs are generally localized to a specific portion of the LV affecting either anterior or inferior wall. Anterior wall infarctions are designated as anteroseptal, strictly anterior or anterolateral/apical depending on the leads that show signs of infarction.

ANTERIOR WALL INFARCTION

The characteristic feature of an anterior wall infarct is the loss of normal R wave progression in the chest leads. Pathologic Q wave will be appeared in one or more of the chest leads.

Anteroseptal infarction

As a result of the damaged septum septal depolarization waves are lost. Thus r waves in leads V1 and V2 disappear and an entirely negative QS complex appears.



Strictly Anterior infarction

Normally leads V3 and V4 shows RS or Rs type complexes. If the infarction occurs in the anterior wall of the LV, the positive R waves that reflect the voltages produced by this muscle area are lost. Instead of that Q waves appears as a part of Qs or QR complex in leads V3 and V4.

Anterolateral or Anteroapical Infarction

An infarction of lateral or apical wall of LV produces changes in more laterally situated leads V5 and V6. With such infarction, abnormal Q waves as a part of Qs or QR complexes appear in leads V5 and V6.

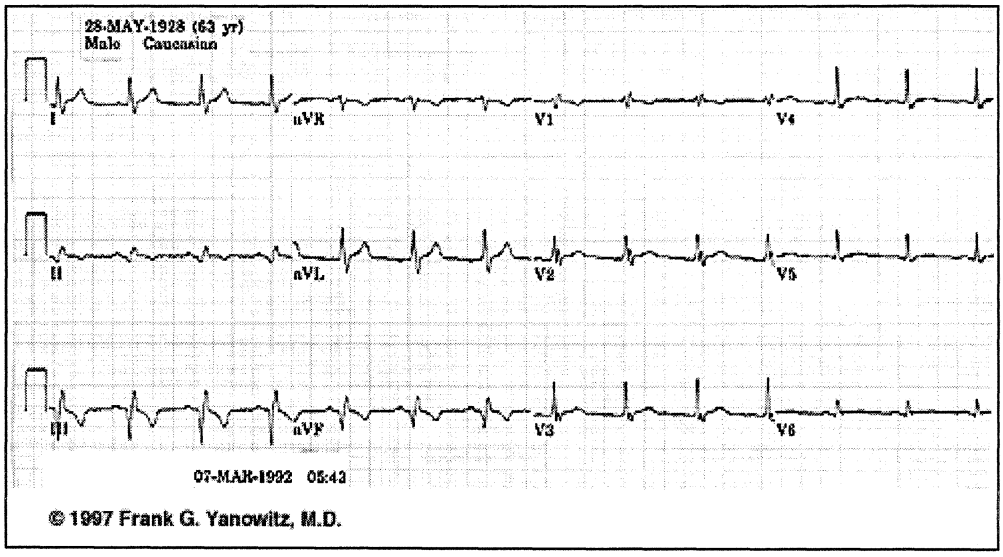
INFERIOR WALL INFARCTION

Infarction of the inferior (diaphragmatic) portion of the LV is indicated by the changes in leads II, III, and aVF. It produces abnormal Q waves inferior leads. Reciprocal ST changes are often observed in anterior leads.

POSTERIOR INFARCTIONS

Infarctions on the posterior side of the LV is difficult to diagnose because characteristic ST elevation may not be seen in any of the conventional 12 lead ECG. Instead tall R waves and ST depressions may our in leads V1-3, reciprocal to the Q

waves and ST elevations that would be recorded at the back of the heart. During the evolving phase anterior chest leads show tall positive T waves reciprocal to T wave inversion on the posterior side. In most of the case posterior infarction extends either to lateral wall of LV producing changes in leads V5-6 or to the inferior wall, producing changes in the inferior leads. Because of this overlap more general term inferoposterior is used when ECG shows changes due to inferior and posterior infarction.

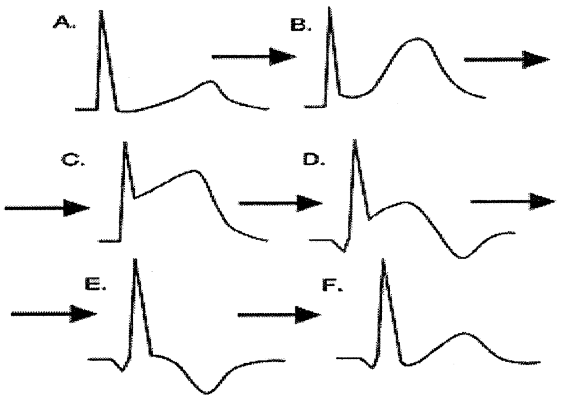


RIGHT VENTRICULAR INFARCTION

Right ventricular infarction is found in every four cases of inferoposterior MI. RV infarction is characterized by ST elevation in leads reflecting the right ventricle such as V1 and V3R to V5R.

Sequence of Q waves and ST-T changes with MI

The earliest sign of transmural ischemia is ST segment elevation. The ST elevation usually persists for hours to days. During this period Q waves begin to appear in the lead that shown ST changes previously. Then ST segments starts to return to isoelectric baseline and T waves become inverted during this evolving phase. The abnormal Q wave persists for weeks or months and even for years after infarction. Occasionally Q waves diminish in size and disappear entirely. In some cases abnormal T wave inversion persists indefinitely.



Evolution of Acute MI

Normal and abnormal Q waves

The normal septal Q waves must be differentiated from the pathologic Q waves of infarction. Normal septal Q waves are characteristically narrow and of low amplitude. Septal Q waves are less than 0.04s in duration. A q wave is abnormal if its duration is 0.04s or more in inferior leads or in leads V3-6. A Q wave of duration more than 0.04s or large qS may be normal variant in lead V1 and rarely in both V1 and V2. An abnormal QS complex resulting from anterior MI sometimes shows a notch as it descends. An abnormal Q wave in only one lead especially lead III is not diagnostic of MI. Q waves in lead III not caused by MI usually disappears during deep inspiration.

Poor R wave progression in hest leads sometimes with actual QS complex in the anterior leads may our with LBBB, LVH, and chronic lung diseases in the absence of MI. prominent Q waves in the absence of MI are sometime called as pseudoinfarct pattern.

Ventricular Aneurysm

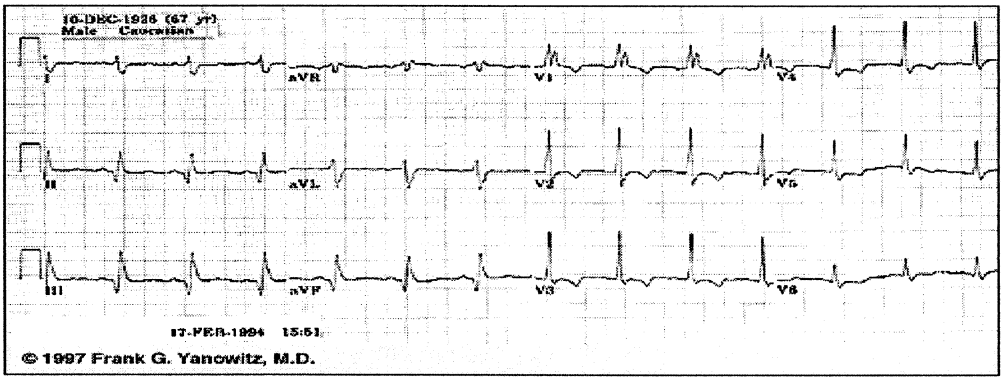
After a large MI a ventricular aneurysm develops for some patients, which are severely scarred area of infarcted ventricular myocardium that does not contract normally. Patient with ventricular aneurysm have persistent ST segment elevation (for several weeks) after an infart.

Diagnosis of MI in the presence of Bundle branch block

RBBB with myocardial infarction

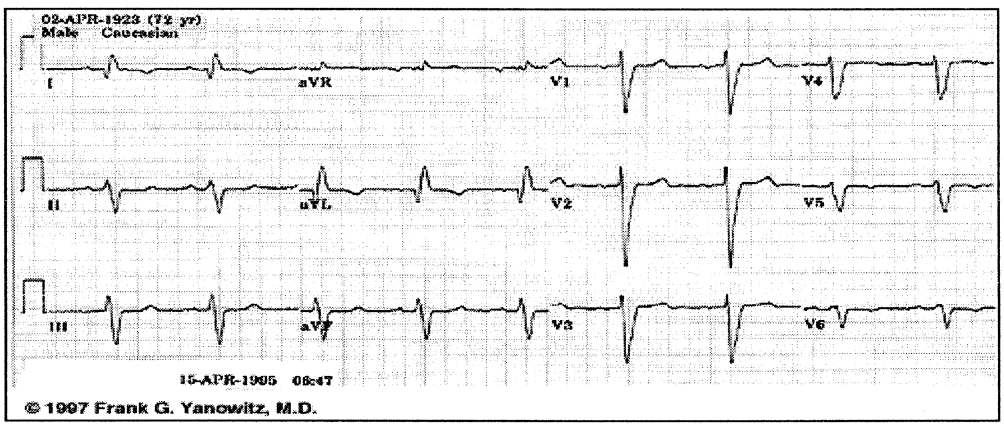
RBBB affects mainly the terminal phase of ventricular depolarization producing a wide R wave in right chest leads and a wide S wave in the left chest leads. MI affects the initial phase of ventricular depolarization, producing abnormal Q waves. When RBBB and infarction occurs together combination of these two patterns are seen. QRS complex is abnormally wide, and lead V1 shows a terminal positive deflection and lead V6 shows a wide S wave. If the infarction is anterior the ECG shows a loss of R wave progression

with abnormal Q waves in the anterior leads and characteristic ST-T changes. If the infarction is pathologic Q waves and ST-T changes are seen in inferior leads.



LBBB with myocardial infarction

LBBB interrupts both the early and late phase of ventricular stimulation. It also produces secondary ST-T changes. Thus LBBB hides all the diagnosis of an infarction making the diagnosis of MI with LBBB complicated. Occasionally patients with LBBB manifest primary ST-T changes indicative of ischemia or infarction. LBBB shows poor R wave progression in the chest leads because of reversed way of ventricular septal activation, this mimics anterior wall MI. so in a patient with LBBB, anterior infarction should not be diagnosed on the basis of poor R wave progression in the right chest leads. However the presence of Q waves as part of QR complexes in the left chest leads with LBBB indicates underlying MI. In addition the appearance of ST elevations in the left chest leads or in other leads with prominent R waves suggests ischemia.



ATRIOVENTRICULAR HEART BLOCK

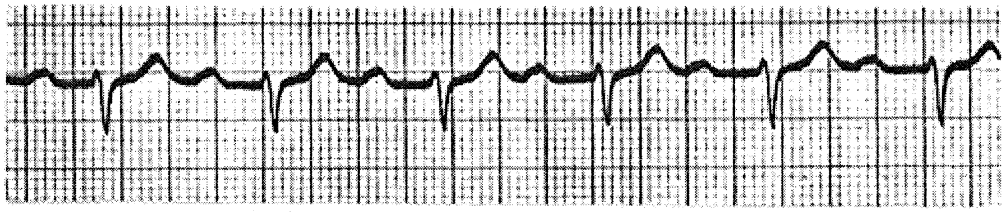
Conduction disturbances occurring in the heart are expressed as heart blocks. A heart block means that refractory period of certain areas of the heart particularly conduction system is abnormally prolonged. It may affect any area of the conduction network but generally the term block refers to indicate atrioventricular (AV) conduction disturbances. In the ECG PR interval is the measure of conduction time between atria and ventricles and normal PR interval is between 0.12 and 0.2 s. heart block in AV junction may be transient, permanent or intermittent and one type may change to another type from time to time.

Classification of AV block

The following classification is generally used according to the degree of the AV conduction disturbance. (1) First degree AV block , (2) Second degree AV block, (3) High degree AV block, (4) Third degree or complete heart block.

FIRST DEGREE AV BLOCK

First degree AV block is characterized by a prolonged PR interval. (More than 0.2 s in adults and 0.18 s or more in children). Here each atrial impulse is conducted to the ventricles. PR interval is generally constant throughout the cycle, but may be altered according to the heart rate. PR interval may prolong as much as 0.8s.



1st degree AV block (PR = 280 ms)

Mechanism: In most of the cases first degree AV block is caused by the prolongation of the refractory period of AV junction, which extends to end of the cycle. In some cases there is a prolongation of the PR interval when there is an impairment of conduction in the common AV bundle is present. Patients with ischemic heart disease may have block of any degree, particularly with inferior wall MI because the right coronary artery that generally supplies inferior wall of the heart also supplies the AV junction. Heart blocks during inferior wall MI tend to be transient. Hyperkalemia may also cause prolonged AV conduction.

First degree AV block is the most common conduction disturbance found in individuals especially in elderly people due to the degenerative changes in the conduction system. First degree AV block is considered to be the most common and earliest finding

in acute rheumatic fever in children. Treatment of this conduction disturbance is not indicated, but the underlying cause must be investigated.

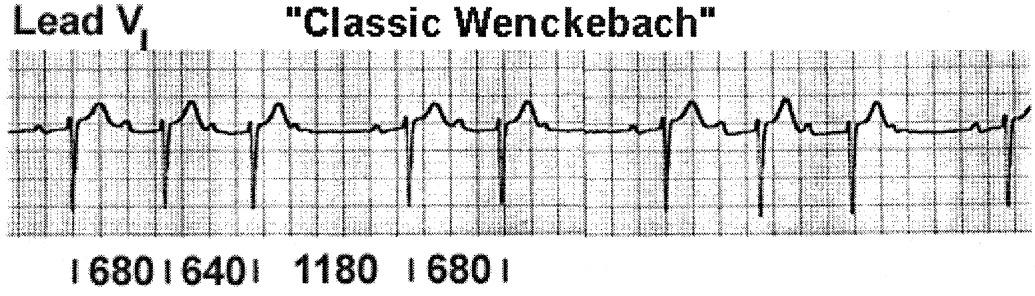
SECOND DEGREE AV BLOCK

Second degree AV block is diagnosed when a P wave is blocked, or it is not followed by QRS, T complexes because of an abnormally prolonged refractory period of the AV node. Second degree AV block has two classifications namely Mobitz type I and Mobitz type II AV block. Mobitz type I AV block is also called as Wenckebach AV block.

Mobitz type I AV block indicates intra nodal block, where as Mobitz type II indicates infranodal block.

Mobitz type I AV block

It is characterized by a progressive lengthening of the PR intervals and a P wave is blocked. The occurrence of a blocked P wave preceded by a progressive lengthening of PR interval is termed as Wenckebach phenomenon. The blocked P wave may occur occasionally, frequently or periodically. The ratio of AV conduction is expressed according to the number of the P wave versus the number of conducting QRS complexes. When one out of four atrial impulses is blocked, the AV conduction ratio is expressed as 4: 3 AV block. Mobitz type II AV block is caused by a block either in the His bundles or in both bundle branches. Two or more consecutively appearing blocked P waves such as 3:1 or 4:1 AV blocks are termed as High degree or Advanced AV block. The Wenckebach cycle also produces a distinct clustering of QRS complexes separated by a pause, that results from the dropped beat. If an ECG shows this kind of group beating its is a particular diagnostic pattern of Wenckebach block.



Diagnostic criteria

Progressive lengthening of PR intervals followed by a blocked P wave.

Progressive shortening of RR intervals followed by a blocked P wave

The largest RR interval is shorter than two cycles of the PP interval, which one is found with the blocked P wave.

Regular irregularity of RR interval when the AV ratio is fixed

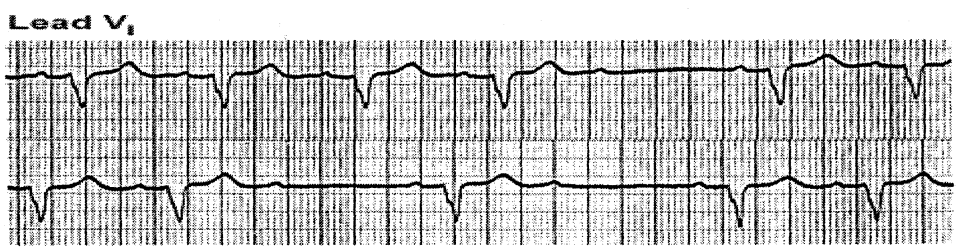
Regular PP intervals

Patients with Wenckebach type of AV block are usually without symptoms unless ventricular rate is slow. Common causes of this block include ischemic heart disease and drugs such as digitalis, beta-blockers and calcium channel blockers. Wenckebach associated with acute inferior wall M I is transient and does not require therapy.

Mobitz type II AV block

Mobitz type II AV block is diagnosed when blocked P wave occur periodically without a preceding Wenckebach phenomenon. The PR intervals of all the conducted beats in Mobitz type II AV block are usually constant, but the PR interval may be prolonged. The ratio of AV conduction may be fixed or vary. The AV ratio is commonly 3:2, 4:3 etc. when 2:1 ratio is present a specification between Mobitz type I and type II is not possible. However if 2:1 AV block is associated with hemi block, bundle branch block the diagnosis is Mobitz type II.

The RR intervals including the blocked P waves are usually multiples of the PP intervals of the conducted beats. The RR interval of all the conducted beats other than RR interval including the blocked P wave is constant. PP interval is also constant if the rhythm is sinus in Mobitz type II block. Since the block represents infra nodal damage QRS complex may exhibit the pattern of hemiblock, bundle branch block or bifascicular block.



Diagnostic criteria

Periodic occurrence of a blocked P wave without grouping of QRS complexes

Constant PR intervals in all conducted beats

QRS complexes may show hemiblock, bundle branch block or bifascicular block

Mobitz type II block is generally a sign of conduction system disease in the infranodal region i.e. His Purkinje system. It often progress into complete heart block. So Mobitz type II block is an indication for pacemaker implantation. It is not seen with digitals toxicity or inferior wall M I, but seen with anterior wall M I.

High degree AV block

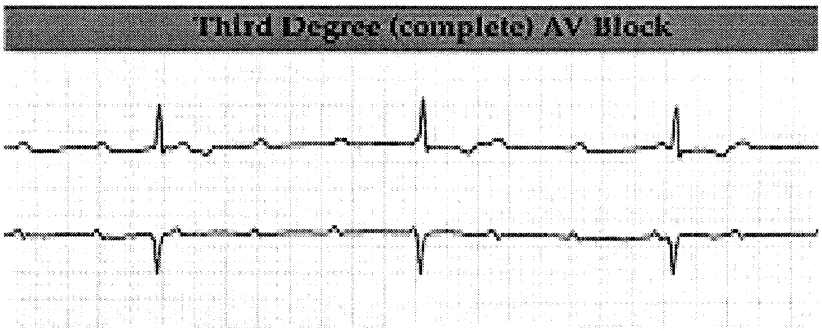
High degree or advanced AV block is diagnosed when the AV conduction ratio is 3:1 or more. It is the most advanced form of incomplete AV block. Common AV conduction ratios are even numbers such as 4:1, 6:1 and 8:1 etc. Odd number ratios are uncommon. Because of slow ventricular rhythm in high degree AV block AV, Junctional escape beats or ventricular escape beats appear to control the ventricles. One or more AV Junctional escape beats occur when AV conduction ratios are 4:1 or higher. When conduction ratios are further increased the number of escape beats exceed those of the conducted beats. In this circumstances conducted beats may occur very rarely, otherwise ECG shows complete AV block. In high degree AV block, incomplete AV dissociation occurs because the sinus P waves and escape waves are independent.

PR interval in high degree AV block will be normal or prolonged for all conducted beats, unless Wenckebach phenomenon occurs. Atrial rhythm will be sinus or ectopic. So the PP interval is generally regular. The RR intervals are almost irregular because of the frequent occurrence of escape beats in addition to the conducted beats. The configuration of QRS complex depends on the origin of escape rhythm. All conducted beats have normal QRS configuration. QRS complex from the AV junction is also normal but the QRS complex of a ventricular escape beat as a result of infranodal block is wide and bizarre.

THIRD DEGREE AV BLOCK OR COMPLETE HEART BLOCK

In complete heart block no atrial stimuli are transmitted to the ventricles and ventricles are paced independently. The atria generally continue to be paced from sinus node. But the ventricles are paced by an escape pacemaker paced below located below the block. The resting ventricular rate in CHB may be lower than 30 bpm or higher as 50 to 60 bpm. The atrial rate is generally faster than the ventricular rate. CHB may also occur in patients whose basal atrial rhythm is flutter or fibrillation. In these cases the ventricular rate is very slow and completely irregular. CHB is caused by a prolongation of the absolute refractory period, so that it occupies the entire cardiac cycle and no relative refractory phase is present. When the atrial mechanism is sinus, the P-P intervals are often regular unless sinus arrhythmia is present. The R-R intervals in AV Junctional or ventricular escape rhythm are usually regular .

Complete AVBlock (3rdDegree) with Junctional Rhythm



Diagnostic criteria

Independent atrial and ventricular activities producing AV dissociation.

Atrial rhythm may be sinus or ectopic

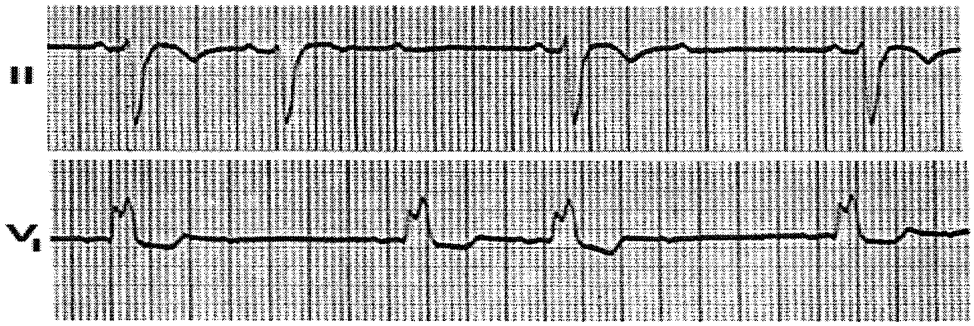
Ventricular rhythm may be AV Junctional or ventricular escape beat

Complete heart is commonly caused by block at the AV junction or bilateral bundles. Depending upon the etiology the HB may be transient or permanent. HB caused by acute anterior wall MI or digitalis toxicities are transient. But anterior wall MI often produces permanent CHB. Common cause of CHB is the degenerative changes in the conduction system, especially in elderly people. HB may be congenital also.

Severe congestive heart failure and Stoke Adams syndrome is frequently occurred in complete AV block. Implantation of permanent pacemaker is always necessary for the treatment of complete AV block.

BIFASCICULAR AND TRIFASICULAR BLOCK

Patients with complete heart block often have other conduction disturbances before the onset of complete heart block. These conduction disturbances are referred as bifascicular blocks. Bifascicular block indicates the blockage of any of two of the three fascicles. For example RBBB with left anterior hemiblock produces RBBB pattern with left axis deviation. RBBB with left posterior hemiblock produces right axis deviation. Similarly a complete heart block indicates blockage of both the anterior and posterior fascicles. Bifascicular block is significant because they make ventricular conduction depend on the single fascicle. Additional damage to this fascicle produce complete AV block or trifascicular block.



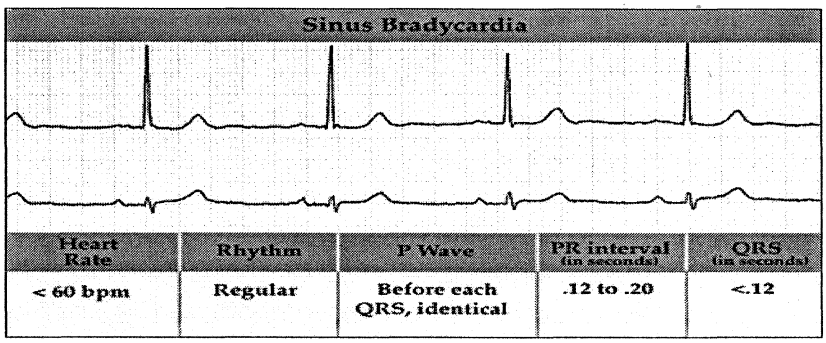
Trifascicular Block: RBBB, LAFB, and Mobitz II 2nd Degree AV Block

CARDIAC ARRHYTHMIAS

There are five necessary criteria for the diagnosis of normal sinus rhythm. Normal axis of P wave, normal P-R interval, constant P wave configuration, normal rate and constant R-R cycle. When any one of these criteria are lacking, a cardiac arrhythmia of sinus origin is present. They include,

Sinus bradycardia

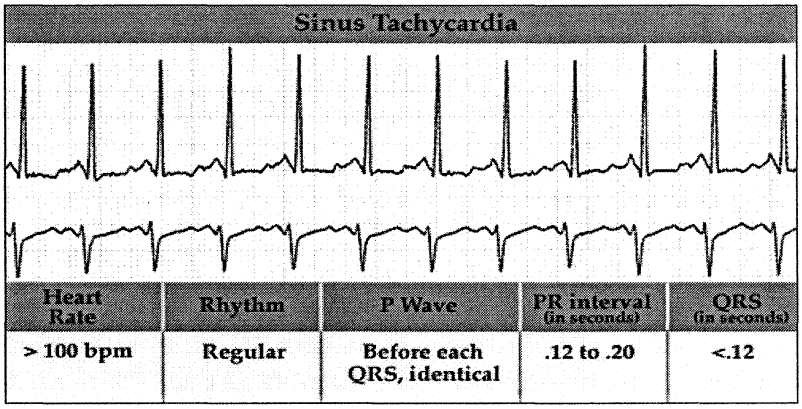
Sinus rhythm with a rate below 60 beats/minute is termed sinus bradycardia. A diagnostic criterion includes normal P wave, constant P-R interval, and rate between 45-60-beats/per minute.



Increased vagal tone is responsible for spontaneous bradycardia. Drugs that increase vagal tone (digitalis) or that decrease sympathetic tone (beta blockers and calcium channel blockers) may cause marked bradycardia. In most of the cases sinus bradycardia is asymptomatic and doesn't need therapy. But patients with markedly slow rate need therapy, which include drugs such as atropine.

Sinus tachycardia

Sinus tachycardia is diagnosed when all the five criteria for a normal sinus rhythm are present except the rate is faster than 100 beats/minute. Usual tachycardia rate is between 101-160 beats/min. diagnostic criteria include normal P wave, constant and normal PR interval, and constant P wave configuration. Common causes of tachycardia include emotion, anxiety or sympathomimatic drugs. Treatment of sinus tachycardia is not required unless the arrhythmia is associated with cardiac or non-cardiac disorders.

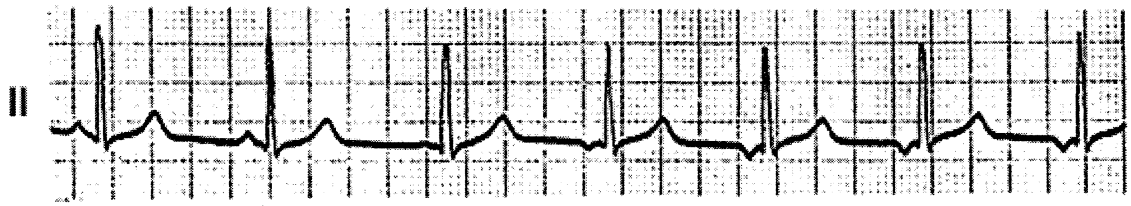


Sinus arrhythmia

Sinus arrhythmia is diagnosed when the P-P or R-R intervals in the sinus rhythm vary 0.16s or more. Sinus arrhythmia is divided into two types – respiratory and non-respiratory sinus arrhythmias. In respiratory sinus arrhythmia the sinus cycle vary in relation to the respiratory cycle. Sinus rate increases with inspiration and slows with expiration. Respiratory sinus arrhythmia is produced by the variation in the vagal tone by reflex mechanism in the pulmonary and systemic vascular system in relation to respiratory cycle. Non-respiratory sinus arrhythmia is the irregular sinus cycle not related to the respiratory cycle. It is found only in diseased heart.

Wandering pacemaker in the sinus node

A wandering pacemaker in the sinus node is one that shifts from one part of the sinus node to another. Thus the configuration of P wave changes from beat to beat, but the P-R interval either remains constant or changes between 0.12 and 0.2s.



Wandering atrial pacemaker

Wandering atrial pacemaker is a benign rhythm change where the pacemaker site shifts from the sinus node into the atrial tissues. P-wave morphology varies with the pacemaker site.

Diagnostic criteria

P wave of sinus origin

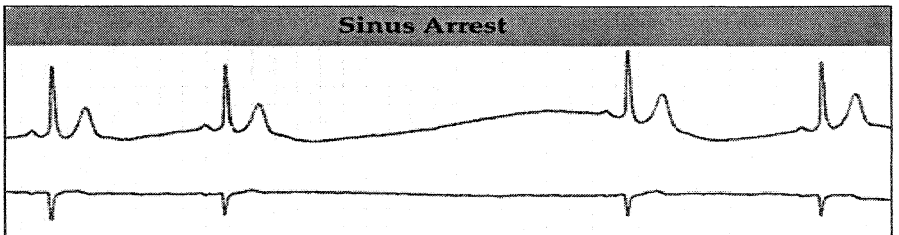
Varying P wave configuration in each lead.

Relatively constant P-R interval (may vary between 0.12s-0.2s)

Slightly irregular or regular P-P cycles.

Sinus arrest

Sinus arrest is the failure of impulse formation in the sinus node. Since no impulse is formed in the sinus node no electrocardiographic wave is produced. Thus sinus arrest is manifested by the absence of the P wave and QRS, T complexes. Sinus arrest may occur in normal people with increased vagal tone or with hypertensive carotid sinus. It may also occur with the over dosage of digitalis or quinidine. It has been observed that sinus arrest occurs following AV Junctional rhythm when the sinus node is suppressed by ectopic discharge from the AV Junctional pacemaker. When sinus arrest occurs AV Junctional escape beats may occur as a physiologic mechanism to control the ventricles.



Please note: trace and grid have been reduced in size

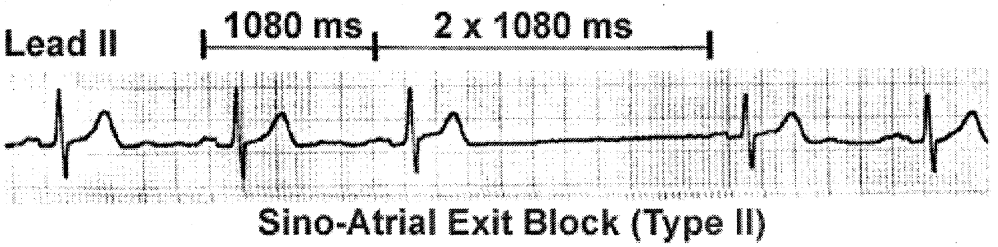
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
N/A	Irregular	Before each QRS identical. New rhythm begins after a pause. The P to P interval is disturbed.	.12 to .20	<.12

Sinoatrial block

Sino atrial block may be classified into first second and third degrees S-A block analogous to A-V block. But only second degree A-V block can be diagnosed with certainty because it is unable to record first and third degree S-A block in normal ECG. Second degree S-A block can be divide into (1) Mobitz type II S-A block, which is comparable to Mobitz type II A-V block, and (2) Wenckebach S-A block which is comparable to Wenckebach A-V block.

Mobitz type II S-A block

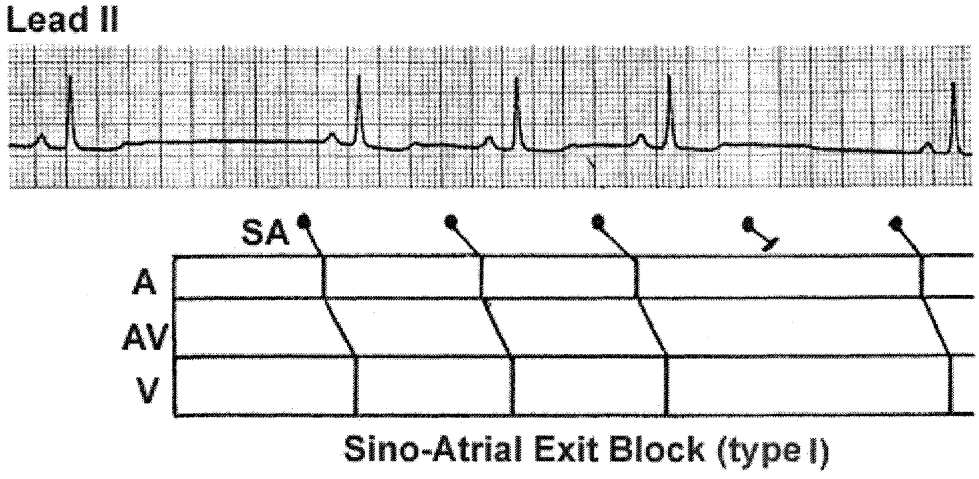
In second degree S-A block, there will be an occasional absence of one or more beats of P, QRS and T complexes. The P-P interval including the S-A block is exactly the multiples of P-P cycles of the basic rhythm. Thus the long P-P interval containing the S-A block will commonly be twice and less commonly three or four times P-P cycles of the basic rhythm. S-A block of this type may occur periodically regular or irregular intervals. When S-A block occurs two or three times continuously atrial standstill occurs unless an ectopic focus takes over to activate the atria.



Wenckebach S-A block (Mobitz type I)

In this type of S-A block, the long P-P intervals because of the S-A block are not the multiples of the P-P cycles of the basic rhythm as in type II S-A block. In Wenckebach

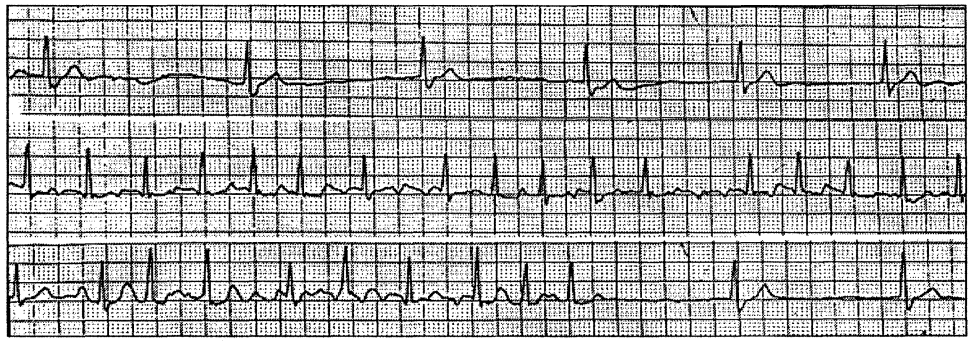
S-A block, the P-P intervals caused by the S-A block may vary. The P-P intervals preceding the longer pause become progressively shorter until a dropped beat occurs. Wenckebach S-A block can be diagnosed only by finding the cyclic shortening of the P-P intervals followed by a long pause. Sinoatrial block is differentiated from the sinus arrest, by a long P-P interval unrelated to the P-P cycle of the basic rhythm



Sino-Atrial Exit Block (type I)

Sick Sinus Syndrome

The term sick sinus syndrome is used to describe a broad spectrum of clinical manifestations. Electrocardiographic manifestations in the sick sinus syndrome may include: (1) persistent and sinus bradycardia (2) Sino atrial block (3) sinus arrest (4) long pause following an atrial premature contraction (5) chronic atrial flutter or fibrillation with slow ventricular rate (6) brady-tachyarrhythmia syndrome. In this case sinus node is severely diseased, leading to complete generator failure.

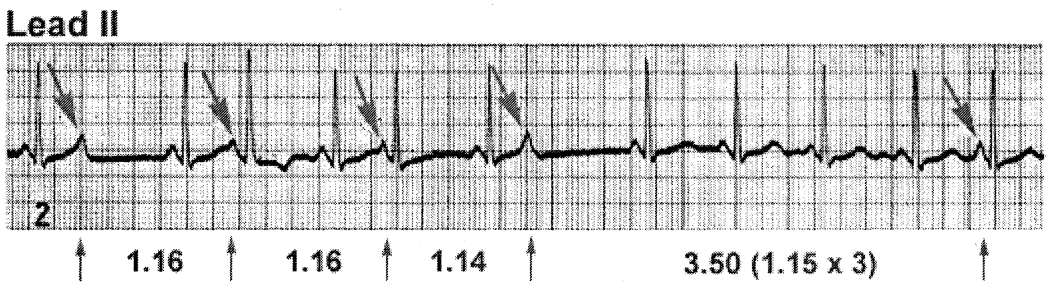


Electrocardiogram exhibiting alternating patterns of bradycardia and tachycardia as seen in patients with sick sinus syndrome.

ATRIAL ARRHYTHMIAS

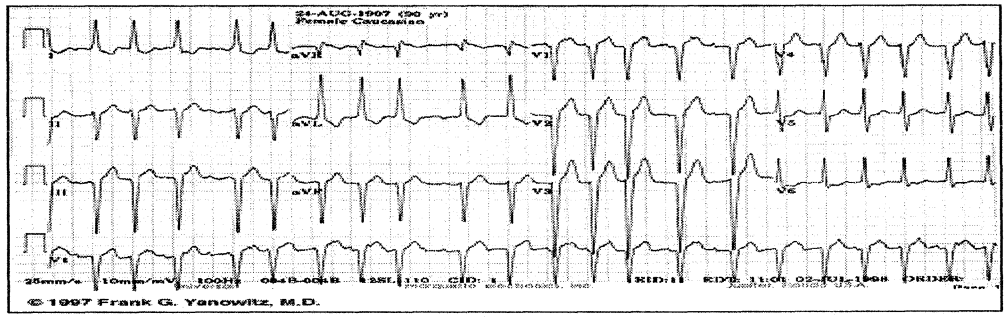
Atrial premature contractions (APCs)

Atrial premature contraction is diagnosed when the ectopic P wave appears earlier than the cardiac cycles of the basic rhythm. APCs may occur from anywhere in the atria outside the sinus node. The ectopic P wave will resemble the P wave of sinus origin when the ectopic focus is located near the sinus node. The ectopic P wave may be conducted retrograde when the ectopic focus is located near the A-V junction. The impulse from the atrial premature focus activates the entire area, including the sinus node. The sinus node is prematurely discharged and its inherent sinus impulse discharge will be suppressed momentarily. The prolongation of impulse in the atria is usually different from that of sinus impulse, but conduction below A-V node is identical to that in normal sinus rhythm.



Atrial tachycardia

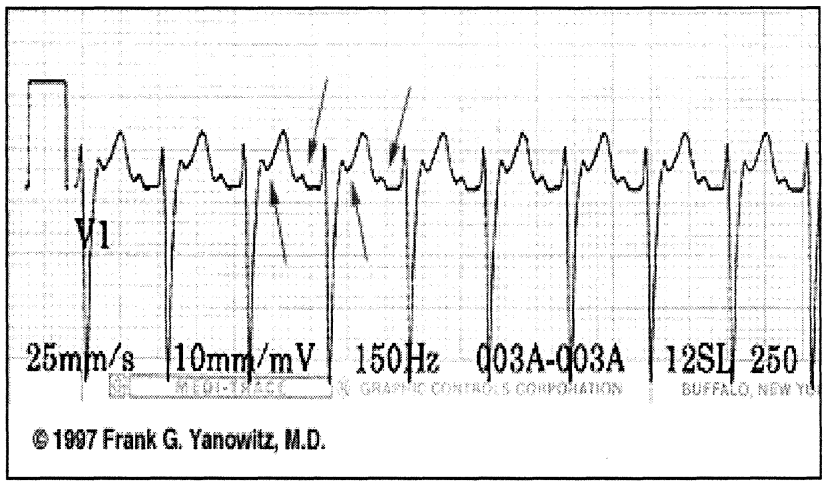
Atrial tachycardia is diagnosed when there is a run of six or more consecutive atrial premature contractions. In most atrial tachycardias the ectopic P wave is often upright in lead II and inverted in lead aVR. The rate of atrial tachycardia may be between 160 to 250 beats per minute.



Atrial flutter

Atrial flutter is characterized by rapid polarization and Repolarization of the atria. Atrial flutter produces characteristic flutter waves. Flutter wave is composed of two parts directed in opposite directions. First portion corresponds to the P wave of atrial depolarization followed by wave of Repolarization, which gives characteristic saw tooth

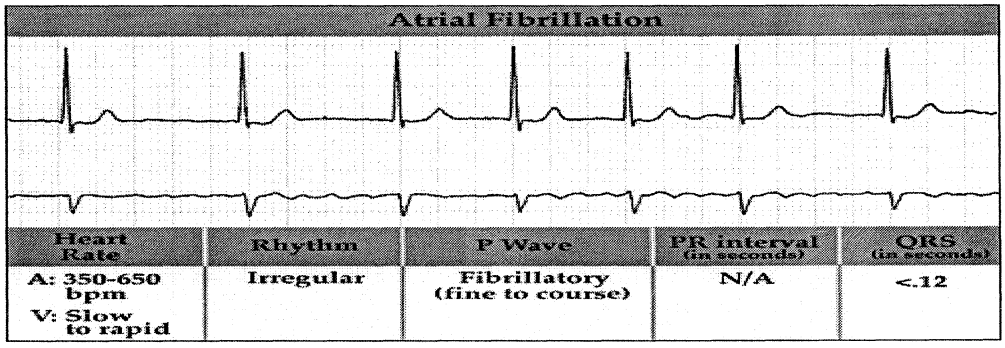
wave appearance. The atrial rate will be in between 250 and 350 beats per min. usually atrial flutter shows 2:1 ventricular conduction.



The arrows point to two flutter waves for each QRS complex. Atrial rate = 280; ventricular rate = 140.

Atrial fibrillation

In atrial fibrillation the atria are stimulated at a very rapid rate, up to 600 beats per min. this fibrillary activity produces irregularly wavy pattern in the place of normal P wave. These waves are called f waves. Ventricular rate may be high as 110-180 beats /min.



A-V JUNCTIONAL ARRHYTHMIAS

A-V Nodal Reentrant Tachycardia (AVNRT)

AVNRT is caused by a rapidly circulating impulse in the AV node area. The term reentry means that the cardiac cycle spins around the A-V node. AVNRT produces a very rapid and regular supra ventricular rhythm with rates between 140 and 250 beats per minute. The reentrant rhythm originates in the A-V nodal area and spreads simultaneously up the atria and down the ventricles. So the P waves are usually hidden in

the QRS complex because the atria and ventricles are activated simultaneously. Because of the retrograde conduction inverted P waves may be seen lead II

AtrioVentricular Reentrant Tachycardia

In patients with atrioventricular reentrant tachycardia there will be a bypass tract between the atria and the ventricles, circumventing the AV node. This bypass tract may cause tachycardia by reentrant mechanism. The impulse initially travels down through the normal conduction system into the ventricles, recycles rapidly up into the atria via the bypass tract, and then goes down the AV node again. Repetition of this large reentrant circuit leads to paroxysmal supraventricular tachycardia.

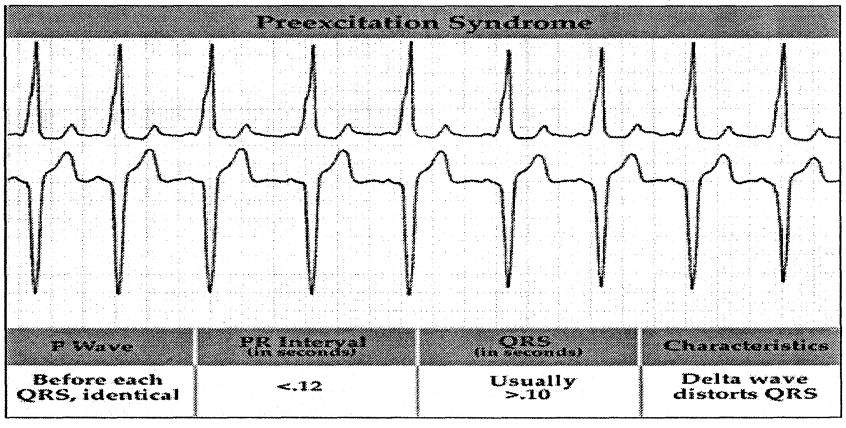
WOLFF-PARKINSON-WHITE SYNDROME (Ventricular Preexcitation Syndrome)

Wolf Parkinson White syndrome (WPW) is an unusual ECG abnormality caused by preexcitation of the ventricles, through an extra pathway between atria and ventricles. This produces the following changes in the ECG.

The QRS complex is widened, giving the appearance of right bundle branch block pattern

The PR interval is shortened because of preexcitation.

The upstroke of QRS is slurred and notched. This is called a delta wave.



AV Junctional escape rhythm

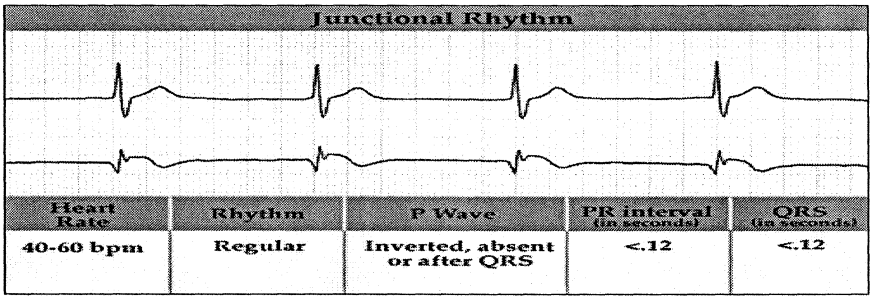
Under certain circumstances the A-V junction may function as an ectopic pacemaker, producing A-V Junctional rhythm. When A-V junction is the cardiac pacemaker the atria is paced in a retrograde fashion. This produces a positive P wave in lead aVR and negative P wave in lead II. The ventricles are depolarized normally, producing a narrow QRS complex.

Diagnostic criteria

Retrograde P wave (positive in lead aVR and negative in lead II) immediately preceding t he QRS complex.

Retrograde P waves immediately following the QRS complex.

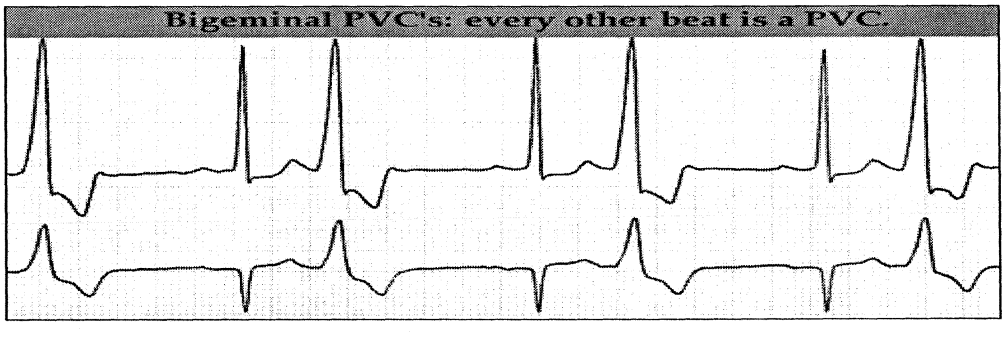
Absent P waves, so that the baseline between QRS complexes is flat.



VENTRICULAR ARRHYTHMIAS

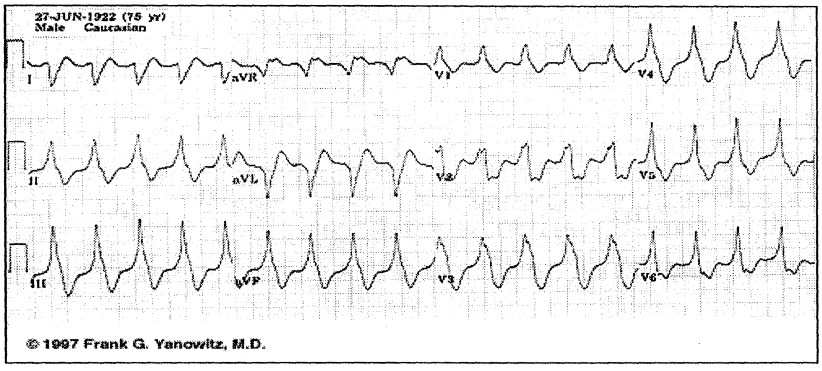
Ventricular Premature Contractions (VPC)

VPCs are premature depolarization arising in the ventricle. They are premature and occur before the next normal beat is expected. The QRS complex is wide. A full compensatory pause always follows a VPC, when the basic rhythm is sinus. The duration of QRS complex is 0.12s or more. A VPC originating from the left ventricle resembles the QRS complex of LBBB pattern and a VPC from the right ventricle resembles RBBB pattern. A VPC from the septum shows positive QRS complexes in right and left leads. The T wave and QRS complexes usually point in opposite directions. VPCs usually precede a P wave. Occasionally they appear just after a P wave before the QRS complex. Sometimes they are followed by a negative P wave because of the retrograde conduction. VPCS may occur in various combinations. If each normal sinus impulse is followed by a VPC, it is called ventricular bigeminy. If a VPC occurs every after two-sinus beat it is called ventricular trigeminy.



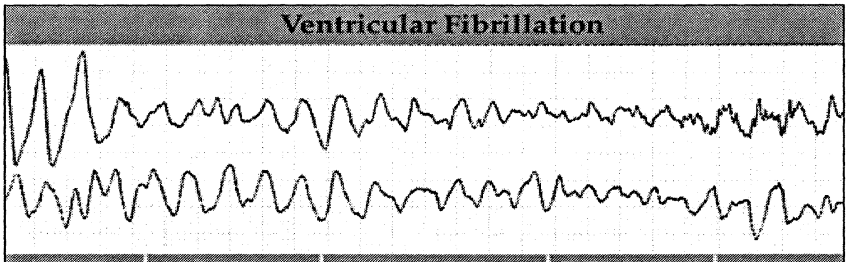
Ventricular Tachycardia (V T)

Ventricular tachycardia is the run of six or more consecutive VPCs. Ventricles beats rapidly unrelated to the atrial mechanism, and the QRS complex is wide and bizarre. VTs are classified according to their duration as sustained and non-sustained VT. Sustained VT lasts for 30 seconds or more with symptoms syncope or near syncope. Non-sustained VT lasts for less than 30 s. VTs are characterized by wide QRS complexes and unrelated to the atrial mechanism. When QRS complex is narrow and has incomplete right bundle branch block pattern, it is considered to be fascicular VT of the left bundle branch. The rate VT is 180-250 usually. VTs are also characterized as monomorphic or polymorphic, depending whether the consecutive VPCs have the same or variable appearances in a single lead.



Ventricular Fibrillation (V F)

With VF the ventricles do not beat in synchronized fashion, but they fibrillate asynchronously. The ECG of VF shows characteristic fibrillatory waves with an irregular pattern that may be fine or coarse. In VF the ventricular rate is 250-600 beats/min.



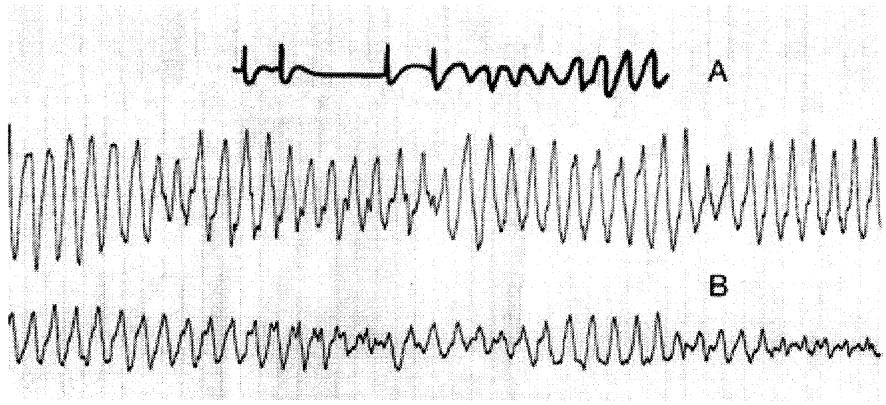
Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
300-600	Extremely irregular	Absent	N/A	Fibrillatory baseline

R on T phenomenon

R on T phenomenon refers to VPCs that that are timed so that they fall near the peak of preceding T wave of normal beat. Those falling on the T wave may cause VT or VF in the course of acute MI or with very long QT intervals.

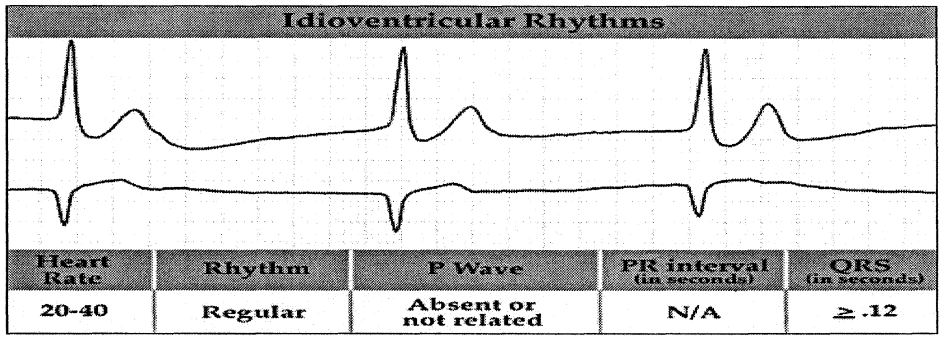
Torsade de Pointes

They are distinct type of polymorphic VTs. Characteristically torsade de pointes is manifested by a long QT interval, polymorphous QRS complexes with varying R-R intervals and fluctuating QRS axes. Ventricular tachycardia is often initiated by an ectopic.



Ventricular escape rhythm

Ventricular escape beat may originate from any part of the ventricle. When six or more ventricular escape beats appears, it forms idioventricular escape rhythm. The QRS complex of ventricular escape beat is wide as 0.16s. The usual rate is 30 to 40 beats per minute.



ECG Of Drug Toxicity and Electrolytic Imbalance

Digitalis Toxicity

Digitalis refers to a class of cardiac drugs with specific effect on the electrical and mechanical functions of the heart. In patients with congestive heart failure it increases the strength of myocardial contractions. It affects the conductivity of the heart. Thus its ant arrhythmic action results primarily from slowing of conduction through the AV node as the result of an increase in vagal tone. Consequently digitalis is used for controlling the ventricular response in atrial flutter and atrial fibrillation. It is also used to treat certain reentrant type of paroxysmal supraventricular tachycardia. But it has a relatively low

margin of safety. The difference between therapeutic and toxic concentrations is very narrow. Digitalis induced cardiac arrhythmias are given below.

A-V Junctional tachycardia, Ventricular premature contractions, Sinus bradycardia, Mobitz type I A-V block, Atrial tachycardia with varying A-V block, Atrial premature contractions, Sinoatrial block or sinus arrest, Complete A-V block, A-V Junctional escape rhythm, Atrial flutter or fibrillation, Ventricular tachycardia or fibrillation.

Certain cardiac arrhythmias are not caused by digitalis toxicity. The non digitalis induced cardiac arrhythmias are given below

Sinus tachycardia, Paroxysmal A-V Junctional tachycardia, Mobitz type II A-V block, Hemiblock, bundle branch block, Bilateral bundle branch block, Complete A-V block.

Digitalis Effect

Therapeutic usage of digitalis shortens the Repolarization time of the ventricles, which shortens QT interval and is associated with a characteristic scooping of the ST-T complex. This phenomenon is called digitalis effect and it is different from digitalis toxicity. Not all patients may show digitalis effect.

Electrolyte Disturbances

Variations in serum concentrations of potassium and potassium can produce marked changes in the ECG.

Hyperkalemia

Hyperkalemia produces changes in both ventricular depolarization and Repolarization. The normal serum potassium concentration is 3.5 to 5 mEq/L. the first change seen with elevated serum potassium concentration is narrowing and peaking of the T waves. The T waves will become tall and they have a characteristic tented shape. With the further increase in potassium, PR intervals become prolonged and the P wave disappears. The QRS complex widens and undulating sine wave pattern of asystole is the lethal stage of Hyperkalemia.

Hypokalemia

With hypokalemia the most common pattern seen in ECG is ST depressions with prominent U waves and prolonged repolarization. The U waves become enlarged and may exceed the height of the T waves.

Hypercalcemia

Ventricular repolarization is shortened by Hypercalcemia, which is reflected as short QT interval in the ECG, due to shortened ST segment. With marked hypercalcemia the T wave appears to take off from the end of the QRS complex.

Hypocalcemia

Hypocalcemia prolongs the QT interval, primarily because of lengthening of the S-T segment. Less commonly flattening or inversion of the T wave may be see. Significant cardiac arrhythmias other than premature beats are not caused by hypocalcemia.

Hypothermia

With hypothermia ECG shows hump like elevation localized to the junction of the end of the QRS complex and the beginning of the ST segment (J point). These waves are called as Osborn waves, which disappears with warming.

Pericarditis

ECG pattern seen with pericarditis resemble those seen with acute MI. The early phase of pericarditis is characterized by ST segment elevation. Major difference between these elevations and those occurring with MI is their distribution. The ST elevations with acute MI are characteristically limited to anterior or inferior leads, showing localized area of infarct. As the pericardium envelops the heart, the ST segment changes occurring with pericarditis are there for more generalized. ST elevations are seen in both anterior and inferior leads. Pericarditis may affect atrial repolarization also, which is reflected as elevation of PR interval in lead aVR and depression of PR interval in other extremity leads and the left chest leads. So in acute pericarditis the PR and ST segments typically point in opposite directions. The important difference between the ECGs of MI and pericarditis is, with MI abnormal Q wave appears due to necrosis of the heart muscle. Abnormal Q waves are never resulted by pericarditis.

Pericardial Effusion

The most common ECG sign of pericardial effusion is low voltage QRS complexes, due to the short-circuiting of cardiac voltages by the fluid surrounding the heart. Low voltage is said to be present when the total amplitude of the QRS complexes in all the extremity leads is 5mm or less. Electrical alterans is another the pattern that can occur with pericardial effusion and tamponade. This pattern is characterized by a beat-to-beat shift in the QRS axis associated with mechanical swinging of the heart in a large accumulation of the fluid.

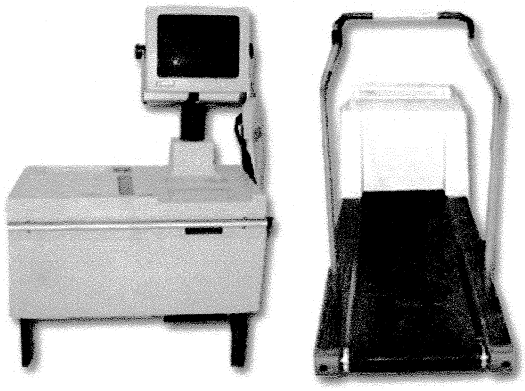
Myocarditis

ECG findings with Myocarditis resembles with that of MI, with ST elevations and Q waves. Atrial or ventricular arrhythmias can occur with Myocarditis.

Pulmonary Embolism

With pulmonary embolism ECG may show sinus tachycardia, ventricular ectopy or atrial arrhythmias. A right ventricular strain pattern (T inversions in V1-V4), with QRS axis shift to right. ST segment depression may be seen indicating subendocardial ischemia. An incomplete or complete bundle branch block pattern may be seen in lead V1.

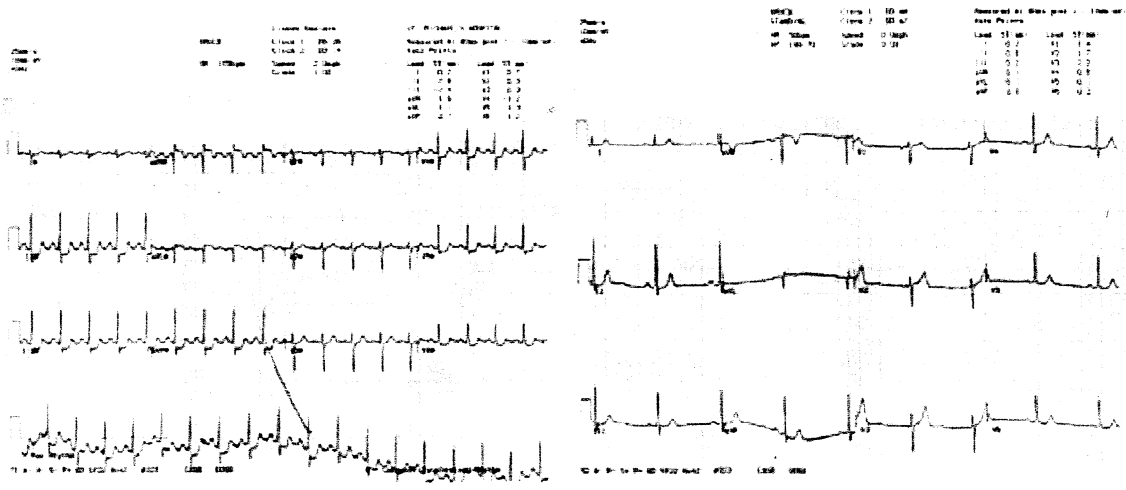
EXERCISE ELECTROCARDIOGRAPHY



Exercise electrocardiography is one of the most important and valuable non-invasive diagnostic test in the clinical evaluation and management of patients with coronary artery disease. Different modalities of exercise ECG test treadmill test and bicycle ergo meter, which are most common. Other forms of exercise test have been evaluated such as induced hypoxia, isometric exercise and atrial pacing. These exercise have not got wide popularity because of difficulties in performing and standardization as well as lack of sensitivity. At present various multistage exercise protocols have been developed for exercise ECG test, using a motor driven treadmill.

