SREE CHITRA TIRUNAL INSTITUTE FOR MEDICAL SCIENCES &TECHNOLOGY

(An Institute of National Importance under Govt.of India)

Thiruvananthapuram-695 011, Kerala, India

PG DIPLOMA IN CARDIAC LABORATORY TECHNOLOGY

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Work Book submitted in partial fulfillment of the requirements for the award of the PG Diploma in Cardiac Laboratory Technology

WORK BOOK

by

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CERTIFICATE

This is to certify that this is the bonafide work book of the laboratory work undertaken by ***Ms. ASWATHY SWAMY A*** *in partial fulfillment of the requirements for the award of PG Diploma in Cardiac Laboratory Technology.*

Place : Thiruvananthapuram

Date:

Prof:Dr.Ajith kumar V K

Head of the department

Dept.Of Cardiology

SCTIMST

PREFACE

This work book, I have done as a part of my training in the Department of Cardiology for PG Diploma in Cardiac Laboratory Technology (PG DCLT) course includes brief details of the equipment used in the Dept. And working involved with the equipments, the routine protocols and the procedures followed in our different labs, number of cases which I have individually done in ECG, ECHO, TMT, HOLTER labs and the cases which I have assisted in Cardiac Cath Lab. PG DCLT is a two years full time residential programme conducted in the Department of Cardiology for Students from Bachelor of Physics community to excel and learn the newer techniques in the field of cardiology. Selection is done by a national level entrance examination. At present Institute offers 3 seats. The students are posted in the Department of Cardiology equipped with all modern medical facilities. During the two year programme, first year posting is in noninvasive cardiology and the second year is in invasive cardiology. The course schedule contains theory classes, practical training, seminar presentations and projects. Diploma is awarded after successful completion of 2 year term based on a written examination with viva-voce and internal assessment.

ABOUT OUR INSTITUTION

Sree Chitra Tirunal Institute for Medical Sciences and Technology (SCTIMST) is an Institute of National Importance under the Department of Science and Technology, Govt. of India. The joint culture of medicine and technology pioneered by its founders more than three decades ago, has come of age and gained unprecedented acceptance in India. The institute has the status of a university and offers excellent research and training facilities. It has three wings: a tertiary referral super specialty hospital, a biomedical technology wing and the Achutha Menon Centre for Health Science Studies.

The Institute focuses on high quality, advanced treatment of cardiac and neurological disorders, indigenous development of technologies for biomedical devices and materials and public health training and research. The institute offers advanced treatment using modern technologies in several specialized areas such as interventional radiology, cardiac electrophysiology, deep brain stimulation for movement disorders, epilepsy surgery, pediatric cardiac surgery, base of skull and vascular surgeries, to name a few. The institute has excellent facilities and teams of professionals dedicated to the development of innovative biomedical devices and products, evaluation of medical devices to global specifications, training in novel medical specialties and research in medical and public health areas of social relevance. The Institute is a Technical Research Centre for Biomedical devices .

DECLARATION

I hereby declare that I have actually performed all the procedures listed in this work book during my course and this work book has not been submitted in any form, whole or part for awarding another degree or diploma at any other university or institution of higher education.

Place: Thiruvananthapuram ASWATHY SWAMY A

Date:

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First and foremost I would like to thank to Head of the Department of Cardiology **Prof: Dr. Ajith kumar V K** and all other faculty members of the department who guided through the different cases of studies and encouraged and helped me in all aspect of my training.

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Aswathy swamy A

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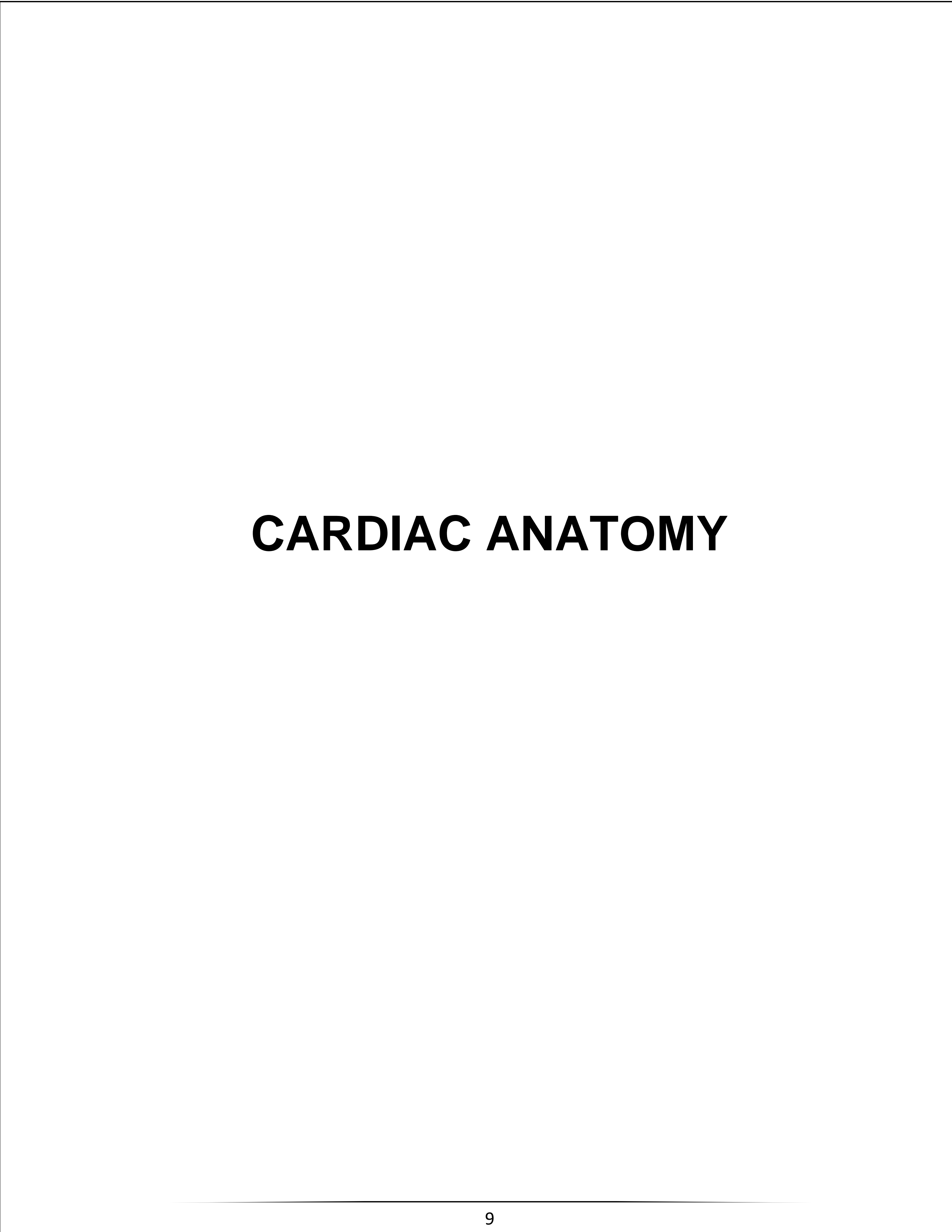
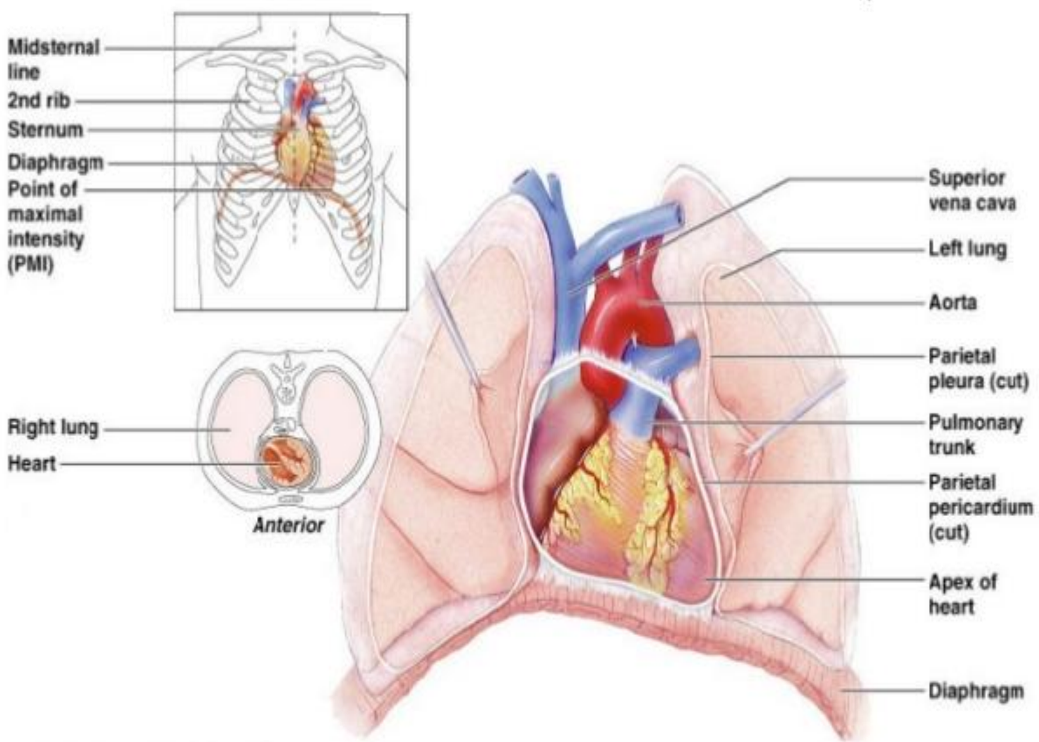


CARDIAC ANATOMY

THE HEART

Andreas Vesalius (1514-1564) is considered the "father" of modern anatomy. The heart is a roughly cone shaped hollow muscular organ. It is about 10cm long and is about the size of the owner's fist. It weighs about 225g in women's and is heavier in men about 310g. It is placed in the middle of chest or thoracic cavity (middle mediastinum), posterior to the body of sternum and the second to sixth costal cartilages and anterior to the fifth to the eighth thoracic vertebrae. The heart rests on the superior surface of the diaphragm. The heart has a base, which is formed by the atria and great arteries, and an apex, which is formed by the junction of ventricles and ventricular septum.



The heart lies in a double walled fibroserus sac called pericardial sac, which is divided into (a) fibrous pericardium and (b) serous pericardium. The fibrous pericardium envelops the heart and attaches onto the great vessels. The serous pericardium is a closed sac consisting of two layers – a visceral layer or epicardium forming the outer lining of the great vessels and the heart, and a parietal layer forming an inner lining of the serous pericardium contain the pericardial fluid, which prevents friction between the heart and the pericardium. The wall of heart is composed of three layers: **(a) epicardium: (b) myocardium: and (c) endocardium.** The epicardium is the outer lining of the cardiac chambers and is formed by the visceral layer of the serous pericardium. The myocardium that are seen predominantly in the left ventricle and inter-ventricular septum alone and includes is the intermediate layer of the heart and is composed of three discernable layers

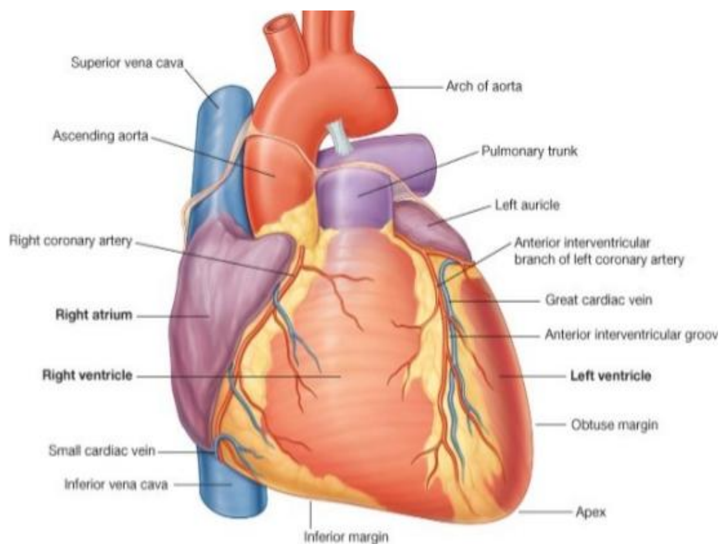
of muscle a subepicardial layer. The rest of the heart is composed mainly of the subepicardial and subendocardial layers. The myocardium also contains important structures such as excitable nodal tissue and conducting system. The endocardium the innermost layer of the heart is formed by endothelium and subendothelial connective tissue.

CARDIAC MUSCLE TISSUE

Cardiac muscle cells make up the myocardium portion of the heart wall. They are relatively short, branched fibers that measure approximately 10-20 micrometers in diameter and 50-100 micrometers in length. Typically each cardiac myocyte contains a single nucleus, which is centrally positioned. The cell is a short cylinder, with striations and a single central nucleus surrounded by a light perinuclear space. These cells branch, and at rectangular intervals, the cytoplasam reveals a transverse dark line called the 'intercalated disc'. It has partial properties both of the skeletal and the smooth muscle of fibres.

CARDIAC SURFACE ANATOMY

Because of its shape, the heart has three surfaces: anterior, inferior and posterior. Often the surfaces are referred to as: sternocostal (anterior), diaphragmatic (inferior) and base (posterior). The apex of the heart is directed downward, forward and to the left.



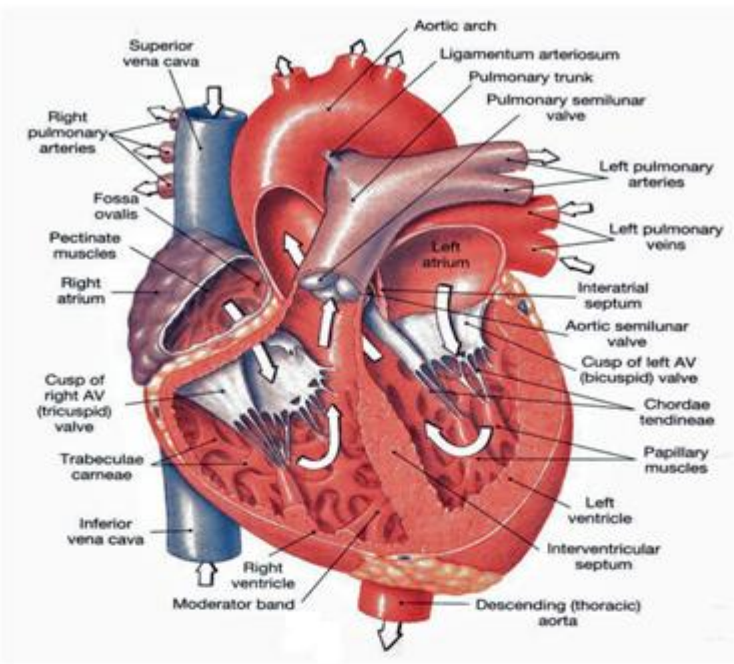
Anterior (Sternocostal) surface: It is formed mainly by the right atrium and right ventricle. They are separated from each other by the vertical atrioventricular groove. The right border of the anterior surface is formed by the right atrium while the left border is formed by left atrium and part of left auricle.

Inferior (Diaphragmatic) surface: It is formed mainly by the right and left ventricles separated by the posterior interventricular groove. The inferior surface of the right atrium into which the inferior vena cava opens, also forms part of this surface.

The base of the heart (posterior surface): It is formed mainly by the left atrium, into which the four pulmonary veins drain. It lies opposite to the apex. Often, the beginners think of the diaphragmatic surface of the heart as its base because of the fact that the heart rests on it, however, it should be kept in mind that the heart does not rest on its base. It rests on the diaphragmatic surface which is not the base. The posterior surface is called the base because it lies opposite to the apex of the pyramidal shaped heart. Each atrium

has an appendage (auricle), a small muscular pouch on the surface of each atrium, resembling an ear lobe. The annulus fibrous, considered as the heart's skeleton, is a plane of connective tissue composed four fibrous rings. Two of these rings from the interface between the atria and the ventricles and constitute an attachment for the muscular fibres and the atrioventricular valves. The two other fibrous rings surround arterial orifices and serve for attachment of the great blood vessels and the semilunar valves.

CARDIAC CHAMBERS

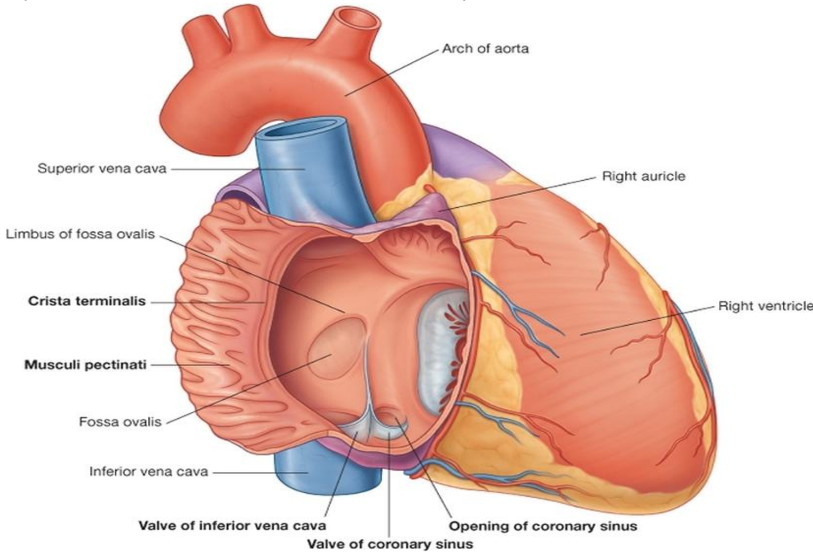


RIGHT ATRIUM

Venous blood returns to the heart via superior and inferior venae cavae into the right atrium, where it is stored during right ventricular systole. During ventricular diastole, blood flows from the right atrium into the right ventricle. The right atrium is larger than the left,

but its wall is somewhat thinner, about **2 mm thick.** The capacity of the right atrial cavity is about **57 millilitres**. The right atrium consists of two regions: Anteromedially, the **right atrial appendage** produces from the right atrium and overlaps the aortic root. On the posterior external surface of the right atrium a ridge, the **sulcus terminalis** (or terminal groove), extends vertically from the superior to the inferior vena cava. This corresponds to an internal muscular bundle, the **crista terminalis**. The sinus node is usually located at the lateral margin of the junction of the superior vena cava with the right atrium and the right atrial appendage.

The inner surface of the posterior and medial walls of the right atrium is smooth, while the surface of the lateral wall and of the right atrial appendage are composed of parallel muscle bundles, the **pectinate muscles**. There are four openings in the right atrium that are: The superior vena cava (svc), which returns blood from the upper half of the body, the opening of SVC has no valve. The inferior vena cava (IVC), that returns blood from the lower half of the body. A rudimentary valve, the **Eustachian valve**, formed by a crescentic fold. The coronary sinus, which returns blood from the coronary circulation.



The **Thebesian valve**, sometimes guards the opening of coronary sinus. The valve is formed by a fold of the lining membrane of the atrium .Right atrioventricular orifice, it lies anterior to the opening for inferior vena cava and is guarded by the tricuspid valve. The posterior cusp and right coronary cusp of the aortic root lean against the medial right atrium, forming a normal slight bulge known as the **torus aorticus**.

The insertion of crista terminalis through the interatrial groove is the origin of an important muscular bundle that runs from the right to left atrium, **Bachmann's bundle.** The Atrioventricular node is located in the lower atrial septum, just above the septal leaflet of the tricuspid valve. The Koch's triangle is the landmark for the AV node bundle: at the angle formed by the tendon of Todaro and the annulus of the septal leaflet of the tricuspid valve. The **fossa ovalis** (oval fossa) is an oval depression in the atrial septum that seperates the right and left atrial cavities. It lies at the lower part of the septum, above and to the left of the orifice of the inferior vena cava.

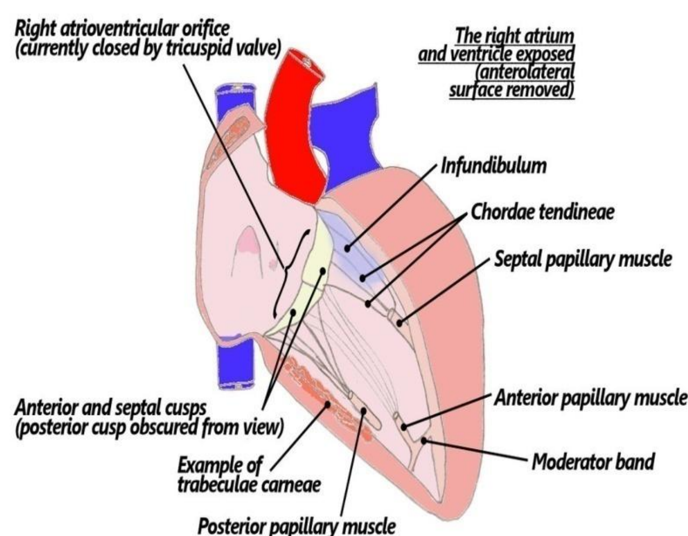
RIGHT VENTRICLE

Right ventricle receives venous blood from the right atrium during ventricular diastole and propels blood into the pulmonary circulation during ventricular systole. The right ventricle is normally the most anterior cardiac chamber, lying directly beneath the sternum. The right ventricle, which normally contracts against very low resistance, has a crescent-shaped chamber and a thin outer wall, measuring **4 to 5 mm thickness**. The anterior right ventricular wall curves over the ventricular septum, which normally bulges into the right ventricular cavity. The wall of right ventricle are much thicker as compared to those of right atrium. They show several internal projecting ridges, which are formed of muscle bundles. These ridges are known as **trabeculae corneae** and they give the walls a spongy appearance. They are of three types:

Type 1: First type of trabeculae consists of **papillary muscles**, which project inward They are attached by their bases to the ventricular wall and their apices are attached by fibrous chords, known as **chordae tendinae**, to the cusps of the tricuspid valve.

Type 2: Second type consists of muscle fibers attached to the ventricular walls in the same way as the first type but they are free in the middle. One of them, known as the **moderator band**, crosses the entire ventricular cavity from septal to anterior wall.

Type 3: It is simply composed of prominent ridges. The right ventricle can be partitioned into an inflow tact, an out flow tract, and an apical trabecular component. The trabecular muscles in the apex of the right ventricle are much coarser than those in the left ventricle.

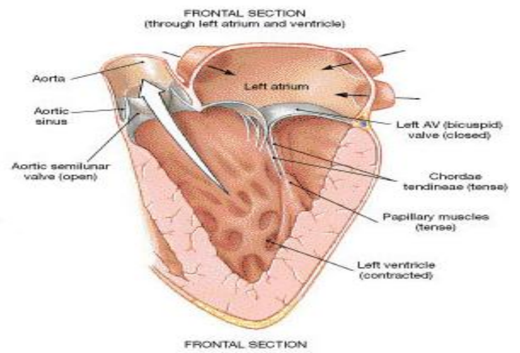


There are two openings in the right ventricle: the right atrioventricular orifice (guarded by tricuspid valve) and the opening for the pulmonary trunk (guarded by the pulmonary valve). The smooth walled outflow tract, also referred to as the **infundibulum**, form the superior part of the right ventricle. It is separated from the inflow tract by a thick muscle, the **crista supraventricularis**. The area of the ventricular wall below the pulmonary valve is called **right ventricular outflow tract (RVOT).**

LEFT ATRIUM

The left atrium receives blood from the pulmonary veins and serves as there servoir during the left ventricular systole and as conduit during left ventricular filling. The left atrium is located superiorly, in the midline and posterior to the other cardiac chambers. The wall of the left atrium is **3mm thick**, slightly thicker than that of the right atrium. The left atrial appendage lies to the left and anterior to the main pulmonary artery. The four pulmonary veins, two right and two left, superior and inferior veins, connect with left atrium posteriorly, conveying oxygenated blood from the lungs. The openings of the pulmonary veins are not guarded by any valve, however, the left atrioventricular orifice is guarded by bicuspid valve. The endocardium of the left atrium is smooth and slightly opaque. Pectinate muscles are present only in the left atrial appendage. The atrial septum is smooth but may contain a central shallow area, corresponding to the fossa ovalis.

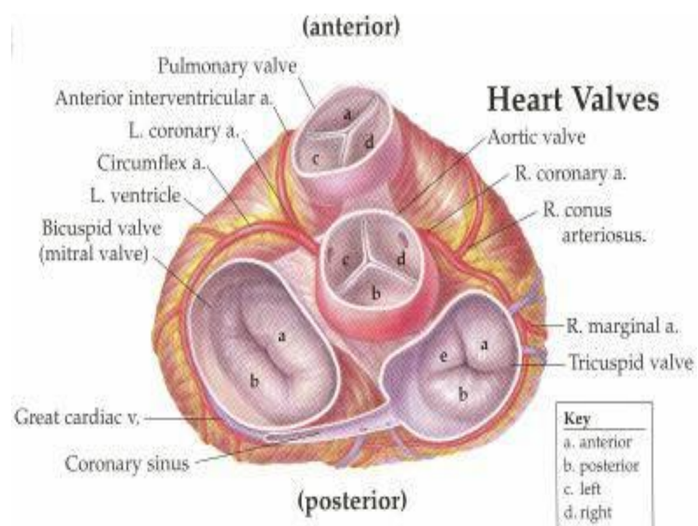
LEFT VENTRICLE



The left ventricle receives blood from the left atrium during ventricular diastole and ejects blood into the systemic arterial circulation during ventricular systole. The left ventricle is roughly bullet shaped, with the blunt tip directed anteriorly, inferiorly, and to the left, where it contributes, with the lower ventricular septum, to the apex of the heart. It is the strongest chamber of the heart. Its walls measuring **8 to 15 mm**, or three times thicker than those of the right ventricle. The reason for extra thick walls is that the left ventricle has to deal with high pressures. The pressure inside the left ventricle is about six times higher than that inside the right ventricle. In cross section, the right ventricle is circular and consequently the right ventricle is crescentic. It is because of the bulging of the interventricular septum into the right ventricle. There are two openings in the left ventricle: the left atrioventricular orifice (guarded by mitral valve, also known as bicuspid valve) and the aortic opening (guarded by aortic valve). The apical portion of the left ventricle is characterized by fine trabeculations.

CARDIAC VALVES

The heart contains four cardiac valves: two semilunar valves and atrioventricular valves. The two semilunar valves, aortic and pulmonic, the outlet orifice of their respective left and right ventricles. The two AV valves, mitral and tricuspid, guard the inlet orifice of their respective left and right ventricles. The four cardiac valves are surrounded by the fibrous tissue forming partial or complete “rings” (valve annulus).



SEMILUNAR VALVES

The aortic valve separates the left ventricular outflow tract from the ascending aorta. The aortic valve has also been called the left semilunar valve and the left arterial valve and has three leaflets, or cusps: the left coronary cusp, the right coronary cusp, and the non-coronary cusp. The inlets to the coronary artery system can be found within the

sinus of Valsalva, superior to the the leaflet attachments and inferior to the sinotubular junction. The left coronary ostium is found midway between the commissures of the left coronary cusp, and almost immediately branches into the anterior interventricular branch and the circumflex branch. The right coronary ostium is found above the right coronary cusp and gives rise to the right coronary artery. The final cusp is named the non-coronary cusp and is positioned posteriorly relative to the other two cusps.

The pulmonary valve separates the right ventricular outflow tract of the right ventricle from the pulmonary trunk. The pulmonary valve can also be referred to as the pulmonic valve, the right semilunar valve, and the right arterial valve. Its three leaflets, or cusps, are difficult to name because of the oblique angle of the valve. Its nomenclature is therefore derived based on the nomenclature of the aortic valve, which lies in proximity to it. The two leaflets attached to the septum are named the left and right leaflets, and correspond to the right and left leaflets of the aortic valve, which they face. The third leaflet is called the anterior leaflet or the non- coronary leaflet (to maintain the nomenclature of the aortic valve).

ATRIOVENTRICULAR VALVES

The mitral valve is also called the bicuspid valve and the left atrioventricular valve. As the name bicuspid valve may suggest, the mitral valve is considered to have two primary leaflets: the anterior and posterior leaflets. The anterior leaflet has also been called the septal, medial, or aortic leaflet, while the posterior leaflet is also referred to as the lateral, marginal, or mural leaflet. Each leaflet is then further broken down into scallops divided by commissures, or zones of apposition. Due to the high variability of leaflet and scallop anatomy, and an alphanumeric nomenclature has been proposed by Carpentier that breaks the leaflets into regions. Three regions are found on the anterior leaflet (A1-A3) with opposing regions on the posterior leaflet (P1-P3). The subvalvular apparatus of the mitral valve consists of chordae tendinae attaching to the anterior and posterior papillary muscles of the left ventricle.

The tricuspid valve, also called the right atrioventricular valve, gets its name because it is generally considered to have three leaflets: the anterior, posterior and septal leaflets. Of these, the anterior, also called the infundibular or anterosuperior, leaflet is typically the largest. The posterior leaflet is also referred to as the inferior or marginal leaflet and the septal leaflet is also referred to as the medial leaflet. Terminating on the ventricular side of the tricuspid valve leaflets, the chordae tendinae are connected to three papillary muscles in the right ventricle. In humans, the three papillary muscles of the right ventricle have highly variable anatomy. The anterior papillary muscle is usually the most prominent, with the moderator band terminating at its head. The moderator band typically originates from the septal papillary muscle. The septal papillary muscle is normally the least prominent.

GREAT CARDIAC ARTERIES AND VEINS

SUPERIOR VENA CAVA

The superior vena cava (SVC) is a large valveless venous channel formed by the union of the brachiocephalic veins. It receives blood from the upper half of the body (except the heart) and returns it to the right atrium. The SVC begins behind the lower border of the first right costal cartilage and descends vertically behind the second and third intercostal spaces to drain int the right atrium at the level of the third costal cartilage. Its lower half is covered by the fibrous pericardium, which it pierces at the level of the second costal cartilage.

INFERIOR VENA CAVA

The inferior vena cava (IVC) drains venous blood from the lower trunk, abdomen, pelvis and lower limbs to the right atrium of the heart. The IVC is formed by the confluence of the two common iliac veins at th L5 vertebral level. The IVC has a retroperitoneal course within the abdominal cavity. It runs along the right side of the vertebral column with the aorta lying laterally of the left. Various other veins drain into the IVC along its course before it passe through the diaphragm at the caval hiatus at the T8 level. It has a short intra-thoraci course before draining into the right atrium.

PULMONARY TRUNK

The pulmonary artery, which is one of the great arteries and is sometime referred to as the main pulmonary artery or pulmonary trunk, is a major blood vesse in the heart through which blood is transported to our lungs from the right ventricle While most arteries transport oxygenated blood to different parts of the body, th pulmonary artery delivers de-oxygenated blood to the right and left lungs In an average person, it has a length of approximately five centimetre (two inches) and a width of about three centimetres (one-point-two inches). Th main pulmonary artery (MPA) is intrapericardial and courses posteriorly an superiorly from the pulmonic valve. It divides into the left pulmonary artery (LPA) and right pulmonary artery (RPA) at the level of the fifth thoracic vertebra. The RPA is longer than the LPA and crosses the mediastinum, sloping slightly inferiorly to th right lung hilus. The LPA represents the continuation of the MPA.

PULMONARY VEINS

The pulmonary veins along with the bronchial veins are part of the venou drainage system of the lungs. The pulmonary veins drain oxygenated blood to th left atrium There are typically four pulmonary veins:

Right superior - drains right upper and middle lobes

Right inferior - drains right lower lobe

Left superior - drains left upper lobe

Left inferior - drains left lower lobe

The pulmonary veins course in the intersegmental septa and as such do not run with the bronchi like the pulmonary arteries do. The superior pulmonar vein takes an oblique inferomedial course whereas the inferior pulmonary veins run horizontally peripherally before taking a more vertical course. They pass through th lung hilum, antero-inferiorly to the pulmonary arteries, forming a shor intrapericardial segment, to drain into the left atrium. The ostia of the inferio pulmonary veins are more posteromedial and the left pulmonary veins being mor superior. There is extensive communication with deep bronchial veins within th lung and with the superficial bronchial veins at the hilum.The pulmonary veins a covered by a short (~9mm) myocardial layer, which is often the focus of atria fibrillation with the left superior pulmonary vein being the foci for almost half of cases.

AORTA

The aorta is the largest vessel in the body. It transports oxygenated bloo from the left ventricle of the heart to every organ. The aorta starts in the heart with the aortic valve; immediately adjacent is the aortic root, followed by the ascendin aorta, the transverse aorta (the aortic arch), the descending aorta, and th thoracoabdominal aorta. The aorta ends in the abdomen after bifurcation (t divid or fork into two branches) of the abdominal aorta in the two common iliac arteries The size of the aorta is directly proportionate to the patient's height and weight. It diameter may range from 3cm (more than an inch) to 1.2cm (half an inch). It is typically the largest in the aortic root and smallest in the abdominal aorta.The aorta is divided as here in seven sections:

Aortic valve: The aorta starts with the aortic valve just below the first branches of the aorta, the coronary arteries.

Aortic Root: The aortic root is the segment above the aortic valve and below th sinotubular junction. The left and right coronary arteries - supplying the heart wit oxygenated blood – arise here from the sinuses of Valsalva.

Ascending aorta: The ascending aorta arises from the upper end of the left ventricle. It is about 5 cm long and enclosed in the pericardium. The ascending aorta is the segment between the sinotubular junction and the largest aortic branch vessel; the innominate (brachiocephalic) artery. This is the only portion of the aorta that doe not give any branch vessels. The ascending aorta is the most anterior (toward the front of the body) portion of the aorta.

Aortic arch: Arch of the aorta is the continuation of the ascending aorta. It is situate in the superior mediastinum behind the lower half of the manubrium sterni. It begin 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; the travels 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 this point the aortic arch continues as the descending aorta. The aortic arch has three branches. The first, and largest, branch of th arch of the aorta is the brachiocephalic trunk, which is to the right and slightl anterior to the other two branches and originates behind the manubrium of the sternum. Next, the left common carotid artery

originates from the aortic arch to the left of the brachiocephalic trunk and then ascends along the left side of the trachea and through the superior mediastinum. Finally, the left subclavian artery comes off of the aortic arch to the left of the left common carotid artery and ascends, with the left common carotid, through the superior mediastinum and along the left side of the trachea. The arch of the aorta 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. below the superior border to the manubrium sterni. Blood flows from the upper curvature to the upper regions of the body, located above the heart - namely the arms, neck, and head.

Descending thoracic aorta: The descending aorta (thoracic aorta) is between the arch of the aorta and the diaphragm muscle below the ribs. It lies in the posterior mediastinum. At the origination point, it is on the left side of the vertebrae. As it descends, it winds around the vertebrae and ends in front. The diameter of the artery is 2.32 centimeters. It has six paired branches: bronchial arteries, mediastinal arteries, esophageal arteries, pericardial arteries, superior phrenic artery, and intercostal arteries. There are nine pairs of the intercostal arteries. The right branches are longer than the left, because the descending aorta (thoracic aorta) is on the left side of the vertebrae. Through its various branches, it supplies blood to the esophagus, lungs, and the chest area, including the ribs and mammary glands.

Abdominal aorta: The abdominal aorta branches to the intestine and the kidneys and divides into left and right common iliac arteries. The branch vessels of the abdominal aorta include the celiac artery, the superior mesenteric artery, the left and right renal arteries, and the inferior mesenteric artery.

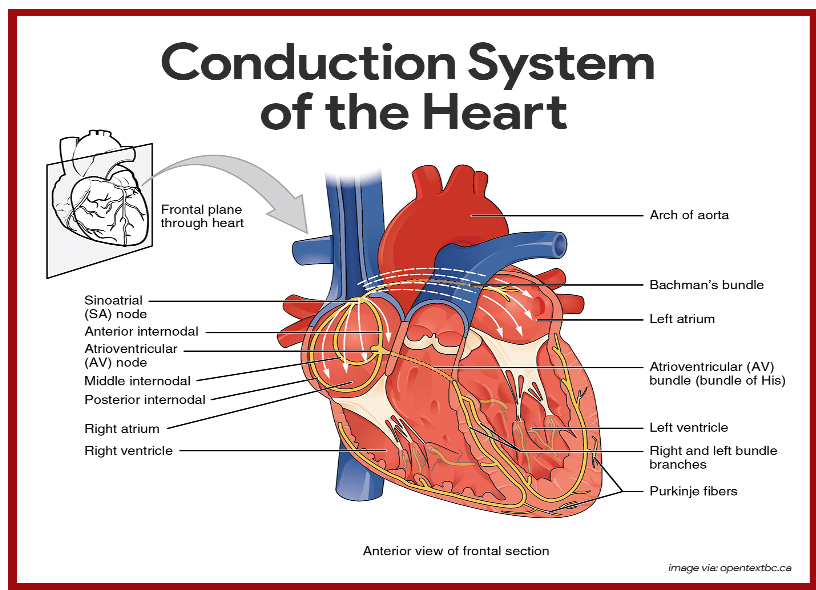
Thoracoabdominal aorta: The thoracoabdominal aorta is the segment starting past the last branch of the aortic arch and ends with the abdominal aortic bifurcation into left and right common iliac artery.

Microscopic anatomy of the aorta:

The aortic wall is made up of three layers

- Inner layer of intima
- Middle layer of media
- Outer layer of adventitia that includes vessels supplying the aortic valve with oxygenated blood.

THE CONDUCTION SYSTEM OF HEART



The electrical conduction system of the heart is composed of sinoatrial (SA) node, internodal atrial conduction tracts, interatrial conduction tract, atrioventicular (AV) node, bundle of HIS, right and left bundle branches, and Purkinje network. The AV node and the bundle of HIS form the AV junction.

The bundle of HIS, the right and left bundle branches, and the Purkinje network are also known as the HIS-Purkinje system of the ventricles. As its sole function, the electrical conduction system of the heart transmits minute electrical impulses from the SA node (where they are normally generated) to the atria and ventricles, causing them to contract.

The SA node lies in the wall of the right atrium near the inlet of the superior vena cava and consists of pacemaker cells that generate electrical impulses automatically and regularly. The AV node lies partly in the right side of the interatrial septum in front of the opening of the coronary sinus tricuspid valve. **Function of conducting system of heart:** The conducting system of heart is responsible not only for generating rhythmical cardiac impulses but also for conducting these impulses rapidly throughout the myocardium of the heart. Thus it aids in coordinated and efficient contraction of different chambers of the heart. The activities of the conducting system of heart can be influenced by the autonomic nerve supply of heart. The parasympathetic nerves slow the rhythm and diminish the speed of conduction, while the sympathetic nerves have the opposit effect.

Internodal pathways: Impulses from the sinuatrial node have been show to travel to the atrioventricular node more rapidly than they can pass through the muscle of the heart. This can be explained by presence of specialized pathways in the atrial wall, which have a structure in between that of the Purkinje fibers and ordinary muscle cells. These specialized pathways are called internodal pathways and there are three of them in the atrial wall.

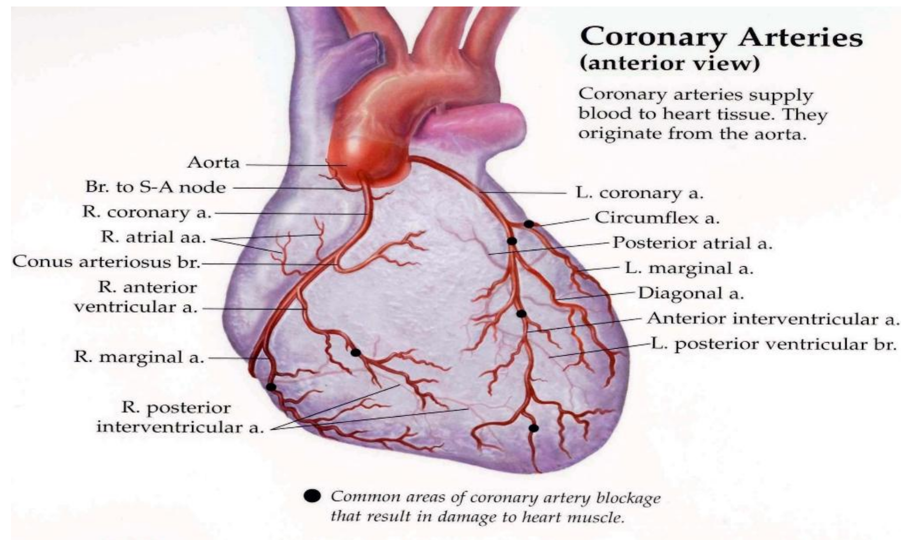
Anterior internodal pathway: It leaves the anterior end of the SA node and passes anterior to the superior vena cava to end in the AV node.

Middle internodal pathway: It leaves the posterior end of the SA node and passes posterior to the superior vena cava to end in the AV node.

Posterior internodal pathway: It leaves the posterior part of the SA node and descends through the crista terminalis to end in the AV node.

CORONARY ARTERIES

The heart muscle, like every other organ or tissue in your body, needs oxygen-rich blood to survive. Blood is supplied to the heart by its own vascular system, called coronary circulation.



CORONARY OSTIA

Location:The coronary arteries originate as the right and left main coronary arteries, which exit the ascending aorta just above the aortic valve (coronary ostia). There are three cusps of the aortic valve: the non-coronary cusp, which contains no ostia, the righ coronary cusp, which contains the ostia of the right coronary artery, and the left coronary cusp, which contains the ostia of the left (main) coronary artery. The ostia of the left and right coronary arteries are located just above the aortic valve, as are the left and right sinuses of Valsalva.

Function: Oxygenated blood is pumped into the aorta from the left ventricle; it then flows into the coronary artery ostia. Blood flow into the coronary arteries is greatest during ventricular diastole.

RIGHT CORONARY ARTERY

Location: The right coronary artery emerges from the right coronary cusp into the AV groove. It descends through the groove, then curves posteriorly, and makes a bend at the crux of the heart and continues downward in the posterior interventricular sulcus. Within millimeters after emerging from the aorta, the right coronary artery gives off two branches: 1) **the conus (arteriosus) artery** which runs to the right ventricular outflow tract, and 2) the atrial branch which gives off the **SA nodal artery** , which runs along the anterior right atrium to the superior vena cava, encircling it before reaching the SA node. The right coronary artery continues in the AV groove and gives off a variable number of branches to the right atrium and right ventricle. The most prominent of these is the right **marginal branch** which runs down the right margin of the heart supplying this part of the right ventricle. As the right coronary curves posteriorly and descends downward on the posterior surface of the heart, it gives off two to three branches. The **AV nodal artery** which branches from the right coronary artery at the crux of the heart and passes anteriorly along the base of the atrial septum to supply the AV node (in 50-60 % of hearts), proximal

parts of the bundles (branches) of His, and the parts of the posterior interventricular septum that surround the bundle branches.

Function: In addition to supplying blood to right ventricle (RV), the RCA supplies 25% to 35% of the left ventricle (LV). In 85 % of patients (Right Dominant), the RCA gives off the posterior descending artery (PDA). In the other 15% of cases (Left Dominant), the PDA is given off by the left circumflex artery. The PDA supplies the inferior wall , ventricular septum , and the posteromedial papillary muscle. The RCA also supplies the SA nodal artery in 60% of people. The other 40% of the time, the SA nodal artery is supplied by the left circumflex artery .

LEFT MAIN CORONARY ARTERY

Location: The left coronary artery (left main coronary artery) emerges from the aorta through the ostia of the left aortic cusp, within the sinus of Valsalva. The plane of the semilunar valve is tilted so that the ostium of the left coronary artery is superior and posterior to the right coronary ostium. The left coronary artery travels from the aorta, and passes between the pulmonary trunk and the left atrial appendage. Unde the appendage, the artery divides (and is thus a very short vessel) into the anterior interventricular (left anterior descending artery) and the left circumflex artery. This bifurcation can often be seen when looking into the left coronary artery ostia with a videoscope. Note that the left coronary artery may be completely absent, i.e., the anterior interventricular and circumflex arteries arise independently from the left aortic sinus.

Function: The coronary arteries supply blood to the myocardium (heart tissue) itself; that is, coronary capillaries deliver oxygenated blood (nutrients) to all of the heart's cells. The left coronary artery and its branches supply the majority of oxygenated blood to ventricular myocardium, and additionally to the left atrium, left atrial appendage, pulmonary artery, and aortic root.

LEFT ANTERIOR DESCENDING

Location: The left anterior descending (LAD, interventricular) artery appears to be a direct continuation of the left coronary artery which descends into the anterior interventricular groove. Branches of this artery, anterior septal perforating arteries, enter the septal myocardium to supply the anterior two-thirds of the interventricular septum (in ~90% of hearts).

Function: In general, the LAD artery and its branches supply most of the interventricular septum; the anterior, lateral, and apical wall of the left ventricle, most of the right and left bundle branches, and the anterior papillary muscle of the bicuspid valve (left ventricle). It also provides collateral circulation to the anterior right ventricle, the posterior part of the interventricular septum, and the posterior descending artery.

LEFT CIRCUMFLEX ARTERY

Location: The circumflex artery branches off of the left coronary artery and supplies most of the left atrium: the posterior and lateral free walls of the left ventricle, and part of the

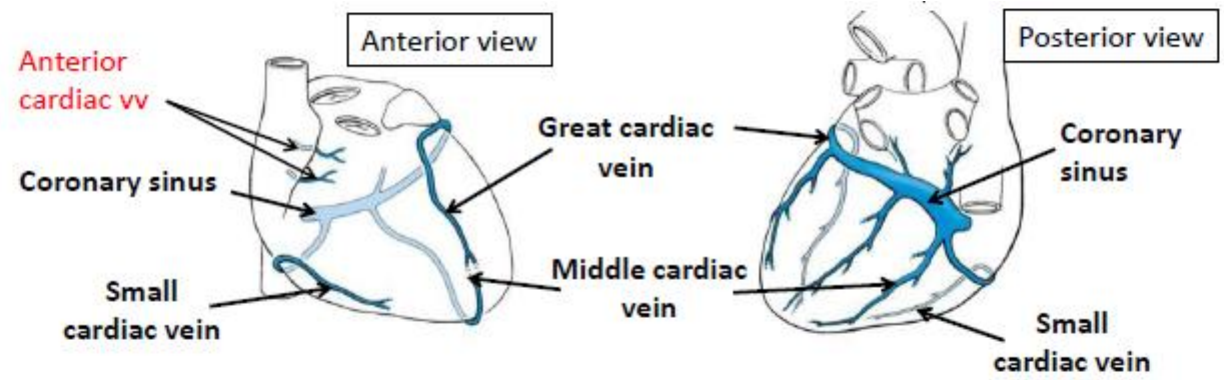
anterior papillary muscle. The circumflex artery may give off a variable number of left marginal branches to supply the left ventricle. The terminal branch is usually the largest of these branches. More likely, the circumflex artery may continue through the AV sulcus to supply the posterior wall of the left ventricle and (with the right coronary artery) the posterior papillary muscle of the bicuspid valve. The left circumflex branches decrease in diameter along their lengths.

Function: In 40-50% of hearts the circumflex artery supplies the artery to the SA node.

CARDIAC DOMINANCE

In about 10% of hearts, the right coronary artery is rather small and is not able to give the posterior interventricular branch. In these cases the circumflex artery, the continuation of left coronary provides the posterior interventricular branch as well as to the AV node. Such cases are called as left dominant.Mostly right coronary gives posterior interventricular artery. Such hearts are right dominant. Thus the artery giving the posterior interventricular branch is the dominant artery.

THE CORONARY VEINS



After flowing through myocardium, most (80%) of the blood is returned to the right atrium by several prominent veins that run along the surface of the heart.

Coronary sinus: Is the largest vein draining the heart and lies in the coronary sulcus , which separates the atria from the ventricle the opening of the IVC and the AV opening. Has a one-cusp valve at the right margin of its aperture.

Receives the great, middle, and small cardiac veins; the oblique vein of the left atrium; and the posterior vein of left ventricle.

Great cardiac vein: Begins at the apex of the heart and ascends along with the IV branch of the left coronary artery continues as the coronary sinus. Turns to the left to lie in the coronary sulcus and continues as the coronary sinus.

Middle cardiac vein: Begins at the apex of the heart and ascends in the posterior IV groove, accompanying the posterior IV branch of the right coronary artery. Drains into the right end of the coronary sinus.

Small cardiac vein: Runs along the right margin of the heart in company with the marginal artery and then posteriorly in the coronary sulcus to end in the right end of the coronary sinus.

Oblique vein of the left atrium(oblique vein of Marshall): Small vessel which descends obliquely on the back of the left atrium and ends in the coronary sinus near its left extremity.

Anterior cardiac vein: Drains the anterior right ventricle, crosses the coronary groove, and ends directly in the right atrium.

Smallest cardiac veins (venae cordis minimae): Begin in the wall of the heart and empty directly into its chambers.

THE ARTERIAL CIRCULATION

The aorta curves upward from the left ventricle of the heart as the ascending aorta, arches to the left as the aortic arch, then drops downward following the spine as the thoracic aorta to finally pass through the diaphragm to become the abdominal aorta. The branches of the parts of the aorta are listed below in their sequence from the heart and the organs served

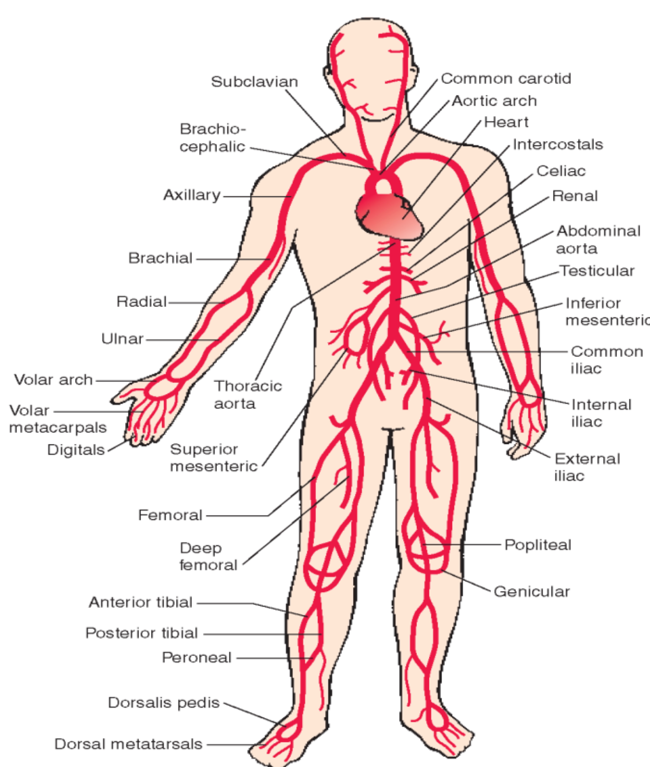
Branches of the Ascending Aorta:

- **Right cononary artery- heart**
- **left cononary artery - heart**

Branches of the Aortic Arch:

- **Brachiocephalic artery**
    - **Right common carotid artery - head & neck**
    - **Right subclavian artery - vessel carrying blood beneath the right clavicle.**
    - **Posterior auricular: vessel carrying blood to the ear.**
    - **Occipital: vessel carrying blood to the head.**

- **Left common carotid artery**
    - **Left internal carotid - brain**
    - **Left external carotid - head & neck**
- **Left subclavian artery**
    - **vertebral artery - brain**



The subclavian artery becomes the **axillary artery**, then continues into the arm as the **brachial artery** which supplies the arm. At the elbow, the brachial artery splits

- **Radial artery** - forearm
- **Ulnar artery** - forearm

Branches of the Thoracic Aorta:

- **Intercostal arteries** - 10 pairs supply the muscles of the thorax wall
- **Bronchial arteries** - lungs
- **Esophageal arteries** - esophagus
- **Phrenic arteries** - diaphragm

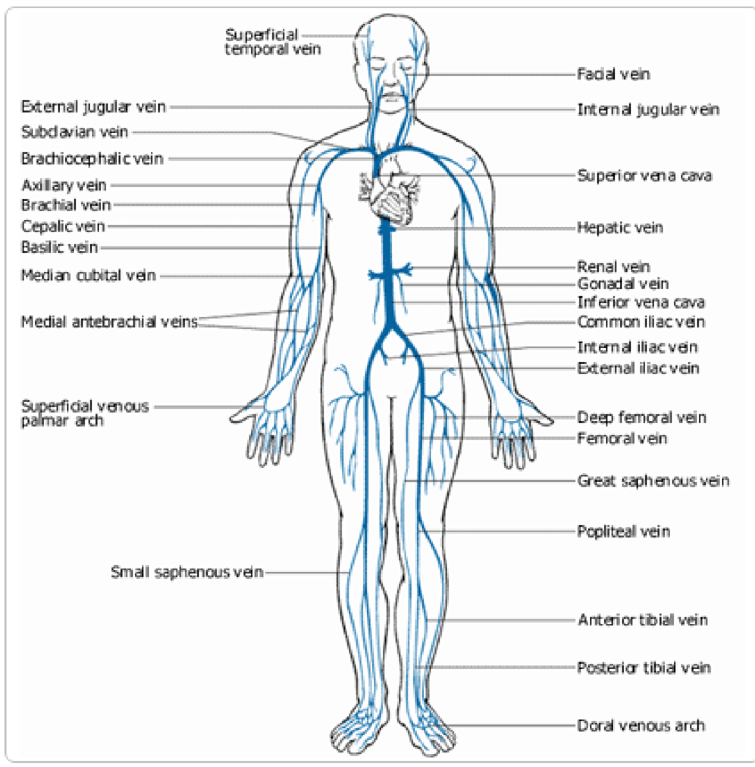
Branches of the Abdominal Aorta:

- **Celiac Trunk**
    - **Left gastric artery** - stomach
    - **Splenic artery** - spleen
    - **Common hepatic artery** - liver
- **Superior mesenteric artery** - small intestine
- **Right and Left Renal arteries** - kidneys
- **Right and Left Gonadal arteries** - called **ovarian arteries** in females (serving the ovaries) and **testicular arteries** in males (serving the testes).
- **Lumbar arteries** - several pairs serving the heavy muscles of the abdomen and trunk walls.
- **Inferior mesenteric artery** - lower large intestine
- **Right and Left Common iliac artieries** - the final branches of the abdominal aorta. Each divides into:
    - **Internal iliac artery** - pelvic organs
    - **External iliac artery** - enters the thigh where it becomes the femoral artery. The femoral artery and its branch, the deep femoral artery , serve the thigh. At the knee, the femoral artery becomesthe popliteal artery, which then splits into:
    - **Anterior and posterior tibial arteries,** which supply the leg and foot.The anterior tibial artery terminates in the dorsalis pedis artery, which supplies the dorsum of the foot.

THE VENOUS CIRCULATION

Although arteries are generally located in deep, well-protected body areas, many veins are more superficial and some are easily seen and palpated on the body surface. Most deep veins follow the course of the major arteries, and with a few exceptions, the naming of these veins is identical to that of their companion arteries.While major systemic arteries branch off the aorta, the veins converge on the vena cava.

Blood returns to the right atrium of the heart through the vena cava. Veins draining the head and arms empty into the superior vena cava and those draining the lower body empty into the inferior vena cava. The veins listed below begin distally and move proximally to the heart.



Veins Draining into the Superior Vena Cava:

- **Radial and ulnar veins are deep** veins draining the forearm. They unite to form the **brachial vein**, which drains the arm and empties into the **axillary vein.**
- **Cephalic vein** provides superficial drainage of the lateral aspect of the arm and empties into the **axillary vein**.
- **Basilic vein** provides superficial drainage of the medial aspect of the arm into the brachial vein.
  The basilic and cephalic veins are joined at the anterior aspect of the elbow by the median **cubital vein**. (This vein is often the site for blood removal for the purpose of blood testing.)
- **Subclavian vein** receives blood from the arm through the **axillary vein** and from the skin and muscles of the head through the **external jugular vein.**
- **Vertebral vein** drains the posterior part of the head.
- **Internal jugular vein** drains the dural sinuses of the brain.
- **Left & Right Brachiocephalic veins** drain the subclavian, vertebral, and internal jugular veins on their respective sides. The brachiocephalic veins join to form the superior vena cava, which enters the heart.
- **Azygos vein** a single vein that drains the thorax and enters the superior vena cava just before it joins the heart.
  Veins Draining into the Inferior Vena Cava:

The inferior vena cava, which is much longer than the superior vena cava, returns blood to the heart from all body regions below the diaphragm.

- **Anterior and posterior tibial veins** and the **peroneal vein** drain the calf and foot. The posterior tibial vein becomes the **popliteal vein** at the knee and then the **femoral vein** in the thigh. The femoral vein becomes the **external iliac vein** as it enters the pelvis.
- **Great saphenous veins** are the longest veins in the body. They receive the superficial drainage of the leg. They begin at the dorsal venous arch in the foot and travel up the medial aspect of the leg to empty into the femoral vein in the thigh.
- Each **Left & Right common iliac vein** is formed by the union of the **external iliac vein and the internal iliac vein** (which drains the pelvis) on its own side. The common iliac veins join to form the **inferior vena cava**, which the ascends superiorly in the abdominal cavity.
- **Right gonadal vein** drains the right male or female sex gland. (The Left gonadal vein empties into the left renal vein superiorly.)
- **Left & Right renal veins** drain the kidneys.
- **Left & Right hepatic veins** drain the liver.

THE PORTAL CIRCULATION

1. Blood flow from the abdominal organs that passes through the portal vein, the sinusoids of the liver and into the hepatic vein before returning to the heart from the inferior vena cava. This pathway permits the liver to process and to detoxify substances entering the body from the gastrointestinal tract.

2. A portal system between the hypothalamus and the anterior pituitary gland. The hypothalamus secretes releasing or inhibiting hormones into the blood; they are carried directly to the anterior pituitary and stimulate or inhibit secretion of specific hormones.

THE FETAL CIRCULATION

Oxygenated blood from the **placenta** enters the fetus through **umbilical vein**. Most of the newly oxygenated blood bypasses the liver via the **ductus venosus** and combines with oxygenated blood in the inferior vena cava. Blood then joins deoxygenated blood from the superior vena cava and empties into the right atrium. Since pressure in the right atrium is larger than pressure in the left atrium, most blood will be shunted through the **foramen ovale**. Some blood does travel from the right atrium to the right ventricle through the pulmonary trunk but most blood bypasses the pulmonary arteries and moves directly to the aorta via the **ductus arteriosus**. Deoxygenated blood returns to the umbilical arteries originating from the internal iliacs near the bladder.

LYMPHATICS OF THE HEART

The lymphatics of the heart consist of small lymph vessels in the connective tissues of the tunica intima and media, which drain into an extensive epicardial lymphatic plexus. Efferent vessels from the epicardial plexus converge in the cardiac sulci to form right and left cardiac collecting lymph trunks, which run along with branches of the coronary arteries towards the root of the ascending aorta and pulmonary trunk.

NERVE SUPPLY OF HEART

Two sets of autonomic nervous fibres innervative the heart and help regulate its action.One set consists of parasympathetic fibres , the other of sympathetic fibres. The **parasympathetic** fibres are located in the vagus nerve, which is the tenth cranial nerve. The sympathetic fibres are termed as cardiac accelerator or augmentor nerves and are postganglionic neurons originating usually in the cervical ganglia and first 5 thoracic ganglia of the sympathetic chain.

These nerves may influence the heart through two chain mechanism:(1) by altering the contractile force of the myocardium, and (2) by increasing or decreasing the heart rate. Generally speaking parasymapathetic stimulation (through the vagus) will decrease the contractile force and the rate of the heart, while sympathetic stimulation will increase both factors.

Any impulse conveyed to the heart or any other effector organ is transmitted through the pathways known as efferent nerves . These regulatory impulse originate in response to sensory impulses which are transmitted to the regulatory center via afferent nervous pathways.

Vagus Nerves

The vagus nerves is composed of both afferent and efferent nerve fibres. The vagal nerve fibres which innervate the heart are divided into right and left bundles, the right vagus and left vagus. The right vahus innervate the SA node , and left vagus innervate the AV node and the Bundle of His. Both vagi innervate the atrial muscle . The ventricular myocardium is said to contain few or no paraysmpathetic fibres; but, paradoxically , vagal stimulation significantly affects the left ventricle.

Vagal stimulation slows the heart rate and decrease its contractile force. It is believed that these effects are due to hyperpolarization of cardiac cells to the point that resting membrane potentials are greatly increased and actoin potentials are severly decreased. Atrial contraction are weakened , and the speed of impulse transmission from the SA node through the atria and AV node decreased .

Vigorous or persistent vagal stimulation can be result in partial heart block or temporary complete ventricular arrest. The arrest does not persist because, in the presence of

impulse inhibition in the SA and AV nodes, a potential pacemaker (ectopic focus) in the ventricles will fire and cause ventricular contraction.This phenomenon is refferd as vagal escape.

The vagal neurons constantly propagate impulses, causing a continous braking ofr inhibiting influence to be exerted on the heart. This is reffered to as vagal tone. Cutting of the vagi or blocking their impulses results in an immediate increase of the heart rate and strength of contraction because the heart is then released from the normal inhibitory influence of vagal impulses.

Augmentor Nerves

The augmentor nerve representing the sympathetic division of the autonomic nervous system, innervate all of the conduction system and the myocardium of the atria and ventricles. In other words , the entire heart is innervated by the sympathetic nervous system. Most sources believes that these contain only efferent nerves.

Since innervation of the heart by the augmentor nerves is vast in comparison to innervation by the vagi, the potential effects of sympathetic stimualtion far surpass those of parasympathetic stimulation. Sympathetic stimulation via the augmentor nerves accelerates the transmission of impulses throughout the conduction system and strenghthens both atria l and ventricular contractions.

Augmentor impulses are thoght to accelerates the heart rate and impulse conduction by increasing the cell membrane permeability to sodium , thereby allowing the threshold voltage to be more rapidly attained. Conversely, Vagal impulses increase the resting potential of cells by increasing their permeability to potassium, making it more difficult to achieve a threshold voltage.

Normally, there is also a continous propagation of augmentor impulses, without which the strength of ventricular contraction would be about 20% below its normal level. Under condition of stress , sympathetic stimulatiom of the heart increases. Maximal sypmpathetic stimulation can increase te force of myocardial contraction up to 200%.

Sympathetic- Parasympathetic Balance

Since there is continous propagation of both sympathtetic and parasympathetic impulses cardiac action at any moment will correspond to the summation or balance of these opposing influences. Under resting conditions, the parasympathetic influence appears to be greater, while stress situations cause the augmentor nerves to control heart action.

Reflex Control

Nervous impulses to the heart are controlled mainly by centers in the medulla oblongata. There is a cluster of cells called the cardioinhibitory center and another cluster known as the cardioaccelerator center. Activation of the cardioinhibitory center causes an increase of parasympathetic impulses to the heart through the vagus, resulting in diminished cardiac action. Conversely, activation of the cardio accelerator would increase the sympathetic stimulation of the heart, having oppsite effect on cardiac action.

Cardiac function may also be influenced by impulses originating in other parts of the brain. These include the motor and premotor cortex, the frontal lobe, portiond of the temporal lobe, the thalamus; and the hypothalamus. These centers help correlate heart actio to such state as exercise, emotional stress and sleep.

Aortic and Carotid Sinuses

The most important afferent pathways which deal directly with cardiac and vascular action originate in the carotid and aortic sinuses. A sinus is a dilated or hollowed- out area(in this case., of a blood vessel), and in the carotid and aortic sinuses are located nerve ending sensitive to special stimuli.

The carotid sinuses are located at the bifurcation of each common carotid artery. They are innervated by the carotid sinus nerves or nerves of hering, which jion the glossopharyngeal nerves to send afferenr impulses to the cardiac centers. The aortic sinus in located in the transverse aortic archand is innervated by afferent fibres of the vagus nerve.

These nerve endings in the carotid and aortic sinuses are sensitive to pressure changes and are therefore often referred to as pressoreceptaors. Because they are helpful in buffering pressure chenges, they are also known as baroreceptors, and nerves themselves are called buffer nerves. Actually , the sensation which stimulates these sensory nerve endings is not pressure , but expansion of arterial wall secondary to rise in pressure.

Stretching of the arterial wall from increases in pressure causes more sensory or afferent impulses to be sent to the cardiac center, which responds by activating cardioinhibitory centre.A fall in blood pressure causes the stretch receptors to dischsrge at a slower rate or to cease dischargung. The cardioinhibitor center will therefore remain relatively inactive , allowing the cardioaccelerator center to compensate for the decreased blood pressure by propagation of impulses through the augmentor nerves. The carotid and aortic sinus mechanism for buffering changes in arterial blood pressure are called the aortic and carotid sinus reflexes.

Since pressure at the locations of the carotid and aortic sinuses is pulsatile, the rate of discharge fromthe stretch receptors is increased during systole, resulting in an inverse relatioship between blood presure and heart rate , which is termed Marey's law of heart.

Aortic and Carotid Bodies

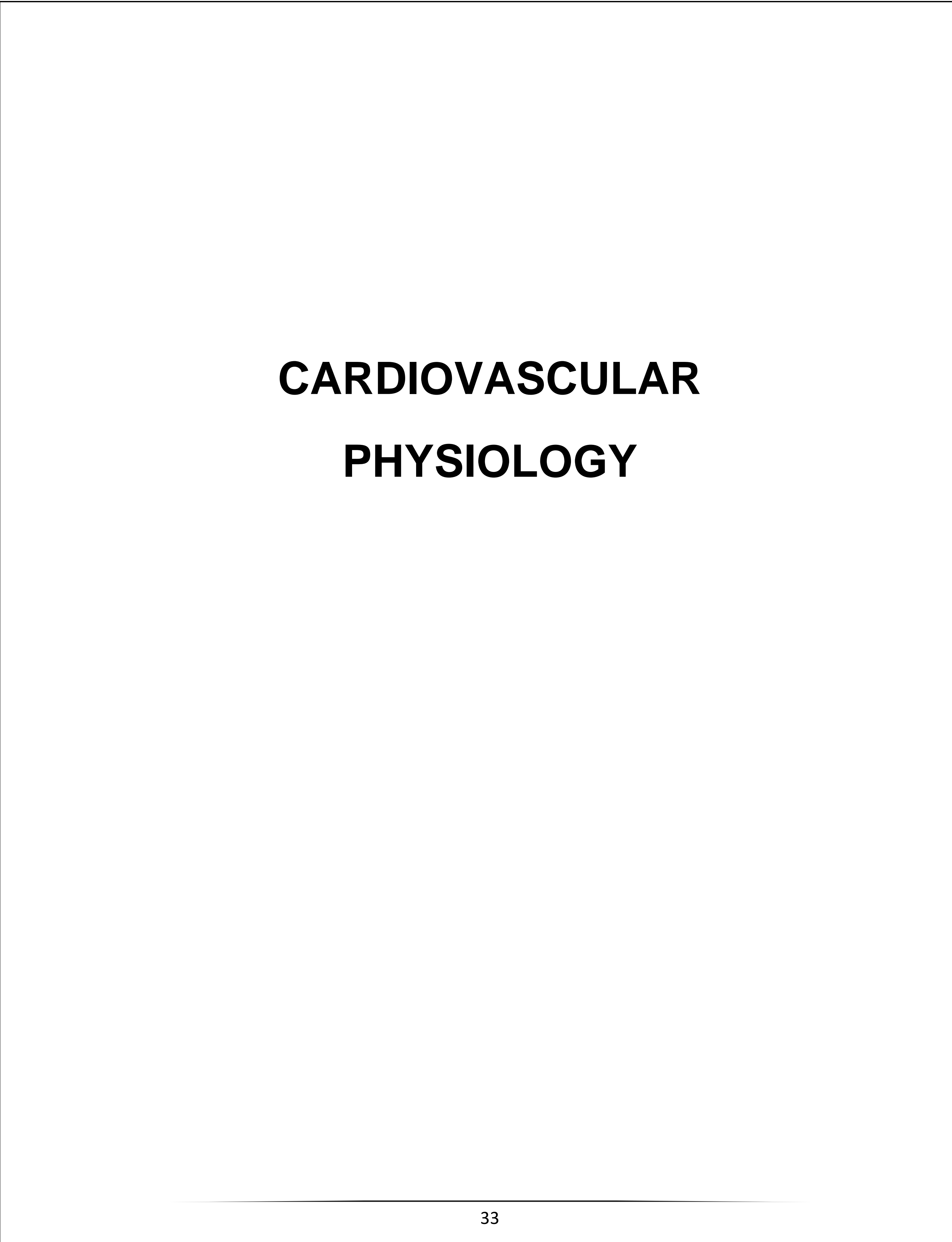
There are other specialized cells, the carotid and aortid bodies, located near the carpotid and aortic sinuses. The carotid bodies are immediately rostral to the bifurcation of region: that is , in the tissue between the pulmonary artery and aorta. The cells are termed chemoreceptors because they are sensitive to the pO_2-and pCO_2 of blood.

It is difficult to isolate the effects on the heart which are triggered by the sensivity of these chemoreceptors to hypoxemia and /or hypercapnea. While it is true that hypoxemia causes an increased cardiac output, the heart action is thught to be secondary to reflex hyperventilation or direct stimulation of the cardiac center in the medulla oblongata for , if un changed. Secondly, stimulation of the carotid bodies decreases the heart rate. Therefore the exact relationship between the chemoreceptors and reflex cardiac function remain unknown.

Low- Pressure Reflexes

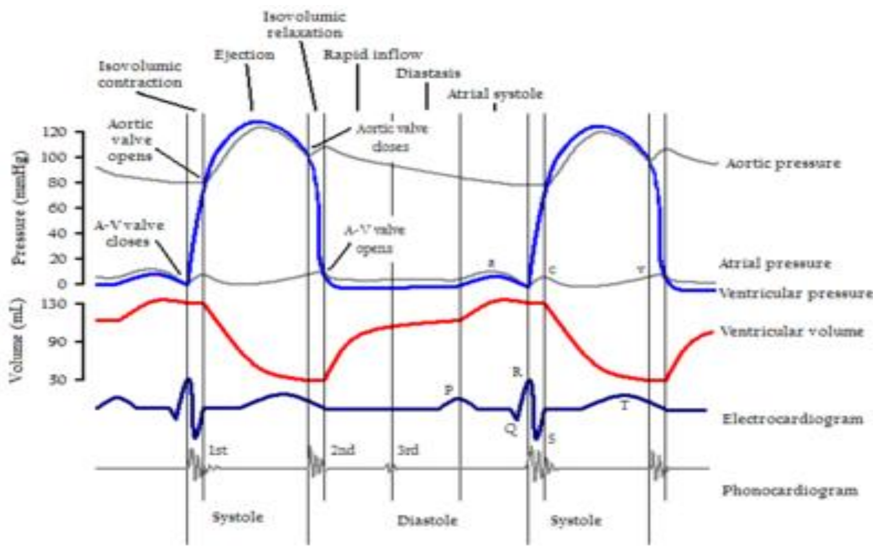
The aortic and carotid sinus reflexes act mainly to inhibit increases in arterial pressure and become less active as pressure is reduced. The sini-aortic nerves have therefore referred to as " high-pressure" receptors. There are other groups of specialized cells wich are "low –pressure" receptors that have been found bith at the juntion of great veins with thier respective atria and in the ventricles.

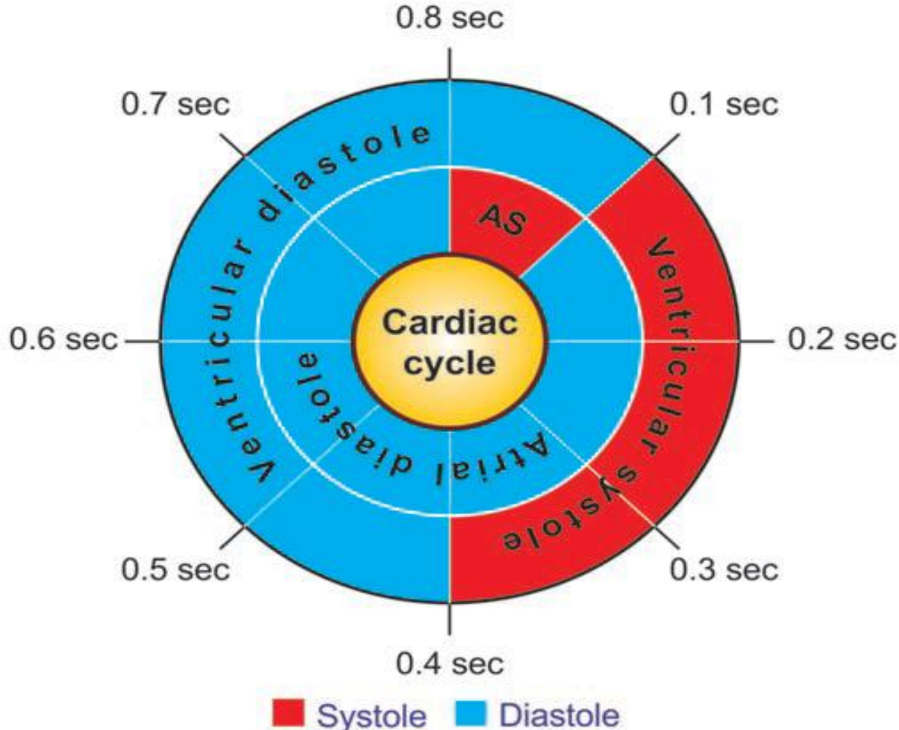
These receptors send sensory impulses though afferent fibres of the vagus nerve . Depending upon their location in the heart, such impulses are propagated at different periods of the cadiac cycle.Sensory impulses from these receptors are said to be produce same response as the " high-pressure" receptors: increased vagal tone and decreased sympathetic tone. While it may seen contradictory that opposite stimuli produce similar responses , the explanation of the " low-pressure" reflexes probably lies in the fact that low pressure is due to low volume and that reflexes the slowing of the heart allows it more time to fill.



CARDIOVASCULAR PHYSIOLOGY

THE CARDIAC CYCLE





The function of the heart is to maintain a constant circulation of blood throughout the body. The heart acts as a pump and its action consists of a **series of events known as the cardiac cycle**. During each heartbeat, or cardiac cycle, the heart contracts and then relaxes. The period of contraction is called systole and that of relaxation, diastole. The blood returns from the systemic circulation to the right atrium and flows via the tricuspid valve to the right ventricle. It is ejected from the right ventricle through pulmonary valve to the lungs. Oxygenated blood returns from the lungs to the left atrium, and flows via the mitral valve to the left ventricle. Finally blood is pumped through the aortic valve to the aorta and the systemic circulation. When the ventricles contract, the atrioventricular valves closes, this will prevent the back flow of blood from the ventricles to the atria. The papillary muscles are shortened and so that they pull chordae tendinae and this prevent the inversion of the valves into the atria. As a result, there is increase in the blood pressure in the ventricles. As soon as the pressure inside the ventricles exceeds that in the pulmonary artery and aorta, the valves guarding the arterial orifices open and blood is driven from the ventricles into the arteries. After this contraction phase the ventricles relax. The semilunar valves prevent the regurgitation of blood from the arteries to the ventricles. With each contraction the cardiac muscle squeezes blood from the heart into pulmonary or systemic circulation. The blood flows from an area of high pressure to low pressure area. The changing of blood pressure inside each chamber acts to open and close the heart valves as blood moves through the heart's chambers and into the circulation.

Stages of the cardiac cycle

The normal number of cardiac cycles per minute ranges from 60 to 80.Each cycle consists of 7 events.

- Phase 1: Atrial systole

Ventrivular systole

- phase2: Isovolumetric contraction
- Phase 3: Rapid ejection
- Phase4: Reduced ejection

ventricular diastole

- Phase 5: isovolumetric relaxation
- Phase 6: rapid filling
- Phase7:reduced filling(Diastasis)

Phase 1:**ATRIAL SYSTOLE**

Atrial systole occurs at the same time as ventricular diastole. It is initiated by the p wave of the electrocardiogram, which represents electrical depolarisation of the atria. Atrial depolarisation initiates contraction of the atrial musculature. It begins when atrial contracts. Just before the atrial contraction, the atrium is full of blood and the ventricle is partially full. The blood pressure inside each chamber is relatively low, but the atrial pressure is somewhat higher than ventricular pressure, ensuring that AV valve remains open. As the atrium contracts, the atrial pressure rise; then lowers as blood flow from atrium the ventricle. Ventricular pressure rises somewhat when it fills with blood, but not enough to exceed the pressure outside in the aorta. Therefore, during atrial systole, the aortic valve remains closed.

Phase2: **ISOVLUMETRIC CONTRACTION**

This phase of the cardiac cycle begins with the appearance of the QRS complex of the ECG, which represents ventricular depolarisation. The ventricle begins to contract and ventricular blood pressure rises rapidly. When ventricular pressure exceeds atrial pressure, AV valve closes. As the ventricle continues to contract, ventricular pressure rises, but it is not yet greater than the pressure outside the aortic valve, so the aortic valve remains closed. During this phase the ventricle is, for a moment, a closed chamber that contains an unchanging volume of blood. Duration of isovolumetric contraction is 0.05 second.

Phase3: **RAPID EJECTION**

When the ventricular pressure exceeds the blood pressure outside the aortic valve, the aortic valve opens. Blood is quickly and forcefully ejected into the aorta. This greatly decreases the volume of the ventricle and increases the blood flow and pressure in the aorta. The pressure in the aorta and ventricle quickly equalize and reach a peak. The duration of this period is 0.09 second.

Phase4:**REDUCED EJECTION**

Near the end of the ventricular systole, muscle fibres contract less forcefully because they are no longer stretched by a large volume. The ventricle continues to empty, but at a slow rate. When the ventricle begins to relax, ventricular pressure declines quickly. Blood pumped into the aorta remains under pressure, the aortic valve closes. The closing of the aortic valve marks the end of the ventricular systole and the onset of the ventricular diastole. Duration is 0.13s.

Phase5:**ISOVOLUMETRIC RELAXATION**

After the aortic valve closes, ventricular pressure decreases rapidly as the walls of the ventricle relax. The blood pressure has not yet dropped far enough to open the AV valve, so for the ventricle is a closed chamber with an unchanged volume of blood. The duration of isovolumetric relaxation period is 0.12s.

Phase6:**RAPID FILLING**

When ventricular pressure drops below atrial pressure, the AV valve opens and blood flows quickly from the atrium to the ventricle. Because blood is flowing passively, atrial pressure drops. Though the inflow of blood causes a slight increase in ventricular pressure, ventricular pressure continues to drop as the ventricles continue to relax. When the atrial and ventricular pressure begins to equalize, blood flow decreases. Duration is 0.11s.

Phase7:**REDUCED FILLING (DIASTASIS)**

Most of the blood that had accumulated in the atrium flow into the ventricle during the rapid filling. Blood continues to flow during the reduced filling phase, although relatively little enters the ventricle. The pressure inside both chambers remains constant. At the end of the reduced filling, both the atrium and ventricles are relatively full and the AV valve remains open. Duration of this phase is 0.19s.

CARDIAC OUTPUT

Cardiac output (CO) is the volume of blood pumped out by the heart in one minute. It is measured in litres per minute. On an average, a woman's heart pumps 41/min and a man's heart pumps 51/min. In an average lifetime, a human heart pumps 150 to 200 million litres of blood. Cardiac output is the product of two relatively independent factors, heart rate and stroke volume.

Cardiac output = Stroke volume x Heart rate

The cardiac output is primarily measured using Fick principle. In 1870, Aldoph Fick described the first method to estimate cardiac output in humans. He postulated the oxygen uptake in the lungs is entirely transferred to the blood and therefore that cardiac output can be calculated knowing oxygen consumption of the body and the difference in oxygen content between in atrial and mixed venous blood.

Cardiac output = O2consumption (ml/min) /AVO2 difference (ml O2/100ml blood) * 10

Another method is indicator dilution technique (thermodilution using a PA catheter). This technique is based on the principle that a single injection of a known amount of an indicator injected into the cental circulation mixes completely with blood and changes concentration as it flows to a more distal location. The change in the indicator concentration is plotted over time, the area under the curve is used to calculate CO.

HEART RATE

Heart rate (HR) is the number of time the heart beats per minute. The average heart rate for an adult at rest is 70 per minute (bpm). A healthy individuals heart rate can increase as much as three times normal rate, when the body requires greater cardiac output.

STROKE VOLUME (SV)

Stroke volume is the amount of blood pumped by left ventricle per contraction. The average stroke volume is 75ml per contraction. Stroke volume also increases in response to metabolic demand.

Heart rate and stroke volume are the dynamic factors in the changing relationships between the blood supply and metabolic demand. The volume of blood contained in the ventricles before the contraction is called end-diastolic volume (EDV). The volume of blood contained in the ventricles after the contraction is end-systolic volume. The stroke volume is expressed by,

Stroke volume = End-diastolic volume- End-systolic volume

Normal valves range from 75 to 133ml for EDV and from 20 to 50ml for ESV .The factors that determine stroke volume are:

- Preload
- Afterload
- Contractility

PRELOAD

Preload is another way of expressing EDV. Therefore, the greater the EDV, the greater the preload .A main factor is ventricular filling time. The faster the contractions are, the shorter the filling time and both the EDV and preload are lower. The relationship between ventricular stretch and contraction has been stated in the Frank- Starling mechanism which says that the force of contraction is directly proportional to the initial length of muscle fibre. So that the greater the stretch of the ventricle the greater the contraction. Any sympathetic

stimulation to the venous system will increase venous return to the heart and ventricular filling.

AFTERLOAD

The ventricles must develop a certain tension to pump blood against a resistance of the vascular system. This tension is called afterload. The thick walled arteries keep blood flowing under fairly high pressure. A ventricular contraction must therefore be strong enough to overcome arterial pressure and force the aortic valve open. The higher the arterial pressure, the stronger a ventricular contraction must be to open the aortic valve.

CONTRACTILITY

The ability of the myocardium to contract, (its contractility), controls the stroke volume which determines the end systolic volume. The greater contraction the greater the contraction the greater the stroke volume and the smaller the end systolic volume. Higher the cardiac output, a longer filling time between contraction and less resistance to filling during diastole. Oxygen availability to the myocardium affects contractility therefore the heart strengths decreases if there is not enough oxygen to meet the metabolic needs to the myocardial tissue.

HEART SOUNDS AND MURMERS

One of the simplest methods of assessing the heart's condition is to listen it using a stethoscope. In a healthy heart, there are only two audible heart sounds, called S1 and S2. The first heart sound S1, is the sound created by the closing of the atrioventricular valves during ventricular contraction and is normally described as "lub". The second heart sound, S2, is the sound of the semilunar valves closing during ventricular diastole and is described as "dub". Each sound consists of two components, reflecting the slight difference in time as the two valves close.S2 may split into two distinct sounds, either as a result of inspiration or different valvular or cardiac problems. Additional heart sounds may also be present and these give rise to gallop rhythms. A third heart sound, S3 usually indicates an increase in ventricular blood volume. A fourth heart sound S4 is referred to as an atrial gallop and is produced by the sound of blood being forced into a stiff ventricle. The combined presence of S3 and S4 give a quadruple gallop. Heart murmers are generated by turbulent flow of blood, which may occur inside or outside the heart. Murmurs may be physiological (benign) or pathological (abnormal). Abnormal murmurs can be caused by stenosis restricting the opening of a heart valve, resulting in turbulence as blood flows through it. Abnormal murmurs may also occur with valvular insufficiency (regurgitation), which allows backflow of blood when the incompetent valve closes with

only partial effectiveness. Different murmurs are audible in different parts of the cardiac cycle, depending on the cause of the murmur.

THE CONDUCTION SYSTEM OF THE HEART

The cardiac cycle is regulated by specialized areas in the heart wall that forms the conduction system of the heart. Two of these areas are tissue mass called nodes; the third is a group of fibers called the atrioventricular bundle. The sinoatrial node, which is located I the upper well of the right atrium an initiates the heart beat, is called the pacemaker. The second node, located in the ineratrial septum at the bottom of the right atrium, is called the atrioventricular node. The atrioventricular bundle, also known as the bundle of His, is located at the top of the interventricular septum; it has branches that extend to all parts of the ventricle walls. Fibers travel first down both sides of the interventricular septum in groups called the right and left bundle branches. Smaller Purkinje fibers then travel in a branching network throughout the myocardium of the ventricles.The order in which the impulses travel is as follows:

- The sinoatrial node generates the electric impulse that begins the heart beat.
- The excitation wave travels throughout the muscle of each atrium, causing it to contract.
- The atrioventricular node is stimulated. The relatively slower conduction through this node allows time for the atria tocontract and completes the filling of the ventricles.
- The excitation wave travels rapidly through the Bundle of His and then throughout the ventricular walls by means of the bundle branches and Purkinje fibers.

PRESSURE WAVES IN THE HEART

Hemodynamics is the study of the dynamic behavior of blood. As blood flows from chamber to chamber, as valves open and close, and as the myocardium contracts and relaxes, pressures are generated in various parts of the heart. These cardiovascular pressures can be measured and monitered through catheters whose tips are placed in the atria, pulmonary artery or systemic arteries

PRESSURE MEASURING DEVICES

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 rapid the sensing

membrane, the lower the sensitivity; conversely, the more flaccid the membrane. This general principle applies tomanometers currently in use.

FREQUENCY RESPONSE

A second crucial property of any pressure measurement is its frequency response. The frequency response of a pressure measurement is defined as the ratio of output amplitude to input amplitude over a range of frequencies of the input pressure wave. Frequency response of a catheter system is dependent on catheter's natural frequency and amount of damping. The higher the natural frequency of the system, the more accurate the pressure measurement at lower physiologic frequencies.

NATURAL FREQUENCY AND DAMPING

A third important concept is the natural frequency of a sensing membrane. Natural frequency is the frequency at which fluid oscillates in a catheter when it is tapped. Frequency of an input pressure wave at which the ratio of output/input amplitude of an undamaged system is maximal. Damping is the dissipation of the energy of oscillation of a pressure management system, due to friction.

STRAIN-GAUGE TRANSDUCER

Pressure measurement system today generally use electrical strain gauges based on the principle of the Wheatstone bridge. The strain gauge is a variable resistance transducer whose operation depends on the fact that when an electrical wire is stretched, its resistance to the flow of current increases. As long as the strain remains well below the elastic limit of the wire, there is a wide range within which the resistance is accurately proportional to the increase in length.

Strain-gauge pressure transducer increased pressure on diaphragm stretches, and increases resistance of G1 & G3 wires, while relaxing G2 & G4 wires. Voltage is applied across the wires and nnbalanced resistance leads to current flow across Wheatstonebridge. The Wheatstone bridge uses this principle in converting a pressure signal to an electrical signal.

TRANSDUCER/MONITOR ZERO AND CALIBRATION

Set- up of the transducer and monitor should be performed 15 to 20 minutes in advance to allow a sufficient warm -up period for stabilization. All transducers are affected by temperature changes, resulting in some drift to the zero baselines. Zero drift may occur within the monitor itself. For these reasons, rezeroing should be performed before infrequent or critical measurement readings, if there is a discrepancy between readings,

whenever there is a change in transducer level or a large environmental temperature shift, or at least every 4 hours. This is true for both disposable and reusable transducers.

- Position the air-reference stopwatch at the patient's midchest level or at the phlebostatic axis. The transducer may be placed on an IV pole, on an arm board next to the patient or directly on the patient's chest or arm.
- Remove the dead-ender cap and open the side arm of the air-reference stopcock.
- Select the appropriate scale on the monitor to correspond to the anticipated pressure range.
- Turn or press zero control knob on the monitor to obtain zero reading
- Adjust the tracing on the oscilloscope to the correct zero position.
- Press and hold the calibration knob on the monitor to read the precalibrated value.
- While depressing the calibration knob, adjust the tracing on the oscilloscope to the appropriate scale position.
- Close the air reference stopcock on the transducer dome and replace the dead-ender cap.

TRANSDUCER CALIBRATION

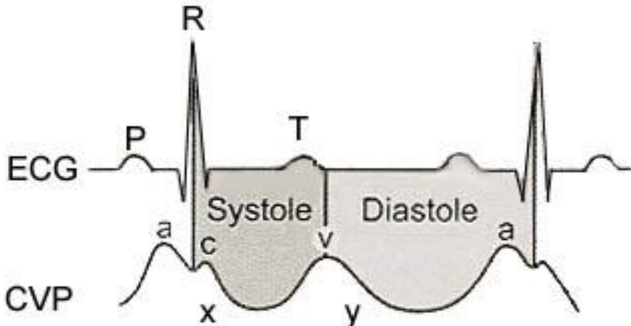
To be assured of obtaining accurate pressure measurements calibration of the transducer is important. Attach a disposable done to the transducer then press zero control knobs on the monitor. Attach a mercury manometer to one of the luer- lock fittings of the transducer dome, using short plastic tubing. Attach a hand bulb to the second luer-lock fitting on the transducer dome. Squeeze the hand bulb to 200mmHg and hold it. The digital readout should display 200mmHg and release the hand bulb and check for a return of the digital readout to zero. Repeat this at low pressures levels also. Remove the calibrating equipment and dome and attach the patient's dome to the transducer. Blood pressure is the force or pressure which the blood exerts on the walls of the blood vessels. The systemic arterial blood pressure, usually called simply arterial blood pressure, is the result of the discharge of blood from the left ventricle into the already full aorta. When the left ventricle contracts and pushes blood into the aorta the pressure produced within the arterial system is called the systolic blood pressure. In adults it is about 120 mmHg (millimetres of mercury) or 16 kPa (kilopascals). When complete cardiac diastole occurs and the heart is resting following the ejection of blood, the pressure within the arteries is called diastolic blood pressure. In an adult this is about 80 mmHg or 11 kPa. The difference between systolic and diastolic blood pressures is the pulse pressure. Arterial blood pressure is measured with a sphygmomanometer and is usually expressed in the following manner:

BP = 122 mmHg or BP

The blood pressure is maintained within normal limits by fine adjustments. Blood pressure is determined by cardiac output and peripheral resistance.

Blood pressure = cardiac output* peripheral resistance

Central venous pressure (CVP), also known as mean venous pressure (MVP) is the pressure of blood in the thoracic vena cava, near the right atrium of the heart. CVP reflects the amount of blood returning to the heart and the ability of the heart to pump the blood into the arterial system. CVP is often a good approximation of right atrial pressure (RAP), however the two terms are not identical, as right atrial pressure is the pressure in the right atrium.



CVP and RAP can differ when arterial tone is altered. This can be graphically depicted as changes in the slope of the venous return plotted against right atrial pressure (where central venous pressure increases, but right atrial pressure stays the same; VR = CVP-RAP). Direct measurement of arterial blood pressure is obtained via a peripheral artery catheter that is connected to a transducer and is a continuous display/recording device or monitor. Intra-arterial or direct blood pressure provides a continuous display of the arterial waveform along with measurements of mean arterial pressure, systolic and diastolic pressure. Indications for the use of direct arterial blood pressure monitoring include shock, critical illness, peripheral vasoconstriction, and intra-operative and post- operative monitoring of high-risk patients. In addition, arterial catheters allow for frequent monitoring of blood gas measurements.

RIGHT ATRIAL PRESSURE

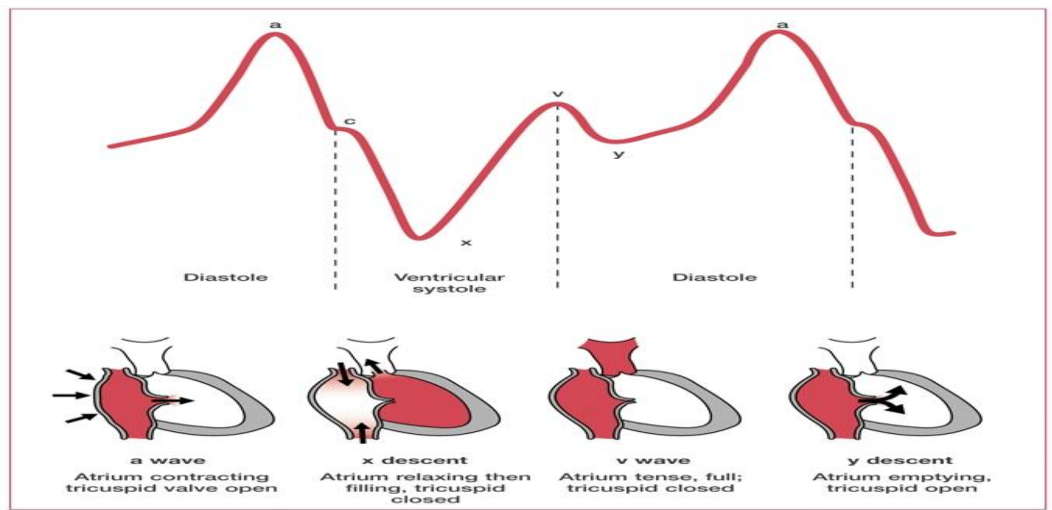
The right atrial pressure waveform normally has three positive waves, an “a” wave a “c” wave and a “v” wave, followed by the x, x’ and y descent. Normal RA pressure is 2 t 6 mmHg.

- **“a”** wave - Atrial systole

- **“c”** wave - Protusion of TV into RA
- **“x”** descent - Relaxation of RA. Downward pulling of tricuspid annulus by RV contraction.
- **“v”** wave - RV contraction.Height related to atrial compliance and amount of blood return. Smaller than a wave.
- **“Y”** descent - TV opening and RA emptying into RV

RIGHT VENTRICULAR PRESSURE

The RV waveform consists of systolic and diastolic phases. In the absence of outflow obstruction, the peak systolic pressure is normally the same as the systolic pressure, whereas the diastolic pressure falls to zero (+_5mm hg).



Normal RV pressure

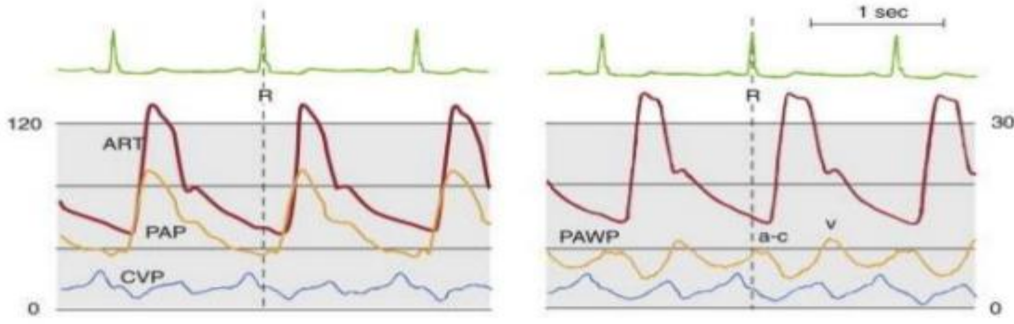
Systolic pressure : 20 to 30 mm Hg

Diastolic pressure : 0 to 5 mm Hg

End-diastolic pressure : 2 to 6 mm Hg

PULMONARY ARTERY PRESSURE

The PA waveform is the right sided circulation arterial pressure waveform and is comparable to the arterial waveform, consisting of systole, the dicrotic notch and diastole. Pressure readings obtained are usually the peak systolic, end-diastolic, and mean pressures.



Temporal relationships between systemic arterial pressure,pulmonary artery pressure, central venous pressure and pulmonary artery wedge pressure

-PAP upstroke precedes radial artery pressure upstroke
-Wedge pressure a wave follows ECG R wave

Normal PA pressure

Systolic pressure : 20 to 30 mm Hg

Diastolic pressure : 8 to 12 mm Hg

End-diastolic pressure : 10 to 20 mm Hg

PULMONARY ARTERIAL WEDGE PRESSURE

The PAWP is obtained during balloon inflation, which occludes PA branch and interrupts forward blood flow. Thus the tip of the catheter only measures that pressure distal to it, generated retrograde by the left atrium (LA). Therefore the PAWP reflects LA pressure and morphologically identical t the RA pressure waveform, consisting of an “a” wave, a “v” wave and occasionally a “c” wave. the “a” wave is absent in patients with atrial fibrillation

• “a” wave– Atrial systole

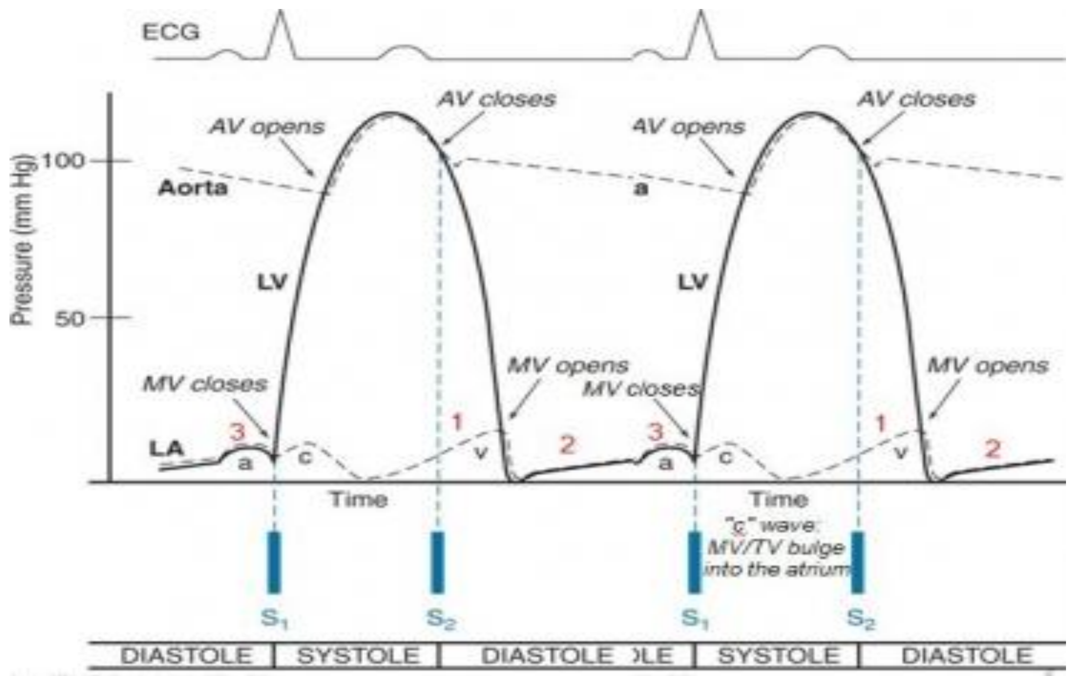
• “c” wave– Protrusion of MV into LA

• “x” descent– Relaxation of LA.Downward pulling of mitral annulus by LV contraction

• “v” wave– LV contraction.Height related to atrial compliance & amount of blood return. Higher than a wave

• “y” descent- MV opening and LA emptying into LV

LEFT VENTRICULAR PRESSURE



SHUNT DETECTION AND QUANTIFICATION

Detection, localiazation and quantification of intracardiac shunts are an integral part of the hemodynamic evaluation of patients with congenital heart diseases.