The exercise ECG has two roles. One role is to determine whether the coronary circulation is capable of increasing the oxygen supply to the myocardium in response to increased demands. During physical exercise myocardial oxygen demands are increased by the increment of systolic blood pressure, the contractile state and the heart rate. The other role of exercise ECG test is to assess the exercise capacity.

The heart extracts 70% of the oxygen from each unit of the blood perfusing the myocardium at rest, so that the oxygen delivery can't be significantly increased by increased extraction. Thus coronary blood flow must increase in order to meet myocardial oxygen demand. In patients with coronary artery disease, coronary blood flow fails to increase adequately to meet the demands of the myocardium for oxygen leading to myocardial ischemia, which can be manifested by chest pain, ST-T wave changes etc.



Preparation and Precaution for Exercise Test

Patient preparation, Complete patient history, Careful consideration of indications and contra indications, Careful consideration of various factors that may cause a false positive or false negative TMT, Careful consideration of the various drug effects and electrolyte imbalance, Basal ECG before the test, Full instruction about the procedure to the patient, Proper skin preparation and electrode placement, Periodic recording of blood pressure during the test, Supervision of the test by a physician, Stop the test when acute symptoms, serious arrhythmias, significant ST changes or Hypotension occurs, Treat the patient for serious arrhythmia or symptoms as needed, Ready availability of cardiopulmonary resuscitate equipments and drugs.

Protocols for exercise ECG test

For an ideal exercise test initial workload should be well within the anticipated physical working capacity and workload should be increased gradually. In some exercise protocols the workload is increased by changing the speed alone with a fixed grade (elevation). Where as in some other protocols grade is increased with fixed speed. For the progressive increment in the workload at least 3 min interval is preferable so that steady state blood pressure and heart rate responses can be achieved. Commonly used protocols are Balke, Bruce, Ellestad, Naughton, and McHenry. There is no significant difference between these tests at maximum oxygen intake (VO2), heart rate and blood pressure.

Some of the exercise protocols are shown in table given below

BRUCE PROTOCOL

Stage	Duration	Speed	Grade	Mets
1	3	1.7	10	5
2	3	2.5	12	7
3	3	3.4	14	10
4	3	4.2	16	13
5	3	5.0	18	16
6	3	5.5	20	19
7	3	6.0	22	22

MODIFIED BRUCE PROTOCOL

Stage	Duration	Speed	Grade	Mets
1	3	1.7	0	1.7
2	3	1.7	5	2.8
3	3	1.7	10	5.4
4	3	2.5	12	7
5	3	3.4	14	10
6	3	4.2	16	13
7	3	5.0	18	13

NAUGHTON'S PROTOCOL

Stage	Duration	Speed	Grade	Mets
1	2	1	0	1
2	2	2	0	2
3	2	2	3.5	3
4	2	2	7	4
5	2	2	10.5	5
6	2	2.5	14	6
7	2	2	17.5	7

Electrocardiographic measurements

The extremity leads in standard 12 lead ECG should be moved to torso to reduce motion artifact. This modification is known as Mason-Likar modification. The arm electrodes are placed in the most lateral aspects of the infra clavicular fossae. The leg electrodes are placed above the anterior iliac crest, below the rib cage. Mason-Likar modification results a right axis shift and increased voltages in the inferior leads, and may produce loss of Q waves in inferior leads.

When the maximum exercise test is compared with the sub maximum exercise test, there appears to be no significant difference in the clinical and practical sense. METS or metabolic equivalents are the multiple of the basal metabolic rates, are commonly used to express the workloads in several of the exercise protocols. In most of the patients with coronary artery disease workloads with 8 METS are sufficient.

INDICATIONS FOR EXECISE ECG TEST

Diagnostic purpose, Confirmation of the diagnosis of CAD, To find out the etiology of chest pain, Assessment of nature of cardiac arrhythmias in relation to exercise, Evaluation purposes, Evaluation of the functional capacity of patients with CAD, Evaluation of efficacy of medical treatment for CAD, Evaluation of efficacy of surgical therapy, Evaluation of prognosis in patients with previous myocardial infarction, Research purposes, Evaluation of antianginal drugs, Evaluation of antiarrhythmic drugs, Evaluation of exercise responses in various cardiovascular disorders.

CONTRA INDICATIONS

Absolute indications, Acute M I, Unstable angina, Serious cardiac arrhythmias, Acute myocarditis or pericarditis, Severe AS, Acute or sever CHF, Acute pulmonary embolism, Relative contra indications, Clinically significant non cardiac disorders, Significant physical handicaps, Mentally unstable or uncooperative patients, Sever anemia or fever, Moderate-severe hypertension, Moderate AS, Various cardiac drug effect or electrolytic imbalance, Fixed rate artificial pacemaker.

INDICATION TO TERMINATE THE TEST

Absolute indications: Patients request, Reduction of heart rate and blood pressure during increased workloads, Significant symptoms or signs as chest pain, ataxia, confusion, cyanosis, Serious arrhythmias: grouped VPCs, VT, VF, Acute MI, Malfunctioning of the equipment. Relative indications: Less serious symptoms, significant chest pain, dizziness, marked fatigue or dyspnea, severe anxiety, leg cramp. Marked (2mm or more) horizontal or downslopping ST segment depression or marked upslopping ST segment elevation. Marked hypertension i.e. systolic b p > 220mmHg or diastolic b p > 110mmHgFailure of b p to raise during increasing workloads (systolic b p rise less than 20mmHg during the first three stages). Frequent VPCs. Persisting supra ventricular tachycardia.

CRITERIA FOR A POSITIVE TMT TEST

Horizontal or upslopping ST segment depression ≥1mm, Horizontal or upslopping ST segment elevation ≥ 1mm

Various kinds of ST segment responses

ST depression

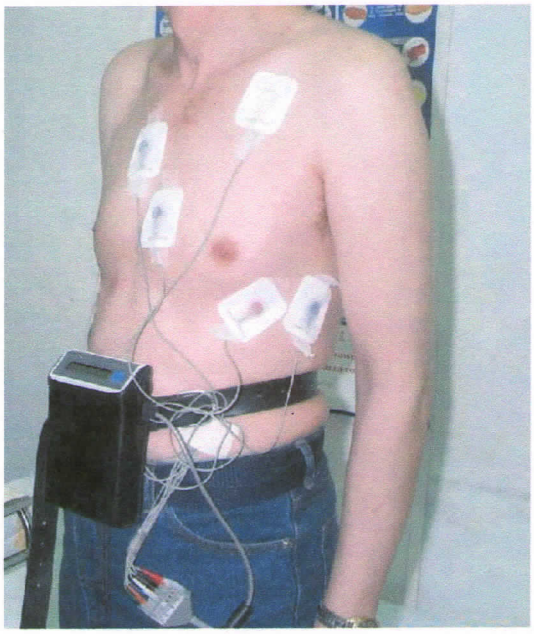
Horizontal, Functional (J point), Down slopping, Up slopping, Slow rising, Sagging.

ST elevation

Horizontal, Upslopping.

HOLTER ELETROCARDIOGRAPHY

The Holter technique utilizes a precordial lead system, which is connected to a portable electrocardiograph monitor. The ECG signals are recorded on a magnetic or semiconductor type recorder, which records 24 hours of ECG. Holter monitor has the sensitivity to detect various cardiac arrhythmias and S-T, T wave changes.



Indications for Holter monitoring

The primary indication for Holter monitoring is to detect arrhythmias. The recorder can be used to identify the episode of angina by the St segment and T wave alteration. The recorder can also be used to evaluate the occurrence of extra systoles or R on T phenomenon. Another important use of Holter monitoring is in the evaluation of antiarrhythmic therapy, by determining whether the incidence of treated arrhythmias have decreased or abolished by the given drug. In patients with permanent pacemakers, Holter monitor can be used to evaluate the status of the pacemaker function.

1. Diagnosis of arrhythmias

Premature contractions, Tachy arrhythmias, Brady arrhythmias, Sick sinus syndrome, Conduction disturbances, W-P-W syndrome.

2. Evaluation of various symptoms to correlate with arrhythmias

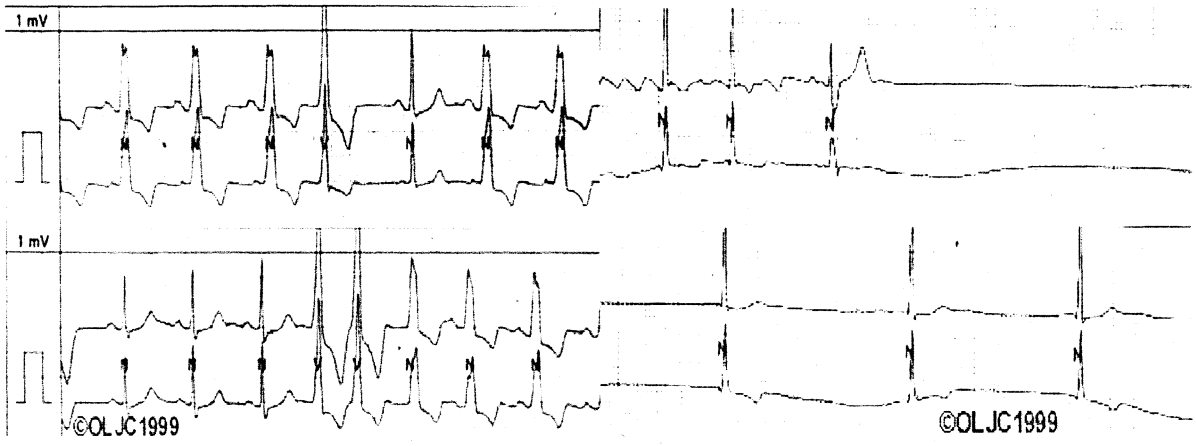
Syncope or near syncope, Lightheadedness, Palpitations, Irregular pulse, Chest pain, Dyspnea, Myocardial ischemia, ST, T wave changes, Classical angina, Atypical angina.

3. Evaluation of antiarrhythmic drug therapy

Efficacy of drugs, Toxicity of drugs.

4. Evaluation of Artificial Pacemaker

Assessment of normally functioning pacemaker, Diagnosis of malfunctioning pacemaker, Slow pacing, Irregular pacing, Failure of sensing or capture, Pacemaker induce arrhythmias.

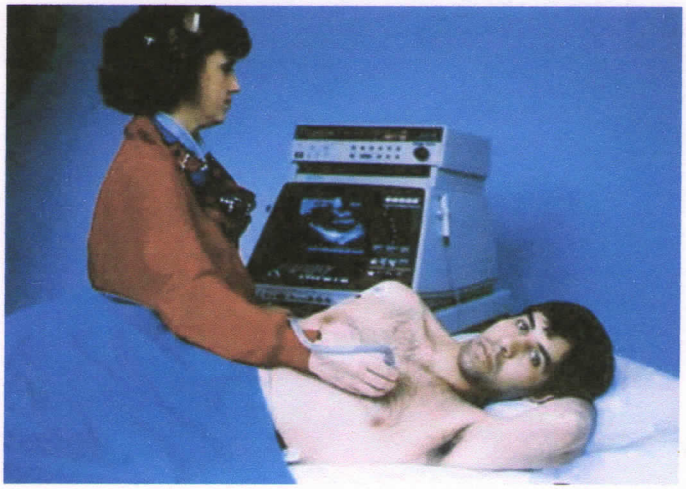


Technical Considerations

Holter monitor system utilizes a bipolar electrode system, consists of three or more electrodes. The exploring electrode (red), indifferent electrode (white), and ground (green). The general application is a bipolar modification of lead V4 or V5. This usually provides a good way of analyzing P, QRS, S-T and T wave abnormalities. QRS complex is upright when this lead system is used. The exploring lead is placed over the fifth rib in the left midclavicular line. For cardiac rhythm analysis a modified form of lead V1 is used. The exploring electrode is placed over the lower sternum, indifferent electrode over the upper sternum, and the ground electrode over the fifth rib in the right midclavicular line.

ECHOCARDIOGRAPHY

Introduction

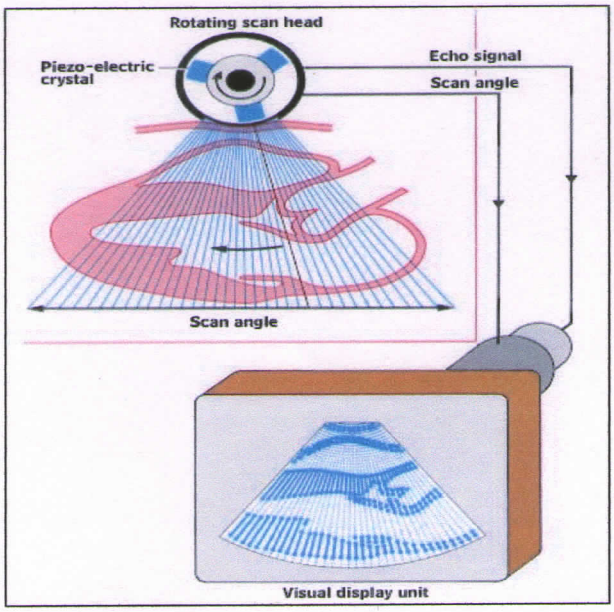


Echocardiography is a unique noninvasive method for imaging the living heart. It is based on detection of echoes produced by a beam of ultrasound (very high frequency sound) pulses transmitted into the heart.From its introduction in 1954 to the mid 1970's, most echocardiographic studies employed a technique called M-mode, in which the ultrasound beam is aimed manually at selected cardiac structures to give a graphic recording of their positions and movements. M-mode recordings permit measurement of cardiac dimensions and detailed analysis of complex motion patterns depending on transducer angulation. They also facilitate analysis of time relationships with other physiological variables such as ECG, heart sounds, and pulse tracings, which can be recorded simultaneously.

A more recent development uses electromechanical or electronic techniques to scan the ultrasound beam rapidly across the heart to produce two-dimensional tomographic images of selected cardiac sections. This gives more information than M-mode about the shape of the heart and also shows the spatial relationships of its structures during the cardiac cycle. A comprehensive echocardiographic examination, utilizing both M-mode and two dimensional recordings, therefore provides a great deal of information about cardiac anatomy and physiology, the clinical value of which has established echocardiography as a major diagnostic tool.

Instrumentation

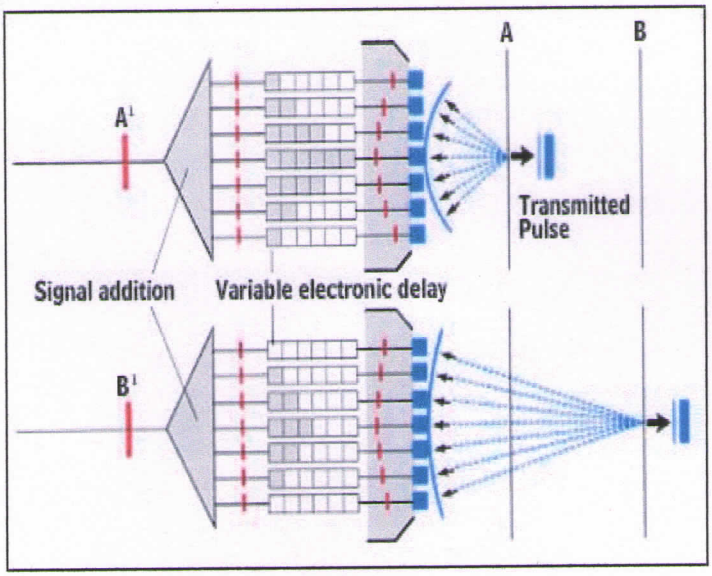
Mechanical Sector Scanners



In the M-mode technique, all the ultrasonic pulses are propagated along the same axis and different parts of the heart are studied by changing the direction of the beam manually. An M- mode echocardiogram is not a "picture" of the heart, but rather a diagram that shows how the positions of its structures change during the course of the cardiac cycle. It is an admirable method for studying a structure like a heart valve, but it does not provide information about the spatial relationships of different parts of the heart to each other. However, this can be accomplished by scanning the ultrasound beam rapidly back and forth across a section of the heart.

Because access to the heart afforded by the ribs and lungs is very limited, almost all cardiac scanners are of the sector type. A mechanical sector scanner can use either an oscillating or rotating scan head. In the rotating type several transducers spin inside a small dome filled with liquid. As each one passes over the heart, it transmits pulses and receives echoes. The echo signals are displayed in B-mode form. Signals from the scan head are used to steer the oscilloscope beam in the same manner as the ultrasound beam. The result is a tomographic image of the heart, showing the structures in the selected scan plane and their motion patterns.

Phased Array Sector Scanners



The ultrasound beam can also be steered electronically, without moving the transducer. Electronically steered, or "phased array" systems typically comprise 96 to 128 small elements (only a few are shown), which are pulsed in a very rapid, precisely controlled sequence. The top element is pulsed first because it is very small, the ultrasound wave it generates is circular. Very soon afterwards, the second element is pulsed, and so on. The individual wavelets combine to make one compound wave that, because of the pulsing sequence, travels at an angle to the axis of the transducer array. Returning echoes do not reach all the transducer elements simultaneously; electronic circuits delay the signals from those arriving first, allowing the remainder to catch up.

Continuously changing the pulsing sequence scans the ultrasound beam in a manner similar to a mechanical scanner. Despite the necessary complexity of the electronic circuitry the phased array technique offers methods for reading the effective beam width not possible with mechanical systems. This is a very important factor in improving image quality. Focusing can be achieved by fitting a plastic lens over the face of the transducer. Phased array systems can provide additional focusing electronically; a lens works by delaying portions of the wavefront and a phased array can achieve the same effect electronically by further modifying the pulsing sequence.

A phased array system can also employ a technique called "dynamic focusing" If a pulse is transmitted across two interfaces, A and B, the echo from A returns first. Its curved wavefront reaches the center transducer elements before those at the edges. The electrical signals from the central elements are delayed to allow those from the edges to catch up. All the signals are then added together (A1). A few microseconds later, echoes from B arrive. This wavefront is less curved, so the delay pattern is altered. In this way the receiver changes its focal distance as echoes from more distant structures arrive. This technique rejects off-axis echoes that reduce effective beam width.

Quality of Images

A single two-dimensional scan comprises a number of radial lines, each displaying echoes in B- mode format. For accurate images both the echoes shown on each B-mode line should accurately reflect the structures encountered by the ultrasound beam, and the two-dimensional image should contain the maximum possible number of lines. Because the ultrasound beam is not a fine, laser-like line, objects that lie off-axis are detected and generate artifactual multiple echoes. This greatly impairs lateral resolution, the major limiting factor in B-mode image accuracy. Reduction of beam width by focusing techniques is therefore necessary to make the beam as narrow as possible. The number of B-mode lines available to form a two- dimensional image is determined by the velocity of ultrasound in the body.

For a 20 cm depth the maximum pulse rate is 3750 per second. Also, the structures being imaged must not move significantly during the period of the scan. A cardiac scan must therefore be completed in 1/60th of a second or less depending on power supply characteristics to 'freeze' valve motions, and a succession of such images made for each cardiac cycle (1 second) to show the motion patterns. If 60 images are made each second, only 75 lines gives 2.5 lines per degree, but if increased to 90 degrees, there is less than one line per degree. Sector scanning systems must therefore compromise between scans per second, scan angle, and line density. summarizes two beams. With poor lateral resolution, multiple images are formed of A and some echoes from B are also detected.

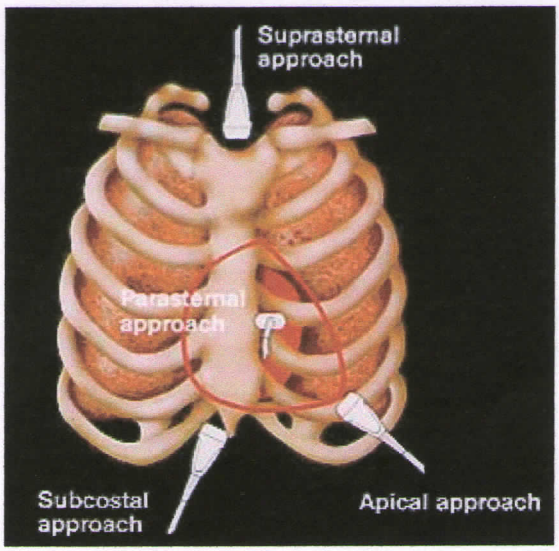
Recording a Display

The simplest method of recording a two-dimensional display is to focus a TV camera on the oscilloscope image and record it on a standard video tape recorder. However, this does not give very good image quality, partly because of the double conversion from electrical to visible image, then back to electrical before obtaining the final visible image, and also because of interaction between the radial scan lines forming the sector image and the parallel lines used for TV. Another possibility is to use a movie camera, but film is expensive and processing it takes time. Furthermore, unless the movie camera shutter is synchronized to the oscilloscope scan rate, there will be unsightly flickering or blank bars across the picture. A much better method is to change the sector image into a TV format by means of a digital scan converter. This comprises a matrix of electronic memory cells (typically 512 x 512 elements). The image intensity at each point on the waveform is assigned a specific number by which is it expressed in digital form on a scale of 0-64 grey levels and its value stored in the appropriate memory cell. The memory matrix can then be "read" in any sequence.

Images from a scan converter can be transferred directly onto videotape. For quantitative analysis, selected scans can be photographed or measured directly from the oscilloscope screen.It is sometimes useful to be able to record M-mode and two-

dimensional images simultaneously. An electronic cursor superimposed on the display is adjusted to the desired position and the appropriate B-mode lines are printed on an M-mode strip-chart recorder. Unfortunately, with a mechanical sector scanner only one such line is available for each two-dimensional scan field (typically 60 per second) and the resulting M-mode recording quality is poor.

Access to the Heart for Echocardiography

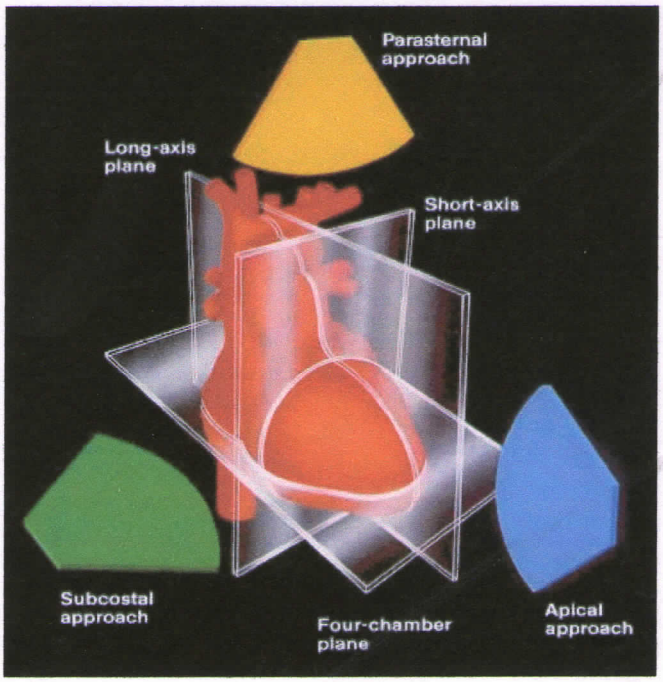


The cardiac outline extends from the junction of the second left costal cartilage with the sternum at the sternal angle to the apex beat, usually in the fifth intercostal space on the midclavicular line. From the apex, the inferior surface of the heart runs across to a point in the right fifth intercostal space about 1 cm from the sternal edge, then it parallels the sternum to a point 1 cm to the right of its junction with the third right costal cartilage. The base of the heart is marked by a line joining the right third costal cartilage and the left second costal cartilage. The left lung does not cover the heart completely and in most individuals beneath the third, fourth and fifth intercostal spaces, for 2 or 3 cm to the left of the sternal border, the pericardium lies directly beneath the chest wall and pleural membranes. This region, termed the left parasternal area, provides the best access for echocardiography. Moreover, it lies over the center of the heart, and the distance from the chest wall to the furthest part of the normal heart is only about 12 cm.

Additional access, important particularly for two-dimensional echocardiography, can usually be obtained from the cardiac apex and by a subcostal route, with the transducer placed near the xiphisternum. The great arteries and the base of the heart can also be visualized from the suprasternal notch. The approaches described above are

available, to a greater or lesser degree, in most adults. In young children, the ribs and lungs do not attenuate the ultrasound beam so severely, and in neonates the transducer can be placed almost anywhere on the precordium. By contrast, in adults with "barrel" chests, or who have hyper inflated lungs, for example as a result of chronic emphysema, it can be almost impossible to obtain any echocardiographic images. Lack of adequate access to the heart is the greatest limitation to echocardiography and a large proportion of the technical skill required to perform the examination lies in being able to find a transducer site from which clear images can be obtained.

Standard Planes for Two- Dimensional Echocardiography

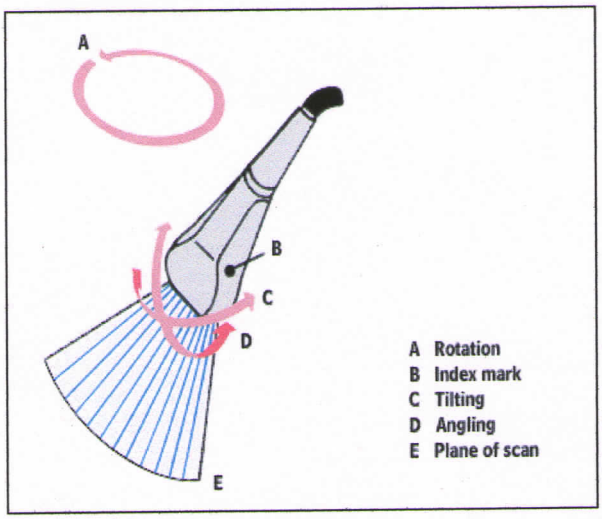


Even with the limited access to the heart afforded by the left parasternal, apical, and subcostal transducer positions, it is possible to direct an ultrasound beam at most cardiac structures. It has been agreed that two-dimensional echocardiographic images should be based on three orthogonal planes. The long-axis plane transects the heart from the aortic root to the left ventricular apex and includes the aortic and mitral valves. On the surface of the body, it is almost perpendicular to the plane of the sternum and runs approximately from the subject's right shoulder to the left kidney. The short axis approximates to the plane of the atrioventricular junction. On the surface of the body, it is at right angles to the plane of the sternum and runs from the left midclavicle to the right hip. The four-chamber plane is at right angles to both the long and short axes. It runs from the apex to the base of the heart and is approximately perpendicular to both the posterior interventricular septum and the interatrial septum. As its name implies, it includes parts of each of the four cardiac chambers. On the surface of the body, it is parallel to the plane of the sternum, and includes both the apex and the right shoulder.

As can be appreciated from the three best echocardiographic approaches to the heart correspond approximately to the points at which the three standard planes intersect. Thus, from the left parasternal transducer position, it is possible to examine the long-axis and short-axis planes. From the apical position, the long-axis and four-chamber

planes can be visualized, and from the subcostal position, the four-chamber and short-axis planes.

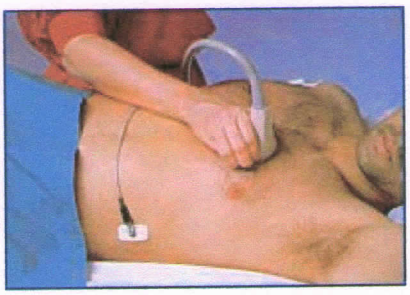
Transducer Manipulation



Much of the skill needed to perform an echocardiographic examination lies in being able to direct the ultrasound beam at cardiac structures of interest in such a way that echoes from them can be detected. The principles of cardiac anatomy and recording technique relevant to M-mode apply equally to two-dimensional echocardiography. The stationary ultrasound beam used for M-mode can be thought of as a flashlight, illuminating only a small area of the heart at a time; a two-dimensional transducer is like a circular saw, rotating around the point where it rests on the chest, with the blade aligned with the index mark on the transducer. The side of the transducer with the index mark corresponds to the right-hand side of the display. When performing a two-dimensional examination, therefore, more complex maneuvers are necessary to order to align the scan plane with the desire anatomic axis of the heart. Manipulation of the transducer can be described as follows. Rotating the transducer pivots the scan plane about the transducer axis. Rotation through 90 degrees is used, for example, to change from the parasternal long axis to the short axis.

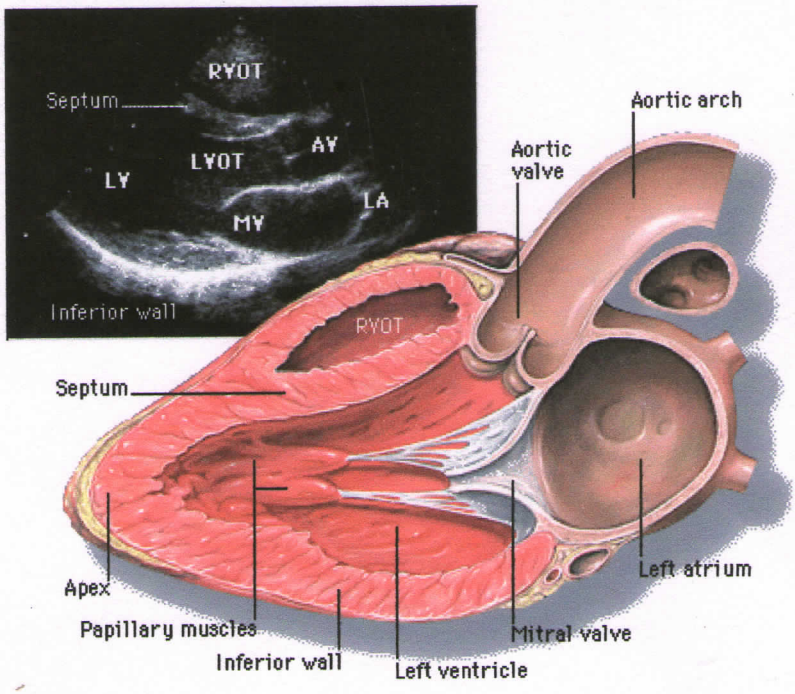
Different Views in Echocardiography

Parasternal Views



To obtain parasternal views, the subject is inclined slightly toward the left lateral position. The transducer is placed over the intercostal space as for an M-mode examination. For the long-axis view, the scan plane is aligned from the right shoulder to the left kidney, with the transducer index mark toward the shoulder so the aorta will appear on the right-hand side of the display. From this position, the transducer is rotated clockwise through 90 degrees to obtain short-axis views (the index mark should now point toward the left midclavicle). Since the right- hand side of the short-axis scan tends to be obscured by lung, the transducer should be positioned as close to the sternal border as possible. It may help to turn the subject more toward the left, and to record during forced exhalation. In difficult subjects, such maneuvers may be necessary even to obtain minimal visualization of cardiac structures.It is normal to obtain a series of parasternal short-axis views, from the apex to the pulmonary artery, by tilting the transducer along the line of the long axis. When the transducer is aimed toward the apex, a tomographic section is obtained at the level of the papillary muscles. Progressive tilting up the long axis shifts the section first to the mitral valve, then to the left ventricular outflow tract, the aortic and pulmonary valves, and finally to the main pulmonary artery.

Parasternal Long-Axis Plane



At the top is the outflow portion of the right ventricle, below which the plane cuts across the anterior portion of the interventricular septum. The aortic root is seen at the right, with the right coronary (upper) and noncoronary (lower) cusps of the aortic valve. Below the aortic root is the left atrium. To the left of the section is the apex of the left ventricle, in which the posteromedial papillary muscle can be seen. Separating the left atrium from the left ventricle is the mitral valve. The longer, anterior leaflet arises from the posterior wall of the aortic root, and the shorter, posterior leaflet is attached at the atrioventricular groove; the plane cuts across the coronary sinus at this point.

The ultrasound image through encompasses the region from the papillary muscles to the aortic valve. It clearly shows the motion of the aortic and mitral valves and permits measurement of the left atrial and aortic root dimensions, as well as those of the left ventricular cacity, interventricular septum, and posterior ventricular wall. This view is used for many purposes, including all types of left ventricular outflow obstruction, mitral and aortic valve vegetations, and mitral valve prolapse. It corresponds approximately to the angiographic right anterior oblique view, but reversed right-to-left. Superior angulation of the transducer allows the scan plane to be extended up to the roof of the left atrium and the proximal part of the ascending aorta, which is useful for detecting atrial masses and for aortic root dissection and aneurysms. With counterclockwise rotation of the transducer and slight medial angulation, the right atrium, tricuspid valve apparatus, and proximal portion of the right ventricular inlet are encountered. This is termed the right ventricular inlet view.

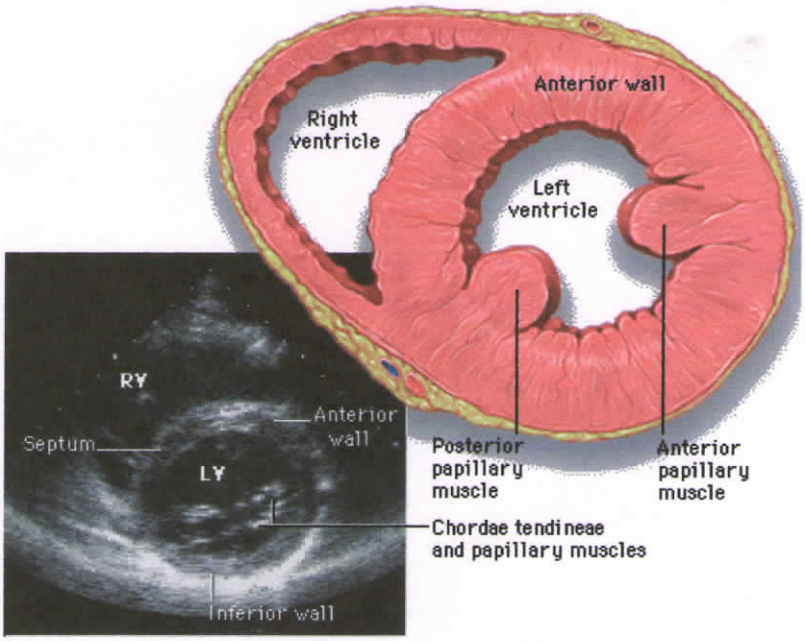
Parasternal Short-Axis Plane

Mitral valve Level

If the transducer is rotated clockwise through 90 degrees from the paraste long-axis view, the short-axis view is obtained. Provided the mitral valve was in center of the sector before rotation, the short-axis section will be at mitral valve le The tomographic section of the heart shows the left ventricle, bounded by thick, musc walls comprising the anterior and posterior portions of the interventricular septum on left and the lateral free wall to the right. Within the left ventricular cavity are the leaflets of the mitral valve, the upper being the anterior and the lower the posterior. the upper left of the left ventricle a portion of the right ventricle is seen as a cresc The section cuts it between the tricuspid valve and the apex of the right ventricle, be the crista.

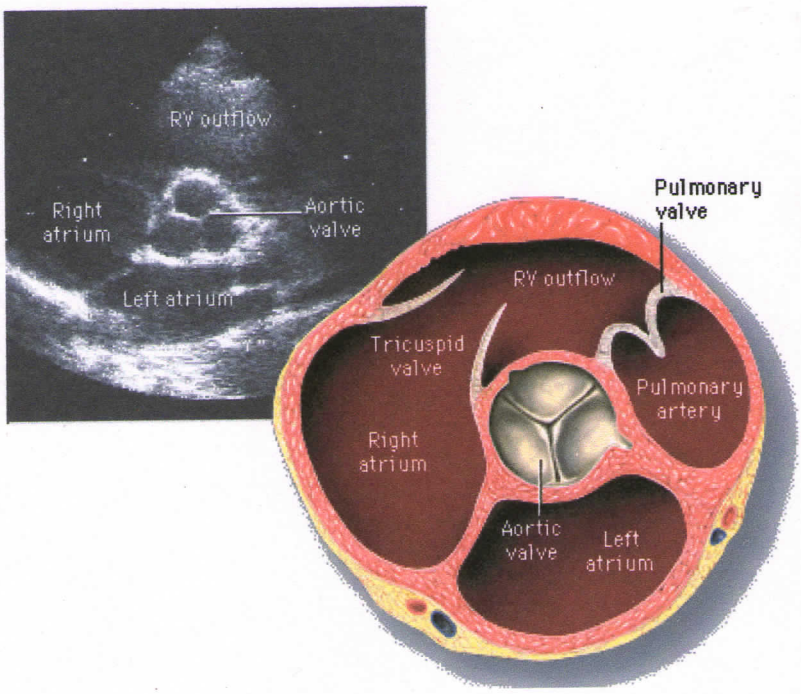
The major feature of the ultrasound image is the motion of the mitral va leaflets that form a shape like the mouth of a goldfish, viewed head-on. The large, cur upper leaflet, and the flatter lower leaflets separate and come together as blood f through the orifice varies. In early diastole, they separate widely during rapid ventricu filling, then partially come together in mid-diastole as the filling rate falls. With the adv of atrial systole, they again separate, before closing completely at the beginning systole, where they remain until the beginning of the subsequent diastole. It is poss from this view to estimate mitral valve orifice area, a measurement that has been sho to correlate well with the actual area assessed at surgery. It therefore forms probably best echocardiographic method for determining the severity of mitral stenosis.

Papillary Muscle Level



If the transducer is tilted toward the apex, the ultrasound beam scans a lower plane. At this level, the left ventricle is sectioned across the middle of its two papillary muscle groups. These are normally easy to visualize and they form a reliable means of identifying ventricular morphology, since the right ventricle has only one prominent papillary muscle. The right boundary of the left ventricle is formed by the lateral free wall and the bottom of the section by the inferior wall. To the left is the muscular interventricular septum. This section cuts the right ventricle very close to its apex. The deep trabeculation in this region may be apparent. In addition to identification of ventricular situs, this view is useful for segmental analysis of wall motion in patients with ventricular disease. For this purpose, the ventricular wall is divided into a number of segments, typically 10 to 15, and the motion and thickening of each is assessed independently. For example, the short-axis views at mitral valve and papillary muscle levels might be divided into five segments each, with an additional four segments derived from the apical long-axis and short-axis views. This provides a disciplined approach to analysis of ischemic disease, but as with all echocardiographic methods, it frequently fails due to technical difficulty in visualizing all the segments adequately.

Aortic Valve Level



Further tilting of the transducer toward the subject's right shoulder, combined with slight rotation clockwise, brings the scan plane above the level of the mitral valve annulus, so that it transects the two atria. In the center is the aorta, with its three cusps visible. Below the aorta is the left atrium, with the interatrial septum running diagonally downward and to the left, dividing it from the right atrium. The plane of the section passes diagonally across the tricuspid valve annulus, above the posteroseptal leaflet, part

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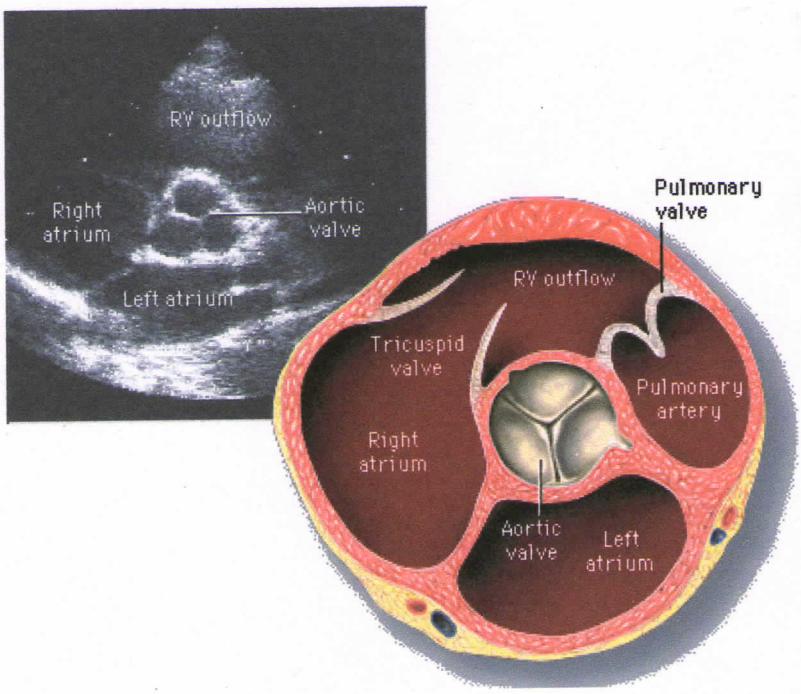

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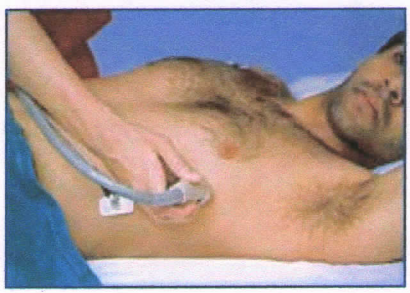
of which is still visible. The outflow tract of the right ventricle arches over the top of the aorta toward the pulmonary valve, the posterior cusp of which is seen.

These features are depicted on the echocardiographic image. There is usually some "dropout" in the atrial septum in the region of the fossa ovalis where it is very thin. While lack of continuity of the atrial septum on the echo display is therefore not diagnostic of an atrial septal defect, this view can be used in conjunction with echocardiographic contrast studies to demonstrate interatrial shunts. Microbubbles, generated by rapid injection of fluid into a peripheral vein, enter the right atrium. Even where the dominant shunt is left to right, there is usually a small amount of flow from right to left at the onset of systole, and this is indicated by passage of a few microbubbles across the interatrial septum into the left atrium.

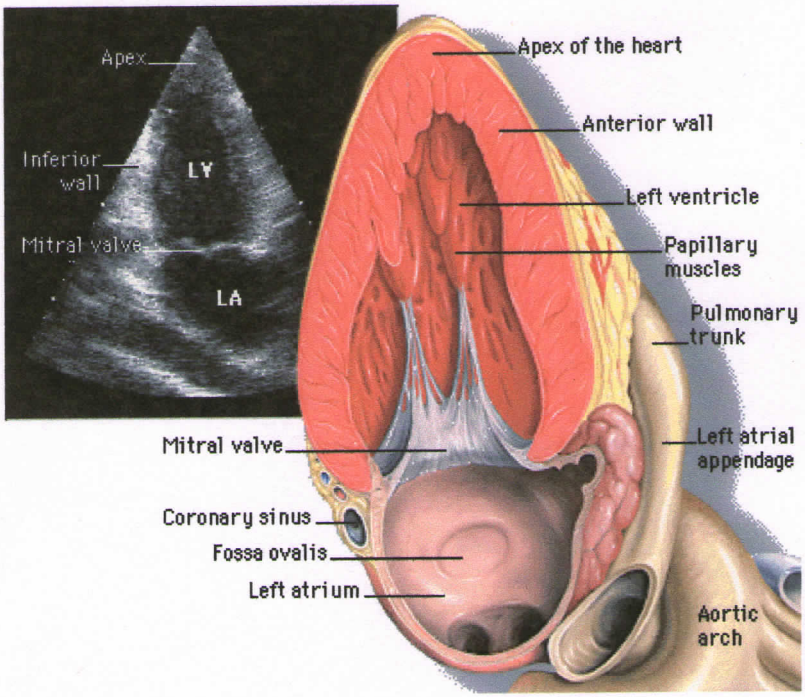
Tilting the transducer slightly further moves the scan plane above the level of the aortic valve. In this position, it is possible to visualize the ostium of the left coronary artery in a substantial proportion of subjects, and that of the right coronary artery in rather less. With further refinements in instrument resolution, it may become possible to detect major stenosis of the proximal coronary arteries with acceptable reliability. Further counterclockwise rotation aligns the scan plane along the main pulmonary artery, seen running posteriorly to the right of the aorta. It is frequently possible to visualize this vessel as far as the bifurcation into right and left pulmonary arteries.

Apical Views

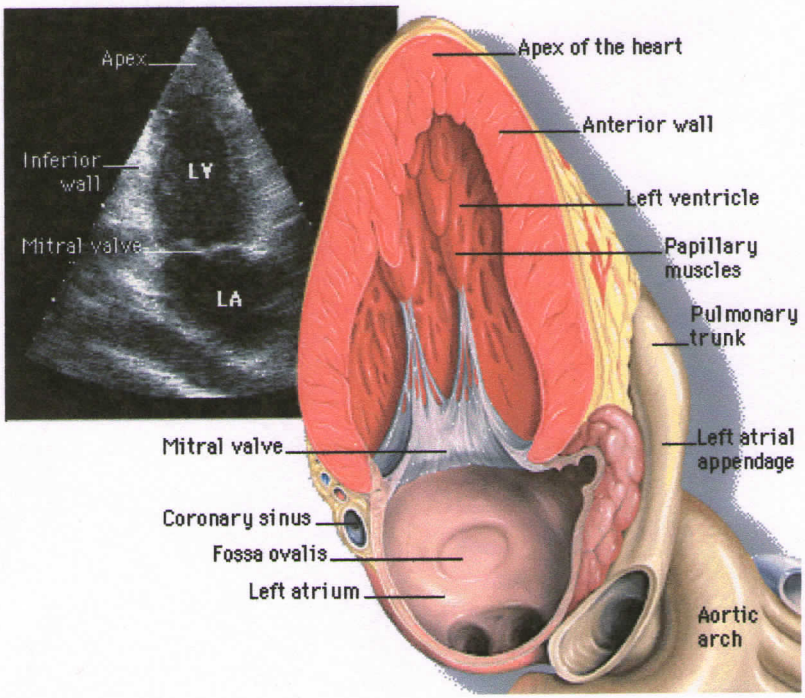


The subject is turned to the left, and the transducer is positioned at the exact point where the maximal apical impulse can be felt, with its axis aimed toward the right shoulder. To obtain a long-axis view, the transducer is rotated so that the index mark points upward. This is a difficult view to obtain in young, slim subjects, because the scan plane is at a right angle to the direction of the ribs and the latter act as a "venetian blind", that greatly limits the field of view. If the transducer is rotated 90 degrees clockwise, the index mark points toward the left axilla. The scan plane is now parallel to the intercostal spaces, affording an easier view showing the four cardiac chambers together with the interatrial and interventricular septam.

Apical Long-Axis Plane



This view is, however, not always easy to obtain, particularly in tall, slim subjects. The direction of the scan is at right angles to the intercostal spaces, and since the transducer axis has to be aimed fairly strongly superiorly, the effect is like looking through a nearly closed venetian flind. It is frequently necessary to position the transducer one intercostal space above the apex, which retains the view of the apical area but tends to obscure the more anterior structures, such as the aortic valve.The ultrasound image is illustrated in which shows the apical region of the left ventricle and also the aortic and mitral valves. Both mitral leaflets are seen. The aortic valve in this view presents a similar target to the pulmonary valve when seen from the parasternal position. When the valve is closed, echoes are detected, but as it opens, the cusps fold away parallel to the aorta and no echoes return to the transducer. The primary application of this view is in assessing the function of the apical region of the left ventricle, since it is the region most prone to development of an aneurysm following infarction associated with occlusion of the left anterior descending coronary artery.



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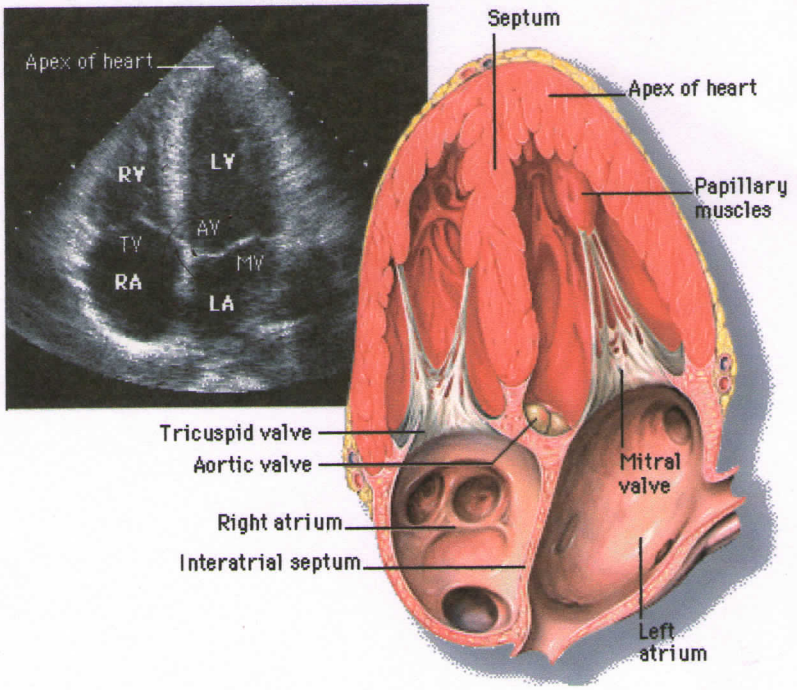
Apical Four-chamber Plane

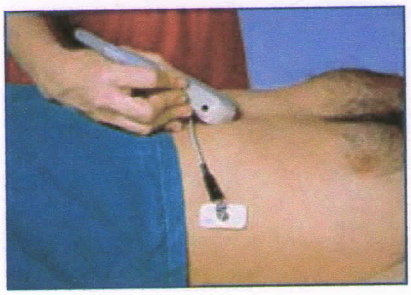


Rotation of the transducer through 90 degrees clockwise from the apical long-axis position turns the scan plane so that it is approximately parallel to the precordium and it passes through all four chambers of the heart. The section shows the interventricular and interatrial septa running down the center, with the left ventricle and atrium to the right, and the right ventricle and atrium to the left. The part of the interventricular septum seen is the posterior septum, whereas the anterior part is seen in the long-axis view. A particularly valuable contribution of echocardiography in the diagnosis of complex congenital heart disease is the ability to identify the cardiac chambers and great vessels. This view is particularly helpful for identification of the two ventricles. In a normal subject, the left ventricle has much thicker walls, but in abnormal hearts this may not be the case, and it is necessary to look for other clues to ventricular morphology. One of these has already been mentioned - the presence in the left ventricle of two distinct papillary muscle groups. The tricuspid valve joins the septum above (i.e., nearer to the apex, and thus below in the body) the mitral. This is because the mitral valve is positioned so that its annulus joins to the septum above the membranous septum, whereas the annulus of the tricuspid valve crosses the membranous septum.

Since the right and left atrioventricular valves are invariably associated with the corresponding ventricles, this provides firm identification of the two ventricles. Further evidence can be obtained from the presence in the right ventricle of the moderator band, and by the more extensive and deeper trabeculation in the right ventricle. Prolapse most commonly affects the posterior mitral leaflet, which occupies two-thirds of the annulus, and is divided into three "mini-cusps". It is most frequently observed in the central of the

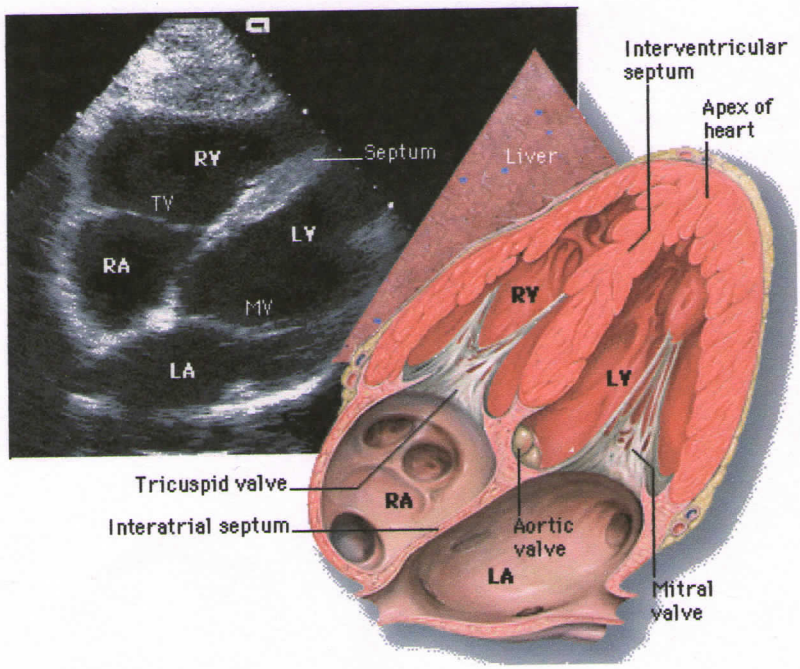
three, presumably because it is less well supported by the chordae tendinae that arise from the papillary muscles beneath the leaflet commissure. The left ventricular outflow tract and proximal portions of the aorta may also be interrogated from the apical approach. With superior angulation of the scan plane from the apical four- chamber view, these structures usually come into view.

Subcostal Views



The subject is placed in a supine position, possibly with a pillow under the kidneys to arch the back upward. The transducer is positioned just to the right of the xiphisternum, with its axis pointing toward the left shoulder and the index mark oriented toward the left hip. It is then pressed down so that the ultrasound beam scans underneath the rib cage in the four-chamber plane. Rotation counterclockwise through 90 degrees, to bring the index mark upward, changes the scan plane to the short axis. Subcostal views are often improved by the subject inhaling; this lowers the diaphragm and pulls the heart down toward the transducer.

Subcostal Four-Chamber Plane



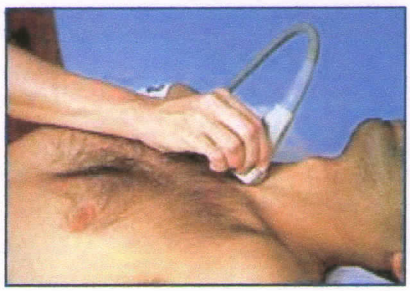
The anatomy of the four-chamber plane seen from the subcostal views is identical to that already described in the apical view. In the body, the inferior surface of the right ventricle, seen at the top, rests on the diaphragm, and the ultrasound beam approaches the heart through a corner of the liver and across the diaphragm. Although the apical region is not seen as well as from the apex, more of the atria are visualized. In addition, clearer echoes are generated by the interatrial and interventricular septam because the ultrasound beam approaches them more nearly at right angles. Thus, while dropout of the echo signals from the septam in the apical view is most likely to be artifactual, consistent absence of echoes from a region of either the interatrial or interventricular septum in the subcostal view suggests the presence of a defect. Two-dimensional echocardiography is helpful for diagnosing these lesions and, in the case of the interatrial septum, it is usually possible to differentiate a defect in the area of the fossa ovalis (secundum type) from a low defect extending down to the mitral annulus (primum) or a sinus venous defect high in the roof of the atria.

The subcostal four-chamber view is the only satisfactory way to visualize the right atrium, and it also affords the best view of the right ventricle, since these chambers like nearest to the transducer. Right atrial morphology can be confirmed by tilting the scan plane inferiorly so that the entrance of the inferior vena cava is seen. In addition to the identification of the atrioventricular valves by their relative levels of attachment to the septum, it is frequently possible to determine ventricular morphology by locating the moderator band within the right ventricle. To a certain extent it is also possible to assess the thickness of the right ventricular walls, and in some patients to diagnose right ventricular hypertrophy. If a pericardial effusion is present, its extent and the degree of separation of the heart from the pericardium should be determined in the subcostal view if pericardiocentesis is contemplated.

Subcostal Short-Axis Views

Short-axis views, similar to those seen from the parasternal position but with the right ventricle or right atrium in the foreground, can be obtained from the subcostal position. However, it is often difficult to produce clear recordings because the transducer must be pressed down in order to scan under the rib cage. In addition, the angle formed by the right and left sixth ribs at the xiphisternum limits transducer mobility. The main application of the subcostal short-axis views is therefore to obtain short-axis images in subjects with hyperinflated lungs where the parasternal approach is impossible. Visualizing cardiac structures from the subcostal approach can also be utilized for M-mode recordings in difficult subjects. The smaller transducer is easier to position beneath the rib cage and with practice most cardiac structures can be visualized. The closest valve to the transducer is the tricuspid, which is seen by aiming the ultrasound beam almost straight up the center line of the sternum. Beyond it is the aortic valve. Slight angulation toward the midclavicular line should reveal the mitral valve. The same direction with the transducer pressed down to align the beam almost parallel with the underside of the rib cage brings the pulmonary valve, farthest from this position, into view.

Suprasternal Views



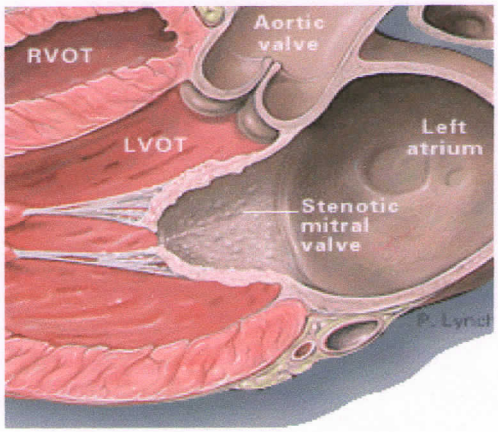
Echocardiographic images of the great arteries can often be obtained by positioning the transducer in the suprasternal notch. The subject lies supine, with the head tilted back over a pillow. If the transducer is positioned so that the scan plane is aligned with the aortic arch, it may be possible to see the ascending aorta passing posteriorly and turning downward, with some of the arteries that supply the head and arms arising from it. Within the loop of the aortic arch, the right pulmonary artery may be seen as a pulsating circular structure.

If the scan plane is rotated through 90 degrees, the aorta will be seen in cross-section, with the right pulmonary artery forming two parallel lines below it. Slight adjustments in scan direction may reveal part of the roof of the left atrium and, if sufficient depth range is available, the mitral valve annulus. Recordings of the aortic arch can be used to diagnose coarctation and to determine both the site of the stricture relative to the subclavian artery and its approximate extent. In patients whose parasternal recordings reveal aortic root aneurysms, knowing whether the enlarged region extends to the origins of the arterial branches can be very helpful as an adjunct to angiography in assessing suitability for surgery.

Although it is not always easy to identify the relevant structures on M-mode recordings, the smaller transducer size and greater depth of penetration allow tracings showing the aorta, right pulmonary artery, and part of the left atrium to be made in the majority of subjects. These are useful in congenital disease for showing the relative sizes of the vessels as a means of assessing severity of shunts, and also in conjunction with contrast injections for identifying the great vessels in patients in whom the combination of transposition and a shunt makes identification from the parasternal position difficult.

Access to the Valves with Echocardiography

Mitral Valve Stenosis



In mitral stenosis, fusion of the commissures between the mitral valve leaflets causes obstruction to left ventricular inflow. The leaflets also become infiltrated with fibrous tissue, which produces shrinkage, thickening and immobility, particularly at the tips of the leaflets. Over a longer period, the fibrotic tissue may calcify and eventually the valve becomes a funnel- shaped structure that impedes blood flow as much by its rigidity as by actual stenosis of the orifice. Chronic elevation of left atrial pressure in mitral stenosis usually causes the chamber to dilate, although the extent to which this occurs depends additionally on atrial wall stiffness. Elevated filling pressure also results in pulmonary venous hypertension, and hence a rise in pulmonary artery pressure. If severe, this leads eventually to right ventricular hypertrophy and the development of tricuspid regurgitation.

On the M-mode recording, thickening of the leaflets and reduction in their mobility can be appreciated. Commissural fusion alters the motion of the posterior leaflet; instead of moving in the opposite direction to that of the anterior, it is pulled forward when the valve opens at the beginning of diastole. In the normal valve, rapid early diastolic filling of the left ventricle means that the valve can partially close in mid-diastole, and it then reopens during atrial systole. This sequence produces the characteristic "M" shape of the recording, with the down slope of the "M" designated as the "E-F slope". With mitral stenosis, however rapid filling is not possible, so the valve leaflets have to remain as widely separated as they can throughout diastole, and the "M" disappears. Another reason for a different pattern of leaflet motion is that atrial fibrillation is frequently present, and this eliminates the late diastolic reopening. The normal left atrial size is below 4 cm in adults and dilates with chronic mitral stenosis, insufficiency, or atrial fibrillation. Patients with severe left atrial dilatation are usually very difficult to convert to normal sinus rhythm with cardioversion. The echocardiogram helps to select patients in which cardioversion might or might not be successful.

Tricuspid Valve Stenosis

Organic tricuspid stenosis is an unusual but important finding. Because of the low pressures in the right side of the heart, significant tricuspid stenosis can produce transvalvular pressure gradients of 1mmHg or less. Echocardiography, in contrast, is a very sensitive method for detecting tricuspid stenosis, and examination of the tricuspid valve should always be included in all patients, particularly in patients with rheumatic aortic or mitral valve disease. An M-mode scan from tricuspid to mitral valve in a patient with both mitral and tricuspid stenosis. Similar changes are evident in both valves, namely reduced mobility, leaflet thickening, and reduced or reversed motion of the posterior leaflet.

Clear two-dimensional visualization of the stenotic tricuspid orifice is not as easy as with the mitral valve. This is due to the lack of an appropriate window by which to access the plane of the open tricuspid valve. In fact, it is a rare patient where the severity of tricuspid stenosis may be planimetered. As with mitral stenosis, the severity of tricuspid stenosis may be estimated by Doppler methods and has been shown to be quite helpful in this regard.

Mitral Valve Regurgitation

Without the use of Doppler, there are no specific signs of mitral regurgitation by either M-mode or two-dimensional echocardiography. The primary echocardiographic features of mitral regurgitation are volume overloading of both the left atrium and the left ventricle. Left ventricular volume overload is recognized by an end- diastolic dimension greater than 5.5 cm, and hyperdynamic wall motion, most noticeable on the interventricular septum. Such profound changes noted by echocardiography require that the hemodynamic load from mitral regurgitation be severe.

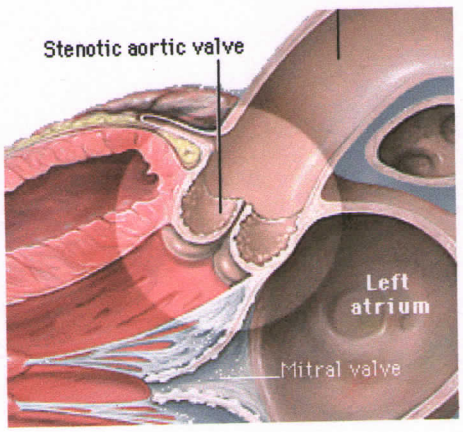
This forms one of the major supports of the whole heart, and the posterior atrial wall consequently moves very little. Expansion of the atrium therefore causes its anterior wall, which is echocardiographically indistinguishable from the posterior wall of the aortic root, to be pushed forward. Thus, vigorous motion of the aortic root indicates high left atrial stroke volume and is a feature of mitral regurgitation (though it is also seen in severe mitral regurgitation in the presence of abnormal ventricular function). Quantification of aortic root motion suffers from the same limitations as measurement of left atrial dimension. Thus, without Doppler echocardiography, the severity of mitral regurgitation can only be deduced from the M-mode or two-dimensional echocardiographic recordings. Direct imaging of the valves, however, frequently reveals the etiology of the regurgitation and provides very useful information for planning surgical approaches. Now that primary valvular reconstruction, rather than replacement is possible, a surgeon experienced in interpretation of echocardiographic data may precisely identify disordered valvular anatomy and predict patient suitability for surgical valvuloplasty.

Tricuspid Valve Regurgitation

As with the mitral valve, there are no specific signs of tricuspid regurgitation on echocardiographic recordings. With the widespread use of Doppler echocardiography it has been learned that small degrees of tricuspid regurgitation are quite common, being present in over 65% of otherwise normal individuals. When tricuspid insufficiency is severe, changes are noted echocardiographically. In the majority of these cases, most functional tricuspid regurgitation is secondary to pulmonary hypertension. Other causes include infective endocarditis and/or abnormalities of the valve itself, such as Ebstein's anomaly.

The primary echocardiographic manifestation of tricuspid regurgitation is volume overload of the right ventricle. The right ventricle becomes enlarged, and the direction of the interventricular septal motion appears to become reversed or "paradoxical". In fact, the left ventricle contracts normally, but superimposed on its motion is the greater movement of the hyperdynamic right ventricle. Contrast echocardiography has been shown to help in the detection of tricuspid regurgitation if Doppler echocardiography is not available. When a bolus of liquid, usually sterile saline, is injected rapidly into a peripheral vein, microbubbles of gas are released. On a two-dimensional display, echoes from these microbubbles can be seen to pass back and forth across the tricuspid valve and also seen passing retrograde into the enlarged hepatic veins; they often continue for several minutes before they are eventually cleared into the pulmonary artery. M-mode recordings show that the contrast echoes first appear in the inferior vena cava during systole, and can be seen passing retrograde into the enlarged hepatic veins.

Aortic Valve Stenosis



Causes of left ventricular outflow obstruction include aortic valve stenosis and subvalvular and supravalvular obstructions. Subvalvular obstruction can be further divided into the discrete diaphragmatic or fibromuscular ring types, and obstruction secondary to septal hypertrophy caused by hypertrophic cardiomyopathy. Aortic valve stenosis can be present from birth due to malformation of the valve, or can develop in later life as a result of calcification either of a congenitally bicuspid valve or a valve inflamed by

rheumatic disease. Mild congenital defects of the aortic valve are common, occurring in about 1% of the population. In most cases, the valve is "bicuspid", either with two equal-sized cusps or with three cusps, two of which are fused together. The patient is usually asymptomatic and there may be little or no murmur.

In adult life, though rarely before the age of forty, some bicuspid valves calcify and become progressively more stenotic. Although histological examination can usually distinguish such cases from rheumatic aortic stenosis. A rough correlation exists between the severity of calcification and the pressure gradient across the valve. Left ventricular hypertrophy may also be detected with two-dimensional echocardiography. Doppler echocardiography provides the most reliable noninvasive means for determining aortic valve peak and mean gradients, as does estimating valve orifice area by the continuity equation.

Aortic Valve Regurgitation

If aortic regurgitation is the sole lesion, its severity may be gauged from the degree of left ventricular volume overload, but the duration and severity of the mitral or septal flutter are of no help in this regard. As with other flow lesions, Doppler echocardiography is now the noninvasive standard for detecting the presence and severity of aortic regurgitation. In rheumatic heart disease, the most common presentation after pure mitral stenosis is a combination of this with aortic regurgitation. The aortic root diameter, measured at the level of the cusps, is normal. The mitral valve abnormality may be confined to slight reduction in amplitude of the posterior leaflet motion without any clinical manifestations. If, on the other hand, the mitral valve is heavily calcified, any flutter caused by aortic regurgitation may be difficult to detect.

Where vegetations on the aortic valve result from the infection, they are best detected by using two-dimensional long-axis and short-axis views. Vegetations larger than 2-3 mm can usually be visualized clearly and their approximate size can be determined, along with the cusps to which they are attached. Aortic valve endocarditis is sometimes complicated by the development of a mycotic aneurysm, which can rupture into one of the other cardiac chambers, or by the spread of the infection to the tricuspid valve. Although M-mode recordings may demonstrate the resulting hemodynamic changes, direct visualization of such defects is normally possible only by two-dimensional echocardiography.

Pulmonary Valve Stenosis

In cases of mild pulmonary stenosis, no abnormality is detectable either by M-mode or two- dimensional echocardiography. With a more severe obstruction, it may be possible to detect right ventricular hypertrophy. Another sign that has been reported, but in our experience is confined to patients with severe obstruction, is an exaggerated "a-dip" on the pulmonary valve echocardiogram. This arises from the fact that the hypertrophied right atrium forcefully injects blood into an already full and stiff right

ventricle during atrial systole. Pulmonary artery pressure is low, and so the sudden increase in right ventricular pressure is sufficient to partially open the pulmonary valve.

Echocardiography can assist phonocardiography in the diagnosis of pulmonary valve stenosis by confirming the origin of the early systolic ejection sound. A recording showing pulmonary valve closure may also help confirm the timing of the later pulmonary component of the second heart sound, although such recordings are routinely possible only in small children. Now that Doppler echocardiography is available, it has been shown to be able to detect, and reliably quantitate, pulmonary stenosis.

Pulmonary Valve Regurgitation

Pulmonary regurgitation causes volume overloading of the right ventricle, indicated on the echocardiogram by enlargement of the right ventricle and reversal of septal motion, as described earlier for tricuspid regurgitation following surgery for pulmonary stenosis. The combination of residual right-sided hypertrophy with overfilling of the right ventricle due to regurgitation gives a very deep pulmonary valve a-dip, and may even open the valve fully before the onset of systole. Doppler, again, has been shown to be quite sensitive in detecting and quantitating the severity of pulmonic regurgitation.

Pulmonary Hypertetion

Pulmonary hypertension causes right ventricular hypertrophy, which may be coupled with volume overload due to associated pulmonary or tricuspid regurgitation. Where pulmonary hypertension develops as a result of a large left-to-right shunt, it reduces the volume of the shunt, eventually reversing it (Eisenmenger physiology). The echocardiographic signs thus reflect both the primary pathology and the degree of pulmonary hypertension. In cases of severe pulmonary hypertension, the right ventricle compresses the left ventricular septum posteriorly, resulting in a characteristic "flattening" of the septum. Elevation of pulmonary artery pressure reduces the amount by which right atrial systole causes the pulmonary valve leaflets to dome upward, and so the a-dip on the M-mode echo recording is reduced. Provided the patient is in sinus rhythm, absence of the a-dip is a fairly reliable sign of pulmonary hypertension.

Stress Echocardiogram

Patients with coronary artery blockages may have minimal or no symptoms during rest. However, symptoms and signs of heart disease may be unmasked by exposing the heart to the stress of exercise. During exercise, healthy coronary arteries dilate (develop a more open channel) than an artery with a blockage. This unequal dilation causes more blood to be delivered to heart muscle supplied by the normal artery. In contrast, narrowed arteries end up supplying reduced flow to it's area of distribution. This reduced flow causes the involved muscle to "starve" during exercise. The "starvation" may produce symptoms (like chest discomfort or inappropriate shortness of breath), EKG abnormalities and reduced movement of the heart muscle. Treadmill is a method for stress echo with bruce protocole. If exercise on a treadmill is not an option (too much stress on the heart)

due to a person's medical condition, a physician may use an intravenous medication called dobutamine. Dobutamine causes the heart to beat faster and will mimic the effects of exercise on the heart.

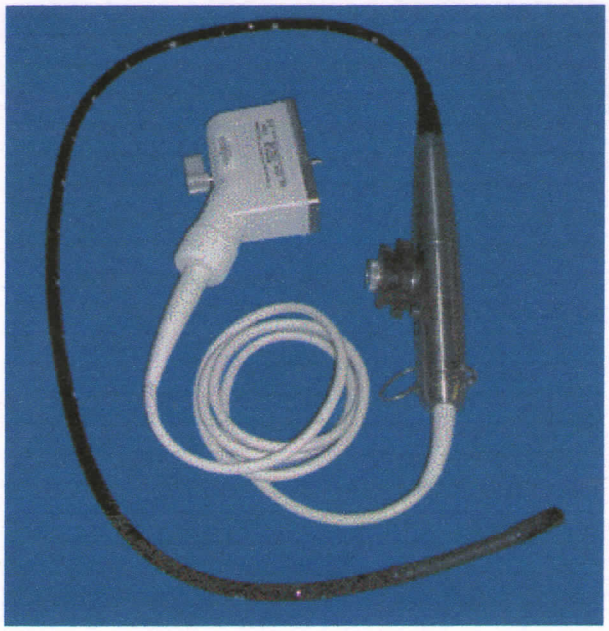
Possible indications for a dobutamine stress echocardiogram may include; to assess the heart's function and structures , to determine limits for safe exercise in patients who are entering a cardiac rehabilitation program and/or those who are recovering from a cardiac event, such as a heart attack (myocardial infarction, or MI) or heart surgery, to evaluate blood pressure during stress testing, to assess stress or exercise tolerance in patients with known or suspected coronary artery disease, to evaluate the cardiac status of a patient about to undergo surgery.

Transesophageal Echocardiography

Transesophageal echocardiography is also known as TEE, or heart scan with endoscopy. TEE uses a small probe guided into the esophagus while a patient is sedated to closely evaluate the heart and blood vessels within the chest. TEE uses a probe with a transducer on the end that is guided through the throat and into the esophagus while your child is sedated. The TEE transducer works the same as the one used in a regular echocardiogram. However, a clearer image can be obtained, because the sound waves do not have to pass through skin, muscle, or bone tissue. Transesophageal echocardiography is used to diagnose abnormalities of the heart, such as thickening of the heart walls, heart enlargement, infections, fluid build up, valve malfunctions, blood clots in the left atrium, and a condition called regurgitation, in which blood flows backward through the partially closed heart valves. It also shows the size of the heart, how well it is pumping, blood flow patterns, and whether there is any damage to the heart tissue. Transesophageal echocardiography provides a better picture of the heart when the patient is obese, has a thick chest wall or bandages on the chest, or is on a ventilator, all of which can interfere with the sound waves in a regular echocardiogram. Transesophageal echocardiography may also be used during cardiac surgery to monitor heart function.

Before the transesophageal echocardiogram begins, patient may be given a mild sedation. Because it is normal to gag if a foreign object is placed in the throat, the back of the throat is sprayed with a local anesthetic to numb the area so the patient won't feel the transducer. The transducer is inside a flexible tube called an endoscope, which contains a fiber optic system that allows the doctor to see where the tube is going. The endoscope is passed down the throat and into the esophagus until it is next to the heart. After the test, it is important to refrain from eating or drinking until the gag reflex has returned--otherwise, the patient may accidentally inhale some of the food or beverage. If a sedative has been given, patients should not drive or operate heavy machinery for at least 10-12 hours. They should avoid consuming alcohol for a day or so, since alcohol

may amplify the effect of the sedative. Transesophageal echocardiography may cause gagging and discomfort when the transducer is passed down into the throat. Patients may also experience sore throat for a few days after the test. In rare cases, the procedure may cause bleeding or perforation of the esophagus or an inflammatory condition known as infective endocarditis. The patient may have an adverse reaction to the sedative or local anesthetic. A TEE Probe picture is given below:



CARDIAC CATHETERISATION

INTRODUCTION

The cardiac catheter is an important tool, whose use over the last 5 years has defined a new area of special expertise within our broader parent speciality. Initially, catheter use was limited to the measurement of pressures and blood flow within the various cardiac chambers, elucidating both the normal physiology and pathophysiology of various cardiac disease states. With the advent of selective coronary angiography in the 1960's the main emphasis of cardiac catheterization began to switch from hemodynamic measurements to evaluation of coronary anatomy prior to bypass surgery.

The cardiac catheter remained a purely diagnostic tool throughout the much of 1970's until Andreas Gruentzig launched the field of cardiac therapeutics in 1977 by introducing his concept of percutaneous transluminal balloon angioplasty. And there by emerging the new field of interventional cardiology.

The interventional cardiology

The scope of what had come to be called "interventional cardiology" broadened further in the 1980's with the introduction of several new therapeutic modalities like coronary stents, valvuloplasty, atheroctomy and laser products .Simultaneously our ability to record electrical signals from within the cardiac chambers (electrophysiology)moved from being a purely diagnostic tool for observing and provoking arrhythmias, to a therapeutic tool by which abnormally conducting tissues can be ablated(Radio frequency ablation)or various devices such as pacemakers and implantable cardioverter defibrillators as the therapy for Brady and tachy arrhythmias.

And the new emergence of a variety of intra cardiac devices for the closure different congenital abnormalities. The device closure of ASD,VSD,PDA,PFO and the coil embolisation of PDA has given a hoon to the advanced interventional cardiology. The interventional cardiology has blossomed and in many ways now be regarded as the dominant discipline within the broad field of cardiac catheterisation as many patients like to avoid surgery.

Indications for cardiac catheterization

Cardiac catheterization is usually recommended to confirm the presence of a clinically suspected condition, define its anatomic and physiologic severity, and determine the presence or absence of associated conditions. Cardiac catheterization may yield information that will be crucial in defining the need for coronary angioplasty, cardiac

surgery, or other therapeutic interventions as well as the timing ,risks and the anticipated benefit in a given patient.

The commonest indication for cardiac catheterization today arises in the patient with an acute coronary ischemic syndrome in whom an invasive therapeutic intervention (PTCA,CABG) is contemplated. The patient with an acute coronary ischemic syndrome has most commonly experienced recent rupture of an atherosclerotic plaque within a coronary artery. Exposure of plaque contents in the flowing blood leads to platelet deposition and coronary thrombosis, which, in turn results in transmutable schema if the thrombus is completely obstructing, or unstable angina if it causes only partial or intermittent occlusion. The goal of cardiac catheterization in such patients is to identify the culprit artery and restore vessel potency by PTCA ,and if needed, intra coronary drug administration. The coronary angiogram(CAG) may reveal all the features of the coronary arterial tree, eg.multivessel disease or left main disease.

Cardiac catheterization is a must for patients with myocardial infarction, unstable angina, TMT positive atypical chest pain and Non ST Elevation MI (NSTEMI). For patients under going valvular surgery it is a routine thing to make sure that there coronary anatomy. For patients with cardiac myopathies it a rule to do a diagnostic catheterization to know the intra cardiac pressures and the chamber anatomy. For kids who have to under go congenital surgeries it is a rule to know the clear cardiac anatomy before surgery (eg. PA anatomy before BDG).

Contraindications to cardiac catheterization

Over the past several years, our concepts of contra indications have been modified by the fact that patients with acute myocardial infarction, cardiogenic shock, intractabel ventricular tachycardia and other extreme conditions have tolerated catheterizations and coronary angiography surprisingly well. At present the only absolute contra indication to cardiac catheterization is the refusal of a mentally competent patient to consent to the procedure.

Among the relative contraindications ventricular irritability can increase the risk and difficulty of left heart catheterization and can greatly interfere with the interpretation of the ventriculography. Hypertension increases pre disposition to ischemia and or pulmonary edema and should be controlled before catheterization. Other conditions that should be corrected is intercurrent febrile illness, decompensated left heart failure, correctable anemia, digitalis toxicity, and hypokalemia. Allergy to radiographic contrast agents is a relative contraindication to cardiac catheterization.

The relative contra indications are;

1. Uncontrolled ventricular irritability: the risk of ventricular tachycardia/fibrillation during catheterization is increased if ventricular irritability is uncontrolled.
2. Uncorrected hypokalemia or digitalis toxicity.

3. Uncorrected hypertension: predisposes to myocardial ischemia and or heart failure
4. Intercurrent febrile illness.
5. Decompensated heart failure: especially acute pulmonary edema, unless catheterization can be done with patient sitting up.
6. Anticoagulated state: prothrombin time>18 sec
7. Severe allergy to radiographic contrast agents.
8. Severe renal insufficiency and/or anuria: unless dialysis is planned to remove fluid and radiographic contrast load.

Different percutaneous approaches

The two main approaches in cardiac catheterization are the Femoral approach and the Brachial approach. Of course the others are there named the trans septal and the apical puncture techniques. And in our lab we most commonly uses the Femoral approach.

Advantages of Femoral approach

The femoral approach does not reqiure arteriotomy and arterial repair and can be performed repeatedly in the same patient at intervals, whereas the brachial approach can rarely be repeated safely more than two or three times. In femoral approach infection and thromboplebitis at the catheterization site are rare ;surgical suture of the skin is not necessary and approach is readily adaptable entry of other variety of vessels. Larger caliber devices like Valvuloplasty balloons and intra aortic counterpulsation balloon can be introduced into femoral artery but difficult in small brachial artery. The femoral approach is clearly a choice of method in a patient with absent or diminished radial or brachial pulsations or the direct brachial approach is unsuccessful.

Advantages of Brachial approach

The direct brachial approaches have advantages in patients with severe peripheral vascular diseases including the abdominal aorta, iliac or femoral arteries; suspected femoral vein or inferior vena cavil vein thrombosis; or forestation of aorta. The direct brachial approach may also have advantage in very obese patients ,for whom the percutaneous femoral approach is technically difficult and bleeding hard to control after catheter removal. Some prefer the brachial approach in patients with hypertension, aortic regurgitation, or wide pulse pressure due to other causes or who are receiving anticoagulant therapy. In these three circumstances, an increased hazard of bleeding has been reported with percutaneous femoral technique. Another advantage for direct brachial approach is the use of a single left heart catheter (sons catheter) for coronary angiography and left ventriculography.

Complications of cardiac catheterization

Because all cardiac catheterization involve the introduction of a foreign object (i.e. catheter) into the circulatory system, it is not surprising that a variety of adverse events can occur. These problems can range from minor problems with no long-term squeal eg.transient bradycardia during CAG to long-term major problems like cardiac perforation or abrupt closure of coronary artery during percutaneous transluminal coronary angioplasty. For the individual patient, the risk of sustaining a complication varies widely depending on demographics (age, gender), the cardiac anatomy (left main coronary artery disease, severe aortic stenos is, diminished left ventricular function), the clinical situation (unstable angina, acute myocardial infarction, cardiogenic shock) as well as the type procedure being performed (diagnostic catheterization, angioplasty, and so on).

By considering all these conditions the physician and the supporting staff can arrive at an accurate estimate of what level of risk is entailed in any given procedure. Familiarity with those risks can be of immeasurable value in: (a) anticipating increased risk of complication, (b) taking extra precautions to avoid them (eg. Placing a temporary pacemaker ready for a patient undergoing PTCA to RCA.) (c) Promptly recognizing complications when they occur (eg. Perforation of the right atrium during trans sepal puncture), and (d) taking corrective and potentially lifesaving action (Eg.pericardiosynthesis for perforation induced tampon).

Specific complications

Diagnostic catheterization: The mortality rate in a diagnostic cardiac catheterization is less than 1%. However, we can include certain patients who are at an increased risk of death during catheterization.

They are;

Age: Infants less than 1 year and the elderly greater than 60 years old are at increased risk of death during cardiac catheterization.

Functional class: Mortality in class IV patients is more than 10 times greater than in class I-II patients.

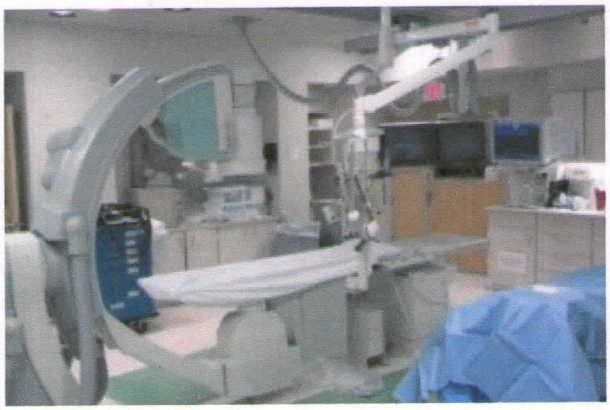
Severity of coronary obstruction: Mortality of patients with left main coronary artery disease is more than 10 times greater than for patients with single vessel

Valvular heart disease: Especially when combined with coronary disease is associated with a higher risk of death at cardiac catheterization than coronary artery disease alone.

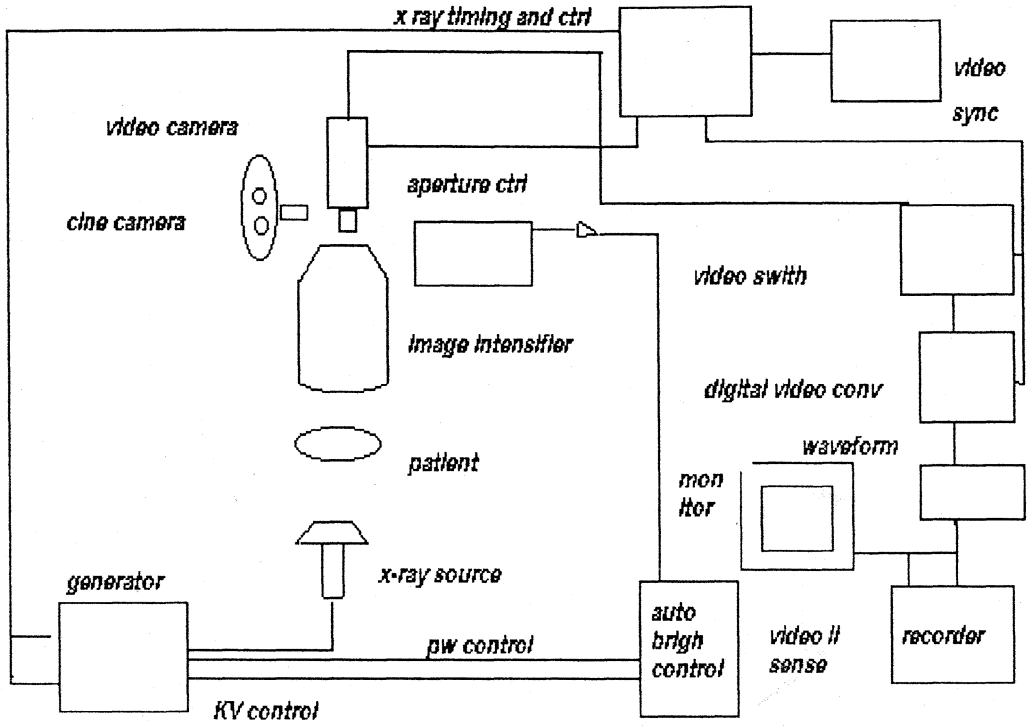
Left ventricular dysfunction: mortality for patients with LV ejection fraction less than 30% is more than 10 times greater than if ejection fraction is greater than 50%

Severe non cardiac disease: Patients with severe renal insufficiency, insulin requiring diabetes, advanced cerebrovascular or peripheral vascular disease, and severe pulmonary insufficiency appear to have an increased risk of death and other major complications from cardiac catheterization

The cardiac catheterization laboratory



As we are all working in cardiac catheterization laboratory it is better to have a good idea about the radiological and radio graphical ideas. The modern cardiac catheterization laboratory consists of patient support table equipment for monitoring of intra cardiac pressures and electrocardiography activity and a floor or ceiling suspended gantry that allows variable angulations of the X-ray beam. The gantry consist of an X-ray tube an image intensifier, a cine camera, and a video camera. The instrumentation of such an imaging system is shown below.



The X-ray generator

The generator is a step up transformer that converts three-phase 480-v line current into the high voltage 70-120 kV needed to power the X-ray tube for the generation of an X-ray beam. The transformer is submerged in a large tank of oil for cooling and insulation.

To be useful in cardiac study the generator must be combined with a cine pulse system, which chops the generator output into brief 4-6 m sec pulses that are required to eliminate motion induced blurring of the rapidly moving coronary arteries. Such pulse is best performed in the secondary or high voltage side of the system, using either vacuum switching tubes or a grid controlled X-ray tube to interrupt transiently generator current. The cine pulse must be capable of handling the 60 to 100-kiwi power output of the generator if that power is to be delivered effectively to the X-ray tube.

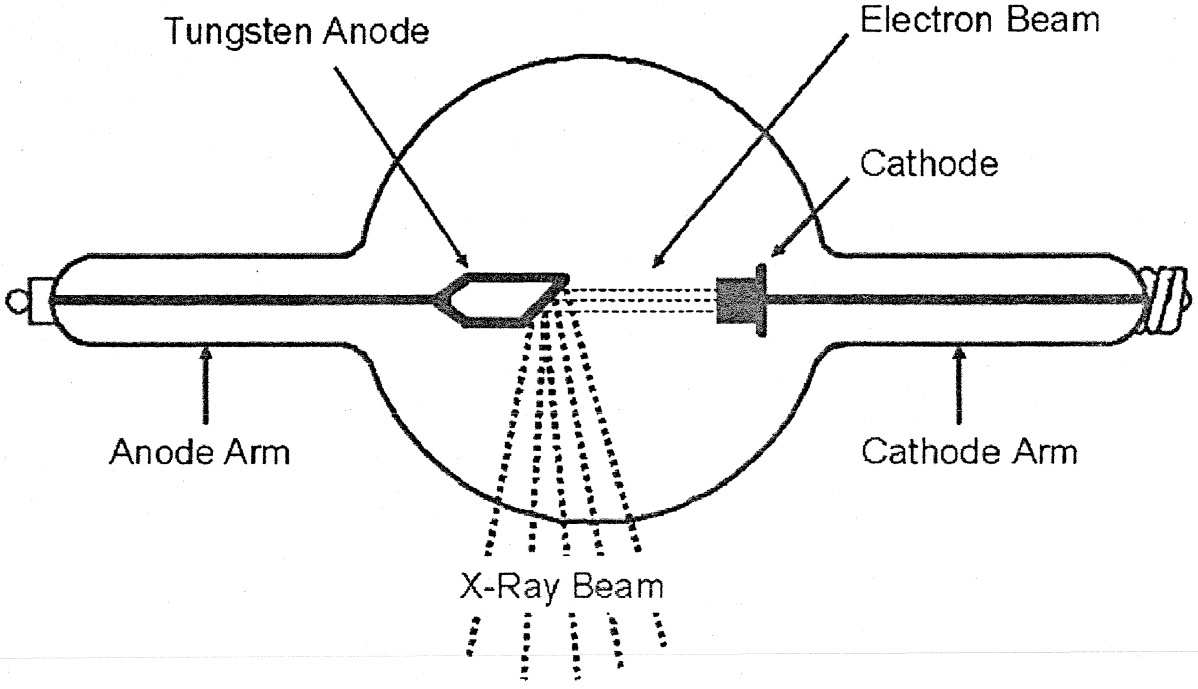
A final portion of the generator function is automatic exposure control, which compensates for changes in the transmission of X-rays to the image intensifier as the beam is panned through structures of differing attenuation. When the sensing photocell (located at the output of the image intensifier) detects a drop in light, the exposure control reacts to increase the number of X-rays that strike the image intensifier. This may be accomplished by increasing one of three factors: kV, this increases the energy of the X-ray photons, which increase their penetrating power; A or milliampere, the electrical current flowing through the X-ray tube increases which increases the number of X-ray photons generated, mess or milliseconds, the duration of each X-ray pulse which

increase the amount of time the X-ray beam is on and thus the total number of X-rays passing through the patient.

In steeply angulated views the automatic exposure system usually must compensate by increasing the kV, because a comparatively small rise in kilovolts will produce the same increase in film blackening as would doubling the am or mess. Although the same increase in film blackening could be obtained by doubling, the Mathis must exceed the power handling ability of the cine pulse system and or X-ray tube and double patient dose. Film blackening could also be increased by widening the pulse width, but doubling the pulse width would cause significant motion blurring and double patient dose. The primary change made by the automatic exposure system to maintain optimal film blackening is thus alteration in the kilovolts.

The X-ray tube

X-rays are produced by energy conversion when a fast moving stream of electrons is suddenly decelerated in the target anode of an X-ray tube. The X-ray tube is made of pyrex glass that encloses a vacuum containing two electrodes. The electrodes are designed so that electrons produced at the cathode (negative electrode) can be accelerated by a high potential difference towards the anode(positive electrode).



Now we can discuss every part of an X-ray tube.

The glass enclosure

It is necessary to seal the two electrodes of the X-ray tube in a vaccum. If gas were present inside the tube, the electrons that were accelerating towards the anode would collide with the gas molecules, lose and cause secondary electrons to be ejected from the gas molecules. By this process additional electrons will be available for the acceleration toward the anode. The purpose of vacuum in the modern X-ray tube is to allow the number and speed of the accelerated electrons to be controlled independently. The size and shape of the X-ray tubes are specifically desighned prevent electric discharge between the electrodes.

The connecting wires must be sealed into the glass wall of the X-ray tube. During operation of the X-ray tube the glass and the connecting wires are heated up to high temperatures. Because of difference in their coefficient of expansion most metals expand more than the glass when heated. This difference of expansion would cause the glass metal seal to break and destroy the vacuum in the tube if special precautions are not taken. Because of this problem special alloys having approximately the same coefficients of linear expansion as Pyrex glass, are generally used in X-ray tubes.

Cathode

The negative terminal of the X-ray tube is called the cathode. In referring an X-ray tube the terms cathode and filament mat be used interchangeably. In addition to the filament which is the source of electrons for the X-ray tube the cathode has two other elements. These are the connecting wires, which supply both the voltage and the current, that heats the filament ,and a metallic focusing cusp. The number(quantity) of X-rays produced depends entirely on the number of electrons that flow from the filament to target(anode) of the tube. The X-ray tube current measured in milliamperes refers to the number of electrons flowing per second from the filament to target. So remember that the number of electrons determines the X-ray tube current.

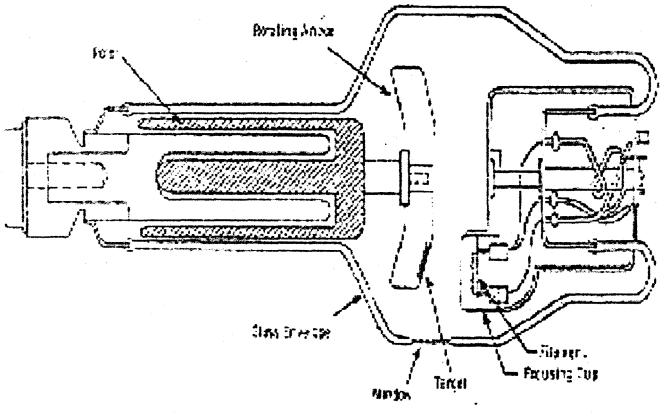
The filament is made of tungsten wire about 0.2 mm in diameter, that is coiled to form a vertical spiral about 0.2 cm in diameter and 1cm or less in length .When current flows through this fine tungsten wire ,it becomes heated. When a metal is heated its atoms absorb thermal energy and some of the electrons in the metal acquire enough energy to allow them to move a small distance from the surface of metal .Their escape is referred to as the process of thermion emission .which is defined as the emission of electrons resulting from the absorption of thermal energy .

The high currents that can be produced by the use of thermion emission are possible because large number of electrons can be accelerated from the cathode to the anode. The number of electrons involved is enormous. The unit of electric current is ampere which may be defined as the unit of flow when 1 coloumb of electricity flows in 1 sec. The coloumb is 6.25*10 18 electrons. Therefore an X-ray tube current of 100mA may be considered as the flow of 6.25*10 17 electrons from the cathode to the anode in 1 sec. Because of the forces of mutual repulsion and the large number of electrons ,this

electron stream would tend to spread itself out and result in the bombardment of an unacceptably large area of the anode. This is prevented by a structure called as the cathode focusing cusp, which surrounds the filament. The focusing cusp is desighned so that its electrical forces causes the electron stream to converge into the target anode in the required size and shape. The focusing cusp usually made of nickel.

Modern X-ray tubes contains three filaments called triple focus. The lrger filaments can be used for larger exposures. It has a stereoscopic angiographic tube .In this tube two fpcal spots are widely separated. When two films are exposed ,using a different focal spot for each film ,a stereoscopic film pair is produced. This tube is useful in angiography when rapid exposure of multiple stereoscopic film pairs is desired. Intervels as short as 0.1 sec between exposures can be obtained with stereoscopic tubes.

Components of an x-ray machine.

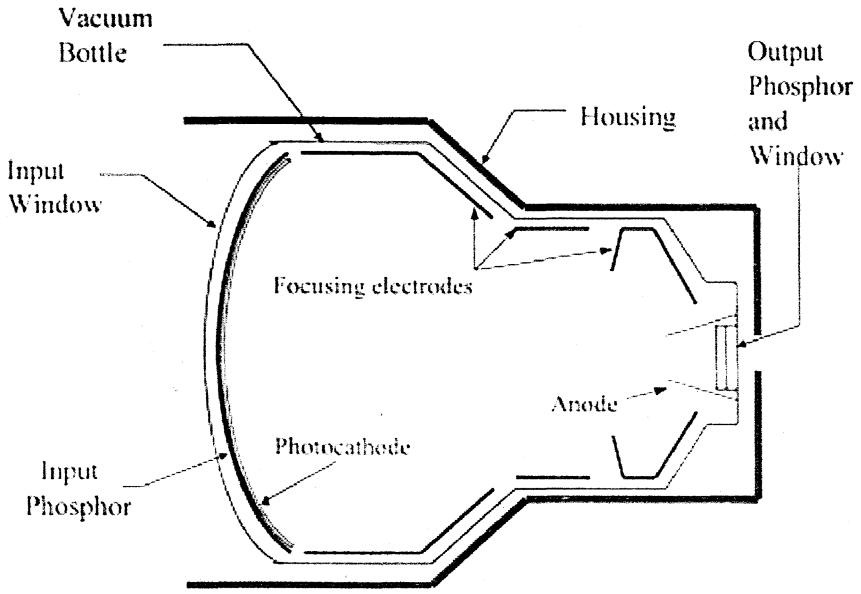


Principal parts of a modern rotating-anode x-ray tube.

Rotating anode X-ray tube

The ability of the X-ray tube to achieve high X-ray outputs is limited by the heat generated by the anode. The rotating anode principle is used to produce X-ray tubes capable of withstanding the heat generated by large exposures. The anode of a rotating tube consist of a large disc of tungsten, or an alloy of tungsten, which rotates at speed of 10000 revolutions per minute(rpm)when an exposure is being made. The purpose of the rotating anode is to spred the heat produced during an exposure over a large area of the anode. To make the anode rotate some mechanical problems must be overcome ,because the anode is contained within the vacuum of the tube. The power to effect rotation is provided by a magnetic field produced by stator coils that surround the neck of the X-ray tube outside the envelope. The magnetic field produced by the stator coils induces a current in the copper rotor of the induction motor and this induced current provides the power to rotate the anode assembly,metallic lubricants like silver are used.

The image intensifier



The x-ray image intensifier converts the transmitted x-rays into a brightened, visible light image. Within an image intensifier, the input phosphor converts the x-ray photons to light photons, which are then converted to photoelectrons within the photocathode. The electrons are accelerated and focused by a series of electrodes striking the output phosphor, which converts the accelerated electrons into light photons that may be captured by various imaging devices. Through this process, several thousand light photons are produced for each x-ray photon reaching the input phosphor. Most modern image intensifiers use cesium iodide for the input phosphor because it has a high absorption efficiency and thus decreases patient dose. Image intensifiers come in various sizes, most having more than one input image size or magnification mode. Modern image intensifiers are specified by conversion factors, which is the measure of how efficiently an image intensifier converts x-rays to light. Because of design restrictions, image intensifiers are subject to inherent and induced artifacts that contribute to image degradation. Both spatial and contrast resolution gradually decrease during the lifetime of the image intensifier because the brightness gain of an image intensifier decreases with time as the phosphor ages. A well-run quality control program for the image intensifier is needed to detect the inevitable changes in settings before they appear on clinical images. An x-ray image intensifier has two major functions: (a) to intercept the x-ray photons and convert them into visible light photons and (b) to amplify or intensify this light signal. The image intensifier creates a large gain (or intensification) in luminance at the output screen compared with that at the input screen. The output screen image can be viewed with closed-circuit television or recorded with film.

Construction and Principles of Operation

In a modern fluoroscopy system, the image intensifier is located opposite the x-ray tube. The image intensifier is contained in a cylindrical protective case because it is a very delicate device under high vacuum and needs to be handled with care. At the entrance end of this protective case, there is usually a mechanical sensory device to prevent the image intensifier from pushing too hard on the patient or other objects, which may cause damage to the image intensifier. Image intensifiers come in various sizes depending on the specific application. Usually, the larger the image intensifier the higher the cost.

The operational principles of an image intensifier can be briefly described as follows. X-ray photons penetrate the input window of the vacuum case. The input phosphor absorbs the x-ray photons and converts them into optical photons (a phenomenon called luminescence). These optical photons are converted to photoelectrons at the photocathode. The photoelectrons are accelerated by the electric field produced by the strong electric potential difference of the image intensifier and are collected at the output phosphor. Each accelerated electron produces many optical photons at the output phosphor.

Image Intensifier Components

An image intensifier consists of the following major components: an input window, an input phosphor and photocathode, several electrostatic focusing lenses, an accelerating anode, an output phosphor screen, and a protective vacuum case.

Input Window

The shape and choice of material for the input window results from a compromise among many factors, such as minimizing patient distance, x-ray absorption, x-ray scatter, manufacturing cost, and mechanical strength of materials. The input side of the image intensifier usually has a convex shape and is generally made of aluminum (Z = 13). The convex shape not only minimizes the patient distance thus maximizing the useful entrance field size, but it also gives the image intensifier better mechanical strength under atmospheric pressure. This aluminum input window is approximately 1 mm in thickness.

Input Phosphor and Photocathode

X rays transmitted through the input window are converted into fluorescent light photons by the input phosphor. The input screen is a substrate made of aluminum coated with a phosphor layer, an intermediate coupling layer, and finally the photocathode layer. The thickness of the input phosphor layer is a compromise between spatial resolution and x-ray absorption efficiency. A thicker phosphor layer has higher x-ray absorption efficiency, which means more x-ray photons can be absorbed and converted to light photons in the phosphor layer. A thicker phosphor layer requires fewer x-ray photons to generate the same amount of light photons at the image intensifier output window, thus

reducing patient dose. However, with a thicker input phosphor layer, more light photons are scattered laterally within the phosphor layer, thus reducing the spatial resolution. Currently, the thickness of an input phosphor layer typically measures between 300 and 450 μm, depending on the image intensifier type and technology used.

To maximize the conversion efficiency from x-ray photons to photoelectrons, the mass attenuation coefficient of the input phosphor material should be matched with the spectrum of the x-rays emerging from the patient. Ideally, the light spectrum of the input phosphor should also match the sensitivity profile of the photocathode. The initial phosphor used in early image intensifiers was zinc-cadmium sulfide (Zincs), whereas the current phosphor of choice is cesium iodide (China). There are several reasons for replacing Zincs with China as the input phosphor material. First, the mass attenuation coefficient of China better matches the x-ray spectrum of the radiation transmitted from the patient.shows the mass attenuation coefficients of the two phosphors in relation to the relative spectral distribution of the transmitted radiation from the patient. The mass attenuation peaks in CsI:Na, compared with those of ZnCdS, are more closely matched to the transmitted x-ray spectrum, thus increasing the absorption of the transmitted x-ray photons. As mentioned, increasing the absorption efficiency decreases the patient's dose. A second advantage for using CsI:Na as the phosphor is that it has a high atomic number from Cs ($Z = 55$) and I ($Z = 53$), which also results in higher x-ray absorption. Consequently, most modern image intensifiers use CsI:Na for the input phosphor material.

The photocathode layer is made of antimony-cesium ($SbCs_3$). To maximize the conversion efficiency from light photon to photoelectron, light emitted from the input phosphor should match the sensitivity spectrum of the photocathode., CsI:Na has a better spectral match to the antimony-cesium compound ($SbCs_3$). This is another reason why CsI:Na is a better input phosphor material than ZnCdS. The photocathode has a thickness of about 20 nm and a photoelectron production efficiency of 10%–15%. Approximately 200 photoelectrons will be created for a single 60-keV x-ray photon absorbed in the input phosphor.

In addition to its high absorption efficiency, CsI:Na can be evaporated onto the substrate in crystal needle form. These needles act like light pipes, in a manner similar to the light propagation in a fiber-optic faceplate, thus reducing cross scatter inside the phosphor screen and yielding better spatial resolution.. The CsI:Na needles are approximately 5 μm in diameter. Input phosphor screens in modern image intensifiers are approximately 300–500 μm in thickness and absorb 60%–70% at 60 keV. Because of the crystalline structure of the needles, the surfaces of the crystals, and the reflectivity of the substrate, approximately 2,600 luminescence photons are generated from each 60-keV x-ray quantum. Of these 2,600 luminescence photons, approximately 1,600 reach the photocathode.

Electron Optics

Photoelectrons are accelerated from the photocathode to the output phosphor by the anode. The accelerated photoelectrons are focused down to the size of the output

phosphor by a series of electrostatic focusing electrodes. The number of photoelectrons within the image intensifier will not increase: Only the speed of the photoelectrons will increase. The total current produced by these photoelectrons is approximately 600 nA (600×10^{-9} A).

The focusing electrodes are very sensitive to external electrical and magnetic fields. Extraneous electrical and magnetic fields (even the earth's magnetic field) may cause image distortions in the image intensifier. This effect must be monitored and controlled for fluoroscopes operated near magnetic resonance imaging units. Furthermore, the high voltages on the electrodes must be kept very stable to guarantee the image quality, since ripple in the voltage will be noticed as periodic variation in image diameter.

On the vacuum side of the output phosphor surface, the anode of the electron optics system has a thin aluminum film coating. This aluminum film allows electrons to pass through, but it is opaque to light photons generated on the fluorescent screen. It stops these photons from being scattered back into the image intensifier and exposing the photocathode. The film also serves as a reflector to increase the output luminance.

Output Phosphor and Window

The output phosphor of the x-ray image intensifier, which typically is called P20, is a fluorescent compound made of silver-activated zinc-cadmium sulfide (ZnCdS:Ag). The emission spectrum of P20 is at a maximum around 530 nm (green light). The P20 layer is very thin, having a thickness of 4–8 μm, and is deposited on the glass output window. Approximately 2,000 luminescence photons are generated for every accelerated 25-keV photoelectron. Because every electron was produced by one light photon, this represents a luminescence gain of 2,000. The output window is carefully designed so that the fluorescent photons reflected back to the input screen are minimized. The luminescence decay time of the output phosphor determines the temporal resolution of the image intensifier.

Image Intensifier Housing

The x-ray image intensifier is enclosed in a metal housing consisting of lead to absorb scattered radiation, mu-metal to shield the electron optics from extraneous magnetic fields, and an outer aluminum shell. On the input side of the housing, the aluminum shell protects the input window of the image intensifier. Although the housing will provide some shielding from external electromagnetic fields, the presence of strong magnetic or electrical fields too close to the image intensifier will degrade image quality.

Magnification Modes and Spatial Resolution

Changing the voltage applied to the electronic lenses inside an image intensifier will change the magnification mode of the image intensifier. In a magnification mode, a smaller area of the input phosphor is used, giving the effect of zooming in on the image. Because the input field size is reduced, the exposure to the input phosphor must be increased to maintain a constant brightness level at the output phosphor. In fact, to maintain the same noise level, the dose quadruples when the magnification is doubled. Modern image intensifiers can perform multiple steps of magnification. It is even possible to have stepless magnification. Each magnification mode yields a different dose rate to the patient. In general, the smaller the field size, the larger the magnification, and the higher the patient dose. The image intensifier exposure rate is typically set to 30 mR/sec for the 25-cm mode, 60 μR/sec for the 17-cm mode, and 120 μR/sec for the 12-cm mode.

Higher magnification modes produce increased spatial resolution. The spatial resolution of an image intensifier is in the range of 4–6 line pairs per millimeter (lp/mm). The final spatial resolution of the fluoroscopic system also depends on other imaging components in the whole imaging chain.

Flat panel detectors

The detectors are based on amorphous silicon technology, which enables the collection of x-rays and their conversion into digital data. The x-rays are transformed into visible light by a scintillator layer of cesium iodide, whose fibre-optic-like structure produces high resolution and a high absorption coefficient. The scintillator is coupled to amorphous hydrogenated silicon (aSi:H) photodiodes, which convert the light into digital data. The light striking each photodiode generates a current, which is subsequently transferred to an external drive via a matrix of aSi:H switching devices. The data are processed by the panel's readout electronics and can then be sent to a workstation or PACS

Flouroscopy and television chain

The primary thing behind a cardiac catheterization is to get a good quality fluoroscopy and a good quality video acquisition.For that the angioscopy machine must be equipped with a good quality imaging.During fluoroscopy essentially all the light from the image intensifier is sent to the television camera.During cine angioscopy , partially silvered mirror is flipped into the light path to divert 90% of the light to the cine camera, allowing only 10% of the light to strike the television camera. This design forces the television camera to adapt to a wide range of input light levels ,which may stretch the cameras usual operating range.This is compensated for in part by an automatic gain control within the television circuitry and by the use of a motor driven iris shutter on the television input lens that can close as needed to avoid light overload during cine runs.

The type of image tube used varies in different television systems.Compared to the standard Vidicon (which uses an antimony trisulfide target),most catheterization labs uses a Plumbicon(lead monoxide target) ,Saticon(selenium arsenic tellurium target) or Primicon (selenium tellurium arsenic target)to maximize image contrast and minimize image carryover or lagso that less than 10% of the image signal carries over into the third video field.

Another important variable is the scanning format. Interlaced scan systems (like broadcast TV) alternately sweep the even and odd numbered lines from the overall raster.This may result in degradation of the television image during 30fps cine angiography,due to misregistration of the anatomic details between the odd and even scan lines, as well as flicker caused by collection of onset of lines when the X-ray beam is on and another set when it is off.Use of a progressive scanning television format can overcome these limitations such that all scans are acquired in numerical sequence each time the X-ray tube is pulsed. A scan converter then picks off the even and odd numbered scan lines for display on a standard interlaced monitor. The other scan variable is the number of scan lines that make up the image. High line systems can theoretically improve vertical resolution on the TV monitor but they also increase amplifier bandwidth and can thus introduce more electronic noise into the displayed image.These limitations can overcome by a digital system.

Digital systems use high speed computers to perform online processing of the television image.In addition digital systems store the images obtained in all cine angiographic runs and allow their play back at variable speed ,magnification and levels of contrast enhancement .These images can be retained as a road map to facilitate the positioning of interventional devices to compare anatomy before and after intervention.And to perform online measurement of stenosis severity .We in our lab are using Digital Imaging and Communications in Medicine(DICOM) standards for storing and retrieving these digital images on recordable compact discs as a potential replacement for cineangiographic film as a dominant archival medium.

Radiation safety

Cardiac catheterization delivers one of the highest levels of patient and operator radiation dose all current diagnostic procedures. So we must be aware about the radiation safety.

Units of measurement

The primary unit of radiation exposure is the rontgen® which is defined in terms of the amount of ionization that the beam produces in air.A more relevant unit is rad or radiation absorbed dose ,which deals with how much heating the radiation beam produces in each gram of a specified material.Gray is a newer unit termed as 1 Gy =100 rads.

Biological effects of radiation

One well known but poorly quantitated risk of radiation is that of genetic damage i.e mutation in the off spring of an exposed individual.Bear in mind that such mutations occur spontaneously and that laboratory studies suggest that very large exposure are required to double the baseline risk of mutation.This means that the major danger of radiation exposure is the somatic risk ,which may include either direct tissue injury or carcinogenesis. The organs most sensitive to radiation induced cancer are the bone marrow ,female breast , and the thyroid.However these organs also have a high incidence of spontaneous malignancy.

Reducing Exposure Dose

The most important factor is to reduce the patient dose.This means the X-ray equipment doses caliberated within national standards and as low as possible without degrading the quality of the fluoroscopic and film images.The temptation to use high fluoro should be avoided other than in emergency situations.If pulse fluoroscopy is available it improves the imaging quality but reduces the patient dose. Distance is another important way of reducing the radiation exposure.The operator can stand as var from the beam as possible to take the advantage of inverse square law .One or two steps further away from the X-ray tube may cut the exposure in half.

PROCEDURES DONE IN CATH LAB

CORONARY ANGIOGRAM(CAG)

Coronary angiography is the only method that is widely available to study the intraluminal anatomy of the coronary arteries. As stated by Gensini "the primary goal of coronary angiogram is identification, localization, and assessment of obstructive lesions within the arteries of the heart.Aquired disorders like atherosclerosis ,spasm, thromboembolus, traumatic injury.As well as congenital anomalies like anomalous coronary artery origin, myocardial bridges ,atresia, fistulae can be detected. Specifically

Coronary angiography is usually performed to determine the presence or absence of atherosclerotic coronary artery disease and to define its severity (degree of stenosis)and extent (number of artery segments involved).

Indications for coronary angiography

Asymptomatic patients

Asymptomatic patients with known coronary artery disease generally those who had a myocardial infarction or have undergone bye pass surgery, angioplasty. Evidence of high risk out come in tread mill tests like down sloping ST segment with >2.0 mm ST segment depression. Abnormal systolic blood pressure response Thallium or scintigraphy study which is positive. Echocardiography which had shown a left ventricular ejection fraction of <40% in rest, with some regional wall motion abnormalities. Marked ECG changes in rest.

Symptomatic patients

The symptomatic patients can be described into three classes as discussed below.

Class I

Angina pectoris that have proved inadequate response to medical treatment PTCA, Thrombolytic therapy, coronary bye pass surgery. Unstable angina pectoris. Acceleration of symptoms with increased severity and frequency of chronic angina within the past two months despite medical management, including the new onset of angina at rest. New onset of unstable angina which is severe or increasing despite medical management. Acute coronary in sufficiency with pain at rest usually of >15 minutes duration associated with new ECG changes. Prinzmetal's or variant angina pectoris. Angina pectoris in association with any one of the following. Evidence of adverse out comes during exercise testing. Coexistence of history of MI or hypertension and ST segment depression in ECG. Episode of pulmonary edema or symptoms of left ventricular failure. Before major vascular surgery ,such as repair of aortic aneurysm ,iliofemoral or carotid arterysurgery.

Class II

Angina pectoris in the following groups. Female patients, 40 years of age with objective evidence of myocardial ischemia by noninvasive testing. Men patients<40 years age. Patients <40years with previous history of MI. Patients who shows a progressively more abnormal Exercise ECG. Patients who cannot be risk satisfied by other means for example unable to exercise because of amputation, arthritis, limb deformity.

Class III

Presence of mild, clinically stable angina in patients who do not have impaired ventricular function. presence of well controlled angina pectoris in patients who are clearly not candidates of bye pass surgery.

Atypical chest pain

Chest pain atypical for angina is defined as single or recurrent episodes of chest pain without the typical location, radiation, character, or inciting factors usually associated with the pain of myocardial ischemia. Chest pain atypical but non invasive tests shows positive results. when the presence of chest pain ,atypical for angina due to coronary artery spasm is suspected. when there are associated signs showing that LV function is impaired. chest pain ,atypical for angina but the non invasive tests are inconclusive. when noninvasive tests are negative but management requires that CAD is to be excluded.

Myocardial Infarction(Acute)

Acute infarction should be considered in three phases .Evolving infarction encompasses the initial hours after the onset of chest pain and is the period whwn the intravenous or intracoronary Thrombolytic therapy is increasingly used for reperfusion. The phase of completed infarction begins after the initial hours and lasts up to pre discharge evaluation. During this phase relatively only small amount of patients suffer complications. The phase of convalescent infarction is the ensuring period up to approximately 8 weeks .

Valvular Heart Disease

When surgery is considered for those patients who had vavular stenosis or valvular regurgitations it is a routine thing to perform a coronary angiography for the following categories. (1) Male patients of >35years age. (2) Female patients of >40 years age.

Congenital Heart Disease

Adult patients who are above 40 years of age are going for an ASD closure of TOF -ICR they should also have a CAG done.

Contraindications to coronary angiogram

The only absolute contra indication is the lack of patients consent. And the relative contra indications are as follows. Recent stroke(within 1 month), progressive renal insufficiency, Active gastrointestinal bleeding, Fever which may be due to infection, Active infection, severe pulmonary, renal or hepatic disease, Severe anemia, Lack of surgical stand by.

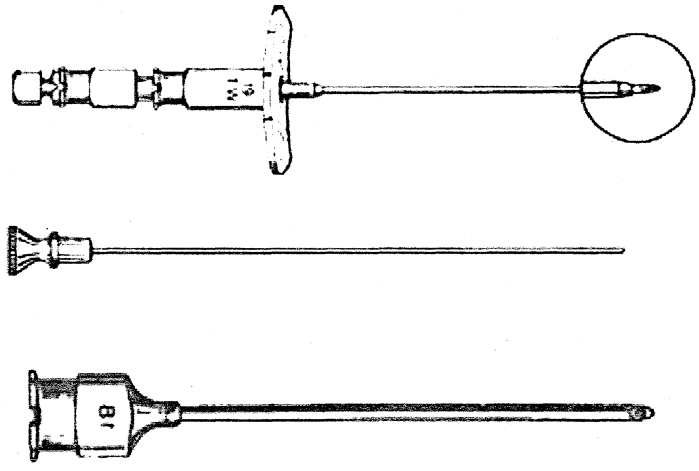
Hardwares used in CAG

Modern catheterization laboratories are equipped with an extensive inventory of catheters , wires and other equipment for use in a wide variety of procedures. An understanding of basic construction and design of these "tools of the trade" is very important relative to the selection of equipment for specific cases, optimal manipulation of the equipment, and avoidance of complications. Discussion of equipment and technique for catheterization will follow in order of use in the procedure, that is first the needles for Percutaneous vascular access; second the varied guide wires available; third sheaths and dilators for establishment and maintenance of a route for catheter insertion and of course ourth the catheters itself.

Needles for percutaneous vascular access

Basic design and Features

Choice of specific needle for vascular access seem to require little consideration.There are two major difference in needle design which result in different insertion techniques, seldinger vs direct, and considerable variations in needle size and shape. The following figures show the Seldinger as well as the direct puncture needles.



The Seldinger needle has a blunted needle bevel and one or two stylets. The innermost stylet is solid and designed to prevent the lumen from becoming blocked with skin and subcutaneous tissue and blood as the needle is inserted into the artery. The hub of the needle must incorporate several features .It must fit firmly on the body of the needle and internally have a smooth funnel into the body to facilitate wire insertion. Most hubs have a female luer lock connector ,allowing attachment to the male luer lock connector on a syringe with an airtight seal. This is important when we withdraw blood from a low pressure vessel like vein and observing blood return.

Needles are sized according to their external diameter and an arbitrary gauging system . With increasing gauge number the diameter decreases. Most 18-gauge needles will allow passage of 0.035 or 0.038 inch standard guide wire. A standard length of 2.75 to 3.0 inches to arterial needles are acceptable. The tip of the needle has a bevel that forms an angle less than acute that of standard injection needles. The needle must of course be sharp enough to avoid tearing the arterial wall on vessel entry and strong enough to pass through calcified arterial walls. Needles for other uses, like pericardiocentesis or direct percutaneous leftventricular puncture, are available in various designs and lengths up to 22cm .

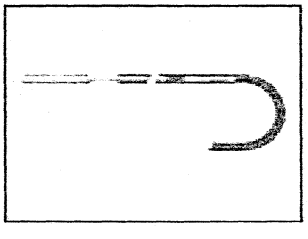
The guide wires

Guide wires are the Heart of cardiac catheterization. They are obtained in different diameters ,lengths ,size and shapes for there respective uses. Guide wire is the one which guides the cardiac catheter through the vascular system and seeks its destination.

Construction

The usual guide wire has mainly two parts one is the coil and the other is the core or mandrel. The coil is usually made by spinning a thin strand of round wire around a metal tube or rod. The coil is advanced over a core or mandrel and fixed at the ends by a soldered bond. At the flexible end or tip a safety ribbon is soldered and runs the length of guide wire adjacent to the core.

All guide wires have an inner safety wire, which is bonded or welded at both ends to prevent separation in the event of guide wire breakage and to contain the coils of the spring wire during manipulation. The wire tips are welded and polished for strength and smoothness to minimize vessel trauma. We usually refer guide wire length in centimeters and the diameters both in inches and in millimeters.



Material

Guide wires are all constructed of fine stainless steel. Some guide wires are coated with Teflon (PTFE-Poly Tetra Flouro Ethylene) to reduce the friction coefficient of the guide wire within the catheter. Care must taken when a Teflon coated guide wire is

passed through a metal needle or catheter hub,as the Teflon coating can become abraded and flake off.

Types

Guide wires basically are of two shapes, straight and J curve and may have either a movable core or a fixed core.

Straight guide wires

The straight guide wire has a straight flexible tip generally 3cm and is appropriate for passage through linear vessels. Since both the proximal (stiff) and the distal(flexible) ends of the straight guide wires appear the same, it is imperative to check the ends before insertion to ensure that the flexible end is inserted. Some straight wires are constructed such that both the ends are flexible to avoid this complication.

J-curve guide wires

The J-curved guide wire generally has a 1.5,3,or 6 mm curved radius at its tip. It is particularly used for passage through tortuous vessels. Many percutaneous catheter introducer sets now supply a 3mm curve J curve wire with the kit. The larger 6mm curve wire is often appropriate for negotiating a tortuous femoral vein. An even larger 15mm radius J-curve is for cases of extreme vessel tortuosity. A multipurpose, double end guide wires offers a long 10cm flexible straight tip at one end with a small 1.5 mm J curve on the other end. J-curve guide wires are packaged with a small plastic sleeve that when advanced over the tip of the guide wire straightens the guide wire and permits it to be introduced into the hub of a needle or catheter.

Fixed core guide wires

Fixed core guide wires have a straight inner core (mandrel) that is fixed at one end and terminates usually 3cm from the tip .The core provides necessary stiffness to the body of the coiled wire ,while allowing flexibility at the tip. Variations in the tapered length 10 or 15 cm of the mandrel alter the degree of tip flexibility and improve the transition from the flexible distal coil to supported mandrel. This is an important feature in PTCA guide wires. Both straight and J-curved guide wires are available with a fixed core.

Movable core guide wires

Guide wires that have the straight core attached only at the distal end are designed to allow movement of the tip of the core for the purpose of increasing or decreasing the length of the flexible tip. This is accomplished by not welding the proximal end of the mandrel core to the end of the guide wire, but instead securing the inner mandrel to a 5cm handle that is attached to the distal end of the wire. Variations in the tapered length of the core (5cm, 10cm,15cm) alter the degree of tip flexibility or in case of J-curve wires the radius of the floppy wire. The ability to increase or decrease the length of the wire's

flexible tip of ten desirable for traversing local areas of tortuosity. On occasion ,re advancement of inner mandrel may be difficult .This may be due to bending or kinking of the un supported flexible tip. In general the inner mandrel should never be forced against a resistance ,if this occurs the entire guide wire should be removed.

Core-flex guide wires

Core-flex guide wires have a solid stainless steel mandrel with a safety wire constructed of spring coil soldered to the end. Although the out side diameter of this guide wire is 0.018inch ,the guide wire offers the same support and rigidity as an 0.035 inch guide wire because of its solid steel mandrel. All usual PTCA wires are Core-flex wires. The rigidity of the guide wire permits percutaneous cannulation with a smaller 22 gauge cannula rather than a standard 18 gauge cannula.

The Exchange guide wires

The exchange guide wire is a straight wire unique only in its length of 260 or 300 cm. It is used exclusively for catheter exchange purposes as it is long enough to allow the removal and insertion of a catheter while the wire tip remains in the selected chamber. This is often used for exchanging catheters in LV if difficulty was encountered in crossing the aortic valve. Exchange guide wires are also used in coronary angioplasty.

Open-end guide wire/catheter

This new type of Teflon wire has an open end and a removable inner mandrel. In this way it can function both as a guide wire and with the mandrel removed as a catheter. This guide is of greatest use in traversing tortuous or stenotic arteries during peripheral angioplasty. Pressures can be monitored during this procedure through the lumen of the guide wire.

Dimensions of the guide wires

The guide wires for CAG are available in sizes ranging from 0.018 to 0.038 inches in diameter. The guide wire most commonly used for coronary angiogram is 0.035inch guide wire as it will support and passes through 6,7 and 8 Fr. catheters. Ideally the catheter tip should fit closely over the guide wire ,with no excess space between the catheter and the guide wire. This promotes the smooth insertion of the catheter into the vessel. Guide wires are available in lengths varying from 50 to 300 cm. The guide wires length is dictated by the length of the specific catheter to be used. The guide wire should be at least 20cm longer than the catheter to allow insertion of the catheter over the wire with some wires always exposed at the hub for manipulation and retension. Usually for coronary angiogram an 145cm wire is used.