QP = Pulmonary blood flow

Qs = Systemic blood flow

QEP = Effective pulmonary blood flow

QES = Effective systemic blood flow

PVR = Pulmonary vascular resistance

SVR = Systemic vascular resistance

PVRI = Pulmonary vascular resistance index

SVRI = Systemic vascular resistance

$13.6 = O_2$ carrying capacity of a gram of Hb.

VO_2- oxygen consumption

CO= VO_2/(13.6*Hb(O_2 content of AO-O_2 content of PA))

BSA=√(weight(kg)*height(cm)/3600

Q_P=VO_2/(13.6*Hb*(PV – PA))

Q_S=VO_2/(13.6*Hb*(AO-MV))

Q_{EP}=VO_2/(13.6*(PV-MV))

Q_{EP}=Q_{ES}

Q_P-Q_{EP} for Left to Right shunt

Q_S-Q_{ES} for Right to Left shunt

PVR={PA_{mean}-PW_{mean}}/CO(l/min)

SVR={AO_{mean}-RA_{mean}}/(CO(l/min)

PVRI=PVR*BSA

SVRI=SVR*BSA

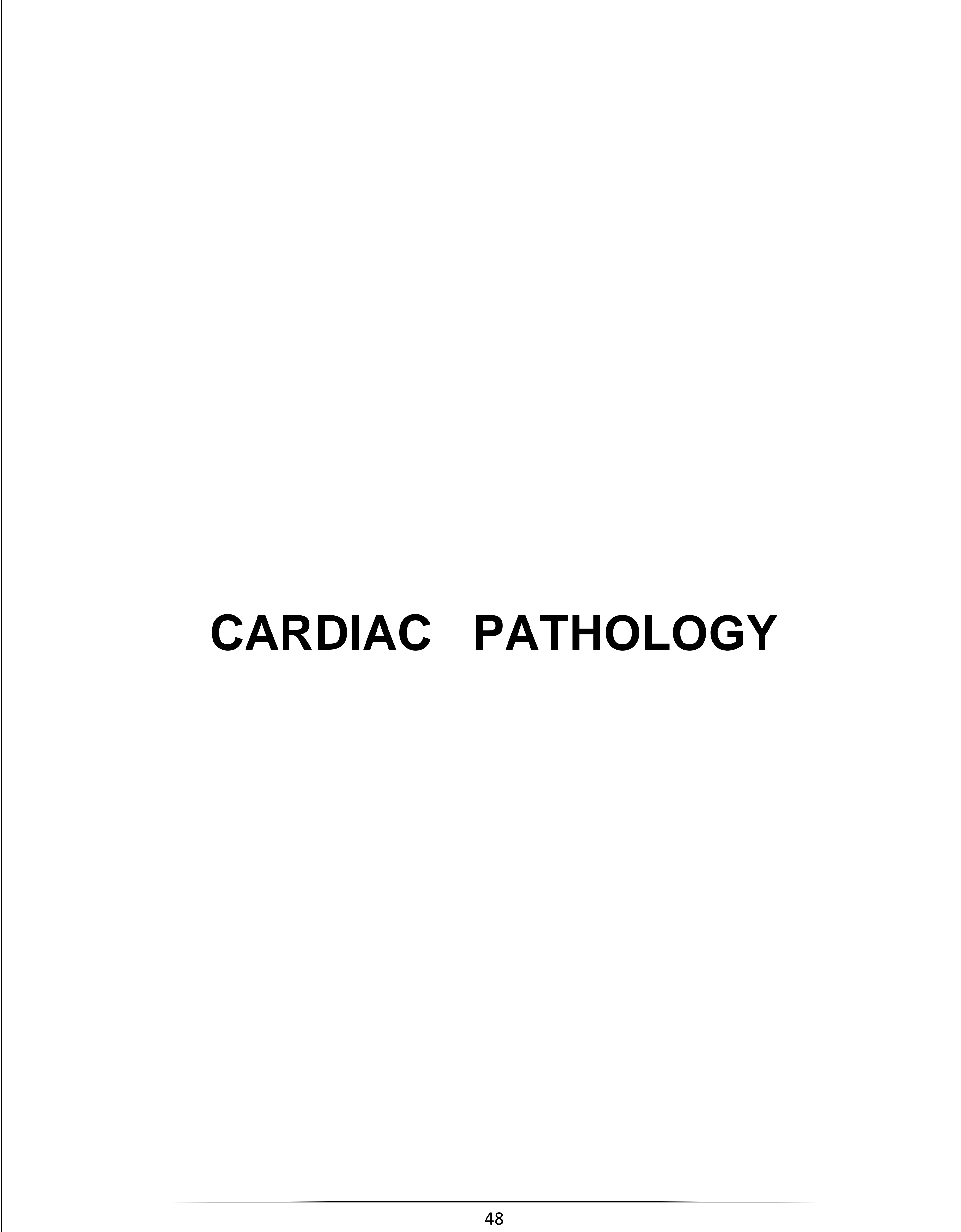
O_2 content of AO={Hb*13.6*O_2 saturation of AO}+{0.03*PO_2}

O_2 content of PA={Hb*13.6*O_2 saturation of PA}+{0.03*PO_2}

O_2 content of PV={Hb*13.6*O_2 saturation of PV}+{0.03*PO_2}

O_2 content of SVC={Hb*13.6*O_2 saturation of SVC}+{0.03*PO_2}

O_2 content of IVC={Hb*13.6*O_2 saturation of IVC}+{0.03*PO_2}



CARDIAC PATHOLOGY

PATTERNA AND CLASSIFICATION OF CARDIOVASCULAR DISEASE

Cardiovascular disease (**CVD**) is a class of diseases that involve the heart or blood vessels Cardiovascular disease includes coronary artery disease (CAD) such as angina and myocardial infarction (commonly known as a heart attack). Other CVDs include stroke, hypertensive heart disease,rheumatic heart disease, cardiomyopathy ,heart arruthmia, congenital heart disease, valvular heart disease, carditis, aortic aneurysms, peripheral artery disease, thromboembolic disease, and venous thrombosis.

CORONARY ARETRY DISEASE(CAD)

Coronary artery disease (**CAD**), also known as **ischemic heart disease** (**IHD**), is a group of diseases that includes: stable angina, unstable angina, myocardial infarction, and sudden cardiac death.[] It is within the group of cardiovascular diseases of which it is the most common type.[] A common symptom is chest pain or discomfort which may travel into the shoulder, arm, back, neck, or jaw. Occasionally it may feel like heartburn. Usually symptoms occur with exercise or emotional stress, last less than a few minutes, and get better with rest Shortness of breath may also occur and sometimes no symptoms are present. The first sign is occasionally a heart attack. Other complications include heart failure or an irregular heartbeat.[

Risk factors include: high blood pressure, smoking, diabetes, lack of exercise, obesity, high blood cholesterol, poor diet, depression, and excessive alcohol. The underlying mechanism involves reduction of blood flow and oxygen due to atherosclerosis of the arteries of the heart.[] A number of tests may help with diagnoses including: electrocardiogram, cardiac stress testing, coronary computed tomographic angiography, and coronary angiogram, among others.

Prevention is by eating a healthy diet, regular exercise, maintaining a healthy weight and not smoking.[Sometimes medication for diabetes, high cholesterol, or high blood pressure are also used.[There is limited evidence for screening people who are at low risk and do not have symptoms.Treatment involves the same measures as prevention. Additional medications such as antiplatelets including aspirin, beta blockers, or nitroglycerin may be recommended.] Procedures such as percutaneous coronary intervention (PCI) or coronary artery bypass surgery (CABG) may be used in severe disease. In those with stable CAD it is unclear if PCI or CABG in addition to the other treatments improve life expectancy or decreases heart attack risk.

Pathophysiology of CAD

Limitation of blood flow to the heart causes ischemia (cell starvation secondary to a lack of oxygen) of the myocardial cells. Myocardial cells may die from lack of oxygen and this is called a myocardial infarction (commonly called a heart attack). It leads to heart muscle damage, heart muscle death and later myocardial scarring without heart muscle regrowth. Chronic high-grade stenosis of the coronary arteries can induce

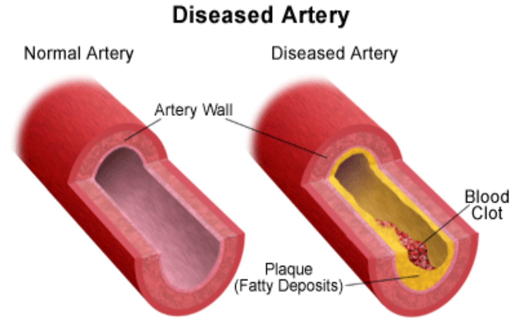
transient ischemia which leads to the induction of a ventricular arrhythmia, which may terminate into ventricular fibrillation leading to death.

Typically, coronary artery disease occurs when part of the smooth, elastic lining inside a coronary artery (the arteries that supply blood to the heart muscle) develops atherosclerosis. With atherosclerosis, the artery's lining becomes hardened, stiffened, and swollen with calcium deposits, fatty deposits, and abnormal inflammatory cells - to form a plaque. Deposits of calcium phosphates (hydroxyapatites) in the muscular layer of the blood vessels appear to play not only a significant role in stiffening arteries but also for the induction of an early phase of coronary arteriosclerosis. This can be seen in a so-called metastatic mechanism of calciphylaxis as it occurs in chronic kidney disease and haemodialysis .

Although these patients suffer from a kidney dysfunction, almost fifty percent of them die due to coronary artery disease. Plaques can be thought of as large "pimples" that protrude into the channel of an artery, causing a partial obstruction to blood flow. Patients with coronary artery disease might have just one or two **plaques**, or might have dozens distributed throughout their coronary arteries. A more severe form is chronic total occlusion (CTO) when a coronary artery is completely obstructed for more than 3 months.

MYOCARDIAL INFARCTION

Myocardial infarction (**MI**), commonly known as a **heart attack**, occurs when blood flow decreases or stops to a part of the heart, causing damage to the heart muscle. The most common symptom is chest pain or discomfort which may travel into the shoulder, arm, back, neck, or jaw. Often it is in the center or left side of the chest and lasts for more than a few minutes. The discomfort may occasionally feel like heartburn. Other symptoms may include shortness of breath, nausea, feeling faint, a cold sweat, or feeling tired. About 30% of people have atypical symptoms. Women more often have atypical symptoms than men Among those over 75 years old, about 5% have had an MI with little or no history of symptoms. An MI may cause heart failure, an irregular heartbeat, cardiogenic shock, or cardiac arrest.



ANGINA PECTORIS: Angina pectoris is a clinical syndrome of CAD resulting from transient myocardial ischemia. There are three overlapping clinical patterns of angina pectoris with some differences in their pathogenesis:

Stable or Typical Angina: Angina pectoris is said to be stable when its pattern of frequency, intensity, ease of provocation or duration does not change over a period of several weeks. Identification of activities that provoke angina and the amount of sublingual nitroglycerin required to relieve symptoms are helpful indicators of stability versus progression. A decrease in exercise tolerance or an increase in the need for nitroglycerin

suggests that the angina is progressing in severity or transitioning to an accelerating pattern.

Prinzmetal’s variant or Accelerating Angina: Angina pectoris is said to be accelerating when there is a change in the pattern of stable angina. This may include a greater ease of provocation, more prolonged episodes, and episodes of greater severity, requiring a longer recovery period or more frequent use of sublingual nitroglycerin. This suggests a transition and most likely reflects a change in coronaryartery blood flow and perfusion of the myocardium. This frequently portends unstable angina or an acute coronary syndrome such as an acute MI. Should a patient transition from a stable to accelerating pattern of angina, acute medical attention is warranted.

Unstable or Crescendo Angina: Unstable angina pectoris occurs when the pattern of chest discomfort changes abruptly. Signs of unstable angina are: symptoms at rest, a marked increase in the frequency of attacks, discomfort that occurs with minimal activity, and new-onset angina of incapacitating severity. Unstable angina usually is related to the rupture of an atherosclerotic plaque and the abrupt narrowing or occlusion of a coronary artery, representing a medical emergency with an incipient acute coronary syndrome and an MI to follow. Immediate medical attention is mandatory.

Risk Factors

- Hypertension
- Obesity
- Cigarette smoking
- Family history
- Stress
- Diabetes mellitus
- High blood lipids
- Rheumatoid arthiritis
- Heavy alcohol consumption

Heart failure

Heart failure (**HF**), often referred to as **congestive heart failure** (**CHF**), occurs when the heart is unable to pump sufficiently to maintain blood flow to meet the body's needs. Signs and symptoms commonly include shortness of breath, excessive tiredness, and leg swelling. The shortness of breath is usually worse with exercise, while lying down, and may wake the person at night.[1] A limited ability to exercise is also a common feature.Chest pain, including angina, does not typically occur due to heart failure.

Types of heart failure

- Left sided heart failure
- Right sided heart failure
- Biventricular heart failure

Left sided heart failure

Failure of the left side of the heart causes blood to back up (be congested) into the lungs, causing respiratory symptoms as well as fatigue due to insufficient supply of oxygenated blood. Common respiratory signs are increased rate of breathing and increased work of breathing (non-specific signs of respiratory distress). Rales or crackles, heard initially in the lung bases, and when severe, throughout the lung fields suggest the development of pulmonary edema (fluid in the alveoli). Cyanosis which suggests severe low blood oxygen, is a late sign of extremely severe pulmonary edema.

Additional signs indicating left ventricular failure include a laterally displaced apex beat (which occurs if the heart is enlarged) and a gallop rhythm (additional heart sounds) may be heard as a marker of increased blood flow or increased intra-cardiac pressure. Heart murmurs may indicate the presence of valvular heart disease, either as a cause (e.g. aortic stenosis) or as a result (e.g. mitral regurgitation) of the heart failure.

Backward failure of the left ventricle causes congestion of the lungs' blood vessels, and so the symptoms are predominantly respiratory in nature. Backward failure can be subdivided into the failure of the left atrium, the left ventricle or both within the left circuit. The patient will have dyspnea (shortness of breath) on exertion and in severe cases, dyspnea at rest. Increasing breathlessness on lying flat, called orthopnea, occurs. It is often measured in the number of pillows required to lie comfortably, and in orthopnea, the patient may resort to sleeping while sitting up. Another symptom of heart failure is paroxysmal nocturnal dyspnea: a sudden nighttime attack of severe breathlessness, usually several hours after going to sleep.

Right sided heart faillure

Right-sided heart failure is often caused by pulmonary heart disease (cor pulmonale), which is usually caused by difficulties of the pulmonary circulation, such as pulmonary hypertension or pulmonic stenosis.

Physical examination may reveal pitting peripheral edema, ascites, and liver enlargement. Jugular venous pressure is frequently assessed as a marker of fluid status, which can be accentuated by eliciting hepatojugular reflux. If the right ventricular pressure is increased, a parasternal heave may be present, signifying the compensatory increase in contraction strength.

Backward failure of the right ventricle leads to congestion of systemic capillaries. This generates excess fluid accumulation in the body. This causes swelling under the skin (termed peripheral edema or anasarca) and usually affects the dependent parts of the body first (causing foot and ankle swelling in people who are standing up, and sacral edema in people who are predominantly lying down).

Biventriucular heart failure

Dullness of the lung fields to finger percussion and reduced breath sounds at the bases of the lung may suggest the development of a pleural effusion (fluid collection between the lung and the chest wall). Though it can occur in isolated left- or right-sided heart failure, it

is more common in biventricular failure because pleural veins drain into both the systemic and pulmonary venous systems. When unilateral, effusions are often right sided.

Pathophysiology

Systolic dysfunction

Heart failure caused by systolic dysfunction is more readily recognized. It can be simplistically described as a failure of the pump function of the heart. It is characterized by a decreased ejection fraction (less than 45%). The strength of ventricular contraction is attenuated and inadequate for creating an adequate stroke volume, resulting in inadequate cardiac output.

Diastolic dysfunction

Insufficient relaxation of the heart muscles during diastole and hence decreased cardiac output. Patient has signs and symptoms of heart failure but ejection fraction is normal i.e. >45-50%. Common in elderly hypertensive patients.

CONGENITAL HEART DISEASE

- **Acyanotic congenital heart disease**

  Acyanotic congenital heart defects may be due to **obstructive** lesions (stenosis) or left-to-right shunts. Lesions with left-to-right shunts include atrial septal defect, ventricular septal defect, and patent ductus arteriosus. **Obstructive** lesions include**pulmonary** stenosis, aortic stenosis, and coarctation of the aorta.

- **Cyanotic congenital heart disease**

  Cyanotic congenital heart disease (CCHD) is a condition present at birth. CCHD causes low levels of oxygen in the blood. A common symptom is a bluish tint to the skin, called **cyanosis**.

Acyanotic CHD L->R	Increased pulmonary blood folw	1) Atrial Septal defect 2) Patent ductus arteriosus 3) Ventricular septal defect 4) Enodcardial cushion defect(AV canal defect) 5) Aorto-pulmonary window

	Decreased systomic blood flow	1) Coarctation of aorta 2) Aortic stenosis
Cyanotic CHD R->L	Increased pullmonary blood flow	1) Tranposition of great arteries 2) Partial or Total pulmonary venous 3) Return 4) Truncus Arteriosus 5) Hypoplastic left heart syndrome 6) Single ventricle 7) Interrupted Aortic arch
	Decreased pulmonary blood flow	1) Tetrology of Fallot 2) Tricuspid atresia 3) Pulmonary atresia 4) Pulmonary vascular obstraictive disease(Eisenmenger's syndrome) 5) Ebstein's anomaly 6) Double outlet right ventricle

PATENT DUCTUS ARTERIOSUS (PDA)

The ductus arteriosus is a communication between the left pulmonary artery and the aortic arch distal to the left subclavian artery. PDA is the failure of the fetal ductus arteriosus to close after birth.

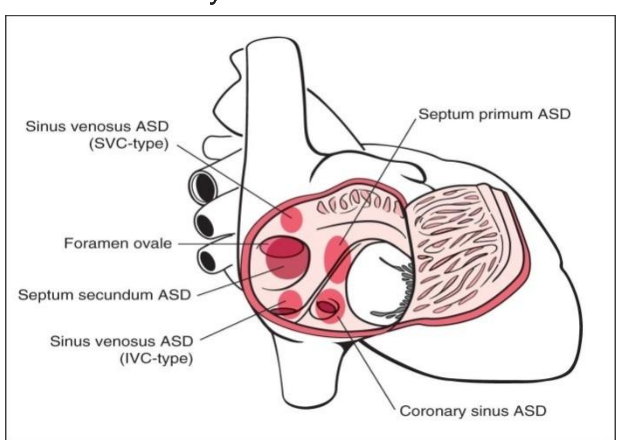
Left to right shunting of blood through the patent ductus results in an increase in pulmonary blood flow. The amount of blood that flows through the ductus, and the degree of symptoms exhibited, is determined by the differences in systemic and pulmonary vascular resistance, and in the circumference and the length of the PDA.

Patent Ductus Arteriosus

ATRIAL SEPTAL DEFECT (ASD)

Atrial septal defect (ASD) is a comnication or opening in the interatrial septum causing shunting of blood between two atria. There are 4 anatomic types:

1. Ostium primum - low in atrial septum, may involve a cleft mitral valve.
2. Ostium secundum - center of the atrial septum. Most common type of ASD.
3. Sinus venosus - high in the atrial septum. Associated with P-TAPVR.
4. Coronary sinus - large opening between the coronary sinus and left atrium.

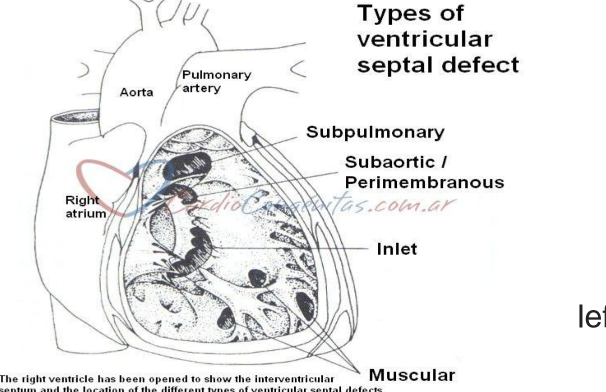


Because of higher pressure in the left atrium, blood is usually shunted from the left atrium across the ASD and into the right atrium. ASD's are restrictive when they are small enough to provide resistance to flow across the septum. ASD's are non-restrictive when the opening is large enough that equal pressures occur in both atria. If the child has a cyanotic congenital heart defect, an ASD can provide an important shunt that allows mixing of oxygenated and venous blood within the atria. This may be necessary to sustain life.

VENTRICULAR SEPTAL DEFECT (VSD)

A ventricular septal defect (VSD) is a communication (or multiple communications) between the right and left ventricles. VSD's are classified by their location in the ventricular septum. There are four anatomic types:

1. Perimembranous inlet VSD
2. Perimembranous outlet VSD
3. Mid Muscular VSD
4. Apical mascular VSD
5. Subaortic VSD
6. Subpulmonic VSD



The right ventricle has been opened to show the interventricular septum and the location of the different types of ventricular septal defects.

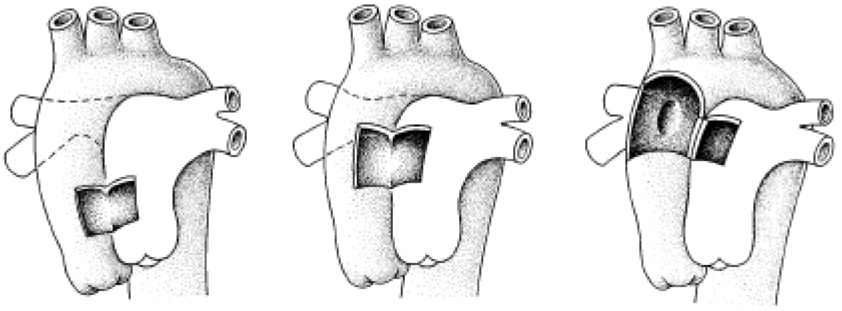
Because of higher pressure in the left ventricle, blood is usually shunted from the left ventricle, across the VSD into the right ventricle, and into the pulmonary circulation. The risk of pulmonary hypertension depends on the size and location of the defect and the amount of pulmonary vascular resistance. Small VSD's restrict the amount of blood shunting from left to right. Large VSD's are non- restrictive, therefore a much higher degree of shunting occurs, and blood flow to the lungs is increased.

AORTO-PULMONARY WINDOW

Aorto-pulmonary window is an opening between the ascending aorta and the main pulmonary artery. There must be two distinct and separate semilunar valves before this diagnosis can be made.

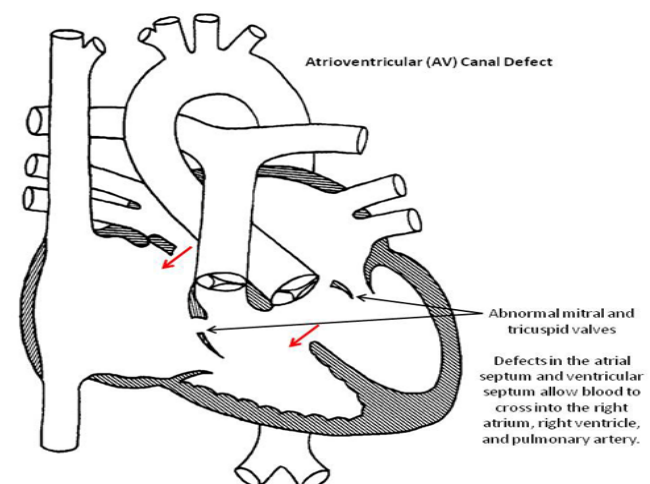
There is left to right shunt, as with a VSD or a persistent PDA. As the pulmonary resistance decreases in the days and weeks after birth, there is an increase in left to right shunting of blood leading to CHF. Without repair, and depending on the size of the defect, varying degrees of irreversible pulmonary vascular disease develops.

Aortopulmonary window may be classified into 3 **types**: **type** I (proximal) defects occur in the proximal part of **aortopulmonary** septum; **type** II (distal) defects occur in the distal part of the **aortopulmonary** septum adjacent to the right pulmonary artery; the **type** III defect is a combination of **types** I and II.



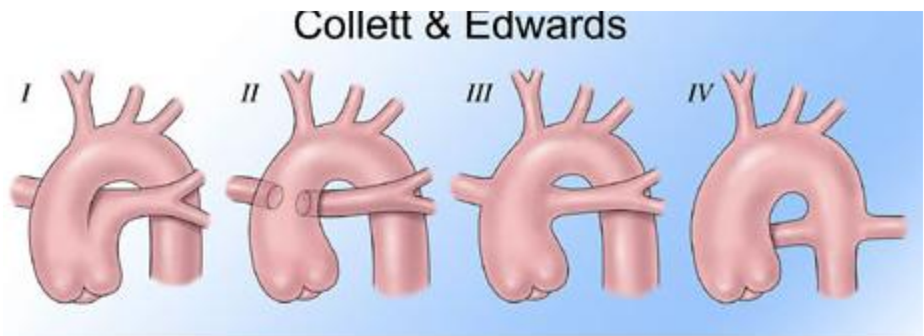
ENDOCARDIAL CUSHION DEFECT(ATRIO VENTRICULAR CANAL DEFECT, AVCD)

Atrioventricular canal (AVC) may be complete or partial. Complete AVC is a communication between both the atria and the ventricles, as well as failure of the tricuspid and mitral valve rings to develop separately. There are often clefts in the septal valve leaflets. The term "canal" is used because the common AV valve orifice and the deficient atrial and/or ventricular septal tissue create a large opening in the center of the heart between the atria and ventricles. There are varying degrees of abnormality in the atrial and ventricular septum and the AV valves, resulting in many variations of severity and symptoms. In the partial form of AVC, only an ASD is present along with a cleft in the mitral valve.



Shunting occurs at the atrial and ventricular levels in a left-to-right direction. The right atrium also receives blood from the left ventricle via the insufficient mitral valve and ASD. Blood can also enter the right atrium via the incompetent tricuspid valve. The excessive volume load on the right side of the heart and subsequent increased blood flow to the lungs, creates a high risk for development of pulmonary vascular disease and pulmonary hypertension.

TRUNCUS ARTERIOSUS



Truncus arteriosus is a rare congenital heart in which a single great vessel arises from the heart, giving rise to the coronary, systemic and pulmonary arteries. This single vessel contains only one valve (truncal valve). The truncus arteriousus overlies a VSD that is almost always seen in conjunction with this defect. There are four major types:

Type I – The most common; a single great vessel arises from the ventricles, and divides into an aorta and a main pulmonary artery (PA).

Type II – There is no PA main segment. The right and left PA's originate from the back of the truncus at the same level.

Type III – The right and left PA arise separately from the lateral aspect of the truncus. There is no main PA segment.

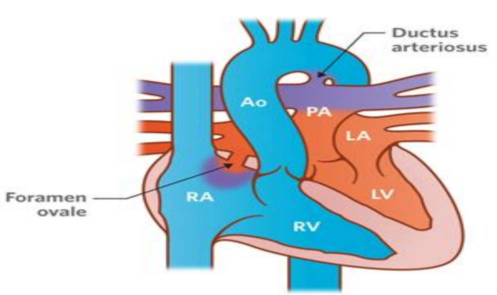
Type IV – No main PA. Pulmonary artery circulation is supplied from the systemic arterial circulation through collateral vessels of the bronchial arteries. This type is currently considered a form of Tetralogy of Fallot with pulmonary arteries.

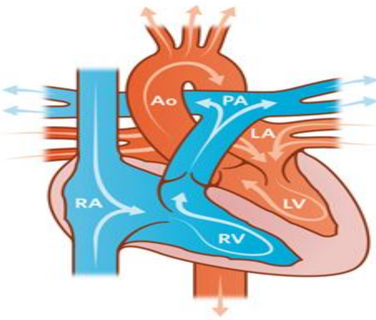
In virtually every case of truncus arteriosus there is a VSD and a single large semilunar valve. Oxygenated and de-oxygenated blood from both the right and left ventricles is ejected into the common great vessel. Both systemic and pulmonary circulations receive mixed venous blood from both ventricles. Pressures in both ventricles are identical. Pulmonary blood flow is increased, therefore pulmonary vascular disease and PAH will develop over time. The amount of blood flow to the lungs varies, depending on the nature of the pulmonary arteries.

D- TRANSPOSITION OF GREAT ARTERIES

In D-Transposition of the Great Arteries (D-TGA) the aorta arises from the anatomic right ventricle and the pulmonary artery arises from the anatomic left ventricle. The most common form of transposition occurs when the ventricles are normally positioned and the aorta is malposed anteriorly and rightward above the right ventricle. A VSD is present in 40% of patients with D-TGA. Abnormal coronary artery patterns present in 33% of cases.

Transposition of the great arteries





Normal heart and circulation

Blood flows from the RA to the RV and out through the aorta, carrying deoxygenated blood to the body. Blood flows from the LA to the LV and out through the PA, carrying oxygenated blood to the lungs. This results in two separate, parallel circulations that require mixing at the atrial, ventricular or ductus arteriosuslevel. The degree of desaturation present will depend primarily on the amount of mixing between systemic and pulmonary venous blood. The extent of inter-circulatory mixing in D-TGA depends on the number, size and position of the anatomic communications.

L-TRANSPOSITOIN OF THE GREAT ARTERIES(C-TGA)

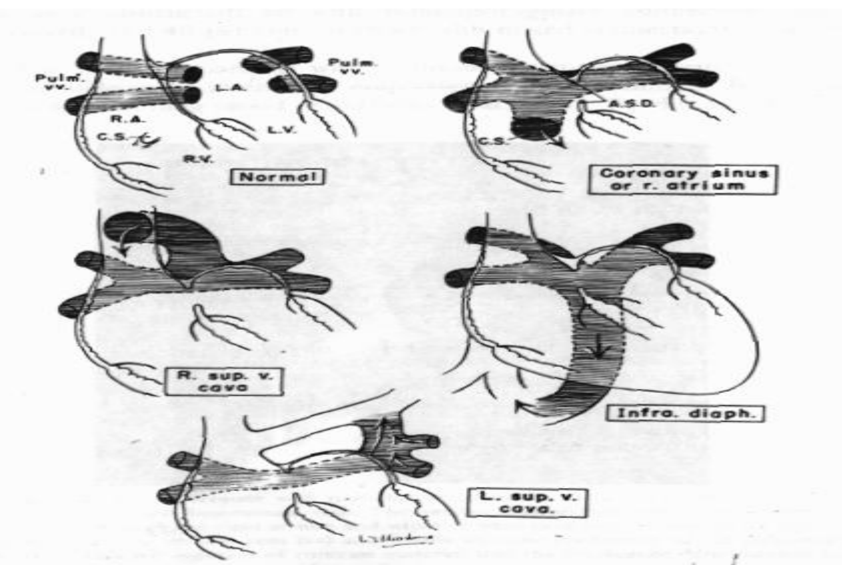
L-TGA, also known as congenitally corrected transposition. This malformation of the heart causes a reversal in the normal blood flow pattern because the right and left lower chambers of the heart are reversed. The L - transposition, however, is less dangerous than a d-transposition because the great arteries are also reversed. This "double reversal" allows the body to still receive oxygen-rich blood and the lungs to still receive the oxygen-poor blood. Most patients with L-TGA have associated cardiac defects (~80%), they include ventricular septal defect and valvar or subvalvar pulmonary stenosis. Patients are asymptomatic when L-TGA is not associated with other defects.

PARTIAL OR TOTAL ANOMALOUS VENOUS RETURN

Total Anomalous Pulmonary Venous Return (T-APVR) results from the failure of the pulmonary veins to join normally to the left atrium during fetal cardiopulmonary development. The pulmonary veins empty abnormally into the right atria via drainage into one of the systemic veins. The infant must have an ASD or a PFO to survive. There are four anatomic variations; defined according to the site of the anomalous connection of the pulmonary vein to the venous circulation and to the right atrium.

- **Supracardiac:** The pulmonary veins join a common pulmonary vein behind the left atrium. This common vein ultimately enters the superior vena cava and right atrium.

- **Cardiac**: The pulmonary venous blood drains into a common pulmonary vein that drains into the right atrium or coronary sinus.
- **Infradiaphramatic**: Pulmonary veins join to form a common pulmonary vein that descends below the diaphragm, through the portal system, then drains via the ductus venosus into the inferior vena cava and into the right atrium.
- **Mixe**d: Pulmonary veins join the systemic circulation at two different sites, or use any combination of systemic venous drainage.



Partial Anomalous Pulmonary Venous Return (P-APVR) results when one or more (but not all) pulmonary veins drain into the right atrium or its venous tributaries. There are two basic pathophysiologic states seen in T-APVR: unobstructed and obstructed. With unobstructed T-APVR, the entire pulmonary venous blood flow is returned to the systemic venous circulation, where there is mixing of the two venous returns in the right atrium. Mixing is virtually complete, each chamber of the heart receiving blood of almost identical oxygen concentration. Right atrial and ventricular dilation are present. The right to left shunting of blood across the PFO/ASD is essential for life. The amount of pulmonary blood flow is regulated by the pulmonary arteriolar resistance and by obstructions of the pulmonary veins. With obstructive T-APVR, the amount of pulmonary blood flow is reduced, causing cyanosis. This decreased flow, along with the systemic venous return, results in low arterial oxygen saturation and right heart failure.

INTERRUPTED AORTIC ARCH(IAA)

Interrupted Aortic Arch (IAA) refers to the congenital absence of a portion of the aortic arch. There are three types of IAA, and they are labelled according to the site of the interruption. IAA is always associated with a PDA and almost always with a VSD. Other cardiac defects may also be present.

- Type A: The ascending aortic arch is intact. The interruption occurs just distal to the left subclavian artery (the isthmus of aortic arch).

- Type B: The aorta is interrupted between the left carotid and the left subclavian artery, as a result the left subclavian artery arises from the descending aortic segment. This type is the most common.
- Type C: The aorta is divided between the innominate and the left carotid arteries. This type is extremely rare.

The ascending aorta receives blood from the left ventricle. The right ventricle perfuses the descending aorta through the ductus arteriosus. The infant will become acidotic and anuric when the ductus arteriosus closes. Ischemia to the liver, bowel and kidneys will occur. Profound ongoing acidosis will result in multi-system failure.

HYPOPLASTIC LEFT HEART SYNDROME

Hypoplastic Left Heart Syndrome (HLHS) is identified as a small, underdeveloped left ventricle usually with aortic and/or mitral valve atresia or stenosis and hypoplasia of the ascending aorta. A large ductus arteriosus is present. The right ventricle is dilated and hypertrophied, and an atrial septal defect is present. The entire systemic output is dependent on flow through the ductus arteriosus..

Because of its small size, the left ventricle is incapable of supporting the systemic circulation. Since outflow from the LV is either severely limited or absent, the systemic circulation must depend on mixed blood flow the pulmonary artery through the PDA. The foramen ovale stays open and left to right shunting occurs. The neonates right ventricle supports both pulmonary and systemic circulations. The PDA supplies the descending aorta with antegrade flow and the aortic arch with retrograde flow to the coronary circulation. Survival beyond birth is dependent on persistent patency of the ductus arteriosus to maintain systemic circulation.

SINGLE VENTRICLE

Both AV valves are connected to a main, single ventricular chamber (i.e., double-inlet ventricle), and the main chamber is in turn connected to a rudimentary chamber through the bulboventricular foramen. One great artery arises from the main chamber, and the other arises from the rudimentary chamber. In about 80% of cases, the main ventricular chamber has anatomic characteristics of the LV (i.e., double-inlet LV). Occasionally, the main chamber has anatomic characteristics of the RV (i.e., double-inlet RV). Rarely does the ventricle have an intermediate trabecular pattern without a rudimentary chamber (i.e., common ventricle). Also, both atria rarely empty through a common AV valve into the main ventricular chamber with either LV or RV morphology (i.e., common-inlet ventricle). Either D-TGA or L-TGA is present in 85% of cases. The most common form of single ventricle is double-inlet LV with L-TGA with the aorta arising from the rudimentary chamber.

PULMONARY STENOSIS

Pulmonary stenosis (PS) is a narrowing that obstructs blood flow from the right ventricle. It may be subvalvular, valvular, supravalvular or in the pulmonary arteries. When this presents in neonates, it is referred to as ‘critical pulmonary stenosis’.

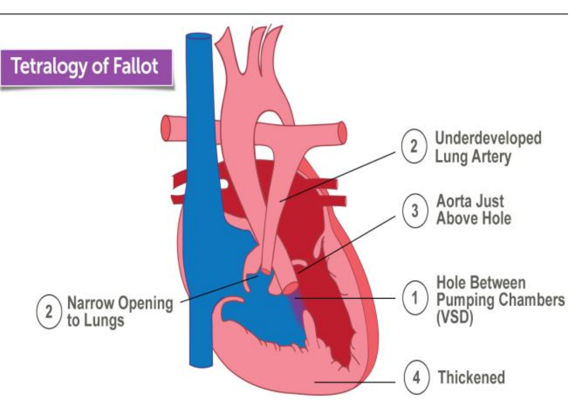
Pulmonary stenosis increases resistance to flow from the right ventricle. To maintain blood flow to the lungs, the right ventricle must generate higher pressures. The greater the pulmonary stenosis, the greater must be pressure. generated by the right ventricle. Because the pressure on the right side is higher, right ventricular hypertrophy is also present. Pulmonary stenosis may be mild, moderate or severe. When severe, the right ventricular hypertrophy may result in a right to left shunting through the foramen ovale

PULMONARY ATRESIA

There is no communication between the right ventricle and lungs so that the blood bypasses the right ventricle through an inter atrial septal defect. It then enters the lungs via patent ductus arteriosus.

TETROLOGY OF FALLOT

Tetrology of Fallot (TOF) is a congenital heart defect characterized by the association of four cardiac abnormalities; malaligned VSD, subpulmonary stenosis, overriding aorta and right ventricular hypertrophy. There is a wide spectrum of right ventricular outflow tract obstruction (RVOTO) in TOF. It may be subvalvular, valvular and/or supravalvular. Typically, there is hypoplasia of the right ventricular outflow tract, stenosis of the pulmonary valve and hypoplasia of the pulmonary annulus and trunk. The right and left pulmonary arteries are usually normal in size.



The hemodynamic changes and the degree of cyanosis that occur as a result of Tetrology of Fallot are directly proportional to the degree of subpulmonary stenosis (right ventricular outflow tract obstruction), and the resulting limitation to pulmonary blood flow. If RVOTO is mild, there is minimal shunting of blood from left to right across the VSD which may result in over circulation and signs of CHF. If severe RVOTO is present, a large amount of blood shunts from right to left, producing systemic arterial oxygen desaturation which can lead to severe hypoxemia and acidosis. As long as the ductus remains open, pulmonary blood flow is adequate.

TRICUSPID ATRESIA

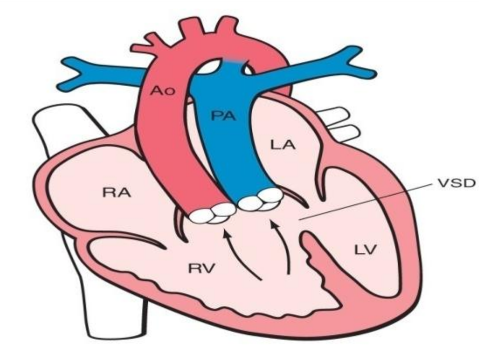
Tricuspid atresia is the failure of development of the tricuspid valve, resulting in a lack of direct communication between the right atrium and right ventricle. The floor of the right atrium is completely muscular. This defect is generally associated with a hypoplastic right ventricle and there is usually a ASD/PFO, VSD and/or PDA. Tricuspid atresia is often associated with some degrees of right ventricular outflow tract obstruction.

Because there is no direct communication between the right atrium and the right ventricle, blood flow is across the atrial septum from right to left via a PFO or an ASD. This results in complete mixing of desaturated systemic venous blood and fully saturated pulmonary venous blood at the atrial level, which is then ejected by the left ventricle. The workload of the left heart is increased due to the volume overload. The oxygen saturation levels are equal in the aorta and the PA.

TRICUSPID STENOSIS

Tricuspid valve dysfunction can result from morphological alterations in the valve or from functional aberrations of the myocardium. Tricuspid stenosis is almost always rheumatic in origin and is generally accompanied by mitral and aortic valve involvement. Most stenotic tricuspid valves are associated with clinical evidence of regurgitation that can be documented.

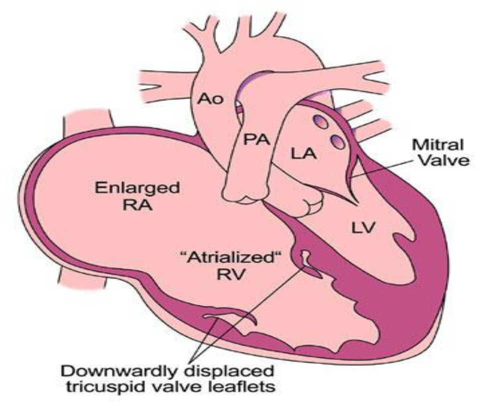
DOUBLE OUTLET RIGHT VENTRICLE (DORV)



Double Outlet Right Ventricle (DORV) spans a wide spectrum of physiology from Tetrology of Fallot to Transposition of the Great Arteries. DORV is a complex cardiac defect where both great vessels (aorta and pulmonary artery), either completely or nearly completely arise from the right ventricle. Most commonly they lie side by side in the same plane, and the aortic and pulmonary valves lie at the same level. A VSD is always present with DORV. The VSD may be subaortic, subpulmonary, or a combination of both. The classification of DORV is dependent on the location and type of VSD. The great vessels may be normally related or transposed. Some types of DORV have two adequately sized ventricles to allow a biventricular surgical repair. Other forms of DORV are associated with a severely underdeveloped left ventricle lending itself to a single ventricle pathway.

EBSTEIN'S ANOMALY

Ebstein's anomaly is a congenital heart defect in which the septal and posterior leaflets of the tricuspid valve are displaced towards the apex of the right ventricle of the heart. The annulus of the valve is still in the normal position. The valve leaflets, however, are to a varying degree, attached to the walls and septum of the right ventricle. A subsequent 'atrialization' of a portion of the morphologic right ventricle (which is then contiguous with the right atrium) is seen. This causes the right atrium to be large and the anatomic right ventricle to be small in size.An enlargement of the aorta may occur; an increased risk of abnormality is seen in babies of women taking lithium during the first trimester of pregnancy (though some have questioned this) and in those with Wolff-Parkinson-White syndrome.



COARCTATION OF THE AORTA

Coarctation of the aorta is a narrowing in the aortic arch. The coarctation may occur as a single lesion, as a result of improper development of the involved area of the aorta, or as a result of constriction of that portion of the aorta when the ductus arteriosus constricts. The coarctation is most often located near the ductus arteriosus; if narrowing is proximal to the ductus it is 'pre-ductal', if it is distal to theductus it is 'postductal'.

Aortic narrowing increases resistance to flow from the proximal to the distal aorta. As a result, pressure in the aorta proximal to the narrowing is increased and pressure in the aorta distal to the narrowing is decreased. Collateral circulation can develop in older children and adults to maintain adequate flow into the distal descending aorta.

AORTIC STENOSIS

Aortic Stenosis (AS) is a narrowing that obstructs blood flow from the left ventricle, leading to left ventricular hypertrophy and/or aortic insufficiency. AS may be mild, moderate, or severe. When this condition presents in neonates, it is referred to as 'critical aortic stenosis'. There are three types of AS, classified according to the location of the stenotic area:

Aortic valve stenosis (tricuspid)

Aortic valve stenosis (bicuspid)

- **Valvular:** Failure of the cusps to separate, creating fusion of the valve, or presence of a bicuspid valve instead of tricuspid. Accounts for 80% of AS.

- **Subaortic:** Caused by the formation of a fibrous ring with a narrowed central orifice below the aortic valve. It may be discrete or diffuse and in infants is seen as a component of a more complex lesion.
- **Supravalvular**: Caused by a fibromembranous narrowing of the aorta above the aortic valve and coronary arteries. Associated with Williams Syndrome.

Whenever there is obstruction to the left ventricular outflow tract, the left ventricle will generate greater pressure to maintain flow beyond the area of resistance. As a result, left ventricular hypertension that is proportional to the degree of aortic obstruction develops, and systemic cardiac output may be compromised. Left ventricular hypertrophy, aortic insufficiency, and failure can develop.

CARDIAC MALPOSITION

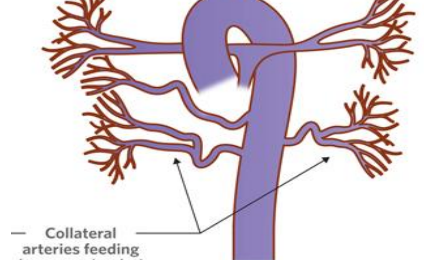
The term cardiac malposition indicates that the heart is abnormally located within the chest.

- Levocardia - The heart is located in the left chest (normal).
- Dextrocardia - The heart is located in the right chest.
- Mesocardia - The heart is located in the middle of the chest.

MAJOR AORTOPULMONARY COLLATERAL ARTERIES (MAPCA'S)

Major aortopulmonary collateral arteries (MAPCA's) are persistent tortuous fetal arteries that arise from the descending aorta and supply blood to pulmonary arteries in the lungs usually at the posterior aspect of hilum. Embryologically, the intersegmental arteries regress with the normal development of pulmonary arteries.

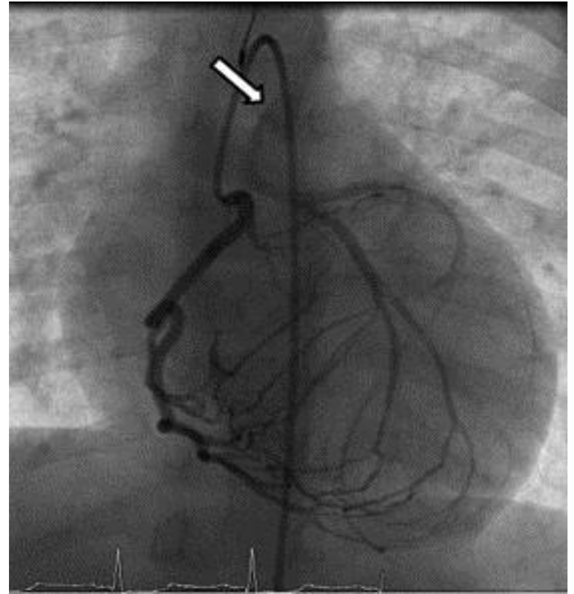
They may persist to supply the pulmonary arteries when there is no flow or very little flow into the pulmonary arteries from the right ventricle. If there is an alternate supply to the pulmonary arteries (e.g. patent ductus arteriosus), then the fetal arteries regress and such patient does not have MAPCAs supply to the pulmonary arteries (e.g. patent ductus arteriosus), then the fetal arteries regress and such patient does not have MAPCAs



ANOMALOUS LEFT CORONARY ARTERY FROM PULMONARY ARTERY(ALCAPA)

Anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) does not present prenatally because of the favorable fetal physiology that includes (1) equivalent pressures in the main pulmonary artery and aorta secondary to a nonrestrictive patent ductus arteriosus, and (2) relatively similar oxygen concentrations due to parallel

circulations. This results in normal myocardial perfusion and, therefore, no stimulus for collateral vessel formation between the right and left coronary artery systems is present.