Desired features of a guide wire

Important considerations in guide wire selections include stiffness, flexibility, and smoothness .These features are important for both patient safety and improved operation.

Stiffness

The stiffness of the guide wire is a function of both the core and the diameter of the wire. The wire must be sufficiently stiff to be pushed forward without collapsing back on itself or coiling within the vasculature. Increased stiffness can be obtained by use of a larger guide wire ,if possible or use a guide wire with heavy duty larger mandrel.

Flexibility

The degree of flexibility of the guide wire is related to the core as well as to the single helix of stainless steel wire around it.

Smoothness

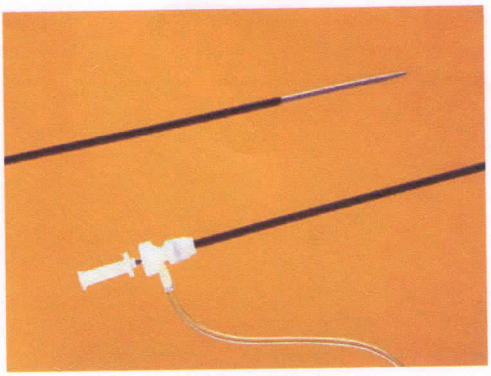
The smoothness of the wire is a result of its surface. Certainly the Teflon coated wires are smoother and produce less friction when passed through catheters. Some guide wires are treated with benzalkonium heparin to reduce thrombogenicity.

The Vascular Sheaths and Dilators

The purpose of putting a sheath(introducer) into the artery is to facilitate the passage and exchange of catheters through a closely fitting sheath. Introducers allow multiple catheter exchanges without or with reduced use of guide wires and with much less patient discomfort.

Design

Introducer sets originally consisted of a tapered dilator within a slightly shorter sheath. The introducer has a gasket seal(hemostasis valve)on the hub of the sheath and a side port tubing with a stopcock attached directly to the hub of the sheath. These things eliminate blood loss during catheter exchange, decrease Thrombolytic complications and reduce the risks of air embolism in the central venous catheterization. The side port tubing conveniently allows the options for fluid or drug administration as well as pressure monitoring.



Longer introducer sets are (23,45 or 60 cm) sometimes very helpful for passing catheters through a tortuous femoral-iliac system. The inner diameter of the introducer is expressed in French system. One French is the one third of a millimeter. 3Fr =1mm. As a standard we are using usual 6Fr. 11cm vascular sheath for diagnostic coronary angiogram and 7Fr.11cm sheath for angioplasty.

The Catheters

In comparison with any other equipment in modern cardiac catheterization catheters are extremely diverse in shape ,design and specific features. Here we will just go through the basics of catheter construction ,and the wide selection of catheters used in coronary angiography describing there merits and demerits of each technique used by different physician. Listed below are the basic characteristics of a standard diagnostic catheter.

Axial control-It is the ability to directly transmit forces from the end of the catheter to the tip.

Body-Segment of catheter between the tip and the hub. Flow rate Ability to deliver high contrast material flow rates within a specified injection pressure range.

Flexibility-Ability of a section of catheter to bend on contact with a resistant surface.

Hub-Fitting on the proximal end of the catheter. Internal diameter-Diameter of the internal lumen of the catheter which determines which guide wire can be accommodated and expected contrast medium delivery.

Maneuverability- Ability to advance a catheter through sharp bends or through tortuous vascular segments. Implies flexibility and torque control.

Memory- Ability to recover and maintain a specific configuration after insertion and guide wire removal.

Pliability-Ability to bend and to be shaped.

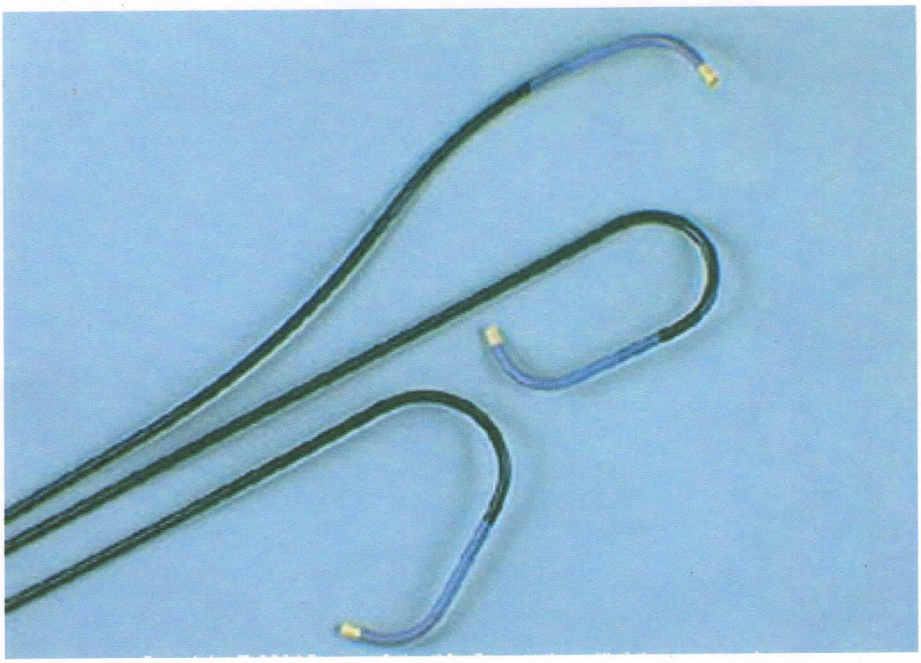
Pressure monitoring characteristics- capacity to accurately transmit pressure from catheter tip to pressure transducer ,a function of length ,internal diameter, and catheter stiffness.

Pushability-Ability to directly transmit forces from catheter hub to the tip longitudinally.

Radiopacity-Ability to visualize the catheter under X-ray

Catheter components

A cardiac catheter is composed of several segments and parts as illustrated in the figure.



Materials used

The original cardiac catheter was rubber ureteral catheters ,then different materials were used, the main considerations given for the material is it must be biocompatible,
thromboresistantandradioopaque.Dacron,polyurethane,polyethylene,Teflon,polyvinychlori de are the materials now used for making cardiac catheters.

Dacron

Catheters made of woven Dacron are very maneuverable and flexible. Most Woven Dacron catheters are covered with polyurethane coating to increase surface stiffness and reduce vascular trauma. Some angiographic catheters are reinforced with a nylon core to increase bursting pressure and there by allow higher flow rates of contrast media. Examples: USCI cournand, Goodale Lubine .

Polyurethane

Catheters extruded from polyurethane have an excellent tensile properties memory. This is a desirable feature of superselective catheters whose tip design may be altered during insertion and manipulation. Polyurethane catheters are also softer than polyethylene and Teflon catheters which reduces the risk of vascular trauma or perforation. This feature often permits the passage of catheter in tortuous vascular system.While polyurethane is associated with increase in thrombogenicity . Polyurethane can be reshaped by imersing in boilng water or exposing to steam for variiying periods of time according to the wall thickness. Examples: Judkins coronary catheters, Pigtail catheter.

Polyethylene

Polyethylene also is utilized in the making of a cardiac catheter. Its degree of stiffness is in between stiffness of polyurethane and Teflon. Because polyethylene also does not soften much with body temperature it also maintains its shape and is so used for selective cannulation. Polyethylene catheters are more thrombogenic than others. Some polyethylene catheters have a stainless steel mesh braid to increase the rotational control of the catheter and catheter bursting pressure. Examples: Cook pigtail angiographic catheters.

Teflon

Teflon catheters are the stiffest vascular catheters. The advantage of Teflon is the extremely low friction coefficient , which reduces the vascular trauma , increases the insertion and passage, and improves flow rates of contrast media. Teflon has poor catheter memory and therefore more appropriate for nonselective catheterization .If needed Teflon can be reshaped but a heat gun of 720 F is needed.Eg.brockenbrough.

Polyvinyl chloride

PVC catheters are the most soft among the catheters available. This characteristic makes the material quite supple and flexible and therefore ideal for flow directed catheters. PVC has a high friction coefficient which may reduce the ease of catheter passage and increase the incidence of venous spasm. They have increased thrimbogenicity and very poor memory. PVC is most hydrophilic with high rate of moisture absorption. Examples: Swan-Ganz catheters, Berman angiographic catheter.

Principles of Catheter Construction

Cardiac catheters may be composed of one or several layers. In most multi layered catheters ,one tube is stretched or "extruded" over another to form a bond. In catheters composed of several layers the components determine the performance characteristics. Most multi layered catheters consist of an inner tube of Teflon ,over which is a tube of nylon , woven Dacron , or stainless steel braiding .A tube of polyethylene or polyurethane is then heated and extruded over the two inner layers to bond firmly as a third or external later of the catheter. Other type might consist of a polyurethane core covered by a stainless steel braiding and an external polyurethane jacket.

The inner Teflon layer provides a smooth surface through which guide wires and contrast agents can pass through , and should be non thrombogenic. The thickness of the filaments and density of wire or nylon braiding in the middle layer and the type of plastic used determines the stiffness and the torque control of the catheter. Components of the external layers are also important .The external layer must be impregnated with a radioopaque material such as barium or bismuth . This process may spften the catheter material and may produce fine pitting of the surface of the catheter leading to increased thrombogenicity. To overcome this materials may be incorporated in the middle layer of the catheter or a coating of silicone is applied over that.

Now many companies are giving as "high flow" catheters . The high flow design incorporates a thinner catheter wall and hence a larger inner diameter for a given external diameter. This allows for higher contrast flow rates at a specific injection pressure. The high flow concept become particularly important in using diagnostic catheters of small diameter and in guide catheters of angioplasty ,where a large lumen is necessary for contrast injection around a balloon catheter or for placement of more than one dilating systems or intracoronary guide wires.

Catheter sizes

Diameter

Catheters for angiographic use are sized by external and internal diameters and length. The internal diameter of a catheter is specified either by actual diameter in thousands of an inch or millimeters, or by the maximum diameter of the guide wire which can be passed through the catheter. The external diameters is expressed in French which is obtained by multiplying the actual diameter in millimeters by 3.0.French sizes from 5Fr. to 8Fr. are used in coronary angiography and usual catheter has a length of 100 cm's. External and lumen diameter measurements in Standard, Thinwalled and super high flow catheters.

French size	External Diameter		Internal Diameter			
			Thin walled		Super high flow	
	Inches	mm	inches	mm	inches	mm
5	0.065	1.67	0.044	1.08	0.052	1.28
6	0.078	2.00	0.050	1.27	0.056	1.42
7	0.092	2.34	0.056	1.42	0.061	1.55
8	0.104	2.64	0.063	1.60	a	a

Techniques for selective coronary angiography

Mainly there are three methods for the selective cannulation of the human coronaries. The Sone's technique, The Judkin's technique and The Amplatz technique.Sone's method is a brachial approach and the other two are femoral approaches the Amplatz technique can be applied from the brachial approach also.

The Sone's Catheter and technique

Through a small antecubital cut down the brachial artery is isolated .The right brachial artery is preferred. Usual CAG is done with a standard 6French catheter and with a length of 80cm.The advantage of the Sone's catheter is that its shaft is rigid at body temperature. This provides excellent torque control in manipulating the catheter through the subclavian and tip control in the high velocity blood flow in the ascending aorta.

The tip of Sone's catheter tapers into 5.5 Fr diameter over the distal 1-2 cm depending upon the type used that is type I longer than type II .The two types have tapered sections of different length. The tapering provides the distal portion much more flexible and favors directing the catheter tip towards the coronary ostium by enabling this portion to a J-shaped tip.The Sones catheter has an end hole and two or four side holes which enables the tip stability during injections. Canulation process is as follows; Advance the catheter top the central aorta . If the subclavian artery is tortuous ask the patient to take a deep inspiration and raise the shoulder or turn the head towards the left when you manipulate the catheter. If it is not successful put a J tip guide wire to negotiate the vessel without causing injury to the vessel.

Advance the catheter to the sinus of valsalva in the Left anterior oblique position and press it against the aortic valve forming a J loop approximately 1.5 inches in the right arch. Attempt to engage the left coronary ostium by moving the catheter up and down in an alternating advancement and withdrawal passion while maintaining its J shape Having the patient in deep inspiration helps in this maneur. Small amount of contrast injections can be done to make sure the catheter position whether it is in the coronary ostium if it is the tip motion will be reduced by gently advancing the catheter stabilize the catheter check the pressure for any kind of Damping if there is Left main disease the pressure forms will be damped .Then proceed with selective injections in different planes .If LMCA disease is suspecting pull the catheter back to the sinus and do an injection there.

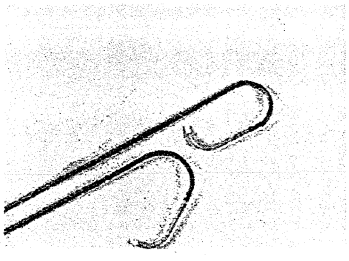
After satisfactory completion of the left angio pull out the catheter from the left ostium maintaining a small loop in the tip. Then rotate the catheter shaft clockwise and slowly withdrawing the catheter tip to phase the tip anteriorly and into the right sinus of valsalva. A rapid in and out movement of the catheter over a few millimeters allow in transmitting this torque.As the catheter tip enters the right coronary sinus it usually falls into the right coronary ostium .Observe the aortic pressures if it appears damped never inject against a damped pressure ,adjust the position of the catheter to get good pressures and then do the injections. Sone's catheter also cab be used for Left ventriculography. The catheter can simply entered into the LV and do the angio there. The major advantage of the Sones method is that a single catheter can be used for all the cannulation.

The Judkins catheters and technique

In 1967 Dr.Melvin Judkins introduced another effective technique for selective cannulation of the coronary arteries in a more simple way which is now used as a standard technique of coronary cannulation world wide .Three catheters are usually needed for a complete study – a Left coronary catheter(LCA), a Right coronary catheter (RCA), and a pigtail angiographic catheter.

Cannulation of the Left coronary artery

The picture shows a Judkins Left coronary catheter.



The Judkins Left coronary catheter is made up of polyurethane and has a length of 100 cms and available in 5 to 8 French diameters. The catheter ha two curves at the distal end the first curve is called the primary curve and it is 90 degree the second curve is called the secondary curve and it is usually 180 degree for an LCA catheter. The rest is the body of the catheter and at the proximal end there is a hub for the attachments of luer locks.For a 6Fr and 7Fr catheters the tip of the catheter tapers into a 5.2 French to enable the entry into the coronary ostium. Usually the Judkins catheters are classified in accordance with the length of the segment between the primary and the secondary curve. It can be 3.0cm, 3.5cm, 4.0 cm, 5.0 cm or 6.0 cm. In accordance with this length we call them JL3.0, JL3.5, JL4.0 etc. This difference in length enables us to choose different catheters for the cannulation of coronaries with usual and dilated aortic roots. Usually for an adult Male we use JL4.0 and for a female a JL3.5 is suitable. For high coronary ostial take off a JL3.0 may be helpful and for dilated aortas due to Aortic stenosis and all a JL5.0 may be helpful to cannulate the coronary ostium.

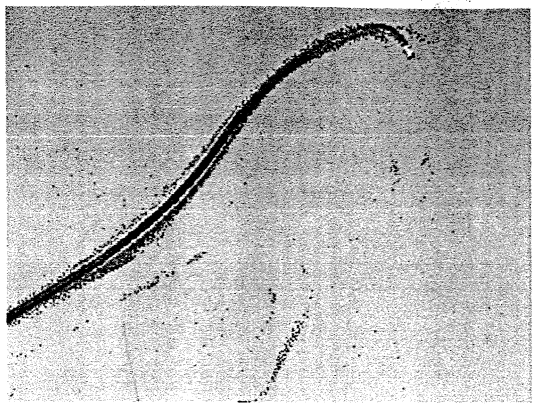
So before choosing the LCA catheter have a look at the chest X-ray and decide the catheter type and go. Insert the coronary artery catheter through the sheath with a safety guide wire ,with the guide wire leading and advance it with the aid of fluoroscopy via the femoral-iliac arteries and into the distal aortic arch. As it reaches the arch advance the catheter first so that the curves of the catheter helps to take the arch curve and goes to the ascending aorta. Remove the guide wire aspirate and flush the catheter and connect to the manifold for pressure recordings , if it seems ok fill the catheter with contrast and slowly advance the catheter down the medial wall of the ascending aorta . If the correct size has been selected the catheter will rotate slightly and seek and engage the coronary ostium without requiring further manipulation

Connect the pressures and look for any damping,if it is there, there may be an LMCA disease.Adjust the catheter for good pressure and inject small amount f contrast into the coronary for confirmation and perform the angiograms in different angulation with a hand injection of 6 to 8 ml of contrast medium over 2 to 3 seconds. After the left coronary injections pullout the LCA catheter with wire inserted .

Cannulation of the Right coronary artery

Similar to the LCA catheter RCA catheter is also made up of polyurethane and has a length of 100 cm, and available at a diameter of 5 Fr. to 8 Fr. It has two curves at the distal end the primary curve of an RCA is 90 degree and the secondary curve is 30 degree only. It also has different types in accordance with the length of the segment between the primary and the secondary curve as 3.5 and 4.0, so we usually refer RCA catheters as JR3.5 or JR 4.0 . RCA catheter tip also tapers into a 5.2 Fr.

The picture shows an RCA catheter.



Advance the RCA catheter to the ascending aorta with a J tip guide wire inserted in the Antero-posterior view and then go to the Left anterior oblique(LAO) view so that the Right coronary origin can be seen clearly. Advance the catheter to 1 to 2 cm above the level of the left coronary ostium and 2 to 4 cm above the level of the aortic valve.Very slowly apply rotation to the catheter hub , do not give excessive torque, as the rotation reaches 60 degree the catheter tip will rotate anteriorly and to the right and will fall 2 to 3 cm into the right coronary sinus. Continue small rotation and observe the catheter tip drops into the right coronary ostium with a jerk. Check the pressures to know wheather there is any damping if so adjust the catheter because the catheter may be in a small conus branch. Proceed with injections in different angulations.

Left ventriculography

Left ventricular angio is usually done with a pigtail catheter. A pigtail angiographic catheter is made up of polyurethane or poly ethylene with a tapered tip, the terminal 5 cm of which is coiled back into itself in a tight loop. The catheter is available with an end hole and two or three pairs of side holes. The catheter has a length of 110cm

The pigtail catheter allows safer passage across cardiac valves and safer injection of contrast medium into the ventricle .The coiled up tip reduces the incidence of ventricular arrhythmias ,catheter recoil, intramyocardial injection and cardiac perforation. The pigtail usually crosses the aortic valve easily but usually the tip must be straightened with a slightly protruding guide wire to cross stenotic aortic valve. The risk of thrombus formation is large with pigtail so effective flushing is needed. After entering the LV the pigtail is aspirated and flushed and then connected to the manifold for the LV pressure. Record LV pressure and then do the LV angio in RAO and LAO CRAN angulations with the help of a pressure injector.

The Amplatz catheter and technique

The Amplatz type of catheter is made up of either polyurethane or poly ethylene .Dr. Kurtz Amplatz designed the catheter for selective cannulation of the Left and Right coronaries. The uniquely designed tip and curve of this catheter are shaped to fit the shape of the sinus of valsalva. The catheter is available in several broad secondary curves AL1.0 to AL4.0 to accommodate variations in the aortic root size .The AL1.0 andAL2.0 are suitable for cannulation of a normal aortic root and the other two are for a

dilated aortic root. The amplatz right coronary catheter is available only in three secondary curves AR1.0 to AR 3.0 .In this AR1.0 is used for patients with normal aortic root and the other two are used for anomalous right coronary artery origin and sometimes for bye pass graft visualization. The Amplatz catheter can be selectively placed in the right or left coronary artery if placement of the Judkins catheter is a failure. It can be used both in femoral as well as in brachial approach.

For catheterization of the left coronary the secondary curve of the AL catheter rests on the noncoronary posterior aortic cusp, with its tip pointing towards the left main coronary ostium. Once the catheter is engaged a slight withdrawal will allow the tip to sit well in the coronary ostium. The right coronary catheter may initially points towards the left coronary sinus , it should be withdrawn slightly and rotated clockwise until the tip points towards the right coronary artery and the secondary curve rests against the left aortic cusp . Slight advancement and withdrawal will engage the right coronary ostium.

Catheters for Artery and Vein grafts

The Judkins and Amplatz right coronary catheters are frequently successful for selective catheterization of the vein grafts. The Left Coronary Bye pass(LCB), and the Right Coronary Bye pass(RCB) are available for the cannulation of the vein grafts. And the Left Internal Mammary Artery (LIMA) for the cannulation of mammary artery grafts.

Left Coronary By pass Catheter

Melvin Judkins designed this catheter for visualization of the left coronary bye pass graft that affixed to the left anterior descending or the left circumflex artery. The primary curve is 90 degree and the secondary curve is approximately 70 degrees. It is made up of polyurethane and has a length of 100cm.

Right Coronary By pass Catheter

This catheter is same as the left except with a primary curve of 110 to 120 degrees and a secondary curve of 30 degrees. It can be used for the visualization of either left or right coronary artery grafts.

Coronary By pass Catheter II

This is employed for visualization of the right coronary bye pass grafts located superiorly on the aorta. It has two oppositely directed circular bends with a small 90 degree curved tip.

Internal Mammary By pass Catheter

Designhned for opacification of the right and left internal mammary arte grafts,this is similar to the Judkins right coronary catheter with a shallower primary cur of 80 to 85 degrees and a 1.5 to 2.0 cm tip.

Cannulation of Grafts

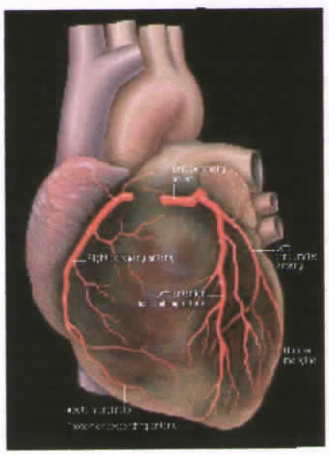
The aortic insertion of the vein grafts are to the antero lateral surface of t ascending aorta 2 to 3 cm above the native coronary arteries. Usually the graft to t right coronary will be the lowest .Above and to the left is the origin of the graft to t LAD, and the most superior and leftward in origin are usually the grafts to the diagor and marginal rteries.

Cannulation of Mammary Arteries

The sharply angled LIMA catheter facilitates the entry into the origin of t mammary artery , which branches off from the subclavian artery at an acute angle .T catheter is first placed in the aortic arch in a neutral position with its tip pointi downward. It is then rotated clock wise until it falls into the subclavian artery . It advanced with a slight anterior rotation of its tip until its tip engages the inter mammary artery, just distal to the origin of the left vertebral artery.

Anatomic distribution of the coronaries

The picture shown here shows the normal course of coronary arteries.



The Left Main Coronary Artery

The left main coronary artery is a short trunk arising from the left aortic sinus running from its ostium through the aortic wall to left and anteriorly in the anterior atrio ventricular sulcus . The Left main trunk varies considerably in size considerably ranging from a few millimeters to 4 cm. As a rule the Left main coronary artery bifurcates into the left anterior descending and the left circumflex. On occasion it may trifurcates into an LAD an LCX and a Ramus inter mediuns.

Angiographic anatomy

In the Antero posterior and in the Right anterior oblique(RAO) projection the left main appears fore shortened and its orifice is not visualized. In the LAO view the LMCA is seen in profile .So the proximal LMCA is seen in LAO and the distal LMCA is seen in RAP projections.

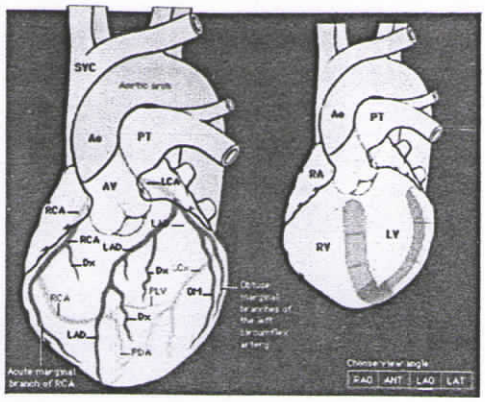
The Left Anterior descending artery

The left anterior descending is usually a direct continuation of the left main coronary artery. It runs in the anterior interventriculr groove and thus varies in position depending upon the presence of right or left ventricular hypertrophy. At sometimes it may leave the interventricular sulcus and cross over the ventricle. The LAD is usually classified into three types in accordance with its approach towards the apex. If the LAD is not reaching up to the apex then it is called a Type I LAD .If it is reaching upto the apex it is then called a Type II LAD, and if it reaches the apex and continues as the posterior ascending artery it is called Type III LAD.

The most important branches of the left anterior descending arteries are branches to the inter ventricular septum , which are called the septal branches. These anterior septal branches anastamose with the posterior septal branches arising from the posterior descending artery. The diagonal branches arise at oblique angles to the LAD to descend diagonally towards the obstuse margin of the heart. The diagonal branches vary in number from 1 to 5 then we name them as D1,D2....D5. They are supplying the anterior wall of the LV.

Angiographic anatomy

The Left anterior descending artery is well seen in cranial angulations with the C-arm in AP,LAO, and RAO projections. Lateral view also helps in visualizing the LAD. The brilliant view for the proximal and mid LAD is an AP Cranial view. It clearly shows the septal branches going deep into the septum and the diagonal branches towards the obstuse margin of the heart. An LAO Cranial view shows the separation of the LAD and the Diagonal it is a best view for diagonal branches. An RAO Cranial view shows the proximal LAD nicely. An LAO Caudal view shows the bifurcation of the LMCA into the LAD and LCX and the proximal part of the LAD can be visualized. The picture below shows the LAD and its branches. It is an LAO Cranial angulation. And an AP CRANIAL



The Left Circumflex Artery

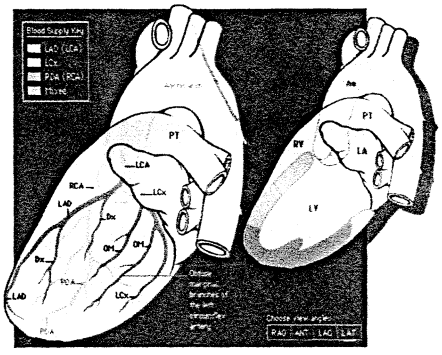
The vessel is a branch of Left main coronary artery. It may v considerably in size depending upon whether the posterior descending artery is a bran of the right coronary or the circumflex coronary itself. In some individuals with stron dominant right coronary artery the circumflex will be too small and may be absent. T circumflex coronary artery measures generally 2mm to 4mm in external diameter . It ru through the left atrioventricilar groove then turns down and goes to the postero-lateral p of the LV . Running parallel to it and superficial or anterior to it is the great cardiac v and it unites with the large atrial vein to the origin of the coronary sinus. If we are runn the venous phase of the angio the coronary sinus just fills exactly over where t circumflex is.

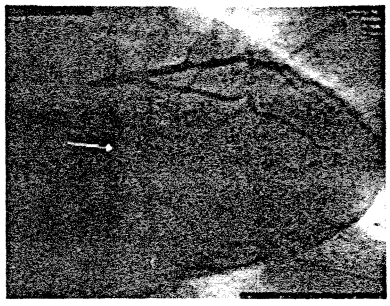
The first branch arising from the circumflex artery is in 40% of the peo the sinus node artery which supplies the SA node –the pacemaker of the heart. Kuge artery may also arise from the proximal portion of the circumflex ,this vessel forms important collateral pathway when the right or left coronary arteries are occluded a supply of blood to the posterior left ventricle. Kugel's artery is a vessel which runs in t inter atrial septum. The next major artery arising from the circumflex is the left at circumflex artery. In case of dominant right coronary artery the LA circumflex may be t only artery arising from the LCX. It supplies the lower portion of the left atrium . Margi branches are important branches arising from the circumflex, they course towards t obtuse margin of the heart and then curve towards the apex, the so called artery is t Obtuse marginal artery which supplies the lateral wall of the left ventricle. Obtuse mar may be one or more so we name them as OM1, OM2...

Angiographic anatomy

The Left circumflex artery is best seen in caudal angulations. An AP Caudal a RAO Caudal are the standard views which are used for the clear view of the LCX . Late as well as LAO positions also shows LCX clearly. On the LAO position the circumf artery proper courses to the left from the left coronary bifurcation and then it tu

downwards and slightly medially following the atrioventricular groove. This helps distinguish it from the marginal branch which remains laterally positioned in this projection. On the lateral film the circumflex artery always hugs the back of the heart and is always posterior to the marginal branch. Next for the proximal portion of the circumflex artery the Spider view(LAO CAUDAL) is usually helpful. This view helps in the proximal LAD,LCX, and their bifurcation. It also shows a Ramus intermedians if it is there. The following pictures shows the circumflex artery in the Lateral as well as in the RAO Caudal angulations.





The Right coronary artery

The Right coronary artery arises from the right aortic sinus. It runs through the right atrtioventricular groove turns posteriorly and continues through the posterior interventricular branches. Its main branches are in 60% SA nodal artery arises form RCA ,which supplies the sinus node. Next is a Conus artery arising from RCA but it is only in 35% in 65% of people the onus artery has separate origin. The Conus artery supplies the RVOT. Next the RCA turns in the acute margin of the heart from there the next artery called the Acute margin arises, which supplies the Right ventricular free wall. On the posterior side the RCA continues through posterior interventricular groove, at the crux of the heart it gives the next branch called the AV nodal branch , which supplies the AV node. Now the RCA continues as the posterior descending artery (PDA) and give several posterior septal branches which supplies the posterior part of the septum. Next branches are the posterior left ventricular branches(PLVB) which supplies the posterior part of the left ventricle. RCA is said to be a Dominant vessel if it continues as PDA and gives rise to posterior left ventricular branches.

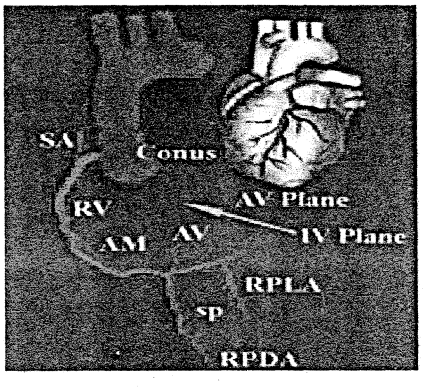
Angiographic Anatomy

The right coronary ostium is pointing somewhat anteriorly and if the patient is catheterized in the AP position, the catheter must point directly at the operator. So the best view to cannulate right coronary artery is the LAO view in which the artery point directly to the right (to the left of the operator). The two most commonly utilized views are

the LAO and RAO projections. Usually a left lateral position is seen to be useful in case of difficulty in cannulation in the LAO position.

In the LAO projection the , the conus branch comes forward towards the pulmonary artery, where as the sinus node branch bends medially and posteriorly towards the superior vena cava. In this position the marginal branch may be difficult to differentiate from the main coronary artery as both appear to have a parallel course. Its identification will be easier in the RAO position. In the LAO and Lateral projections the crux may be identified by the presence of an inverted 'U' .The posterior descending artery is usually foreshortened in the LAO Cranial view it is seen nicely opened in AP Cranial view. It is usually goes slightly downwards and to the left as it courses in the intervenricular groove, RAO also give good view of PDA. In the RAO projection the Conus branch is usually well seen and easy to identify as it passes anteriorly over the pulmonary outflow tract. The Lateral view is good for visualization of the mid RCA and an LAO Caudal is useful for the RCA ostium. An AP Cranial will be useful for the PDA ostium.

The following pictures shows the RCA in the LAO Cranial projection.



In some people the Right coronary artery may have an extra bend just after the proximal part of the RCA, which makes the Right Coronary artery looks like a Shepherds stick so such an RCA is called a Shepherds Crook RCA, which is of importance when we consider that RCA for any kind of intervention .The right coronary artery is the major source supply to the conduction system of the heart , so take adequate precautions doing a coronary angiogram. Never inject contrast into an RCA which is wedging, try to adjust the catheter to get a good pressure form then only inject otherwise the patient may go into serious arrhythmias like ventricular fibrillation.

Description of lesion characteristics

It is a need to assess the coronary arterial tree after the coronary angiogram. If there we must be able classify the type of lesions because if we are planning for an

intervention of for a surgery or only for medical follow up like that. The American College of Cardiology /American Heart Association task force had classified the lesions into three arbitrary types called Type A,B and C. That is fully based on the lesions pathophysiologic characteristics but based on the anatomic positions also we can classify them as Angulated lesions, Bifurcation lesion, Ostial lesions. Now we can discuss each type detailed.

Type A Lesions

These are the lesions which has a high success rate of more than 85% on revascularisation, these are low risk lesions for an intervention. Type A lesions will be discrete (less than 10mm length) , concentric and readily accessible lesions. The lesion will be on a nonangulated segment with a smooth contour. There will be little or no calcification and no thrombus. They will not be in ostial location ,without any major branch included and will be subtotal only.

Type B Lesions

Type B lesions has only moderate success rate of 60 to 85%. They are tubular (10 to 20 mm length), eccentric and with moderate tortuosity of proximal segments . The lesion will be on a moderately angulated segment of >45 degree with an irregular contour. There will be moderate to severe calcification and some thrombus may be present. They re total occlusions of less than three months old , and ostial in location and may be a bifurcation lesion requiring double guide wires.

Type C Lesions

Type C lesions has success rate of less than 60% and are high risk. They are diffusely diseased (greater than 20 mm length), with excessive tortuosity of proximal segment and may be extremely angulated segments with > 90 degrees. They are of Total occlusions greater than three months old.

Angulated lesions

Here the lesions will be located within an angulated segment of coronary and are not very simple for an intervention. Earlier balloon catheters were non confirmable and when inflated at vessel bend points suddenly straightened up exerting non coaxial stress. This rapid chande in shape resulted in high risk of dissection. But introduction of conformable balloons which are constructed with polyethylene terephthelene shown improved action.

Bifurcation lesions

Severe stenosis which are located at the branching of two vessels are called bifurcation lesions. For example the LAD and Diagonal branch and the LCX an OM branch point. They are associated with low success rate and high risk. For such

bifurcation we are doing the "kissing balloon angioplasty" in which two wires are passed one through the main vessel and the other wire is passed into the major branch to protect it.

Calcified lesions

They are the most difficult lesions for an intervention in which only rotational atherectomy is possible.

Eccentric Lesions

Most coronary atheroma narrow the vessel lumen circumferentially, though usually some what eccentrically, and PTCA is successful in most of these lesions. Some plaque arise predominantly from one half to three quarters of the circumference of the vessel wall , sparing the remaining segment.

Ostial lesions

Lesions in the beginning of a vessel is termed as Ostial lesions. Such lesions have an elastic recoil even after PTCA .

Complications of coronary angiogram

Coronary angiogram is a relatively safe procedure now. There will not be usually any serious complications and if to the max death is only 0.1% only. The main complications are as follows. Death, Renal insufficiency, Myocardial Infarction, Cerebrovascular Accidents, Arrhythmias like VT or VF, Allergic response to contrast, Brady arrhythmias.

CARDIAC CATH AND HEMODYNAMIC MONITORING

Introduction

The cardiac catheterization other than coronary angiography has its own role in the diagnostic as well as therapeutic intervention. Our main aim in doing a cardiac Cath is recording the intra cardiac pressures, taking blood samples for the calculation of cardiac output and doing angiography in different chambers and related vessels. The cardiac catheterization for adults are usually for the valvular lesions like Mitral stenosis, Mitral regurgitation, Aortic stenosis, Aortic regurgitation. Here the pressure measurements and the gradients are important to decide whether the patient needs a surgery or he an go on medical follow up for some more years.

Indications for Cardiac Cath

1. Valvular lesions like Mitral Stenosis, Aortic stenosis.
2. Valvular insufficiencies like Mitral regurgitation and Aortic regurgitation.

3. Cardiac myopathies like Hypertrophic cardiac myopathy,HOCM.
4. Disease like Endomyocardial fibrosis.
5. Diseases of the pericardium like constrictive pericarditis.
6. Congenital heart disease.

Contra indications for Cardiac Cath

There is no absolute contra indication for not doing a Cath except the patient refusal.

Mitral and aortic stenosis

Determining the severity of a valvular stenosis based on the pressure gradient and flow across the valve is an important aspect of the evaluation in patients with valvular heart disease. The measurement of the pressure gradient alone often is insufficient to distinguish significant from insignificant valvular stenosis. In patients with aortic stenosis, a true transvalvular pressure gradient should be obtained whenever possible. Although measuring the gradient between the left ventricle and the femoral artery is convenient, downstream augmentation of the pressure signal and delay in pressure transmission between the proximal aorta and femoral artery may alter the pressure waveform and introduce errors. This is especially important in patients with a low pressure gradient and cardiac output.

In many patients, left ventricular-femoral artery pressure gradients may suffice as an estimate of the severity of aortic stenosis, especially if the gradient is high and the cardiac output is preserved. The normal aortic valve area is 2.6-3.5 cm^2 in adults. Valve areas of 0.8 cm^2 or smaller represent severe aortic stenosis. In patients with Mitral stenosis, the valve gradient usually is measured using the left ventricular and pulmonary capillary wedge pressure. The pulmonary wedge pressure tracing must be realigned with the left ventricular tracing for accurate mean gradient determination. However, the most accurate method uses the left atrial and left ventricular pressure. This requires a transseptal catheterization approach. The normal Mitral valve area is 4-6 cm^2, and severe Mitral stenosis is present with valve areas smaller than 1.0-1.2 cm^2.

Mitral regurgitation

The severity of Mitral regurgitation can be graded based on the amount of contrast regurgitation from the left ventricle through the incompetent Mitral valve into the left atrium, using the opacification of the left atrium as a guide. The opacification is graded as follows:

Grade 1+ (mild): Regurgitation essentially clears with each beat and never opacifies the entire left atrium.

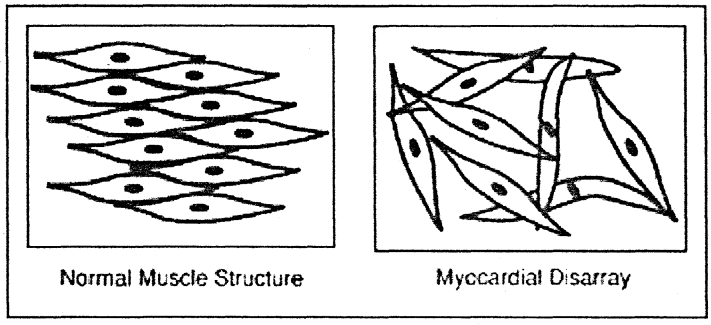
Grade 2+ (moderate): Regurgitation does not clear with 1 beat and opacifies the entire left atrium after several beats.

Grade 3+ (moderately severe): The left atrium is opacified completely and achieves equal opacification to the left ventricle.

Grade 4+ (severe): The entire left atrium is opacified within 1 beat and becomes denser with each beat, with associated refluxing into the pulmonary veins during systole.

Hypertrophic Cardiomyopathy

Cardiomyopathy is a condition in which the muscle of the heart is abnormal in the absence of an apparent cause. This terminology is purely descriptive and is based on the Latin derivation. There are four types of cardiomyopathy: Hypertrophic (HCM), Dilated (DCM), Restrictive (RCM) and Arrhythmogenic Right Ventricular (ARVC). The main feature of Hypertrophic Cardiomyopathy is an excessive thickening of the heart muscle (hypertrophy literally means to thicken). Heart muscle may thicken in normal individuals as a result of high blood pressure or prolonged athletic training. In Hypertrophic Cardiomyopathy, however, the muscle thickening occurs without an obvious cause. In addition, microscopic examination of the heart muscle in Hypertrophic Cardiomyopathy shows that it is abnormal. The normal alignment of muscle cells is absent and this abnormality is called myocardial disarray.



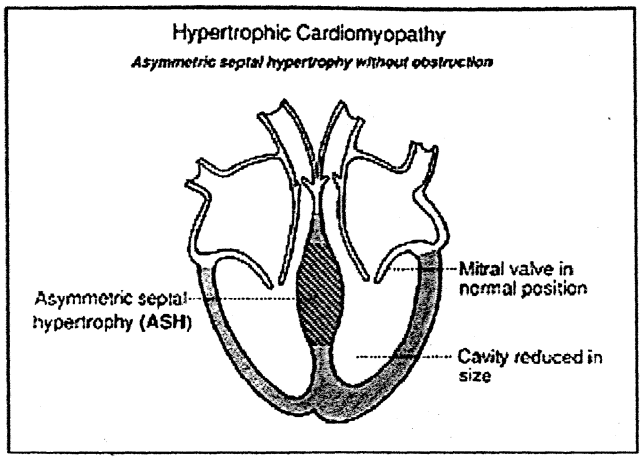
These diagrams contrast the regular, parallel alignment of muscle cells in a normal heart with the irregular, disorganized alignment of muscle cells or myocardial disarray found in some parts of the heart in Hypertrophic Cardiomyopathy.

The heart in hypertrophic cardiomyopathy

The major abnormality of the heart in Hypertrophic Cardiomyopathy is an excessive thickening of the muscle. The distribution of muscle thickening or hypertrophy is variable. The left ventricle is almost always affected and in some patients the muscle of the right ventricle also thickens.

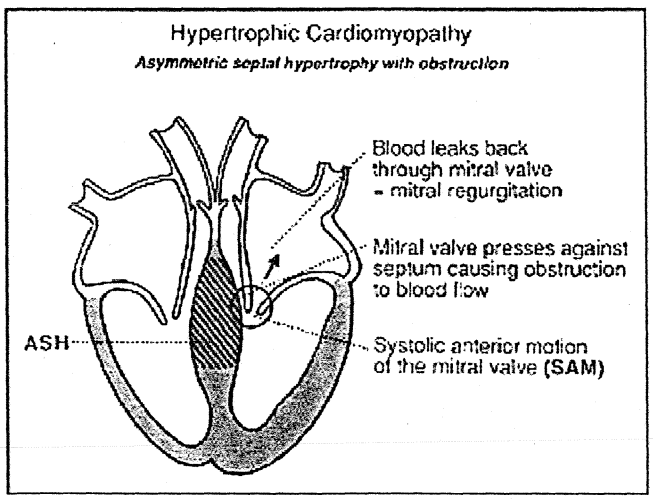
Asymmetric Septal Hypertrophy

Hypertrophic Cardiomyopathy where the muscle thickening occurs predominantly in the septum or dividing wall between the right and left sides of the heart.



This diagram shows the commonest form of Hypertrophic Cardiomyopathy where the muscle thickening occurs mainly in the upper part of the septum. Note that the Mitral valve maintains a normal position. It can be seen from Figure that the hypertrophy is usually greatest in the upper septum, in the area where blood flows out of the heart into the aorta or outflow tract. The muscle thickening in this region may be sufficient to narrow the outflow tract. In such cases during the ejection of the blood flow from the heart, the mitral valve touches the septum (there should normally be a considerable gap between these structures, (Figure). This narrowing of the outflow tract interferes with the normal ejection of blood. It causes turbulent blood flow and sometimes obstruction to flow. The turbulent flow produces a murmur which is audible with a stethoscope. In such patients, the abnormal position of the mitral valve may cause it to leak. This is called mitral regurgitation and may also cause a murmur.

Hypertrophic Cardiomyopathy



In some cases of asymmetric septal hypertrophy obstruction to the outflow of blood from the heart may occur as shown here. Note that the mitral valve now touches the

septum, blocking the outflow tract (Systolic Anterior Motion of the Mitral Valve or SAM). Some blood is leaking back through the mitral valve (mitral regurgitation).

Catheters used in Cardiac Cath

The catheters are usually classified as the left heart catheters and right heart catheters and angiographic catheters. We can now discuss the catheters detailed.

General-purpose right heart catheters

The Cournand catheter

Originally designed by Andre cournand in 1939, the cournand catheter was the first one made specifically for use in right heart. The cournand catheter is a standard wall, end hole ,Woven Dacron catheter with an outer coating of polyurethane. It has very gradual distal curve which lends itself well to passage through the right heart .This is an all purpose catheter in cardiac cath for pressure measurements and blood sampling. The catheter is available in 5 to 8 frenches with 100 to 125 cms.

Lehman catheter

The Lehman catheter is a thin walled version of the cournand catheter with a slightly shorter distal curve. Its thinner wall increases the inside diameter of the catheter and decreases its stiffness.

Goodale-Lubin catheter

The Goodale-Lubin (GL) catheter is constructed of woven Dacron coated with poly urethane. Its unique characteristics are the two laterally opposed side holes near the distal open end it is referred as the birds eye. It is the most common catheter used in the cardiac catheterization lab for pressure measurement and blood sampling .A 6Fr.GL is of 100m length and a 7 Fr. GL is of 125m length. There are standard wall and thin wall versions of this catheter. The curve of the standard wall resembles cournand and that of thin wall resembles Lehman catheter. This catheter is not suited for obtaining a good pulmonary wedge pressure.

Balloon flotation catheters

Balloon floatation catheters are polyvinyl chloride multilumened , right heart catheters with a balloon at the tip ,which, when inflated with CO2 caries the catheter along with the blood flow through the right side of the heart and into the pulmonary artery allowing measurements of PA pressure and PA wedge pressure(PAWP). The PAWP an be obtained through the distal port by the inflation of the balloon of the catheter , which carries the catheter tip forwards and occludes the lumen of the vessel proximal to the tip. Standard adult sizes are from 5 to 7.5 French with a length 110 cm. pediatric sizes are 4 to 5 Fr. with a length of 60 cm.

The ease and accuracy of obtaining the PA wedge pressure has improved with the use of Swan-ganz catheter. Its floatation feature allows passage without fluoroscopy, when used in Cath lab it reduces the flouro time. Because the inflated balloon protrudes slightly beyond the tip of the catheter the trauma to the endocardium can be reduced incidence of arrhythmias and cardiac perforation an be reduced. The soft ,flexible nature of the PVC balloon floatation catheter may prevent difficulties in passage , with looping the catheter in the right atrium or in the right ventricle and predisposing to catheter knotting. Prolonged catheter insertion time further softens the catheter and prevent successful passage out to PA injection of iced saline may stiffen the catheter temporarily.

Balloon floatation catheters are available with two to five lumens , a distal thermistor for the thermo dilution method for cardiac output , and a ventricular pacing lead. The two lumen balloon has a distal port of PAWP and another one for balloon inflation. The three lumen catheter has one extra lumen for RA pressure. The four lumen catheter has one thermistor and computer connecting cables for thermo dilution techniques are there. Balloon catheter without recessed balloons require use of a sheath that is one half or one french greater than the size of the catheter is needed to accommodate the balloon.

Angiographic catheters

The Gensini catheter

The Gensini catheter is made up of woven Dacron with a polyurethane coating and three pairs of laterally opposed oval side holes within 1.5 cm of its open tip. The tip is tapered to provide a lose fit over the appropriate size guide wire. The catheter is intended specifically for percutaneous insertion into the right and left sides of the heart for retrograde aortic and ventricular as well as pulmonary and vena caval angiographic studies. The Gensini catheter is available in sizes 5 through 8 Fr. with 80, 100, and 125 cm in length. The tapered tip of 5 and 6 Fr. fits and 0.025inch wire 7 and 8 Fr. fit an 0.035 inch guide wires.

The straight tip of this catheter is more arrhythmogenic than the tip of the pigtail catheter and often recoils during the injection of contrast medium, particularly often at high rates. The risk of intramyoardial injection and myocardial perforation is also increased with this straight tip and end holed catheter.

National Institute of Health Catheter(NIH)

The NIH catheter is a closed end side holed catheter with a gentle curve. It is available in two versions made by USCI and COOK international. The USCI version has six side holes near its flexible tip. It is a thin wall catheter constructed of Woven Dacron reinforced with a nylon core, which permits injections at very high flow rates. The COOK catheter has four or six side holes near its tip and is constructed of polyethylene with an inner stainless steel braid , which imparts rotational control to the catheter shaft and allows higher injection pressures.

The NIH catheter is used for angiographic visualization of the Right ventricle the arterial or pulmonary vasculature. The NIH is available in sizes 5 through 8 Fr. and lengths 50,80,100 and 125 cm. The NIH catheter is stiff and may recoil and cause extra systoles . The risk of cardiac perforation is considerable in experienced hands.

Eppendorf catheter

The Eppendorf catheter is a thin walled catheter of Woven Dacron with polyurethane coating . Its distinguishing feature is that only the area 20 cm proximal to the hub of the catheter is reinforced with nylon fibers , thus making this catheter less stiff than the NIH catheter while permitting the torque control and the ability to withstand high pressure injections.

Lehman ventriculographic catheter

The Lehman ventriulography catheter is a thin wall closed end ,Woven Dacron catheter with polyurethane coating. The unique feature of this catheter is its slightly curved tip tapered to five French with four side holes beginning 2.5 cm from the tip. This catheter is usually stable in LV and is designed primarily for LV angio. It is available in sizes through 5 to 8 French with 80,100 and 125 Lengths. The tapered tip makes this catheter useful for traversing a tight aortic valve . If the LV hamber is very small it is possible for the curved tip of the lehman catheter to come in contact with the walls of the catheter before the side holes are in the chamber.

Van Tassel angled pigtail catheter

The Van Tassel catheter is similar to high flow pigtail catheter with polyurethane over a thin nylon core. The unique feature of this end holed catheter is an angle of 145 degree 7 cm from the tip. This design is helpful in crossing a stenotic aortic valve because it permits best alignment of the catheter as it approaches the aortic valve orifice. Once the tip is in the ventricle the angle lifts the tip away from the posterior wall of the ventricle and thus reduces myocardial irritability and thus reduces myocardial irritability and the risk of intramyocardial injection.

Berman angiographic catheter

The Berman angiographic catheter is a double lumen PVC catheter with side holes near the balloon tip. The catheter is intended for selective pulmonary angiography in adults or for right and left heart catheterization and angiography in infants and small children. It allows a single catheter for hemodynamic pressure monitoring and angiography it gives PAWP as the side holes are proximal to the balloon.

TERUMO RADIFOCUS GUIDE WIRE

The Terumo guide wire is a very useful wire when we are dealing difficult conditions like crossing a tight pulmonary valve, crossing an aortic valve which is stenotic,

traversing a tortuous artery segment ,crossing a PDA like. It has a more recent design consisting of an elastic alloy core coated with a polyurethane jacket and a hydrophilic coating. This provides for pliable tip, a smooth outer coating, and excellent torque control. This wire design should reduce thrombogenicity and resistance to wire advancement.

THE BLOOD FLOW AND CARDIAC OUTPUT

Introduction

Since we are dealing about the blood, a fluid, and its flow through a vessel we must be familiar with some laws of physics just to calculate two things one is the flow and the other is the resistance to this flow. For that we can use the Poiseuille's Law.

Poiseuille's Law

Being a man who needs simplicity throughout this book I just tells you that Poiseuille's Law an be stated as follows;

$$Q=3.14(Pi-Po)r4/8*eta*l$$

Where Q is the volume of blood flow, Pi-Po is inflow pressure- outflow pressure, r is the radius of the tube, l is the length of the tube, Eta is the viscosity of the fluid.

Taking into account all these factors just think about what happens to the blood flow through a vessel, we an say that the volume of blood flow is directly proportional to the gradient (pressure difference) of pressure, and the fourth power of the vessels radius. And of course inversely proportional to the length of the vessel and the viscosity of the blood. And the Hydraulic resistance, R, can be defined analogy to Ohm's law as the ratio of the mean pressure drop , Delta P, to volume of flow Q across the vascular circuit, so the vascular resistance can be calculated as,

$$R=Pi-Po/Q = 8*eta*l/pi*r4.$$

Estimation of vascular resistance

We can simply apply the equation for hydraulic resistance, to calculate for the vascular resistance. So in front of us there is systemic vascular resistance, total pulmonary resistance and pulmonary vascular resistance.

$$\text{Systemic vascular resistance} = \frac{\text{Mean aortic pressure-Mean RA pressure}}{\text{Systemic blood flow}}$$

$$\text{Total pulmonary resistance} = \frac{\text{Mean PA pressure}}{\text{Pulmonary blood flow}}$$

$$\text{Pulmonary vascular resistance} = \frac{\text{Mean PA pressure - Mean LA pressure}}{\text{Pulmonary blood flow}}$$

The flows are volume flows and are expressed in litre per minute and the mean pressures are expressed in millimeters of mercury. The unit is a Hybrid Resistance Unit(HRU) or sometimes it is termed as woods units.

Oxygen transport

The amount of oxygen that is delivered into the tissues is determined by the flow of rate(cardiac output), the oxygen saturation of arterial blood and the hemoglobin concentration of blood.

Oxygen transport = cardiac output * Arterial oxygen saturation * hemoglobin * 1.34.

Oxygen saturation of blood

Oxygen taken up in the lungs combines chemically and reversibly with the hemoglobin in the blood (oxyhemoglobin) approximately 1.34 cc of oxygen can combine with every gram of saturated hemoglobin. The percentage of total available oxyhemoglobin is referred to as the oxygen saturation of the blood. Normally, arterial blood is fully saturated up to 99% and venous blood has a saturation of 65 to 75 %.

Oxygen content of blood

The actual amount of oxygen that combines with the hemoglobin to form oxyhemoglobin is a product of the total oxygen carrying capacity and the percentage of hemoglobin that saturated with oxygen. This is referred to as the oxygen content of 100 ml of blood and can be calculated as follows,

Oxygen content= Hemoglobin * 1.34ml / gm * So2.

For E.g. The Oxygen content of a fully saturated arterial blood in a patient with 15gm hemoglobin is : 15*1.34*97=19.5 ml. The normal value of arterial oxygen content is 19 to 20 ml/100ml, and that of venous is 12 to 15 ml/100ml.

Oxygen consumption (Vo2)

The amount of oxygen(ml) actually diffused into the body's tissues per minute is referred to as the oxygen consumption. Under most conditions oxygen consumption reflects the tissue demand for oxygen. For the average person at rest oxygen

consumption is approximately 230 to 250 ml/minute (or 125 ml/minute/M2). Oxygen consumption varies directly with alterations in metabolic rate, temperature, and physical work. In our lab we manually calculate the oxygen consumption by finding out the patients Body Surface Area from the nomogram and then multiplying it with the oxygen uptake of different ages.

Arterial-venous oxygen difference

Subtraction of the oxygen content of venous blood from the oxygen content of arterial blood provides the A-Vo2 difference. Normally, the tissues extract approximately 25 to 30 % of available oxygen from hemoglobin. Therefore the oxygen saturation of mixed venous blood is normally between 65 and 75% when arterial blood is almost fully saturated. This results in a venous oxygen content of approximately 14% to 15% when subtracted from a normal arterial oxygen content of 19 vol% gives an A-Vo2 of 4 to 5 vol%. The normal difference between arterial and venous oxygen content is 3.o to 5.5 %. An A-Vo2 difference greater than 5.5 vol% is associated with reduced oxygen delivery and an increased oxygen extraction by the tissues. The A-Vo2 difference reflects the adequacy of cardiac output to match tissue oxygen demand and has long been used clinically to reflect directional changes in cardiac output.

Fick's technique of calculating cardiac output

The fick's formula says that the cardiac output can be calculated by taking the ratio of the oxygen consumption to the arterio venous oxygen difference.

$$\text{Cardiac output} = \frac{\text{Oxygen consumption(Vo2)}}{\text{A-Vo2 difference}}$$

A-Vo2 difference=Arterial oxygen content-Venous oxygen content

PRESSURE MEASUREMENT

What is a pressure wave?

Force is transmitted through a fluid medium as a pressure wave, and an important objective of cardiac catheterization is to assess accurately the forces and therefore the pressure waves generated by various cardiac chambers. For, example a ventricular pressure wave may be considered a complex periodic fluctuation in force per unit area, with one cycle consisting of the time interval from the onset of one systole to the onset of the subsequent systole. The number of times the cycle occurs in one second is termed as the fundamental frequency of cardiac pressure generation. Thus a fundamental frequency of two corresponds to a heart beat of 120 bpm

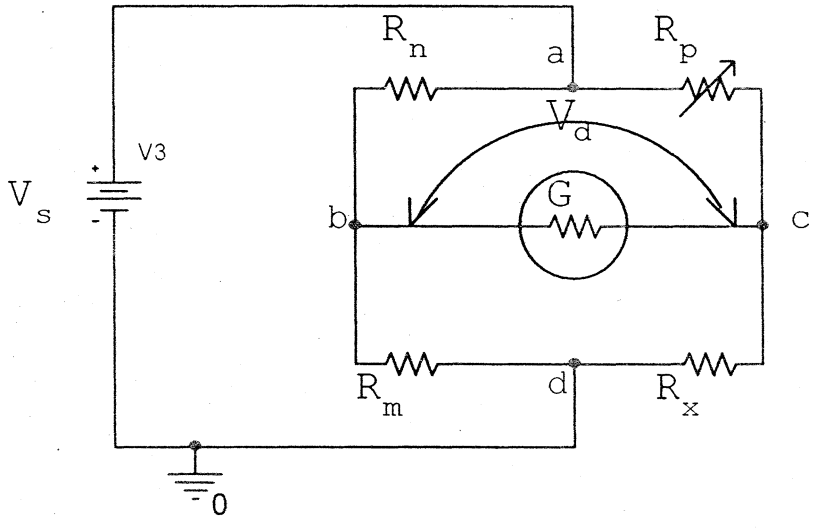
The electrical Strain Gauge transducer

Pressure measurement systems in use today are essentially all electrical strain gauges and employ the principle of the wheat stones bridge.

Wheatstone Bridge

In 1843, the English scientist Charles Wheatstone invented the bridge circuit bearing his name. It is widely used for rapid and precise resistance measurements. Also, similar bridges can be used to measure capacitance or inductance.

Although a commercial Wheatstone bridge may contain precision resistors, switches, and potentiometers to protect the meter movement, and perhaps a variable voltage source, the simplified circuit of Fig. 4.4 will be used here to illustrate the basic principles. The resistances R_n, R_m, and R_p are known, and the resistance R_x is to be determined. Meter G is a sensitive current meter.



In the use of the bridge, the resistance of R_p is adjusted until the meter movement G shows no deflection. Then V_d, the detector voltage, is zero, which means that nodes b and c must be at the same potential. Consequently, the voltage drop from a to b equals that from a to c. Also, the voltage drop from b to d equals that from c to d. Further, since the meter movement current is zero, the current I_1 in resistor R_n also flows in resistor R_m. Similarly, the current I_2 flows through both resistors R_p and R_x. Since $V_{ab} = V_{ac}$, then $I_1R_n = I_2R_p$. Also, since $V_{bd} = V_{cd}$, then $I_1R_m = I_2R_x$. The division of equations yields

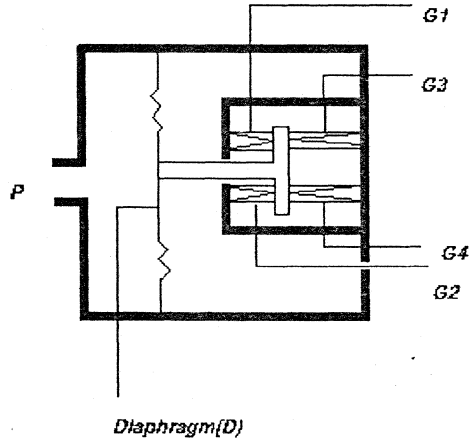
$$\frac{I_2R_x}{I_2R_p} = \frac{I_1R_m}{I_1R_n} \quad \text{which reduces to} \quad R_x = \frac{R_m}{R_n}R_p$$

which is the bridge balance equation. With the bridge balanced, this equation can be used to determine the resistance R_x to an accuracy that is nominally the same as the precision of the resistances R_m, R_n, and R_p.

During preliminary adjustments, when the bridge may be far from a balanced condition, a variable series resistor must protect the meter movement. The value of this resistor must be large compared with that of the meter movement. If, however, the voltage supply V_s is variable, the variable series resistor is not needed. Then, the V_s can be gradually increased from 0 V to some maximum voltage, with repetitive attempts to balance the bridge at intermediate values of voltage. This is an equally safe and accurate method for balancing the bridge.

The strain gauge transducer

The figure shown here is the circuit diagram of a strain gauge transducer,



The strain gauge is a variable resistance transducer whose operation depends on a simple phenomenon : when a wire is stretched its electrical resistance increases. As long as the strain remains well below the elastic limit of wire, there is a wide range within which the increase in resistance is accurately proportional to the increase in length. From the schematic representation the of a pressure transducer pressure is transmitted through port P and acts on diaphragm D, which is vented to atmospheric pressures on its opposite side.

The manner of attachment is such that the increased pressure on diaphragm stretches and therefore increases the resistance of G1 and G2 and decreases the resistance of G3 and G4. In the Wheatstone bridge G1,G2,G3 and G4 are connected electrically and attached to an electrical voltage. If all four resistance are equal , then exactly half the voltage of battery will exist at the junction of G1 and G4 and at the junction of G2 and G3and therefore no current will flow between the output terminals.

However when pressure is applied the resistance are un balanced ,so the junction of G1 and G4 becomes negative , and a current will flow across the out put terminals.

Since movement of the diaphragm D is necessary to produce current flow in the Wheatstone bridge a certain volume of fluid must move through the catheter and connecting tubing to record a pressure. Balancing a transducer is simply a process whereby a variable resistance is interpolated into the circuit so that an arbitrary baseline pressure the voltage across the output terminal can be reduced to zero.