Shortly after birth, as the circulation becomes one in series, pulmonary artery pressure and resistance decrease, as does oxygen content of pulmonary blood flow. This results in the left ventricular myocardium being perfused by relatively desaturated blood under low pressure, leading to myocardial ischemia; low pressure is more important in causing decreased myocardial perfusion.

Initially, myocardial ischemia is transient, occurring during periods of increased myocardial demands, such as when the infant is feeding and crying. Further increases in myocardial oxygen consumption lead to infarction of the anterolateral left ventricular free wall. This often causes mitral valve papillary muscle dysfunction and variable degrees of mitral insufficiency.

Collateral circulation between the right and left coronary systems ensues. Left coronary artery flow reverses and enters the pulmonic trunk due to the low pulmonary vascular resistance (coronary steal phenomena). As a result, left ventricular myocardium remains underperfused. Consequently, the combination of left ventricular dysfunction and significant mitral valve insufficiency leads to congestive heart failure (CHF) symptoms (eg, tachypnea, poor feeding, irritability, diaphoresis) in the young infant. Inadequate myocardial perfusion likely causes significant chest pain and these symptoms of myocardial ischemia may be misinterpreted as routine infantile colic.

DISORDERS OF THE LUNG AND PULMONARY CIRCULATION

:PULMONARY HYPERTENSION

The pulmonary circulation is a low pressure system. Pulmonary hypertension exists when the systolic pressure exceeds 30 mmHg. Pulmonary hypertension is a rare lung disorder in which the arteries that carry blood from the heart to the lungs become narrowed. As a result, the blood pressure in the areteries rises far above normal levels. This abnormally high pressure strains the right ventricle of the heart, causing it to expand in size. Overworked and enlarged, the right ventricle gradually becomes weaker and loses its ability to pump enough blood to the lungs. This could lead to the development of right heart failure.

PULMONARY VENOUS HYPERTENSION

It's increased blood pressure in the pulmonary veins (carrying blood away from the lungs, to the heart). Pulmonary venous hypertension is most often caused by congestive heart failure. A damaged mitral valve in the heart (mitral stenosis or mitral regurgitation) may contribute to pulmonary venous hypertension.

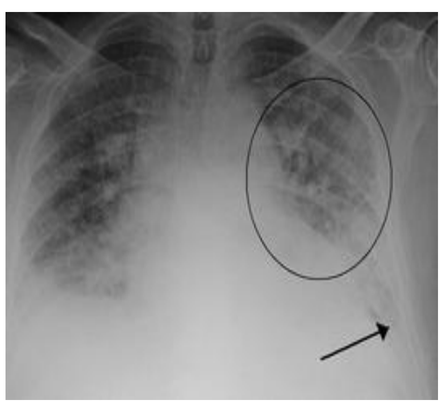
Pulmonary embolism, pulmonary thrombosis and pulmonary infraction are related conditions. Pulmonary embolisms are most often caused by deep vein thrombosis, a condition in which blood clots form in veins deep in the body. The blood clots that most often cause pulmonary embolisms begin in the legs or pelvis.

CHRONIC THROMBOEMBOLIC DISEASE

In rare cases, a blood clot to the lungs (pulmonary embolism) is never reabsorbed by the body. Instead, a reaction occurs in which multiple small bloodvessels in the lungs also become diseased. The process occurs slowly, and gradually affects a large part of the pulmonary arterial system

PULMONARY EDEMA

Pulmonary edema is fluid accumulation in the tissue and air spaces of the lungs.[1] It leads to impaired gas exchange and may cause respiratory failure. It is due to either failure of the left ventricle of the heart to remove blood adequately from the pulmonary circulation (cardiogenic pulmonary edema), or an injury to the lung parenchyma or vasculature of the lung (noncardiogenic pulmonary edema). Pulmonary edema, especially acute, can lead to fatal respiratory distress or cardiac arrest due to hypoxia. It is a cardinal feature of congestive heart failure.



Types

Cardiogenic

- Congestive heart failure which is due to the heart's inability to pump the blood out of the pulmonary circulation at a sufficient rate resulting in elevation in wedge pressure and pulmonary edema - this may be due to left ventricular failure, arrhythmias, or fluid overload, e.g., from kidney failure or intravenous therapy.
- Hypertensive crisis can cause pulmonary edema as the elevation in blood pressure and increased afterload on the left ventricle hinders forward flow and causes the elevation in wedge pressure a nd subsequent pulmonary edema.

Non-cardiogenic

- Neurogenic causes (seizures, head trauma, strangulation, electrocution).
- Acute respiratory distress syndrome.
- Negative pressure pulmonary edema in which a significant negative pressure in the chest (such as from an inhalation against an upper airway obstruction) ruptures capillaries and floods the alveoli.

Signs and Symptoms

The most common symptom of pulmonary edema is difficulty breathing, but may include other symptoms such as coughing up blood (classically seen as pink, frothy sputum), excessive sweating, anxiety, and pale skin. Shortness of breath can manifest as orthopnea (inability to lie down flat due to breathlessness) and/or paroxysmal nocturnal dyspnea (episodes of severe sudden breathlessness at night). These are common presenting symptoms of chronic pulmonary edema due to left ventricular failure.

RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

Rheumatic fever (**RF**) is an inflammatory disease that can involve the heart, joints, skin, and brain. The disease typically develops two to four weeks after a streptococcal throat infection. Signs and symptoms include fever, multiple painful joints, involuntary muscle movements, and occasionally a characteristic non-itchy rash known as erythema marginatum. The heart is involved in about half of cases. Damage to the heart valves, known as rheumatic heart disease (RHD), usually occurs after repeated attacks but can sometimes occur after one. The damaged valves may result in heart failure, atrial fibrillation and infection of the valves.

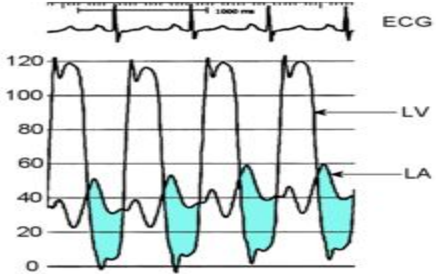
DISORDERS OF THE CARDIAC VALVES

Valvular diseases are various forms of congenital and acquired disease which cause valvular deformities. Many of them result in cardiac failure. Rheumatic heart disease is the most common form of acquired valvular disease. The valvular deformities may be two types: stenosis and insufficiency:Insufficiency and regurgitation are synonymous terms that describe an inability of the valve to prevent backflow of blood as leaflets of the valve fail to join (coapt) correctly. Stenosis is characterized by a narrowing of the valvular orifice that prevents adequate outflow of blood. Stenosis can also result in insufficiency if thickening of the annulus or leaflets results in inappropriate leaf closure..

MITRAL VALVE DISEASE

MITRAL VALVE STENOSIS:

Mitral stenosis is generally rheumatic in origin. Less common causes include bacterial endocarditis, LibmanSacks endocarditis, endocardial fibroelastosis and congenital parachute mitral valve.. The appearance of the mitral valve in stenosis varies according to the extent of involvement. Generally, the valve leaflets are diffusely thickened by fibrous tissue and/or calcific deposits, especially towards the closing margin. There are fibrous adhesions of mitral commissures and fusion and shortening of chordae tendineae. In less extensive involvement, the bases of the leaflets of mitral valve are mobile while the free margins have puckered and thickened tissue with narrowed orifice; this is called as '**purse-string puckering'.** The more advanced cases have rigid, fixed and immobile diaphragm-like valve leaflets with narrow, slit-like or oval mitral opening, commonly referred to as 'button-hole' or **'fish-mouth'** mitral orifice. Left atrial pressure (LAP) is equal to pulmonary venous pressure and this is equal to pulmonary capillary wedge pressure (PCWP).Mean LA pressure will be elevated for severe mitral stenosis having mitral valve area less than 1.0cm^2



"If pressure difference between PCWP or LA pressure to PA pressure is more than 10mmhg than it is considered as reactive pulmonary hypertension"

MITRAL REGURGITATION

All the causes of mitral stenosis may produce mitral insufficiency, RHD being the most common cause. In addition, mitral insufficiency may result from non- inflammatory calcification of mitral valve annulus (in the elderly), myxomatous transformation of mitral valve (floppy valve syndrome), rupture of a leaflet or of the chordate tendineae or of a papillary muscle. A few other condition scause mitral insufficiency by dilatation of the mitral ring such as in myocardial infarction, myocarditis and left ventricular failure in hypertension.

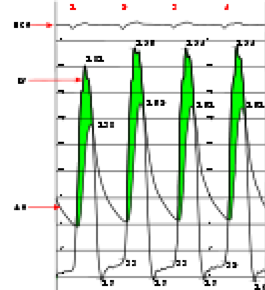
Over time, however, there may be decompensation and patients can develop volume overload (congestive heart failure). Symptoms of entry into a decompensated phase may include fatigue, shortness of breath particularly on exertion, and leg swelling. Also there may be development of an irregular heart rhythm known as atrial fibrillation..

AORTIC VALVE DISEASE

AORTIC STENOSIS

It is of two main types: non-calcific and calcific type,

1. Non-calcific aortic stenosis: The most common cause of non-calcific aortic stenosis is chronic RHD. Other causes are congenital valvular and subaortic stenosis and congenitally bicuspid aortic valve.



2. Calcific aortic stenosis: Calcific aortic stenosis is more common type. Various causes have been ascribed to it. The seinclude healing by scarring followed by calcification of aortic valve such as in RHD, bacterial endocarditis, Brucella endocarditis, Monckeberg's calcific aortic stenosis, healed congenital malformation and familial hypercholesterolaemic xanthomatosis.

The aortic cusps show characteristic fibrous thickening and calcific nodularity of the closing edges. Calcified nodules are often found in the sinuses of Valsalva. In rheumatic aortic stenosis, the commissures are fused and calcified, while in nonrheumatic aortic stenosis there is no commissural fusion.

AORTIC REGURGITATION

In about 75% of patients, the cause is chronicRHD. However, isoated aortic insufficiency is less often dueto rheumatic etiology. Other causes include syphilitic valvulitis, infective endocarditis, congenital subaortic stenosis (congenitallybicuspid aortic valve), myxomatous degeneration of aortic valve (floppy valve syndrome), traumatic rupture of the valve cusps, dissecting aneurysm, Marfan's syndrome and ankylosing spondylitis. The aortic valve cusps are thickened, deformed and shortened and fail to close. There is generally distension and distortion of the ring.

TRICUSPID VALVE DISEASE

TRICUSPID STENOSIS

Tricuspid stenosis is a narrowing or blockage of the tricuspid valve. Tricuspid stenosis causes the right atrium to become enlarged, while the right ventricle does not get enough blood. Tricuspid stenosis is usually associated with rheumatic fever. Often, people with tricuspid stenosis also have mitral stenosis.

TRICUSPID REGURGITATION

It means there is a backflow of blood from the lower-right chamber (the right ventricle) to the upper-right chamber (the right atrium), either because of a damaged tricuspid valve or an enlarged right ventricle. Blood is pushed back into the major veins instead of moving

forward into the lungs, where it can pick up oxygen. The main cause of tricuspid regurgitation is an enlarged right ventricle. This may be caused by any disorder that causes right ventricular failure, such as increased pressure in the pulmonary artery. Damage to the tricuspid valve may be caused by rheumatic fever.

PULMONARY VALVE DISEASE

Pulmonary valve disease is relatively uncommon. Pulmonary stenosis is usually of congenital origin. Other causes of pulmonary stenosis include rheumatic heart disease and malignant carcinoid. Obstruction of the outflow tract of the right ventricle may occur in hypertrophic cardiomyopathy and mediastinal tumours. Pulmonary regurgitation is usually secondary to pulmonary hypertension, but occasionally occurs as a consequence of infective endocarditis, as a complication of the surgical relief of pulmonary stenosis, as a congenital anomaly.

MYOCARDIAL DISEASE

Involvement of the myocardium occurs in three major forms of diseases already discussed—ischaemic heart disease, hypertensive heart disease and rheumatic heart disease. In addition, there are two other broad groups of isolated myocardial diseases:

MYOCARDITIS:

Inflammation of the heart muscle is called myocaditis. It’s usually forms part of a generalized infection (particularly viral) but can also be due to physical and chemical agents. Myocarditis is divided into four main etiologic types: Infective myocarditis, Idiopathic (Fiedler’s) myocarditis, Myocarditis in connective tissue disease and Miscellaneous types of myocrditis.

CARDIOMYOPATHY

The term cardiomyopathy refers to disease process involving heart muscle. The WHO definition of cardiomyopathy also excludes heart muscle diseases of known etiologies. However, the term cardiomyopathy has been loosely used by various workers for myocardial diseases of known etiology as well e.g. alcoholic cardiomyopathy, amyloid cardiomyopathy, ischaemic cardiomyopathy etc. This controversy is resolved by classifying all cardiomyopathies into two broad groups:

- Primary cardiomypathy: Disease confined to heart muscle and not arising from any other identifiable disease process.
- Secondary cardiomyopathy: heart muscle diseases arising as part of a more generalized disorder, which closely resemble the clinical characteristics of a primary cardiomyopathy.

Three types of functional impairment are obsereved in patients with cardiomyopathy:

- **Dilated:** The ventricles are dilated with impaired function.
- **Hypertrophic**: The left ventricle is inappropriately thickened, but contractile function is preserved.
- **Restrictive:** Diastolic filling is impaired.

PERICARDIAL FLUID ACCUMULATIONS

Accumulation of fluid in the pericardial sac may be watery or pure blood. Accordingly, it is of two types: hydropericardium (pericardialeffusion) an haemopericardium. The bulk of current evidence indicates that with**normal** cardiac volumes, the effective **pericardial pressure** ranges from 0–1 mm Hg to (at most) 3–4 mm Hg. The **pericardial** space between the parietal and visceral layers **normally** contains 15–50 mL of fluid, and the reserve volume of the **pericardium** is relatively small.

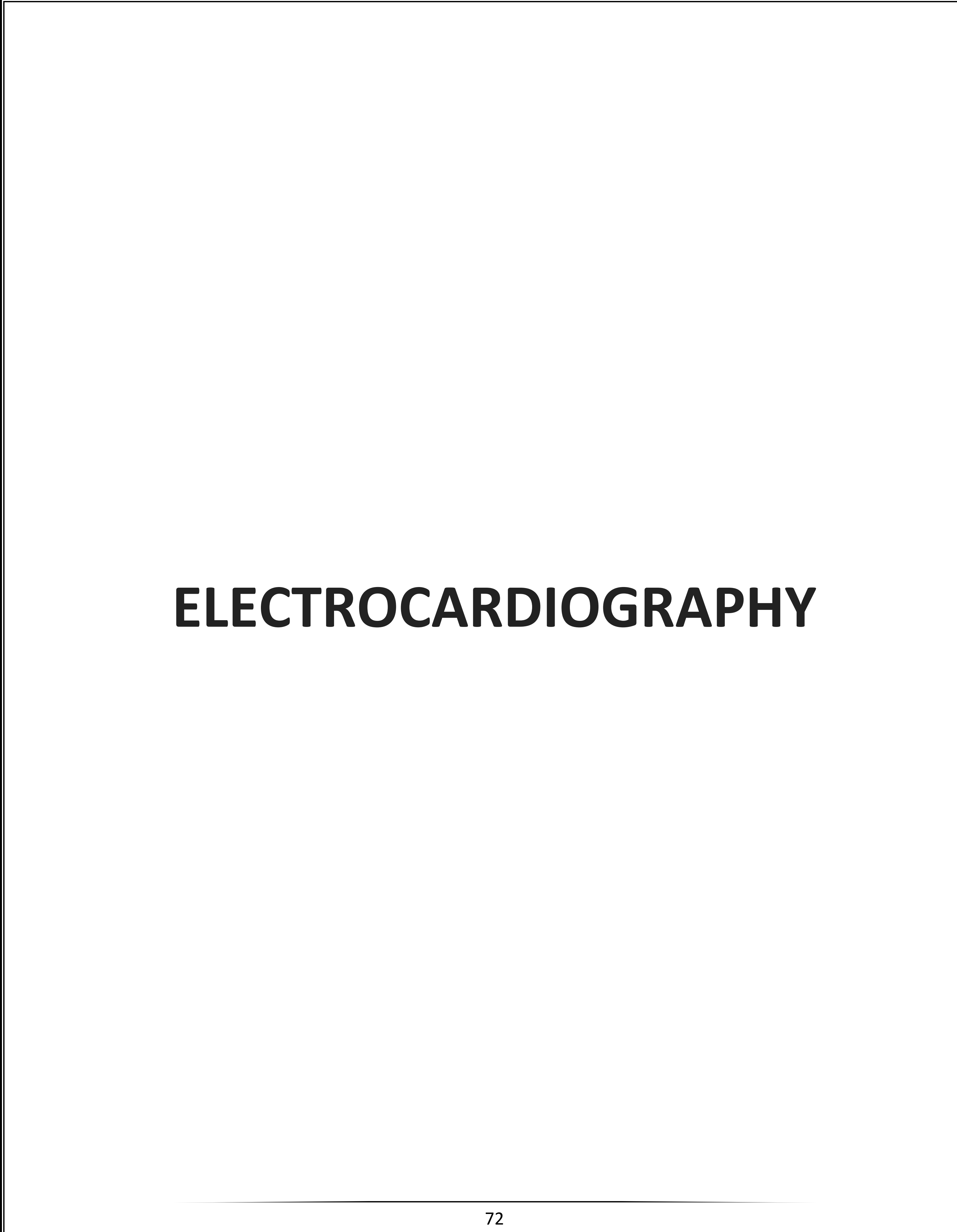
- Hydropericardium (pericardial effusion): Accumulation of fluid in pericardial cavity due to non inflammatory cause. Because of the limited amount of space in the pericardial cavity, fluid accumulation leads to an increased intrapericardial pressure which can negatively affect heart function. A pericardial effusion with enough pressure to adversely affect heart function is called **cardiac tamponade**.
- Haemopericardium: Accumulation of pure blood in the pericardial sac.

PERICARDITIS

Pericarditis is the inflammation of the pericardial layers and is generally secondary to diseases in the heart or caused by systemic diseases. Primary or idiopathic pericarditis is quiterare. Based on the morphologic appearance, pericarditis is classified into acute and chronic types, each of which may have several etiologies.

TUMOURS OF THE HEART

Cardiac tumours are rare. The commonest is the myxoma which occurs most frequently in the left atrium, but occasionally in the other chambers. It varies from 1 to 8 cm in diameter, and is usually attached by a pedicle to the atrial septum. Because its position may vary with posture, transient or complete obstruction of the mitral valve may result. The tumour may prolapsed into the left ventricle and cause mitral regurgitation.



ELECTROCARDIOGRAPHY

ELECTROCARDIOGRAPHY

It is the technique by which the electrical activities of the heart are studied. The spread of excitation through myocardium produces local electrical potential. The causes flow of small currents through the body fluids particularly extracellular fluid. These small currents can be picked up from the surface of the body by using suitable electrodes and recorded in the form of electrocardiogram. This technique was discovered by Dutch physiologist, Einthoven Willem who is called the father of ECG.

Electrocardiogram is a written record of the heart, while electrocardiograph is an instrument with which it is recorded. The same is true for telegram. The graph of voltage versus time produced by this noninvasive medical procedure referred to as an electrocardiogram.

THE CARDIAC ELECTRO PHYSIOLOGY

The most important muscle type in a vertebrate is cardiac muscle, which causes the heart beat. The cells that make up are called cardiac muscle cell or cardiomyocytes. Pacemaker cells, electrical conducting cells, myocardial cells these are the three types of cardiac cells. There are four basic properties of cardiac cells,

1. **Excitability:** It is the knack of cardiac cells to respond to a suitable amount of stimuli and produce an electrical potential, also called electrical impulse. This electrical impulse spreads across the heart, causing it to beat. It is due to the cellular action potential.

2. **Conductivity**: It is the ability of cardiac cells to transfer the action potential generated at the sino-atrial node from cell to cell.

3. **Contractility:** It is a cardiac muscle cell's ability to transform an electrical signal originating at sino-atrial node into mechanical action.

4. **Rhythmicity**: Rhythmicity is a property of cardiac muscle cells which describes their ability to contract regularly without the involvement of any nerves. Cardiac muscle cells do not need any neural stimulation to perform their functions. The action potentials originate in the sino-atrial node and do not involve any nervous activity. The action potential then moves through the entire heart again without any neural input. This property of cardiac cell is also called **automaticity**.

Action potential

The **cardiac action potential** is a brief change in voltage (membrane potential) across the cell membrane of heart cells.This is caused by the movement of charged atoms (called ions) between the inside and outside of the cell, through proteins called ion channels. The cardiac action potential differs from action potentials found in other types of

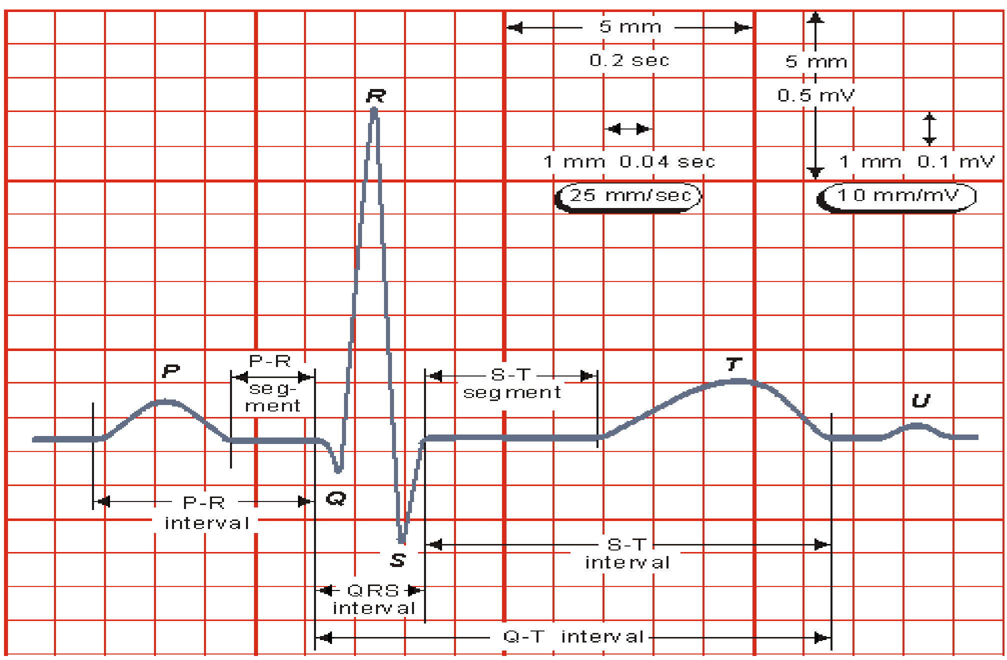
electrically excitable cells, such as nerves. Action potentials also vary within the heart; this is due to the presence of different ion channels in different cells.

The action potential in typical cardiomyocytes is composed of 5 phase (0-4).

Conduction pathway

Depolarization occurs in the sinoatrial (SA) node; current travels through internodal tracts of the atria to the atrioventricular (AV) node; then through Bundle of His, which divides into right and left bundle branches; left bundle branch divides into left anterior and posterior fascicles.

WAVES OF NORMAL ELECTROCARDIOGRAM



The waves of ECG recorded by Limb Lead II are considered as the typical waves. Normal electrocardiogram has the following waves namely P,Q,R,S and T.

'P' wave is the atrial **depolarization**

'QRS' complex is the ventricular **depolarizations**

'T' 'wave is the ventricular **repolarization**.

'P' WAVE

It is a positive wave and the first wave in ECG.

Duration:100ms.

Amplitude: 0.1 to 0.12mV.

‘QRS’ COMPLEX

Q wave is a small negative wave. It is continued as the tall R wave, which is a positive wave. R wave is followed by a small negative wave, the S wave.QRS complex is obtained because of the depolarization of ventricular musculature. Q wave is due to the depolarization of basal portion of interventricular septum. R wave is due to the depolarization of apical portion of interventricular septum and apical portion of ventricular muscle near the atrioventricular ring.

Duration : 80-100ms

Amplitude: Q wave: 0.1 to 0.2mV; R wave: 1mV; S wave: 0.4mV.

‘T’ WAVE

T wave is due to the repolarization of the ventricular musculature.

Duration:200ms

Amplitude: 0.3mV.

Atrial repolarization is not recorded as a separate wave in ECG because, it is

merged with QRS complex.

‘U’ WAVE

It is a rare and insignificant wave of ECG. It is supposed to be due to repolarization of papillary muscle.

INTERVALS AND SEGMENTS OF ECG

‘PR’ INTERVAL

It is the interval between the onset of P wave and the onset of Q wave. P-R interval signifies the atrial depolarization and conduction of the impulses through AV node.

Duration: 120-200ms

‘QT’ INTERVAL

It is the interval between the onset of Q wave and the end of T wave. Q-T interval indicates the ventricular reploralization.

Duration: 400-420ms

‘ST’ SEGMENTS

It is the time interval between the end of S wave and the onset of T wave. It is isoelectric. Elevation of S-T segment occurs in acute myocardial infarction.

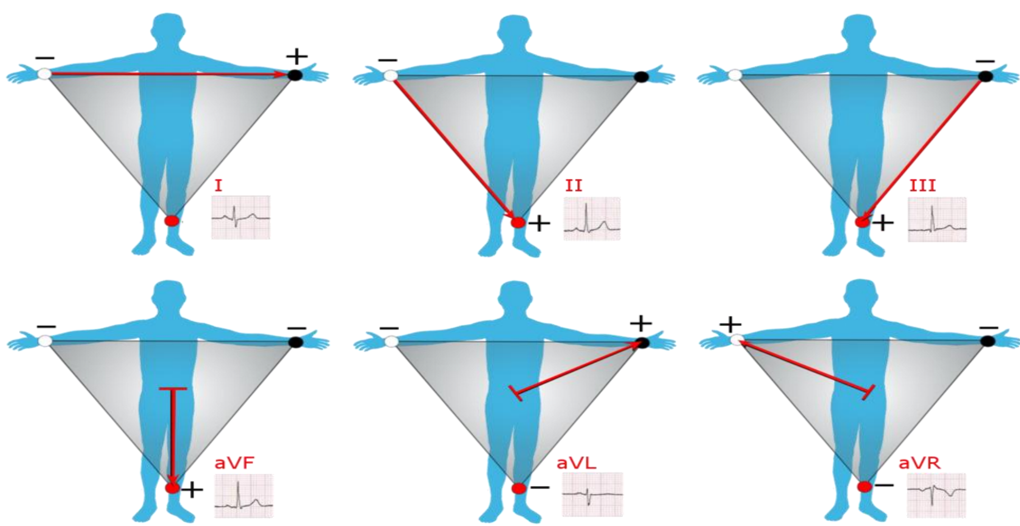
Duration: 80ms

‘RR’ INTERVAL

It is the time interval between two consecutive R waves. It is the duration of one cardiac cycle.

Duration: The normal duration of R-R interval is 60-100ms

ECG LEADS



Two arrangements, bipolar and unipolar leads.

Bipolar Lead: One in which the electrical activity at one electrode is compared with that of another. By convention, a positive electrode is one in which the ECG records a positive (upward) deflection when the electrical impulse flows toward it and a negative (downward) deflection when it flows away from it.

Unipolar Lead: One in which the electrical potential at an exploring electrode is compared to a reference point that averages electrical activity, rather than to that of another electrode. This single electrode, termed the exploring electrode, is the positive electrode.

Limb Leads: **I, II, III, aVR, aVL, aVF** explore the electrical activity in the heart in a frontal plane; i.e., the orientation of the heart seen when looking directly at the anterior chest.

Standard Limb Leads: I, II, III; bipolar, form a set of axes 60° apart

Lead I: Composed of negative electrode on the right arm and positive electrode on the left arm.Gives the voltage between left arm and right arm.

Lead I =LA-RA

Lead II: Composed of negative electrode on the right arm and positive electrode on the left leg. Gives the voltage between left leg and right arm.

Lead II=LL-RA

Lead III: Composed of negative electrode on the left arm and positive electrode on

the left leg.Gives the voltage between left leg and left arm.

Lead III=LL-LA

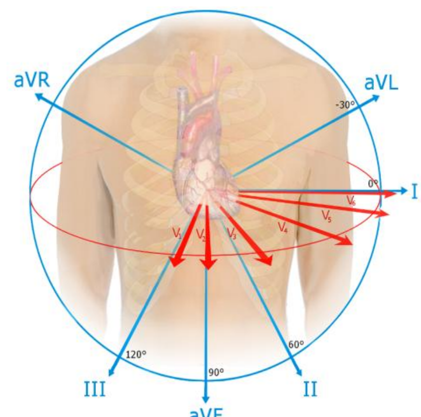
Augmented Voltage Leads:**aVR, aVL aVF**; unipolar ; form a set of axes 60° apart but are rotated 30° from the axes of the standard limb leads.

The common lead, Wilson's central terminal V_W, is produced by averaging the measurements from the electrodes RA, LA, and LL to give an average potential across the body:

$$V_W = 1/3(RA+LA+LL)$$

. In a 12-lead ECG, all leads except the limb leads are unipolar (aVR, aVL, aVF, V_1, V_2, V_3, V_4, V_5, and V_6). The measurement of a voltage requires two contacts and so, electrically, the unipolar leads are measured from the common lead (negative) and the unipolar lead (positive).

CHEST LEADS (PRECORDIAL LEADS)

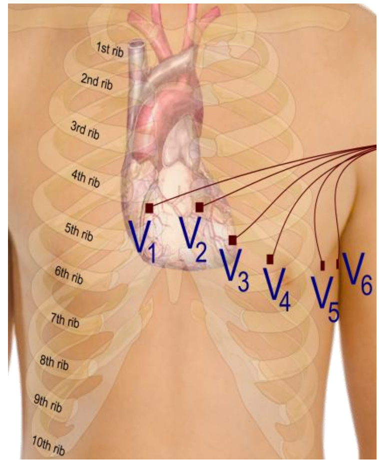


VI, V2, V3, V4, V5, V6, explore the electrical activity of the heart in the horizontal plane; i.e., as if looking down on a cross section of the body at the level of the heart. These are exploring leads.

Reference Point for Chest Leads: The point obtained by connecting the left arm, right arm, and left leg electrodes together.

Sternal angle (Angle of Louis): It is the anterior angle formed by the junction of the manubrium and body of the sternum. The sterna angle is a palpable clinical landmark. Which attach to the 2nd rib. It refers to the precordial leads position.

VI: Positioned in the 4th intercostal space just to the right of the sternum.



V2: Positioned in the 4th intercostal space just to the left of the sternum.

V3: Positioned halfway between V2 and V4.

V4: Positioned at the 5th intercostal space in the mid-clavicular line.

V5: Positioned in the anterior axillary line at the same level as V4.

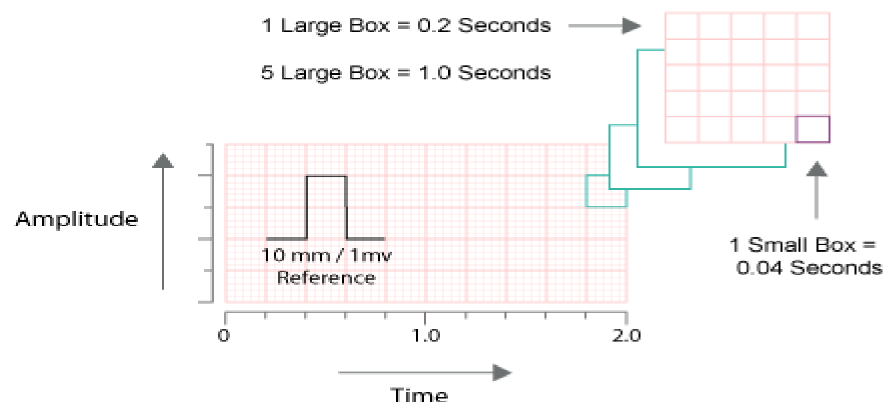
V6: Positioned in the mid axillary line at the same level as V4 and V5.

V1 and V2: Monitor electrical activity of the heart from the anterior aspect, septum, and right ventricle.

V3 and V4: Monitor electrical activity of the heart from the anterior aspect.

V5 and V6: Monitor electrical activity of the heart from the left ventricle and lateral aspect.

Dimensions of Grids on ECG Paper and calibration deflection:

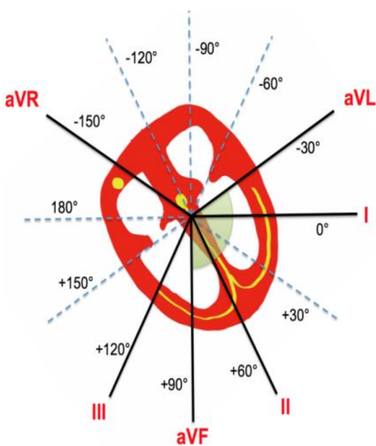


Horizontal axis represents time. Large blocks are 0.2 seconds in duration, while small blocks are 0.04 seconds in duration. Vertical axis represents voltage. Large blocks are 5mm, while small blocks represent 1mm. The movement of thepaper can be adjusted in **two speeds, 25 mm/sec and 50 mm/sec. Usually, the speed of the paper during** recording is fixed at 25 mm/sec.

The first deflection usually seen in the ECG is a square calibration wave. This should be exactly 10mm high from the top margin of the baseline to the top of thesquare wave.

THE CARDIAC AXIS

The average direction of spread of the depolarisation wave through the ventricles as seen from the front is called the 'cardiac axis'. It is useful to decidewhether the axis is in a normal direction or not. The direction of the axis can bederived most easily from the QRS axis complex in leads I, II and III.



A normal 11 o' clock-5 o'clock axis means that the depolarizing wave is spreading towards leads I, II and III, and is therefore associated with a predominantly upward deflection in all these leads; the deflection will be greater in lead II than in I and III. Normal axis is a mean vector between +90 degree and 0 degree.

Right axis Deviation: It is usually caused by right ventricular hypertrophy. In right axis deviation the direction of depolarisation is distorted to the right (1 – 7 o'clock). Right axis deviation is a mean vector of > +90 degree.

Left axis Deviation: It is the general direction of depolarisation becomes distorted to the left. This causes the deflection in lead III become –Ve. Left axis deviation is a mean vector

more negative than -30 degrees. Iso-Electric: When the deflection is perpendicular on the lead, this is called iso- electric. This QRS is neither +Ve or –Ve.

Classification	Angle	Notes
Normal	−30° to 105°	Normal
Left axis deviation	−30° to −90°	May indicate left ventricular hypertrophy, left anterior fascicular block, or an old inferior q-wave myocardial infarction
Right axis deviation	+105° to +180°	May indicate right ventricular hypertrophy, left posterior fascicular block, or an old lateral q-wave myocardial infarction
Indeterminate axis	+180° to −90°	Rarely seen; considered an 'electrical no-man's land'

Determining the axis of the mean vector: Check lead aVF.

if aVF is positive, check lead I

if lead I is positive, axis is normal.

if aVF is negative, check lead II.

if lead II is positive, it is in the gray zone.

if lead II is negative, there is left axis deviation.

HEART RATE

The rate of ventricular depolarization can be calculated by measuring the distance between each QRS complex. The approximate heart rate can be calculated using the formula:

heart rate= 300/number of large boxes between QRS complexes

Rate and rhythm

In a normal heart, the heart rate is the rate in which the sinoatrial node depolarizes as it is the source of depolarization of the heart. Heart rate, like other vital signs like blood pressure and respiratory rate, change with age. In adults, a normal heart rate between 60 and 100 beats per minute (normocardic) where in children it is higher. A heart rate less than normal is called **bradycardia** (<60 in adults) and higher than normal is **tachycardia** (>100 in adults). A complication of this is when the atria and ventricles are not in synchrony and the "heart rate" must be specified as atrial or ventricular (e.g., atrial rate in **atrial fibrillation** is 300–600 bpm, whereas ventricular rate can be normal (60–100) or faster (100–150)).

In normal resting hearts, the physiologic rhythm of the heart is **normal sinus rhythm (NSR**). Normal sinus rhythm produces the prototypical pattern of P wave, QRS complex, and T wave. Generally, deviation from normal sinus rhythm is considered a **cardiac arrhythmia**.

Artifacts

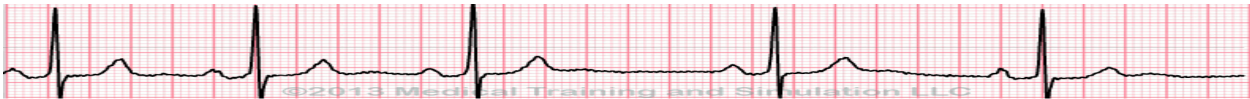
An EKG tracing is affected by patient motion. Some rhythmic motions (such as shivering or tremors) can create the illusion of cardiac dysrhythmia. Artifacts are distorted signals caused by a secondary internal or external sources, such as muscle movement or interference from an electrical device.

Improper lead placement (for example, reversing two of the limb leads) has been estimated to occur in 0.4% to 4% of all EKG recordings, and has resulted in improper diagnosis.

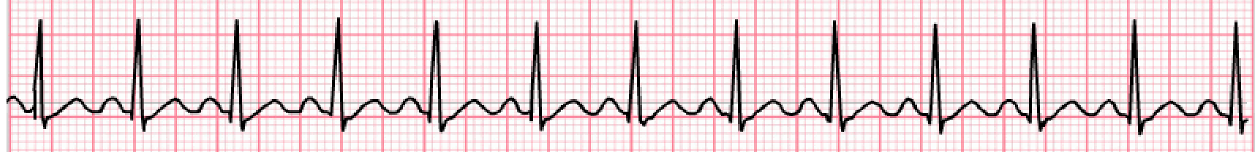
SINUS RHYTHM DISTURBANCE

Sinus rhythms originate in the sinoatrial node. Diagnosis of sinus rhythms requires examining leads II and aVR for the correct polarity of the P waves. The P wave is always positive in lead II and negative in lead aVR. A P wave will precede each QRS complex, and the P-R interval should be constant.

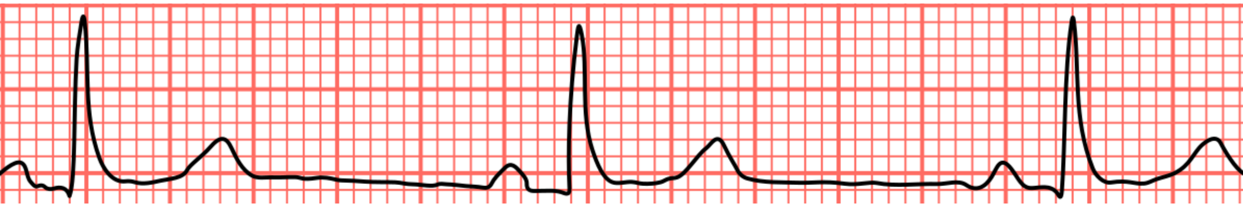
Sinus Arrhythmia: Under normal physiological conditions in a normal healthy person, the heart rate varies according to the phases of respiratory cycle. Heart rate increases during inspiration and decreases during expiration. This type of rhythmical increase and decrease in heart rate in relation to respiration is called sinus arrhythmia or respiratory sinus arrhythmia (RSA).



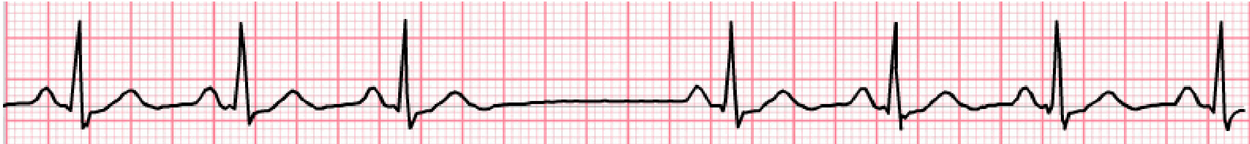
Sinus Tachycardia: Sinus rhythm with a rate >100 beats per minute. With fast rates, P waves may merge with preceding T waves and be indistinct. Can originate from the sinoatrial node, atrial muscle, or atrioventricular junction. Often referred to as supraventricular tachycardia without specifying site of origin.



Sinus Bradycardia: Sinus rhythm with a rate <60 beats per minute.



Sinoatrial Block: Refers to failure of the sinus node to function for one or more beats. In this condition, there are simply one or more missing beats; i. e., there are no P waves or QRS complexes seen. Fortunately, when the sinus fails to function for a significant period of time (sinus arrest), another part of the conduction system usually assumes the role of pacemaker. These pacing beats are referred to as escape beats and may come from the atria, the atrioventricular junction, or the ventricles.

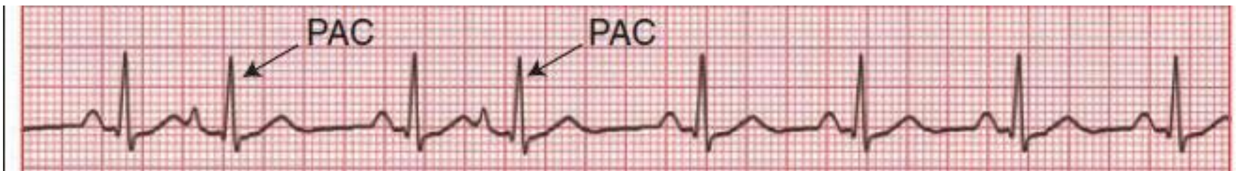


Sick Sinus Syndrome: In elderly people, the sinus node may undergo degenerative changes and fail to function effectively. Periods of sinus arrest, sinus tachycardia, or sinus bradycardia may occur. It may caused by age or coronary artery disease.

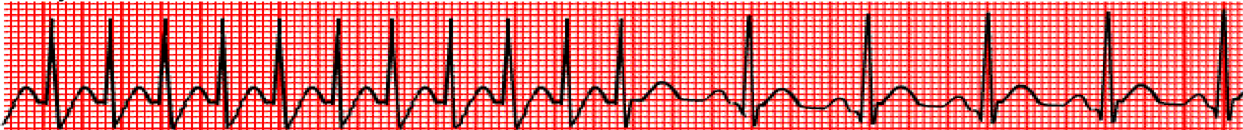
ATRIAL ARRHYTHMIAS

Include premature atrial beats, paroxysmal atrial tachycardia, multi-focal atrial tachycardia, atrial flutter, and atrial fibrillation. Because the stimuli arise above the level of the ventricles, the QRS pattern usually is normal.

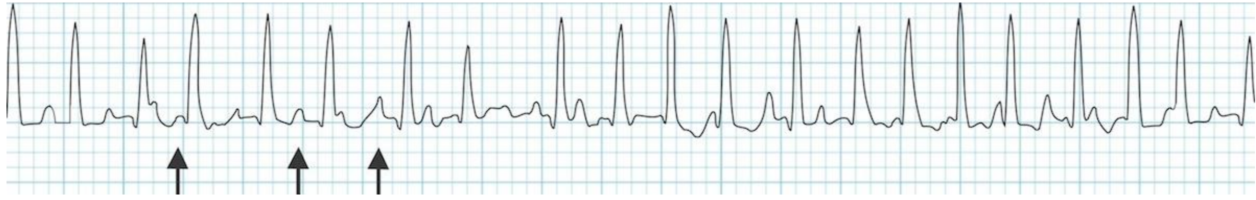
Premature Atrial Contraction (PAC): An ectopic beat arising somewhere in either atrium but not in the sinoatrial node. Occurs before the next normal beat is due, and a slight pause usually follows. The P wave may have a configuration different from the normal P wave and may even be of opposite polarity. The P-R interval may be shorter than the normal..



Paroxysmal Atrial Tachycardia (PAT): PAT usually occurs at a regular rate, most commonly between 150 and 250 beats per minute. P waves may or may not be sinus tachycardia.

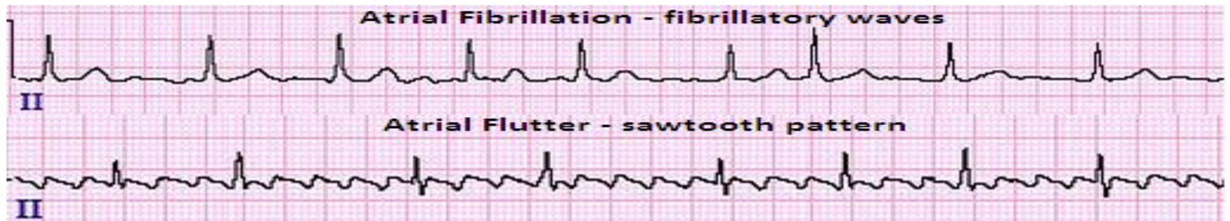


Multi-Focal Atrial Tachycardia (MFAT): Results from the presence of multiple, different atrial pacemaker foci. This rhythm disturbance is characterized by a tachycardia with beat-to-beat variation in P wave morphology.



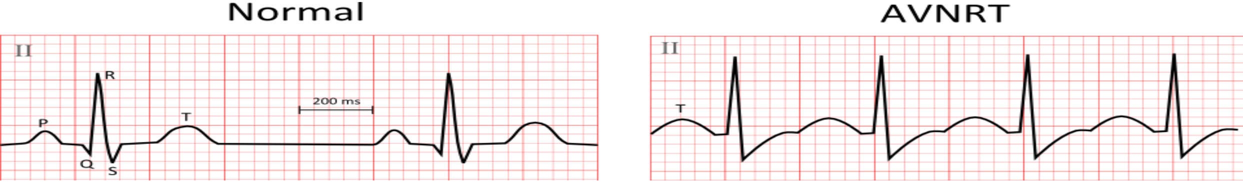
Atrial Flutter: An ectopic atrial rhythm. Instead of P waves, characteristic sawtooth waves are seen. The atrial rate in atrial flutter is usually about 300 beats per minute. However, the AV junction is unable to co rate is less-usually 150, 100, 75, and so on, beats per minute. Atrial flutter with a ventricular rate of 150 beats per minute is called a 2:1 ratio because of the atrial rate (300) to the ventricular rate(150).

Atrial Fibrillation: Here the atria are depolarized at an extremely rapid rate, greater than 400 beats per minute. This produces a characteristic wavy baseline pattern instead of normal P waves. Because the AV junction is refractory to most of the impulses reaching it, it only allows a fraction of them to reach the ventricles. The ventricular rate, therefore, is only 110-180 beats per minute. Also characteristic of atrial fibrillation is a haphazardly irregular ventricular rhythm.



Atrioventricular Nodal Reentral Tachycardia: It is a common type of supraventricular tachycardia. It accounts for the many of the fast heart rate that start in the upper part of the heart. AVNRT can cause symptoms of any age. It typically is not a result of other forms

of heart disease. It is caused by an abnormal or extra electrical pathway in the heart a kind of “short circuit”.

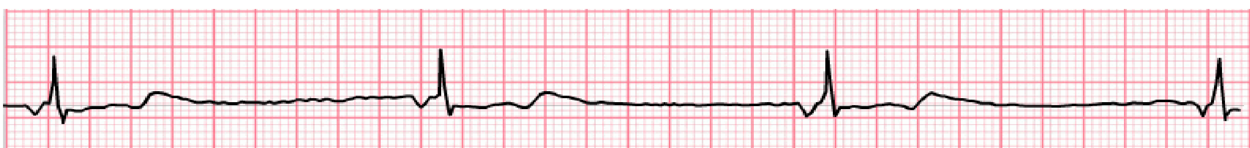


Atrioventricular Reentrant Tachycardia

During AVRT, the electrical signal passes in the normal manner from the AV node into the ventricles. Then, the electrical impulse pathologically passes back into the atria via the accessory pathway, causing atrial contraction, and returns to the AV node to complete the reentrant circuit (see figure). Once initiated, the cycle may continue causing the heart to beat faster than usual. Initiation of AVRT may be through a premature impulse of atrial, junctional, or ventricular origin.

JUNCTIONAL RHYTHMS

The three types of junctional rhythms are premature junctional contractions, junctional tachycardia, and junctional escape rhythms. Junctional rhythms arise in the AV junction. P waves, when seen, are opposite their normal polarity. They are called retrograde P waves. These P waves may precede, be buried in, or follow the QRS complex. Since the stimulus arises above the level of the ventricles, the QRS complex is usually of normal configuration.



ATRIOVENTRICULAR BLOCKS(AV BLOCK)

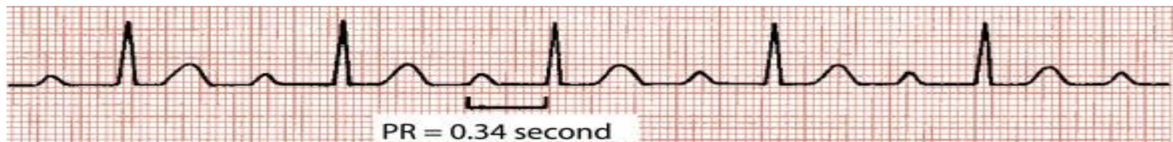
Atrioventricular block (**AV block**) is a type of heart block in which the conduction between the atria and ventricles of the heart is impaired. Under normal conditions, the sinoatrial node (SA node) in the atria sets the pace for the heart, and these impulses travel down to the ventricles. In an AV block, this message does not reach the ventricles or is impaired along the way. The ventricles of the heart have their own pacing mechanisms, which can maintain a lowered heart rate in the absence of SA stimulation.

The causes of pathological AV block are varied and include ischaemia, infarction, fibrosis or drugs, and the blocks may be complete or may only impair the signaling between the SA and AV nodes. Certain AV blocks can also be found as normal variants, such as in

athletes or children, and are benign.Strong vagal stimulation may also produce AV block. The cholinergic receptor types affected are the muscarinic receptors.

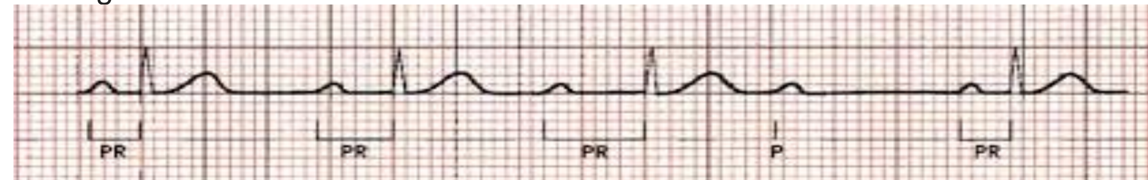
There are three types:

- **First-degree atrioventricular block** - The heart's electrical signals move between the upper and lower chambers of the heart.PR interval greater than 0.20sec.



- **Second-degree atrioventricular block** - The heart's electrical signals between the upper and lower signals of the heart are slowed by a much greater rate than in first-degree atrioventricular block. **Type 1 (Mobitz 1, Wenckebach**): Progressive prolongation of PR interval with dropped beats (the PR interval gets longer and longer; finally one beat drops) . **Type 2 (Mobitz 2, Hay):** PR interval remains unchanged prior to the P wave which suddenly fails to conduct to the ventricles.

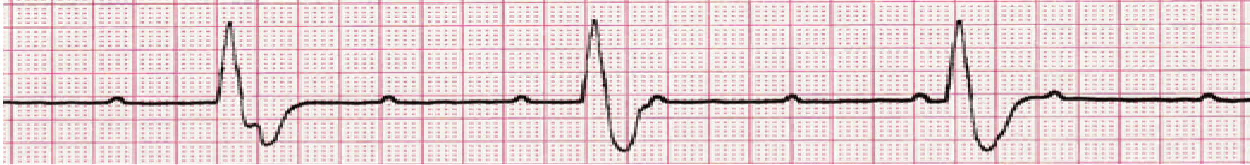
  - ✓ **Mobitz I(Wenckebach**) is characterized by a reversible block of the AV node. When the AV node is severely blocked, it fails to conduct an impulse. Mobitz I is a progressive failure. Some patients are asymptomatic; those who have symptoms respond to treatment effectively. There is low risk of the AV block leading to heart attack.



  - ✓ **Mobitz II** is characterized by a failure of the His-Purkinje cells resulting in the lack of a supra ventricular impulse. These cardiac His-Purkinje cells are responsible for the rapid propagation in the heart. Mobitz II is caused by a sudden and unexpected failure of the His-Purkinje cells. The risks and possible effects of Mobitz II are much more severe than Mobitz I in that it can lead to severe heart attack. The characteristic ECG picture is that of a series of non-conducted P waves; e.g., 2:1, 3:1, 4:1, block.

Third-degree atrioventricular block - No association between P waves and QRS complexes. The heart's electrical signals are slowed to a complete halt. The characteristic ECG picture is: (1) P waves are present and occur at a rate faster than the ventricular rate; (2) QRS complexes are present and occur at a regular rate, usually <60 beats per minute;

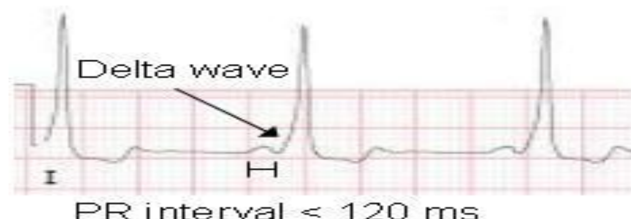
and (3) the P waves bear no relationship to the QRS complexes. Thus, the P-R intervals are completely variable. The QRS complex may b of normal or abnormal width, depending on the location of the blockage in the AV junction..



PRE-EXCITATION SYNDROME

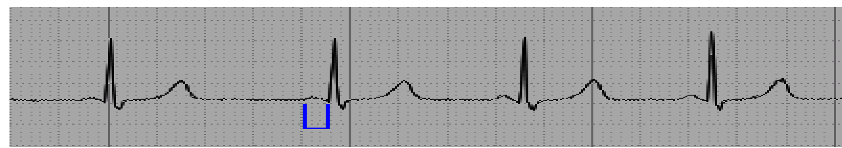
Pre-excitation syndromes refer to clinical Conditions in which the wave of depolarization bypasses the atrioventricular node as it passes from the atria to the ventricles. The time required for the wave to leave the sinoatrial node and arrive at ventricular muscle (P-R interval) is, therefore, shortened. Two pre-excitation syndromes exist (1) the Wolff-Parkinson-White syndrome, and (2) the Lown-Ganong- Levine syndrome.

Wolff-Parkinson-White Syndrome (WPW): Patients with WPW possess an accessory pathway of depolarization, the bundle of Kent. Three electrocardiographic criteria for WPW are: **(1) a short P-R interval, (2) a wide QRS complex, and (3) a delta wave**.



The QRS complex is widened by the delta wave in exactly the same amount as the P-R interval is shortened. The delta wave is a slurring of the initial portion of the QRS complex produced by early depolarization. The major clinical manifestation of WPW is recurrent tachycardia.

Lown-Ganong-Levine Syndrome (LGL): LGL is the result of some of the internodal fibers' (James fibers) bypassing the major portion of the atrioventricular node and terminating in the bundle of His.The three criteria for LGL are: (**1) a short P-R interval without a delta wave, a) anormal QRS, and (3) recurrent paroxysmal tachycardia**. It should be noted that,unlike in WPW, episodes of tachycardia are required for the diagnosis of LGL.



INTRAVENTRICULAR CONDUCTION DISTURBENCE

In the normal process of ventricular depolarization, the electrical stimulus reaches the ventricles by way of the atrioventricular (AV)junction. Then the depolarization wave spreads to the main mass of the ventricular muscle by way of the right and left bundle branches. The right bundle branch is undivided, while the left divides into anterior and posterior fascicles. Normally the entire process of ventricular depolarization occurs in less than 0.1 seconds. Any process that interferes with normal depolarization of the ventricles may prolong the QRS width.

Right Bundle Branch Block (RBBB): Septal depolarization results in a small R wave in V1. Left ventricular depolarization results in an S wave. Right ventricular depolarization produces a second R wave. The delayed depolarization of the right ventricle causes an increased width of the QRS complex to at least 0.12 seconds. Hence, RBBB is characterized by an R-R1 configuration in lead V1 with a QRS complex > 0.12 seconds. RBBB occasionally can be seen in normal subjects.

Incomplete RBBB: This shows the same QRS pattern as a complete RBBB; however, the QRS duration is between 0.1 and 0.12 seconds.

Left Bundle Branch Block (LBBB): Blockage of conduction in the left bundle branch prior to its bifurcation results primarily in delayed depolarization of the left ventricle.In LBBB, the septum depolarizes from right to left, since its depolarization now is initiated by the right bundle branch. Next the right ventricle depolarizes, followed by delayed depolarization of the left ventricle, giving an R-R1 configuration in lead V6 and a QRS interval 0.12 seconds. Hence, LBBB is characterized by an R-R1 configuration in lead V6 and a QRS interval > 0.12 seconds. Unlike RBBB, LBBB alwaysis a sign of organic heart disease.

Incomplete LBBB: This shows the same QRS pattern as a complete LBBB; however,the QRS duration is between 0.1 and 0.12 seconds.

Fascicular Blocks (hemi-blocks): These are blockages of transmission that also may occur in the anterior orposterior branches (fascicles) of the left bundle branch. The main effect of a fascicular block is to markedly change the QRS axis without changing the shape orduration of the QRS wave form.

Left Anterior Hemiblock: This results in left axis deviation (-30 degrees or more).

Left Posterior Hemiblock: This results in right axis deviation (+90 degrees or more).

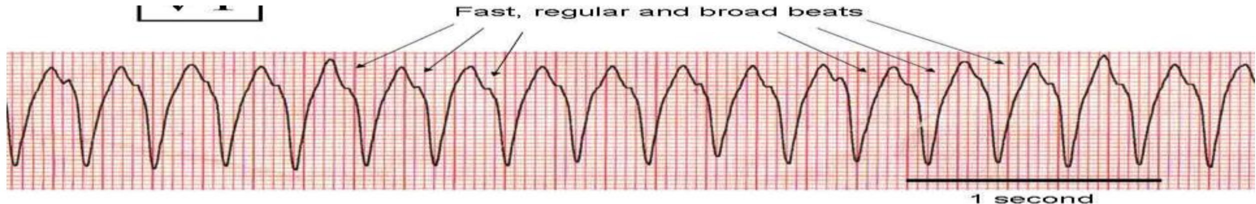
VENTRICULAR ARRHYTHMIAS

Ventricular tissue is capable of spontaneous depolarization. When this occurs, a premature ventricular contraction (PVC) is initiated. Because the depolarization wave arises in the myocardium, it usually does not follow the normal path of ventricular

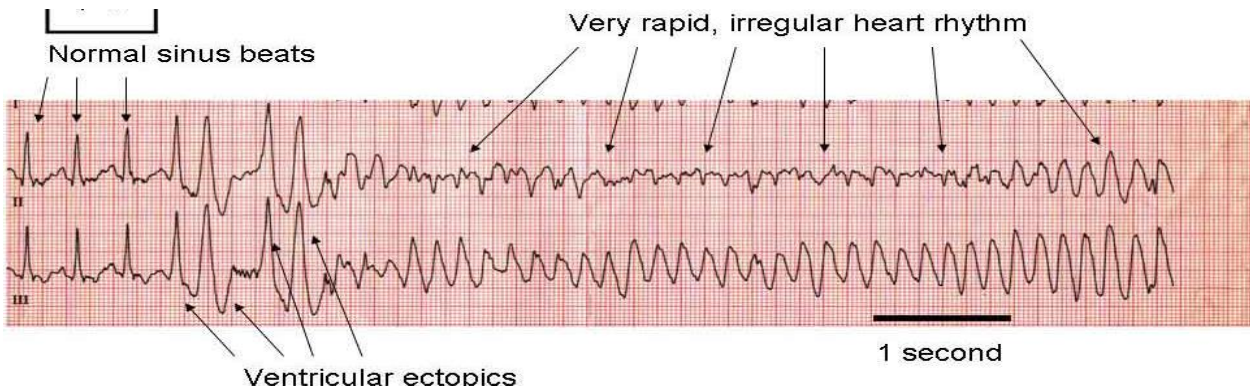
depolarization. Therefore, the QRS complex is prolonged and bizarre in shape. In addition to PVCs, ectopic ventricular beats produce ventricular tachycardia and sometimes ventricular fibrillation. Ventricular escape rhythms also occur.

Premature Ventricular Contractions (PVC): PVCs are premature beats arising from the ventricles, and are analogous to premature atrial contractions and premature junctional contractions. PVCs have two major characteristics: (1) they are premature and arise before the next normal beat is expected (a P wave is not seen before a PVC), and (2) they are aberrant in appearance. The QRS complex always is abnormally wide; the T wave and the QRS complex usually point in opposite directions. The PVC usually is followed by a compensatory pause. PVCs may be unifocal or multifocal. Unifocal PVCs arise from the same ventricular site, and as a result have the same appearance on a given ECG lead. Multifocal PVCs arise from different foci and give rise to different QRS patterns.

Ventricular Tachycardia: This is defined as a run of 3 or more PVCs and may occur in bursts or paroxysmally. They may be persistent until stopped by intervention. The heart rate is usually 120 to 200 beats per minute. Ventricular tachycardia is a life- threatening arrhythmia.



Ventricular Fibrillation: This occurs when ventricles fail to beat in a coordinated fashion and, instead, twitch asynchronously. The beats are sometimes divided into coarse and fine rhythms.



ATRIAL ENLARGEMENT

To evaluate atrial enlargement by looking on the P wave morphology in the LeadII and lead V1 of ECG. The right portion of the P wave is generated by the right atrium and the left by the left atrium.

Right atrial enlargement: tall P wave in laed II and Lead V1.

Left atrial enlargement: Wide or notched P wave in Lead II and Lead v1.

VENTRICULAR HYPERTROPHY

The ECG normally reflects left ventricular depolarization because left ventricular mass is much greater than right ventricular mass.

Right Ventricular Hypertrophy (RVH): When right ventricular muscle mass become great enough, it causes alterations in the positivity of the right chest leads. In the absence of myocardial infarction or right bundle branch block, the diagnosis of RVH can be made when right axial deviation is present and when R > S in lead V1 or S > R in lead V6.

RVH with Strain (systolic overload): In addition to RVH criteria, T wave inversion and usually ST segment depression are present in the right chest leads. (ST segment T wave changes are not present in diastolic overload.)

Left Ventricular Hypertrophy (LVH): Hypertrophy of the left ventricle causes an increase in the height and depth of the QRS complexes. LVH is present when the sum of the S wave in V1 and the R wave in V5 or V6 (whichever is larger) > 35 mm. Accuracy in diagnosing LVH can be improved by considering limb lead criteria; i.e., if the sum of the R wave in lead I and the S wave in lead III > 25 mm., LVH is said to be present when either the chest lead criteria or limb lead criteria is met

LVH with Strain (systolic overload): In addition to criteria for LVH, T wave inversion and ST segment depression occur in the left chest leads. (ST segment and T wave changes are not present in diastolic overload.)

MYOCARDIAL ISCHEMIA

Due to insufficient oxygen supply to the ventricular muscle. It may be transient, causing angina pectoris, or more severe, causing the death of a portion of heart muscle (myocardial infarction).

Subendocardial Ischemia: Produces classic angina and subendocardial myocardial infarction. Involves the inner layer of ventricular muscle.

Transmural Ischemia: Produces Prinzmetal's angina and transmural myocardial infarction. Involves the entire thickness of the ventricular wall.

Classic Angina: Produces transient ST segment depression (except in lead aVR, which may show reciprocal ST segment elevation). Not all patients with coronary artery disease show ST segment depression during chest pain.

Prinzmetal's Angina: Atypical angina that occurs at rest or at night and results in ST segment elevation. Thought to be caused by transient transmural ischemia due to vasospasm. May occur in individuals with otherwise normal coronary arteries.

MYOCARDIAL INFARCTION

According to waveform morphology we can divide MIs as

- STEMIs and NSTEMIs
- Q wave MI and Non Q wave MI

According to age

- Acute vs.subacute vs. “old”

Through anatomic localization we can identify the affected wall and the culprit vessel.

Acute coronary syndrome can be divided into ST elevation MI s(STEMI) and Non ST elevation MIs (NSTEMI).in STEMIs there should be an ST elevation >=2mm in the anatomically contiguous leads. In the same way reciprocal changes such as ST depression and / or T wave inversion can also be seen corresponding reciprocal leads.

In Non ST elevation MIs(NSTEMI) and Unstable Angina (UA) may or may not have ST depression and /or T wave inversions. Sometimes EKG may be completely normal. NSTEMI and UA differentiated only by elevation of biomarkers(e.g.tropnin).

Other common EKG findings during MIs are

- Pathologic Q waves
- New QRS axis deviation
- Poor R wave progression
- Conduction block(e.g.AV block, Bundle branch block)\

Anatomic localization

Acute STEMIs can be localized to a specific anatomic region . subacute or old MIs which have resulted in pathologic Qwave s(a ‘Q wave MI”) can also be localized to an antomic region , though this is not completely reliable , and assume the cause of the Q wave must be an old Infarct (which may nat be true.) . however , acute NSTEMIs and old non Q wave MIs cannot be accurately localizaed . The location of st- t changes (e.g. ST depressions, T wave inversions) seen in these situations do not correspond to the location of iinfarcted territory.

The heartmuscle itself is very limited in its capacity to extract oxygen in the blood that is being pumped. Only the inner layers (the endocardium) profit from this oxygenrich blood.

The outer layers of the heart (the epicardium) are dependent on the coronary arteries for the supply of oxygen and nutrients. With aid of an ECG, the occluded coronary can be identified. This is valuable information for the clinician, because treatment and complications of for instance an **anterior wall infarction** is different than those of an **inferior wall infarction**. The anterior wall performs the main pump function, and decay of the function of this wall will lead to decrease of bloodpressure, increase of heartrate, shock and on a longer term: heart failure. An inferior wall infarction is often accompanied with a decrease in heartrate because of involvement of the sinusnode. Longterm effects of an inferior wall infarction are usually less severe than those of an anterior wall infarction.

The heart is supplied of oxygen and nutrients by the right and left coronary arteries. The left coronary artery (the **Left Main** or LM) divides itself in the **left anterior descending** artery (LAD) and the **ramus circumflexus** (RCX). The **right coronary artery** (RCA) connects to the **ramus descendens posterior**(RDP). With 20% of the normal population the RDP is supplied by the RCX. This called **left dominance**.

Below you can find several different types of myocardial infarcation. Click on the specific infarct location to see examples.

Help with the localization of a myocardial infarct

localization	**ST elevation**	**Reciprocal ST depression**	**coronary artery**
Anterior MI	V1-V6	None	LAD
Septal MI	V1-V4, disappearance of septum Q in leads V5,V6	none	LAD-septal branches
Lateral MI	I, aVL, V5, V6	II,III, aVF	LCX or MO
Inferior MI	II, III, aVF	I, aVL	RCA (80%) or RCX (20%)

Posterior MI	V7, V8, V9	high R in V1-V3 with ST depression V1-V3 > 2mm (mirror view)	RCX
Right Ventricle MI	V1, V4R	I, aVL	RCA
Atrial MI	PTa in I,V5,V6	PTa in I,II, or III	RCA

The localisation of the occlusion can be adequately visualized using a coronary angiogram (CAG). On the CAG report, the place of the occlusion is often graded with a number (for example LAD using the classification of the American Heart Association.

The cardiomyocytes in the subendocardial layers are especcially vulnerable for a decreased perfusion. Subendocardial ischemia manifests as ST depression and is usually reversible. In a myocardial infarctiontransmural ischemia develops.

In the first hours and days after the onset of a myocardial infarction, several changes can be observed on the ECG. First, **large peaked T waves** (or hyperacute T waves), then **ST elevation**, then **negative T waves** and finally **pathologic Q waves** develop.

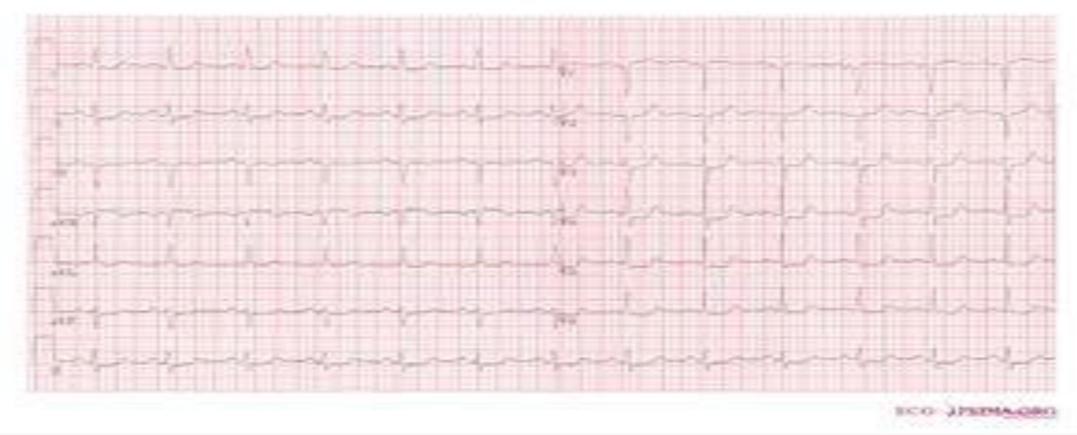
Wellens syndrome or sign (see image) can be an early ECG warning sign of critical anterior ischemia before the development of overt mocardial infarction.

Evolution of the ECG during a myocardial infarct

Time from onset of symptoms	ECG	Changes in the heart
minutes	hyperacute T waves (tall T waves), ST-elevation	reversible ischemic damage

Hours	ST-elevation, with terminal negative T waves, negative T waves (these can last for days to months)	onset of myocardial necrosis
Days	Pathologic Q Waves	scar formation

Subendendocardial Ischemia



An example of subendocardial ischemia with diffuse ST depression

Subendocardial ischemia is ischemia that is not transmural. It is mostly caused by demand ischemia where energy supply to cardiomyocytes is insufficient for the work force, e.g. during extreme hypertension, aortic valve stenosis, extreme left ventricular hypertension, anemia, atrial fibrillation with rapid ventricular response. On the ECG often

diffuse ST depression is present. Cardiac enzymes (CK-MB, Troponine) may or may not be elevated depending on the severity.

Transmural Infarction: The infarcted area remain in a depolarized (negative) state. A normal variant - early repolarization - often occurs in younger individuals and may be confused with myocardial infarction. With early repolarization, however, the T wave is distinct from the elevated ST segment, whereas with myocardial infarction, It is incorporated into it. The loss of positivity in the infarcted area is responsible for the characteristic Q waves that develop in the leads exploring the infarcted area. Keep in mind that a normal ECG may exhibit small Q waves in leads I, V5, and V6 that represent only normal septal depolarization. Q waves, to be considered diagnostic of acute myocardial infarction, must (1) have a duration of at least 0.04 seconds or (2) have a depth equal to 25% or more of the height of the R wave.

Pseudo Infarction Syndromes: LBBB and Wolff-Parkinson-White usually hav significant Q waves. Left ventricular aneurysm after extensive infarction may show persistent ST segment elevation. Pericarditis may show ST segment elevation and subsequent T wave inversion; however, there is no Q wave formation. Patients with idiopathic hypertrophic subaortic stenosis often may have significant Q waves due to distortion of the normal pattern of depolarization. Dramatic alterations of ST segments and T waves may occur with increased intracranial pressure.

PATTERNS CAUSED BY DRUG AND ELECTROLYTE EFFECTS

The drugs digitalis and quinidine produce major effects on an ECG that have considerable clinical significance. Two electrolytes-potassium and calcium-also produce significant ECG effects.

Digitalis: Changes include modification of the ST-T contour, slowing of AV conduction, and enhancement of ectopic automaticity. Digitalis may produce characteristic scooping of the ST-T complex. The ST segment and T wave are fused together, and it is impossible to tell where one ends and the other begins. This may occur when digitalis is in the therapeutic range. With toxicity, digitalis can cause virtually any arrhythmia and all degrees of atrioventricular block.

Quinidine: Increases repolarization time and, hence, prolongs the Q-T interval. In toxic doses, may widen the QRS complex and cause ST segment depression.

Potassium: Hyperkalemia produces tall, peaked T waves, widening of the QRS complex, and prolongation of the P-R interval. Hypokalemia produces flattening of the T waves, which may unmask U waves. T waves may become inverted, and ST segment depression may occur.

Calcium: Hypercalcemia shortens ventricular repolarization time, resulting in a shortened Q-T interval. Hypocalcemia prolongs the Q-T interval.

NON-SPECIFIC ST-T WAVE ABNORMALITIES

Non-specific abnormalities of the ST-T wave segment are diagnosed when the repolarization complex is abnormal but does not indicate a particular diagnosis.Factors such as temperature, hyperventilation, and anxiety can influence the ST-T complex.

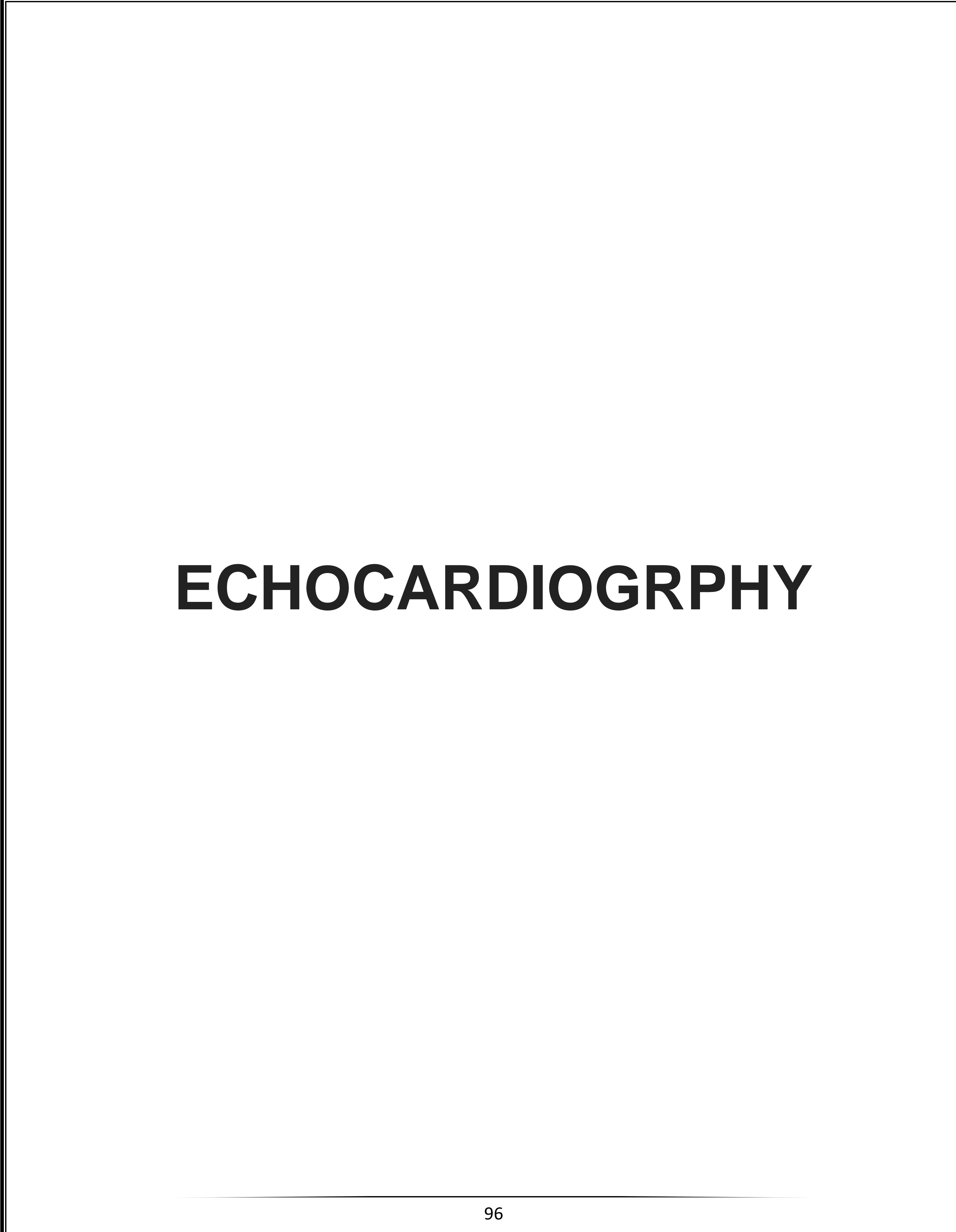
LOW VOLTAGE COMPLEXES

Can be caused by pericardial effusion, obesity, diffuse myocardial fibrosis, infiltration of the heart muscle by substances such as amyloid, and hypothyroidism.

ECG FILTERS

Filters used to remove unwanted noise. In ECG work, the signal levels are very small, so it is necessary to use filtering. This noise may come from an unstable dc offset from electrode/ body interface , muscle noise, electrical nise from equipment in the environment and from within the ECG equipment itself, such as from internal DC/ DC converters. Mains noise (50/60Hz), muscle noise and drift in DC offset due to patient movement all fall in the same frequency range as typical ECG. We are using low pass filter in which high frequency components not allows to transfer. The most common hardware is the series of resistor/capacitor.

Whereas high pass filters are opposite to that of low pass filters,which allows high frequency components to pass through. In hardware, a single pole filter can be madeout of a capacitor in series with a resistor. The main intention of a high pass filter in ECG works is to remove the DC offset which in turn is largely caused by the electrode/gel/body interface. Notch filters combine in both high pass low pass filters to create a small region of frequencies to be removed. For ECGs the main target is to remove 50Hz to 60Hz.



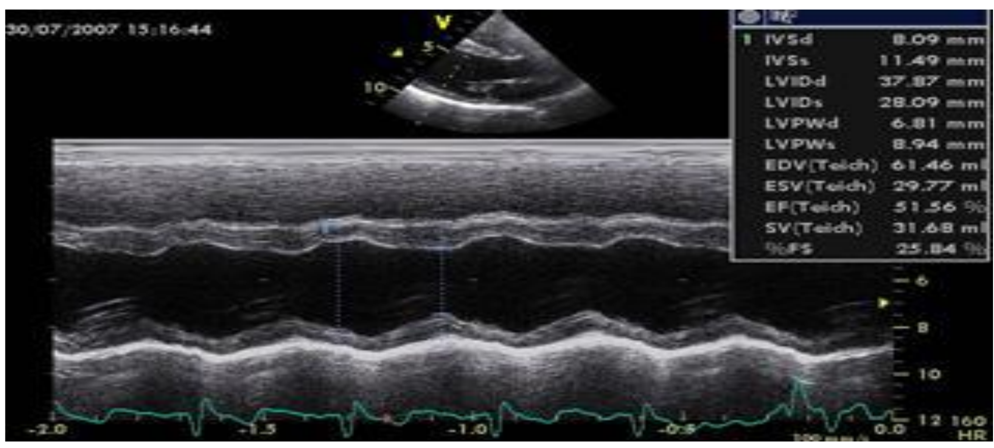
ECHOCARDIOGRPHY

PRINCIPLES OF ULTRASOUND

Sound whose frequency is above what is audible to human ear (more than 20KHz) is known as ultrasound. The technique of using ultrasound to examine heart is known as echocardiography. Electricity and ultrasound are two different forms of energy that can be transformed from one to the other by special crystals made of ceramics such as barium titanate. Ultrasound relies on the property of such crystals to transform electrical current of changing voltage into mechanical vibrations or ultrasound waves. This is known as the piezoelectric effect. When the current is passed through a piezoelectric crystal the crystal vibrates. This generates ultrasound waves which are transmitted through the body by the transducer which houses several such crystals.

Most of these ultrasound waves are scattered or absorbed by the tissues, without any obvious effect. Reflected ultrasound waves again distort the piezoelectric crystals and procedure an electric current. These reflected echoes are processed by filtration and amplification, to be eventually displayed on the cathode ray tube. Diagnostic ultrasound for cardiovascular imaging uses frequencies ranging from 2 to 30 Mh ; adult transthorasic frequencies range from 1 to 5 MHZ , tansesophagal frequencies from 5 to 7 Mhz ,and intravascular ultrasound frequencies from 20 to 40 Mhz, 15 to 20 for pediatric.

M-MOD E



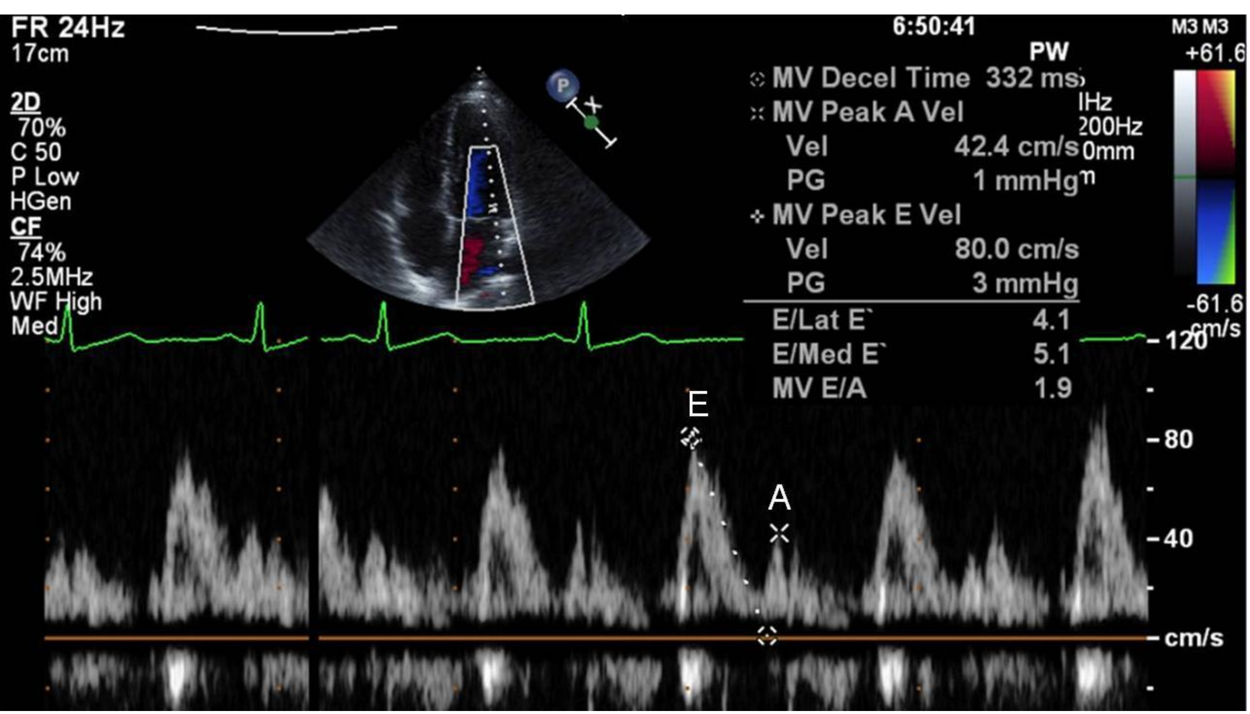
M-mode (motion) echo displays a narrow ultrasound beam of information within the heart along the Y-axis(vertical axis) and displays it according to time on the X- axis (horizontal axis). Heart structures are displayed with respect to motion and time. M-mode echo provides high temporal resolution and provides information regarding both the structure and function of the heart. M-mode echo predated the existence of two-dimensional echo and although it is not as commonly used in the pasr M-mode can still be useful to describe motion of structures of the heart with respect to cardiac cycle.

2-D

2-D echo was developed from similar concepts of M-mode. Because Mmode only allowed a very narrow and focused area of interrogation of the heart, it was limited, 2-D echo provided a wider area investigation into the structure and function of the heart within a 90-degree scanning sector. Thus, 2-D echo provides a more complete investigation of the entire structure and function of the heart.

DOPPLER

Doppler affords clinician a powerful and integral tool for assessing heart function. This is based on the Doppler principle. It describes the change in reflected sound wave frequency compared with the transmitted sound wave frequencies generated from the transducer. These sound waves are reflected off of moving red blood cells. This change in frequency is related to the velocity of moving red blood cells through the heart, which can be used to describe the hemodynamics of blood flow through the heart.

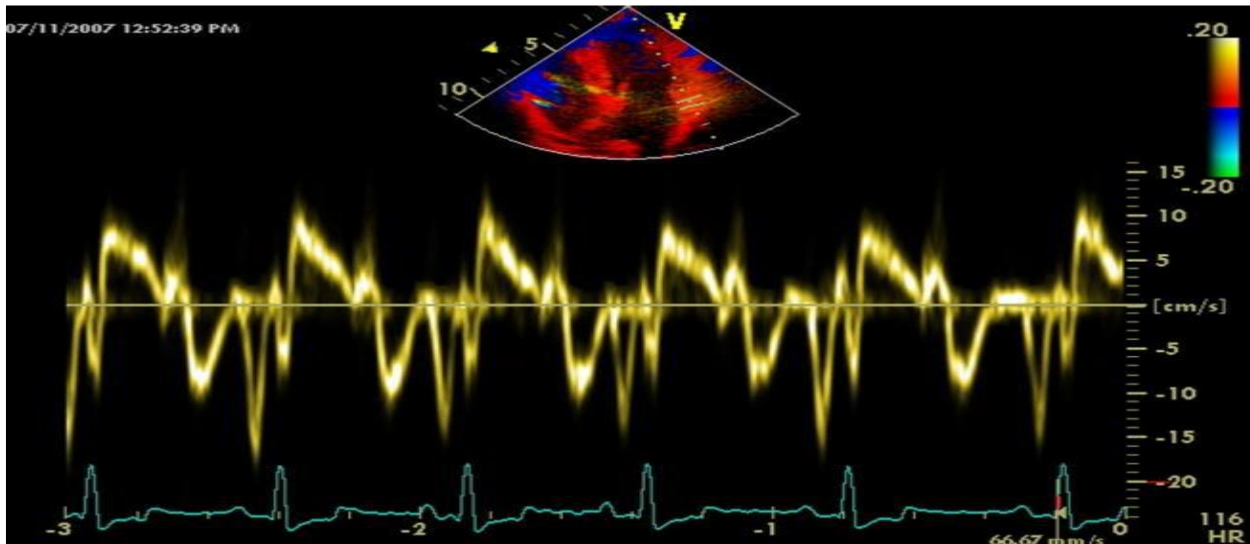


One important feature of Doppler interrogation is the angle of the transmitted frequency as compared to blood flow. To achieve the most accurate estimate of the velocity of blood flow, the angle of the transmitted sound wave should be parallel to blood flow. As the angle increases, so does the error, which results in underestimation of blood flow velocity. Because lungs and bones limit the number of imaging spaces that can be used to interrogate blood flow through the Heart. There are three types of of Doppler techniques: pulse wave, continues wave, and color. Pulse wave Doppler is used for the assessment of blood flow at specific location and is useful for velocities less than 2 m/s. Continues wave Doppler is used for assessing velocities along the entire pathway of the sound wave and is used for

velocities up to 8 m/s. Continues wave Doppler as its name suggests is continuously transmitting and receiving sound waves using separate crystals . This technique is unlike all other echo techniques , such as 2-Decho, pulse wave Doppler , and color Doppler, which all use the same crystal to transmit and receive sound waves and predominantly spend the majority of time receiving sound waves . Color Doppler is a pulse wave technique in which multiple points in a specified sector are sampled. Depending upon the direction and turbulence of blood flow, a color is encoded upon a 2-D image. This technique is useful for visualizing the presence of blood flow, the presence of turbulent blood flow, and shunt.

TISSUE DOPPLER AND STRAIN IMAGING

Tissue Doppler imaging records the motion of tissue or other structures with a velocity or frequency shift much lower than that of blood flow. Doppler echocardiography for blood flow measures the velocities of red blood cells. However, the velocities of myocardial tissues are much lower but with large amplitudes than those produced by blood. Therefore pulsed wave Doppler was modified to record the low velocities of myocardial tissue and to reject the high velocities generated by blood flow. TDI can also be displayed in color mode. A major limitation of tissue velocities recording is that tissue velocities measured by TDI may overestimate or under estimate the active component or function of the tissue because of translational motion or tethering, respectively . Strain and strain rate imaging can overcome this limitation by measuring the actual extend of stretching or contraction.



The clinical application of TDI and strain imaging are increasing and provide incremental diagnostic and prognostic value over standard 2D and blood pool Doppler echocardiography. They have been used successfully in assessing regional and global systolic and diastolic function. Myocardial strain becomes abnormal during the early stage of myocardial ischemia as well as in myopathies and appears to be more sensitive for identifying ischemic segments during stress echocardiography. TDI has been used most

commonly to evaluate diastolic function and to estimate diastolic filling pressures. Both strain and tissue Doppler imaging allow reliable determination of cardiac timing intervals ,which is useful in assessing cardiac function and LV interventricular mechanical dysynchrony.

PATIENT PREPARATION

Transthorasic echo is performed by placing an ultrasound transducer on the patient's chest and images are obtained through the chest wall. The patient is positioned in the left lateral decubites position for the examination and then is moved to the supine position to complete the image set. Occasionally, the patient is positioned in the right lateral decubitus position. Electrocardiogram electrodes are placed on the patients's skin to acquire a continues ECG rhythm. The blood pressure and heart rate should always be recorded at the time of the examination as these measurements affect cardiovascular hemodynamic. Ultrasound waves have significant attenuation through air, a coupling gel is used between the transducer and the patient's skin to eliminate any air.

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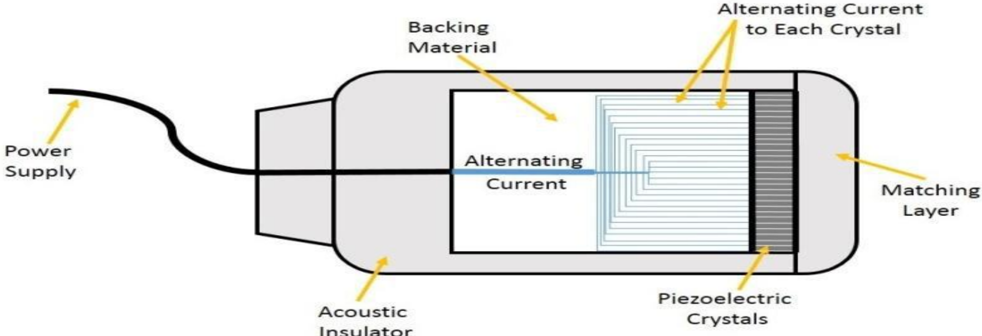
AXIS OF THE HEART

The heart situated obliquely in the chest with the apex toward the left. Imaging is not therefore performed in a straight axial or sagittal orientation. Instead, most of the cardiovascular imaging is performed along the axis of the heart and not the axis of the body. There are two standard axes of the heart; long and short. In the long axis views, the heart is imaged from the base to the apex. The short axis of the heart perpendicular to this axis.

TRANSDUCERS

The use of ultrasound for imaging became practical with the development of piezoelectric transducers. Piezoelectric substances or crystals rapidly change shape or vibrate when an alternating electric current is applied. It is the rapidly alternating expansion and contraction of the crystal material that produce the sound waves. Equally important is the fact that a piezoelectric crystals will produce an electric impulse when it is deformed by reflected sound energy. Such piezoelectric crystals form the critical component of ultrasound transducers. Although a verity of piezoelectric materials exist. Most

commercial transducers employ ceramics, such as ferroelectric, barium titan, and lead zirconate titanate.



Ultrasound transducer very small carefully arranged piezoelectric elements that are inter connected electronically. The frequency of transducer is determined by the thickness of these elements. Each element is coupled to electrodes which transmit current to the crystals, and then record the voltage generated by the returning signals. An important component of transducer design is the dampening materials, which shortens the ringing response of the piezoelectric material after the brief excitation pulse. Transducer design is critically important to optimal image creation. The proximal or cylindrical portion of the beam is referred to as the *near field* or Fresnel zone. When it begins to diverge, it is called the **far field** or **Fraunhofe**r zone. Most ultrasound systems are equipped with a selection of transducer with a range of capabilities and limitations. The frequency of transducer used for cardiac imaging often depends on body habitus and patient size. For large patient or thickchested individuals, a 2.0 or 2.5-MHZ transducer may be necessary to provide adequate penetration. Children and smaller adults can generally be adequately imaged using a 3.5 or 5.0 MHZ transducer. For infants and children, a 7.0 or 7.5 MHZ transducer is often ideal.

STANDARD ECHO WINDOWS

Standard echo windows are important for two reasons:

1. Penetration of ultrasound wave is good , without much masking of image or absorption of ultrasound by ribs and lungs.
2. Standardized echo images can be compared with studies peformed by different observers or different occasions by same observer.

Standard locations on the anterior chest wall are used to place the transducer, which are called “echo windows”.

- ❖ Left parasternal
- ❖ Apical
- ❖ Subcostal
- ❖ Right parasternal
- ❖ Suprasternal

Transthoracsic echo may be technically difficult to perform in the foolowing situations:

a) Severe morbid obesity
b) Chest wall deformity

c) Pulmonary emphysema

Parasternal Long Axis View

- Transducer position: left sternal edge; 2nd and 4th intercostal space
- Marker dot direction:points towards right shoulder.
- Structure seen:
    - Proximal aorta
    - Aortic valve
    - Left atrium
    - Mitral valve
    - Interventricular septum
    - Posterior wall
    - Right ventricle
    - Pericardium

Most of the studies begin with this view. It sets the stage for subsequent echo windows.

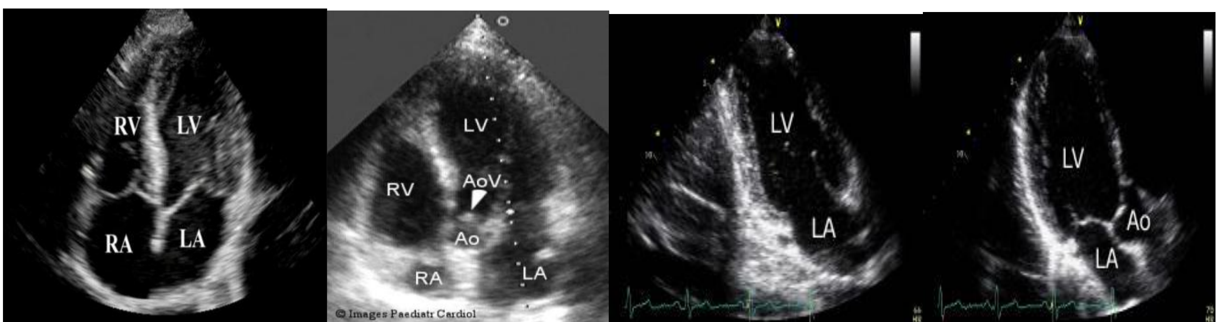
Parasternal Short Axis Views

- Transducer position: left sternal edge ; 2nd and 4th intercostal space
- Marker direction: points towards the left shoulder (90^0 clockwise from PLAX)

By rotating the transducer clockwise, the patient's lateral wall is placed to the observer's right and the medial wall is to the observer's left. A useful reference pointto begin the short axis examination is the tip of the anterior mitral valve leaflet. By rotating the transducer slightly and adjusting the tilt of the plane, the left ventricle can be made to appear circular and both leaflets of the mitral valve will demonstrate maximal excursion. As in all short axis views, the left ventricle is displayed as is viewed from the apex of the chamber. When properly recorded, the short axis view in this plane corresponds roughly to the mid left ventricular level and allows optimal recording of mitral leaflet excursion, mid left ventricular wall motion, and visualisation of a portion of the right ventricle. Moving to a more basal plane, the short-axis view approaches the level of the aortic annulus and the aortic valve, right ventricular outflow tract, coronary Ostia, left atrium, right atrium, interatrial septum, tricuspid valve, pulmonary valve, and proximal pulmonary artery can also be recorded. By moving the transducer to a lower interspaced and angling the scan plan more apically, the image will sweep through the papillary muscle level and then the left ventricular apex.

APICAL

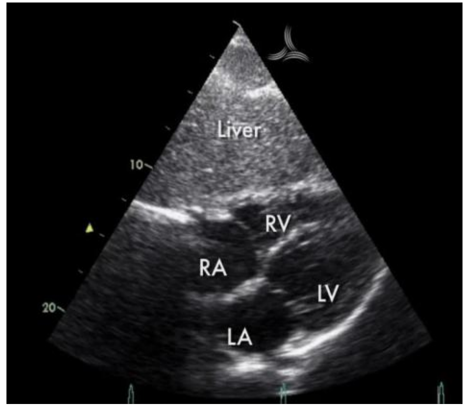
With the patient rotated to the left and the transducer placed at the cardiac apex, a family of long axis images is available. Once the apical window is located, the transducer is pointed in the general direction of the right scapula and then rotated until all four chambers of the heart are optimally visualized. This occurs when the full excursion of



the both mitral and tricuspid valves is recorded and the true apex of the left ventricle lies in the near field. The normal true apex can be identified by its relatively thin walls and lack of motion. When properly adjusted, this image includes the four chambers, both atrioventricular valves, and the interventricular and interatrial septal. By tilting the transducer into a shallower angle relative to the chest wall, resulting in a more anterior scan plane the left ventricular outflow tract, aortic valve, and aortic root can be recorded. This is frequently referred to as the five chamber view. By rotating the transducer counterclockwise approximately 60 degrees, an apical two chamber view is recorded. If the transducer position is returned to the four chamber orientation and then rotated clockwise 60 degrees, an apical long axis view is recorded, characterized by the presence of both mitral and aortic valves in the same plane.