Calibrating and Zeroing of transducer

Once the transducer is in your laboratory before you are starting recording the intra cardiac pressures you must calibrate and adequately Zero the transducer. Before measuring the intracardiac pressures we are taking all the pressures above the atmospheric pressures. The atmospheric pressures is 760mm of mercury, and all the cardiac pressures are above this so by saying that the systolic pressures is 120 mm of mercury it means that the real pressure is 120+760 mmHg. So we have to tell this thing to the transducer that is what we are doing by Zeroing it we tell the transducer to measure from this lowest point only or the Zero level of the transducer is 760 mmHg. For doing this open the transducer to the atmospheres and see whether the oscilloscope is showing its pressure signal line exactly over the Zero line other wise make it so.

For calibrating the transducer you are telling a known pressure to the transducer and make sure he is showing the same. That is we manually give a pressure of 100mmHg to the transducer with the help of sphygmomanometer and see how much is the transducer showing in the oscilloscope if it is wrong tell him that it is 100mm of Hg by entering the value. So from the very next impulse it will measure the correct pressure.

Desired features of a pressure measuring system

1.Sensitivity

The Sensitivity of a measurement system is defined as the ratio of the amplitude of the recorded signal to the amplitude of the input signal. The more rigid the sensing membrane the lower the sensitivity conversely the more flaccid the sensing membrane higher the sensitivity.

2.Frequency Response

A second crucial property of a pressure measuring system is its frequency response. The frequency response of a pressure measuring system may be defined as the ratio of the output amplitude to the input amplitude over a range of frequencies of the input pressure wave. To accurately measure the pressure the frequency ratio must be constant over a sufficient range of frequency variation .Otherwise the amplitude of the major frequency components of the pressure wave forms may be attenuated while minor components are amplified, so that the recorded pressure wave form becomes a distorted

caricature of the physiologic event. The range of good frequency response can be obtained by stiffening the membrane ,but it will be narrowed by making the membrane more flaccid because the flaccid membrane cannot respond well to higher frequencies. Thus Frequency response and sensitivity are related reciprocally and one can be obtained by sacrificing the other.

Natural Frequency and Damping

A third important concept is the natural frequency of a sensing membrane and how it determine she degree of damping required for optimal recording. If the sensing membrane is shock excited in the absence of friction, it would oscillate for an indefinite period of time in simple harmonic motion. The frequency of this motion should be the natural frequency of a system. Any means of dissipating the energy of this system, such as friction, is called Damping. The dynamic characteristics of such a system are largely determined by the natural frequency and the degree of damping the system possess.

Pressure waves

The pressure wave has a systolic component, which is due to the isovolumic contraction of the ventricle, a diastolic component, which is due to the isovolumic relaxation of the ventricle and a mean pressure. There is no mean pressure for the ventricular pressures because there systolic and diastolic pressures are widely different.

Pulse pressure

The difference between the systolic and diastolic pressure is called the pulse pressure.

$$Pp = Ds - Dp.$$

Mean pressure

$$\text{Mean pressure} = \frac{\text{Diastolic pressure} + \text{Pulse pressure}}{3}$$

CHAMBERS AND PRESSURE WAVE FORMS

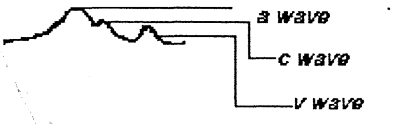
Introduction

An understanding of the normal pressure waveform morphologies is necessary to comprehend the abnormalities that characterize certain pathologic conditions. Simply stated whenever a fluid is added or compressed within a chamber the pressure usually

rises usually when the fluid exits from a chamber or the chamber relaxes the pressure usually falls. One exception of this rule is the early phase of ventricular diastolic filling , when ventricular volume increase after mitral valve opening but pressure continues to decrease because of active relaxation.

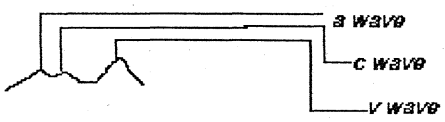
Atrial pressure

The picture shows a typical right atrial pressure tracing.



The right atrial pressure waveform has three positive deflections the a, c, and v waves. The 'a' wave is due to the atrial systole and follows the P wave of the ECG .The height of the a wave is depending on the atrial contractility and the resistance to right ventricular filling . The x descend follows the a wave and represents relaxation of the atrium and downward pulling of the tricuspid annulus by right ventricular contraction. The x descent is followed by a small wave called the 'c' wave due to the protrusion of the tricuspid valve into the right atria. The atrial pressure then peaks into another wave the 'v' wave which represents right ventricular systole. The height of the 'v' wave depends upon the atrial compliance . Usually the right atrial 'a' wave is bigger and the 'v; wave is smaller. The y descend occurs after the 'v' wave reflects the tricuspid valve opening and atrial emptying into the ventricle. Right atrial pressures falls during inhalation and rises during exhalation.

The left atrial pressure waveform is similar to that of the right atrium, although normal left atrial pressure is high reflecting the high pressures of the left side heart. In the left atrium as opposed to the right atrium the 'v' wave is generally higher than the 'a' wave. This occurs because the left atrium is constrained posteriorly by the pulmonary veins. The height of the 'v' wave reflects the left atrial compliance. The picture below shows the left atria; pressure tracing.



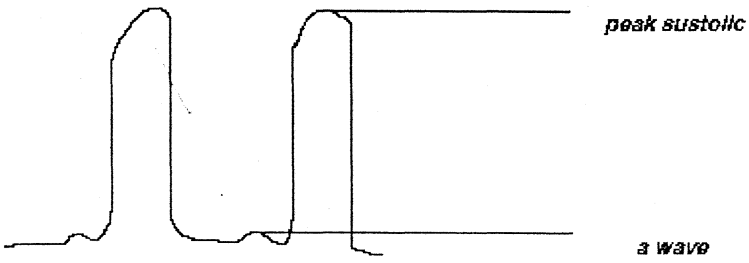
Pulmonary artery wedge pressure

The pulmonary artery wedge pressure waveform is similar to the left atrial pressure waveform but it is slightly damped and delayed as a result of transmission through the lungs. The 'a' wave 'v' wave and x, y descend are seen but the ;c; wave will

not be seen .So with a PA wedge pressure we can see the LA pressure without entering the Left Atrium.

Ventricular pressure

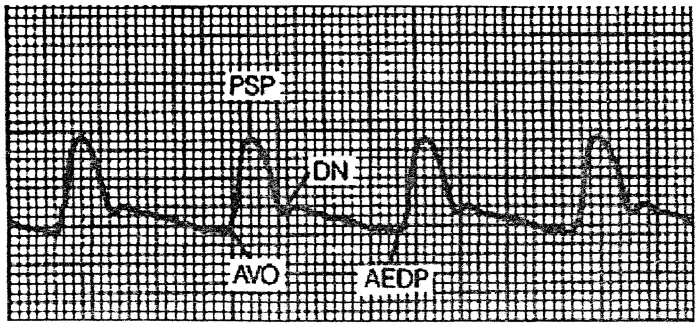
The right and left ventricular waveforms are similar in morphology. They differ mainly with respect to their magnitudes .The picture shows the tracings of a left ventricular pressure.



The durations of systole and isovolumic contraction and relaxation are longer and the ejection period shorter in the left than the right ventricle. There may be a small systolic gradient between the right ventricle and the pulmonary artery. Ventricular diastolic pressure is characterized by an early rapid filling wave during which most of the ventricle fills a slow filling phase and an 'a' wave denoting atrial systolic activity. End diastolic pressure is generally measured at the 'c' point which is the rise in pressure at the time of isovolumic contraction. When the 'C' point is not clearly seen a line is drawn from the R wave on the simultaneous ECG to the ventricular pressure wave form , and this is used as the end diastolic pressure(EDP).

Great vessel pressures

The contour of the central aortic pressure and the pulmonary artery pressure tracing consist of a systolic wave , the incisura (indicating closure of the semi lunar valves, Dicrotic notch) and a gradual decline in the pressure until the next systole. The following figure shows an aortic pressure wave form.



The pulse pressure reflects the stroke volume and compliance of the arterial system . The mean aortic pressure more accurately reflects peripheral resisternce.

Normal pressures of the chambers

Chambers	Range of pressures
Right atrium	
'a' wave	2-7
'v' wave	2-5
mean	1-5
Right ventricle	
Peak systolic	15-30
End diastolic	1-7
Pulmonary artery	
Peak systolic	15-30
End diastolic	4-12
Mean	9-19
PA wedge	
Mean	4-12
Left atrium	
'a' wave	4-16
'v' wave	6-21
Mean	2-12

Left ventricle	
Peak systolic	90-140
End diastolic	5-12
Aorta	
Peak systolic	90-140
End diastolic	60-90
Mean	70-100

ANGIOGRAPHY OF THE CARDIAC CHAMBERS

Cardiac Ventriculography

A left ventriculogram is usually doing for those patient who found to have significant coronary artery disease, or who had a regional wall motion abnormality in Echo, or for post MI patients immediately after the coronary angiogram. We are already discussed the catheters using for an LV angio, The pigtail catheter is the usual catheter using in our lab for LV angiography. The angio can be done in a plane RAO projection and an LAO Cranial view. The picture below shows an LV angiogram with segments marked.The first segment marked is the anterolateral and the next one is the anterobasal, The next is in order the posterobasal, diaphragmatic and apical segments. By drawing the LV for its End diastolic and End systolic volumes we can calculate the Ejection fraction of the LV.

LV angiogram is usually doing for the assessment of Mitral Regurgitation also. The categories are given below.

The severity of Mitral regurgitation can be graded based on the amount of contrast regurgitation from the left ventricle through the incompetent Mitral valve into the left atrium, using the opacification of the left atrium as a guide. The opacification is graded as follows:

Grade 1+ (mild): Regurgitation essentially clears with each beat and never opacifies the entire left atrium.

Grade 2+ (moderate): Regurgitation does not clear with 1 beat and opacifies the entire left atrium after several beats.

Grade 3+ (moderately severe): The left atrium is opacified completely and achieves equal opacification to the left ventricle.

Grade 4+ (severe): The entire left atrium is opacified within 1 beat and becomes denser with each beat, with associated refluxing into the pulmonary veins during systole.

Right ventricular angiography

Right ventricular angiography is usually done in patients with congenital heart disease, especially to prove the RV anatomy and then to profile Ventricular Septal Defects (VSD). The right ventricular angio is usually done with a NIH catheter or sometimes with a Berman angiographic catheter. The angio can be done in Anteroposterior view for anatomy of RV and in Lateral view to show the Right ventricular outflow tract and the pulmonary valve. Usually an RV angiogram is done to profile a ventricular Septal Defect, the vies for different VSD's are given below.

Type of VSD seen	Views to profile VSD
Perimembraneous VSD (SA) VSD	LAO 60 CRANIAL 20
Inlet VSD	LAO 40 CRANIAL 40
Muscular VSD If Anterior	RAO
If Posterior	LAO 60 CRANIAL 20
Sub pulmonic VSD	RAO

Pulmonary angiography

Indications

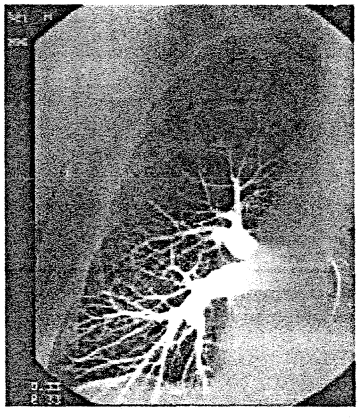
Pulmonary angiography is indicated in the following conditions: Suspected pulmonary embolism that cannot be excluded or confirmed by other studies. Suspected peripheral PA stenosis. Suspected anomalous pulmonary venous drainage. Suspected arteriovenous fistulas. For PA anatomy if it is not clear by Echocardiography.

Contraindications

There are no absolute contraindications to pulmonary angiography only the relative contraindications are: Miderate to severe Pulmonary Artery Hypertension(PAH) associated with RV failure. Severe hypoxemia.

Methods

Usually we are doing the pulmonary angiography with a NIH catheter, sometimes with a pigtail catheter also. If u cannot enter the PA with an NIH the best method is to enter the PA with a GL or with a balloon wedge pressure catheter and then put an exchange wire and then exchange it for a pigtail and do the angio. If NIH is tracking then it is the best catheter to do the angiography. The Usual projection for the opacification of the Branch PA's are LAO 15degree CRANIAL 20 degree. This projection clearly shows the PA anatomy.



INTERVENTIONAL PROCEDURES

PERCUTENEOUS TRANSLUMINAL CORONARY ANGIOPLASTY (PTCA)

INTRODUCTION

Attempts of nonsurgical catheter treatment of obstructive vascular disease date back to 1964, when Charles Dotter and Melvin Judkins described their techniques of peripheral vascular dilation using progressively larger catheters and forecast applications of such techniques to the coronary arteries. But the ultimate result for a successful coronary angioplasty was to Andreas Gruentzig, who designed a small flexible catheter with a distensible balloon at the tip and successfully done the job in 1977. The keys to success appear to be the use of small flexible catheters permitting passage to distal vessels , and the capability of inflating the balloon to a predetermined diameter under increasingly higher inflation pressures. This permitted a controlled compression or splitting of focal atheromatous lesions without major trauma to the entire vessel wall or adjacent segment of the vessel.

Indications of PTCA

Indications of coronary angioplasty is still evolving, when this procedure is to be considered, four important questions must be asked (1) Can the procedure be performed with high probability of success? (2) Will a successful result benefit the patient? (3) Is it better than other available treatment options? (4) Can the procedure be performed safely? A definite indication exist if answer to all these questions is 'yes'. A relative contraindication exist if the answer to any one of it is 'no'. A strong contraindication is present if the answer is 'no' to two or more questions.

The indication and contraindications are still evolving and are determined by (1) the experience and judgment of the person performing the procedure, (2) the technical and support facilities available, (3) the design of available equipment, (4) the capability and experience of the cardio vascular surgical back up team. And (5) the individual, clinical and angiographic characteristics of the patient.

Classic indications

Classic indication exist for the patient who is a candidate for aortocoronary bye pass surgery and has single vessel coronary artery stenosis with the following findings.

1. Recent onset angina not well controlled with medications.
2. Objective evidence of myocardial ischemia.
3. Proximal discrete stenosis.
4. Concentric non calcified lesions.
5. Subtotal obstruction.
6. Good left ventricular function.

Angioplasty in these cases can be performed safely ,with a high probability of success and with substantial benefit to the patient. It is the procedure of choice when medical treatment has not controlled the patient angina.

Extended indications

The extended indications are , Proximal left anterior descending artery or dominant right coronary artery stenosis with demonstrable ischemia, with or without angina. Multiple discrete lesions in the in a single vessel (there are no absolute morphologic contraindication) selected cases of multivessel coronary artery stenosis with relatively refractory angina and demonsration of myocardial ishemia. Acute evolving myocardial infarction with or without the use of Thrombolytic therapy. Saphenous vein bye pass grafts stenosis, preferably discrete and at the distal anastomatic site. Restenosis after a first or second successful angioplasty. Variant angina in the presence of fixed anatomic obstruction. Atherosclerosis with high grade obstruction following cardiac transplantation. Selected patients who are not acceptable candidates for CABG but may benefit from palliative angioplasty, such as patients with severe obstructive lung disease, advanced age, disseminated malignancy, or prior CABG with high risk for reoperation.

Relative contraindications

There are few absolute contraindications to coronary angioplasty. As with indications, contraindications are still evolving and are highly subject to the experience and judgment of the operator and the support available at the facility. Some generally accepted contraindications for elective coronary angioplasty are discussed below.

Left main coronary artery stenosis constitutes a relative contraindication, where morbidity and mortality of coronary angioplasty are high and restenosis may have a clinically malignant presentation and where CABG has proven long term benefits. Most of the exceptions to this are in the category of 'protected' left main lesions where previous surgery has been partially successful. There are also rare emergency situations where angioplasty may be used before surgery to stabilize the patient who has acute left main artery occlusion. "left main equivalent" (when the vessel to be dilated supplies most of the viable left ventricle and the contra lateral vessel supplying an area of old myocardial infarction is obstructed) also represents a relative contra indication.

Additional relative contra indications are, Three vessel coronary artery disease with disabling angina where long term benefits of surgical revascularisation are well established. Critical valvular disease not caused by acute myocardial ischemia. Chronic total occlusions ; if patency is reestablished the chance of resolution is high. Variant angina with documented coronary artery spasm : response of these patients to angioplasty has been unpredictable with a high chance of reoccurrence ; these patients are best treated with vasodilators. Stenosis at the orifice of vessels ; Disease here may not well respond to dilation.

Risk and complication of PTCA

Death	0.1- 0.5 %
Failure to dilate	5 – 10 %
Acute occlusion	2 %
Emergency surgery	2-3 %
Myocardial infarction	1-2 %
RV puncture and pericardial tamponade	1%
Coronary artery aneurysm	rare
Coronary artery rupture	rare
Coronary artery emboli	rare
Iatrogenic left main stenosis due to catheter trauma	rare
Vascular complications	same as diagnostic
Arrhythmias	slightly more

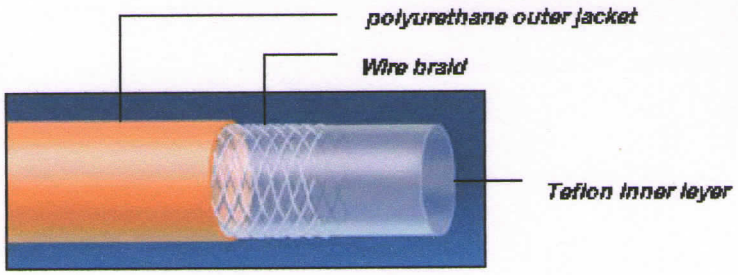
Angioplasty equipment

Advances in coronary angioplasty have been directly related to improvements in equipment design and construction. The physician as well as the technologist must be intimately familiar with the equipment. The Equipment used are The guiding catheter,

The coronary guide wires, the haemostatic apparatus, The Indiflator, The Balloon catheter, The stent catheter.

Guiding catheters

The picture below shows the important parts of a guiding catheter.



Practically all the coronary catheters used for diagnostic angiography are available as guiding catheters, but with different construction. These catheters are made of three layers an inner layer of Teflon to reduce friction between guiding and dilation catheters , a middle layer of either Epoxy and fiber braid or a wire braid to permit torque control and an outer layer of polyurethane providing stiffness and ability to retain preformed shape.

The femoral guiding catheter is 100 cm long and are available in 6 to 8 Fr. outer diameters traditionally the tip of the catheters are short but not tapered , increasing their potential to cause intimal injury. There are also differences in the internal diameter but it depends upon the manufacturer, anyway it has a greater diameter than the diagnostic counter part. The following table shows the difference internal diameter of a guiding versus diagnostic catheter.

Diagnostic catheter	External diameter	Internal diameter	Length	Maximum Guidewire
5 Fr.	1.67	1.14	100	0.038
6 Fr.	2.00	1.27	100	0.038
7 Fr.	2.33	1.55	100	0.038

Guiding catheter	External diameter	Internal diameter	Length	Maximum Guidewire
6 Fr.	2.08	1.80	100	0.038
7 Fr.	2.39	2.08	100	0.038

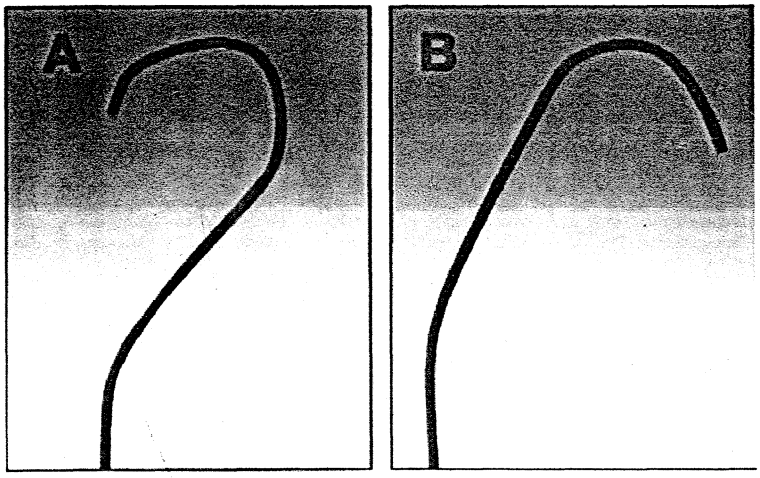
Choice of catheters

The factors considered for choice of the catheter are fit of the diagnostic catheter, and anatomy of the proximal coronary anatomy, aorta, and coronary lesion. There is no single catheter better for all cases. A most important requisite for successful angioplasty is a guiding catheter that (1) will provide proper coaxial seating in the coronary ostium without occluding the flow and (2) is capable of providing support and 'power' for the passage of the dilating catheter through high grade or distal stenosis. Careful review of the diagnostic angiogram and the seating of the catheters used if the diagnostic angiography provide helpful clues.

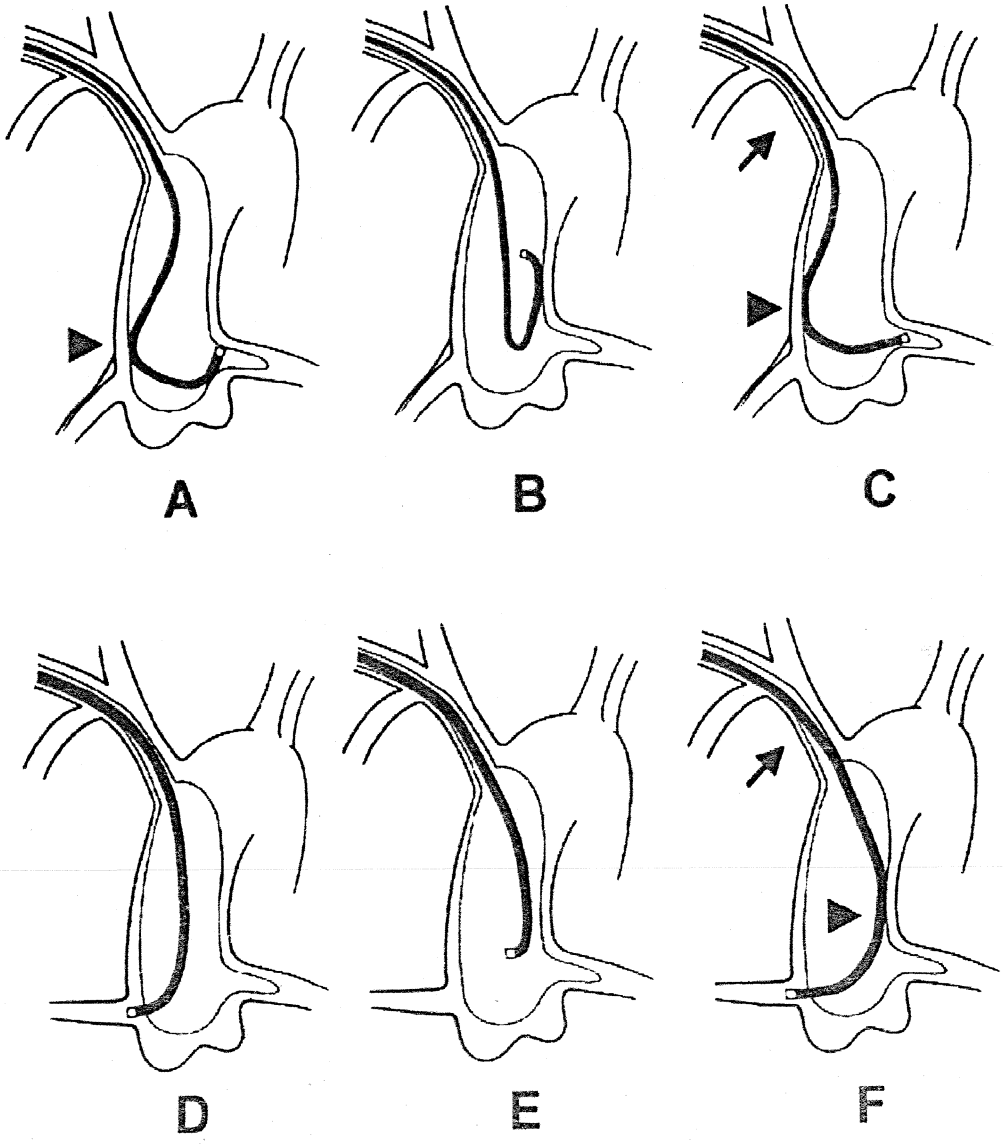
Usually in our lab a Usual Judkins is used for a normal sized LMCA and an Extra Back Up (EBU) is used if the LMCA is long. And usually for the right Judkins right with side holes are used. But the disadvantage of the side holed catheter is, even though there is damping of pressures the catheter will show a good pressure wave and we get fooled by this catheter, and the angiogram will be sub standard.

The Brachial PTCA and the Ikari guiding catheter

The Ikari catheters are shown in Figure 1. At present, the 6 French (Fr) Heartrail shaft has an inner lumen of 0.071 inches (Terumo, Tokyo, Japan). The mechanism whereby the Ikari catheter operates is based on our hypothesis that the lower back-up support in the upper limb approach is due to a different route of approach used by the guide catheters. The biggest difference is the angle between the right subclavian and the brachiocephalic arteries, which causes a reverse angle on the guide catheters. Judkins catheters designed for TFI are inappropriate to fit this angle. Therefore, we incorporated the reverse angle in the design of the guide catheter (Figure 2).



Ikari catheters (A) Ikari catheter left 4.0 (IL 4.0). (B) Ikari catheter right 1.5 (IR 1.5).



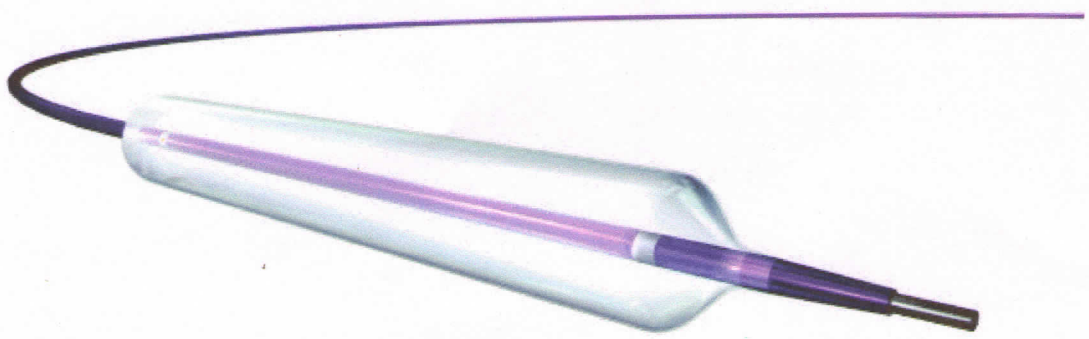
Mechanisms of the Ikari catheter. (A) When Judkins left catheter is applied via a right upper limb approach, the catheter sometimes does not engage coaxially. The distal tip may injure the top of the left main trunk. (B) With Judkins left, the catheter easily loses engagement when the device is pushed into a tight lesion. (C) Ikari L fits the awkward angle at the brachiocephalic artery (black arrow). The angle induces the catheter to attach on the other side of the aorta against the left coronary ostium (triangle). In Judkins, the attachment is limited to a small point of contact (triangle, A) but with Ikari catheter the contact area is much wider (triangle, C). This generates a coaxial engagement and good back-up support (C). (D) When the Judkins right catheter is applied via the right upper limb, the catheter usually does not attach on the other side of the aorta. (E) The catheter easily loses engagement when the device is pushed into a tight lesion. (F) However, the Ikari right catheter fits the angle at the brachiocephalic artery (black arrow). The catheter attaches on the other side of the aorta (triangle). This generates a coaxial engagement and good back-up support.

At present, the Ikari left (IL) catheter is available in three sizes, designated IL 3.5, IL 4.0 and IL 4.5. These represent lengths that correspond to the reverse side of the aorta to the left coronary ostium, and are the same sizes as Judkins catheters. IL 4.0 is the regular size. The Ikari R (IR) catheter has four sizes, designated IR 1.5, IR 2.0 and IR 2.5. These correspond to lengths of the distal tips. The first choice is usually IR 1.0 or IR 1.5. When the tip length is longer, the back-up is stronger but more difficult to engage.

In the Ikari left catheter, the actual length of each site is 0.8 cm at the first short distal line. At the second straight line, the length is 1.1 cm for IL 3.5, 1.3 cm for IL 4.0 and 1.5 cm for IL 4.5. The length is 1.1 cm at the third straight line and 5.9 cm at the fourth straight line. In the IR catheter, the length of each site for the distal straight line is 1.0 cm (IR 1.0), 1.3 cm (IR 1.5), 1.8 cm (IR 2.0) and 2.3 cm (IR 2.5). The length is 1.0 cm at the second angled line and 6.0 cm at the third straight line.

The Balloon catheter

The picture given here shows a balloon catheter using in PTCA.



Types of balloon catheters

There are three types of angioplasty balloon catheters over the wire, monorail and fixed wire balloon catheters.

Over the wire angioplasty balloon catheters

A standard over the wire angioplasty balloon catheter has a central lumen through out the length of the catheter for a guide wire and another separate lumen for the balloon inflation. These balloons are approximately 145 too 155 cm long and can be used with guide wires of various dimensions (0.010 to 0.018). The balloon catheter may have a guide wire lumen that is large enough for pressure measurement and distal contrast injection with the wire in place.

In these systems, the guide wire and balloon catheter move independently. The major advantage is ability to maintain artery access with the guide wire beyond the lesion while exchanging one balloon catheter for another. Using standard technique after the balloon is tracked over the wire the wire may be removed from the balloon if distal injections or pressure measurements are needed, or the wire may be removed or reshaped and reintroduced through the central lumen. A regular wire can be extended from 145cm to 300 cm to help maintain the distal position while the balloon catheter is completely withdrawn over the guide wire and another balloon catheter is exchanged over it .

Over the wire angioplasty balloon catheters have few limitations. These catheters are in general slightly larger than the rapid exchange and fixed wire catheters and their use with small guiding catheters (6Fr) may be more difficult. Despite the very low balloon profile, over the wire balloons may also need very good catheter back up support.

Rapid-exchange (monorail) angioplasty balloon catheters

Rapid-exchange balloon angioplasty catheters were developed to improve exchanging angioplasty balloon catheters by single operators. Rapid exchange catheters have only a variable length of the catheter shaft containing two lumens. One lumen runs the entire length of the catheter and is used for the balloon inflation. The other lumen which extends only a portion of the catheter shaft, houses the guide wire. Because only a limited portion of the balloon requires dual lumen rapid exchange catheters are smaller improving contrast visualization of the artery. Rapid exchange balloon catheters address certain inherent limitations of over the wire systems. First over the wire requires a long guide wire an unnecessary manure for monorail type. We usually use a guide wire of length 175 cm for monorail balloons. Second a single operator can use a rapid-exchange balloon without the aid of other assistants to maintain distal guide wire position.

Limitations of monorail catheters include the need for excellent guiding catheter support and difficulty with simultaneous manipulation of the guide wire, the balloon catheter and the guiding catheter. There may be excessive blood loss at the rotating

hemostatic valve during removal of the balloon catheter. Care must also be taken when the balloon and guide wire are advanced through the guiding catheter to the coronary ostia. If the monorail balloon is advanced in front of the wire, the wire may come out of its short lumen, necessitating the reassembly of the balloon and the guide wire. When monorail catheter with very short rails are used in distal grafts , the free portion of the aorta may loop and kink during the balloon catheter pull back a problem which is difficult to diagnose when a radio opaque wire is used.

Perfusion balloon catheters

The perfusion catheter is a variant of the conventional monorail balloon catheter system with multiple side holes proximal and distal to the balloon communicating directly with the central lumen of the catheter allowing the arterial blood to flow through the proximal side holes, down the central lumen, and out of the distal side holes with the balloon inflated in the lesion. This catheter permits perfusion of the myocardium during balloon inflation.

The perfusion catheter is of particular value when the patient is unable to tolerate the ischemia induced with routine balloon inflations. A perfusion catheter is selected to reduce severe angina, Hypotension from compromised ventricular function, or when prolonged inflations are required. Its disadvantages are related to its bulky size, which makes it difficult to track through the tortuous arterial segment and limited artery opacification.

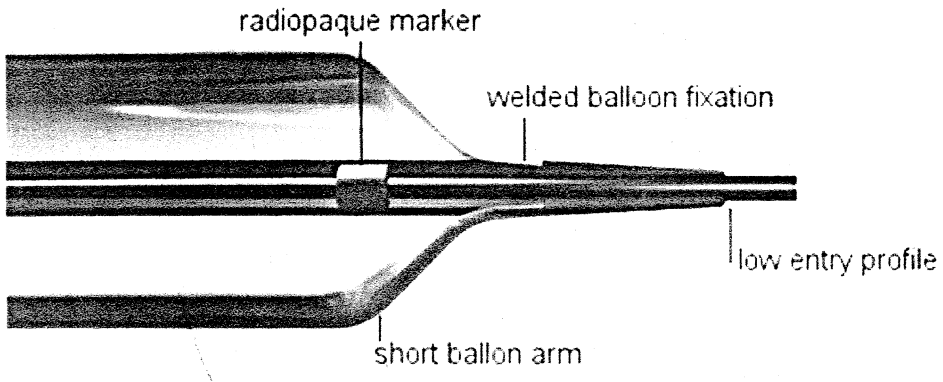
Fixed-wire angioplasty balloon

The fixed wire catheter has the balloon mounted on a central hollow wire with a distal flexible steering tip. The proximal end of the catheter consist of a single non removable port connected to a thin metal tube (hypotube). A core wire extends from the hypotube to the distal steerable tip. This assembly is coated with a thin plastic shaft that enhances flexibility. Fixed balloons have only one enclosed lumen for balloon inflation.

Here the wire cannot be advanced independent of the balloon and the balloon cannot be exchanged without removing the entire system. Since the wire is attached to the distal end of the balloon, there is no central balloon lumen resulting in a lower total profile than the over the wire system. Its principle advantages relate to its low profile, enabling passage through very tight stenosis, and good contrast visualization of the lesion being dilated around the balloon catheter. Fixed wire balloons are particularly useful for distal stenosis, subtotal stenosis, and lesions located in tortuous vasculature. Additionally they may be particularly helpful when small guiding catheters are used.

The limitations of fixed wire catheters include lack of the inherent safety advantage of over the wire and rapid exchange systems because the balloon is mounted on a guide wire. To exchange this catheter for a different balloon size the catheter is removed completely and the lesion is recrossed with a new balloon catheter. A narrowed or dissected lesion may not permit recrossing or advancement of balloon catheter.

The picture below shows a close view of a balloon catheter.



What is Compliance?

The plastic material of the balloon determines its compliance and tensile strength. The main difference in balloons is in its compliance and noncompliance. Inflation of a compliant balloon above nominal pressures will lead to further expansion of the balloon approximately 10% to 20% over the rated diameter. Non compliant balloons on the other hand remains very close to their rated diameter , even when inflated several atmospheres above nominal pressure. Most balloons are coated with low friction surface polymers that facilitate lesion crossing.

Balloon mechanics

The mechanical aspects of balloon angioplasty apply the following fundamental principles,

According to Laplace's law ,wall stress increases with radius. At a given pressure, a larger balloon undergoes more wall stress than a small balloon ,promoting balloon rupture. The arterial segment next to the lesion has a larger luminal radius than the lesion. Thus artery sites adjacent to the lesion may be traumatized. At higher pressures, balloons weaken over time. The balloon burst pressure may be decreased during subsequent inflations of the same balloon. When inflating a balloon above the rated burst pressure, consider limiting the number and duration of inflations.

Balloon diameters always increases with increase in pressure. Noncompliant balloons will grow in diameter by less than 10% over nominal pressures . Compliant balloons increase their diameter by >20%.The balloon diameter- pressure relation is usually linear reflecting the compliance characteristics.

Balloon does not return to original dimensions when deflated. At any given pressure the balloon diameter during a subsequent inflation will be larger than during the first inflation. When dilating two lesions with a compliant balloon, consider approaching the smaller lesion first.

Selection of balloon size

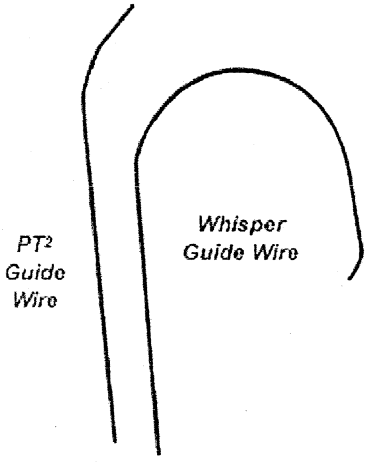
The selection of balloon size is subjective. The balloon size is selected always a 0.5 mm less than the original size of the vessel. If we are dilating an artery with a balloon of same size as of artery it may dissect the segment. More over the selection is operator dependent.

The coronary guide wires

The coronary guide wires are the heart of coronary intervention. They play an important role in coronary angioplasty as they are the carriers of different balloons and stents into the coronaries and their ability helps us to reach the destination point. Like the usual guide wires the coronary guide wires also has the two configurations, the J tip and the straight tip.

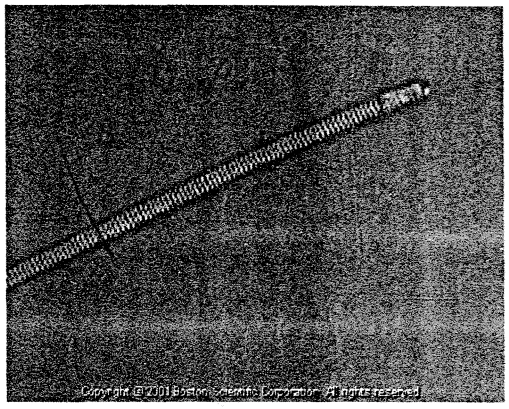
J Tip guide wires

All the coronary guide wires are core flex wires in which the tip of the core is welded to a floppy coil or a shaping ribbon. In J tipped guide wire the core is just ending to 3 cm from the tip and the distal most tip is making an angle of 30 degree and hence the J curve. The core is usually coated with a Hydrophilic polymer till 23 cm from the tip and then only Teflon coating is there. A J tip guide wire is used because the 30 degree angle makes less trauma to the intima and reduce the possibility of dissection in the coronaries. So it is always safe to go with a J tip coronary wire. The picture below shows a J tip guide wire.



Straight tip guide wires

Some coronary wires are manufactured without the 30 degree bending in the tip of the guide wires and they are usually meant for traversing straight coronaries. The core to tip configuration is same like that of the J tip coronary wire. They are prone to trauma to the intima so they are less used in interventions. The figure below shows a straight tip.



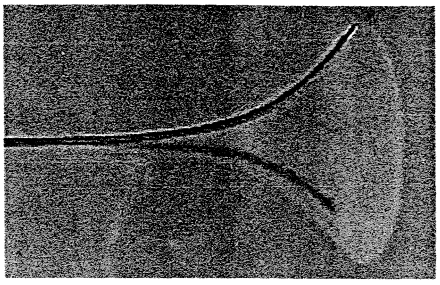
Types of coronary guide wires

Based on the tip flexibility the guide wires are Floppy and Intermediate.

Floppy tip guide wires

A floppy tip guide wire is the one whose core will reach 2 cm short of distal tip. The core tapers and ends to the floppy tip, the floppy tip is usually a platinum coated coil or a shaped ribbon. Floppy wires are available in J tip and in straight configuration. A floppy wire does not make any injury to the intima it is smooth. A floppy wire is usually meant for type A lesions. A subtotal lesion in a less tortuous artery and if the lesion is in the proximal or in the mid vessel we can surely opt a Floppy wire. The 0.014 inch Galeo Floppy wires are usually used in our lab, it has the following features.

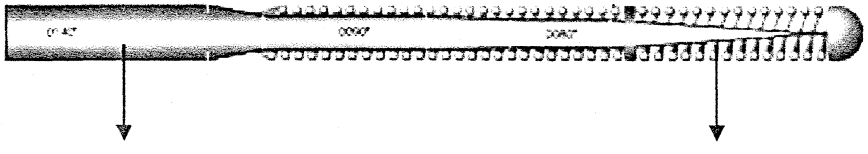
Galeo®/Galeo Hydro coronary guide wires offer a unique combination of push, flexibility and torque transmission. 1:1 handling is guaranteed by the direct connection of coil and core wire (Controlled Transmission Platform). The special BIOCoating® across the entire length of the distal coil optimizes wire movement, catheter movement and crossability. The unique hydrophile Hydro-X coating, applied over the distal 12 cm, optimizes the sliding properties and ensures maximum haemo-compatibility of the Galeo Hydro. All guide wires have a usable length of 175 cm with a radiopaque 3 cm tip. They are moderately visible via x-ray along the entire length and can be extended by 150 cm using the Galeo EW (Extension Wire). Galeo/Galeo Hydro ES 0.014" guide wires are ideally suited to stent procedures. Example: Galeo floppy.



The picture above shows the tip of a Galeo floppy wire with straight tip.

Intermediate wires

This is the second type of coronary guide wire which gives more stiffness to the tip of the guide wire. In this type of guide wires the core of the guide wire tapers to 1cm from the distal tip of the guide wire. By making such a configuration the tip will give you an intermediate support to cross tight coronary lesions. These wires are usually used for total occlusions and chronic total occlusions(CTO). The coating of this wire is similar to the floppy wire. The figure below shows the manufacturing of such an intermediate wire.



Core of the guide wire Intermediate Tip.

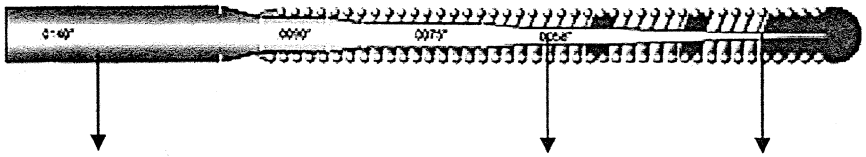
Example; Galeo intermediate, Cross IT.

Extra support guide wires

These are the guide wires used for a tortuous segments especially it helps to make the segment straight. Here the core of the guide wire will be a stiffer one than that of a usual guide wire. By making the core a stiffer one we can increase the stiffness of the shaft. And there by it gives an extra support through out the procedure. The tip of an extra support wire can be a floppy or an intermediate. These type of guide wires help in accessing a distal lesion easily. So we always prefer an extra support wire for a distal

lesion or for a tortuous coronary artery. The figure below shows the manufacture of such an Extra support wire.

Example: Galeo Hydro, Balance Middle Weight(BMW) wires.



Stiffer shaft material Straight tip More radio opaque tip

Coatings on a guide wire

All the coronary guide wires are coated with polymers to reduce the friction. The main coatings are a hydrophilic coating and a micro glide coating. An optional lubricious coating preferably is disposed about the strands, about the polymer layer, if it is present, or about both the strands and the polymer layer. Typically, the proximal portion of the core wire will already have a lubricious coating on it and does not require further coating. Suitable lubricious coatings include hydrophilic materials such as polyvinylpyrrolidone (PVP), polyethylene oxide, polyethylene glycol, cellulosic polymers, and hydrophilic maleic anhydride, or hydrophobic materials such as silicone, PTFE, or FEP.

These coatings are typically applied by dip coating or spray methods, and heat curing may be used. For example, cure temperatures up to about 70°C are used for silicone coatings, and several hundred degrees may be required for PTFE coatings.

In addition to the lubricious coating, bioactive coatings may be applied over all or part of the guide wire. Such coatings also may incorporate materials such as heparin, hirudin and its analogs, or other drugs. These coatings typically are applied by dip coating. Bioactive coatings are desirable to prevent blood clotting or for delivery of drugs to a specific site.

Characteristics of guide wires

The following are the desirable or necessary characteristics of an angioplasty guide wire.

Visibility

Angioplasty wires must be easily seen under fluoroscopy. Usually the tip of guide wire is coated with Gold or platinum to make the tip more radio opaque .

Steerabilitiy (Torque control)

Angioplasty wires must be steerable to permit passage into tortuous vessels and branches. Now all the wires are constricted in this manner.

Flexibility

Angioplasty wires should be soft at the tip to permit non traumatic passage through the coronary arteries and reduce the chance of dissection. Tip flexibility is achieved by ending the tapered core several centimeters before the tip. On the other hand some tip stiffness is needed to provide trakability and steerability to permit passage through fresh thrombus or a chronic total occlusion. The Teflon coating of the tip increases the Stiffness.

Shapability

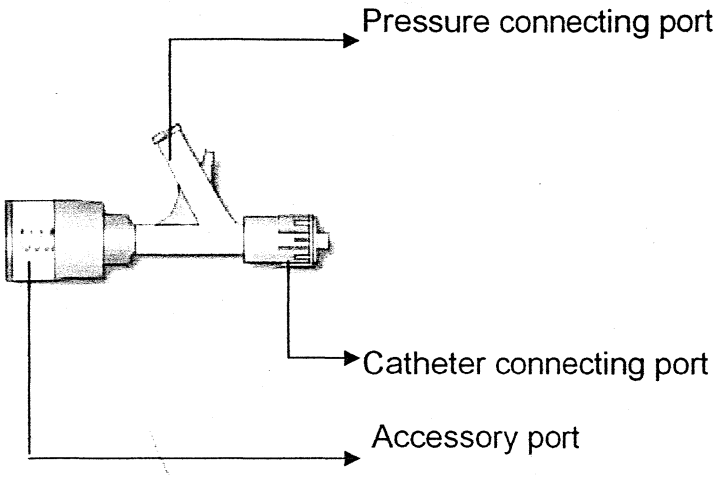
Angioplasty wires should have tips that can be shaped into various size curves to suit the individual requirements, and these curves should be retained. The introduction of tip forming ribbons at flexible wire tips had made this possible. Extending the tapered core of the guide wire to its tip (Full- body wires) is an alternate method that increases the stiffness of the tip as well as the potential for intimal trauma.

Trackability

Trackability refers to the ability of the wire to follow the tip when the tip has engaged in a side branch. Angioplasty wires should possess a degree of trackability to permit passage into side branches with acute angles. Trackability also refers to the ability to advance dilation catheters over the wire. This function would require some stiffness in the wire thus both extreme flexibility and trackability are not possible in a single wire.

Haemostatic accessories

These are playing a good role in intervention because we are more concerned with the blood loss for a patient . The usual adapters we are using are The Touhy Boarst, The Co-pilot. The picture shown here describes a Co-pilot.



Features

Engineered to Significantly Reduce Bleeding

The new COPILOT Bleedback Control Valve / RHV is designed to significantly reduce the blood loss commonly associated with interventional procedures. And, unlike conventional rotating hemostatic valves, COPILOT eliminates the need for RHV adjustments during the procedure.

The Difference Is in the Design

COPILOT's unique bleedback control seal features an innovative push-and-release mechanism. Opening the seal with a simple push facilitates the easy insertion and removal of devices. Once the seal is closed (released position), devices can still be moved freely. Compatible With RX and OTW Platform Interventional Devices. Switching to COPILOT is simple and seamless. Guidant has designed COPILOT to be completely compatible with a wide range of interventional devices on both rapid exchange and over-the-wire platforms.

Coronary Stents

A stent is a small metal coil, slotted tube, or mesh structure that is placed in a coronary artery to keep it open. It is a permanent implant that will remain in your coronary artery. The stent helps hold the artery open, improves the flow of blood, and relieves symptoms of coronary artery disease. Before placing a stent, doctors will usually perform a coronary angioplasty, a non-surgical procedure that is used to open or widen the passageway in the narrowed coronary artery. Although angioplasty is successful in most cases, it also has limitations. In particular, the blockage in the artery may recur. To help keep the artery open, your doctor may decide to implant a coronary stent. A coronary stent is a small metal coil, slotted tube, or mesh structure that is placed in an artery to keep it open. It is mounted on a balloon catheter and delivered to the site of blockage.

When the balloon is inflated, the stent expands and is pressed against the inner wall of the coronary artery. After the balloon is deflated and removed, the stent remains in place, keeping the artery open. Experience has shown that the use of a coronary stent may improve the success rate of angioplasty and reduce the rate of restenosis.

Types of stents

Stents can be classified according to their mechanism of expansion (self-expanding or balloonexpandable), their composition (stainless steel, cobalt-based alloy, tantalum, nitinol, inert coating, active coating, or biodegradable), and their design (mesh structure, coil, slotted tube, ring, multi-design, or custom design).

Stent Designs

As of the time of this writing, 2 stent designs are currently approved for coronary implantation in the United States:

- The Palmaz-Schatz stent, the most widely used of these devices, is a small, slotted, stainless-steel tube. It is about half-an-inch long, is as narrow as a piece of spaghetti, and weighs as little as a straight pin.
- The Gianturco-Roubin (Cook) stent, is a single stainless-steel coil wire shaped like a clam shell and mounted on a balloon tipped catheter.

There are over 30 other stent designs in the world, and some of them are currently being under clinical investigation in the United States. It is very likely that several of them will be approved for use in this country.

Slotted tube design

It is a type of manufacturing the stents in which a single metal tube is taken(316L stainless steel) and the part is cut to make the tube in a mesh like form by discarding the parts which is removed the rest of the tube will look like a stent. A Palmaz-Schatz stent is a slotted tube design. The problem with the slotted tube is that the thickness of the strut is high and less flexibility.

Coiled wire Stents

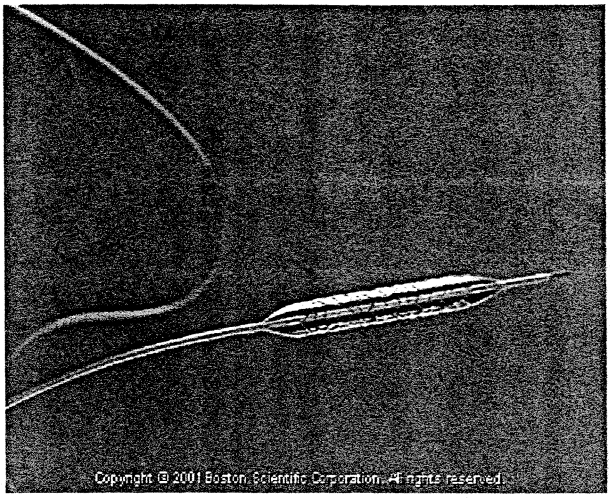
In this type of stents a single wire is coiled to make a mesh. A flat coil design with cross linking spine maintaining even spacing. The circular coils enhance flexibility, stability on balloon and vessel conformity with a single longitudinal strut.

Hybrid Stents

In Hybrid stents the strut design is made by the slotted tube technology and the struts are welded together at junctions. But the welding at junctions markedly reduces the flexibility and increases the strut thickness.

LASER Cut Stents

All the modern stents are made by this technology in which a metal tube is finely shaped by passing LASER through it so that we can get a fine shape of stent. The use of LASER technology is the accuracy in making a design and there is no need for welding. So we can reduce the strut thickness markedly and make the stent very flexible. The picture below shows a modern LASER cut stent.



Palmaz-Schatz Stents

The Palmaz-schatz stent is a balloon expandable stainless steel stent with a tubular slotted design. The standard length is a 15mm stent consisting of 7mm tubular slotted segments with an 1mm central articulation. The stent is available with a spiral central articulation and also in variable length. The stents provides good radial support and lesion coverage and is a first choice for vein grafts, ostial lesions and many type of native coronary lesions. The stent has a limited flexibility , and this draw back can be overcome with a short or disarticulated Palmaz-Schatz stents.

Gianturco-Roubin stent

The Gianturco-Roubin stent is a balloon expandable, stainless steel flexible coiled stent that is available in 12mm and 20mm in length. The flexibility allows for relatively good trackability in tortuous vessels or lesions with long dissection and can be delivered in the bailout situation.

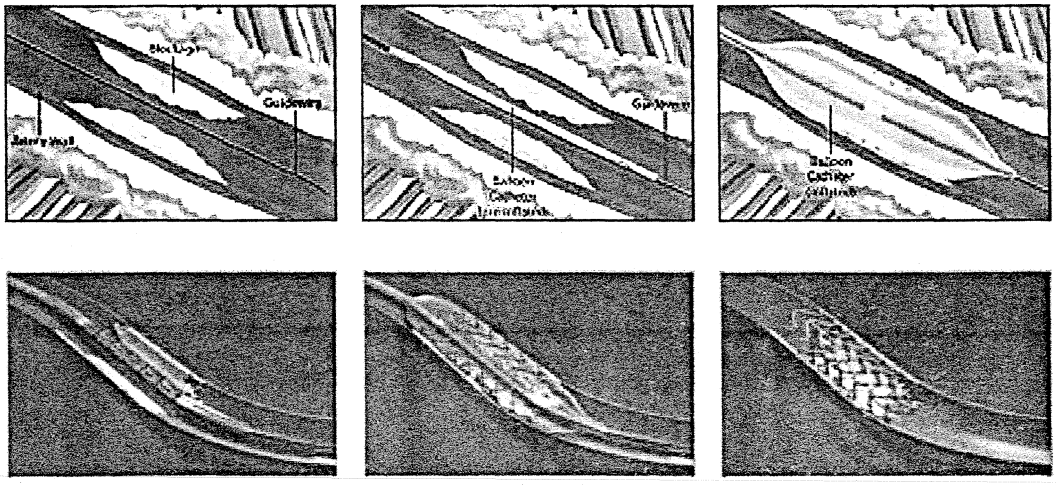
Desirable characteristics of an ideal stent

Flexibility, Trackability, Low unconstrained profile, Radioopaque, Thromboresistent, Biocompatible, Reliable expandability, High radial strength, Circumferential coverage, Low surface area, Hydrodynamic compatibility.

The flexibility and trackability of a stent is mainly decided by the strut thickness, so by reducing the strut thickness we can make the stent more flexible and trackable but as the strut thickness comes down the radial strength goes off . So we have to compromise any one of these. Now the most trackable stent has a strut thickness of 0.0036 inches i.e. 0.09mm thickness.

The procedure

The guiding catheter is advanced into the coronary and cannulate the ostium of the coronary. The next step is advancing a guide wire into the coronary in which the lesion is situated. Once the wire is in a safe deep position the PTCA balloon catheter is advanced into the coronary and exactly over the stenosis. Dilate the balloon catheter to the nominal pressure and look for any waste if it is there go more pressures and till the waste disappears. Deflate the balloon and see by an angiogram whether the segment is opened up and then take the balloon out make always an angiogram with the balloon in the coronary so that we can measure the length of the segment to be stented. As we have an idea about the diameter and the length of the coronary segment to be stented we can select a stent. The selection of stent is always an operator dependent factor but as a general rule always go for a flexible and trackable stent. If possible a Drug Eluting can be used. Adequately position the stent and do repeat angiograms to make sure that the stent is covering the lesion completely if suitable position is attained inflate the balloon and deploy the stent at nominal pressures or if the segment is still big you can go up to higher pressures and over inflate the stent. Deflate the balloon and see by angio how the vessel looks if it is satisfactory the procedure can be ceased. Always keep an eye on the hemodynamic parameters during the procedure, and the medications like Heparin. The pictures shown below respectively shows the above said procedure.



Special techniques in Angioplasty

Bifurcation lesions ("kissing Balloon angioplasty")

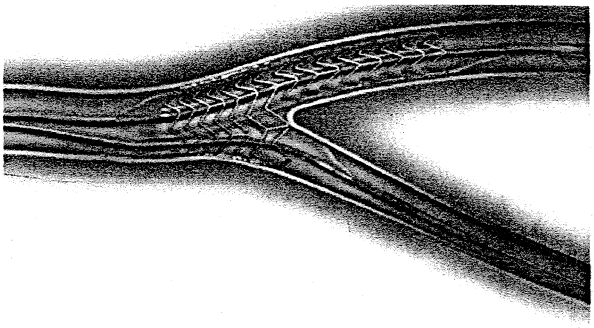
Bifurcation lesions most involve the LAD and its diagonal branches or circumflex and marginal vessels but could also involve the right coronary system. If a side branch vessel originates at the site of the stenosis to be dilated there is a risk of side branch occlusion. This risk is higher if the ostium of the side branch is stenosed. Such occlusion may be well tolerated and may show spontaneous recanalization but may also cause Myocardial infarction. They can frequently be prevented by a double guide wire technique. In order to determine the need for this technique carefully assess the angiogram with the following questions.

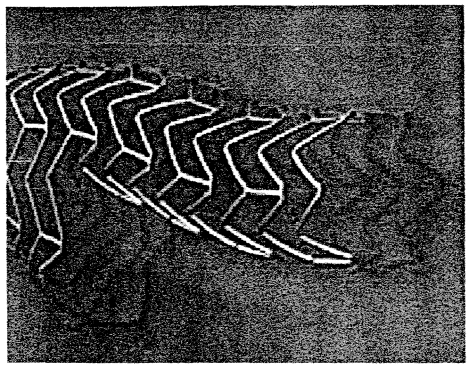
Does the side branch involved originate from the area of the stenosis itself? Is the side branch large enough to warrant an independent bye pass if the patient were to have bye pass surgery? Is there a stenosis at the origin of the branch vessel? If the answers of all these questions are positive, the patient should be considered a candidate for the double wire and sequential dilation technique. This is done with a single guiding catheter and two guide wires and two balloons. The balloon size and the wire size is dictated by the anatomy of the vessel involved. Long duration and low pressure inflations are used in these double guide wire sequential inflation methods. This technique is more appropriate for bifurcation stenosis in the LAD. The steps involved are:

1.Position the first guide wire across the stenosis that requires more manipulation. The more difficult vessel is negotiated first to minimize excessive manipulation of the second guide wire while the second guide wire is in place. After both the wires are well sited in the coronaries go to next step.

2. Determine the sequence of dilations to be performed. The largest vessel supplying the largest area of myocardium is dilated first. Proceed with the dilations now.

In the "kissing balloon angioplasty" the both the balloons are advanced into the stenosis and keep exactly in the bifurcation and inflate both the balloons simultaneously for a long time. Now the balloons look kissing each other. But this technique has a high incidence of Restenosis and major adverse cardiac events. The picture below shows the implantation of a bifurcation stent after a "kissing balloon angioplasty" and the stent is in close up.





The Primary angioplasty

Angioplasty that is carried out after the occurrence of an acute Myocardial Infarction and before giving any Thrombolytic therapy is called a primary PTCA. While the pathophysiology of acute myocardial infarction is not completely clear, there is ample evidence to support the concept that acute thrombosis is the principal mechanism, usually occurring at the site of plaque rupture. In the overwhelming majority of patients, occlusive thrombus is the inciting event, and in more than half of patients, occlusion occurs in vessels that are not highly stenotic (ie, less than 75%).

Modern therapy for acute myocardial infarction involves rapid and effective reperfusion. Numerous studies have demonstrated that muscle necrosis is a time-dependent process, with brief periods of ischemia causing cell dysfunction, stunning (loss of function but preserved viability), and eventually death. The necrotic process starts in the subendocardium, usually after about 20 minutes of coronary obstruction, and progresses to transmural and complete infarction in 4 to 6 hours. Thus, the most effective reperfusion strategies are those that are started early after the onset of the occlusion. In addition, recent observations indicate that optimal salvage of myocardium requires nearly complete reperfusion. Restoration of partial blood flow appears to be minimally helpful in reducing mortality and improving ventricular function. In other words, the goal should be to restore coronary blood flow to normal.

Prolonged occlusion leads to muscle necrosis and reduced left ventricular function, and long-term mortality in patients with coronary disease is exponentially related to the degree of left ventricular function. When the left ventricular function, measured by ejection fraction, falls to less than 30%, mortality is 30% or greater. Salvaging even small amounts of myocardium in patients with reduced left ventricular function is critically important. Finally, a number of studies covering time spans from 1 to 5 years have shown that an open artery results in better long-term survival, regardless of the ability to salvage ischemic myocardium (6). It is suggested that improvement is due not only to better left ventricular function, but to improved healing of the myocardium during the recovery phase and a significant reduction in arrhythmic complications, particularly ventricular fibrillation and ventricular tachycardia. Therefore, the primary driving principle is to reperfuse the

ischemic myocardium as soon as possible to maximize myocardial salvage and restore normal blood flow.

Role of primary PTCA

Because of the limitations of thrombolysis, interest in immediate cardiac catheterization and primary percutaneous transluminal coronary angioplasty (PTCA) to establish reperfusion is high.

Theoretically, primary PTCA has a number of distinct advantages over thrombolysis. First, it can be used in most patients with infarction, including those not eligible for thrombolytic therapy. It confirms the diagnosis, particularly in patients whose ECGs are not definitive. It allows immediate assessment of restored blood flow, which helps the operator obtain better reperfusion in a higher percentage of patients. As a result of improved reperfusion, there may be a reduction in mortality and morbidity and restoration of left ventricular function. Finally, the knowledge gained about the patient's entire coronary anatomy can be helpful in deciding which patients would benefit from later surgery. Early studies confirm these benefits and show a recanalization rate of more than 90%, restoration of normal flow in 85% to 95% of patients, a low hospital mortality rate, and a favorable 1-year outcome.