THE SUBCOSTAL EXAMINATION

Tranducer position : under the xiphisternum.
Marker dot position: points towards the left shoulder.
Better images are obtained with the abdomen relaxed and during phase of inspiration. The subcostal view is particularly useful when transthoracic echo is technically difficult because of the following reasons :

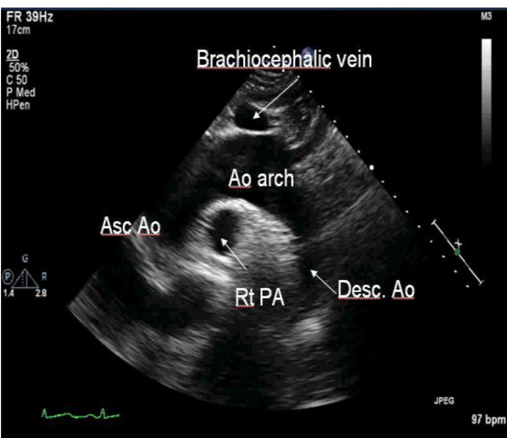


- Severe obesity
- Chest wall deformity
- Pulmonary emphysema
- The following structures such inferior vena cava, descending aorta, interatrial septum, pericardial effusion can better seen from this view.

SUPRASTERNAL VIEW

For suprasternal view, the patient lies with the neck hyperextended by placing a pillow under the shoulders. The head is rotated slightly towards the left. The position arms and legs and the phase of respiration have no bearings on this echo window.
Transducer position: suprasternal notch
Marker dot direction: points towards the left jaw.

Both ascending and descending segments of the aorta as well as the origin of the innominate, left common carotid, left subclavian, and right pulmonary arteries. Because of the proximity of the arch to the transducer, a 90 degrees sector may not be wide enough to simultaneously record both ascending and descending segment of the aorta. Angulations of the transducer is necessary for a complete recording in such patients. From this position , the transducer can be rotated 90 degrees to provide the perpendicular plane, which demonstrates the arch in short axis orientation. From ,this view , the right pulmonary artery and left atrium can usually be recorded. By adjusting the scan plane leftward and slightly anteriorly, the superior venacava also be visualized.

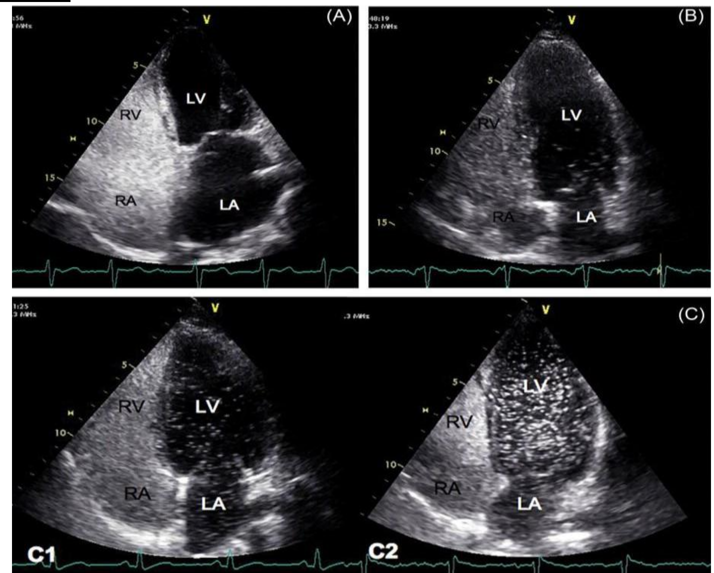


RIGHT PARASTERNAL VIEW

In this view transducer is placed between 2nd and 4th intercostal space on the right sternal edge. Marker dot direction is points towards the left shoulder. Aortic root and aortic arch can be clearly seen in this view.

SPECIAL ECHO TECHNIQUES

CONTRAST ECHO



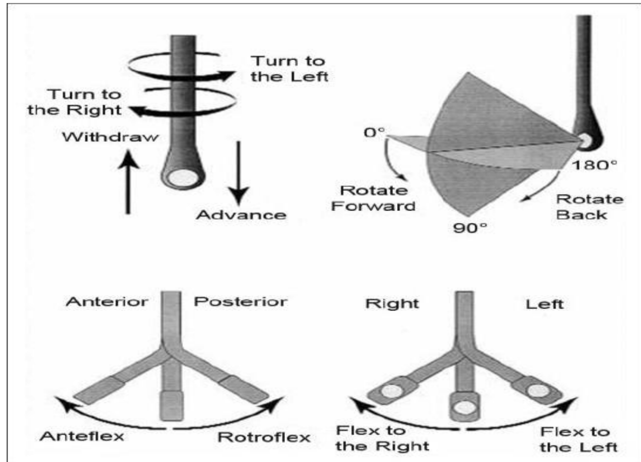
There are several purposes for the use of contrast in echocardiography. The diagnostic indication will detect the specific type of contrast. One simple classification system of echo contrast agents is whether the contrast agent crosses the pulmonary vasculature microcirculation. Some contrast agents remain in the venous circulation while others will be in the venous and arterial circulation Echo contrast agents are composed of bubbles.

The size of the bubbles determines whether they will cross the pulmonary vasculature microcirculation. Red blood cells cross the pulmonary capillaries Saline micro bubbles are commonly used for detection of shunts, primarily because the bubbles are too large to cross the pulmonary vasculature microcirculation. Saline micro bubbles are injected into an intravenous catheter and opacify the right side of the heart. Because the micro bubbles are larger than red blood cells, they cannot cross the pulmonary vasculature and are absorbed by the lungs. Therefore, they should not opacify the left heart structures. Saline micro bubbles that do appear in the left side of the heart are indicative of a right to left shunt (ASD, PFO, pulmonary arterial venous malformation). Saline micro bubbles are created by connecting a two-way stopcock to an intravenous catheter with two 10-cc syringes. One syringe is filled with 9-cc of normal saline and 1-cc of air and then transferred to the empty syringe back and forth. This action makes bubbles of air throughout the saline. The full 10-cc is then quickly injected into the intravenous catheter. Emphasis is placed on quickly so as to achieve bolus opacification of contrast and not streaming of contrast of the right heart structures .

STRESS ECHO

Stress Echo is performed in combination with continuous 12- lead ECG monitoring to improve the diagnostic accuracy of coronary artery detection or risk stratification of patents with known coronary artery disease. Image acquisition is performed in the same standard views as, described previously and optimal endocardial definition is necessary. Sometimes, contrast is needed to provide endocardial definition to visualize wall motion. Global and regional wall motion is compared from rest to stress periods. A normal finding: a myocardial segment has normal function at rest and become hypo kinetic or akinetic at stress. The preferred method of stress testing is exercise, provide the patient is able. With echo, exercise is usually done on a treadmill or supine bicycle. For treadmill exercise, rest images are performed while the patient is lying on the examination bed. The patient then exercise on the treadmill with continues ECG monitoring. When peak exercise is achieved, the patient is quickly moved from the treadmill back to the examination bed and imaging is repeated. This sequencing requires practice and coordination. The longer it takes to perform the imaging after peak exercise, the heart rate decreases and so does the sensitivity of the test. Image acquisition should occur within 60 seconds of the termination of peak exercise. For those patients who cannot exercise, dobutamine is the most common stress agent used; however, it should be understood that dobutamine does not completely simulate the physiological state of exercise. A nurse should be present during dobutamine infusion since medication need to be given intravenously. Starting doses are between 5 and 10mcg/kg/min, increasing by 10 mcg/kg/min increment every 3 minutes to a peak of 40 mcg/kg/min. Doses of atropine can be given and physical maneuvres can be performed in addition to dobutamine infusion to achieve 85% of the maximal predicted heart rate.

TRANSESOPHAGEAL ECHO



Principle: during echocardiography, a balance has to be struck between tissue penetration image resolution. Low frequency transducers have good penetration (less attenuation) but relatively poor resolution. On the other hand, high frequency tranducers have poor penetration (more attenuation) but better resolution. Anatomically speaking, the esophagus in its mid- course is strategically located posterior to the heart and anterior to he descending aorta. This provides an opportunity to interrogate the heart and related mediastinal structures with a high frequency transducer positioned in the esophagus for better image resolution. The technique is known as transesophageal echocardiography(TEE).

A miniature transducer is mounted onto a probe or gastroscope similar to the one employed for upper gastrointestinal endoscopy. The scope is advanced to various depths in the esophagus to examine cardiac and related structures. By manoevring the transducer and angle of beam from controls on the handl, different views of the heart are obtained. This 'back-door' apporoach to echocardiography has both advantages and disadvantages.

Advantages

- Useful alternative to transthoracic echo if the latter is technically difficult due to obesity, chest wall deformity, emphysema or pulmonary fibrosis.
- Useful complement to transthoracic echo because of better image quality and resolution due two reasons.
- Absence of acoustics barrier between the ultrasound beam and ribs cage, chest wall lung tissue.
- Greater proximity to the heart and therefore the ability to use higher frequency probe with vastly improved image quality an precise spatial resolution.
- Useful supplement to transthoracic echo, which cannot examine the posterior aspect of the heart. structures such as left atrial appendage, desending aorta and pulmonary veins can only be visualized by TEE

Disadvantages

It is a semi-invasive procedure which is uncomfortable to the patient, more time consuming and carries a small risk of serious complication such as oropharyngeal or esophageal trauma, cardiac arrhythmias and laryngo-brochospasm. It requires short term sedation oxygen administration and ECG monitoring since, there are chances of hypoxia, arrhythmia and angina. Rarely, respiratory depression or allergic reactions may occur. TEE in contraindicated in the presence of active bleed nor coagulopathy, esophageal abnormalities, unstable cervical arthritis and poor cardiopulmonary status.

Complications with TEE

Major

- **Esophageal rupture or perforation**
- **Laryngospasm or bronchospasm**
- **Sustained ventricular tachycardia**

Minor

- **Retching and vomiting**
- **Sore-throat and hoarseness**
  Blood-tinged sputum
- **Tachycardia or bradycardia**
- **Hypoxia and ischemia**
- **Transient BP rise or fall**

Contraindications TEE

Absolute

- **Uncooperative patient**
- **Poor cardiorespiratory status**
- **Esophageal obstruction**
- **Trachea esophageal fistula**
- **Active bleed or coagulopathy**

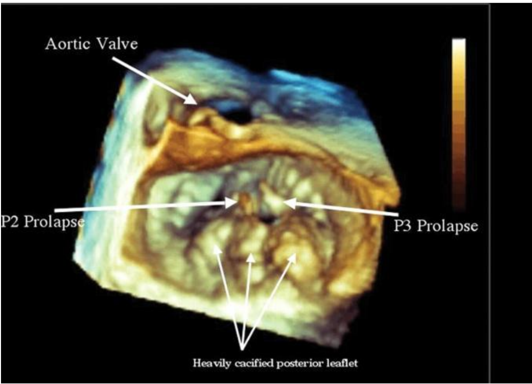
Relative

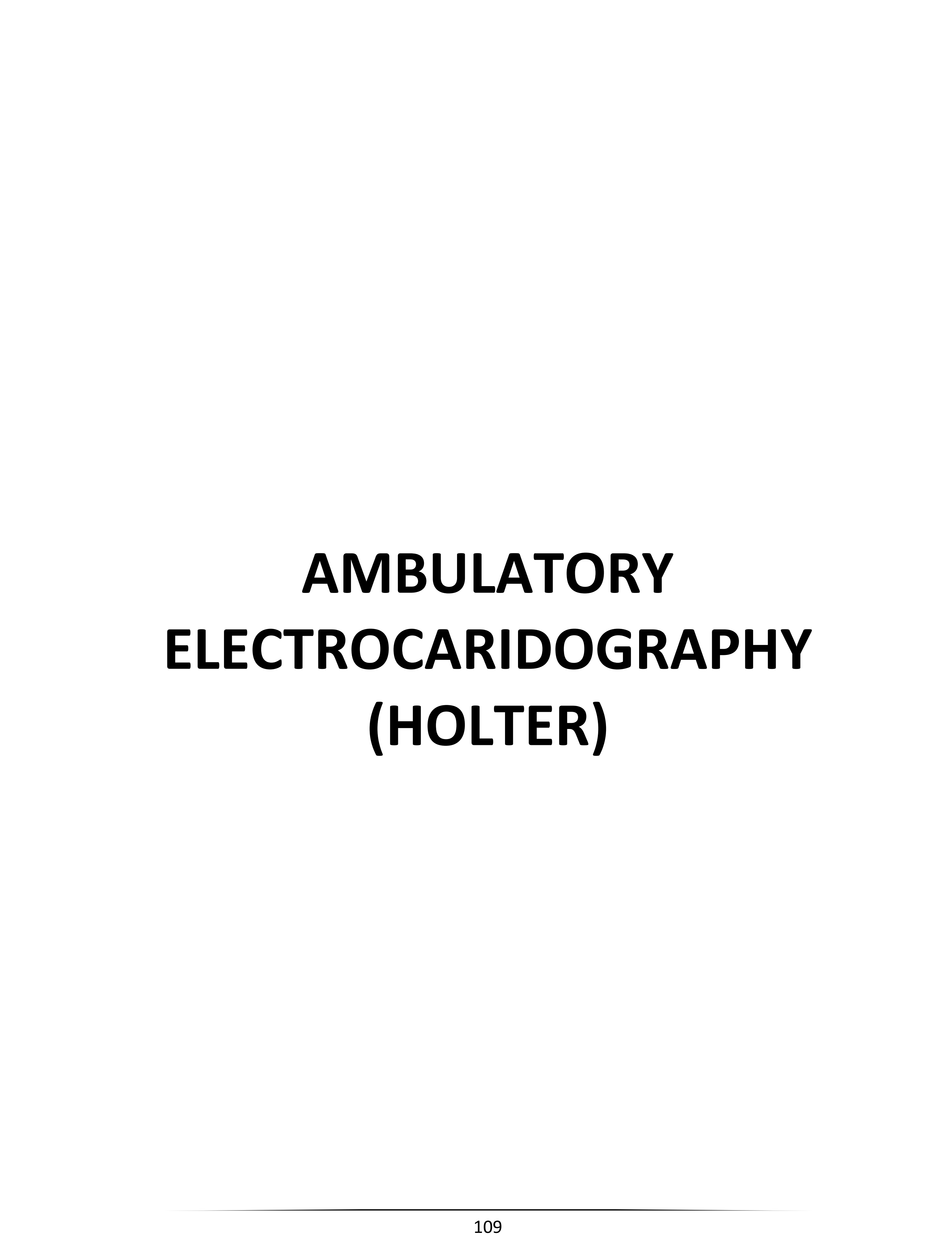
- **Large esophageal varices**
- **Prior esophageal surgery**
- **Unstable cervical arthrirtis**

3-DIMENSIONAL ECHOCARDIOGRAOHY

3-dimensional echocardiography (3D echo) obviates the need for cognitive 3D reconstruction of 2D image planes and the compulsion of making geometric assumption about the cardiac structures for quantification. Besides its application in ventricular volumetric assessment,e 3D echo is particularly useful to study asymmetrical stenotic valve orifices, eccentric regurgitant jets picked up by color doppler and complex structural relationships observed in congenital heart diseases. The 3D echo can be seen from various

projections by rotating the images. It has the potential to reduce the time required for complete cardiac image acquisition





AMBULATORY ELECTROCARIDOGRAPHY (HOLTER)

HOLTER MONITORING

In medicine, a Holter monitor (often simply "Holter" or occasionally ambulatory electrocardiography device) is a portable device for continuously monitoring various electrical activity of the cardiovascular system for at least 24 hours (often for two weeks at a time). The Holter's most common use is for monitoring heart activity (electrocardiogram or ECG), but it can also be used for monitoring brain activity (electroencephalography or EEG) or arterial pressure. Its extended recording period is sometimes useful for observing occasional cardiac arrhythmias or epileptic events which would be difficult to identify in a shorter period of time. For patients having more transient symptoms, a cardiac event monitor which can be worn for a month or more can be used.

The Holter monitor is named after physicist Norman J Holter, who invented telemetric cardiac monitoring in 1949. Clinical use started in the early 1960s. When used for the heart, (much like standard electrocardiography) the Holter monitor records electrical signals from the heart via a series of electrodes attached to the chest. Electrodes are placed over bones to minimize artifacts from muscular activity. The number and position of electrodes varies by model, but most Holter monitors employ between three and eight. These electrodes are connected to a small piece of equipment that is attached to the patient's belt or hung around the neck, and is responsible for keeping a log of the heart's electrical activity throughout the recording period.

ELECTRODE PLACEMENT CHART
7 -LEAD 3 -CHANNEL

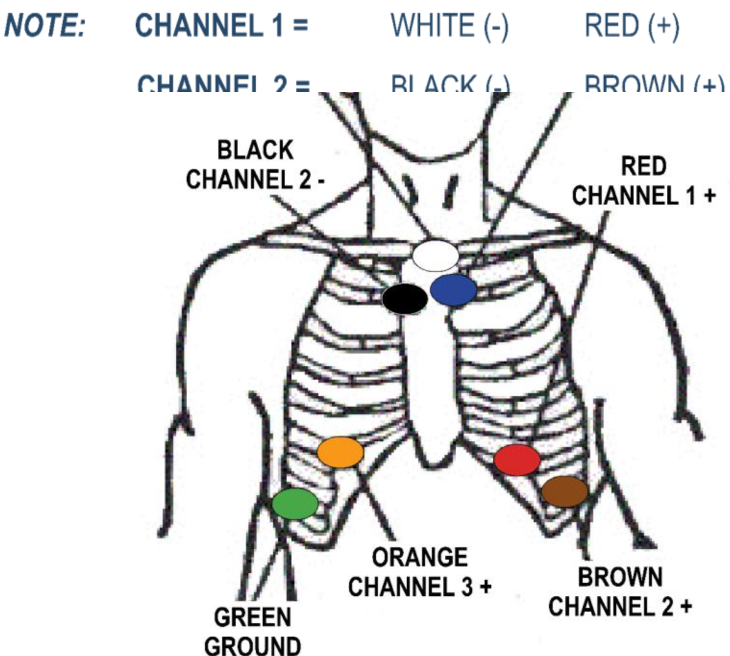
Seven leadwires are utilized to create a three channel ECG recording

WHITE:	**Channel 1 (-).** Place at the top of the sternum
RED:	**Channel 1 (+).** Place on the left side at the V3 position on a rib
BLACK:	**Channel 2 (-).** Place at the top of the sternum, adjacent to the white lead
BROWN:	**Channel 2 (+).** Place on the left side at the V5 position on a rib
BLUE:	**Channel 3 (-).** Place at the top of the sternum, adjacent to the white lead
ORANGE:	**Channel 3 (+).** Place on the right side at the V1 position on a rib
GREEN:	**Ground.** Place on the right side opposite V5 position on a rib

NOTE:	CHANNEL 1 =	WHITE (-)	RED (+)
	CHANNEL 2 =	BLACK (-)	BROWN (+)

ORANGE: **Channel 3 (+).** Place on the right side at the V1 position on a rib

GREEN: **Ground.** Place on the right side opposite V5 position on a rib



DATA STORAGE

Older devices used reel to reel tapes or a standard C90 or C120 audio cassette and ran at a 1.7 mm/s or 2 mm/s speed to record the data. Once a recording was made, it could be played back and analyzed at 60x speed so 24 hours of recording could be analyzed in 24 minutes. More modern units record an EDF-file onto digital flash memory devices. The data is uploaded into a computer which then automatically analyzes the input, counting ECG complexes, calculating summary statistics such as average heart rate, minimum and maximum heart rate, identifying ectopics such as PVCs, PACs and labelling it by the application software in the computer. This will make the operator to reassess the analysed data from the computer more easier.

COMPONENTS

Each Holter system consists of two basic parts – the hardware (called monitor or recorder) for recording the signal, and software for review and analysis of the record. Advanced Holter recorders are able to display the signal, which is very useful for checking the signal quality. Very often there is also a “patient button” located on the front site allowing the patient to press it in specific cases such as sickness, going to bed, taking pills…. A special mark will be then placed into the record so that the doctors or technicians can quickly pinpoint these areas when analyzing the signal.

RECORDER

The size of the recorder differs depending on the manufacturer of the device. The average dimensions of today's Holter monitors are about 110x70x30 mm but some are only 61x46x20 mm and weigh 99 g. Most of the devices operate with two AA batteries. In case the batteries are depleted, some Holters allow their replacement even during monitoring. Most of the Holters monitor the ECG just in two or three channels. Depending on the model (manufacturer), different counts of leads and lead systems are used. Today's trend is to minimize the number of leads to ensure the patient's comfort during recording. Although 2/3 channel recording has been used for a long time in the Holter monitoring history, recently 12 channel Holter's have appeared. These systems use the classic Mason-Liker lead system, thus producing the signal in the same representation as during the common rest ECG and/or stress test measurement. These Holters then allow to substitute stress test examination in cases the stress test is not possible for the current patient. They are also suitable when analyzing patients after myocardial infarction. Recordings from these 12-lead monitors are of a significantly lower resolution than those from a standard 12-lead ECG and in some cases have been shown to provide misleading ST segment representation, even though some devices allow setting the sampling frequency up to 1000 Hz for special-purpose exams like the late potential. Another interesting innovation is the presence of a triaxial movement sensor, which records the patient physical activity, and later shows in the software three different statuses: sleeping, standing up, or walking. This helps the cardiologist to better analyze the recorded events belonging to the patient activity and diary. Holter monitoring is a very useful part of an ECG. Some modern devices also have the ability to record a vocal patient diary entry that can be later listened to by the doctor.

ANALAZING SOFTWARE

When the recording of ECG signal is finished (usually after 24 or 48 hours), it is up to the physician to perform the signal analysis. Since it would be extremely time demanding to browse through such a long signal, there is an integrated automatic analysis process in each Holter software which automatically determines different sorts of heart beats, rhythms, etc. However, the success of the automatic analysis is very closely associated with the signal quality. The quality itself mainly depends on the attachment of the electrodes to the patient body. If these are not properly attached, electromagnetic disturbance can influence the ECG signal resulting in a very noisy record. If the patient moves rapidly, the distortion will be even bigger. Such record is then very difficult to process. Besides the attachment and quality of electrodes, there are other factors affecting the signal quality, such as muscle tremors, sampling rate and resolution of the digitized signal (high quality devices offer higher sampling frequency). The automatic analysis commonly provides the physician with information about heart beat morphology, beat interval measurement, heart rate variability, rhythm overview and systems also perform spectral analysis, ischemic burden evaluation, graph of patient's activity or PQ segment

analysis. Another requirement is the ability of pacemaker detection and analysis. Such ability is useful when one wants to check the correct pacemaker function.

Indication:

- Palpitation
- Light -headedness or dizziness(syncope)
- Chest discomfort
- Shortness of breath
- The patient has found to have significant cardiac or conduction disorder and holter monitoring is necessary as a part of the evaluation and management.
- The patient has a heart condition associated with high incidence of serious cardiac arrhythmias and/or Myocardial ischemia.
- To evaluate the effect cardiac medication.
- The patient has a pacemaker and clinical findings suggest possible pcemaker malfunction.

It may be used to diagnose:

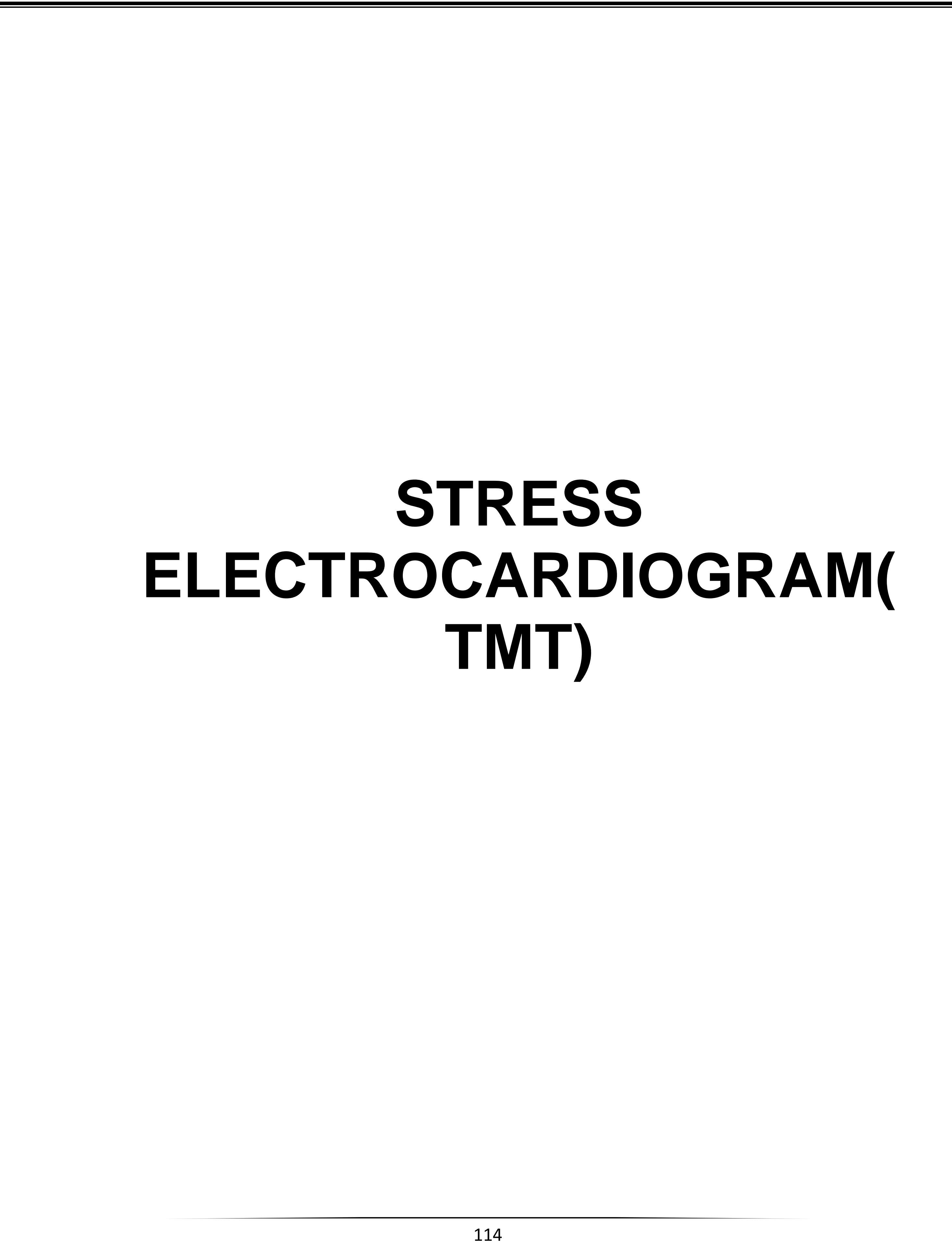
- Atrial fibrillation or flutter
- Multifocal atrial tachycardia
- Palpitations
- Paroxysmal supraventricular tachycardia
- Reasons for fainting
- Slow heart rate (bradycardia)
- Ventricular Tachycardia

Patient preparation

- Make awareness about the monitoring modality.
- Clean the lead position with spirit which reduces the skin resistances.
- Place the leads properly and comfortable to the patient.

Instructions to the patient

- Please read the instructions carefully. Failure to allow these instructions may require the procedure to be rescheduled.
- You can take all your medications before this test.
- Wear comfortable clothing.
- You will be unable to bath during monitoring.
- Take care of the lead positions.
- Keep a dairy mentioning every events and time during monitoring with you.



STRESS ELECTROCARDIOGRAM(TMT)

INTRODUCTION

Exercise is the body’s most common physiologic stress, and it places major demands on the cardiopulmonary system. For this reason exercise can be considered the most practical test of cardiac perfusion and function. Exercise testing is a non invasive tool to evaluate the cardiovascular system’s response to exercise under carefully controlled conditions.

Bayes' theorem:

‘The posttest probability is proportional to the pretest probability’

BASIC PRINCIPLES

Two basic principles of exercise physiology are physiologic principle and path physiology.

The **physiologic principle** is total body oxygen and myocardial oxygen uptakes are distinct in their determinants and in the way they are measured are estimated. Total body or ventilator oxygen uptake is the amount of oxygen that is extracted from inspired air as the body performs work. Myocardial oxygen uptake is the amount of oxygen consumed by the heart muscle. Myocardial oxygen uptake estimated by the product of heart rate and systolic blood pressure.

The second principle is **the path physiology**, considerable interaction takes place between the exercise test manifestations of abnormalities in myocardial perfusion and function. The electro physiologic response and angina are closely related to myocardial ischemia, whereas exercise capacity. Systolic blood pressure and heart rate responses to exercise can be determined the presence of myocardial ischemia.

INDICATIONS

There are many indications for exercise testing which includes

- Evaluation of patient with chest pain
- Determination of prognosis and severity of disease
- Evaluation of the affects of medical and surgical therapy
- Screening for latent coronary disease
- Early detection of labile hypertension
- Evaluation of congestive heart failure.
- Evaluation of arrhythmias
- Evaluation of functional capacity and formulation of an exercise prescription
- Evaluation of congenital heart disease

CONTRAINDICATIONS

There are many contraindications for exercise testing which includes absolute contra indication and relative contra indication.

Absolute

- Patient with an acute MI.
- Patients suffering from acute myocarditis or pericarditis.
- Patients exhibiting signs of unstable progressive angina.
- Patients with second or third degree heart block.
- Patients with known severe left main disease.

Relative

- Aortic stenosis.
- Suspected left main equivalent.
- Severe hyper tension, idiopathic hypertrophy.
- Sub aortic stenosis.
- Severe ST segment depression at rest.
- Congestive heart failure.

PROCEDURE

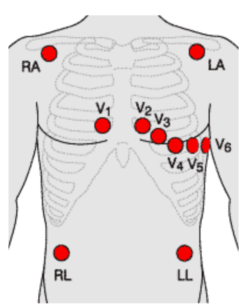
EQUIPMENT NECESSARY IN EXERCISE LABORATORY

- Treadmill with specially designed electrode and cables.
- 12 channel continuous ECG monitor and recorder.
- Sphygmomanometer and stethoscope.
- Cardio-pulmonary resuscitative equipments including defibrillator, oxygenator etc.,
- Emergency drugs.

PATIENT PREPARATION

The patient should be instructed not to eat or smoke at least 2 to 3 hours prior to the test and to come dressed for exercise. A brief history and physical examination should be performed to rule out any contra indications to testing.Specific questioning should determine which drugs are being taken, and potential electrolyte abnormalities should be considered.It is generally no longer considered necessary .The patient should receive careful explanations of why the test is being performed and of the testing procedure. Including its risk and possible complications. Proper skin preparation is essential for the performance of an exercise test. The general areas for electrode placement should be shaved if they have hair and should be clean with an alcohol-saturated gauze pad.

MASON-LIKAR ELECTRODE PLACEMENT



Because of a 12-lead ECG cannot be obtained accurately during exercise. The electrodes are placed at the base of the limbs for exercise testing. Right and left arm electrodes should be placed far laterally on the edge of the shoulders. The ground electrode can be on the back of the cardiac field and the left leg electrode should be below the umbilicus. The V1, V2 and V4 precordial electrodes

should be placed in the appropriate interspaces and V3,V5 and V6 aligned according to them.

HEART RATE

The maximum heart rate with exercise is a fundamental physiologic parameter. The maximum achievable heart rate is unique for each patient but can be estimated by using regression equations that adjust for the patient age, the most familiar equations is HRmax=220-Age.

There is considerable variability with this equations, especially in patients with CAD who are taking beta-blockers, Newer equation is HRmax=164-(0.7*age).

Although, numerous devices have been developed to measure the blood pressure during exercise. The time proven method of the physician holding the patients arm with a stethoscope placed over the brachial artery remains the most reliable method to

BLOOD PRESSURE MEASUREMENT

obtain the blood pressure. If systolic BP appears to be increasing slightly or decreasing, it should be taken again immediately, If a drop in systolic BP of 10 to 20mm Hg or more occurs, or if it drops below the value obtained in the standing position prior to testing, the test should be stopped.This is particularly important in patients who have heart failure, a prior myocardial infarction, or are exhibiting signs or symptoms of ischemia. An increase in systolic blood pressure to 115mmHg are also indications to stop the test.

TREAD MILL PROTOCOL

- Bruce protocol
- Naughton protocol
- Weber protocol
- ACIP(asymptomatic cardiac ischemia pilot)
- Modified ACIP

The treadmill protocol should be consistent with the patient’s physical capacity and the purpose of the test. In healthy individuals, the standard **Bruce protocol** is popular, and a large diagnostic and prognostic data base has been published using this protocol. The Bruce protocol has 3- minute periods to allow achievement of a steady state before work -load is increased, In older individuals or those whose exercise capacity is limited by cardiac disease, the protocol can be modified by two 3-minute warm-up and 5%grade **(modified Bruce).**

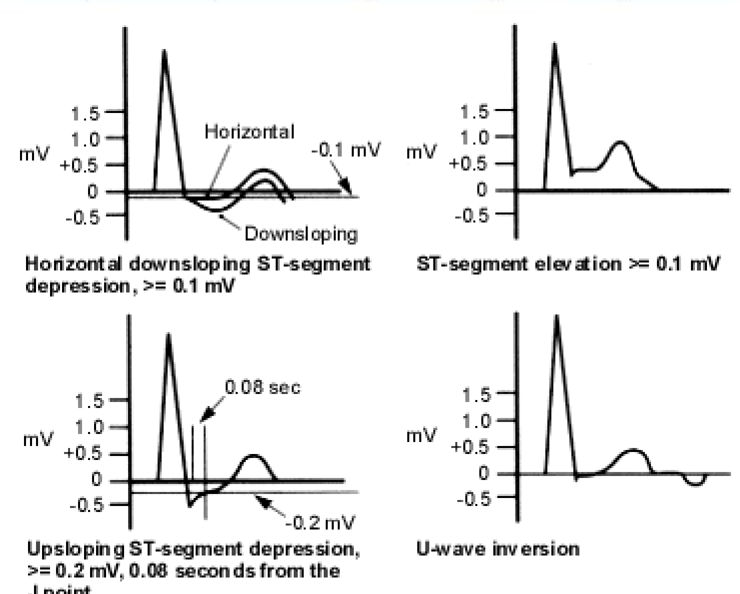
ELECTROCARDIOGRAPHIC RESPONSES

In normal persons the PR, QRS and QT intervals shorten as heart rate increases, P amplitude increases, and the PR segment becomes progressively more down sloping in the inferior leads. J point depression is a normal finding during exercise. In patients with CAD, exercise can cause an imbalance between myocardial oxygen supply and demand, which can result in an alteration in the ST segment of the electrocardiogram. These changes are

the foundation of the exercise test clinically. The diagnostic electrocardiographic criteria and leads that exhibit abnormalities during exercise have been the source of significant debate.

Bruce Protocol

Stage	Minutes	% grade	km/h	MPH	METS
1	3	10	2.7	1.7	5
2	6	12	4.0	2.5	7
3	9	14	5.4	3.4	10
4	12	16	6.7	4.2	13
5	15	18	8.0	5.0	15
6	18	20	8.8	5.5	18
7	21	22	9.6	6.0	20



1.00 mm or greater ST segment depression that is horizontal or 60 to 80 millisecond after the J-point .ST-segment depression greater than 1 mm that is downsloping is generally indicative of more severe CAD. Most ischemic ST changes occur in the lateral precordial leads.The significance of STsegment elevation depends on the presence or absence of Q waves. When ST elevation occurs in the presence of resting electrocardiogram ,it is usually indicative of severe transmural ischemia ,it can be arrhythmiogenic ,and it localizes the ischemia .conversely ,exercise –induced ST-segment elevation occurring in lead with Q waves is more common and is related to the presence of dyskinetic areas .

TEST TERMINATION

ABSOLUTE

• Drop in systolic BP >10mmHg from baseline despite an increase in workload, when accompanied by other evidence of ischemia
• Moderate to severe angina
• Increasing nervous system symptoms
• Signs of poor perfusion
• Technical difficulties in monitoring electrocardiogram or systolic blood pressure
• Subject's desire to stop
• Sustained VT
• ST elevation >=1mm in leads without diagnostic Q waves.

RELATIVE

• Drop in systolic BP >=10mmHg from baseline despite an increase in workload, in the absence of other evidence of ischemia.
• ST or QRS changes such as excessive ST depression.
• Arrhythmias other than sustained VT, including multifocal PVCs, SVT, triplets of PVCs, heart block or bradyarrythmias.
• Fatigue, shortness of breath,wheezing ,leg cramps, or claudication.
• Development of bundle branch block or intraventricular conduction delay that cannot be distinguished from VT.
• Increasing chest pain.

FALSE POSITIVE AND FALSE NEGATIVE RESPONSES

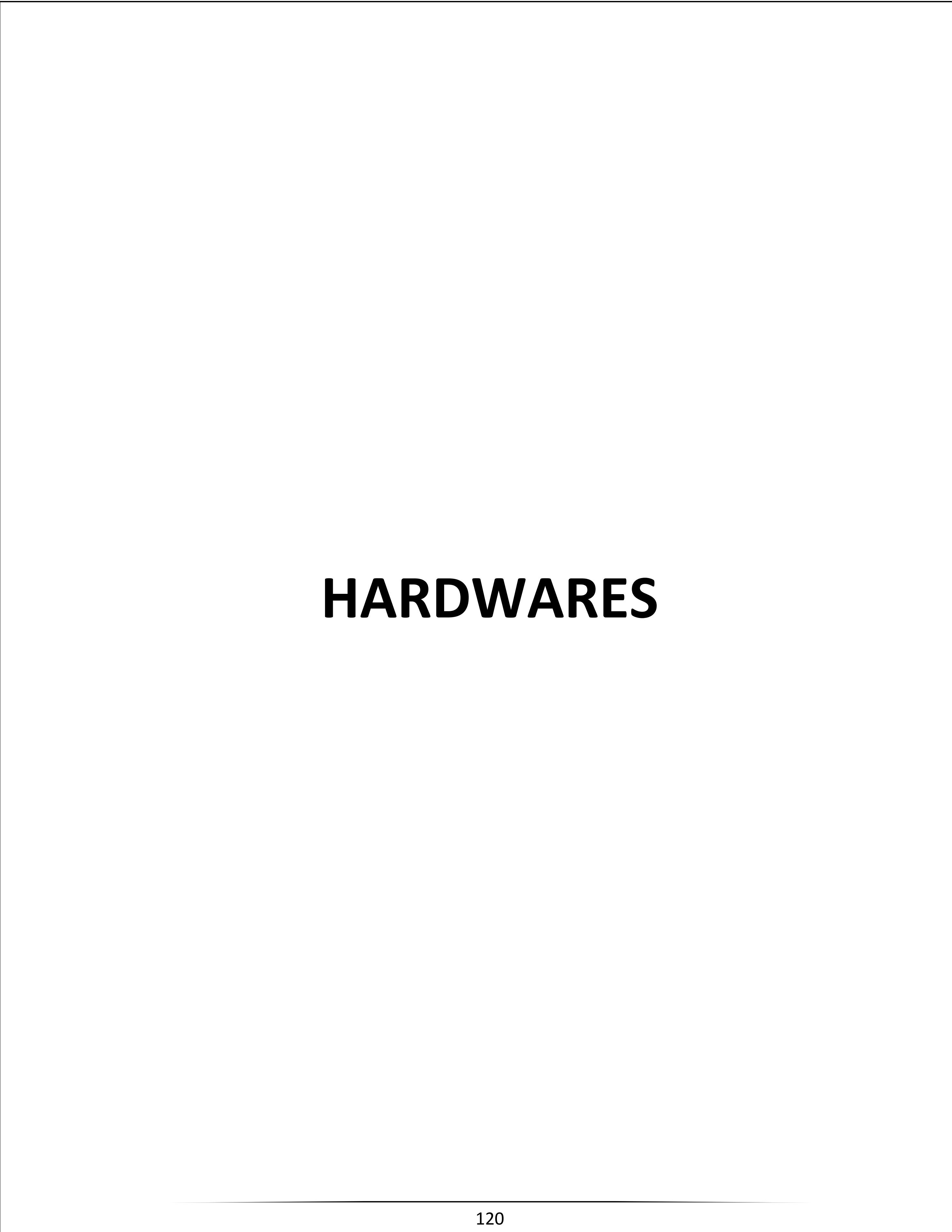
The factors associated with false –positive or false – negative responses should also be considered before the test. A false positive response is defined as an abnormal exercise test response in a person without significant heart disease and causes the specificity to be decreases. A false negative response occurs when the test is normal in person with disease and causes the sensitivity of the test to be reduced.

False – Positive

• Resting repolarisation abnormalities ,cardiac hypertrophy ,accelerated conduction defects.
• Digitalise.
• Non ischemic cardiomyopathy ,hypokalemia ,vasoregulatory abnormalities ,mitral valve prolapse.
• Pericardial disease, coronary spasm in the absence of CAD, anemia female gender.

False – Negative

• Failure to reach ischemic threshold secondary to medications.
• Monitoring an insufficient number of leads of leads to detect ECG changes.
• Angiographically significant disease compensated by collateral circulation.
• Musculoskeletal limitations preceding cardiac abnormalities.



HARDWARES

TOOLS FOR CATHETERIZATION

Tools for catheterization, for either diagnostic or monitoring purposes consists of catheters, guidewires, needle, introducers, transducers, and protective sleeve adaptors.

VASCULAR ACCESS NEEDLE

Percutaneous vascular access into either the artery or the vein is gained via introduction of a needle. Percutaneous cannulation needles frequently referred to as Seldinger needle. Vascular needles are constructed of rigid stainless steel with a bevelled tip possessing two sharp cutting edges. This type of point aids in puncturing the vessel and smoothly sliding through it. Percutaneous entry needles can be categorized according to the number of parts: One-part needle, Two-part needle and Three-part needle.

Sizes: The size of the needle is measured in needle gauge, which refers to outside diameter.

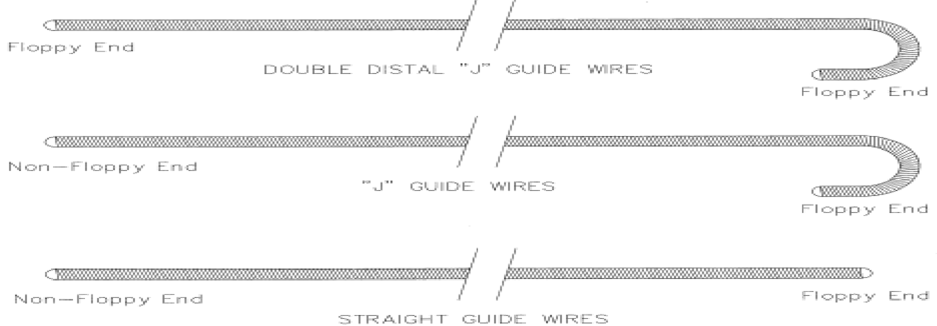
Types: Reusable stainless needles, such as the Cournand, Riley, and Potts-Cournand needles have been traditionally used in the cardiac catheterization laboratory for percutaneous cannulation of the femoral or brachial artery or vein. The peel-away needle is used most commonly for transvenous pacemaker insertion into the subclavian vein.

GUIDEWIRES

Guidewires are very delicate devices used in cardiac catheterization to straighten a catheter curve, traverse vessels, and permit percutaneous insertion of an introducer or catheter. Guidewires are constructed of the stainless steel. Some guidewires are also coated a thin film of Teflon to reduce the friction coefficient of the guidewire within the

catheter. Some Guidewire is a hydrophilic coated. The Terumo Glide Technology hydrophilic coating allows smooth and rapid movement of the guidewire through tortuous vessels, and it enables crossability over difficult lesions. It is able to resist kinks and retains its shape for consistent reliability. It has a 1:1 torque ratio and enhanced radiopacity.

Guidewires basically are of two shapes, straight and ‘J’ curve, and may have either a fixed or movable core.



Straight guidewire: It has a flexible tip (generally 3 cm) and is appropriate for passage through vessels of linear configuration.

J-Curve guidewire: It has generally a 1.5, 3, or 6 mm curved radius at its tip. Use of the J-curve guidewire for negotiating the external jugular vein has been associated with a higher incidence of successful passage than the use of a standard straight wire.

Fixed core guidewire: It is a type of guidewire with a rigid core that is fixed at the proximal end of the wire but is usually unattached at the distal end.

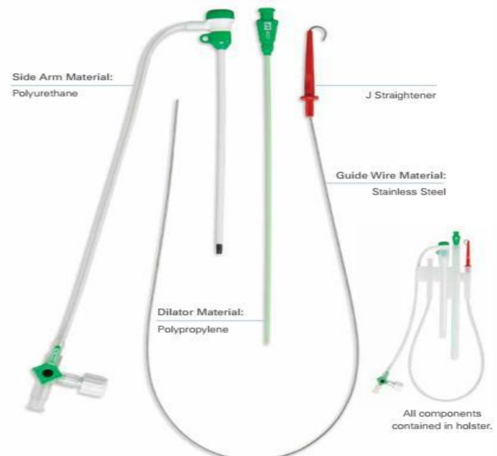
Movable core guidewire: Guidewires that have the straight core attached only at the distal end are designed to allow movement of the tip of the core for the purpose of increasing or decreasing the length of flexible tip.

Sizes: Most guidewires are available in sizes ranging from 0.018 to 0.038 inch (0.4 to 1.0mm). Guidewires are available in length varying from 50 to 300 cm. In cardiac catheterization laboratory, a 150cm long guidewire is commonly used.

Desired features: Important considerations in guidewire selection include stiffness, flexibility, and smoothness.

SHEATHS AND DILATOR

Vascular sheaths generally contain a removable dilator, a diaphragm that leakage of blood or air into the sheath, and a sidearm connected to a threeway stopcock, which allows the operator to record pressure measurements, flush the sheath, and infuse medications. The dilator is made up of stiff plastic that allows it to pass through fibrous subcutaneous tissue or atherosclerotic/calcified vessel. For femoral and brachial diagnostic catheterizations, 7F, 6F and 5F catheter sheaths are most commonly used.



For radial cases 4F or 5F or 6F catheter sheath, although larger sized sheaths are sometimes used in interventional cases. The length of sheath used routinely varies from 6 to 35 cm. For most cases, an 11cm sheath is adequate. Longer sheaths of 25 to 35 cm are selected when one encounters tortuous femoral and iliac arteries to facilitate torque control of the diagnostic catheter.

FRENCH CATHETER SCALE

The French catheter scale (most correctly abbreviated as Fr, but also often abbreviated as FR or F) is commonly used to measure the outer diameter of cylindrical medical instruments including catheters, needles etc. In the French Gauge system as it is also known, the diameter in millimeters of the catheter can be determined by dividing the French size by 3, thus an increasing French size corresponds with a larger diameter catheter. The following equations summarize the relationships:

D (mm) = Fr/3 or Fr = D (mm)*3
1 Fr = 0.335 mm or 0.013 inches

CATHETERS

A catheter is a hollow flexible tube that can be inserted into a body cavity, duct or vessel. Catheters thereby allow injection of fluids. The process of inserting a catheter is catheterization. The ideal catheter is one that provides the most accurate information (hemodynamic pressure and angiographic opacification) with no risk to the patient Some basic design features that are important for successful catheterization include material

used, curve, shape, flexibility, memory, catheter tip, end and side holes, catheter hub, and radiopacity.

MATERIAL

Dacron: Catheter made of woven Dacron are very maneuverable and flexible. Most woven Dacron catheter are covered with a polyurethane coating to increase surface smoothness and reduce vascular trauma. Examples: Goodale-Lubin catheter, and Sones catheter.

Polyurethane: Catheters extruded from polyurethane have an excellent tensile properties memory (they recover their original shape well at body temperature). Which reduce the vascular trauma or perforation. Polyurethane can be reshaped if immersed in boiling water or exposed to steam for varying periods of time, depending on wall thickness. Examples: pigtail angiographic catheters and the original Judkins coronary catheters.

Polyethylene: Polyethylene is utilized in both performed and custom-made catheters. Its degree of stiffness lies somewhere between the stiffness of polyurethane and that of Teflon. Because polyethylene dose not soften much at body temperature, it also maintains its shape and thus is very popular for selective catheterization. Examples: National institute of health catheter (NIH), Cournand catheter.

Teflon: Teflon catheters are stiffest vascular catheters. The advantage of is its extremely low friction coefficient, which reduces vascular trauma, increases ease of insertion and passage, and improves flow rates of contrast media. Examples: Introducer sheath.

Polyvinylchloride: PVC catheters are softer than catheters of the mentioned above materials. This characteristic makes the material quite supple and flexible and therefore ideal for flow directed catheters. PVC has a high friction coefficient. Examples: Flow directed catheters.

CURVE SHAPE

The curve shape of catheter tip is an important consideration in catheters used for selective angiographic studies. Performed catheters are available in an array of primary and secondary curvatures to facilitate selective placement and positional stability of the catheter. A nonspecific, gentle curve in performed catheters is adequate for many purposes, including pressure measurements and nonselective catheter placement.

FLEXIBILITY

The construction of the catheter determines its flexibility and the safety and ease with which it can be manipulated. The ability to finely control the rotation or torque of the catheter's tip by manipulation of the catheters hub is an essential feature for successful selective catheterization.

MEMORY

Memory is the ability of the catheter to retain its original performed shape and is a function of the material used in catheter construction. Catheters with performed curves often must be straightened with a guidewire to permit passage through the vascular

system. It is important that upon removal of the guidewire, the catheter resumes its original performed curve.

TIP

The catheter tip should be neither blunt nor too sharp and should be soft and flexible. Catheters with a slightly tapered ("bullet-nose") tip produce the least amount of trauma during insertion and reduce the risk of vascular dissection, which can occur with the passage of blunt-tip catheter.

END AND SIDE HOLES

Most of the cardiac catheters have an end hole at the tip of the catheter, permitting percutaneous introduction over a guidewire. Although the presence of an end hole at the tip of the angiographic catheter allows more complete flushing of the catheter, thus reducing the risk of thrombus formation at the tip, the absence of an end hole (with only side holes) reduces the risk of intramural injection of contrast medium and catheter recoil during angiography.

HUB

Whether the catheter hub is made of plastic material, it should not be significantly smaller than the lumen of the catheter. Tapered hubs permit easier insertion of a guidewire.

SIZES

Diameter: The outside diameter of cardiac catheter continues to be measured in French (Fr) gauge (1 Fr = 0.335 mm). The inside diameter of the catheter will vary depending on the thickness of the catheter wall. The catheter size selection represents a compromise between use of the largest size possible for more accurate pressures and faster delivery of large boluses of dye and use of the smallest size possible to reduce the risk of vascular trauma, thrombus formation, or bleeding.

LENGTH

Catheter length selection is determined by the insertion site and the desired eventual location of the catheter tip. In general, the catheter should be no longer than necessary to reach the intended location. Right heart catheters for adult are generally 100 to 125 cm long, with a shorter catheter suitable for internal jugular vein insertion. Left heart catheters are generally 100 to 110 cm long; this is usually sufficient for placement of the catheter tip in the left ventricle via the femoral artery approach.

RIGHT HEART CATHETERS

GOODALE-LUBIN CATHETER

The Goodale-luben is constructed of woven Dacron coated with polyurethane. Unique characteristics are the two laterally opposed side holes near the distal open end. It is reusable. The Goodale-Lubin catheter is a standard catheter used in cardiac catheterization laboratory for routine right heart pressure measurements and blood

sampling which has 45^0 angle at its tip. It available in sizes 4 through 8 Fr, with 80, 100, and 125 cm lengths.

BALLOON FLOTATION CATHETER

Balloon flotation catheters are PVC, multilumened, right heart catheters with a balloon at the tip, which, when inflated with air, carries the catheter along with the blood flow through the right side of the heart and into the pulmonary artery, allowing measurement of PA pressure and PAWP. The balloon flotation catheter is particularly useful for catheterization in children, in whom exact anatomy is not always predictable. Standard adult sizes are 5, 6, and 7 Fr, 110 cm long. Pediatric sizes are 4 and 5 Fr, 60 cm long. The balloon capacity of the different sizes varies and is marked on the hub of the catheter.

COURNAND CATHETER

Originally designed by Andre Cournand in 1939. The Cournand catheter is a standard wall, end-hole radiopaque woven Dacron catheter with an outer coating of polyurethane. This is multipurpose right heart catheter. The Cournand catheter is available in sizes 5 through 8 Fr, with 100 and 125 cm length.

LEHMAN CATHETER

The Lehman catheter is a thin wall variation of the Cournand catheter with a slightly shorter distal curve. Its thinner wall increases the inside diameter of the catheter and decreases its stiffness. It is reusable.

ANGIOGRAPHIC CATHETERS

GENSINI CATHETERS

The Gensini catheter is made of woven Dacron with a polyurethane coating and three pairs (six) of laterally opposed oval side holes within 1.5 cm of its open tip. The tip is tapered to provide a close fit over the appropriate-size guidewire. The catheter is intendend specifically for percutaneous insertion into the right or left side of the heart for purposes of retrograde aortic and ventricular as well as pulmonary and vena caval angiographic studies. The Gensini catheter is available in sizes 5 to 8 Fr and lengths of 80, 100, and 125 cm.

NATIONAL INSTITUTES OF HEALTH CATHETER (NIH)

NIH catheter is closed-end, side-hole catheter with a gentle curve. It is a thin-wall catheter constructed of woven Dacron reinforced with a nylon core, the NIH catheter has six side holes near its flexible tip. The NIH catheter is used for angiographic visualization of either the RV or LV, the arterial or pulmonary vasculature, and the great veins. NIH catheter is available in sizes 5 to 8 Fr and length 50, 80, 100 and 125 cm.

LEHMAN VENTRICULOGRAPHY CATHETER

It’s a thin-wall, closed-end, woven Dacron catheter with polyurethane coating. The unique feature of this catheter is its slightly curved tip, tapered to 5 Fr, with four side holes

beginning 2.5 cm from the tip. This catheter is usually fairly stable in the LV and is designed for primarily ventriculography. Sizes 5 through 8 Fr, with 80, 100 and 125 cm lengths.

PIGTAIL ANGIOGRAPHIC CATHETERS

A pigtail angiographic catheter is made of polyurethane or polyethylene with a tapered tip, the terminal 5 cm of which is coiled back onto itself in a tight loop ("pigtail"). The catheter is available with an open or closed end with 4 to 12 nonlaterally opposed side holes located in the terminal 5 cm of the catheter. This is most commonly used catheter for left ventricular and aortic angiography and is also successfully used for pulmonary angiography. Pigtail catheter to allow safer passage across cardiac valves and safer injection of contrast medium into ventricles and great vessels without causing any trauma or perforation. The Van Tassel angled pig tail catheter is employed for angiographic studies of the LV and aorta in patients with normal or stenosed aortic valves. This design is particularly helpful for crossing a stenotic aortic valve. The unique feature of this end hole catheter is a 145_0 or 155_0 angle 7 cm from the tip.

BERMAN ANGIOGRAPHIC CATHETER

The Berman angiographic catheter is a double-lumen PVC catheter with side holes near the balloon tip. The catheter is intended selective pulmonary angiography in adults or for right and left heart catheterization and angiography in infants and small children.

CORONARY ANGIOGRAPHIC CATHETER

JUDKINS-TYPE CORONARY CATHETER

Coronary angiography can be completed using Judkins catheters from the femoral approach in more than 90% of patients. Judkins catheters have special preshaped curves and tapered end-hole tips. The Judkins left coronary catheter has a double curve. The length of the segment between the primary and secondary curve determines the size of the catheter (i.e., 3.5, 4, 5, or 6 cm). The proper size of the left Judkins catheter is selected depending on the length and width of the ascending aorta.

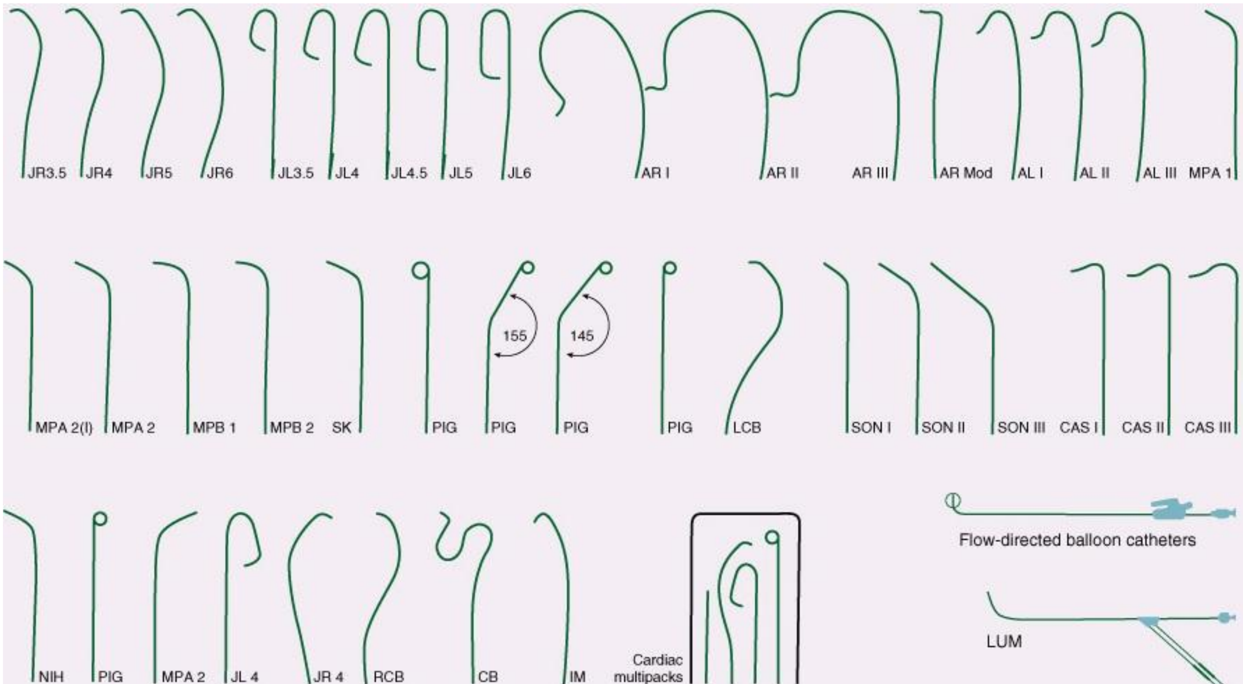
The Judkins right coronary catheter is sized by the length of the secondary curve and comes in 3.5, 4 and 5 cm sizes. The 4cm catheter is adequate in most cases.

THE BRACHIAL TYPE OR RADIAL TIG CATHETER

The TIG is an end-hole catheter, with a single side hole and is well suited for the transradial approach. This catheter is user friendly; a single catheter helps to cannulate both left and right coronary without exchanging like Judkins catheters.

AMPLATZ-TYPE CATHETER

The left Amplatz-type catheter is a preshaped half-circle with the tapered tip extending perpendicular to the curve. Amplatz catheter sizes (left 1, 2 and 3 and right 1 and2) indicates the diameter of tip curve. The catheter for selective catheterization of the left or right coronary artery or anomalous origin from the percutaneous femoral approach. The modified curve of Amplatz are also available.



MULTIPURPOSE CATHETER

MP catheters are catheters that are primarily straight with an end-hole and two side holes placed close to the tapered tip. Preshaped, mildly angled configurations are also available. The MP catheter can be used for left and right coronary injections.

CORONARY BYPASS CATHETERS

It is specifically designed and curved for post-operative opacification of right and left coronary bypass graft from the femoral percutaneous approach.

Left coronary bypass catheter: This catheter for visualization of a left coronary bypass graft affixed to the left anterior descending or circumflex artery. The tip has a 90_0 bend with a secondary curve of approximately 70_0.

Right coronary bypass catheter: This catheter is the same as the left, except for a $110\ ^0$to 120^0 bend of the catheter tip. It is used for visualization of either right or left coronary bypass graft.

Internal memory bypass catheter: Designed for opacification of both right and left internal memory artery bypass grafts, this is similar to the Judkins right coronary catheter with a shallower primary curve ($80\ ^o$ to 85^0) and a 1.5 to 2 cm tip.

PACING ELECTRODES

These are no-lumen catheters with two platinum electrodes located 1 or 2.5 cm apart at the tip of the catheter and two electrode connectors at the base. They are employed for temporary atrial or ventricular pacing. Standard pacing electrodes come in sizes 5, 6 and 7 Fr, 110 cm long.

MANIFOLD

A variety of manifold systems exist. One common design is a three component manifold that has three stopcocks attached. The first stopcock is connected to a pressure transducer, the second is attached to flush solution and third is attached to the contrast agent choice.

GUIDE CATHETERS

Guiding catheters are like angiography catheters only difference is that guiding catheters are more stiffer & firm as it carries Balloon catheters, PTCA wires and stent delivery system. Mild stiffness comes due to the wire braided design.

- Most designs are derived from standard Judkins and Amplatz diagnostic catheters with some relaxation of acuteness.
- Soft or atraumatic tip.
- The ideal guide catheter align coaxially with the proximal portion of the target vessel without obstructing it and bracing sufficiently against other parts of aortic root to provide axial support to pass dilating catheter.
- Stiffer shaft, larger internal diameter, a shorter and angulated tip (110 vs 90 degree) and reinforced construction (3 vs 2 layers).
- Larger catheter – better opacification, guide support and pressure monitoring at the cost of increased risk of ostial trauma, vascular complications and possibility of kink of shaft.
- Outer layer – polyurethane or poly ethylene for overall stiffness, middle layer composed of a wire matrix for torque generation and inner coating of Teflon for smooth passage of balloon catheter.

Limitations of Judkins

- Primary curve is fixed – may not be co axial with index artery.
- Difficult to pass balloons – catheter takes90 degrees to treated artery.
- Point of contact with root of aorta is very high.

EBU catheter (Extra Back Up catheter)

- Larger lumen guide balancing flexibility and support
- Workhorse construction suitable for various anatomies and takeoffs
- Flexible distal segment enables you to engage for backup support
- Supportive secondary curve for backup support and curve retention
- Enhanced visualization
- Larger lumens to maximize contrast flow for enhanced visualization
- Radiopaque marker band and nylon shaft
- Full-wall technology construction
- Robust, thinner walls without compromising support or curve retention

• Full range of coronary and peripheral curves.
• 5F – 8F sizes available

RCA interventions

• JR or hockey stick guide usually preffered.
• If extra support needed – CTO, tortuosity – AL1 – larger secondary curves rests on aortic root.
• MP guide – abnormal take off of RCA from aorta especially inferior orientations
• 3DRC (Three dimensional right curve) – tortuous or bend anatomy, posterior and superior take off of RCA.

SVG (saphenous vein grafts) and LIMA

• Usual – JR
• MP or AL1 – abnormal positions and take offs
• Left coronary bypass catheter or right coronary bypass catheter
• Internal mammary artery (IMA) catheter is designed for both right and left Internal Mammary arteries. It is shaped like a JR catheter but with a steeply angled tip (80 to 85^0).

BACK UP FORCE

Three factors were found to be associated with back up force:
1. Catheter size
2. Angle (theta) of the catheter on the reverse side of the aorta
3. Contact area

• The angle (theta) determines the vertical vector that can dislodge the guiding catheter. A smaller Cosθ results in a greater backup force. This suggests that the lower position is preferable as the point of contact on the reverse side of the aorta because the angle approaches 90 degrees.
• The larger French size guiding catheter produced a significantly greater backup force
• Backup (EBU/XB) type catheters have greater backup force than a JL4 with deep engagement.

SIDE HOLES

Side holes are useful where the pressure gets frequently damped as in RCA interventions, CTO interventions or sole surviving artery or left main interventions

Advantages:

• Prevent catheter damping (occlusion of the coronary ostium). Allow additional blood flow out of tip, to perfuse the artery.
• Avoid catastrophic dissections in the ostium of the artery.

Disadvantages

• Suboptimal opacification
• Reduction in back up support provided because of weakness of catheter shaft and the kinking at side holes.

Guide can cause

- coronary spasm
- non-coaxial alignment

Improve the delivery of coronary stents to complex lesions.Child catheters 4/5 F 100cm. Mother catheter - 6 F guiding catheter 100cm. Superior trackability of the 4F child catheter. Increased backup support of the mother-child system. 4F mother-child system provided > 90% success rate.

TORTUOUS VESSELS

- At least two or more than 75 degree bend proximal to the target lesion or one proximal bend 90 degrees.
- LAD – XB, EBU
- LCX – EBU
- Deep seating of guide – RCA and Cx clockwise or retract balloon.

TECHNIQUES TO STABILIZE A GUIDE

1. Second angioplasty wire/Buddy wire-advanced parallel to the first one. Straightens tortuous vessel and provides better support for device tracking.
2. Second wire in side branch- useful in anchoring the guide.
    a. Provides for better backup and retraction of the guide when necessary, without loss of position.
    b. Also prevents the guide from being sucked in beyond the LM when pulling back balloon catheters
    c. Cause unnecessary denudation of endothelium in that vessel.
3. Change to a stronger guide.
4. Anchoring balloon.
    a. Second small balloon (1.5-2mm diameter) inserted in a small proximal branch.
    b. Inflated at 2ATM- anchor the guide.

5. Change the current sheath to very long sheath.
6. Double guide technique.
    a. insert a small guide in current guide.

COMPLICATIONS

- Embolism – air, atheroma, thrombus.
- Dissection of coronary, aorta, abdominal aorta or iliac artery
- Frequent bleed backs.
- Aortic debris – 24 – 65%, JL and MP.
- Least with JR.
- Less forceful injections till guide is coaxial
- More aggressive guide – more chance of dissection

CORONARY GUIDE WIRES

Guidewire selection is an essential component of successful coronary intervention.

PURPOSE OF THE GUIDEWIRE

Guidewires are used to facilitate successful delivery of interventional devices. Guidewires have the following functions:

- To track through the vessel,
- To access the lesion.
- To cross the lesion atraumatically.
- To provide support for interventional devices.

STRUCTURE OF GUIDE WIRES

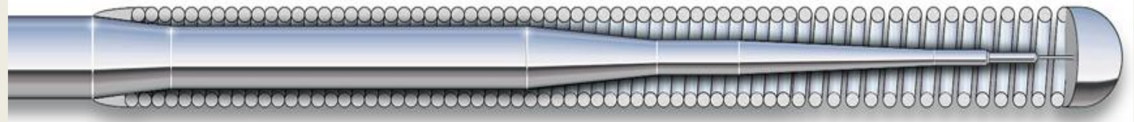
CORE

The inner part of the guidewire is referred to as the core. It extends through the shaft of the wire from the proximal to the distal part where it begins to taper. It is the stiffest part of the wire that gives the stability and steerability to the guidewire from its proximal end to the distal tip. The core material affects the flexibility, support, steering and trackability of the wire while its diameter influences the flexibility, support and torque of the wire. The central core gives an adequate rail support. This material will be stainless steel, nitinol or elastinite.

Core material:

The most popular core materials are stainless steel and nitinol, and other metal alloys.

- Stainless steel provides excellent support, transmission of push force and torque characteristics; these wires also have good shape ability ,however it is less flexible in comparison to newer core materials and more susceptible to kinking.



- Nitinol is a super-elastic alloy of nickel and titanium designed for resiliency and kink resistance. It provides excellent flexibility and steering and is much more durable than stainless steel. Since nitinol maintains its shape and resists kinking, it is less susceptible to deformation during percutaneous coronary intervention (PCI) and could be used for treatment of multiple lesions and lesions with tortuous anatomy. However, it has less torqueability than stainless steel.

Core diameter:

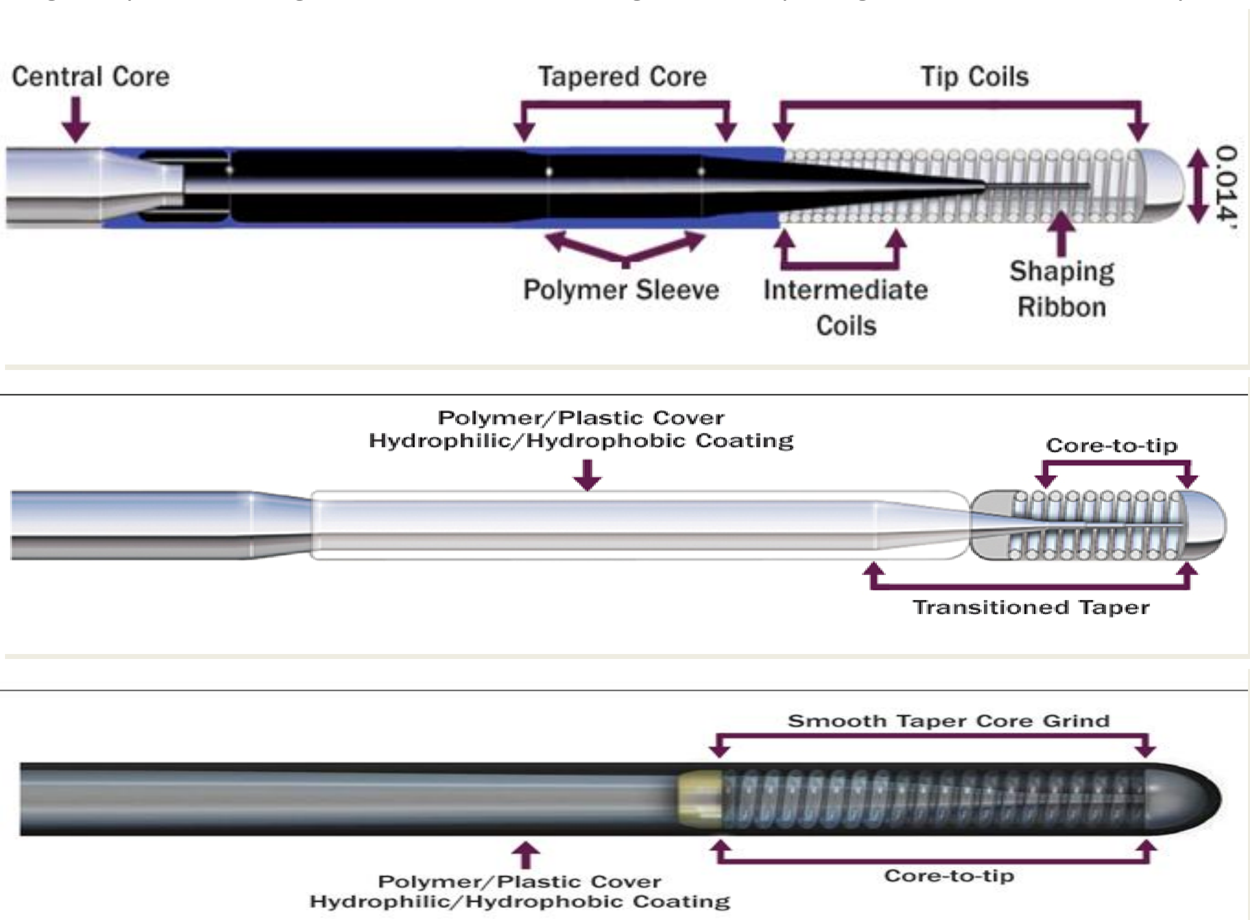
The diameter of the core influences the performance of the wire. Larger diameters improve the support, while smaller diameters have the opposite effect and enhance the

Prolap

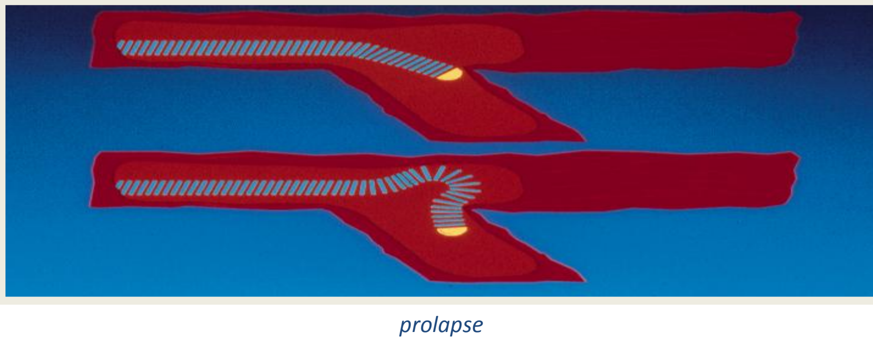
flexibility of the wire. Larger diameters allow 1:1 torque response where the distal tip of the wire turns one turn for every turn on the proximal end for optimal steerability.

Core taper:

The core of a guidewire usually is tapered along its length. The taper may have variable length and it may be either continuous or segmental. Shorter taper and smaller numbers of widely spaced gradual tapers enhance the support and transmission of push force, while longer tapers and larger numbers of more segmental tapering enhance the flexibility.



Shorter tapers produce increase in longitudinal support and also increase in tendency to prolapse.



prolapse

Distal tip:

- Flexible, radio-opaque part.
- Consists of spring coil extending from distal untapered part of central core to distal tip weld.
- Integrates tapered core barrel (as well as shaping ribbon in 2-piece wire).
- Spring coil-variable length (1-25cm) with a radio-opaque section located at its terminal end.
- Distal tip weld- short (≤2mm) compact cap forming the true distal end of the wire - to decrease trauma while the wire is traversing vessels.

COATING OF THE GUIDEWIRE

The coating is the outer covering on the core that keeps the overall diameter consistent and influences the wire performance. It affects the lubricity and tracking of the guidewire. It creates the "tactile feel" of the guidewires. The purpose of the coating is:

• To reduce frictions by facilitating the movement of the wire within the coronary anatomy and across the lesion, helping the wire negotiate tortuous anatomy,

• To improve deliverability by facilitating the movement of interventional equipment over the wire.

The type and length of coating may vary. Most often coating is applied to the distal 30 cm of the wire. Two types of coatings are used:

• Hydrophilic coatings attract water and are applied over the entire working length of the wire, including tip coils. When dry, the coating is a thin, nonslippery solid. Upon contact with liquids, such as saline or blood, the coating becomes a slippery gel-like surface that acts to reduce friction with the vessel walls and increase trackability. Hydrophilic coating provides a lubricious, low friction feel inside the vessel and more trackability. They are recommended for operators with some experience as they carry the risk for subintimal movement, dissection and perforation of coronary artery.

• Hydrophobic coatings are silicone based coatings which repel water and are applied on the working length of the wire, with the exception of the distal tip. They require no activation by liquids to create a "wax-like" surface and to achieve the desired effect – to reduce friction and increase trackability of the wire. Silicone coating has higher friction, more stable feel inside the vessel.

GUIDEWIRE MAIN CHARACTERISTICS

Characteristics Definition

Torque control Is an ability to apply rotational force at a proximal end of a guidewire and have that force transmitted efficiently to achieve proper control at the distal end **a**bucking or kinking, to navigate anatomy of vasculature

Steerability : Is an ability of a guidewire tip to be delivered to the desired position in a vessel

Flexibility: Is an ability to bend with direct pressure.
Prolapse tendency: Tendency of the body of a wire not to follow the tip around bends
Radiopacity/visibility: Is an ability to visualise a guidewire or guidewire tip under fluoroscopy.
Tactile feedback: Is tactile sensation on a proximal end of a guidewire that physician has that tells him what the distal end of the guidewire is doing
Crossing: Is an ability of a guidewire to cross lesion with little or no resistance
Support: Is an ability of a guidewire to support a passage of another device or system over it.

CLASSIFICATION OF GUIDEWIRES

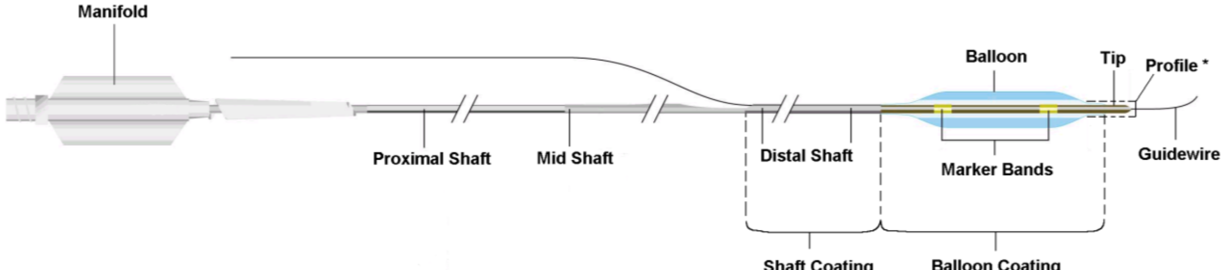
There is no uniform classification of coronary guidewires.
Based on:
1. Tip flexibility (floppy/soft, intermediate, and stiff).
2. Tip coating (hydrophilic, hydrophobic, no coating).
3. Tip style (one-piece core-to-tip, two-piece core with shaping ribbon).
4. Tip tapering (tapered, untapered).
5. Core construction material (stainless steel, Nitinol, high-tensile stainless steel).
6. Device support (light, moderate support, extra support).
7. Target lesion type (workhorse/frontline wires, CTO wires, wires for tortuous lesions, etc.)
8. Specific purpose guidewires, e.g., pressure wire, marker wire (with markers or length indicators)), rotablator wire, wiggle wire, etc.

	FLOPPY/SOFT	INTERMEDIATE	STIFF
Asahi	Light (0.5 g) Soft (0.7 g) Route (0.8 g) Rinato (0.8 g) Fielder (1.0 g) Fielder FC (0.8 g) Fielder XT (0.8 g) Grand Slam (0.7 g)	Intermediate (3.0 g) Miracle 3 (3.0 g) Miracle 4.5 (4.5 g) ULTIMATEbros 3 (3.0 g)	Miracle 6 (6.0 g) Miracle 9 (9.0 g) Conquest (9.0 g) Conquest Pro (9.0 g) Conquest Pro 12 (12 g) Conquest Pro 8-20 (20 g)
Abbott Vascular Hi-Torque series	Balance (0.6 g) BMW (0.6 g) BMW Universal (0.7 g) BHW (0.7 g) Extra S'Port (0.9 g) Floppy II (0.4 g) Whisper LS (0.8 g) Whisper MS (1.0 g) Iron Man (1.0 g)	Cross-IT 100 (1.7 g) Cross-IT 200 (4.7 g) Whisper ES (1.2 g) Pilot 50 (1.5 g) Pilot 150 (2.7 g) Pilot 200 (4.1 g) Progress 40 (4.8 g)	Cross-IT 300 (6.2 g) Cross-IT 400 (8.7 g) Standard (5.4 g) Progress 80 (9.7 g) Progress 120 (13.9 g) Progress 120T (12.5 g) Progress 200T (13.3 g)
Medtronic	Intuition (1.0 g)	ProVia 3 (3 g) ProVia 6 (6 g) Persuader 3 (3 g) Persuader 6 (6 g)	ProVia 9 (9 g) ProVia 12 (12 g) ProVia 15 (15 g) Persuader 9 (9 g)
Boston Scientific	Forte Floppy (0.6 g) ChoICE Floppy (0.8 g) PT Graphix (0.8 g) Luge (0.9 g) Kinetic (1 g)	Platinum Plus (2.1 g) ChoICE PT Floppy (2.1 g) PT2 LS (2.5 g) PT2 MS (2.9 g) PT Graphix Intermediate (1.7 g)	
J&J		Shinobi (7 g) Shinobi Plus (8 g)	
Terumo	Runthrough NS Floppy/Hypercoat (1 g)	Runthrough NS Intermediate (3.6 g)	Crosswire NT (5.5 g)

CORONARY BALLOON

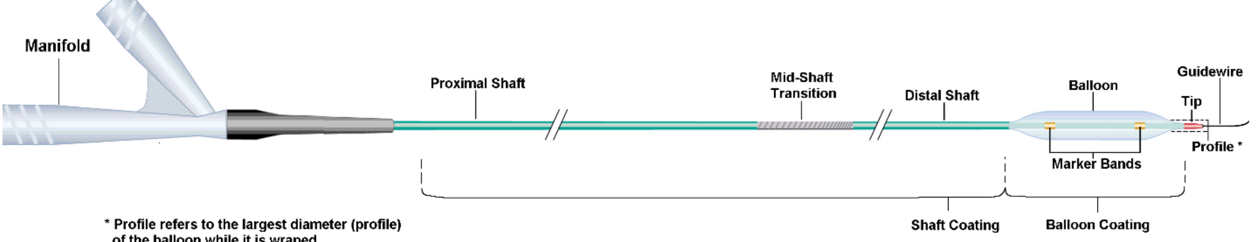
Coronary angioplasty began by using a balloon catheter to compress plaque within a clogged coronary artery, a procedure referred to as a plain old balloon angioplasty (POBA). The main uses of angioplasty balloon catheters are for predilatation of the lesion, post dilatation of the lesion and for stent delivery. In addition to these there are several other uses of the balloon catheters. Most of the Balloon catheters now used in angioplasty are Rapid Exchange (Rx) design because of easiness to exchange guide wire.

Monorail or Rapid Exchange (RX) Balloon Catheters: Rapid-exchange (RX) or "monorail" balloon catheters have become the current standard for percutaneous coronary interventions (PCI). In this balloon catheter too there are two lumens, doesn't extend along the entire course of the balloon. It starts from distal end but finishes well before the proximal end of the balloon catheter. The main advantage of the system is that it allows a rapid exchange of interventional devices over the same standard length guidewire (rather than exchange of different guidewires over the same balloon). Thus the system has led to significant reduction in procedural and fluoroscopy time. Monorail system also seem to add to the safety of percutaneous interventions, due to a variety reasons, namely lower doses of contrast medium, shorter duration of fluoroscopy, and smaller devices diameters.



Another type of balloon we used are Over The wire balloon (OTW balloon)

Over-The-Wire (OTW) Balloon Catheters: These are the classical balloon catheters which have two lumens running through the entire course of balloon catheter. One lumen is for passage of guidewire and the other lumen for connecting with an indeflator for balloon inflation / deflation. This system is more useful when enhanced trackability is required and also in procedures requiring multiple guidewire exchanges and balloon upsizing i.e. chronic total occlusions (CTO).



TERMS USED IN CONTEXT OF BALLOON ANGIOPLASTY

Balloon Diameter: nominal inflated balloon diameter measured at a specified pressure

Balloon Length: typically refers to the working length or the length of the straight body section.

Nominal Pressure: refers to the pressure at which when balloon is inflated it achieves the listed balloon diameter. It is usually 6-8 atm.

Mean Burst pressure (MBP): is the average pressure where 50% of balloons will burst (usually measured at the body temperature).

Rated Burst Pressure (RBP): the pressure below which 99.9% of the balloons will not burst upon single inflation.

Balloon Profile: is the maximum diameter of the balloon when mounted on a catheter in its deflated and wrapped condition or the smallest hole through which the deflated wrapped balloon can pass.

Balloon compliance: it is defined as change in balloon diameter per atmosphere of inflation pressure. It is an index of stretchability of the balloon. While more compliant balloons have the ability to mold according to vessel shape in general they have an inherent disadvantage because they can over-dilate a vessel (optimal balloon: vessel ratio is 0.9-1.1) making it prone to complications like dissection, vessel rupture, abrupt closure and other ischemic complications.

Pushability: It refers to the ability to advance the balloon across the lesion.

Trackability: It refers to the ease of tracking balloon over the guide wire up to the target lesion.

Non Complaint Balloons: They are ultra high-strength, thin walled balloons, generally fabricated from PET and used for coronary and peripheral angioplasty, stent delivery and other dilatation procedures. Rated burst pressure typically ranges from 18-24 atm, these balloons grow in diameter by <10% over nominal pressure upon inflation to high pressures. The RBP can be increased by increasing thickness of balloon, but this also increases stiffness of balloon and balloon profile.

Semi Compliant Balloons: These are standard balloons used for preparation /predilatation of lesion before stent implantation. They have nominal pressure of 6-8
atm. And RBP of around 12-18 atm. Compliant balloons may increase in diameter by greater than 20% upon inflation to higher pressures.

Cutting Balloon: The cutting balloon is equipped with microatherotomes to incise the plaque at three or four points along it circumference. This scoring or cutting of the plaque is purported to produce the controlled and better dilation. The cutting balloon is commonly used for ostial lesions or in-stent restenotic lesions. Cutting balloon features three or four atherotomes (microsurgical blades), which are 3-5 times sharper than conventional surgical blades. The blades which are fixed longitudinally on the outer surface of a non-complaint balloon, expand radially and deliver longitudinal incisions in the plaque relieving its hoop stress. Use of the cutting balloon device is contraindicatd in situation

where the cutting balloon device would be passed through the side cell of previously placed stent as the deflated cutting balloon device could become entangled in the stent. Coronary artery spasm in the absence of a significant stenosis.

The AngioSculpt Scoring balloon: Catheter from Abbott Vascular is essentially a modification of cutting balloon technique. It has a flexible nitinol scoring element with three rectangular spiral struts which work in tandem with a semi complaint balloon to score the target lesion. Balloon inflation focuses uniform radial forces along the edges of the nitinol element, scoring the plaque and resulting in a more precise and predictable outcome. It has a low crossing profile (2.7F), and 0.014" system compatible with 6F guiding catheters, a semi-compliant balloon material.

Drug Eluting Balloon: Drug eluting balloons (DEB) could offer a viable alternative to drug eluting stents (DES) in the treatment of coronary artery disease. These balloons may also offer a valuable new option to treat patients in whom stent implantation is not desirable including small vetssels, in-stent restenosis, bifurcation side-branch, and possible acute ST-elevation MI. currently 4 DEBs are available:SeQuentTM Please, Medrad Interventional's CotavanceTM with Paccocath technology, Eurocor's DIORTM Paclitaxel-coated and Invatec's IN.PACTTM Amphirion paclitaxel-eluting balloon for peripheral use. The Pantera Lux Paclitaxel releasing PTCA balloon catheter is intended for the treatment of CAD with simultaneous release of Paclitaxel to the vessel wall. The balloon surface is homogeneously coated with a delivery matrix incorporating 3µg Paclitaxel per mm^2. Lesions which are untreatable with percutaneous transluminal coronary angioplasty(PTCA). Target lesion in left main coronary artery. Coronary artery spasm in the absence of a siginificant stenosis. Renal insufficiency or allergy to contrast media. Cardiogenic shock of the patient. Patients with an ejection fraction<30%. Allergy , intolerance or hypersensivity to Paclitaxel or structurally related compounds and/or to the delivery matrix n-Butyryltri-n-hexyl Citrate (BTHC). Inflate the balloon in standard PTCA techniques, depending on the patient situation and vessel morphology the inflation should be kept for a period of 30s.

Y CONNECTOR (ADJUSTABLE HEMOSTASIS DEVICE)

The Y connector is an accessory device that minimizes back-bleeding while the balloon catheter is inserted into the guiding catheter. This device allows the injection of contrast media and pressure monitoring through the guiding catheter, regardless of balloon catheter position. It comes with a rotating valve to prevent back bleeding while advancing the balloon/stent catheter through guide catheter.

INFLATION DEVICE

A disposable syringe device is used to inflate the balloon on the balloon catheter with precise measurement of the inflation pressure in atmospheres (atm). Balloons are generally inflated at pressures of 4 to 20 atm. Although stents may be inflated at 10 to 18 atm. It features braided high pressure tubing with a rotating luer fitting, a luminescent

analog pressure gauge, and a handle. Pressure balloon inflation are generated by squeezing on the handle or by turning angle in clockwise.

TORQUE (TOOL) DEVICE

A small cylindrical pin vise clamp slides over the proximal end of the angioplasty guidewire, permitting the operator to perform fine manipulations of the guidewire by turning the torque tool (i.e., pin vise on the guidewire).

GUIDEWIRE INTRODUCER

A very thin, needle like tube with a tapered conical opening helps the guidewire to be inserted into balloon catheters and through Y adapters.

CORONARY STENTS

Stents are small expandable tubes used to treat narrowed or weakened arteries in the body. In patients with coronary artery disease (CAD), stents are used to open narrowed arteries and help reduce symptoms such as chest pain (angina) or to help treat a heart attack.

There are currently five types of stents available:

- Dual Therapy Stent (DTS)
- Bioresorbable Vascular Scaffold (BVS)
- Bio-engineered Stent
- Drug Eluting Stent (DES)
- Bare Metal Stent (BMS)

Dual Therapy Stent (DTS)

Dual Therapy Stent (DTS) is the latest type of coronary stent. It is a first-ofits- kind stent therapy designed to not only reduces the likelihood of the re-narrowing of the artery or of having to undergo a repeat procedure, but also help the healing process of the artery. It combines the benefit of DES and bio-engineered stents and is the only stent to contain a drug with active healing technology.

The DTS has coating both inside and outside, which reduces the likelihood of blood clots, inflammation and helps the healing process of the artery. The stent surface facing the artery wall contains a drug that is released to help stop the artery blocking again without the worry of swelling or an inflammatory response. The drug is delivered from a bioresorbable polymer that will degrade over time. The side of the stent which faces blood flow is coated with antibodies, which promote natural healing and helps the healthy artery function properly.

The Bio-Vascular Scaffold (BVS)

The Bio-Vascular Scaffold (BVS) is a drug eluting stent on a dissolvable type of scaffold platform which can be absorbed by the body over time. Like some of the currently available Drug Eluting Stents (DES), BVS is coated with a drug released from a polymer that disappears over time to reduce the likelihood of the artery re-narrowing (restenosis). The

scaffold itself is absorbed overtime. Unlike with the DTS, there is no active element to promote artery healing.

Bio-engineered Stent

Bio-engineered Stent is also known as antibody-coated stent. This type of stent differs from DES because it does not contain a polymer and does not use a drug. As a result, it helps to speed up the cell lining of the artery (endothelialization), promoting natural healing.

The antibody on the stent's surface attracts circulating Endothelial Progenitor Cells (EPCs) which come from human bone marrow and help speed up the formation of healthy endothelium. This provides rapid coverage over the stent's surface helping to reduce the risk of early and late thrombosis blood clots.

Drug eluting stent(DES)

DES is a peripheral or coronary stent (a scaffold) placed in to narrowed, diseased peripheral or coronary arteries that slowly releases a drug to block cell proliferation. This Prevent fibrosis that, together with thrombi, could otherwise block the stented artery, a hprocess called restenosis. The stent is usually placed within the peripheral or coronary artery by an interventional cardiologist or interventional radiologist a during an oehond. angioplasty procedure.

Drug eluting stents generally consist of three parts- the stent platform, a polymer coating that binds the drug to the stent and releases drug. The stent platform itself is an expandable framework, generally with an elaborate mesh-like design to allow expansion, flexibility and in some cases the ability to make /enlarge side opening for side vessels. The DES were stainless steel alloy composed of iron,nickel and chromium amd were based on existing bare metal stents. These stents were hard to visualize with medical imaging, posed a risk of causing allergic responses, and were difficult to deliver and subsequent new alloys were brought to bear, namely Co-Cr and platinum chrome, with improved performance. Subsequent ,bioresorbable stents have been developed in which the stent itself dissolve overtime. As of 2009, material that had been explored included Mg,polylactic acid,polycarbonate polymers, and salicylic acid polymers. Resorbable stents have held the promise of providing an acute treatment that would eventually allow the vessel to function normally, without leaving a permanent device behind.

One to three or more layers of polymer can be used in the coating eg,.a base layer for adhesion, a main layer that holds and elutes the drug into the arterial wall by contact transfer, and sometimes a top coat to slow down the release of the drug and extend , extend its effect. The first few drug-eluting stents to be licensed used durable coatings. the first immunological reactions at times and some possibly led to thrombosis, which has driven experimentation and development of new coating approaches.

The drug is manly to inhibit neointimal growth(due to proliferation of smooth muscle cells) which would cause restenosis. Much of the neointimal hyperplasia seems to be caused by

inflammation. Hence, immunosuppressive and antiproliferative drugs are used. Sirolimus, Paclitaxel and everolimus were previously used for other medical applications and have been included in licenced DES.

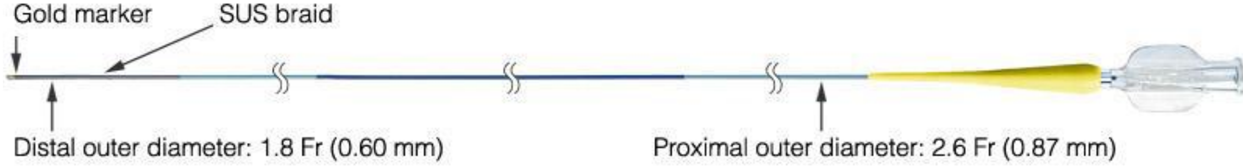
Bare metal stents

Bare metal stents are usually stainless steel and have no special coating. They act as scaffolding to prop open blood vessels after they are widened with angioplasty. As the artery heals, tissue grows around the stent, holding it in place. However, sometimes an overgrowth of scar tissue in the arterial lining increases the risk of re-blockage.

MICROCATHETERS

A microcatheter is a single-lumen device that can be loaded on the GW in order to track it to the target lesion. The TRACK FORCE (**TRACKABILITY**) needed to reach the lesion should be as low as possible. The microcatheter is used to help the physician to cross the lesion, previous to the balloon dilatation/stenting, giving mechanical support to the guidewire and enhancing its ability to transmit PUSH FORCE **(PUSHABILITY)** to the occlusion.

Fine cross (Terumo): Tapered inner and outer lumen provide an optimal balance between guide wire support and crossability. Fully stainless steel braided shaft provides strong guide wire support. PTFE coated inner layer facilitates manipulation of the guide wire 2.6 Fr (0.87 mm) proximal diameter enables buddy wire technique in 6 Fr (I.D. ≥ 0.070'' - 1.78 mm) guiding catheter. Large inner diameter ensures optimal guide wire handling



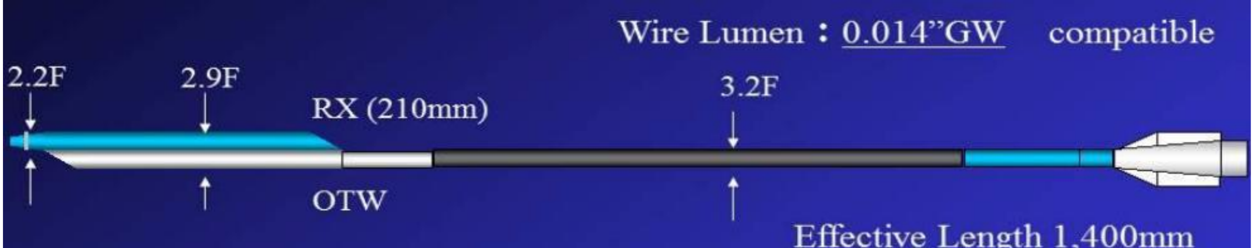
Corsair (Asahi): For increased success with Antegrade and Retrograde approaches to CTO's:

- Kink resistant tapered tip (.016") eases access to complex channels
- Ease in crossing and dilating micro-channels or lesions
- Improves wire support and wire manipulation Hydrophilic coating applied to distal 60cm section

CRUSADE

The Crusade Catheter is a dual lumen device studied for the treatment of stenosis close to the bifurcations, when the guidewire doesn't provide the right support to engage the side branch.

The Crusade catheter allows the operator to deliver a second guidewire through the OTW lumen while leaving the guidewire in the monorail lumen in place. The maximum supported diameter of the guidewires in the Crusade Catheter is 0.014".



GUIDELINER CATHETER

- GuideLiner catheter is a coaxial guiding catheter extension delivered through standard guiding catheter on a monorail.
- Comprises a flexible 20 cm straight extension connected to a stainless steel push tube.
- Permits very deep intubation of the target vessel, thus providing backup support to facilitate stent delivery across heavily calcified lesions in tortuous vessels.

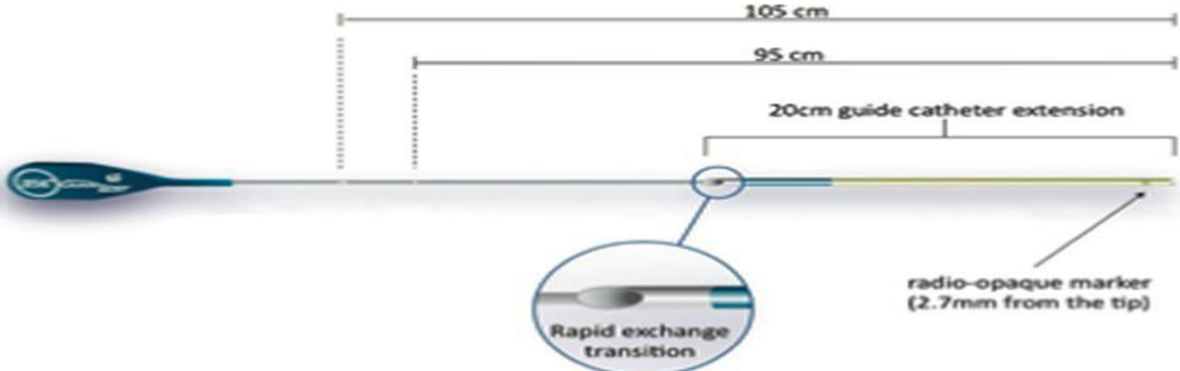


Figure 1. The GuideLiner catheter. This consists of a flexible 20 cm straight guide extension connected to a stainless-steel push tube.