Five major randomized clinical trials on angioplasty have been reported. In each of these trials, patients presenting within 6 to 12 hours after the onset of chest pain with ST-segment elevation were randomly assigned to receive a thrombolytic agent--streptokinase in two trials and tissue plasminogen activator (TPA) in three. The primary end point of these studies included improvement of left ventricular function, coronary patency, revascularization up to 30 days after the procedure, reinfarction, and death. In all of the studies, thrombolytic therapy was initiated within 30 to 60 minutes of presentation, more rapidly than primary PTCA, even though angioplasty was performed, on average, 60 minutes after randomization. The centers that participated in these studies had personnel who were, in general, highly skilled at performing primary PTCA, and success rates ranged from a low of 80% to a high of 98%. In three of the five studies, mortality at 30 days after myocardial infarction was lower in angioplasty-treated patients than in the thrombolysis group. The largest of the five trials, the GUSTO IIb trial (14), involved 1,138 patients randomly assigned to receive "front-loaded" (accelerated) TPA or primary PTCA. At 30 days, PTCA had a small but significant advantage, with a mortality of 5.7% compared with 7% for thrombolysis. The combined composite end point of death, stroke, and reinfarction was statistically significant. However, at 6 months this difference was lost.

The recent meta-analysis of these trials supports the theory that primary PTCA performed in hospitals with highly skilled operators within 60 minutes of the onset of acute myocardial infarction reduces mortality to a greater extent than thrombolysis (15). The mechanism for this improved outcome is probably a higher reperfusion rate and better myocardial salvage, as demonstrated in a number of studies. In addition, primary PTCA offers the advantage of less risk of recurrent ischemia, since the infarcted artery has less residual stenosis than after thrombolysis alone.

Angioplasty of chronic total occlusions(CTO)

Coronary angioplasty of chronic total occlusions has been limited by a relatively low success rate and a high average restenosis rate of 53%.Since the introduction of percutaneous transluminal coronary angioplasty by Gruentzig[1] in 1977, this revascularization technique has been applied for an increasing number of indications. In coronary artery disease chronic total occlusions are still considered to be a relative contraindication for percutaneous transluminal coronary angioplasty. It has been estimated that approximately 10% of all angioplasty procedures are currently undertaken for chronic total occlusions.

The approach of a chronic total occlusion is entirely different from the usual angioplasty, We always prefer an intermediate wire, with an 1.5/6.0 mm balloon support to cross the tight lesion. The balloon must have a hydrophilic coating. Once we are sure that the wire is in the lumen do serial dilations with the small balloon and open up the vessel step by step. Since we are using a wire of intermediate thickness a chance of dissection is there. Once the vessel is slightly opened up by giving NTG we can take an angiogram and step to a higher sized balloon. If the lesion is calcific we must go for a coronary atherectomy.

Angioplasty of By pass grafts

The growing population of patients with aged saphenous vein grafts has become a clinical challenge of increasing magnitude. Efforts to treat these patients with trans-catheter interventions have been limited by distal emboli-zation and restenosis. The use of intravascular stents has been a major breakthrough in improving the short- and long-term clinical outcomes of such patients. Future techniques, which promise to reduce in-stent restenosis and improve long-term outcomes, include the use of drug-coated stents.

The clinical and technical aspects of balloon angioplasty in saphenous vein bypass grafts vary from those in native coronary arteries. Venous conduits are larger vessels with less tortuosity and calcification and no side branches, making bypass graft lesions readily accessible to guidewires and balloon catheters. However, because of the friable nature of graft atheroma, the outcome of balloon dilation of a vein graft lesion is more unpredictable. It carries the increased risk of distal embolization of atherothrombotic debris and periprocedural myocardial infarction. Risk factors for distal embolization include grafts older than 3 to 5 years, long lesions, and the presence of thrombus. Furthermore, the long-term clinical and angiographic results of balloon angioplasty are limited by the high incidence of restenosis at the treated lesion site. Restenosis exceeds 50% for lesions located at the ostium or within the body of the graft. Plokker et al. reported the long-term outcome of 454 patients treated with balloon angioplasty for venous bypass graft lesions. After a 5-year period, 26% of the patients died and 48% experienced a major nonfatal cardiovascular event such as myocardial infarction, repeat bypass surgery, or repeat angioplasty. After 5 years, only 26% of the patients survived event free. In addition to periprocedural complications and restenosis, older

patient age, comorbid conditions, and progression of atherosclerosis in sites remote from the initial lesion contribute to unfavorable clinical outcomes after vein graft interventions.

The landmark randomized trials, STRESS and BENESTENT, demonstrated the superiority of stent placement over balloon angioplasty in native coronary arteries. However, these trials excluded patients with vein graft lesions. Many observational studies have reported successful treatment of vein graft disease by stent placement with relatively low restenosis rates. Self-expanding stents, such as the less-shortening Wallstent, possess several features desirable for vein graft intervention: relatively large surface area coverage, availability of long devices for diffuse disease, and suitability for large vessels 4 mm in diameter. Furthermore, the self-expanding mechanism minimizes the pressure needed for balloon inflation after deployment, which may reduce embolic complications acutely and restenosis long-term. Whether these theoretical advantages translate into true clinical benefit has yet to be demonstrated.

Distal protection devices in saphenous vein graft intervention

It is a known case that the Saphenous vein grafts are more prone to thrombus and their intervention surely end in distal embolization. So we always start the procedure with a distal protection device in the distal vessel. A number of techniques have been used to aspirate thrombus and other macroparticulate debris from SVGs during percutaneous intervention. Early methods included using two guiding catheters and occluding the distal vessel with a balloon catheter and performing an aspiration thrombectomy using an aspiration device while a balloon was inflated within the SVG. These methods were tedious to perform and were reserved for patients at highest risk for complications caused by degenerated SVG disease.

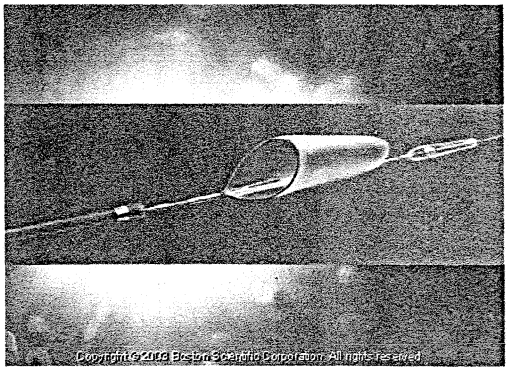
The PercuSurge Guardwire (Medtronic AVE, Santa Rosa, CA) consists of a 210-cm angioplasty wire constructed using a 0.014-inch nitinol hypotube with a distal 35-mm radiopaque, shapeable, steerable tip. A 5.5-mm elastomeric balloon inflatable between 3.5 and 5.0 mm is positioned at the distal portion of the wire. The Guardwire tip is advanced across the stenosis and the balloon inflated in a segment that is relatively free of disease to block the blood flow into the distal portion of the vessel. Balloon angioplasty and stent placement are then performed over the Guardwire. After stent deployment and after dilation, an export aspiration catheter is used to remove the particulate debris. A 20-mL locking syringe is attached to the aspiration catheter to generate a vacuum and to serve as the collection chamber. After aspiration of the microparticulate debris, the Guardwire balloon is deflated and distal flow to the vessel restored

Filters

As an alternative to occlusion devices, a number of porous membrane filters mounted on the tip of coronary guidewires have been developed to capture and retrieve embolized materials during SVG intervention. In general, the porous membrane filters are supported by an array of superelastic nitinol, which is restrained by an outer delivery sheath until the tip is delivered across the lesion. After retraction of the delivery sheath,

the self-expanding filters support the filter against the wall of the SVG. Although liberated particles larger than the filter pore size are trapped within the filter, histologic examination of the retrieved material suggests that particles smaller than the pore size may also be removed, likely due to the creation of fibrin strands within the filter. Once the SVG intervention has been completed, a recovery sheath is advanced to close the filter structure, trapping any collected debris and reducing the filter diameter sufficiently to allow recovery back into the guiding catheter. These filters are generally easier to use because they maintain coronary blood flow; however, they may not routinely capture particles that are smaller than 80 microns.

Increased experience with filter devices have presented additional challenges. Although the embolic protection filtersgenerally use a 0.014' guidewire for over-the-wire delivery of balloons and stents, the early generation, higher-profile delivery systems may pose challenges in some anatomic subsets, such as highly angulated, superior take-offs of left coronary SVGs or tortuous lesions. Use of a 0.014' or 0.018' extra-support buddy wire may also straighten the vessel sufficiently to allow passage of the device into the SVG. Predilation with a small (2.0 mm) balloon catheter may allow passage of the high profile delivery sheath. Many of the new devices also require a 15- to 24-mm segment of nondiseased SVG distal to the lesion to allow positioning of the embolic protection device and advancement of the balloon and stent. Another challenge is in the event that the filters may fill with particulate debris and reduce anterograde flow. Simply removing the filter wire without aspiration may release a column of static particulate debris into the distal microcirculation-in this circumstance, aspiration of the column of material with an aspiration catheter before filter removal may be useful. The figure shows a filter device.



CORONARY ATHERECTOMY

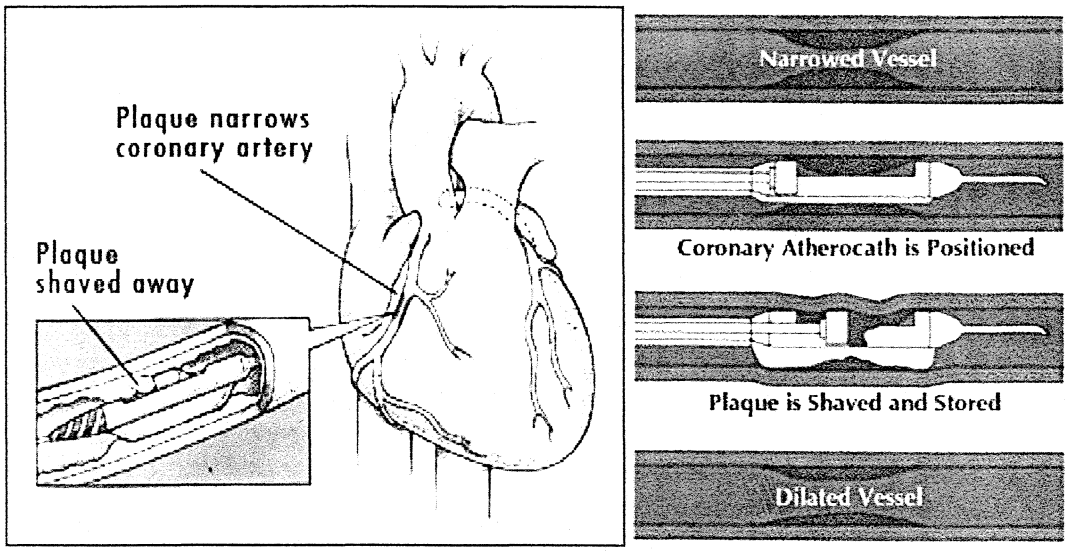
Introduction

The motivation for introducing mechanical atherectomy ,like other new devices was an attempt to overcome some of the limitations of conventional balloon angioplasty. These are the choice of interest when we come across a calcified lesion. These include the family of Directional coronary atherectomy, Transluminal extraction catheter, and Rotational atherectomy.

Directional coronary atherectomy

Directional coronary atherectomy is a catheter intervention that allows us to shave out specific types of narrowing in the coronary artery. This procedure may be preferable to balloon angioplasty. Some of the factors that determine angioplasty versus atherectomy are the location of the blockage, the shape of the blockage, the size of the artery and whether clots are present in the artery. All of these considerations are up to the discretion of the interventional cardiologist.

When it is determined that directional coronary atherectomy is appropriate, the pre-existing catheter in the femoral artery needs to be exchanged for a larger catheter. This is due to the greater bulkiness of the directional coronary atherectomy device. A guiding catheter which allows us to place the guide wire and the directional coronary atherectomy device across the lesion is placed in the aorta at the opening of the coronary artery. Once this guiding catheter is in place, an appropriately sized atherectomy device is chosen. Often, if the narrowing is extremely tight, it may need to be predilated with a small angioplasty balloon in order to allow easier passage of the atherectomy device across the narrowing. After this, the atherectomy device is then placed across the narrowing.



The atherectomy device is a large balloon catheter. However, the balloon is only wrapped around a portion of the circumference of the device. It allows for placement of a window in the metal housing on the side of the catheter and this window is then pushed against the plaque by the balloon and a cutting piston is then advanced through the device in order to shave off the plaque tissue that has been placed in the window. This plaque tissue is then pushed into the nose cone of the device and it will be removed later during the procedure. Typically, during this procedure, your doctor will take four cuts at a time before removing the device from the coronary artery and examining the result. On occasion, your doctor will have to again place the device across the narrowing and take multiple cuts. Your doctor may take anywhere from four to more than twenty cuts in a given blockage in order to obtain a good angiographic result. The amount of tissue removed at the end of the procedure can be variable and is dependent upon the amount

of plaque present at the site of the narrowing. At the end of the procedure, occasionally a balloon may need to be placed in order to smooth out the angiographic result, although this occurs less frequently. You are then sent back to your room where a period of bedrest is required. The sheath will be removed from your groin after the specific amount of time the cardiologist orders.The pictures shown below are the steps of a Directional coronary atherectomy.

Transluminal Extraction coronary atherectomy.

The transluminal extraction catheters consist of several components. The TEC catheter itself has a conical cutting head with two stainless steel blades attached to the distal end of a hollow torque tube, a hand held motor drive unit that attaches to the proximal end of the cutting catheter , a battery pack that attaches to and serves as a power source for the motor drive unit, and a vacuum bottle that attaches to the motor drive unit for collection of aspirated atheroma , thrombus and other debris. A trigger on the handle of the motor drive unit activates cutting blade rotation at 750 rpm, and a sliding lever on top of the motor drive unit permits advancement or retraction of the cutter over the guide wire. The Tec guide wire is specially designed with a 300 cm long 0.014 inch stainless steel shaft, a 2 cm floppy tip, and 0.021 inch ball at the tip of the guide wire to prevent wire entrapment in the cutter. A specially designed rotating hemostatic valve with a side arm adaptor connects the guiding catheter to the manifold for contrast material injections and permits infusion of warm, pressurized, heparinized Ringer lactate solution during atherectomy. This infusion results in a slurry of blood and tissue that is then continuously aspirated as the cutter is activated. Special 10 French tungsten braided soft tip guiding catheters are recommended for all TEC procedures.

The Procedure

Since the ideal device size has never been established, device selection remains empirical. In general the largest clutter used should be 1.0 to 1.5 mm smaller than the normal vessel diameter to achieve a final cutter to artery ratio of 0.6 to 0.7. TEC cutters are available in different diameters ranging from 5.5 French ,6.0,6.5,7.0,7.5 Frenches. After engaging the coronary ostium with the guiding catheter, the target lesion should be crossed with the TEC wire using a bare wire technique. The floppy tip of the guide wire should be positioned in the distal vessel so the stiff shaft of the wire is across the lesion. Once the guide wire is in proper position, the TEC cutter should be advanced up to the lesion, and the infusion of warmed lactated Ringer solution should begin. The operator then depresses the trigger to activate the cutter rotations and slowly advances the lever to traverse the entire lesion. Two to five passes should be made slowly through the lesion until there is no further resistance to cutter advancement. After retracting the cutter into the guiding catheter, repeat angiography should be performed to determine for a larger device or adjunctive angioplasty.

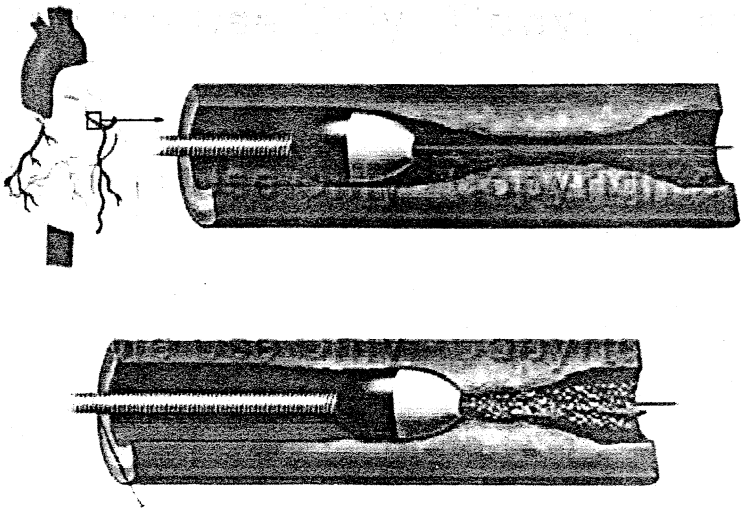
Rotational coronary atherectomy-ROTABLATOR

The high speed mechanical rotational atherectomy device or Rotablator consist of an olive shaped stainless steel or brass burr whose surface is embedded with diamond chips measuring 30 to 120 micro meter in diameter. The burr is attached to a hollow flexible drive shaft that permits passage of a steerable movable 0.009inch guide wire with a 0.014 inch platinum coil at its tip. The drive shaft is encased within a Teflon sheath through which warm heparinised Ringer lactacte solution is pumped into the sheath to lubricate and cool the drive shaft and burr. A compressed air turbine rotates the drive shaft at 150,000 to 200,000 rpm. Burrs for coronary use are available in diameters of 1.25,1.5,2.0,2.15,2.25 and 2.5 mm.

The procedure

Conventional coronary angioplasty guiding catheters may be used for Rotablator atherectomy as long as their lumen is at least 0.020inch larger than the largest burr to be used. If difficulty is anticipated in crossing the lesion then the lesion can be crossed with a conventional angioplasty wire and then can be exchanged for a Rotablator guide wire using a suitable transport catheter.

Once the guide wire is across the lesion, the burr should be advanced to within a few centimeters of the rotating hemostatic valve, with the lines of compressed air and the tachometer attached to the drive console. Now the burr can be advanced into and through the guiding catheter. Some resistance is normally encountered as the burr is passed around the arch of the aorta or around the primary curve of the guiding catheter. This must be overcome by firm traction on the guide wire to pull the device around these curves and into the proximal vessel . It is important to note that the guiding catheter is well seated in the coronary ostium while advancing the burr into the vessel to prevent the kinking of the guide wire. Once the burr has been advanced to 1 to 2 cm proximal to the target lesion, the lever should be unlocked and pulled all the way back to its proximal limits so as to take up any slack in the drive shaft that might otherwise cause the burr to lurch forward into the lesion upon activation. Under fluoroscopy the burr is then activated by stepping on the pedal and then platform speed is adjusted before engaging the lesion(160,000 to 1800,000 rpm) . The lever is then advanced to bring the spinning burr slowly into the lesion. The lever is then advanced to bring the spinning burr slowly into the lesion. The following picture shows a Rotablator in the coronary.



PERCUTANEOUS TRANSVENOUS MITRAL COMMISSUROTOMY (PTMC)

Introduction

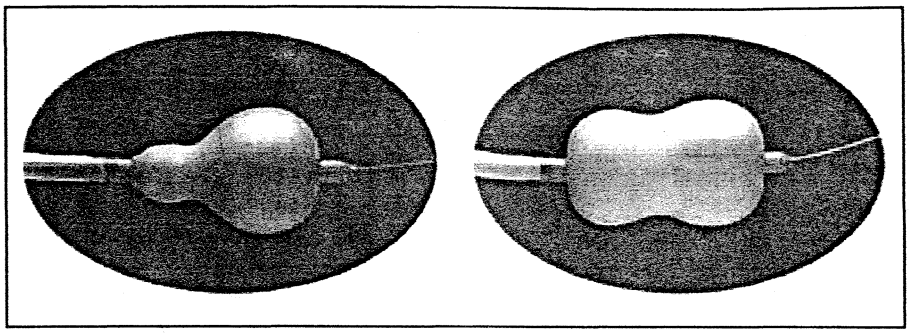
Inoue described the single balloon technique of mitral commissurotomyin1984 ,having first performed the procedure in 1982. The Inoue balloon and the double balloon techniques are the most commonly used techniques for the Mitral commissurotomy. Today the most frequently used and our lab is using the Inoue technique. On an average there is 80% increase in the valve area. Balloon inflation results in splitting of the fused commissures with reductions in the trans mitral pressure gradient, the mean left atrial pressure, and the pulmonary artery pressure. The cardiac out put and the mitral valve area increase. The most important complication of the Mitral Valvotomy is Mitral regurgitation.

The technique

The Inoue balloon

The Inoue device differs substantially from conventional balloons. It is constructed of two layers of latex with a nylon mesh in between. The latex is compliant, whereas the nylon mesh limits the maximum inflated diameter of the balloon and gives it its unique shape and three stage inflation characteristics. The front half of the balloon inflates, giving an appearance of a balloon floatation catheter. The proximal half of the balloon inflates next creating a dumbbell appearance. When it is positioned across the Mitral valve, this facilitates positioning of the balloon device in the valve orifice. Last the central portion of the balloon inflates, resulting in commissurotomy. The distensibility of the latex material allows each balloon to be inflated over a 4mm range of diameter sizes . A single balloon can thus be used to effect sequential dilation of the valve, by inflating it to a serially larger

diameters without removing it from the patient. The picture below shows an Inoue balloon and its three stage dilation.



Other Hardwares used for PTMC

As an interventional procedure the PTMC hardwares and its handling are of importance for a person in the field of intervention. The main hardwares used in PTMC are the Mullins sheath dilator, the Brockenbrough needle, the LA wire, the 14 Fr.dilator, the Inoue balloon, the straightener, the LV stylet and the Vernier caliper.

The Mullins dilator is a Teflon made sheath and dilator pack and for a Inoue technique we use the dilator alone. The sheath is an 8 French with a length of 59 cm and the dilator has a length of 67 cm and an internal diameter of 0.034 inches so we can use an 0.032 inch wire through the Mullins dilator.

The Brockenbrough needle is a 70 cm length needle whose tip tapers into an 0.032 inch diameter and is used for the septal puncture before the Valvotomy.

The LA wire is a core flex wire, and has a diameter of 0.025 inches and has a length of cm. It is usually used after the puncturing if the septum and for the entry of the Left atrium and we put a 14 French dilator through the LA wire.

The 14 french dilator is used for dilating the septum before the balloon entry to make an easy tract for the balloon which is at least an 11 French diameter.

The Inoue balloon is packed with a straightener, which is used to make the tip of the balloon straight for an easy percutaneous entry in to the vein. The proper technique must be used for the entry of the straightener into the balloon otherwise it can damage the balloon.

The next is an LV stylet which is used for the LV entry. As the balloon crosses the septum it may look to the lateral wall of LA so that we can put the stylet into the balloon and make the balloon curved, the tip of the LV stylet has a 70 degree big "J" curve so that it can turn the tip of the balloon into the Stenotic Mitral valve and helps in directing the balloon in accordance with operators wish.

A short connector is attached to the inflating lumen of the Inoue balloon for the attachment of a luer lock syringe with contrast which is under 1:4 dilution.

Patient evaluation

Evaluation of two dimensional echocardiography and Transeasophageal echocardiography are essential before mitral Valvotomy. Transeasophageal echocardiography before PTMC is useful for the detection of atrial thrombi. Left atrial clot is a relative contra indication for PTMC. In usual cases when we note an atrial thrombi, it is good to keep the patient under anticoagulation therapy for 3 to 12 months and then reassess for PTMC.

Selection of balloon size

Maximum expected inflated balloon diameter may be selected based on the patients height. This value provides a guideline for balloon selection, and with stepwise technique a first inflation is always at a diameter smaller than the maximum possible for selected balloon (usually 4mm less than the maximum). Usually a simple formula is used for selecting the maximum Inoue balloon size:

$$\text{Maximum balloon diameter} = \frac{\text{Height in cm}}{10} + 10.$$

For patients with pliable valves, the first inflation can be made with a balloon 2 or 3 mm smaller than the reference size, and then increased in increments of 1mm until either a maximal diminution of gradient has occurred or mitral regurgitation has begun to worsen significantly. There are a number of special considerations in balloon size selection. Smaller balloons than initially estimated may be useful in patients with advanced age, sub valvular disease.

Balloon preparation

The balloon catheter lumen is flushed with saline. Diluted contrast (4 saline: 1 contrast) can be injected into the balloon to purge air from the inflate deflate channel to the balloon and the stop clock is closed on the vent lumen.

The precaliberated balloon inflation syringe is filled to the calibration corresponding to the smallest inflated diameter (for a normal 28mm balloon this should be 24mm in diameter). After connecting the inflation syringe to the inflation port and checking that all connections are secure, the balloon is slowly inflated over a period of 10 to 20 second so that the nylon mesh may be slowly stretched without risking rupture.

The balloon is allowed to deflate passively in a bath of flush solution. Small bubbles will escape from within the mesh layer of the balloon .The balloon is then inflated rapidly and the inflated diameter is measured using calipers to verify that the

precalibrated inflation syringe achieves the correct minimum inflated diameter. If the balloon does not inflate to the desired diameter small amounts of contrast are added or subtracted to achieve proper calibration.

The next step in balloon preparation is to stretch the balloon catheter along its long axis causing it to become more slender . A metal tube is inserted into the central lumen of the balloon over the guide wire and advanced until it locks into the metal hub at the proximal end of the balloon. This tube is called the slenderizing tube.

The procedure

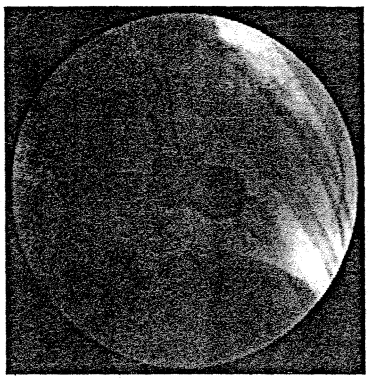
As a procedure of importance in the Haemodynamic of the patient the PTMC ia always preceded and followed by a cardiac catheterization. The Femoral artery and the femoral veins are punctured and a GL is advanced for a right heart catheterization the RA,RV,PA, pressures and the Aortic and Pulmonary saturations are taken for the calculation of the cardiac output.

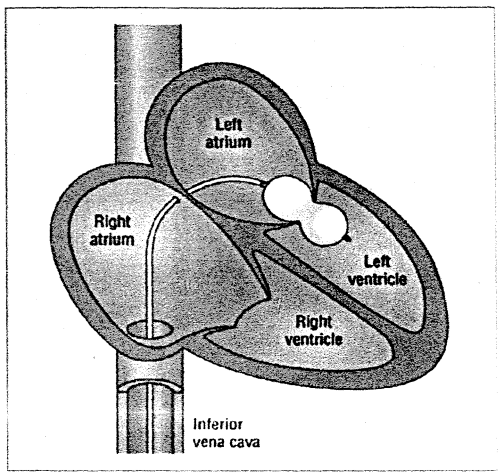
The next step in the procedure is the Septal puncture. The GL is advanced into the SVC and is then exchanged for the Mullins dilator now the Mullins dilator is brought back in such a way that the Mullins dilator will face the Inter atrial septum. Now the Brockenbrough needle is advanced into the Mullins and the position is viewed in the RAO view , and then can go to the lateral view and the septum is visualized by a small contrast injection into the fossa of the septum as it stains the septum the puncture needle can be advanced into the septum which results in the septal puncture and the needle can be withdrawn now and see for the pressures and make sure that the pressures are showing the LA pressures.

Now record the Left atrial pressures with mean and put 14 French dilator to dilate the septum to make an easy entry for the 11 French balloon. Advance a pigtail catheter through the arterial sheath and is advanced into LV to record the Left ventricular end diastolic pressures which equals the LA mean pressure and for the calculation of the gradient which is helpful in calculating the valve area.

The prepared balloon is now advanced over the LA wire and when the balloon reaches the Left atrium take the LA wire back and put the stylet inside. Now go to the RAO view and try to cross the valve. One must have a three dimensional idea about the anatomy to know the valve position, otherwise just inflate the distal bulb of the balloon then we can see the valvular motion deflate it and try there to enter the valve once the balloon entered the valve inflate the distal bulb then give a tug in the balloon so that the waste of the balloon correctly sit on the commissures now go up with the maximum inflation.

The picture below show the dilation of the Mitral valve with an Inoue balloon.





As the dilation is over the balloon is taken back to Left atrium and the left atrial pressures are checked with mean. A decrease in LA pressures indicate that the balloon inflation has resulted in a commissurotomy . The valve can be reviewed using Echocardiography to see whether the commissures has opened and how much is the Mitral regurgitation. If the results are showing good we can stop with the dilations but still the LA pressures are high we can go for further dilations and stop.

Complications of PTMC

The two most serious complications of PTMC are cardiac tamponade and acute Mitral regurgitation. The recognition of pericardial tamponade requires a high degree of suspician on an ongoing basis. Continuous attention to the pulsatile fluoroscopic cardiac borders is important. It is not reasonable to perform PTMC without the ability to perform pericardiocentesis as well.

Acute Mitral regurgitation is easily recognized by the left atrial pressure magnitude and waveform. It occurs in some cases with a single balloon inflation, or after even a half mm diameter increment in balloon inflation size.

Calculation of Stenotic valve Area

The Gorlin Formula

The first hydraulic formula that the Gorlins used was Torricelli's law, which describes flow across a round orifice:

$$F=AVC \qquad (1)$$

F = flow rate, A = orifice area, V = velocity of flow, and C = coefficient of orifice contraction. The constant C compensates for physical phenomenon that except for a

perfect orifice, the area of stream flowing through an orifice will be less than the true area of the orifice.

Rearranging the terms of equation (1),

$$A = \frac{F}{VC} \quad \text{(2)}$$

The second hydraulic principle used in derivation of Gorlin's formula relates pressure gradient and velocity of flow.

$$V\rho V = C\rho C\rho 2\rho g\rho h \text{ or} \quad \text{(3)}$$

$$V = C\rho sqrt(2\rho g\rho h)$$

Where V is the velocity of flow, C is the coefficient of velocity, g is the acceleration due to gravity, and h is the pressure gradient in cmH2O.

Combining equations (2) and (3), we get,

$$A = \frac{F}{C\rho 44.3\rho sqrt(h)} \quad \text{(4)}$$

Where C is an empirical constant accounting for co efficient of orifice contraction and coefficient of velocity. The expression of h is in mmHg.

It is obvious that the antegrade flow across the Mitral and tricuspid valve occurs in diastole where as the flow across aortic and pulmonic valve occurs in systole. So we can make the above equation as the valve orifice area A in cm2 is,

$$A = \frac{CO/DFP \text{ or } SEP\ (HR)}{44.3C\rho sqrt(\blacktriangle p)} \quad \text{(5)}$$

where CO is the cardiac output, DFP is the diastolic filling period, SEP is the systolic ejection period ,Hr is the heart rate, C is the empirical constant and p is the pressure gradient. The empirical constant for the tricuspid, aortic and pulmonic valves are assumed as 1.0. The empirical constant for the Mitral valve is taken as 0.7. But when the DFP is measured from the left ventricular versus PAW or left atrial tracings then the empirical constant should be 0.85.

Mitral valve Area

By rearranging the terms of equation (5) one sees that for mitral valve area ,

$$\blacktriangle P = \left[\frac{CO/(HR)(DFP)}{\sqrt{(MVA)(44.3)(0.85)}} \right]^2$$

Where ▲P is the trans Mitral pressure gradient, and MVA is the Mitral valve area.

BALLOON AORTIC VALVULOPLASTY(BAV)

Introduction

Balloon aortic Valvuloplasty showed great promise as an alternative to surgical aortic valve replacement when the procedure was initially described. Balloon dilation of the aortic valve results in an immediate increase in the aortic valve area, with the expected fall in transvalve pressure gradient and a rise in cardiac out put. Most patients show immediate clinical improvement.

Indications

There are currently five clinical situations in which balloon aortic Valvuloplasty is useful, they are,

Cardiogenic shock/ pre-op for aortic valve replacement (AVR), Severe LV failure/bridge to AVR, Prior to major non cardiac surgery, Diagnostic test for low gradient, low out put AS with poor LV function in consideration of surgery, First, patients who present with aortic stenosis and cardiogenic shock may be stabilized for short term.

Hardwares used

The balloon aortic Valvuloplasty uses, the straight tip guide wire for the crossing of the stenotic aortic valve the best option is a TERUMO Radifocus straight tip wire. The catheter can be an RCA or an AL-1 . Try to cross the aortic valve in plane LAO angulation, care must be taken not to enter the coronaries as the Terumo wire can easily cause a dissection. Next is an Amplatz extra support wire with exchange length, a temporary pacemaker lead, The balloon catheter.

Terumo wire is used because of its tapering at its tip, and the hydrophilic coating. It is the better option to cross a stenotic site. The Right coronary catheter shape helps us to point towards the cusps of the aortic valve for an easy entry and is trackable over the terumo wire. Amplatz Left 1 is another good option to cross the valve. The Amplatz extra support wire is used to support the balloon catheter and to exchange for the terumo wire. The floppy tip of the Amplatz wire should be at least 6 cm to loop in the Left ventricle

without making any significant ectopics. The temporary pacing lead is a bipolar lead for the pacing during inflation.

The procedure

The first part of the balloon aortic Valvuloplasty is doing an aortic root angiogram for the measurement of the aortic annulus. A pigtail catheter is advanced into the aortic root and a root angiogram is done in plane LAO angulation. Then the annulus can be measured, in accordance which we can select the balloon size. Usually we go for a size less than the aortic annulus i.e. if the annulus is 20mm we go for an 18mm balloon.

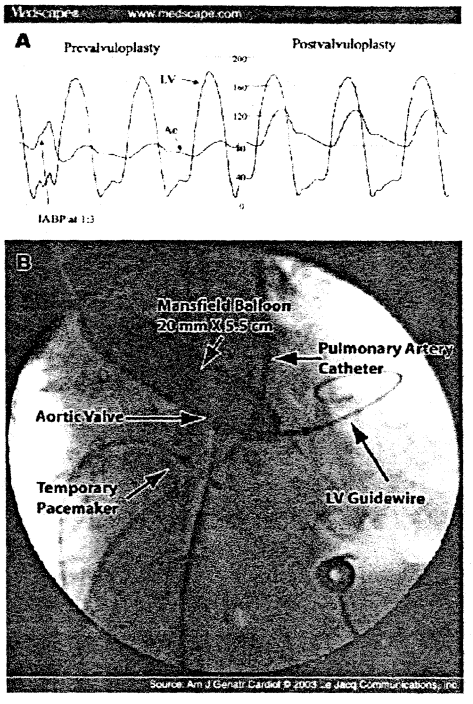
Once the root angiogram is over we can try to cross the valve we usually first try with a straight Teflon with an RCA or AL-1, if it is not crossing the valve we go for a straight terumo wire and it will surely cross the valve. As the wire crosses track the catheter over it and the trans valvular gradient can be taken with a tracker catheter. Now exchange the catheter for an Amplatz Extra support guide wire. Frequent ventricular ectopics will come at this time the defibrillator should be ready within limits for an emergency and quick cardioversion. Because for a patient with aortic stenosis gets a VT there will not be any cardiac output so a quick cardioversion is needed.

The temporary pacemaker lead is now advanced into the Right ventricular apex for pacing during the inflation of the balloon. Pacing is carried out during BAV because when the balloon is in the aortic valve the LV ejection will through the balloon away from the valve. So we pace the heart at a rate of 250 beats per minute so that at that time there will not be any output so the balloon will be in situ and can result in a good dilatation. The balloon must be deflated after 15 seconds because pacing at a higher rate for more than 15 seconds may results in serious ventricular arrhythmias like ventricular fibrillation.

The Valvuloplasty balloon is now prepared with 1:4 diluted contrast and is nicely profiled for an easier entry into the aortic valve. The Tyshack balloon needs a sheath while the Mansfield balloons are sheath less. A sheath is exchanged for the balloon. A pigtail is always kept in the ascending aorta for the aortic pressure measurement during the inflation of the balloon. Now the balloon is taken it must be very clear that there not a single small air bubble in the balloon. The balloon is positioned midway across the aortic valve. Since the LV function is poor the balloon usually sits in position.

Make sure that every thing is ready specially the temporary pacemaker , start pacing and at the same time start inflating the balloon a nice waste can be seen if the balloon is at the correct position, watch the pressure wave form the pressure will go to zero then quickly deflate and stop pacing. The pressure will come up. Now take the trans valvular gradient if it is reduced marginally we can stop there or you can go for further dilatations.

The figure below shows the BAV procedure



Complications of BAV

The major complications of aortic Valvuloplasty are ventricular perforation from the catheter or guide wires used in the left ventricle , and femoral arterial complications due to large sheath necessary for the balloon. Since the balloon catheter abrades the ventricular septum during balloon inflations, bundle branch block may occur that requires pacing in some cases, and rarely permanent pacemaker implantation. If the balloons are oversized severe aortic regurgitation will be a result. Bicuspid aortic valve may resist dilatation more than degenerative tricuspid valves. Serious ventricular arrhythmias like Ventricular tachycardia or ventricular fibrillation may result.

Each therapeutic dilation causes a transient but substantial stress on the left ventricle. Out flow obstruction is acutely worsened, chamber dilation occurs, ventricular pulse generation decreases, and coronary perfusion pressure drops. Several technical factors probably cause this disastrous syndrome.

BALLOON PULMONARY VALVULOPLASTY

Introduction

The potential use of balloon dilation to treat congenital pulmonary stenosis evolved naturally from the early surgical experience with mechanical dilation of stenotic pulmonary valves. The balloon pulmonary Valvuloplasty is usually done in infants who had congenital Pulmonary stenosis.

Indications

Pulmonary stenosis congenital or acquired,Tetrology of Fallot's.

Hardwares used.

A Goodale Lubin catheter, an NIH, a Terumo wire, a balloon wedge pressure catheter, an Amplatz extra support exchange wire, a tracker catheter, the Valvuloplasty balloon.

The procedure

The right heart catheterization except PA pressures are taken with the GL, an NIH is advanced into the RVOT and an RVOT angiogram is done in the lateral view to profile the pulmonary annulus and the pulmonic valve. Once the angiogram is over the pulmonary annulus is measured and the balloon size can be determined. For BPV we can choose a high size balloon than the pulmonary annulus because Pulmonary regurgitation is not problematic as its counterparts. If we get an annulus of 18 then we can go for a balloon of 20mm.

Now try to cross the valve with a terumo wire and a balloon wedge pressure catheter, it is not a very difficult issue to cross the valve. Once the valve is crossed the trans valvular gradient is measured and recorded. The wire is then exchanged for an Amplatz extra support guide wire. Now get the balloon ready with a 4:1 contrast dilution with out any air bubble in the system. Track the balloon over the wire and inflate the balloon exactly at the valve in the Lateral angulation. Once the first inflation is over use the tracker catheter to measure the transvalvular gradient if it seems satisfactory we can conclude otherwise we can go for further dilations.

PEDIATRIC INTERVENTIONS

PDA COILING AND DEVICE CLOSURE

Introduction

Patent Ductus arteriosus (PDA) is a common congenital heart defect, which is usually identified in childhood, but may nit be recognized until late in life. Closure of PDA is indicated to prevent bacterial endarteritis, congestive heart failure, and or pulmonary vascular disease. Trans catheter occlusion became common place in the early 1990's after demonstration that Gianturo coils were effective for closure of small PDA. Recent advances in device technology allow trans catheter closure of virtually all PDA's regardless of size and configuration.

Back ground

Percutaneous trans catheter occlusion of PDA began in 1967 in Berlin when Porstmann used an Ivalon plug. The plug was hand made after an aortogram defined the shape and size of the Ductus. A venoarterial loop was established , and the plug pushed through the femoral artery in a retrograde fashion into the ductus arteriosus. However due to the large sheath sizes required the technique was limited to older children and adults. Widespread use of this device was not achieved. In the following paragraphs we can describe different devices and techniques used for the closure of the Ductus Arteriosus.

Ivalon Plug

Porstmann helped to develop the Ivalon(Polyvinyl alcohol polymer) plug and was the first to use it clinically. The Ivalon plug was a cone shaped plastic foam device. The shape and size of the plug were carved from the large plug in the catheterization lab after reviewing the lateral descending aortogram and measuring the PDA. The required a trans cardiac guide wire loop from the femoral artery and aorta, through the PDA and right heart then out the femoral vein. The plug was advanced over this guide wire loop and pushed into the PDA. Despite the rather un refined features of the device the result were very good. The device has a number of limitations including requiring a 16 to 22 French sheath in the femoral artery, the femoral artery diameter has to be larger than the plug diameter, the PDA has to be conical shaped and if the plug embolises it would be to the aorta.

Rashkind Umbrella

Rashkind initially deviced a single umbrella device. The device consisting of a thin metal frame work that was covered by a thin circle of polyurethane foam. There were several hooks on the umbrella to help attach the device to the PDA. Occasionally the hooks were caught on vessels other than the PDA and misadventure ensued.

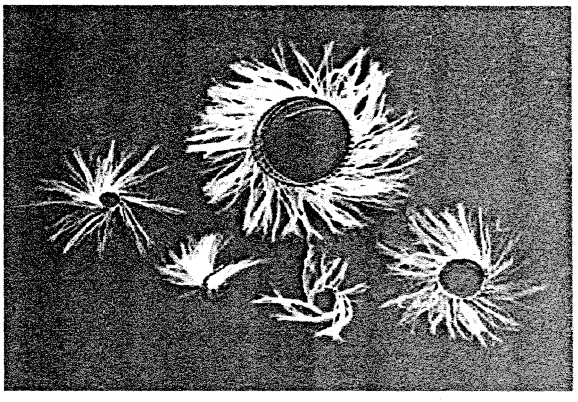
Rashkind redesigned the device as two umbrellas facing each other without hooks, there were two sizes. A three arm, 12mm device for PDA's with minimum diameter less than 4mm, and a four arm,17mm device for PDA's with a minimum diameter 4 to 7 mm. The 12 and 17 mm device were delivered through 8 and 11 French sheath respectively. Based on today's standard the result of the double umbrella device was un unacceptable because of residual shunting.

Gianturco embolization coils and detachable coils

The first to report result of using the Gianturco embolization coil for transcatheter PDA closure. The Gianturco embolization coil had been used for occlusion of various vessels including arterio venous fistulas, aorto pulmonary collateral vessels, hemorrhaging vessels, and vessels supplying tumors.

Device description

The picture below shows a Gianturco embolization coil.



The Gianturco embolization coil is a stainless steel wire, 0.004 inch to 0.008 inch diameter that is wound into a helical configuration called the primary windings. These primary windings are similar to a guide wire that was no inner core. The windings come from various length ranging from 2 to 20 cm, the windings are shaped into a coil that has several loops, the coil diameter ranges from 2 to 20 mm. In general, the larger diameter the coil the longer coil length is needed to provide enough coil loops for occlusion.

The diameter of the wire(primary windings) ranges from 0.014 inches to 0.043 inches. White Dacron strands are added to the wire to promote clot formation. One end of the coil has a smooth rounded bead the same diameter as the coil, this is the end of the coil that is inserted first into the catheter, the other end of the coil has a slightly larger coil diameter, the larger size ensure that the coil is pushed through the catheter, preventing the coil and pusher wire from overlapping within the catheter.

The most common coil diameter used for PDA closure is 0.038 inches. Small PDA may be closed using a 0.035 inch coil diameter. Larger PDA's may require a stiffer coil, 0.052 inch in diameter. Note that the 0.038 inch coil requires a catheter lumen of 0.038 inch, a larger catheter lumen may be too large and the coil may loop inside the catheter. A smaller catheter lumen on the other hand will not allow the coil to pass through it.

It is important to understand the sizing description of each coil. Every coil has three measurements. The first measurement is the coil wire size, the second is the length of the coil and the third is the diameter of the coil as it loops. For example an 0.038-8-6 coil has a wire size of 0.038 inch and has a coil length of 8 cm and has a diameter of 6 mm. The number of loops of a coil can be calculated using the equation ,

$$\text{No of coils} = \frac{L}{\pi * D}$$

Where L is the length of the coil and D is the diameter when it coils.

For the 0.038 inch coil the coil diameter not 0.038 inch it is 0.028 inch or 0.032 inch. But due to the Dacron threads adding to the overall diameter of the coil, a 0.038 inch catheter lumen is required.

Coil closure technique

A prograde right heart catheterization is completed. The RA, PA and the RV pressures are recorded and the blood sampling is done. A descending aortogram is performed with a pigtail catheter at the level of the PDA. Standard fluoroscopy projections are posteroanterior and straight lateral; some PDA's and ampulla's are best visualized in an RAO at 30 degree angulation. It is crucial to measure the PDA minimum diameter during systole, as this measurement can be 30% larger than the diastolic dimension. This is important as this PDA minimum dimension is used to select the coil diameter. A number of techniques have been developed for the closure of PDA.

Successful transcatheter PDA closure is significantly dependent on choice of the correct size coil. Unfortunately there is no chart or specific rule that can define which size of the coil to use, this is due to the tremendous heterogeneity of the PDA shapes and sizes. Two main guidelines must be followed : the coil diameter should be at least twice as large as the minimum PDA diameter, and the coil should be long enough to make three or four loops-preferably four; one loop in the main pulmonary artery and the three loops in the aortic ampulla.

One technique is the retrograde deployment of the coil. The lateral fluoroscopy is used during most of the coil implant. Using a 5 French RCA or a GL the PDA is crossed into the Main Pulmory Artery. The 0.038 inch coil is pushed through a Multipurpose catheter with a 0.038 inch Teflon wire such half to quarter loops outside the catheter. The catheter and coil are slowly pulled back until the coil contacts the PDA, causing a slight

flex of the coil. The Teflon wire is held steady as the catheter is pulled back , exposing the coil in the aorta. As more coil is exposed in the aorta, this segment of coil forms loops in the aorta. When all the coils out of the catheter, the loops in the aorta are pulled into the ampulla, setting the coil in position. As the loops of the coil are being deployed in the aorta, it is important to make sure that additional coil is not advanced into the PDA resulting obstruction in the LPA. After 10 minutes an angiogram is performed to show whether there is any residual shunt or not. Several interventionalists devised techniques to provide better control of the coil during implant . Four techniques are mentioned below.

One, Moore placed a balloon wedge catheter from the MPA through the PDA prior to the coil placement. As the coil is being exposed in the descending aorta, the balloon is inflated in the aorta, then pulled back in to the PDA. The balloon stabilizes the segment of coil in the PDA as the remainder of coil is exposed in the aorta. As the coil retracts into the PDA, it is stopped by the balloon. As the balloon deflates slowly, the coil moves into the ampulla.

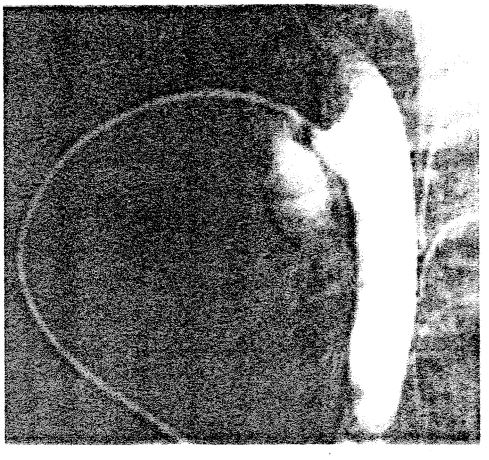
Two, Kuhn and Latson developed a catheter to hold on to the coil during implant. The distal end of an end holed catheter is heated with a hot air gun, then pulled out to taper the catheter and lumen diameter. The catheter is cut in this tapered segment, and a 0.032 inch wire is advanced through the catheter. This catheter provides a snug fit to implant a 0.038 inch coil. If during coil implant, the coil position is not optimal, since the coil is held by the tapered catheter tip, the coil can be pulled out of PDA and out of the patient through the femoral sheath.

Three, Ing and Sommer used an Amplatzer goose neck retrieval snare to grasp the half loop of the coil in the MPA. Then the remainder of the coil was deployed into the aorta . if needed, more coil could be pulled through the PDA into the MPA. When the coil was in good position, it was released by the snare. If the coil was not in good position, the coil could removed through the sheath.

Four, Hays used a three French bioptome to hold onto the 0.038 inch coil during implant. The coil can then be delivered through a 6 French Mullins sheath. This technique is more useful when we are dealing with multiple coil implantation so that we can tie all the coils together and catch it by the bioptome and taken into a loader sheath which is at least one French size sheath less than the delivery sheath. For example if we are going to put a 0.052 inch coil and two 0.038 inch coils we take an 8 French Mullins sheath and take a 7 French loader sheath. If the coil was not well positioned, this technique allows us to pullback the coils back to the sheath and looks for a good position. Bioptome allows a controlled delivery of the coils.

The retrograde approach (through the arterial side) is now only used for very small PDA's which cannot be crossed through the venous side. The most frequent technique used is the prograde(through the venous side) approach. The prograde approach requires crossing the PDA from the MPA using a wire and an end hole catheter. The coil can be delivered through this end holed catheter or exchange it for a large sheath.

The prograde approach, delivering the coil through the transseptal sheath, provides excellent coil control and easy coil retrievability. An end hole catheter is used to cross the PDA into the descending aorta. Over a Teflon wire the catheter is exchanged for a suitable transseptal sheath. The sheath tip is placed in the mid thoracic aorta, then the wire and the dilator are removed. The coil is modified so the bioptome can hold it tightly. The smooth ball of the coil tip is pulled 0.2 mm away from the windings using a hemostat. The ball is grabbed by the bioptome jaws, which are held then in the closest position. The coil and the bioptome are then pulled into the loader sheath, and then advanced into the hub of the transseptal sheath. The coil is advanced out of the transseptal sheath, such that three loops of coil are formed out side the sheath in the descending aorta and ¾ loops remains in the sheath. The sheath coil and the bioptome are pulled as one unit back into the ampulla. If the coil is well positioned in the PDA , the sheath is slowly pulled back into the MPA. Then the coil is released by opening the bioptome jaws. An angiogram can be done at the end of the procedure to know whether there is any residual shunt. The pictures below shows the implantation of a PDA coil.

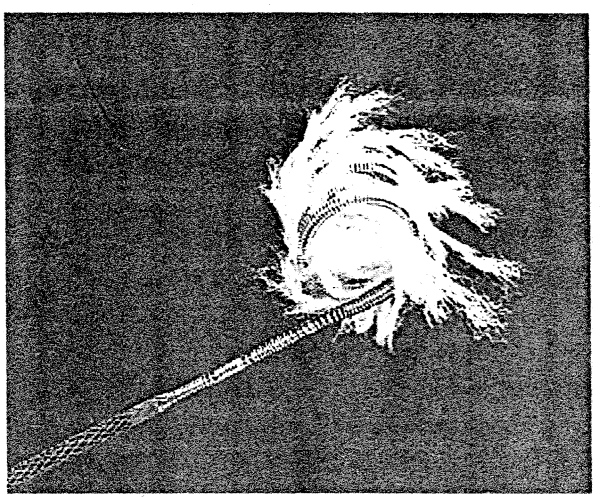


Detachable Coils

The Gianturco embolization coils are available with a detachable mechanism. This detachable coil is named as Jackson coil. Recently a very similar coil, named Flipper coil has been used. The coil has a mandrel which is inserted through the hollow core of the windings to straighten the coil during insertion into the catheter. When the coil is near the tip of the catheter , the mandrel is removed , which allows the coils to loop. There are helical female type threads on the proximal end of the coil. The delivery wire, which has male threads screws into the proximal end of the coil, affording a controlled mechanism to advance and retrieve the coil from the catheter. When the coil is in appropriate position, the delivery wire is unscrewed from the coil releasing the coil.

The Flipper coil is implanted using the same technique as the standard coil. The flipper coil comes in coil diameters of 3,5,6.5 and 8 mm and various lengths to produce

three to five loops. It requires a six French catheter with an internal lumen of 0.041 inch. Although the coil is reportedly a 0.038 inch wire, it does not appear to have the same strength as the standard 0.038 inch coil and is more likely to pull through the PDA. Thus instead of using the standard coil recommendations of implanting a coil diameter that is twice as large as minimal PDA diameter, a Flipper coil diameter should be 2.5 to 3 times larger than the minimal PDA diameter. The Dacron threads on the Flipper coil are shorter than the standard coil, thus preventing threads on the distal coils from getting tangled on threads on the proximal coil; tangled threads make it more difficult to pull the coil back into the catheter. A Flipper coil is shown below.



Duct-Occlud, Nit-Occlud

The Duct occlud device was the next device available for the PDA closure. It consisting pf a stainless steel coil that has primary windings and secondary loops, there are no Dacron threads on the coil. There were two different coil shapes; the standard device has an hour glass shape, and the reinforced device is a single cone shaped with double windings. The largest diameter of the coil should be as large as ampulla diameter. The coil is straightened by inserting a nitinol core wire and then advanced through a sheath.

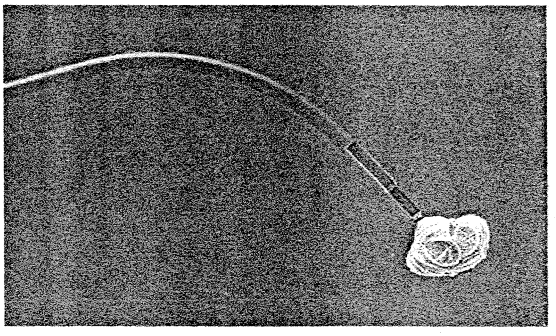
The Duct-Occlud is implanted like the other coils prograde approach is used. Through a 4 French or 5 French catheter placed through the PDA into the aorta , the coil and wire are advanced. As the coil is pushed out of the catheter, it returns to its conical shape. The catheter and coil are pulled back into the ampulla, then the remainder of the coil is advanced out of the catheter. The device is made of nitinol.

DEVICES

Gianturco-Grifka Vascular Occlusion Device

The Gianturco-Grifka vascular occlusion device consists of two basic components, a flexible nylon sack and an occluding filler wire. The sack is constructed from two pieces of tightly woven nylon. The sack is attached to an end hole catheter that has an everted flare on its distal tip; the sack fits tightly over the flared catheter tip. The sack catheter is inside an end holed release catheter; the release catheter is used to push the sack off the sack catheter. A modified guide wire is advanced through the end hole catheter into the sack; this filler wire is a 0.025 inch stainless steel guide wire with three modifications: the stiff inner core is removed, there is a J curve on the distal tip, and the proximal tip ends in a smooth ball. The filler wire is advanced through the sack catheter by a pusher wire, a slightly stiffer wire that also has a smooth ball shape on its distal tip. The balls on the end of each wire overlap and are contained within a thin plastic catheter. As long as the balls are overlapped and rest inside the plastic catheter, the wires are attached. Once inside the sack, the filler wire coils filling the sack, thus occluding the vessel lumen, and providing transmural pressure to maintain the sack position in the PDA. When all of the filler wire is pushed into the sack, two wires are separated. Then the coil filled sack is released from the catheter. To release the sack, the release catheter is advanced against the sack and held in this position. By pulling the sack catheter with a steady firm pull, the everted flare on the sack catheter is pulled through the neck of the sack. The sack is delivered to the PDA through a 8 French Mullins transseptal sheath.

A Gianturco-Grifca vascular occlusion device is shown here.



Amplatzer Duct Occluder

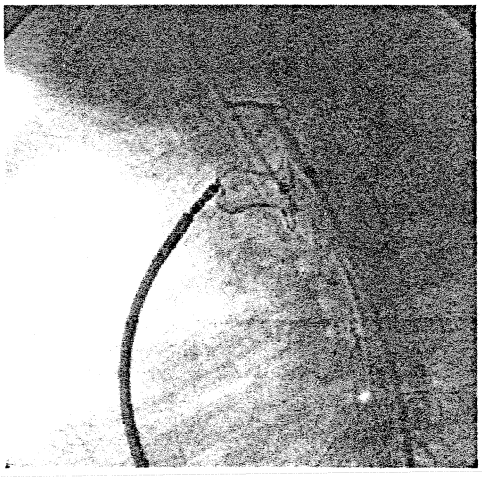
There are a number of PDA's that are still difficult to occlude with the above said devices due to larger size of the PDA. Recently Amplatz developed a new occlusion device for PDA.

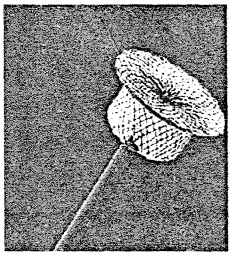
The Amplatzer Duct Occluder(ADO) is a self expanding occlusion device made from 0.003 inch or 0.005 inch nitinol wires configured into a mesh frame work. The nitinol wire has memory properties that allow it to be delivered to the PDA through a small transseptal sheath, then the device returns to its original shape as it is pushed out of the

sheath. The device is a mushroom shaped, with a flat circular retention disk and a cylinder shaped body. The retention disk rest on the aortic side of the ampulla. The cylindrical body, expanded in the PDA, creates radial force to maintain the device in PDA and occlude the PDA lumen. There is polyester fabric inside the device to help induce thrombosis. The cylindrical body has a recessed screw connector that has female threads; the male threads of the delivery cable are screwed into this threaded connector.

The cylindrical body is very slightly tapered, which results in two diameters; the diameter nearest the retention disk is 1-2 mm greater than the proximal end near the screw connector. The retention disk is 4-6 mm larger than the cylinder nearest the disk. For example an 8/6 device the cylindrical body is 6mm diameter at the screw connector , and 8mm diameter at the retention disk, and the retention disk is 12mm. The length of the device is 5-8mm.The device is available in 5-4,6-4,8-6,10-8, and 12-10.

The aortogram is done in the Lateral view. The minimum PDA diameter, the aortic ampulla diameter, and the length between these two diameters are measured. The size of the device is selected such that the smallest device diameter should be at least 2mm larger than the smallest PDA diameter. The device is delivered by the prograde approach. The 6 to 8 French sheath in accordance with the device can be put into the PDA over an exchange wire. The Amplatzer Duct occluder is screwed into the delivery cable, then unscrewed half turn (to make sure that the device is not over tightened onto the cable). The device and the cable are inserted through the transseptal sheath. Just the retention disk is advanced out of the sheath into the aorta. The sheath and the device are pulled simultaneously until the retention disk contacts the ampulla. The sheath is pulled back to allow the cylindrical body expand with the PDA. When an appropriate device position is confirmed the cable is unscrewed using the plastic wise. An Amplatzer Duct Occluder and it in situ are shown here.





Complications of occluder devices

Because of the size of the device needed to occlude a large PDA, if this device were to embolize, the complications could be profound. The GGVOD is more likely to

embolize into the pulmonary artery, the ADO would probably embolize into the aorta. Possibly the ADO could be captured by a snare catheter and pulled back into a transseptal sheath 2- 4 French size larger than the sheath size used to implant it. Stenosis of the Left pulmonary artery can occur due to the device pressing on the origin of the LPA.

ASD DEVICE CLOSURE

Introduction

The Atrial septal defect is a congenital heart disease, in which the inter atrial septum is not intact and makes an abnormal connection between the right and left atrium. This disease may not make the patient symptomatic so we cannot diagnose this defect in child hood so the defect is seen in adults also. As a therapeutic intervention, the Device closure of ASD is of importance in the cardiac catheterization lab. Different techniques are used early in the closure and the previous devices used are: King's and Mill's device, Rashkind devices, Buttened device, Clamshell devices, The angel wings das device, and the amplatzer septal occluder. Blockaid septal occluders are also used in our lab. We are mainly using the Amplatzer septal occluder and the Blockaid device.

The Amplatzer Septal Occluder

The Amplatzer Septal occluder (ASO) is a unique device that combines the advantages of being a double-disk device with a self centering mechanism. It is an important device for the closure of Atrial septal defect and Patent foramen Ovale(PFO), and fontan fenestration.

The Device

The ASO device is constructed from a 0.004 to 0.0075- inch Nitinol (55% nickel and 45% titanium) wire mesh that is tightly woven into two flat disks. There is a 4 mm connecting waste between the two disks that corresponds to the thickness of the atrial septum. Nitinol has super elastic properties with shape memory. This allows the device to be stretched into almost linear configuration and placed in a small sheath for delivery and then reform its original configuration within the heart when not constrained by the sheath. Nitinol also has been proven to have excellent biocompatibility. The device size is determined by the diameter of its waste and is constructed in various sizes, ranging from 4 to 40 mm (with 1 mm increment up to 20 mm and; 2 mm increment up to the largest device currently available at 40mm). The two flat disks extend radially beyond the central waist to provide secure anchorage. Patients with ASD usually have left to right shunt. Therefore the left atrial disk is larger than the right atrial disk. For devices 4 to 10 mm, the left atrial disk is 12 mm and the right atrial disk is 8 mm larger than the waist. However, for devices larger than 11 mm, the left atrial disk is 14 mm larger and the right atrial disk is 10 mm larger than the connecting waist. Both disks are angled slightly toward each other to ensure firm contact of the disk to the atrial septum. Three Dacron polyester patches are sewn securely with polyester thread into each disk and the connecting waist

to increase the thrombogenecity of the thread is laser welded to the right atrial cable to the device.

Device. A stainless steel sleeve with a female disk. This sleeve is used to screw the delivery

The sheath size for an amplatzer Septal Occluder is shown below.

Device size	Recommended sheath size
4 mm	6-7 French
5 mm	6-7 French
6 mm	6-7 French
7 mm	6-7 French
8 mm	6-7 French
9 mm	6-7 French
10 mm	6-7 French
11 mm	7 French
12 mm	7 French
13 mm	7 French
14 mm	7 French
15 mm	7 French
16 mm	7 French
17 mm	7 French
18 mm	8-9 French
19 mm	8-9 French
20 mm	8-9 French
22 mm	9 French

24 mm	9 French
26 mm	10 French
28 mm	10 French
30 mm	10 French
32 mm	10 French
34 mm	12 French
36 mm	12 French
38 mm	12 French

Patient Selection

In theory, ASD device can be used in any patient with an ostium secundum ASD if there is an adequate rim of tissue more than 5mm from the margins of the defect to the adjacent Mitral and tricuspid valves, SVC,RUPV and coronary sinus. The presence of an anterior rim is not essential for the Amplatzer. Defects up to 33 mm anatomic diameter by 2D TEE (40 to 42 mm balloon stretched) were closed success fully using the ASO. The shunt is calculated using the equation,

$$Qp/Qs = \frac{\text{Saturation of aorta} - \text{Mixed venous sturation}}{\text{Saturation of PV} - \text{saturation of PA}}$$

$$\text{Where mixed venous saturation is} = \frac{2^* \text{SVC} + \text{IVC}}{3}$$

Device Implantation Technique

The procedure is done under continuous trans esophageal echocardiography, or intracardiac echocardiography. A femoral artery and a femoral vein punctures are taken. The artery is used for monitoring of the pressure and for blood gas sampling. The presence of partial anomalous pulmonary venous drainage should be excluded by entering all the pulmonary veins.

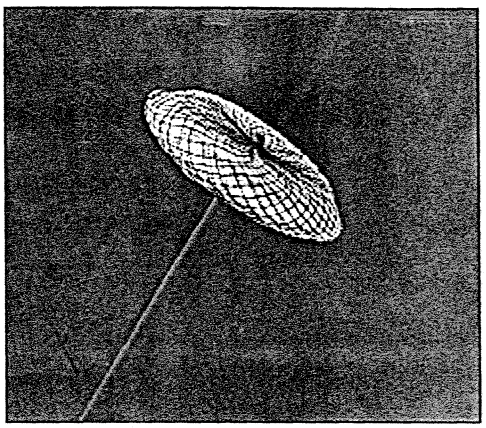
An end hole catheter is (multi purpose or GL) is manipulated through the ASD into the left upper pulmonary vein. Using an exchange length Amplatz extra stiff guide wire, a sizing balloon catheter is exchanged for the catheter. Balloon sizing of ASD is very important step. We have an AGA sizing balloon which have markers inside the balloon for calibration. The balloon is positioned in the middle of the septum across the defect. The balloon is inflated under fluoroscopic and echocardiographic guidance until waisting and disappearance of shunt occurs. The stretched diameter of the ASD is then measured both echocardiographically and cine angiographically. Once the stretched diameter of the ASD is determined, the balloon is removed, keeping the guide wire in the left upper pulmonary vein. The device size is choosed +2 mm from the stretched ASD diameter, depending on the size of the patients left atrium and the total length of inter atrial septum.

The proper size delivery sheath is advanced over the guide wire to the left upper pulmonary vein. Both dilator and wire are removed, keeping the tip of the sheath in the left upper pulmonary vein. Extreme care must be exercised not to allow passage of air into the sheath. The device is then screwed into the tip of the delivery cable, immersed in normal saline and drawn into a loader underwater seal to expel air bubbles out of the system. The loader containing the device is attached to the proximal hub of the delivery sheath. The cable with the ASO device is advanced to the distal tip of the sheath, taking care not to rotate the cable while advancing it in the long sheath to prevent premature unscrewing of the device. Both cable and delivery sheath are pulled back as one unit to the middle of the left atrium.

The left atrial disk is deployed first under fluoroscopic and echocardiographic guidance. Part of the connecting waist should be deployed in the left atrium, very close to the atrial septum. While applying constant pulling of the entire assembly and withdrawing the delivery sheath off the cable , the connecting waist and the right atrial disk are deployed in the ASD itself and in the right atrium respectively. Proper device position can be verified by the TEE. If adequate position is achieved we can deploy the device there by rotating the plastic wise which is attached to the distal end of the delivery cable. A hand injection can be done through the delivery sheath into the Right atrium for the adequate positioning of the Device.

The Device

The picture below shows an amplatzer septal occluder.



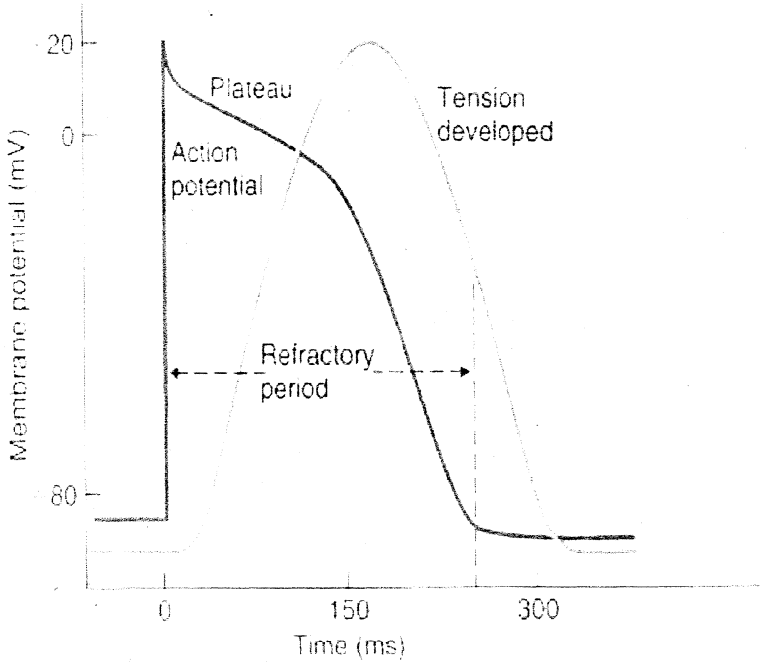
Complications of ASD Device

Reported complications are rare and can be managed in the catheterization laboratory. The most common complication is the device displacement and the device embolization. Which needs the transcatheter snaring of the Device. Arrhythmias were reported in a very rare cases.

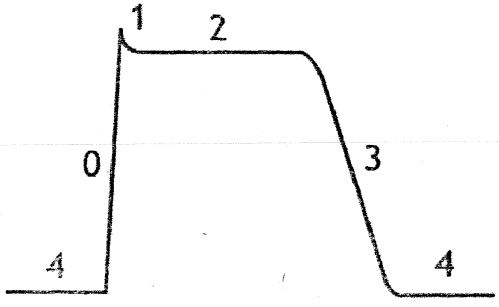
THE CARDIAC ELECTROPHYSIOLOGY

Basics of electrophysiology

The cardiac Muscle cell has some special properties like Automaticity, conductivity, Ionotropicity and exitability. A cardiac muscle cell in its resting state has its out side positive and inside negative. This is called its resting state or the cell is in polarized condition. Once the cell is excited the Depolarization of the cell occurs resulting in an action potential of the cell. The picture below shows an action potential curve of the cardiac cell.



Phases of the cardiac action potential



The cardiac action potential has five phases.

The standard model used to understand the cardiac action potential is the action potential of the ventricular myocyte. The action potential has 5 phases (numbered 0-4). Phase 4 is the resting membrane potential, and describes the membrane potential when the cell is not being stimulated.

Once the cell is electrically stimulated (typically by an electric current from an adjacent cell), it begins a sequence of actions involving the influx and eflux of multiple cations and anions that together produce the action potential of the cell, propogating the electrical stimulation to the cells that lie adjacent to it. In this fashion, an electrical stimulation is conducted from one cell to all the cells that are adjacent to it, to all the cells of the heart.

Phase 4

Phase 4 is the resting membrane potential. This is the period that the cell remains in until it is stimulated by an external electrical stimulus (typically an adjacent cell). This phase of the action potential is associated with diastole of the chamber of the heart. Certain cells of the heart have the ability to undergo spontaneous depolarization, in which an action potential is generated without any influence from nearby cells. This is also known as automaticity. The cells that can undergo spontaneous depolarization the fastest are the primary pacemaker cells of the heart, and set the heart rate. Usually, these are cells in the SA node of the heart. Electrical activity that originates from the SA node is propagated to the rest of the heart. The fastest conduction of the electrical activity is via the electrical conduction system of the heart.

In cases of heart block, in which the activity of the primary pacemaker does not propagate to the rest of the heart, a latent pacemaker (also known as an escape pacemaker) will undergo spontaneous depolarization and create an action potential. The mechanism of automaticity is still unclear. Depolarization of SA and AV nodal cells largely depend on a net increase in intracellular positive charge. Mechanisms include a decrease in the net K^+ outward flow, and a time-dependent increase in flow of Na^+ and Ca^{2+} ions.

Phase 0

Phase 0 is known as the rapid depolarization phase. The slope of phase 0 is determined by the maximum rate of depolarization of the cell and is known as V_{max}. This phase is due to opening of the fast Na^+ channels and the subsequent rapid increase in the membrane conductance to Na^+ (g_{Na}) and a rapid influx of ionic current in the form of Na^+ ions (I_{Na}) into the cell. The ability of the cell to open the fast Na^+ channels during phase 0 is related to the membrane potential at the moment of excitation. If the membrane potential is at its baseline (about -85 mV), all the fast Na^+ channels are closed, and excitation will open them all, causing a large influx of Na^+ ions. If, however, the membrane potential is less negative, some of the fast Na^+ channels will be opened earlier, causing a lesser response to excitation of the cell membrane and a lower V_{max}.

The maximal fast inward Na^+ current is generated when the membrane potential is at the normal resting potential (-85 to –95 mV). If the resting membrane potential is reduced to a low enough level, the increase in fast inward Na^+ current may be inadequate to produce a response, making the fiber unexcitable

The fast Na^+ channel, The fast sodium channel is made up of two gates, the m gate and the h gate. It is the interaction of these two gates that allows Na^+ to enter the cell through this channel. In the resting state, the m gate is closed and the h gate is open. Upon electrical stimulation of the cell, the m gate opens quickly while simultaneously the h gate closes slowly. For a brief period of time, both gates are open and Na^+ can enter the cell across the electrochemical gradient.

Phase 1

Phase 1 of the action potential is due to closure of the fast Na^+ channels, causing an abrupt end to the depolarization of the cell. The transient net outward current is due to the movement of K^+ and Cl^- ions. Phase 0 and 1 together correspond to the R and S waves of the ECG.

phase 2

This "plateau" phase of the cardiac action potential is sustained by a balance between inward movement of Ca^{2+} (I_{Ca}) through L-type calcium channels and outward movement of K^+ through the slow delayed rectifier potassium channels, I_{Ks}. The sodium-calcium exchanger current, $I_{Na,Ca}$ and the sodium/potassium pump current, $I_{Na,K}$ also play minor roles during phase 2. Phase 2 of the action potential corresponds to the ST segment of the ECG.

Phase 3

During phase 3 of the action potential, the K^+ channel is still open, allowing more K^+ to leave the cell and accumulate in the extracellular space. This net loss of positive charge causes the cell to repolarize. The k^+ channels close when the membrane potential is restored to about -40 to -45 mV. Phase 3 of the action potential corresponds to the T wave on the ECG.

The cardiac electrophysiologic studies

The Modern era of intra cardiac electrocardiography began in the late 1960 by recording the His bundle potential in humans. The introduction of programmed stimulation expanded this technique. The Most attention was in understanding the mechanism of Wolf-Parkinson-White syndrome and other supraventricular arrhythmias. Most recently the Ventricular tachycardia is being assessed by Electrophysiology. Here we will discuss the indications and techniques of doing an electrophysiology study.

Indication

EPS has been used in the evaluation of a wide variety of cardiac arrhythmias. It is mainly indicated in cardiac arrest, unexplained recurrent syncope, recurrent sustained ventricular tachycardia, WPW syndrome with symptomatic tachycardia, Other supraventricular tachycardias, and Bradycardias.

Sinus node disease

For patients with sinus node disease, the decision regarding implantation of pacemaker depends upon the EP study and the patients symptoms. In some patients persistent symptoms may be associated with only mild ECG abnormalities. In this case determination of sinus node recovery time is useful, as a markedly prolonged sinus node recovery time(SNRT) is an indication for implantation of a permanent pacemaker. However a normal SNRT does not exclude a symptomatic Sick Sinus Syndrome. The sino atrial conduction time is a sensitive but nonspecific indication of sinus node dysfunction.

Atrioventricular block

For most patients with atrioventricular block the decision regarding a pacemaker can be made on history and the surface ECG without intracardiac recordings. Patients who have symptoms require a pacemaker and who have no symptoms for high grade infra nodal blocks. The intracardiac recordings will be helpful in patients with AV Wenkebach block associated with bundle branch block for concealed bypass.

Intraventricular conduction delay

Patients with symptomatic bifascicular block may have intermittent complete heart block that has escaped detection on monitoring. For these patients, demonstration, during EPS of pacing induced infranodal block or a markedly prolonged HV interval.

Supraventricular tachycardia

In Patients with Supraventricular tachycardia, EPS allows selection of drug therapy, and in cases refractory to drug therapy, it allows the selection of non medical forms of therapy such as specific antitachycardia pacemakers and anti tachycardia surgery.

Wolf-Parkinson-White syndrome

EPS is a must for patients with WPW syndrome for the mapping of the accessory pathway and its ablation.

Ventricular tachycardia (VT)

EPS is indicated for all patients with recurrent, sustained VT. This arrhythmia can be reproduced in the EP lab in 905 of patients using the techniques of programmed stimulation. EPS is mandatory for patients who are planning for an Implantable Cardioverter defibrillator(ICD) implantation.

Contraindications

EPS is contraindicated (1) when acute factors make the findings unrepresentative of the patients usual state(electrolyte abnormality, acute ischemia, and drug toxicity) and (2) when the patients underlying cardiac disease makes it likely that induced arrhythmias will be extremely difficult to terminate and carry a high risk of death.

Risks of EP study

Induction of serious arrhythmias are the main risk of EP study. Atrial fibrillation, or induction of hemodynamically unstable VT or VF.

Equipment

The basic equipment required for EPS is described below,

Fluoroscopy unit: Adequate imaging can be obtained with catheterization laboratory equipment.

Programmable Stimulator: The stimulator must be electrically isolated and allow introduction of one to three precisely timed premature stimuli, during both paced and spontaneous rhythm. It should allow selection of the pacing out put, interval between stimuli, and length of pause after each stimulation sequence. It must allow a rapid changeover from pacing modes used to induce arrhythmias to pacing modes used to terminate arrhythmias.

Multichannel physiologic recorder: The physiologic recorder must allow the simultaneous filtering, amplification and recording of a minimum of three surface ECG leads and four intracardiac signals. The recorder should be connected to a switch box

that allows selection of intra cardiac signals. Recordings must be possible at paper speeds ranging from 25mm/sec to 200mm/sec.

Resuscitation Equipment: All standard resuscitation equipment should be available in EP lab. We used to have two functioning defibrillators present in the laboratory , especially in VT study we used to connect defibrillator pads to the patient for an emergency cardioversion.

Intracardiac electrode catheters: Quadripolar catheters are used mainly in EP study with an electrode distance of 1cm and a decapolar catheter is used in coronary sinus mapping with the same electrode placing.

Intracardiac electrograms

For an Electrophysiology study we usually put the quadripolar electrodes in the High Right Atrium(HRA), The HIS bundle, the Right ventricular apex and a decapolar catheter in the coronary sinus(CS). For a Quadripolar electrodes the distal two electrodes are used for pacing and the proximal electrodes are used for recording the intracardiac ECG. For a decapolar catheter the distal two paces and the others records the electrogram.

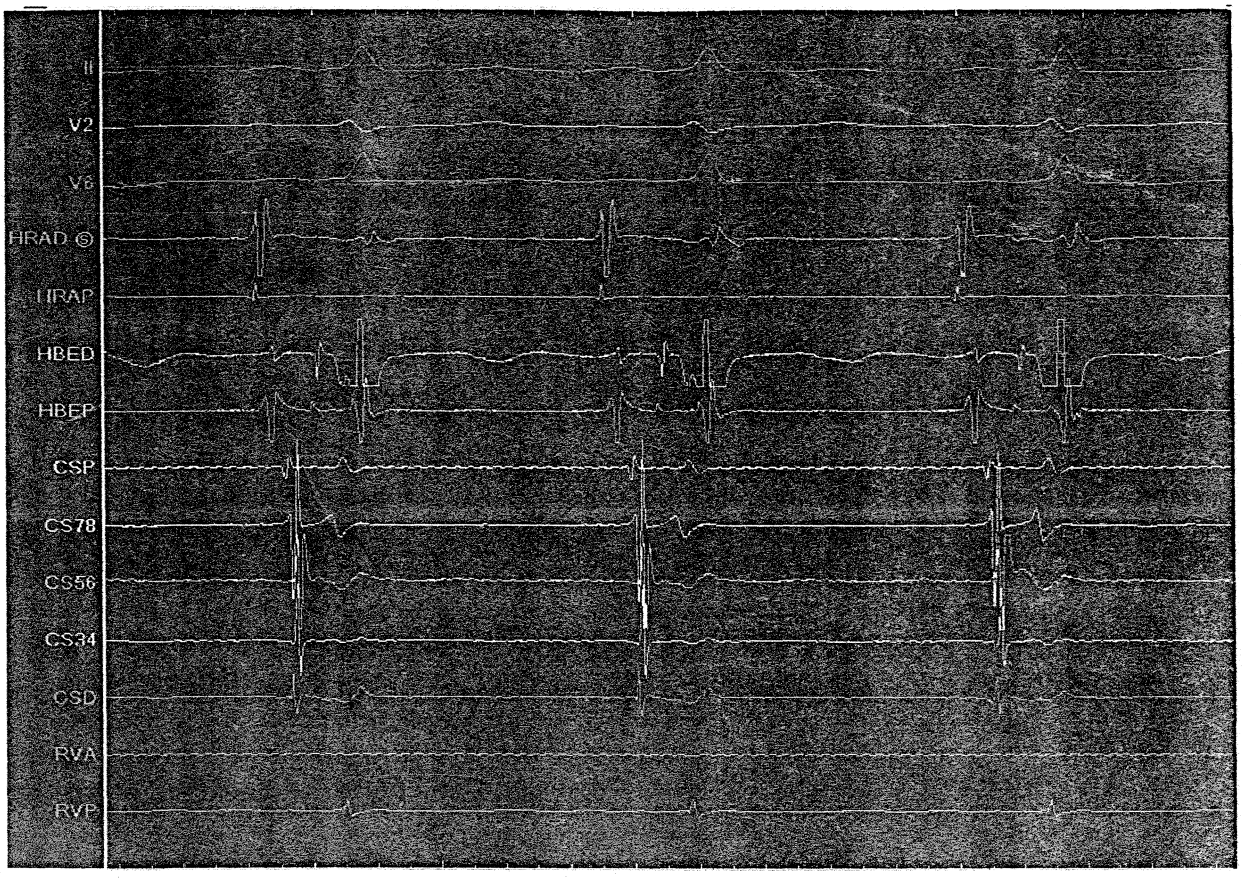
The His Bundle Electrogram

In a simple way the His bundle Electrogram consist of three deflections which are usually biphasic. The first one is an 'A' wave, next is an 'H' wave and the next is a 'V' wave. Our point of interest is in the AH, HV intervals.

AH interval: The AH interval represents conduction time from the low right atrium through the AV node to the His bundle. The AH interval is measured from the earliest onset of the atrial electrogram and the His bundle recording to the earliest deflection of the His bundle deflection. Normal AH interval is 60 to 125 msec. The AH interval is very sensitive to autonomic influences.

HV interval: The HV interval represents conduction time from the proximal his bundle to the ventricular myocardium. The HV interval is measured from the earliest deflection of the His bundle potential from the baseline to the earliest onset of ventricular activation recorded from multiple surface ECG' leads or from ventricular electrogram in the His bundle recording. The normal HV interval is 35 to 55 msec. The HV is not affected by the autonomic tone and is relatively constant.

The figure below shows a His Bundle electrogram with surface ECG;



Techniques of programmed stimulation

Type of stimuli

Standard EPS stimulator uses bipolar, cathodal square wave stimuli of 1.5 to 2.0 msec duration and twice the diastolic threshold. The threshold is determined by pacing at a high out put, gradually decreasing the out put during pacing, noting the output at which capture becomes inconsistent. If the threshold is greater than 1 to 2 mA, one should consider repositioning the catheter.

Incremental Pacing

The stimulator allows the administration of stimuli at any fixed rate desired or at any cycle length desired. The stimulator is synchronized to the native QRS complex, set for the desired interval, and activated. We can generally decrease the cycle length and go for higher rate this is called an incremental pacing. The heart rate and cycle length are related to each other by the equation,

$$\text{Heart rate} = \frac{60000}{\text{Cycle length}}$$

For example,

If the cycle length is 1000 then the heart rate is 60 and the vice versa. Incremental pacing is used in evaluating the sinus node function, for evaluating AV conduction, inducing and terminating supraventricular and ventricular arrhythmias.

Premature stimulation (Extra stimulus)

Basic drive versus native rhythm. The stimulator allows introduction of precisely timed premature stimuli during sinus rhythm, during paced rhythms, and during tachycardias. Many electrophysiologic properties can vary with heart rate, it is important to perform stimulation at multiple cycle lengths to maximize the induction of tachycardia. Suppose the patient has an intrinsic rate of 60 that means the cycle length 1000 msec, so we start with a paced cycle length of 900 msec and go down till we get an adequate information.

Single premature stimuli for measuring the refractory period. In general, extrastimuli are administered after every eight beats of basic drive. The extra stimulus is administered late in the cycle and progressively brought closer and closer to the last beat of the basic drive. The extra stimulus is moved toward the preceding beat until capture does not occur. The interval at which capture does not occur defines the effective refractory period . The beats of the basic drive are often called S1 and the premature stimulus is often referred to as S2.

Double and Multiple stimuli. The extra stimulus can be expanded to include more than one extrastimulus. In administering a second premature stimulus(S3), the first premature stimulus(S2) is set 10 msec beyond the refractory period. S3 is administered late in the cycle and gradually moved towards in 10 msec decrements until it no longer captures. This procedure can be repeated for a third extrastimulus(S4) and so on.

Premature stimulus is used in measuring refractory periods, initiating SVT and VT, defining the mechanism of SVT and VT, and terminating SVT and VT.

Protocols in Electrophysiology

1. Evaluation of sinus node function

(a) Sinus node recovery time (SNRT)

The sinus node recovery time is determined by pacing the high right atrium near the sinus node. Pacing rates just above sinus beat is selected and is maintained for 60 seconds and then abruptly discontinued. The maximal sinus node recovery time is defined as the longest pause from the last paced atrial depolarization to the first sinus return cycle at any paced cycle length. The corrected SNRT is equal to the sinus node recovery time minus the patients intrinsic sinus cycle length, and should be less than 550 msec.

(b) Sinoatrial conduction time (SACT)

Sino atrial conduction time can be determined by the extrastimulus method. The atria should be paced for eight beats at a fixed rate that is only slightly higher than the sinus rate. The return cycle after the eight beats is assumed to represent the intrinsic activity of the sinus node plus conduction time out of the sinus node. The SACT is determined by subtracting the basic cycle length from this cycle. The difference is divided by two and is considered normal when it is between 50 and 125 msec.

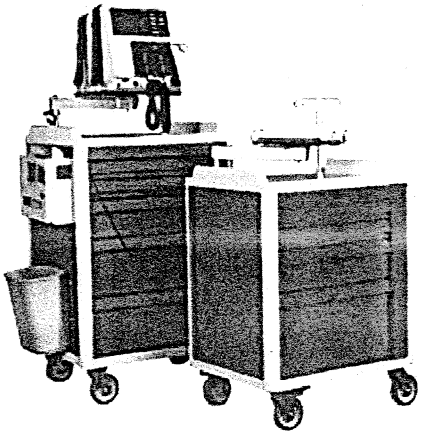
(c) Incremental Atrial pacing (IAP)

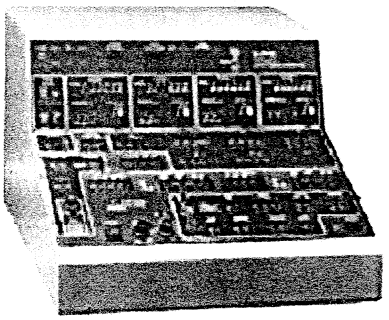
This is pacing the atrium by increasing the rate stepwise.

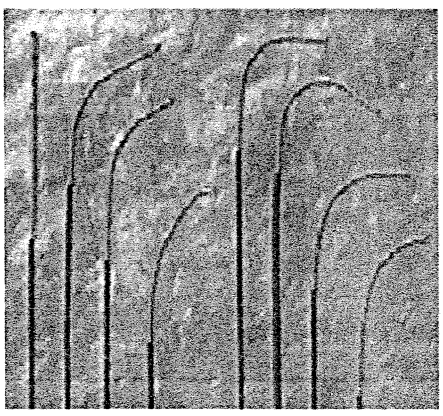
(d) Incremental Ventricular Pacing (IVP)

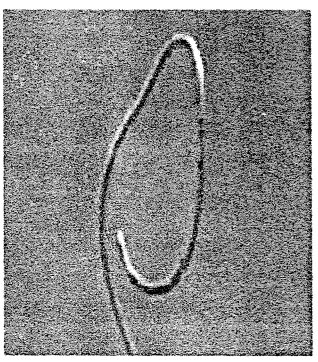
This is pacing the ventricle by increasing the rate stepwise.

Pictures of EP catheters and pictures of machines are given below,









CARDIAC PACING

Permanent pacemakers

Elmquist and Senning implanted the first artificial pacemaker in Stockholm in 1958. Now the pacemakers becoming increasingly compact, reliable and sophisticated.

Indications

The primary indication for a permanent pacemaker implantation is symptomatic bradyarrhythmias. In occasional situations severe assymptomatic bradyarrythmias are also indicated for a permanent pacemaker implantation.

The most common indications can be classified as follws; Acquired atrioventricular block, Complete heart block, Mobitz type II second degree AV block, Mobitz type I second degree AV block, when associated with symptoms, Atrial flutter or fibrillation with slow ventricular response when associated with Symptoms, AV block associated with Myocardial infarction, Bundle Branch Block associated with fixed or transient Mobitz type II AV block or Complete block, Sick Sinus Syndrome, Sinus bradycardia and arrest when associated with symptoms, Tachy-Brady Syndrome.

Contraindications

There are very few contraindications to permanent pacemaker implantations. Implantations are contraindicated in patients with active infections or who are on active anticoagulation therapy.

Equipments

Pulse generator, Ventricular and atrial leads, Sheath with puncture needle and 0.035 inch guide wire, Pacemaker systems analyzer(PSA).

Pulse generator

The pulse generator consists of electronic circuitry, based on digital chips, and has a power source for generating the electric stimuli usually all the present pacemakers use Lithium iodine batteries which lasts up to 7 to 10 years. The entire unit is hermetically sealed in a stainless steel or titanium housing to isolate the contents from the biologic environment.

Pacing Leads

Mainly two types of leads are available unipolar and bipolar. In the unipolar system the cathode (negative electrode) is at the distal tip of the lead, and the body of the pacemaker is the anode (positive electrode). In the bipolar systems the distal tip of the

lead (ring electrode) is the cathode and; a ring electrode usually 1 cm proximal to the tip, is the anode. The conducting tip of a lead will be platinum, and has an insulation of silicone rubber. The leads may be a Tined one (passive fixation) or a helix with a screw in mechanism (active fixation). The tip and ring connectors are made up of stainless steel. The lead tip is coated with steroids to reduce the endocardial inflammation the drug used is dexamethasone acetate.

Ventricular Leads

The ventricular leads are available with a length between 58 cm to 60 cm. Its insulation is silicone rubber and polyurethane, with removable stylets, they are thin and very flexible. So these stylets helps us for manipulation and positioning of a lead in the proper position of the ventricular myocardium. All the ventricular leads are supplied with straight stylets and we can reshape them into a J stylets so that it can seek its position at the ventricular apex. There will be a soft and a hard stylet. Now we are using the active fixation lead for cases with Tricuspid regurgitation and dilated ventricles. In this the tip electrode(cathode) is a Helix which can be screwed into the endocardium with 1.5 to 2 loops.

Atrial leads

The most commonly used atrial leads has a J configuration, which allows entry into the atrial appendage. They have a length of 53cm. There are J shaped stylets which are suitable for the positioning of the atrial lead in the atrial appendage. They also are available as tined and screw in types. The most atrial leads we are using are screw on leads because the atrial lead may come out.

The pulse generator

The pulse generator is the important part of the pacemaker system. The pulse generator has mainly the following components.

The sense amplifier: It receives the unipolar/bipolar heart signal P or R wave, filters and amplifies it. The signal is then passed on to the P/R detection unit depending on when in the interval the signal is received.

The P/R detection logic: This compares the signal to the programmed P/R sensitivity via a level detector and passes the result(capture no capture) on to the control Logic unit.

The control Logic: This unit decides if and when the pulse generator should stimulate the heart. The decision is based on the received signal and the value of all the programmed parameters stored in the Control Logic. Normal stimulation is mainly based on received signal from the P/R detection Logic and occurs when a command is send to the pace pulse output circuit.

The charge pump: the charge pump recharges the output capacitors to the stimulation amplitude. When the PG is close to RRT, the increased charging time results in a longer stimulation interval in which to reach a stimulation amplitude.

The Pace Pulse Out put: This circuit emits impulses with pulse amplitudes and widths determined by the control unit.

More over the pulse generator contains a memory unit, Telemetry unit, and telemetry coil, Programmable functions of a pulse generator

Pacing Modes

AOO, AAI, VOO, VVI, VDD, DDD are the common pacing modes. The classification of pacing modes can be accomplished best by the NASPE code as follows.

Position I: The first position represents the chamber to be paced, It may be ventricle (V) alone, atria(A) alone or both the chambers(D).

Position II: The second position represents the ability of the pacemaker to detect (Sense) spontaneous atrial depolarizatios (A), or spontaneous ventricular depolarizations (V), or both (D).

Position III: This position indicates the response to sensing, means what the pacemaker does when it detects a spontaneous depolarization. We will discuss this mode in detail later.

Position IV: This position indicates the level of programmability of the pace maker and rate modulation. The new generation pacemakers are rate responsive (R), a form of adaptive pacing in which the pacing rate is adjusted automatically for the patients need by sensing various physiologic parameters. When the rate modulation is not there this position indicates the degree to which the pacemaker can be programmed. It may be simply programmable (P), Multiprogrammable (M), or Communicating (C).

Position V: This position, if occupied, designate the presence of antitachycardia pacing, cardioversion, or defibrillation capabilities independent of anti Brady arrhythmia pacing function.

Response to sensing by a pulse generator

The response to sensing can be of the following four types,

O = None This means that there is no response to sensing that is the pacemaker will asynchronously paces the chamber without any sensing of spontaneous depolarization.

I =Inhibited When a spontaneous depolarization is detected, the pacemaker will not give a stimulus, this is because the pacemaker timing is reset by sensing as in VVI or AAI mode.

T =Triggered In this type even though a spontaneous depolarization is detected, the pacemaker will stimulate the chamber. Of course the stimulus has no effect in the chamber. If what has been sensed is a spurious noise signal, the stimulus may be effective. The single chamber triggered modes are often used in assessing pacemaker function and in identifying the effects of muscle potential sensing and extracorporeal electromagnetic interference.

D = Dual This denotes the simultaneous presence of triggered and inhibited pacing. In the VDD mode, ventricular pacing is inhibited when the escape timing is reset by ventricular sensing. The special use of "triggering" concept means that ventricular stimulation is evoked, after a suitable atrioventricular interval, upon sensing of a spontaneous atrial depolarization. In the DDD mode sensing results in inhibition of a pending stimulus in the chamber where the sensing occurs; at the same time, atrial sensing triggers ventricular stimulation after an appropriate interval.

Pacing polarity

The pacemaker can pace in bipolar as well as in unipolar mode.

Sensing polarity

The pacemaker can sense in bipolar as well as in unipolar mode.

Basic rate

The automatic rate is the interval between consecutive atrial or ventricular paced stimuli. This setting can be programmed in between 30 and 150 beats per minute. Usual shipped rate may be 60 or 70 bpm.

Magnet rate

A magnet will be used to check the function of a pulse generator, a magnet on the pulse generator makes it to pace in asynchronous mode. The usual magnet rate of a pulse generator is 100 bpm. It is not a programmable value.

Pulse Amplitude

It is the Voltage of a pulse delivered by the pulse generator for the stimulation of the Myocardium. It is measured in Volts. Usually an voltage of 1 Volt is needed for the successful stimulation of the Myocardium. The present pulse generators allows us to program it up to 4.5 volts or more in case of a chronic threshold change.

Pulse width

It is the time duration of a pulse delivered by the pulse generator for the stimulation of the myocardium. It is measured in milliseconds. Usually a pulse width of 0.5 ms is used for stimulation. We can program pulse width also to higher values.

P/R sensitivity

Sensitivity is the ability of the detection system of the pacemaker to recognize the intrinsic cardiac signal and to use that signal to control the output of the pacemaker. The sensing circuit recognizes the R or P wave by its amplitude and slew rate, where slew rate is the rate of change of voltage amplitude with time. The capability to recognize slew rate allows the sensing circuit to differentiate between the QRS and the T wave. Lower numbers indicate that the unit is more sensitive to intrinsic activity, and higher numbers are less sensitive. It is measured in milliVolts.

Refractory period

Pulse generators operating in a sensing mode incorporates a refractory period, immediately following a pacemaker output or a sensed event, the pacemaker ceases to be responsive to detectable signals for a pre determined period. This prevents pulse generator from detecting the terminal portion of the depolarization signal and in some times the repolarization signal which might result in timing errors. This is usually measured in milliseconds and can be programmed between 250 to 550 msec. Usually a refractory period of 300 to 330 msec is used.

Impedance

It is the measure of opposition of the passage of current through the lead body to the myocardium. It is measured in Ohms and can be calculated by using the Ohms law.

$$R = V / I$$

A very low impedance shows that there is a current drainage any where in the lead and a very high resistance indicates repositioning of the lead.

Escape rate and Hysteresis

The escape rate is the interval between the last sensed beat and the first pacemaker beat to follow.

Hysteresis occurs when the escape interval is longer than the programmed interval. Even though the pulse generator may be programmed to pace at 72 bpm it will not begin pacing until the rate falls below 60 bpm, giving the patient a greater opportunity for conducted sinus beats. Hysteresis is used to avoid competition between the intrinsic activity and the paced events.

AV interval

The AV interval is programmed within normal physiologic range (0.12 to 0.20 seconds) for maximal cardiac output.

Parameters of the lead

The R/P wave is checked first, this represents the sensitivity; a P wave of 3 to 5 mV and an R wave of 10 to 15 mV are satisfying.

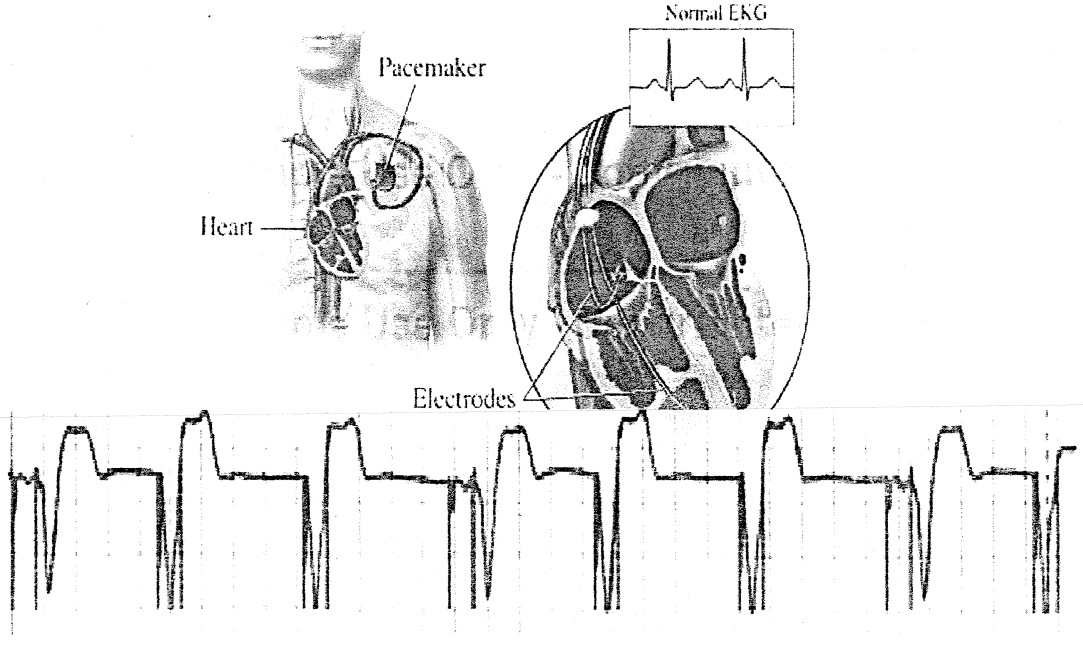
Threshold test, the chamber is paced first at a rate of 80 to 100 and an output of 5.0 volts, at this time the resistance is noted. Then slowly decrease the voltage till the capture is lost, the value just above it which is consistently pacing is the threshold voltage. A current threshold of less then 1 mA and a voltage threshold of less than 1 V are desirable.

Pace at maximum amplitude to make sure that no diaphragmatic stimulation occurs.

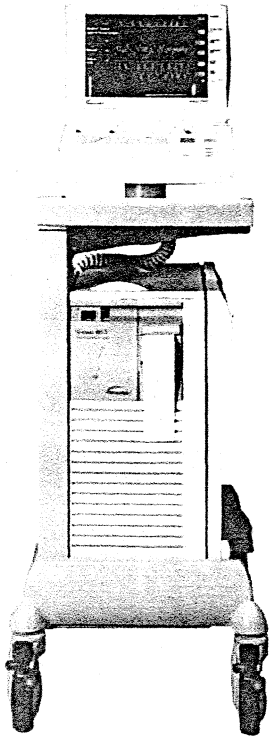
Pacemaker syndrome

The pacemaker syndrome is a group of symptoms related to adverse hemodynamic effects of ventricular pacing. The symptoms include fatigue, dizziness, and pulmonary congestion. This syndrome is most common in patients with sick sinus syndrome who manifest with a VA conduction during ventricular pacing.

The figure below shows a patient with a pacemaker in situ and shows a tracing of ECG from a pacemaker patient.



INTRAAORTIC BALLOON PUMP (IABP)



An intraaortic balloon pump (IABP) is a device that increases blood flow to the heart muscle and decreases the heart's workload through a process called counterpulsation.

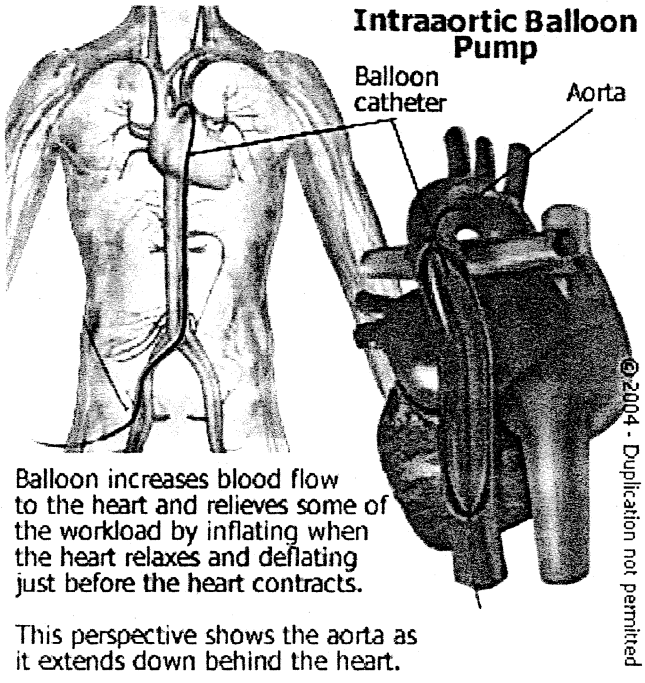
In the early 1950s, researchers discovered that the heart is doing two different kinds of work. The first type of work occurs during the pumping part of the heartbeat (the systole), during which the heart must overcome the blood pressure in the aorta in order to pump blood through it. The second type of work occurs during the relaxed part of the heartbeat (the diastole), during which the heart receives blood via backflow from the aorta. This was described as the difference between pressure work and flow work, respectively. The flow of blood to the heart muscle through the coronary arteries mainly occurs during the diastole. A healthy heart is normally able to accomplish both types of work effectively, but a weakened heart will have more difficulty.

Counterpulsation was introduced as a strategy by which the blood flow to the heart muscle could be increased during diastole (less flow work). The increased blood flow would provide the heart with more oxygen, which would improve circulation, cardiac output and the heart rate. As a result, the technique relieves, for example, the pain and discomfort from angina.

In 1958, Harvard researchers documented positive results from counterpulsation in laboratory studies, and animal studies in the early 1960s supported these results. The IABP arose from these early studies.

The device contained a small balloon that was inflated inside the aorta when the heart muscle relaxed, which increased pressure inside the heart chambers and increased blood flow to the coronary arteries. Then the balloon was deflated just before the heart muscle pumped, creating a vacuum effect that aided blood flow from the heart, thus reducing systolic blood pressure and increasing the heart's ability to pump blood. In this way, the device was shown to improve blood flow to the heart muscle and reduce the workload of the heart muscle. The gas used to inflate the balloon is either carbon dioxide (which has fewer consequences in the rare event of a balloon bursting) or helium (which has the fastest ability to travel or diffuse). Because it is placed in the aorta, patients with aorta-related problems (e.g., aortic dissection, aortic regurgitation) are not generally eligible for this treatment.

Practical aspects of use of balloon pumps



Drive units

- Operation is based on recognition of the fact that the interval between electrical and mechanical events of cardiac cycle is variable, varying from patient to patient and in a given patient, with heart rate
- In a given patient the systolic time interval at a given heart rate can be used to predict the STI at other rates. Drive units are preprogrammed with families of STI/heart rate curves. At the initiation of pumping the operator estimates the intervals from the QRS complex to aortic valve opening and closure and enters them into the machine. The drive unit then selects the STI/heart rate curve that most adequately corresponds to those values to calculate balloon timing parameters
- Current drive units unable to realise maximal benefits of IABP during many arrhythmias

Balloon catheters

- 30-40 ml displacement volume
- Usually have central lumen concentric with and inside the helium channel. Leads to catheter tip and allows use of guide wire to introduce catheter and also allows recording of central arterial pressure, thus eliminating need for a separate arterial line to obtain waveform required for IABP

IABP insertion

The most common strategy for inserting an intraaortic balloon pump (IABP) is a catheter-based procedure. This procedure is done in the operating room, a catheterization laboratory or a hospital's intensive care unit (ICU). In an emergency, it may be performed in the patient's hospital room before the patient is moved to the ICU. Just prior to the procedure, the area to receive the catheter is shaved (if necessary) and sterilized to prevent infection, and a mild sedative may be given to calm the patient. Once the patient is made comfortable, heart monitoring begins, an intravenous (I.V.) line is inserted and the area to receive the catheter is numbed with local anesthesia. The injection of the local anesthesia may result in a brief period of discomfort. This is normal and should be no cause for concern. Once the local anesthesia takes effect, the physician will prepare to insert an IABP-tipped catheter. The IABP-tipped catheter is usually inserted through the femoral artery in the groin and guided all the way up to the aorta. Once inserted in the aorta, the catheter remains there until the IABP is removed.

During balloon insertion the following steps are important:

1. Heparinise patient prior to insertion of catheter providing there are no contraindications such as recent surgery. After cardiac surgery patient should be given low-molecular weight dextran at 20 ml/hr instead of heparin. (Do not exceed total daily dose of 10 ml/kg)
2. Prep skin
3. Fully collapse balloon applying 30 ml vacuum with 60 ml syringe
4. Insert needle into femoral artery at 45° and pass it through both walls of artery. Withdraw needle until strong pulsatile jet of blood is obtained
5. Pass guidewire through needle and advance until tip is in thoracic aorta. Wire should pass very easily
6. Pass sheath over wire in similar manner to insertion of PA catheter sheath
7. Pass balloon over guidewire through sheath. Must be inserted to at least the level of the manufacturer's mark (usually double line) to ensure that entire balloon has emerged from sheath
8. Balloon should be positioned so that the tip is about 1 cm distal to the origin of the left subclavian artery. If fluroscopy is not available during insertion the distance from the angle of Louis down to the umbilicus and then to the

femoral artery insertion site should be measured to approximate the distance the balloon should be advanced and the position should be checked on CXR

9. Remove wire. Return of blood via central lumen confirms that the tip is not subintimal and has not caused a dissection.
10. Flush central lumen with heparin saline and connect to transducer to monitor intra-aortic pressure
11. Monitor Doppler ankle pressures and compare with preinsertion value

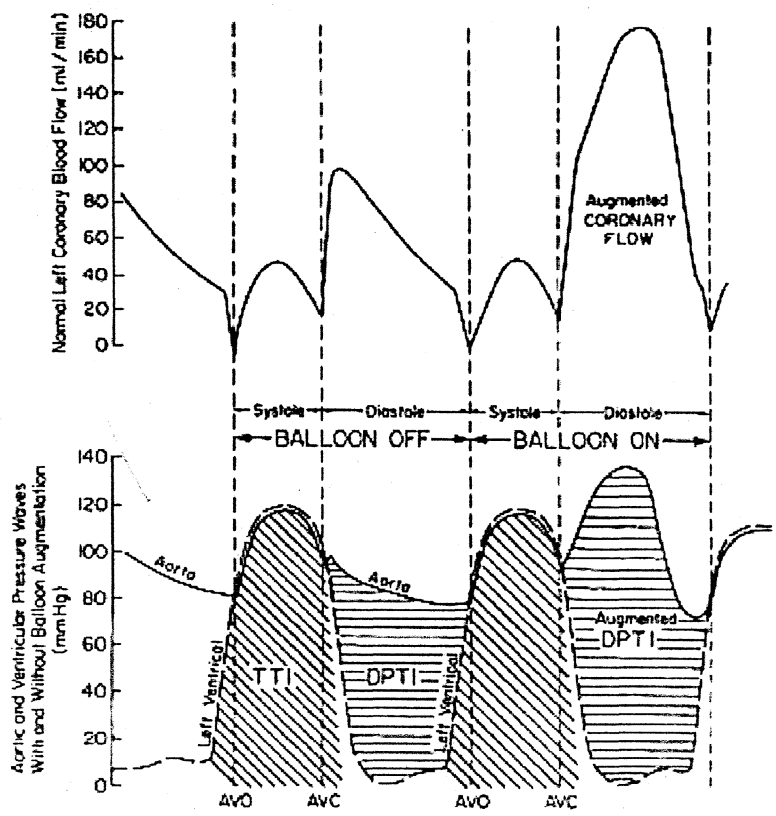
The IABP is connected through the catheter to a computer console at the base of the patient's bed, and patients will often feel its rhythmic pulsing as it controls the inflation and deflation of the IABP. The console includes a number of electrical devices, including:

- Controls for adjusting the IABP's inflation and deflation
- Emergency backup power supply
- Monitor for recording electrocardiogram (EKG) and pressure wave data

If the catheter is inserted through the femoral artery in the groin, patients are often asked to keep that leg straight. A critical care team monitors the patient's progress and should be notified immediately if the patient feels any sudden discomfort or wetness where the catheter was inserted.

Balloon pump timing

- Using central aortic pressure waveform and ECG identify dicrotic notch (aortic valve closure)
- Determine time delay between R wave and aortic valve opening and closure and enter these values into the pump
- Turn on pump and compare assisted and unassisted waveforms to determine whether timing is optimal
- If inflation is too early or deflation too late the balloon waveform is superimposed to varying degrees over the LV systolic component of the central aortic pressure waveform (ie inflation starts when the aortic valve is still open). This results in an increase in afterload which may result in premature valve closure and increase LV work. Also, ventricular emptying is incomplete, stroke volume decreased, cardiac output decreased and myocardial oxygen demand increased. In addition, can increase shunting in patients with a septal defect
- If inflation is too late or deflation is too early diastolic augmentation is suboptimal
- Balloon inflation can be triggered by R wave, arterial waveform or pacing spike. Latter is a potentially lethal mode as loss of capture may result in balloon inflation during systole as the pump will continue to follow the pacing rate rather than the ventricular contraction rate



a) Inflation of the balloon during diastole (augmentation of the aortic diastolic pressure) increases coronary blood flow (DPTI).

b) Deflation of the balloon occurs just prior to the onset of systole and reduces impedance to left ventricular ejection (TTI). This results in less myocardial work, decreased myocardial oxygen consumption and increased cardiac output.

Benefits and risks with IABP

The IABP offers the following potential benefits:

- Improved circulation
- Lower heart rate and decreased workload of the heart
- Improved efficiency of the heart's pumping
- Increased supply of oxygen to heart tissues and decreased demand for it

- Less pressure resistance in the aorta when the heart pumps (during the systole)
- More pressure in the aorta when the heart is relaxing, thus increasing blood flow to the heart muscle (during the diastole)

The risk of complications has dropped as the IABP has evolved over the last 40 years. However, there are some complications that are still reported in some patients, which include:

- Damage to the aorta or femoral artery
- Heavy bleeding (hemorrhaging), occasionally from the site of insertion
- Infection
- Lack of oxygen-rich blood to either a limb (limb ischemia) or an organ (visceral ischemia), due to narrowed blood vessels
- Tearing or bursting of the balloon, releasing gas into the bloodstream with potentially dangerous results

In addition, not all patients are suited for IABP. Patients who may not be recommended for IABP use include:

- Those with conditions of the aorta, including aortic regurgitation, abdominal aortic aneurysm or aortic dissection
- Those with uncontrolled bleeding
- Those with severe peripheral vascular disease

Longevity of IABP use

The intraaortic balloon pump (IABP) usually remains implanted for a short period of time, such as the following situations:

- Before, during or after open-heart surgery or balloon angioplasty (in high-risk patients)
- During acute attacks of angina
- In emergency situations (e.g., heart attack, heart failure, very low blood pressure due to cardiogenic shock)
- Throughout waiting periods as patients prepare for either a heart transplant or a mechanical heart

In most cases, an IABP is needed for a few days. Sometimes it is used for a week, and in rare cases for a full month.

Variants and alternatives to IABPs

Another early variation of aortic counterpulsation used cuffs that were wrapped around the lower legs and timed to alternately empty and fill with water in the same rhythm as the patient's heartbeat. Today, the technique of enhanced external counterpulsation (EECP) uses cuffs filled with air instead of water. This is more comfortable for the patient and easier for the physician. The cuffs fill sequentially, filling with air all around the lower leg, rather than suddenly filling all at once.

There are no reported risks of complication. However, EECP is not recommended in patients with certain factors, including:

- Uncontrolled arrhythmia
- Uncontrolled heart failure
- Bleeding or clotting problems
- Blood pressure higher than 180/110

Moreover, EECP is a scheduled, non-emergency procedure rather than an emergency procedure, for which an intraaortic balloon pump (IABP) may still prove to be more effective.

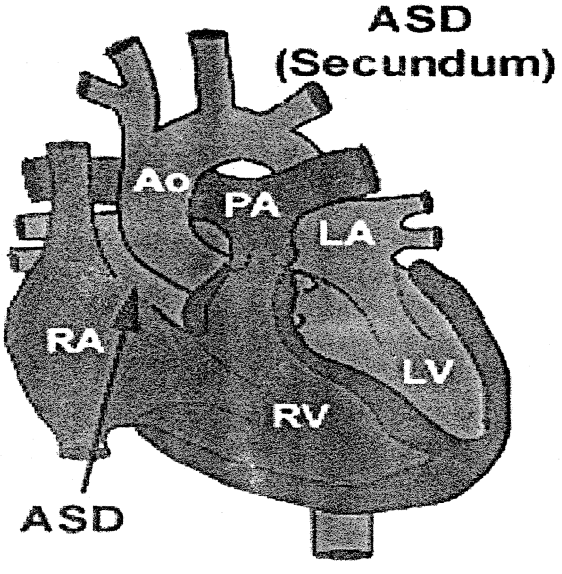
CONGENITAL HEART DISEASES

Congenital heart defects are classified into two classes – cyanotic and acyanotic defects.

ACYANOTIC DEFECTS

ATRIAL SEPTAL DEFECT (ASD)

ASD is defined as the communication across the atrial septum. ASD is an acyanotic defect with left to right shunting. The atrial septum has two parts; the septum primum which is a thin mobile tissue followed by the secondum septum, which is thick and muscular in nature. There are several types of ASDs depending on their anatomical positioning.



There are several types of ASD depending on their anatomical positioning.

Sinus Venosus ASD

In this category ASD occurs at the entrance to the superior vene cava or inferior vene cava, close to the opening of the right atrium. In approximately 90% of cases this type of ASD is associated with abnormal pulmonary venous connection.

Ostium Secundum ASD

It occurs from a deficiency in the fossa ovalis and is located in the center of atrial septum. If the septum primum is normal the defect may be classified as patent foramen ovale (PFO).

Ostium Primum ASD

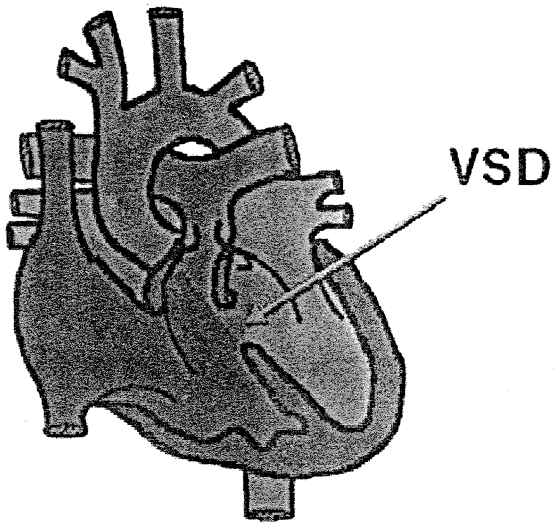
It is located in the bottom end of the septum, adjacent to atrioventricular valves.

Coronary Sinus ASD

It is a rare kind of ASD in which unroofing of the coronary sinus causes right to left shunting of blood from the coronary sinus into the left atrium. This results in cyanosis.

VENTRICULAR SEPTAL DEFECT (VSD)

VSD is the communication between the left and right ventricles. A VSD is an acyanotic lesion with left to right shunting. The ventricular septum has two parts: a small membranous septum and large muscular septum consisting of muscular inlet and outlet portions.



The classification of VSD is as follows,

Perimembranous VSD

It is located within the membranous septum extending towards the muscular septum. It is commonly associated with interrupted aortic arch if the septum is posteriorly malaligned or with tetrology of Fallot.

Muscular VSD

It is located in the muscular or trabecular portion of the ventricular septum. It is also known as trabecular VSD. The rim is totally made up of muscle. It can be single or multiple.

Inlet VSD

In this classification, all or part of the atrial ventricular septum is absent. It lies beneath the atrioventricular valves and is associated with complete atrioventricular septal defect.

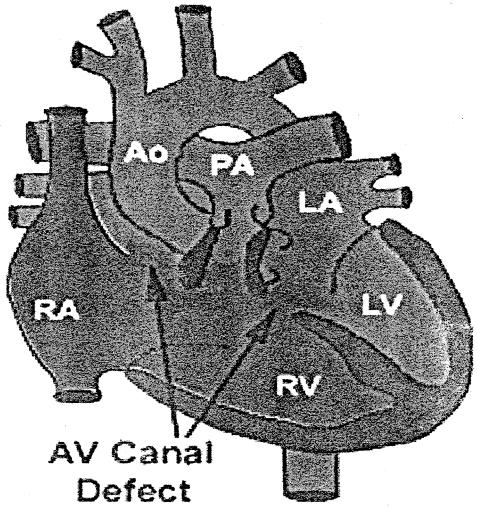
Outlet VSD

These kinds of VSDs are located within the infundibular septum and limited by the pulmonary valve.

Doubly Committed VSD

This is a VSD close to both semilunar valves. If the VSD size is large there will be a large left to right shunt, the high systemic pressure will be directed to pulmonary artery resulting in pulmonary artery hypertension. Pulmonary resistance will rise protecting the lungs from being flooded with blood, subsequently irreversible changes occur in the pulmonary artery vasculature. If surgical correction is not done before this stage, the pulmonary vascular resistance will become higher than the systemic resistance. The shunt becomes reversed and the patient will be cyanotic. This is termed as Eisenmenger's complex.

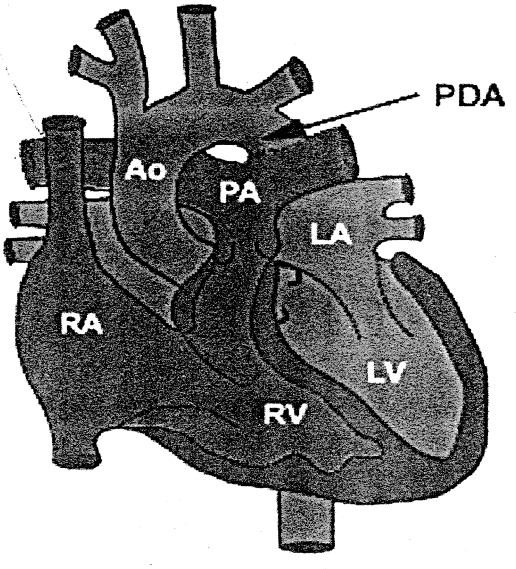
ATRIOVENTRICULAR SEPTAL DEFECT(AVSD)



This is usually a large defect involving both the atrial (ASD) and the ventricular (VSD) septums, which allows blood to pass freely between the two ventricles and the atriums. The valve apparatus at the junction between atriums and ventricles is "shared" - there being effectively only one valve instead of the normal two. Blood flow and pressure in the lung circulation is substantially increased. Early surgical repair is needed in most cases (in the first four to six months).

PATENT DUCTUS ARTERIOSUS (PDA)

The ductus Arteriosus is the communication between the aorta and left pulmonary artery. In the fetus blood flows from the right ventricle into the pulmonary artery and from the pulmonary artery through the ductus into the aorta. Shortly after the birth the walls of the ductus contracts and degenerative changes in the walls of the ductus converts it to a cord of fibrous connective tissue called ligamentum arteriosum. If the ductus does not close, a left to right shunt from aorta to the pulmonary artery is established called patent ductus Arteriosus.



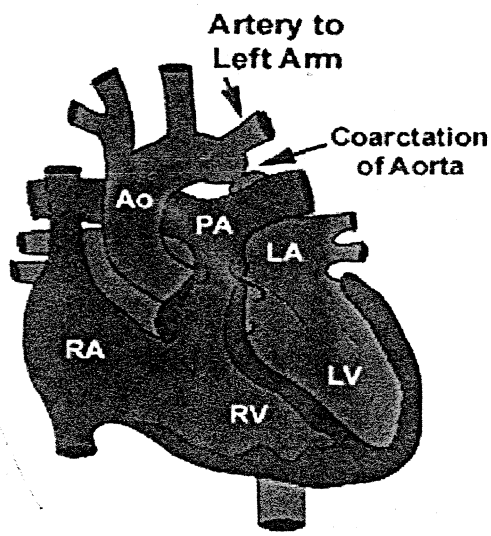
PARTIAL ANOMALOUS PULMONARY VENOUS DRAINAGE (PAPVD)

In this defect one of the three pulmonary veins drain into the right atrium, coronary sinus or vene cave. It is often associated wit sinus venosus type of ASD.

OBSTRUCTIVE DEFECTS

COARCTATION OF AORTA (CoAo)

Coarctation of aorta is a narrowing or constriction of the aorta which occurs in the thoracic and very occasionally in the abdominal aorta. It becomes significant when there is a pressure gradient of more than 20mmHg across the aortic narrowing. Their classification is as follows,



Preductal CoAo

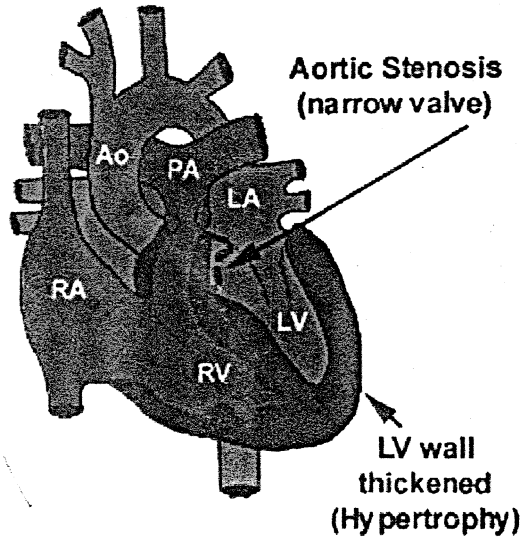
The coarctation is found proximal to the ductus Arteriosus. In the Preductal form, fetal blood flow is unchanged in the presence of a PDA. Blood from the right ventricle enters the ductus arteriosus and discharges into lower aorta. The left ventricular blood flows to the upper extremity of the body. Once the ductus arteriosus closes and pulmonary vascular resistance falls it will lead to heart failure.

Postductal CoAo

The postductal coarctation, the obstruction has implications for the fetus as the constriction reduses blood flow from the ductus arteriosus.There for,the contents of the right ventricle,which normally flow through the ductus arteriosus.,are impeded. The left ventricle still perfuses the upper body and head via the ascending aorta . As this obstruction affects the fetus, a variable colleteral circulation develops, from the proximal to the distal aorta.

AORTIC STENOSIS (AS)

This is a condition of obstruction of left ventricular outflow tract. The stenosis may occur at a valvular, subvalvular and supra valvular level. Obstructions of blood flow out of the left ventricle will increase left ventricular pressure leading to left ventricular hypertrophy.



Valvular AS

The aortic valve is thickened with fused cusps. It is usually bicuspid, instead of normal three cusps. A unicusp can occur in the severely stenotic valve. This is normally found in neonate and is termed as critical aortic stenosis.

Subvalvular AS

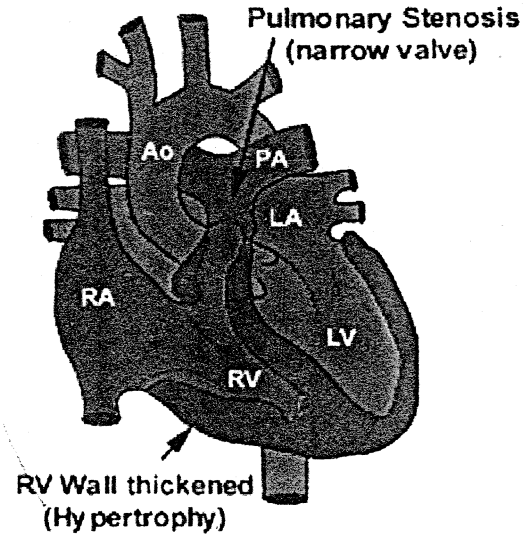
The stenosis is normally attributed to a fibrous membrane beneath the aortic valve being attached to the mitral valve and intraventricular septum.

Supravalvular AS

This stenosis is related to either a fibrous constriction above the aortic valve. The left ventricle is often hypertrophied with reduced blood flow in diastole. In a mild stenosis the pressure gradient between the aorta and left ventricle is less than 30mmHg in systole. In moderate stenosis the pressure gradient is less than 50mmHg and in sever stenosis it is more than 80mmHg and up to maximum of 200mmHg.

PULMONARY STENOSIS (PS)

Refers to any lesion that obstructs the flow of blood from right ventricle to the pulmonary artery. In this case right ventricular pressure increases to maintain normal cardiac output, which results in the hypertrophy of the right ventricle and its failure. According to the level of the obstruction pulmonary stenosis is classified as,



Valvular PS

The narrowing is at the level of the pulmonary valve.

Subvalvular PS

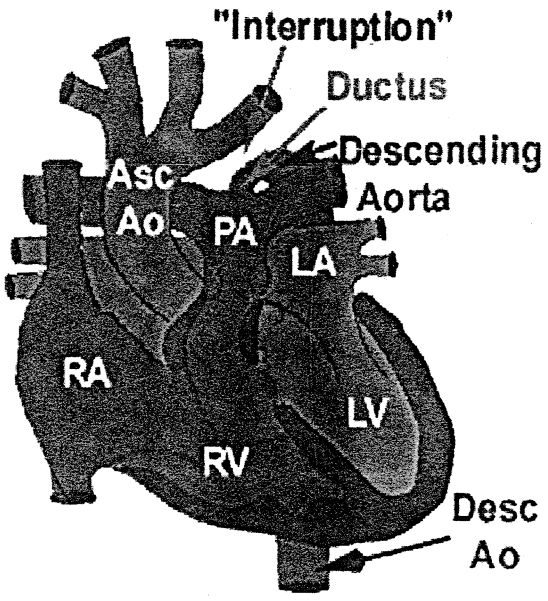
Here the narrowing is below the level of the valve, in the infundibular region of the right ventricle. It is commonly associated with TOF.

Supravalvular PS

The narrowing is above the level of the valve, in the main pulmonary artery itself.In mild stenosis the gradient between right ventricle and pulmonary artery will be less than 30 mmHg, in moderate PS gradient is less than 50mmHg and in severe PS gradient can be as high as 200mmHg.

INTERRUPTION OF THE AORTIC ARCH (IAA)

Part of the Aorta is absent and this leads to severe obstruction to blood flow to the lower part of the body. The ductus allows flow to the lower circulation before birth but as it closes in the newborn period, blood pressure in the lower circulation becomes inadequate and major symptoms develop. Most affected infants develop severe symptoms (difficulty breathing and impaired kidney function) in the first week of life and need urgent surgery. Most affected infants also have a large VSD. Sometimes other defects may be present.



CYANOTIC HEART DISEASES

TETROLOGY OF FALLOT (TOF)

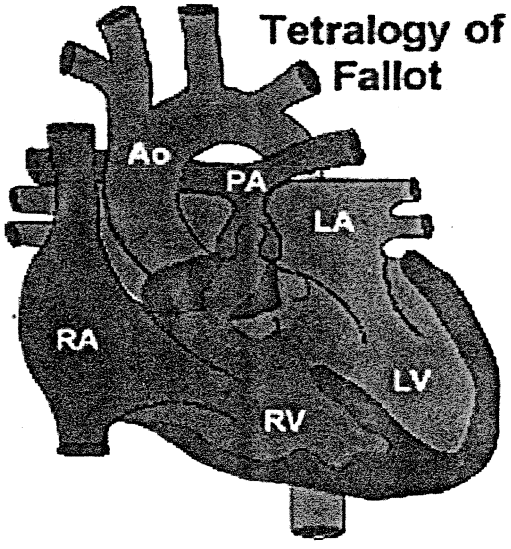
It is a cyanotic lesion with right to left shunting characterized by four anomalies:

. ventricular septal defect

. Right ventricular outflow tract obstruction (RVOTO)

. Overriding aorta

. Right ventricular hypertrophy



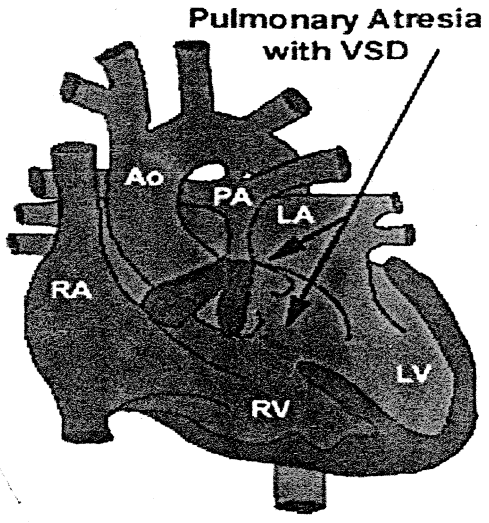
Here the pressures on both the ventricles are the same. The venous blood enters the right ventricle via the right atrium. Most of the blood flows into the area of least resistance, that is the left ventricle and to the aorta. Subsequently there is little flow of deoxygenated blood into the lungs, and reduced flow from the lungs, giving rise to a hypoplastic left ventricle.

Cyanotic Spell

This is representative of a sudden severe reduction in pulmonary blood flow. It is a unique feature of TOF. It produces hypercyanosis, accompanied by crying. The baby becomes pale, breathless, sweaty, and unconscious with hypoxia, acidosis, hypercarpnea, myocardial hypoperfusion and cerebral hypoperfusion leading to brain damage. The baby can die during a spell, it necessitates emergency surgery.

PULMONARY ATRESIA WITH VSD (PA VSD)

Pulmonary atresia with ventricular septal defect may be thought of as the extreme form of tetrology of Fallot, with severe pulmonary stenosis and no communication between the right ventricle and pulmonary artery. This is a cyanotic lesion with right to left shunting across the VSD. The positioning of atresia may vary. In two thirds of cases pulmonary blood flow is dependent upon the presence of a PDA and in the rest major aortopulmonary collateral arteries (MAPCAs) will develop.



The classification is dependent on the size and origin of the pulmonary blood supply.

. Type I has a well developed pulmonary arteries. The pulmonary blood flow is reliant upon the presence of PDA.

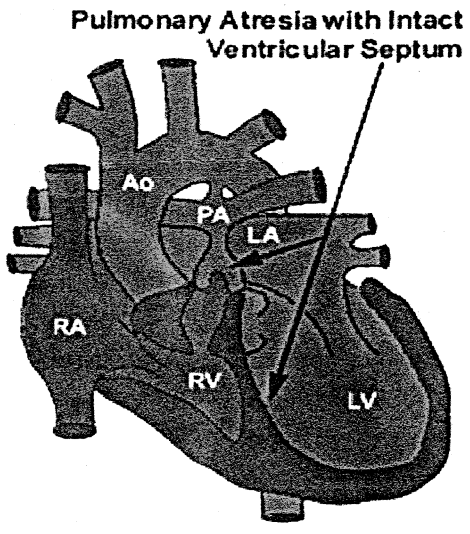
. Type II has well developed pulmonary arteries, but the main pulmonary artery is absent. Pulmonary blood flow is reliant upon the presence of PDA.

. Type III has hypoplastic pulmonary arteries, which can be disconnected from each other. The main pulmonary artery is absent and the pulmonary blood supply is via MAPCAs and small PDA.

. Type IV has no pulmonary arteries, no MPA, no PDA. Total pulmonary blood floe relies on MAPCAs.

PULMONARY ATRESIA WITH INTACT VENTRICULAR SEPTUM

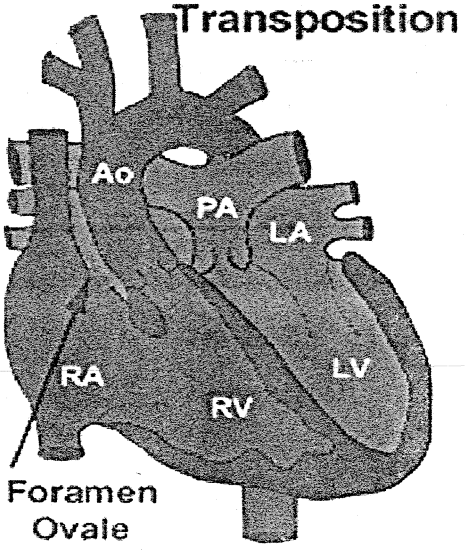
This can be described as hypoplastic right heart syndrome in which pulmonary valve is atritic with varying degrees of right ventricle and tricuspid valve hypoplasia. The pulmonary arteries are usually of normal size due to the blood supply from PDA. It is a cyanotic defect with left to right shunt at atrial level.



Blood enters the right atrium from the vene cave and either flows through the tricuspid valve into the hypoplastic right ventricle or passes directly across the intra atrial communication. If the blood passes into the right ventricle, it will not be able to the MPA due to the atritic valve, so flows back into the atrium due to regurgitation. This causes hypertrophy of the right atrium with an increase in right atrial pressure causing reopening of foramen ovale, blood flows into the left atrium. This mixing of venous blood with pulmonary blood flow causes cyanosis.

TRANSPOSITION OF GREAT ARTERIES

The aorta arises from the right ventrcile anteriorly and the pulmonary artery from the left ventricle posteriorly. It can be described as atrioventricular concordance with ventriculoarterial discordance. It is commonly associated with other anomalies such as ASD, VSD, DORV or PS.



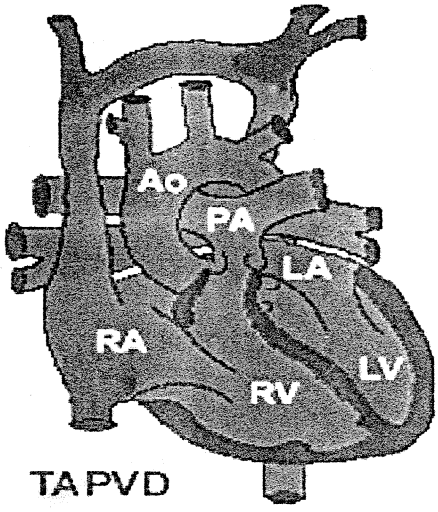
The aorta is dextrotransposed (D-transposition)), lying anteriorly to the pulmonary artery, causes two separate circulations. Venous blood enters the right atrium from the vene cave, passes through the tricuspid valve into the right ventricle. From here the deoxygenated blood enters the aorta into the systemic circulation. Oxygenated blood form the lungs enters the left atrium, passes to the left ventricle and to the pulmonary artery thus reentering the lungs. The baby will die if there is no shunt exists.

TOTAL ANOMALOUS PULMONARY VENOUS CONNECTION (TAPVC)

TAPVC is described as the failure of pulmonary veins to drain into the left atrium. Instead they connect directly or indirectly to the right atrium by way of the systemic veins. The classification of TAPVC are related to where the pulmonary veins connect to the systemic venous circulation. There are four types:

Supracardiac

The pulmonary veins connect to the systemic veins by a vertical vein which then joins the SVC and empties into the right atrium.



TAPVD

Cardiac

The pulmonary veins connect directly to the coronary sinus or the right atrium.

Infra Cardiac

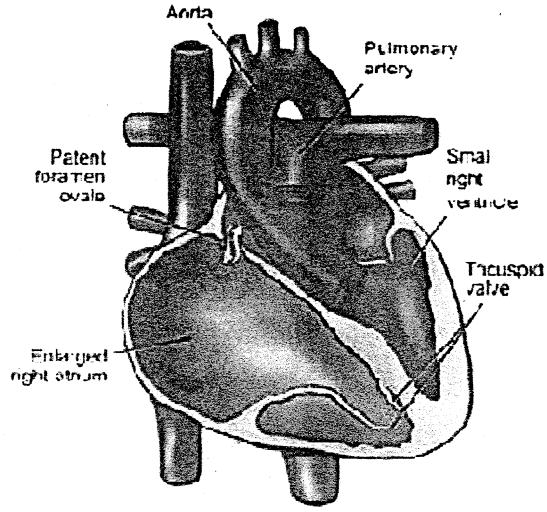
The pulmonary veins joins to form a vertical vein which empties into the ductus venosus, portal vein, hepatic vein or IVC, then emptying into the right atrium. Obstruction occurs more frequently with this type.

Mixed

Here at least one pulmonary vein is connected to a different systemic vein from all other remaining anomalous veins.

EBSTEIN'S ANOMALY OF THE TRICUSPID VALVE

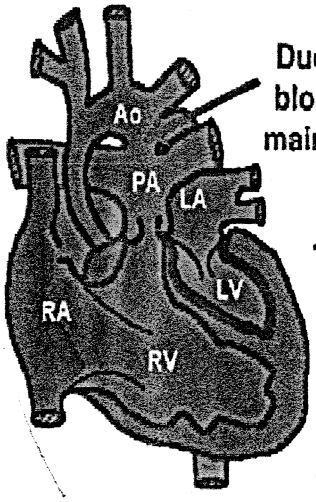
Ebstein's anomaly is defined as malformed and displaced tricuspid valve. The posterior and septal leaflets of the tricuspid valve are displaced downwards and into the right ventricle. This creates an atrialised right ventricle, where by part of the right ventricle is integrated into the right atrium. This causes in a small hypoplastic right ventricle, which cannot eject its blood efficiently causing reduction in pulmonary blood flow and varying degrees of cyanosis. It usually associated with an ASD or PDA.



HYPOPLASTIC LEFT HEART SYNDROME(HLHS)

The left side of the heart is very poorly formed and cannot support the main circulation (round the body). The left ventricle and aorta are abnormally small (hypoplastic). This is amongst the most severe forms of heart defect.

Affected infants usually become severely symptomatic soon after birth (as the ductus closes). This is one of the most serious cardiac malformations and leads to death in the newborn period in almost all affected babies, unless surgery or Heart Transplantation can be offered.



DEFECTS THAT MAY BE CYANOTIC OR ACYANOTIC

DOUBLE OUTLET RIGHT VENTRICLE (DORV)

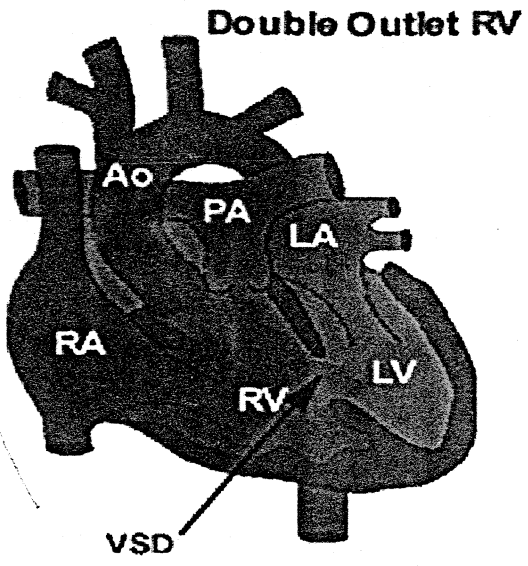
It is a defect in which the aorta and pulmonary artery originate from the right ventricle. A ventricular septal defect is always present with DORV and the position of the VSD classifies the type of DORV. They are:

Subpulmonary VSD without pulmonary stenosis

Subaortic VSD without pulmonary stenosis

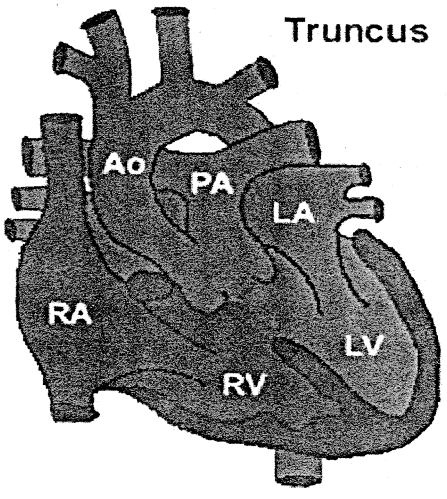
Subaortic VSD with pulmonary stenosis

Doubly committed VSD



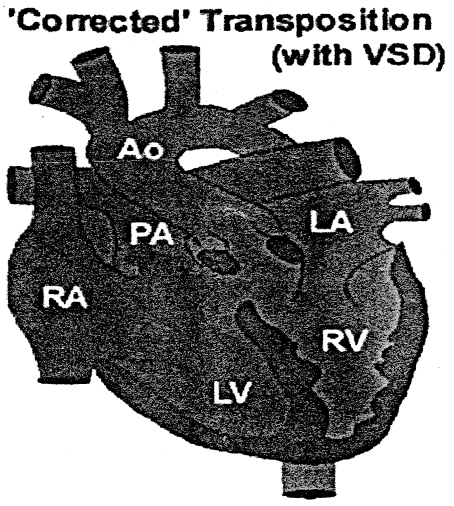
TRUNCUS ARTERIOSUS

Truncus arteriosus is a defect in which the truncus arteriosus fails to divide in the developing fetus. Therefore, the two great vessels are united to form a common great vessel providing both systemic and pulmonary circulations.

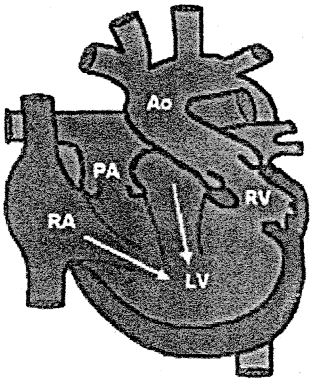


CORRECTED TRANSPOSITION OF THE GREAT ARTERIES (CTGA)

In CTGA the aorta lies anteriorly and the pulmonary lies posteriorly. This is similar to TGA, except that the aorta is on the left of the pulmonary artery instead of the right and left ventricle transposed.



SINGLE VENTRICLE (SV)



Both atriums connect to LV (arrows)

This is one of a group of very complex defects where both Atriums connect to the same ventricle and/or one ventricle is absent or very tiny. In most cases the infant develops symptoms in the early weeks of life - either with 'Cyanosis' (Blueness of the skin) or with breathlessness and failure to gain weight normally. Many affected patients have associated defects in the heart or main arteries, including such problems as Pulmonary Stenosis (PS), Pulmonary Atresia, other valve abnormalities or Coarctation of the Aorta, etc. Such problems may cause sever obstruction to blood flow to the lungs or into the main circulation and may require urgent surgery. The RV (usually very small) may be on the opposite side of the heart to normal. This is similar in some respects to the situation with "Corrected Transposition.

CARDIAC PHARMACOLOGY

In cardiac pharmacology there are many different types of drugs that treat heart disease. A wide variety of cardiac drugs used to reduce or prevent the pain of an angina lower BP, help regulate the heart rhythm, help to prevent the formation of blood cloats, help the pumping action of the heart, lower blood fats and help prevent the future attacks.

Different types of drugs are following below:-

Antiarrhythmic drugs

Propafenone

Prolongation of the PR interval due to blocking at the AV –node, intermittent or paroxysmal atrial fibrillation. The drug has an additional mild β –blocking effects. Indications are PSVT, tachyarrhythmias which include flutter or fibrillation and paroxysmal re-entrend tachycardias. Contra indications are increase digoxin plasma level without digitalis toxicity, severe pulmonary obstructive disease, impaired /hepatic function. Dose 150mg(tab).

Atenolol

It is a cardio selective β-block .It reduces resting and exercise include heart rate as also myocardial contractivity. Also reduces the systolic and diastolic blood pressure. The reduces in BP and heart rate results in reduced myocardial work and oxygen requirement. Anginal attacks are reduced in intensity and frequency, exercise tolerance is improved. Indication are it reduces the incidence of VF in patient with acute MI, increase VF. The contra indications are sinus bradycardia, untreated cardiac failure, cardiogenic shock and bronchial asthma. Interaction is in combination with verapamil it may precipitate heart in patient with impaired LV function. Dosage; in arrhythmias 50-100mg/day, hypertension 25-50mg once daily.

Adenosine

This drugs lows conduction time through the AV-node, can interrupt the reentry pathways through the AV-node, and can restore normal sinus rhythm in patient with paroxysmal supraventricular tachycardia, including PSVT associated with Wolf-Parkinson-White syndrome, ECG changes suggestive of rhythm disturbance. Side effects are hypersensitivity, 2^{nd} or 3^{rd} degree AV block and Sick Sins Syndrome, Palpitation, asthma, Hypotension, bradycardia, headache, nausea and metabolic taste. Dose 3mg/ml.

Propanalol

A non selective β-blocker heart rate and cardiac output are reduced. BP reduced as is myocardial work load. In anginal patients exercise tolerance is increased and attacks reduced. Reduces rennin activity. Indications are hypertrophic subaortic stenosis and hypertrophic cardiomyopathy. Side effects are sinus bradycardia, heart block greater than 1stdegree,untreated cardiac failure, hyperglycemia, dizziness, anxiety, allergic reactions, stomach discomfort, vomiting.

Quinidine

A class 1-a antiarrhythmic drug reduces rate of depolarization phase of cardiac action potential, slows depolarization prolongs refractory period. Exerts vagal blockage, controls atrial fibrillation, ventricular and supraventricular tachycardias. Indication is atrial and ventricular tachyarrhythmias. Side effects are hypersensitivity, heart block, prolonged QT interval, digitalis intoxication, ventricular arrhythmias, muscle weakness, nausea, headache, dizziness, and blurred vision. Interactions are increases plasma digitalis concentration. Enhance effect of antihypertensive, vasodilators, myocardial depressants, oral anticoagulant and non-depolarizing muscle relaxants. Dose: Entopic beats usually 100-300mg orally daily. Children 25-100 mg/day according to age.

Verapamil

It inhibits entry of calcium ions in to arterial smooth muscle as well as the myocytes and conducting tissue. These action lead to reversal and prevention of coronary artery spasm, reduction of after load through peripheral vasodilatation and reduction in ventricular rate in patient with chronic atrial flutter or fibrillation and reduction in the occurrence of paroxysmal SVT. It reduces BP relives angina and control arrhythmias. Contraindications are cardiogenic shock,2nd or 3rd degree AV block severe brachycardia, Sik Sinus Syndrome, acute phase of MI, Hypo tension, less frequently allergic reaction, bradycardia, pulmonary edema, peripheral edema, skin rash, fatigue, head ache. Dose 40 mg tab.

Procainamide

It is an amide derivative of the local anesthetic procaine which was formed to have antiarrhythmic activity. It is an alternative drug to quinidine. For IV use it is safer than quinidine and has a more rapid action. Indications are premature ventricular contraction and tachycardia, atrial fibrillation, paroxysmal atrial tachycardia, cardiac arrhythmias associated with anaesthesia and surgery. Side effects are AV block, nausea, vomiting, weakness, mental confusion, hypotension. Interactions are inhibits sulfonomides enhance the effects of some β-blockers, effects enhance by amiodarone ,alcohol increases elimination. Dose 250mg (tab), 100mg/ml (inj).

Lignocaine

It is a local anesthetic which stabilizes the neuronal membrane and inhibits the ion movement which are necessary for conduction of impulses. In the heart it reduces phase four depolarization, decreases automatically, duration of action potential and effective refractory period is reduced. Contra indications are bradycardia, hypotension, dizziness, drowsiness, muscle twitching, hypertension. Interactions are cimetidine increases plasma concentration and toxicity, propanolol reduces clearance by approximately 40% and absorption reduces by adrenaline. Dose adults 50-100mg IV under ECG monitoring, children bolus dose of 1 mg/Kg followed by infusion at 30mcg/Kg/min.

β-Blockers

It block the action of hormones such as adrenaline that make the heart beat faster and more vigorously. These are very effective in preventing attack of angina but they work too slowly to be use full in relieving an attack of angina. They effective in lowering raised blood pressure and in reducing risk of a further heart attack in people who have already had one . Some β-blocker help control abnormal heart rhythm. The disadvantage is a tendency to constrict the air passages. For this reason they should avoid the people who suffer asthma or wheezing .It reduces the force of the heart beat. side effects include fatigue, tiredness, cold hand and feet. Other less side effects include nausea, diarhhoea, skin rashes.eg: atenolol and metaprolol.

ACE Inhibitors

These drugs are very effective in treating and preventing heart failure. They are recommended for most patient with hear failure and are also used to lower high BP. Also after the heart attack, many patient will benefit from an ACE inhibitor. Angiotensine Converting Enzyme is a chemical which has a powerful narrowing effect on the blood vessels. It is produced as a result of the action of the angiotensin converting enzyme. ACE inhibitors can reduces the activity of this enzyme. ACE inhibitors can causes skin rashes and very occasionally a major allergic reaction. Some people develop a persistent, dry, irritating cough.

Cardiac Glyosides

This increases the contractility, cardiac rate decreases partly medicated through the vagal and extra vagal action. Refractory period is increased in atrium and ventricle by the therapeutic dose decrease in AV node and purkinje fibers. Drugs are digoxin, Amrionone which is a stong positive inotropic effect even in digitalis failure, decreased peripheral resistence, under investigation of congestive heart failure. Drug interaction is quinidine causes significant increase of dioxin level in the plasma, increase the toxicity.

Diurectics

This increases the output of water and salt in urine. They are particular valuable in heart failure, in which there is too much water and salt in the body. It is also effective in lowering the BP. There are main three types; thiazide, loop diurectics and potassium sparing diurectics. In treating high BP diuretic may be used alone or with β-blockers, calcium channel blockers, ACE inhibitor or other drugs. If you are taking a diuretic, you should avoid salty foods. Mannitol is osmotic diuretics reduce edema whether specific organized intravenous administration. Furosemide(lasix) inhibits re-sbsorption of Na in the ascending loop of Henle causing the additional excretion of K.

Calcium channel blockers

It is used to control high BP after cardiac transplant. You need a regular inflow of calcium for the muscle cells in the heart to work normally. It reduces the amount of calcium entering the muscle cells of the arteries (including coronary artery) and cause them to relax and widen. They have the effect of increasing the blood supply to the heart and reducing the work, the heart has to do to pump blood around to the body. Nifedipine increase the heart rate at rest, diltiazem and verapamil tend to reduce the natural increase in heart rate. Which usually happen when you exercise. The combination of the nifidipine and β-blockers may effective, where there is a danger that the heart rate may become too slow if β-blockers are combined with diltiazem or verapamil. Also used to reduce the high BP. Side effects include dizziness, fatigue slow heart rate faintness, swelling of ankles, feeling sick and vomiting.

Potassium channel activators

There are very new types of drug given to relieve angina. They have a similar effect to nitrate as they relax the walls of the coronary arteries and so improve blood flow. Unwanted effect of this is a headache, dizziness.

Acute Hypotension

Epinephrine (Adrenaline)

Catecholamine action is exerted both α and β receptors mainly β causes mixed dilatation and constriction, the arterioles in the skin mucus are constricted, arterioles in skeletal muscle are dilated, result is decrease in the total peripheral resistance. The ion tropic and force of contraction are increases. Then increase cardiac output. Uses are cardiac arrest or stokes Adams syndrome, acute asthmatic attack.

Phenylephrine Hydrochloride

It acts as a vasoconstriction, decrease the heart rate and stroke output increase, also constricts venous capacitance system. This is used to the stage of acute hypotension state.

Mephentermine sulphate

Helpful in controlling arrhythmias by decreasing the AV conduction time, cause slight increase in venous and peripheral resistance.

Nitrates

Nitrates relax the muscle in the walls of the veins and arteries (including the coronary arteries) and make them wider. They are very useful in angina and in preventing predictable attack. Glyceryl trinitrate tab put under the tongue relieves angina quickly. They may also cause flushing, dizziness or even fainting. It can give in an aerosol spray, to spray under the tongue which action is much quick than the tablet. Oral nitrates are isosorbide; mononitrate and dinitrate are drug in preventing angina. Unwanted effects are headache, flushing, dizziness and fainting can happen with nitrates but most common with glyceryl trinitrate. Dose 50 mg in dissolved in 250 ml of 5% dextrose; given intravenously at the rate of 0.5-5 micrograms/kg body weight/min.

Dopamine

It is adrenergic sympathetic agents that increase myocardial contractility through β adrenergic receptor stimulation. It is a positive inotropic and vasoconstrictive agent. Although increase cardiac output. If inotropic therapy is needed in acute pulmonary edema associated with hypotension. Also vasodilatory action on the renal and mesenteric circulation. Both dopamine and dobutamine can include atria and ventricular concern in-patient with myocardial ischemia or acute infarction. Dose 2-500mg/kg/min IV.

Dobutamine

This is an inotropic agent use for blood pressure support and increase HR, myocardial ischemia, increase risk of arrythmias. Contraindications are hypertrophic heart diseases and severe aortic stenosis. Dose 2-40 mg/kg/ min IV.

Digoxin

Digoxin is used in atrail fibrillation. This arrhythmia makes the heart rate quickly and irregularly; this may lead eventually to heart failure. Digoxin slows the heart rate but does not restore its irregularity. It may cause nausea; less frequently can cause vomiting,

palpitation and fainting. Side effects are atrial and ventricular arrhythmias, heart blocks. Dose 0.0625-0.3125 mg/day.

Aspirin

It is also effective in preventing blood from clotting. I reduce the risk of dying after heart attack and of having a further heart attack. Also useful for patient with angina and used to prevent blood clotting in the vein grafts in CABG, for treating patient with coronary artery disease. Side effects are bleeding, ulcer etc. Contraindications are bleeding tendency. Dose 80-325 mg.

Atropine

This is an anticholinergic treatment of bradycardia and heart block. Side effects are dry mouth, confusion. Contraindications are myocardial ischaemia, asthma, ileus and glaucoma. Dose: 0.6 to 2 mg (max) IV.

Abciximab (Reopro)

A new glycoprotein II b/ III a receptor antagonist. Use for complicated PTCA/PTCS procedures, also studied for use in unstable angina and acute myocardial infraction. Side effects are allergic reaction and bleeding tendency. Dose bolus+infusion.

Captopril

Used for the treatment of hypotension, heart failure and post MI remodeling. It is an angiotensine converting enzyme inhibitor. Side effects are impair renal function, anemia. Contraindications are hypercalemia, renal artery stenosis, outflow obstruction. Dose: 25-150 mg.

Carvedilol

Alpha and β-blocker with vasodilator activity. Used for tretment of congestive heart failure. Start at low dose and titrate up slowly in 2 weekly time. New studies showed that it reduses mortality in class II-IV heart failure patients. Side effects are asthmatic attack, bradycardia, worsening of heart failure.

Adrenaline

Its indication is drug of choice in anaphylactic shock. Acts on α and adrenergic receptor sites. It inhibits the intestinal contraction and enhances contraction of voluntary muscle. Dose: 0.1-0.2 mg through IV.

Sodium Bicarbonate

Indication is metabolic acidosis. It decreases concentration of hydrogen ion and increase pH. Dose= Body weight in Kg* deficit of HC.03*0.3.

Calcium sulphate

This is given when there is hypocalcaemia and cardiac emergencies. Action is it stimulates myocardial excitability and increases contractility. Dose 0.5 to 1ml of 10% solution with cardiac monitoring through IV.

Diazepam

It is a pre operative medication to reduce anxiety and tension. It acts on the thalamus and hypothalamus to induce calming effect. Dose 0.3 mg/kg body weight/ day through oral route.

Hydralazine

This is a direct vasodilator. Treated as malignant hypertension, heart failure. Side effects are edema and hypotension. Contraindications are myocardial ischaemia, severe mitral valve disease. Dose 10-400 mg/day IV.

DEPARTMENTAL EQUIPMENTS

INTEGRIS H 5000 F (PHILIPS)

The floor-mounted monoplane Integris H 5000 F is the dedicated system of cardiac procedures. It brings excellent digital imaging performance to cardiac suite. The large diameter of the polydiagnost G stand and patient sensing of bodyguard allow high rotation and angulations speeds of up 25 degree per second. New fully integrated, all digital imaging chain is based on CCD technology optimized for complex cardiovascular applications. It provides high speed, high resolution imaging with true 1024 matrix.

Specifications

Environmental requirements

Ambient temperature	: 10 – 35 °C
Humidity	: 20 – 80 %

Mains

440 V +/- 10 %, 50 & 60 Hz, 3 Phase

X-ray generator

Microprocessor controlled 100 kW high frequency convertor generator

Voltage range	: 40 –150 kV
Maximum current	: 1000 mA at 100 kV, 800 mA at 125 kV

X-ray tube

MRC 0508

Power	: 0.5/0.8, 45/85 kW
Anode heat storage capacity	: 2400 kHU anode
Continuous heat dissipation	: 3500 W

TV chain

XTV 16, CCD camera with proprietary digital out put

Examination light

Light intensity	: 30000 Lux
Focusable light field size	: 14 –25 cm
Lamp type	: Halogen 22.8/24 V 50 W

Automatic wedge filter

Two semi-transparent wedge-shaped filters automatically or manually adjusted.

Digital acquisition

Direct link with XTV 16 image chain
Programs for digital dyanamic acquisition
Frame speeds (frames/second) at 512 * 512 image resolution.

Frame speed (frames/second)

50 Hz	: 12.5, 25, 30
60 Hz	: 15, 30, 60
II sizes	: 9 inch, 7 inch, 5 inch

BV 300 (PHILIPS)

BV 300 is a mobile diagnostic X-ray image acquisition and viewing system

* 9" or 12" triple-mode field of view
* Fixed or rotating anode X-ray tube technology
* 200, 2000, 100000 image capacity.

A unique combination of CCD camera and anamorphic lens that delivers high quality image details.

COROSKOP_ DIGITRON (SEIMENS)

Coroskop-digitron is biplane cath system with DSA and cine angiography support. Equipped with Seimens Polvdors 100 – high frequency X-ray generator with microprocessor control.

ADVANTX DLX_LCV (GE)

A single plane system equipped with high rated grid controlled tube with air-cooling system. Focal spots available are 1.1, 0.6, 0.3, 0.30b, 0.20b and 0.15b. Maximum KVP and mA are 120 and 1000 respectively. System is provided with cine attachment. Other facilities are last image Hold (LIH), auto injection of contrast, edge enhancement, noise filtering, roadmap etc, image processing includes different subtraction modes, pixel shifts, gray scale inversion, re-masking, image filter, image integration, integrated mask, maximum opacification, temporal scale, landscape, shutter, stenosis analysis, cardiac analysis and annotation. Archive media: Digital Audio Tape (DAT) & V.T.R

DICOM

Introduction

The **Digital Imaging and Communications in Medicine** (DICOM) standard is a detailed specification that describes a means of formatting and exchanging images and associated information. The standard applies to the operation of the interface, which is used to transfer data in and out of an imaging device. DICOM relies on computer industry standard network connections, and devices that address the communication and storage of digital images from diagnostic modalities such as CT, MRI, PET, Nuclear Medicine, Ultrasound, x-ray, CR, Digitized film, video capture and HIS/RIS information. It also supports the connection of networked printers, such as laser images (cameras). DICOM is the result of an alliance potential users of the standard (members of the American college of Cardiology and American college of Radiology) with the campanies that manufacture medical equipment (members of the National Electrical Manifactures Association – NEMA). The DICOM effort really began in 1984, and it was orginaly called the ACR/NEMA standard. Now, DICOM has been ebraced by other world wide standards organizations outside of cardiology and radiology. For example, DICOM has been adopted by the Committee European de Normalization (CEN TC 251) and the Japanese Industry Association for Radiation apparatus (JIRA).

The DICOM standard has now been implemented in an increasing number of medical products from various vendors. The rapid adoption of DICOM by the medical imaging industry is opening new opportunities for health care organizations to increase the quality and cost effectiveness of patient care.

THE NAME – DICOM

Version 3.0 of the ACC/ACR-NEMA standard is called the digital imaging and communication in Medicine (DICOM) standard to reflect the contribution of other international organization as well as the standard ability to expand beyond support of cardiology and radiology images, to include images from endoscopy, surgery and pathology.

DICOM REVIEW STATION

In general, A DICOM Review station is to the cine-projector as the CD-R disc is to the interchange aspect of cine film. In order describe the concept of a DICOM Review station, let as make an analogy to both 35mm cine film and the cine film projector. There are five basic tasks that the cardiologist is faced with when using cine film. This tasks include: acquisition- the X-ray cardiac angiographer performs an X-ray exam(diagnostic or interventional) on a patient and acquires the exam to 35mm cine film. Film developing: the cine film must be processed so that it can be used for" review". Review: - the cardiologist must load the film into a 35mm cine projector. The cine projector provides the basic image display functionalities required to diagnostically assess the clinical

severity of most morphological effects. The physician can then view the entire exam by controlling the cine projector (e.g.:- fast forward, reverse, pause...). The cine projector does enable the cardiologist to perform basic image processing such as zoom, roam or brightness adjustment. Interchange- the cardiologist can send the film across town, across the state, across the country or around the world for review on any 35mm projector. This exchange process enable physician to communicate with other people (i.e. Physicians, patients etc). Archive: - after the patient has been treated/ discharges; the cine film (exam can then we archived longer term storage (e.g.:-a ware house).

So what is the analogy between (cine projector and cine film) and (DICOM Review stations/ CD-R)? .The basic DICOM Review station provides the exact functionality of a 35mm projector. A primary difference, however, is that a DICOM Review station can (although not mandatory) provide random access to the digital acquisition sequences (runs) corresponding to a specific exam. With cine projector, however, the image data is stored sequentially on the film real and the user must fast forward to position film to a specific acquisition run. In addition, the benefits of "digital" technology enable the DICOM Review station to provide the same image processing capabilities as those found on high-end digital X-ray imaging systems. For e.g.:-the image sequences can be transferred from the CD-R interchange into the DICOM Review station. The DICOM Review station can provide edge enhancement filtering or quantitative analysis on the digital images. It is not difficult to see that the DICOM standard will enable applications that improve clinical productivity, image quality, and clinical care. So, is DICOM Review station like a cine projector? Yes.. But it can be much more! Loading images into a DICOM review station when a user receives a DICOM CD, the following sequence of events are required to view the images.

- Load DICOM CD into the DICOM Review station drive.
- Select the sequences to be viewed

Import the sequences from the DICOM CD into the review station: the images are stored in a loss less compressed format on the DICOM CD, there fore they must be compressed by the DICOM Review station. This can be done by specialized hardware or by soft ware. Import performance is limited by both the CD-R drive performance as well as the decompression approach used by the review station.

CD-R (compact disc – recordable)

The AdHoc group selected the CD-R as the standard medium for exchange of cardiac digital angiographic examinations. The standardization process has resulted in a sufficiently detailed definition of how images are stored on a compact disc to permit the manufactures of imaging equipment to read and write and digitally – recorded study, regardless of the source of the original image data.

KEY FACTS ABOUT THIS MEDIUM INCLUDE

- In 1996, the standard speed CD-R drive is 4X (600KB/second) performance. Most CD-R drives are back wards compatible and can provide read and write rates at 1X (150KB/second) and 2X (300KB/second) speeds.
- Continuiting investment in research and developmenting CD technology will guarantee substantial improvementing performance in coming years, resulting in increased storage capacity and data transfer rates.
- CD-R is a "write once" non-erasable medium, physically robust (after writing), unaffected by electromagnetic fields, and relatively in sensitive to dust and scratching. Digital recording on CD-R is virtually error free (error rates is less than once false bit in 1000,000,000,000 bits).
- CD-R storage capacity is 650MB which permits recording of up to 4800 frames per disc at a 512X512X8 bit resolution utilizing 2:1 loss less compression. As evidence from the above statistics, this capacity is sufficient to record at least 99% of all cardiovascular examinations on a single disc. Standard CD-R media are sold and referencing minutes of storaging capacity as this technology is derivative of the audio CD-ROM disc. A 650 CD-R is a 74minute disc.

The Ad Hoc group recognizes that current CD-R technology lags sufficient speed to enable direct viewing of angiogram at a 30 frames/second. The committee did not consider lack of real time retrieval to represent a great disadvantage for an interchange medium. The CD-R disc permits random access to any exam sequence. Thus, the relatively low retrieval rate is partially compensated by rapid access to any sequence of interest. Further more, review stations with CD-ROM drives can be equipped with image buffers allowing perfecting of the entire exam followed by real time review as required. CD media and drive technologies are still in a state of evolution towards higher performance. Traditionally, write and read data rates have been reference to a single speed performance of 150KB/second. Currently, the most technically advanced drives have 6 to 8times this performance (0.9MB/Second to 1.2MB/second respectively). An 8X drive, for e.g.: enables images to be stored and retrieved at a rate of approximately 10 frames/second, therefore requiring approximately 3 minutes and 42 seconds for an average study containing 22 is a 74 minute disc.

LOG BOOK

ELECTROCARDIOGRAM PERFORMED

During my posting in ECG lab, I personally took around 10,000 ECGs. Some of the patient datas are given below,

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER
1	Rajan C	56/M	9102912
2	Mathew Eapen U	67/M	188404
3	Gopalakrishnan K	48/M	216288
4	Ambi N	36/F	242348
5	Kavitha S	18/F	242316
6	Vinod V	25/M	241259
7	Abil B	20/M	197865
8	Faizal M	30/M	242353
9	Rosy T	38/F	242357
10	Nikhil G	9/M	242394
11	Victoria J	22/F	242477
12	Shanthi V	56/F	241707
13	Kadeeja A	78/F	242502
14	Chamy G	67/M	242506
15	Hamza M	45/M	236715
16	B/O Beena	1/M	237988
17	Shajida D	12/F	242537
18	Prema S	22/F	9107056
19	Joel B Babu	15/M	242567
20	Raveendran Nair K	60/M	198311

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER
21	Murugeswari S	24/F	240726
22	Mohanan Nair A	56/M	242156
23	Joy G	23/M	221590
24	Salim S	33/M	242416
25	Rev Fr Thomas	73/M	9409645
26	Kamalam V	40/F	236338
27	Salambi S	32/F	198207
28	Manickan A	56/M	242475
29	Chandralekha S	30/F	215530
30	B/O Sreeja	1/F	244730
31	B/O Sony Rose	1/M	242514
32	Ajith B	11/M	221253
33	Bappu S	67/M	232821
34	Ebin S	32/M	229961
35	Manju S	21/F	225680
36	Asma Beevi M	54/F	242047
37	Lalithamma V	67/F	242437
38	B/O Nisha	1/M	232435
39	Dileep R	8/M	231531
40	B/O Shyni	1/F	242830
41	Aiswarya S	18/F	242805
42	Sushoban N (DR)	56/M	221271
43	Abdul Kareem I	67/M	236814
44	B/O Devipriya	1/F	242072
45	Baby K	34/F	242989

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER
46	Lalitha Kumari A	45/F	227516
47	Ouseph O	77/M	239094
48	Syamala D	45/F	192827
49	Basheer A	34/M	243346
50	Saleena N	23/F	242336
51	Sandhya A	19/F	218970
52	Bindhu V	21/F	243045
53	B/O Sreeja	1/M	242473
54	Nabeesa F	25/F	242965
55	Meenakshi A	45/F	229909
56	Chandy D	55/M	240817
57	Chinnamma C	70/F	182152
58	Sabitha C	28/F	9806188
59	Salini L	22/F	9105612
60	B/O Surabhi	1/M	243067
61	Jaya S	36/F	243430
62	Selin John V	27/F	243304
63	Aperna B Raj	20/F	230852
64	Anu G	21/F	9704653
65	Sandhya V	26/F	227727
66	Shobha Albert S	45/F	9501152
67	Ramachandran Nair M	56/M	242546
68	Sarala V	67/F	244857
69	Jijo R	19/M	244757
70	Ismail B	45/M	244799

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER
71	B/O Seema	1/F	245058
72	Gomathi S	50/F	243692
73	Yoonis A	25/M	245111
74	Elsi C	37/F	245072
75	Aravindan G	37/M	219291
76	Sujatha S	33/F	217166
77	Indira S	40/F	237954
78	B/O Sury	1/M	9707057
79	Anooja I	16/F	245057
80	Kavya J	14/F	241301
81	B/O Vanithamani	1/M	243609
82	Remya S	26/F	243936
83	Soman C	45/M	193386
84	B/O Sini	1/F	242683
85	Retnamma S	67/F	240612
86	Gopi C	48/M	219431
87	Usha P	38/F	243980
88	Ramlath S	42/F	8907347
89	James A	30/M	224945
90	Santha C	51/F	243852
91	Aneefa E	23/M	244681
92	B/O Suma	1/M	244669
93	Jeena L	23/F	9604430
94	Radhakrishnan K (DR)	58/M	9707098
95	Leela V	56/F	235127

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER
96	Akhila J	26/F	244716
97	Muraleedharan S (DR)	45/M	9302998
98	Basila Sherin N	27/F	243857
99	Cherian R	67/M	242906
100	Mallika S	32/F	239541

TMT PERFORMED

During my TMT posting I had assisted around 1500 TMT cases. Some of them are given below,

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER
1	Kuriakose P	57/M	256707
2	Nazeer S	48/M	256665
3	Parvathy Dasan K	38/M	245362
4	Remani L	29/F	257642
5	Gomathy M	53/F	258539
6	Devasahayam T	64/M	257807
7	Mohammed Kunju M	46/M	258299
8	Leela C	38/F	258971
9	Thankappan D	38/M	9707306
10	Sarojini L	69/F	255808
11	Savithri S	42/F	256987
12	Moideen Bava J	55/M	254328
13	Jerome I	46/F	9605747
14	Alexander Samuel S	57/M	9707167
15	Ambika Pillai H	40/F	223843
16	Elizebeth John S	56/F	249951
17	Sasidharan Nair R	62/M	251476
18	Mariammal S	43/F	211018
19	Chinnamma T	67/F	221954
20	Lekha D	22/F	250388
21	Vasantha T	49/F	251152
22	Haridasan N	33/M	251601
23	Kunjamma Chacko A	78/F	9803141

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER
24	Wilson S	45/M	254383
25	Philip P	49/M	191154
26	Soman C	45/M	193386
27	Leelakumari P	53/F	249995
28	Anilkumar B	38/M	247124
29	Mohanachandran V	59/M	233784
30	Khadeeja K	41/F	220660
31	Thresiamma Joseph T	61/F	249167
32	Ameenel Beevi H	56/F	250438
33	Padmakshy S	53/F	225552
34	Radha K	50/F	242861
35	Sulochana V	48/F	250179
36	Subaida J	59/F	251082
37	Lowerence T	38/M	238222
38	Leela B	42/F	229762
39	Sivankutty A	38/M	250022
40	Oommen T	65/M	250704
41	Babu C	47/M	250301
42	Sarojam S	60/F	248743
43	Abdul Aziz M	50/M	250681
44	James T	45/M	248008
45	Sathish Chandran C	60/M	227534
46	Shanavas P	30/M	245201
47	Vincent M	46/M	247128
48	Sreerangan T	62/M	249081
49	Kunjuraman Nair K	66/M	249016
50	Reghu D	50/M	247881

HOLTER PERFORMED

During my Holter posting I have assisted around 750 Holter connections. I have also assisted the Holter analyzing. Some of the cases are given below,

SL NO	PATEINT NAME	AGE/SEX	DIAGNOSIS	HOSPITAL NUMBER
1	Savithri K	38/F	SSS, Syncope	8903967
2	Surendran S	36/M	Palpitation	187101
3	Sivani K	51/F	Palpitation	232090
4	Robin C	16/M	ASD, Syncope	243880
5	Bennet O	57/F	Palpitation	232727
6	Elizebeth M	24/F	Palpitation	243204
7	Shiny S	21/F	SSS, Syncope	9905380
8	Balakrishnan A	69/M	HCM, Syncope	245019
9	Nivya Chandran K	8/F	PO OP Sennings	9801392
10	Sreenivasan Achari S	63/F	Dyspnea	243142
11	Mari Infant Thomas	46/F	CHB	9807538
12	Blessan Kurian I	13/M	DCM	244098
13	Joseph K	70/M	PPI	9007029
14	Lalitha J	61/F	AS	218710
15	Jayakumar K	34/M	RVOT VT	250829
16	Narayanan Swamy S	50/M	CAD, Syncope	236892
17	Santha B	40/F	PPI	239372
18	Luis P	60/M	SSS, Syncope	242580
19	Soman N	51/M	Palpitation	9508932
20	Duraipandi M	27/M	PPI	248128
21	Mohandas R	67/M	MS, AS	9104053
22	Manju R	59/M	PPI	256991

SL NO	PATEINT NAME	AGE/SEX	DIAGNOSIS	HOSPITAL NUMBER
23	Ramanan M	45/M	CHB	257677
24	Stanley V	54/M	CAD, Syncope	256082
25	Yonesan M	53/M	PPI	256630

ECHO ASSISTED

During my posting in Echo lab, I have assisted the doctors in doing the TTE and TEE. Some of the patient datas are given below,

SL NO	PATEINT NAME	AGE/SEX	DIAGNOSIS	HOSPITAL NUMBER
1	Manu M	16/M	PAVCD	246321
2	Kailas Vishnu S	1/M	TAPVC	256688
3	Jayakumari J	35/F	MS, AS	9603353
4	Priyadharsini M	3/F	SAVSD	246331
5	B/O Sali	1/M	VSD, PS	256674
6	Jithu N	5/M	ASD	243165
7	Pushpakala S	30/F	MR	254760
8	Thankappan S	50/M	MVP, MR	256714
9	Chendunny A	72/M	AS	256706
10	Meharunisa S	50/F	DCM	256715
11	Ayyappan S	16/M	TOF	256710
12	Kumari K	53/F	AS, AR	256500
13	Anwar Sadiq B	16/M	VSD, AS	254003
14	Christeena S	4/F	TOF	239396
15	Nazeer Ahammed S	28/M	PO OP VSD	9008067
16	Jaison J	3/M	dTGA, PAH	252892
17	Sajeena Navas V	23/M	ASD	250175
18	Sonu M	14/M	VSD, MS, MR	239090
19	Sujith Xavier S	7/M	VSD	222402
20	Essaki Raja S	17/M	ASD, PAH	250351
21	Alima Beegam G	24/F	MS, MR	252372
22	Selva Bharathy T	15/M	RHD, MS, MR	253338
23	Indirakumari V	52/F	AS, AR	201842

SL NO	PATEINT NAME	AGE/SEX	DIAGNOSIS	HOSPITAL NUMBER
24	Santhi Mohini V	24/F	MS, AS, MR, AR	200182
25	Sulajakumari N	36/F	MS, AS	241527
26	Eswari T	17/F	PDA	256890
27	Drishya C	5/F	PO OP BDG	184171
28	Chandrasekharan S	35/M	CAD, RWMA	241744
29	Ajina S	7/F	PO OP PDA	250644
30	Jose Varkey H	45/M	HCM	257022
31	Irshad Ahammed V	13/M	PDA, PAH	257832
32	Gokul Babu J	7/M	dTGA	258058
33	Anandavalli D	27/F	ALCAPA	256397
34	Devishri S	3/F	HOCM	258774
35	Jayasudha M	24/F	HOCM	258951
36	Bhagya Lekshmi L	6/F	PS	259120
37	Geetha R	23/F	TOF	259495
38	Nisha S	18/F	TS, TR	259508
39	Anseer S	35/M	PDA	231457
40	Liju G	27/M	VSD	243854
41	Harilal N	30/M	DCM	259597
42	Sahal C	7/M	DORV	259655
43	Merin Joseph T	2/F	PA, VSD	259676
44	Jamsheera S	20/F	DIRV	9004680
45	Jishnu D	12/M	LVOTO	189277
46	Sundari N	51/F	RHD, MS	259766
47	Renjith P	14/M	SVASD	259807
48	Aswathi C	19/F	TOF	208324
49	Shreyamol S	23/F	SPVSD	257119
50	Ashna B	15/F	PDA	259853

CARDIAC CATHETERISATION

I have personally assisted the cardiologist in doing more than 150 coronary angiograms and 75 cardiac catheterization (adult and pediatric) some of the cases are given below and is done more than 3500 coronary angiograms and 1000 cardiac catheterization with senior technicians. I have also assisted PPI, PTMC, BAV, BPV, ASD DEVICES, PDA DEVICES, PDA COILS, BAS, VSD DEVICES & EPS some of them are given below.

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER	DIAGNOSIS	PROCEDURE
1	Shajahan A	42/M	248395	AWMI	CAG
2	Vimala A	43/F	250999	AWMI	CAG
3	Soudamini A	56/F	215016	IPWMI	CAG
4	Archana G	10/F	9602738	VSD	CATH
5	Philip P	54/M	9601058	AWMI	PTCA
6	Haridasan M	53/M	248202	IWMI	CAG
7	Subramanian M	43/M	250846	IPWMI	PTCA
8	Neelakanta Pillai M	63/M	253231	ILMI	PTCA
9	Abraham K	52/M	247963	NQMI	CAG
10	Saritha A	25/F	8607833	PS	CATH
11	Athira P	10/F	188089	PDA	PDAC
12	Azeez A	56/M	252899	NSTEMI	CAG
13	Vishnu R	3/M	250468	PDA	PDAC
14	Ashida P	4/F	234174	PDA	PDAC
15	Somasundaram V	58/M	241914	NSTEMI	CAG

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER	DIAGNOSIS	PROCEDURE
16	Girija Devi B	54/F	249360	IWMI	CAG
17	Vineeth T	17/M	244879	PDA	CATH
18	Jenisha M	6/F	250490	PDA	PDAD
19	Faziludeen H	39/M	248783	PWMI	CAG
20	Anu N	7/F	204758	PDA	PDAD
21	Kochammini C	78/F	250983	IWMI	CAG
22	Soman A	56/M	250137	IWMI	CAG
23	Ajina S	1/F	250649	PDA	PDAC
24	Radhika R	6/F	257851	PDA	PDAC
25	Rajan P	49/M	249793	ASMI	CAG
26	Murugan L	40/M	249042	AWMI	CAG
27	George V	60/M	9008728	IPWMI	CAG
28	Govindan P	64/M	253167	IWMI	PTCA
29	Varkey K	61/M	253241	AWMI	PTCA
30	Peethambaran V	51/M	199649	IWMI	PTCA
31	Shihabudeen M	84/M	249991	NQMI	CAG
32	Anirudh J	1/M	247074	PDA	PDAD
33	Rajendran C	53/M	256784	AWMI	PTCA
34	Shahul Hameed J	58/M	248166	IWMI	CAG
35	Joykutty C	48/M	249045	AWMI	CAG
36	Dasan G	52/m	259896	LWMI	CAG
37	Alyarkunju T	54/M	250172	NSTEMI	CAG
38	Brijith T	45/M	8805821	PWMI	CAG
39	Abubakkar M	55/M	257006	AWMI	PTCA
40	Viswanatha Shenoy H	59/M	249253	ASMI	CAG

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER	DIAGNOSIS	PROCEDURE
41	Damodharan V	69/M	192766	AWMI	CAG
42	Jubairya M	58/F	248247	IWMI	CAG
43	Thomas K	40/M	237255	MS	PTMC
44	Unnikrishnan V	41/M	249045	IWMI	CAG
45	Celis T	37/F	215561	IWMI	CAG
46	Shajahan M	40/M	249406	MS	PTMC
47	Ibrahim Kutty H	39/M	9006099	AWMI	CAG
48	Thambi E	50/M	247761	AWMI	CAG
49	Assis Rawther M	53/M	9705859	IWMI	CAG
50	Narayanan Kutty S	69/M	250497	MS	PTMC
51	Lalithammal H	62/M	250547	ASMI	CAG
52	Sasi P	54/M	248849	IWMI	CAG
53	Sreedevi amma R	52/F	9000911	IWMI	CAG
54	Ganeshkumar C	16/M	249248	AS	BAV
55	Manuel Nesan I	54/M	249054	AWMI	CAG
56	Easwari G nair	17/F	249998	PS	BPV
57	Prakash M	26/M	9002431	TS	CATH
58	Unnithan N	70/M	194598	ASMI	CAG
59	Samraj P	76/M	194598	IPWMI	CAG
60	Joise Karunakaran S	56/M	242549	AWMI	CAG
61	Harikumar J	38/M	253495	IPLWMI	PTCA
62	Girija S	42/F	233761	AWMI	PTCA
63	Panchali V	46/F	232074	PWMI	CAG
64	Bhaskaran K	58/M	251090	RVMI	PTCA
65	Philip M	25/M	253504	AS	BAV

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER	DIAGNOSIS	PROCEDURE
66	Vasantha V	44/M	259474	AWMI	CAG
67	Raveendranath C	75/M	8900505	ASMI	CAG
68	Sugathan K	23/M	251226	PS	BPV
69	Krishnan Nair S	70/M	251072	AWMI	CAG
70	Santhamma C	61/M	258987	ILWMI	CAG
71	Bhuvanendran N	48/M	259239	IPWMI	CAG
72	Sulfikar S	37/M	253040	AWMI	PTCA
73	Sasidharan P	38/M	253219	ILWMI	PTCA
74	Nazer Kunju A	50/M	257870	AWMI	CAG
75	Sahadevan P	44/M	253314	AWMI	PTCA
76	Preetha R	27/F	254829	HOCM	CATH
77	Fasiludeen M	50/M	244703	TOF	CATH
78	Kalavani I	2/F	255622	PDA	PDAD
79	Molly Chandy J	71/M	9708660	AWMI	PTCA
80	Sundari N	28/M	254638	AS	BAV
81	Asokan N	59/M	253640	NQMI	PTCA
82	Kassim M	15/M	245441	PDA	PDAD
83	Sathy C	51/M	251598	ASMI	PTCA
84	Swapna Mathew M	26/F	253511	ASD	ASDD
85	Chacko M	49/M	257452	VSD	CATH
86	Salini M	20/F	253196	ASD	ASDD
87	Asharaf H	40/M	254452	IPWMI	CAG
88	Vijayan Achary S	65/M	257988	IWMI	CAG
89	Salma P	21/F	251797	ASD	ASDD
90	Reji V	39/M	255579	ASD	CATH

SL NO	PATEINT NAME	AGE/SEX	HOSPITAL NUMBER	DIAGNOSIS	PROCEDURE
91	Samuel Y	68/m	252041	AWMI	PTCA
92	Ebrahim A	25/M	253822	PS	BPV
93	Thilakarajan K	53/M	250737	IPWMI	PTCA
94	Abdul Rawaf K	15/M	227918	SAVSD	CATH
95	Pushpavathi S	60/M	254023	AWMI	PTCA
96	Vasanthakumari K	49/M	253587	AWMI	PTCA
97	Kumari S	38/F	256487	ASD	ASDD
98	Kunhu Muhammed M	40/M	258603	ASD	ASDD
99	Rama Iyer V	53/M	9808402	IWMI	CAG
100	Santha Haridasan K	45/F	9608805	SVASD	CATH

PPI PERFORMED

SL NO	PATEINT NAME	AGE/SEX	DIAGNOSIS/MODE		HOSPITAL NUMBER
1	Kamalabai C	48/F	Pause	VVI	259482
2	Mariamma Beevi N	58/F	CHB	VDD	9702854
3	Abdul Kareem M	63/M	CHB	VVI	258873
4	Sujatha R	30/F	CHB	VVI	204083
5	Thankachy P	70/F	AV Block	DDD	256939
6	Basheer M	64/M	VT	ICD	8806008
7	Pushpavally P	41/F	SSS	VVI	8906648
8	Suseela K	46/F	Pause	AAI	246750
9	Leelamma Raghavan O	71/F	CHB	VVI	259051
10	Sivadasan K	35/F	SSS	VVI	252175
11	Krishnan Namboodiri S	64/M	SSS	DDD	252175
12	Madhavan Pillai G	71/F	CHB	VVI	257079
13	Gouri B	14/F	SSS	VVI	253887
14	Karthyayani M	70/F	SSS	AAI	245407
15	Chandra Babu N	52/M	DCM, VT	COMPO	232746
16	Sajan Domanic P	13/M	CHB	VVI	255552
17	Rajagopalan C	67/M	CHB	DDD	254039
18	Cletus S	61/M	CHB	VVI	257033
19	Sainaba Beevi V	56/F	SSS	VDD	9006214
20	Leelamma Chacko O	48/F	AF	VVI	255700
21	Simimol N	18/F	CHB	VDD	255165
22	Prabhu S	17/M	SV, CHB	VVI	8908030
23	Geevarghese M	47/M	DCM	BVP	250731
24	Balachandran Nair S	40/M	Pause	DDD	246360
25	Suresh G	24/M	SSS	VVI	245781

I have assisted the following number of noninvasive and invasive cases

ECG	10,000
TMT	1,500
HOLTER	750
CAG	3,500
CATH	1,500
PTCA	750
PTMC	500
BAV	20
BPV	20
PDAC	150
PDAD	75
ASDD	100
BAS	20
VSDD	2
CoA STENTING	1
PPI	100
EPS	100