CLOSURE DEVICE

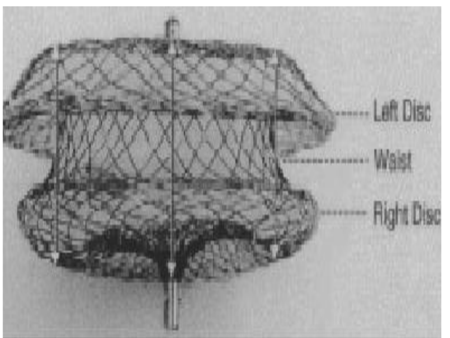
AMPLATZER DEVICES

Septal occluder: This device consists of two self-expandable round discs made of 0.004 to 0.005 inch Nitinol wire mesh that are linked together by a short (4 mm) connecting waist, corresponding to the thickness of the atrial septum. The left atrial disc extends 7 mm radially around the connecting waist and the right disc 5 mm. The left disc is slightly larger than the right because of the higher left atrial pressure. Both discs are angled slightly toward each other to ensure firm contact with the atrial septum. The prosthesis is filled

with Dacron fabric to facilitate thrombosis. Prostheses are currently available in sizes ranging from 4 to 32 mm at increments of 1 mm.

Duct occluder: The **Amplatzer Duct Occluder** is a self-expandable, mushroom-shaped device made from a 0.004 inch thick Nitinol wire mesh (a thin retention disc, 4 mm larger in size than the diameter of the device ensures secure positioning in the mouth of PDA). The PDA is closed by the induction of thrombosis, which is accomplished by polyester fibres sewn securely into the device. Platinum marker bands are applied to the wire ends and laser welded. The shape is then formed by heat treatment. All devices are cone-shaped and 7 mm in length, with a recessed screw, sizes are given from the larger to the smaller diameter.

Duct occlude II: the Amplatzer duct occluder II is self expanding Nitinol mash device for the occlusion of the PDA. The device configuration is a central waist with 2 retension discs. The central waist is designed to fill the ductus and the two retension discs are designed to be deployed in the pulmonary artery and aortic side of the ductus.



g. 1 : Amplatzer septal occluder

cribriform 1

Cribriform device: Amplatzer Multi-Fenestrated Septal Occluder - “Cribriform” were specifically designed to seal ASDs and multi fenestrated ASDs respectively. Amplatzer Multi-Fenestrated Septal Occluder -“Cribriform” were specifically designed to seal ASDs and multi fenestrated ASDs respectively. The left atrial disc is smaller than the right atrial disc.

Muscular VSD occluder: the two disks are linked together by a short cylindirical waist corresponding to the size of the ventricular septal defect.

Cocoon ,occlutech, pushmed ,Heart R are other such devices used.

ADO1 Sizes

Device diameter in DTA	Device diameter in PA	Length	Minimum recommended sheath(amplatzer delivery system)
5mm	4mm	5mm	6 Fr, 180^0 curve
6mm	4mm	7mm	6Fr, 180^0 curve
8mm	6mm	7mm	7Fr,180^0 curve
10mm	8mm	8mm	7Fr,180^0 curve
12mm	10mm	8mm	7Fr,180^0 curve
14mm	12mm	8mm	8Fr,180^0 curve
16mm	14mm	8mm	8Fr,180^0 curve

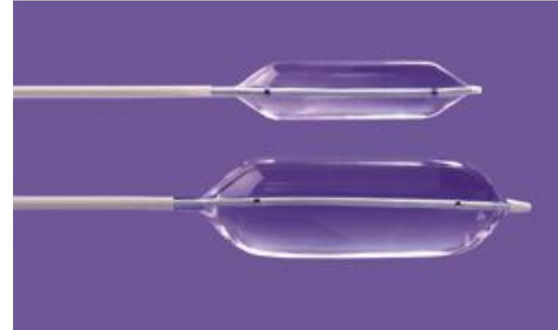
ADO2 DEVICES

Recorder number	Waist diameter(mm)	Device diameter(mm)	disc diameter(mm)
3-4	3	4	9
3-6	3	6	9
4-4	4	4	10
4-6	4	6	10
5-4	5	4	11
5-6	5	6	11
6-4	6	4	12
6-6	6	6	12

VALVULOPLASTY BALLOON

Catheter Characteristics

The Z-MED™ high pressure dilatation catheter is a coaxially constructed catheter with a distally mounted non-compliant high pressure balloon. The catheter exhibits a comparable profile to other marketed catheters by also combines this profile with a high pressure balloon suitable for resistant stenosis. The Z-MED™ balloon catheter is available in over 50 configurations ranging in diameter from 2.0 to 40.0 mm.



Radiopaque Marker

Platinum marker bands facilitate reliable positioning of the balloon.

Balloon

The Z-MED™ PTV balloon has a low deflated profile that maintains tip flexibility. The exceptionally low profile balloon requires the smallest introducer possible. Nominal dimensions are maintained over the entire length of the balloon.

Maximum Trackability

The distal shaft through the balloon is highly flexible for exceptional maneuverability. This, combined with the pushability of the coaxial shaft, provides outstanding tracking performance.

Sizes

The balloon diameters are starting 4 up to 30 mm.

first described by Gianturcoand colleagues 5 more than 20 years ago. The most commonly used coil embolization materials available include the Gianturco stainless steel coil (Occluding Spring Emboli; Cook, Bloomington, Indiana) and the platinum microcoil (TargetTherapeutics, Santa Monica, California). The Gianturco coil is constructed of stainless steel wire of varying helical diameters and lengths to which Dacron fibers have been attached to increase thrombogenicity. There are at least 15 different loop sizes and lengths of the wire Gianturcoand coils from the smallest diameter of 1mm to th largest 15 mm.

BIOPTOME

There are two basic types of bioptomes: (a) stiff devices that are maneuvered independently through the vasculature and (b) more flexible devices that can be positioned only with the aid of a long sheath or introducing catheter. A bioptome is a small pincer-shaped cutting/grasping instrument used in medicine for taking biopsy specimens.

It can either be built into or threaded through a vascular catheter or endotracheal tube that delivers the bioptome directly to the desired site of tissue sample.

INOUE TYPE BALLOONS

The Inoue balloon catheter has a unique design that allows inflation of the balloon to the facilitate crossing the mitral valve. A calibrated syringe produces a step-wise incremental valve dilation. Selection of balloon size is based on patient height.

ACCURA BALLOON

The accura double lumen PTMC balloon dilation catheter has an 11 F, 80 cm shaft and three stages of expandable balloon.

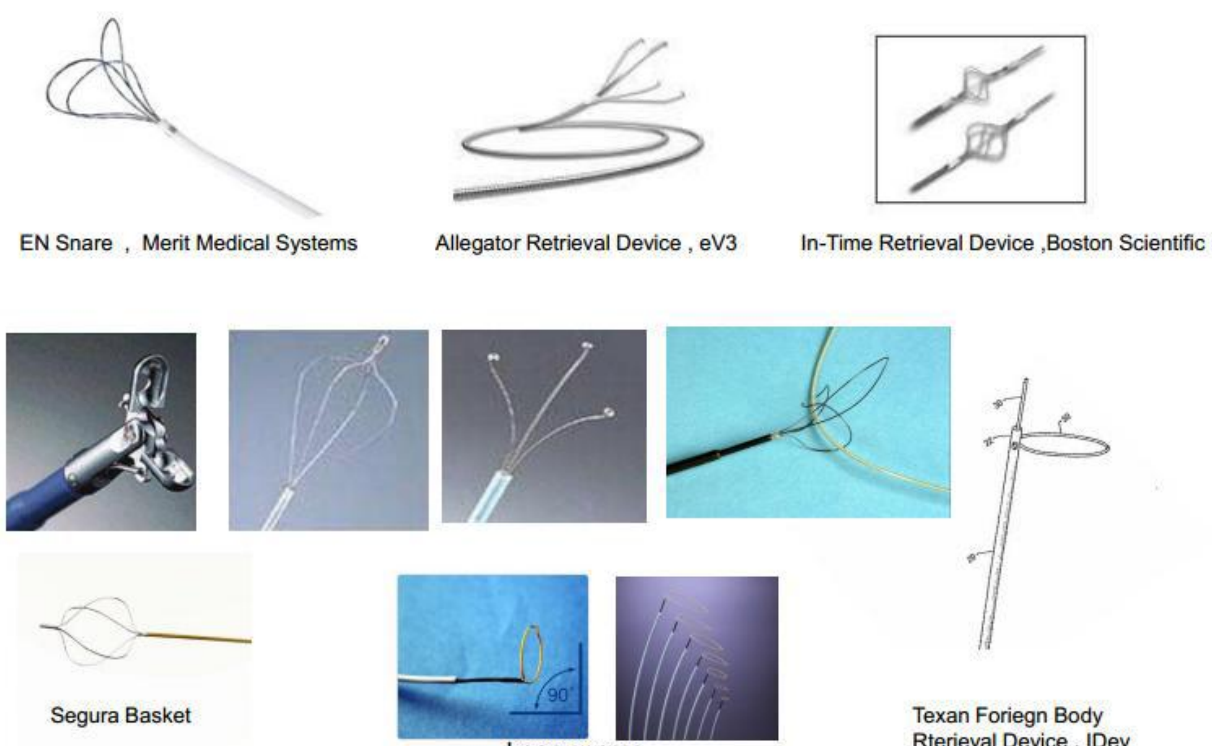
INFERIOR VENA CAVA FILTERS

IVC filters are typically collapsible cone-shaped arrays of six struts (wires) of steel, of titanium, or nickel-titanium (nitinol), with hooks (barbs) on the wire ends to secure the filter to the vena cava wall. Other filter shapes are also used—for example, the bird’s nest IVC filter, which is a random array of wires extending in various directions; the shape is reminiscent of a bird’s nest. There are basically two types of IVC filters, permanent and optional, commonly referred to as retrievable. Permanent filters are designed to remain in the patient without the ability to be removed. Permanent filter design should permit significant fixation to the vena cava wall to prevent migration over the patient’s life. Optional (retrievable) filters are designed to remain permanently in the patient or to be removed when it is no longer warranted, such as when the risk of PE has subsided or when the patient no longer has a contraindication to anticoagulation therapy. Retrievable filters should also achieve fixation to the vena cava wall, but their structure must have the ability to be altered (e.g., collapsible) at the time of removal with catheter-based retrieval devices to facilitate safe removal.

THROMBUS ASPIRATION SYSTEM

Aspiration embolectomy, catheter embolectomy is also used for aspiration embolectomy ,where the thrombus is removed by suction rather than pushing with a balloon. It is a I in rapid and effective way of removing thrombi in thrombolic occlusions of the limb arteries below the inguinal ligament. Export advance aspiration catheter by Medtronik has full wall variable braiding technology improves deliverability by increasing kink resistance , resulting in optimal thrombus aspiration capability. Guide catheter compatibility:6F (min ID 0.070”).

FORIGN BODY RETREIVAL CATHETERS



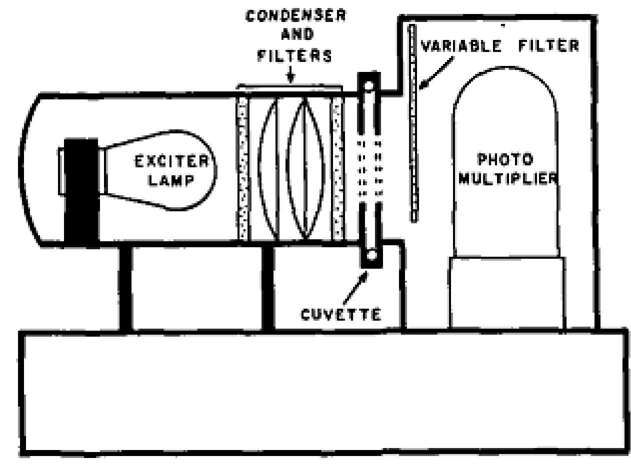
PULSE OXIMTERY

Oxygen saturation is the percentage of Hb that carries O_2. Working principle of pulse oximeter is Beer-Lambert's Law, which states that amount of light absorbed is proportional to concentration of light absorbing substance and length of the path that light travels through the absorbing substance. Here Hemoglobin is the light absorbing substance , when the concentration of oxyhemoglobin increases saturation will also increases. But absorption varies with wavelength of light.

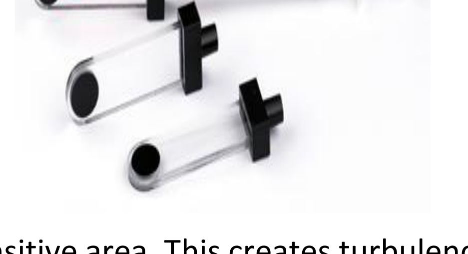
A typical pulse oximeter consist of an electronic processor and a pair of small LEDs facing a photo diode. The finger (absorbing substance) is placed between these source and detector. One is red light (660nm) and other is infrared light(960nm). Absorption of these lights varies significantly between oxy hemoglobin and deoxy hemoglobin.

Oxyhemoglobin absorbs more IR light and allow more red light to pass through where as deoxyhemoglobin viceversa. This ratio of absorption is taken to a processor and displaying on the monitor.

CUVETTE OXIMETRY



'IG. 1. Diagrammatic illustration of densitometer.



The fundamental principles upon which oximetry is based are the facts as demonstrated by (1) oxyhsemoglobin transmits visible red light (620-770 m^) to a much greater degree than does reduced hemoglobin, and (2) that Beer's law of optical absorption may be applied to the hemoglobin of whole blood in various saturations with oxygen. The instrument employed consists basically of a light source with transmission of light through a cuvette containing blood to a photosensitive element. The light source is of a wavelength of 630 nm and at this wavelength the transmission of light through blood is directly related to its oxygen saturation. The photosensitive element consists of a multiplier tube capable of a gain of one million and with an output sufficiently high to permit the use of a fast-response galvanometer (125 c.p.s.). The cuvette is constructed so that the entering blood changes its course in a 90° turn just prior to reaching the photosensitive area. This creates turbulence in the flow and prevents streaming of the blood. The light source, cuvette and phototube form a small unit remote from the power supply, permitting the use of the densitometer close to the field of operation.

ACTIVATED CLOTTING TIME

This test is used to monitor effectiveness of high dose heparin therapy.

- The detection mechanism comprises a magnet located inside the test tube and two solid state magnetic detection positioned within the test well. One is at 0^0 (precision aligned magnet) and other at 90^0.
- When the test tube is inserted in to the test well the detector at 0^0 senses the presence of magnet in the test tube as the tube rotates.
- As clot forms fibrine strand causes the magnet to rotate with the tube such that its presence is sensed by the detector at 90^0.
- Once it is detected the clot formation the instruments activates an audible indicator and displays clotting time.

- Precautions : sample to be taken in non-heparinized tube. Use only dry syringes. Tube has to be prewarmed. It has to be done immediately because the activation starts at the moment blood gets in contact with activator.
- Normal ACT is 107s to 150s.
- For sheath removal < 170s.

CLEANING AND STERILIZATION

Most catheters used today are intended for one-time use only. Catheters suitable for reuse should be cleaned in the following way:

- Immediately after use, flush the catheter manually with 20 to 40 ml of normal saline and rinse the outer surface with normal saline or sterile water.
- Connect the catheter to a pressure flush system and flush with water for 30 to45 minutes.
- Cleanse the catheter by soaking in solution of hydrogen peroxide or such disinfectant solution for 30 minutes .
- Flush the catheter with 1000 ml of H_2O_2 solution with either a pressure flush system or gravity drip setup.
- Rinse the catheter with 100 ml of distilled water using a pressure flush system or a gravity drip setup.
- Dry the catheter.

Three types of sterilization methods using such as

1. Hot sterilization
2. Chemical sterilization
3. Gaseous sterilization

Hot sterilization

Boiling and Steeming(autoclave)

Steem is most effective method at100^0. It is enough to kill vegetative bacteria only much higher temperature is needed to kill spores. In industrially high pressurized closed vaccum chambers are used for steem sterilization at130^0 C. it must ensure that packets emoved from autoclave are dry.

Chemical sterilization

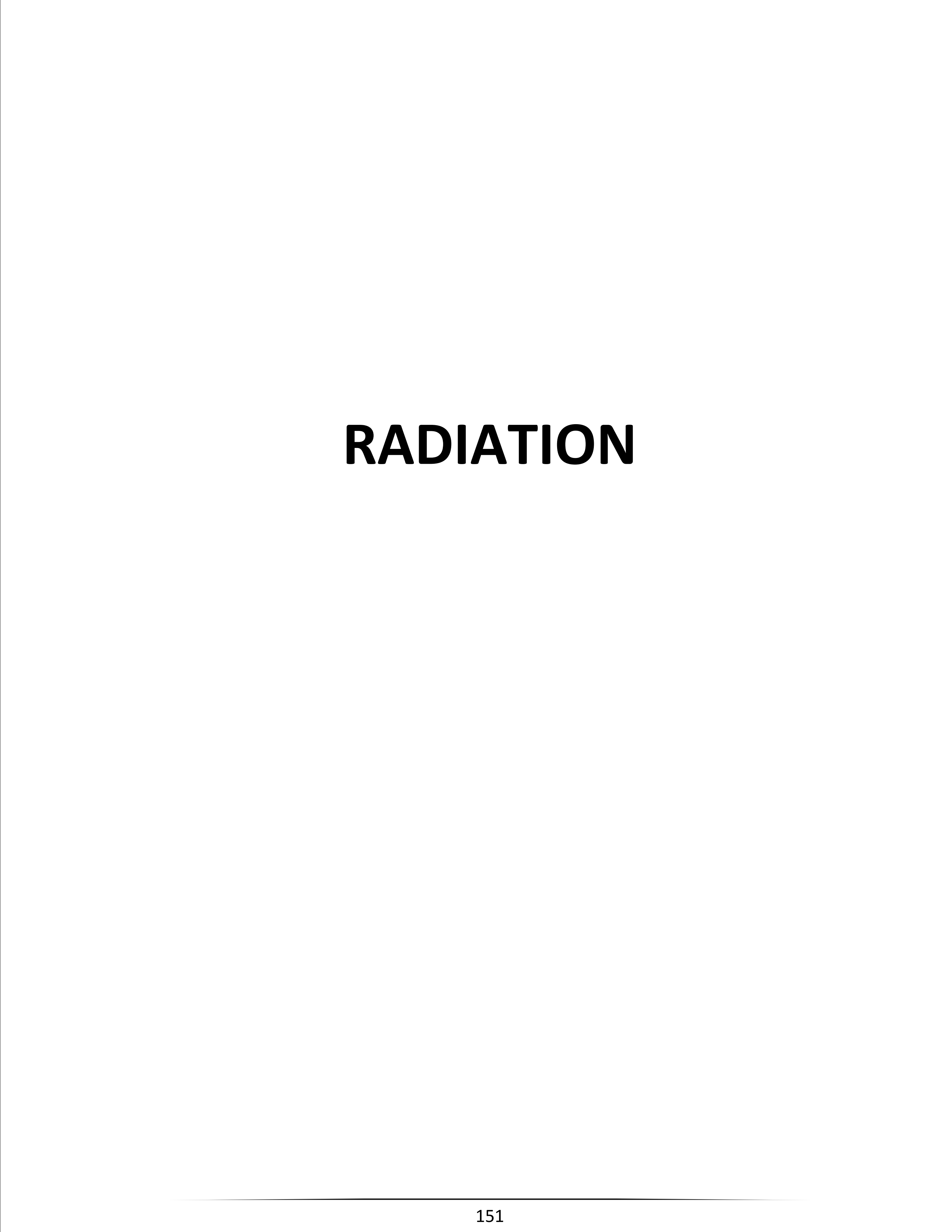
Using cidex(Glutaraldehyde) has a wide range of antimicrobial acivity. It kills microbacteria in 20 to 60 minutes and spores in 3 to 10 hrs. once activated alkaline solution of cidex remains active for 14 to 28 days. The particle to be sterilized to be fully immersed in the solution and lumens are flushed with cidex .

Gaseous sterilization

a) Ethylene trioxide(ETO)
   Using low temperature combination with ETO gas. This sterilization cycle includes removal of air from chamber followed by humudification. Air must be evacuated below atm pressure to ensure the deep penetration of the miiosture. The ETO gas must be vapourized and heated before it is admitted to the evacuated chamber. An effective combination of sterilization should be within the following range such as
   ETO-700 to 1000mg/L
   Temperature-45 to 65^0 C
   Humidity- 70%
   Sterilizing time -4 hrs.
b) Formaldehyde sterilization
c) Plasma sterilization-using hydrogen peroxide at plasma state.

DISINFECTION OF LAB (FUMIGATION)

Disinfection of lab is done by formaldehyde (formalin). Formalin is an aqueous solution containing 37 to 40% formaldehyde, this may be diluted with water or alcohol (1:1). This solution is vaporous in the room. The room has to be airtight for effective disinfection. The vapors are allowed to be in the cabin for 12 hrs.



RADIATION

The cath lab professionals obligated to minimize and reduce risks to other personnel, themselves and their patints. Radiation is invisible and can easily ignored , but it shuld be respected.

X-ray is a form of electromagnetic radiation with its very short wavelength and very high frequency , each x-ray photon can pass through solid matter and captured to film or monitor.

- **X-ray dose and measurement:**Important radiation safety programs must manage patients and staff safety by reducing exposure to x-ray to a level that is as low as reasonably achievable(ALARA). X-ray dose measurement can be seen by the following:
    - **Fluoroscopic time(FT):** it is the time during a procedure that fluoroscopy is used. Since FT doesn't include cine acquisition imaging, it is not a useful description of patient radiation dose.
    - **Total Air Kerma(AK)** at the interventional reference point is the procedure cumulative x-ray energy delivered to air at the interventional reference point. It is used to monitor patient dose as it is associated with deterministic skin effects, through it is not the true peak skin dose.
    - **Air Kerma Product(PKA, Gy/cm^2)** is the sum of the product of instantaneous air kerma and x-ray field area.
    - **Peak Skin Dose(PSD,Gy)** is the maximum dose received by any local area of patient skin. When PSD increases , the probability and severity of deterministic skin effects increases. PSD is estimated by air kerma and x-ray geometry details.
- Scattered radiation is the produced when the x-ray beam interacts with the patient and is redirected rather than absorbed completely. If scattered radiation reaches the image receptor , it contributes to noise and reaches the image receptor ,it contributes to noise and reduces the image contrast created as the primary x-ray beam interacts with the anatomic structures. Scattered radiation is also the principle source of exposure for the patients body parts that lie outside the field of the primary x-ray beam and also for the staff. The amount of scatter increases with increases in the intensity of the x-ray beam and the size of the x-ray field.

Radiation production and control

Generators : the cine fluoroscopic x-ray generators controls and delivers electrical power to the x-ray tube. It heats the x-ray tube's filament to produce a beam of electrons that are accelerated towards the target anode. The flow of electrons towards the anode is not continuous, but is separated to 15 to 60 pulses per second(pulsed fluoroscopy).

X-ray tubes: the x-ray tube is a device that converts a portion of electrical energy delivered by the generator into x-rays. The x-ray tube consists of an evacuated glass or metal housing that contains a tungsten filament, and an anode disc which rotates at more than

10000rpm. Electrons are emitted from the filament and accelerate toward the anode under the influence of electric field supplied by the generator.

Fluoroscopy : provides a real time x-ray image for guiding manipulations. This allows significantly lower x-ray input doses than those needed for acquisition. Many systems offer variable fluoroscopic frame rates. Decreasing the frame rate saves dose at the expense of visual smoothness of the transition between frames.

Acquisition /cine : generates images of sufficient quality for single frame viewing. Higher x-ray input rates are needed to reduce image noise and optimize visualization with a per frame dose approximately 15 times greater than fluoroscopy.

X-RAY DETECTION AND RECORDING

The x-ray image formed by the interaction of the x-ray beam and the patient must be detected and transformed into a visible format. The image intensifier captures modulated x-ray beam emerging from the patient and converts it into a visible light to be viewed on an output screen. Image intensifier are equipped with various sized image fields that alter the image resolution. The smaller the input screen diameter, the smaller the field size and the sharper the resolution. To obtain optimal cine angiograms, clinicians should be aware of the following principles:

- The image intensifier should be as close to the patient's chest as possible,
- The position optimizes the image detail and decreases scatter radiation.
- The patient should be instructed to take a deep breath and hold it before the start of cine angiography. This pulls the diaphragm downward and out of the field of view.
- All ECG electrodes, lead wires, metal snaps on the pateint's gown and jewellery should be out of the field of view before the start of procedure. Keep wires and IV lines from hanging down under the table to prevent interference with the x-ray tube that may result in wires or lines from being pulledout.
- Use of collimators eliminates unwanted lung field brightness and helps optimize exposure settings.
- **Cineangiographic Frame Rates**

  Minimizing radiation exoposure to patient and the operator os controlled one of the primary safety concerns in the catheterization laboratory. Patients undergoing +/- ad-hoc PCI or planned PCI , will be randomized to conventional settings(15 fps and 15 pps) or low frame rate settings(7.5 fps and 15 pps) or low cine settings (7.5 fps and 15pps). The patient radiation dose,patient radiation dose area product (DAP), and fluoroscopy time will be measured. This will help to reduce radiation exposure.

Radiation safety

Four principles in radiation safety should be self evident.

- The less exposure, the less chance of absorbed energy interaction.
- No known level of ionizing radiation is a permissible dose or absolutely safe.
- Radiation exposure is cumulative. No washout phenomenon occurs.
- Clinicians are obliged to minimize and reduce risks to other personnel and themselves.

Methods to Limit Radiation.

- Wear lead aprons: 0.5mm or more thickness provides 80% protection.
- Limit the fluoroscopic or cine angiographic time.
- Use collimators.
- Reduce distance between the x-ray source and the patient.
- Maximize the distance between the x-ray source and the operator and assistants.
- Limit the milli amperes /Kv as much as possible for an adequate image.
- Use slower panning and provides good initial angiographic setup. Angled views almost double the radiation.
- Keep the image inteifier as low as possible.
- Use extra shielding(thyroid , glasses,table shields).

Radiation hazards in cath lab

1. **Deterministic effects**

A deterministic effect describes ionizing radiation induced damage where a Dose threshold exists, and for which the severity of damage increases with increasing Dose above that threshold. Allowable whole body radiation per year is **5Rem(Roentgen equivalent man).**

1Rem(CGS system) =0.01 Sv (SI)

Deterministic effects includes:

a) Erythema
b) Desquamation
c) Cataracts
d) Decreased WBC count
e) Fibrosis
f) Hair lose
g) Thyroid disease
h) Neuro- degenerative disease
i) Organ atrophy(decrease in tissue mass of an organ due to either a decrease in size of individual cells(cellular atrophy) a decrease in number of cells or both).

j) Sterility

2. **Stochastic effect**

   Effects that can occur by chance and which may occur without a threshold level of dose, whose probability is proportional to the dose and whose severity is independent of dose. The main stochastic effect is cancer.

3. **Non stochastic effects**

   The severity of which varies with the dose and for which threshold is believed to exist. Generally result from the receipt of a relatively high dose over a short time period.

CARDIAC CATHETERZATION DIAGNOSTIC STUDIES AND PROCEDURES

INTRODUCTION

Cardiac catheterization is the insertion and passage of catheters through the arteries and veins to the heart to obtain x-ray images and measure pressure in the heart. The x-ray images or angiography are performed to view the coronary arteries and diagonoisis coronary artery disease as well as identify diseases of the aorta and pulmonary vessels. Besides providing diagnostic information , the cardiac catherization performs catheter basal intervention to treat cardiovascular disease.

History

The first cardiac catheterization was performed by Claude Bernard in 1844 by passing catheters in to both the right and left ventricles of a horse using the jugular vein and carotid artery. This procedure began an era of investigation of cardiovascular physiology in animals that resulted in important techniques used today. These techniques include pressure manometry and the Fick cardiac method.

The first cardiac catheterization on a human being was performed by Werner Forssman. At the age of 25, he passed a catheter through the one of his left antecubital veins and guided it by fluoroscopy until the catheter entered his atrium. He then walked to the radiology department , where the catheter position was documented by chest x-ray.

Further developments came rapidly. Retrograde Left Heart Catheterization was first reported by Zimmeraman and Limon Lason in 1950. Seldinger developed a percutaneous technique for both left and right heart catheterization in 1953. Selective coronary arteriography was developed by Sones in 1959. This technique was modified for percutaneous approach by Judkins in 1967. In 1970, a balloon -tipped flow guided catheter technique was introduced the technique of coronary angioplasty in 1977.

Indications for cardiac catheterization

- Acute MI
- Assessment of coronary vasculature after abnormal non invasive studies.
- Assessment of coronary artery disease in patients with confusing clinical pressentations or unexplained chest pain.
- Assessment of vascular disorders and cardiomyopathy to determine the need for surgical correction.
- Medical clearance for surgical procedures.
- Evaluation of aortic dissection.

Contraindications

The only true contraindication for cardiac catheterization is patient refusal. List of relative contraindication is that require special attention before proceeding with cardiac catheterization.

- Severe uncontrolled hypertension
- Ventricular arrhythmias.
- Acute stoke or recent cerebrovascular accident(<1 month).

- Severe anemia.
- Active gastrointestinal bleeding.
- Allergy to radiographic contrast.
- Acute renal failure.
- Uncompensated congestive heart failure(patient cannot lie flat).
- Febrile illness or untreated infection.
- Electrolyte abnormalities.
- Anticoagulation and clotting disorders.
- Pregnancy
- Uncooperative patient.

Complications.

- Death
- Heart attack.
- Lethal arrhythmias
- Cardiovascular accident or stroke.
- Bleeding
- Perforation of a vessel.
- Dissection of a blood vessel.
- Infection.
- Allergic reaction to the contrast or dye.
- Air embolism.
- Congestive heart failure.
- Vasovagal reaction.

Patient preparation

Preparing the patient is an important part of the cardiac catherization. Key information can be obtained and the procedure itself can be optimized with proper patient preparation.

- **Welcome patient:** it is natural for the patient to be nervous. Show confidence and understanding with your greeting. The first impression can determine the course of the patient visit.
- **Patient identification:** identify the right patient for the right procedure.
- **Obtain vital signs:** baseline vital signs: blood pressure, heart rate and rhythm, respiratory rate and pulse oximetry are important to know before the procedure. The baseline values can helps to determine if vital signs during the procedure are trending toward a complication.
- **Obatain 12 lead ECG:** a baseline ECG will be valuable information for the procedure in recognizing any changes that can take place.
- **Review history:** ask the patient about their previous medical history. Complete a head to toe medical history and record of any surgeries. Ask if the patient has had a prior cardiac catheterization. Ask if the patient has any cardiac risk factors such as: Diabetes, Family history, Elevated cholesterol, Hypertension and tobacco use.
- **Review medical record:** complete a copy of the patients home medication record that includes: medication, dose, route, freuquency and when last taken. Important

medications to be watchful for are Coumadin(Warfarin), Glucophage(Metformin) and Viagra. These medications can lead to complications associated with the cardiac catheterization.

- **Verify allergies:** it is important to know that the patient is allergic to so that these medications can be avoided. Ask the patient if they are allergic to IV dye, Iodine or shellfish products. If so then they will need to be premedicated prior to their procedure.
- **Review the current lab results:** just like baseline vital signs, recent lab results are important to know prior to the procedure. Abnormal will need to be addressed prior to the procedure. Severe abnormals may postpone the procedure to another day. These are just some of the measures to prevent the complications associated with cardiac catheterization.
- **Check peripheral pulses:** since the procedure involves a puncture of the arterial system baseline peripheral pulses will be important to compare with post procedure assessment.
- **Check pregnancy status:** female patients of child bearing age should be asked about pregnancy status.
- **Verify the IV line is patent:** A patent IV line should be there so that IV fluids and medications can be administered.
- **Verify NPO status:** The patient should be NPO for a minimum 6hrs prior to their procedure. This reduces that risk of aspiration of stomach contents into the airway during the procedure.
- Verify that necessary paperwork is completed.
- **Review procedure with patient :** Administer premedications as ordered . patients with IV contrast allergies will need to be premedicated prior to cardiac catheterization. This includes Benadryl 50mg IV, Pepcid 20mg IV, and solucorlet 100mg IV. Patients with elevated creatinine and diminished renal function may require: Mucomyst 1200mg PO and IV hydration with 3amps of sodium bicarbonate in Litre of NS.

Room preparation

The patient room and table should be cleaned according to hospital policy prior to placing the patient onto the examination table. Gather the necessary supplies for the procedure. This should include as exam pack, sterile gloves, sterile gowns, sheath and catheters. Position the patient centered at examination table. Be sure to tuck the armboards inclose enough to the patient to avoid interference with the C-arm as it rotates. Have the patient lay their arms down in the armboards and explain to them to keep their arms to their side throughout the procedure to prevent contamination to the sterile field and to not interference with the procedure.

Be sure to keep non-radiolucent items away from the area to be examined. Non radiolucent ECG cables should be positioned along the shoulders and then down along the left side. It is good practice to place the

noninvasive BP cuff on the opposite side of the IV, unless contraindicated. SPO_2 monitoring is essential for conscious sedation.

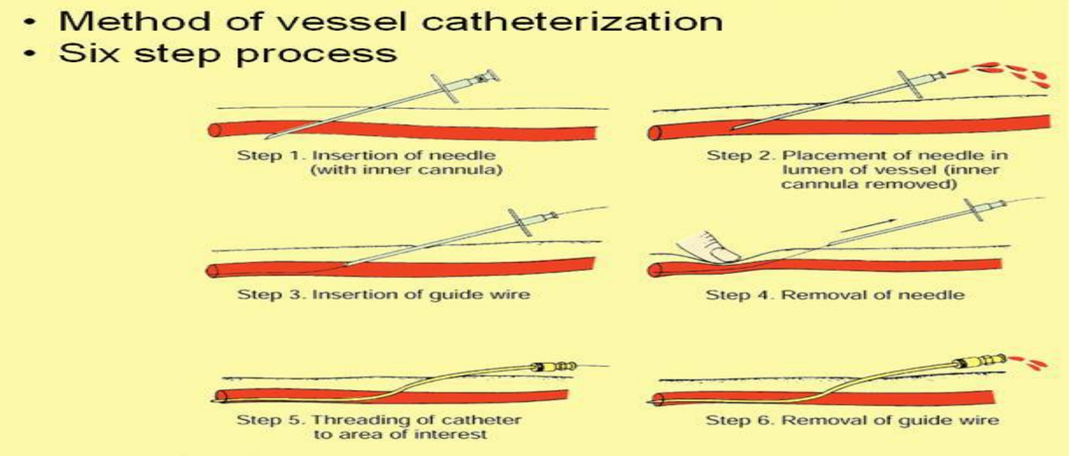
The scrub technician should then begin to prepare the procedure table with the necessary supplies needed for the case. Fill flush syringes with heparinized saline. Drape the patient according to sterile techniques. If groin access is being performed, expose the correct bulls eye of the drape and place over the groin site. Proceed to unfold the drape to completely cover the patient from shoulders down to the end of the table. Placing a sterile towel below the groin site between the legs is helpful in collecting blood loss during the procedure. Place sterile bags over the image intensifier , x-ray shield and any other equipment that may be attatched to the table that may contaminator the clinicians hands and equipments used for procedure.

PERCUTANEOUS VASCULAR ACCESS

The site of access is determined by the planned investigation and the anticipated anatomic and pathologic conditions of the patient.

THE SELDINGER TECHNIQUE

Modified Seldinger only punctures one side of vessel – this Percutaneous method can be used for arteries or veins - describes the method of catheter introduction that is not a direct stick.



FEMORAL ARTERY APPROACH

The operator should identify anatomical landmarks prior to giving local anesthesia, such as the inguinal ligament, which traverse from the anterior superior iliac spine to the pubic tubercle. The femoral artery generally crosses the inguinal ligament at an imaginary point that is located one-third from the medial aspect and two thirds from the lateral aspect of the ligament. The femoral pulse is then palpated approximately two finger breadths (2 to 3 cm) below the inguinal ligament, marking the site of arterial acces . One can also use fluoroscopy to identify the femoral head. The optimal access location would be at the site

over the inferior border of the femoral head. This approach is especially useful in obese patients. Locating the optimal site of entry is important. Entry sites above the inguinal ligament may lead to an increased risk of retroperitoneal bleeding, while entry sites that are too low may result in development of arteriovenous fistula or pseudoaneurysm.

FEMORAL VEIN APPROACH

The femoral arterial pulse is the landmark for the femoral vein. The femoral vein is located approximately 1 cm medial to the femoral artery; sometimes it is located partially behind the artery. Vein puncture may be successful only after several attempts, because venous pressure is low, it may be difficult to see unassisted back bleeding from the needle on entry. A 10 ml syringe with 5 ml of saline can be attached to the Seldinger needle and gently aspirated during the needle advancement.

RADIAL ARTERY APPROACH

Compared with the femoral artery, the radial artery is easily accessible in most patients and is not located near significant veins or nerves. The superficial location of the radial artery enables easy access and control of bleeding. No significant clinical sequelae after radial artery occlusion occur in patients with a normal Allen's test because of the dual blood supply to the hand through the ulnar artery. The results of the oximetric Allen test are divided into three grades of waveform during radial artery occlusion: type A, no change in pulse wave; type B, a damped but distinct pulse wave; and type C, loss of phasic pulse waveform. Radial artery cannulation can proceed with either type A or type B and is not recommended for type C. The patient should be well sedated and comfortably positioned. Several positioning techniques have been suggested. Arm abduction at a 70-degree angle on the arm board or placement immediately next to the femoral access site has been used for sheath insertion. Before complete sheath introduction, use of a vasodilator (nitroglycerin, verapamil) and intra-arterial lidocaine reduces artery spasm.

BRACHIAL ARTERY APPROACH

The brachial and radial pulses of both arms should be palpated. The brachial artery is approximately 3 to 5 diameter. The strongest brachial pulse is generally located 1 to 2 cm above the elbow crease. The antecubital fossa is then sterilized and draped.

BRACHIAL VEIN APPROACH

The antecubital vein anatomy varies greatly among patients a successful vein puncture depends on visual identification of an adequately sized antecubital vein. The vein located on the lateral antecubital area should not be used because of their course through the cephalic venous system over deltoid muscles, making catheter advancement through the relatively sharp turns in the shoulder area difficult.

INTERNAL JUGULAR VEIN APPROACH

The internal jugular vein is lateral to the carotid artery, medial to external jugular vein, and usually just lateral to the outer edge of the medial head of the sternocleidomastoid muscle. To identify landmarks, the operator instructs the patient to lie supine without a

pillow under the head and, in the case of right internal jugular, with the head turned 30 degrees to the left. Patient with low venous pressure may be placed in the Trenelenburg (head lower than feet) position. Many physicians and critical care societies recommend the use of ultrasound to guide access.

HEPARIN

Catheterization from the femoral artery had a higher incidence of major complications than catheterization from the brachial artery. One difference was that brachial catheterization used systemic heparinization to avoid thrombosis in the smaller diameter brachial artery. When systemic heparinization was adopted in femoral procedures, the rates of complications became standard practice to achieve full intravenous heparinization (5000 U) immediately left sided sheath was inserted. Lesser amounts of heparin (2,500 to 3,000) were used, particularly in smaller patients, and additional heparin (up to a total 50 to 70 U per Kg) was given procedure went on to a coronary intervention. This type of higher heparin dosing is routinely monitored by an activated clotting time (ACT) machine in the cardiac catheterization laboratory, and titrated to an ACT of roughly 300 seconds.

RIGHT HEART CATHETERIZATION

Only the right heart catheterization can provide data regarding mean left heart filling pressure (the pulmonary capillary wedge, left ventricular end diastolic pressure), detect pulmonary arterial hypertension, measure the cardiac output, and detect left-to-right intracardiac shunts.

Indications for right heart catheterization:

- Congenital heart disease, including atrial septal defect, ventricular septal defect, patent ductus arteriosus.
- Pulmonary thromboembolic disease
- Suspected pericardial tamponade or constriction
- As part of endomyocardial biopsy or electrophysiologic studies
- As part of left heart catheterization and coronary angiography in complicated cases with left ventricular failure, previous myocardial infarction, or cardiomyopathy
- Valvular heart disease

The right heart can be approached through the femoral, internal jugular or subclavian veins. Once the inferior vena cava or superior vena cava is reached, the catheter is advanced through the right atrium, right ventricle, and pulmonary artery to a distal pulmonary vessel. Right ventricular irritability may be noted when the catheter tip passes through the right ventricle. The course of the catheter is followed with pressure monitoring through the catheter and with fluoroscopy. When indicated, blood samples are taken, and pressure are recorded as the catheter is advanced. If left heart catheterization is planned, the catheter may be left in the distal pulmonary vessel, so that simultaneous left ventricular and pulmonary artery wedge pressure waveforms can be from the pulmonary artery to the right ventricle and from right ventricle to the right atrium. These

measurements are used to determine valve gradients and to evaluate pulmonic and tricuspid valve function. Blood sample can also be taken as the catheter is withdrawn for detection of left to right shunts. If pulmonic or tricuspid valve disease is suspected, contrast can be injected for digital imaging of the right atrium, right ventricle, or pulmonary artery.

CARDIAC OUTPUT STUDIES

The method of cardiac output determination includes the Fick oxygen method, indicator dilution technique, and the thermodilution method.

Fick Cardiac Output: The **Fick** formula, proposed by Adolph Fick, is considered the "gold standard" of cardiac output. It determines blood flow by measuring oxygen uptake.

Require 5 elements:

- Hb (obtained from blood sample).
- Aortic O_2 saturation(arterial blood sample.)
- PA O_2 saturation (PA sample).
- Hb O_2 carrying constant-1.36(each gram of Hb can carry 1.36 ml of O_2.

FICK CO=O_2 consumption /AVO$_2$ difference *10.

CO =VO$_2$/13.6*Hb(Ao-PA)

Thermodilution and Indicator Dilution Method: thermodilution and indicator dilution methods are based on principle that if a known amount of an indicator is added to an unknown quantity of flowing liquid and concentration of the indicator is then measured downstream, the time course of its concentration gives a quantitative index of the flow. Applied to the circulatory system, the amount of indicator, and the time during which the first circulation of the substance occurs can be used to compute cardiac output.

The thermodilution technique using cold or room temperature dextrose or saline injectate solution is the most frequently used cardiac output determination method in cardiac catheterization laboratories. The benefits of the thermodilution technique are that (1) it is performed over a short period and is, therefore, more likely to be recorded during a period of study state; (2) it is most accurate in patients with normal or high cardiac output; (3) the indicator used is inert and inexpensive; (4) it does not require an arterial puncture; and (5) the computer analysis curve is reasonably simple to interpret. Drawbacks of this method include its unreliability in the presence atrial fibrillation, significant tricuspid regurgitation, and its tendency to overestimate cardiac output in patients with low cardiac output.

LEFT HEART CATHETERIZATION

Left heart catheterization is used to perform coronary angiography for the evaluation of coronary anatomy, to obtain pressure measurements to evaluate mitral and aortic valve function, and to perform left ventriculography to evaluate left ventricular function.

The two main approaches into the left heart are retrograde entry through the aortic valve by either percutaneous femoral, which is the most common, or brachial approach and trans septal entry from the right atrium. The progress of the catheter in both approaches is followed by fluoroscopy and pressure measurement. For mitral valve studies, simultaneous pulmonary artery wedge and left ventricular pressures or simultaneous left atrial and left ventricular pressures are recorded to evaluate pressure difference across the valve. To evaluate aortic valve function, pullback pressure is recorded as the catheter withdrawn from the left ventricle to the aorta. Digital imaging may be performed during contrast injection of the left atrium, left ventricle, or aortic root to evaluate valve function further.

Trans septal Left Heart Catheterization: The trans septal approach to left heart catheterization involves crossing from the right atrium to the left atrium through the fossa ovalis. This technique is infrequently done for diagnostic catheterizations but can be used in the rare situation when retrograde left heart catheterization is not possible due to severe aortic stenosis or a prosthetic valve that cannot be adequately evaluated by echocardiogram or transesophageal echocardiography. More common uses of the trans septal approach include mitral valvuloplasty, electrophysiology studies requiring access to the left atrium or left ventricle, and transcatheter closure of patient foramen ovale or atrial septal defect.

VENTRICULOGRAPHY

The left ventriculogram is an integral part of every coronary arteriographic study and provides information about LV wall motion and overall function of the heart. Abnormal wall motion indicates the presence of coronary ischemia, infarction, aneurysm, or hypertrophy. Left ventriculography also provides quantitative information, such as the ventricular volumes during systole and diastole, the ejection fraction, the rate of ejection, the quality of contractility, the presence of hyperdynamic hypertrophic contraction, and valvular regurgitation. Ventriculography may be performed before or after coronary angiography. Coronary angiography is routinely performed first because ventricular function can be obtained through noninvasive methods in case of complications that terminate the study prematurely. In patients with LMCA or aortic stenosis, left ventriculography has often been when postprocedural hypotension is anticipated. Low-volume, non-ionic, low-osmolar contrast ventriculograms can be performed while little or no induced hypotension.

Setup of Contrast Media Power (High-Pressure) Injectors

Because contrast media is viscous and great force is necessary to inject contrast rapidly through small catheters, power injectors are always used to deliver a preset contrast volume over a brief period. Three steps are critical to safe ventriculography: (1) contrast loading, (2) air bubble clearing from the injection syringe and high-pressure connecting tubing, and (3) correct injector settings. The most important step of the setup is to clear all air and bubbles from the transparent pressure injector syringe and tubing before an injection. This is an obligation of all physicians and nurses in the laboratory. There is no excuse for injection of air during contrast ventriculography.

Typical injection pressure settings for ventriculography are as follows:

- Flow rate (10 to 16 ml/sec).
- Total volume (20 to 50 ml)
- Pressure limit (800 to 1200 psi)
- Rise time (0.2 to 0.5 second)

Indications and Complications of Ventriculography:

Indications of left ventriculography:

- Identification of LV function for patients with coronary artery disease, myopathy, or valvular heart disease
- Identification of ventricular septal defect
- Quantitation of the degree of mitral regurgitation
- Quantitation of the mass of myocardium for regression of hypertrophy or other similar research studies.

Indications of right ventriculography:

- Documentation of tricuspid regurgitation
- Assessment of pulmonary stenosis
- Assessment of RV dysplasia for arrhythmias
- Assessment of abnormalities of pulmonary outflow tract
- Assessment of right to left ventricular shunt.

Complications:

- Cardiac arrhythmias, especially nonsustained VT do not require treatment. Sustained VT and ventricular fibrillation require immediate cardioversion.
- Intra myocardial contrast media “staining” during power injection
- Embolism (thrombi or air)
- Contrast-related complications

- Transient hypotension (<15 to 30 seconds) was common with ionic high osmolar contrast media.

Catheter Position

The optimal catheter position for left ventriculography is one of that avoids contact with the papillary muscles or is positioned too close to the mitral valve so that mitral regurgitation is not produced artificially. For most catheters a mid-cavity positions seems best because contrast material fills most of the LV chamber and apex, and catheter during injection dose not interfere with mitral valve function. In this position pigtail catheter side holes are fell below the aortic valve, which improves chamber opacification. Angled (145 degree) pigtail catheters and helical tip designs (halo catheter) may provide better quality ventriculography with less induced ectopy and mitral regurgitation. Standard left ventriculographic views are (1) a 30 degree RAO that visualize the high lateral, anterior, apical, and inferior LV walls and (2) a 45- to 60-degree LAO, 20-degree cranial angulation that best identifies the lateral and septal LV walls. The LAO with cranial angulation provides a view of the interventricular septum. For right ventriculography a Berman balloon tipped or NIH catheter produces excellent opacification. The angiographic projection of right ventriculography is not standardized. An AP cranial or lateral projection is commonly used to visualize the septum and right ventricular outflow tract (RVOT). Injection rates range from 8 to 10 ml/sec for volume of 20 to 30 ml.

Regional Left Ventricular Wall Motion: The normal pattern of LV contraction has been defined as a uniform, concentric, inward motion of all points along the ventricular inner surface during systole. Uniform wall motion depends on the cooperative and sequential contraction of the heart muscle, producing maximal effective work at minimal energy costs. This coordinated contraction is called synergy. Uncoordinated contractions of LV wall motion are given names according to the severity of asynergy. Abnormal LV motion is particularly obvious in patients with severe coronary artery disease or cardiomyopathy. Several methods exist to analyze LV wall motion. The RAO and LAO left ventriculograms are divided into five segments. Points are assigned to the following wall motion abnormalities.

- Normal contraction
- Moderate hypokinesis (a diminished, but not absent, motion of one part of the LV wall is weak or poor contraction)
- Severe hypokinesis
- Akinesis (total lack of motion of a portion of the LV wall, i.e., no contraction)
- Aneurysm-dyskinesis (paradoxical systolic motion or expansion of one part of the LV wall, i.e., an abnormal outward bulging during systole).

ASCENDING AORTOGRAPHY

Ascending aortography may be necessary in the course of coronary angiography and interventions.

Indications:

- Aortic aneurysm or aortic dissection
- Aortic insufficiency
- Nonselective visualization of coronary bypass graft
- Supravalvular aortic stenosis
- Brachiocephalic or arch vessel disease
- Coarctaion of the aorta
- Aortic –PA or aortic to right sided heart (e.g., sinus of valsalva fistula) communication.
- Aortic periaortic neoplastic disease
- Arterial thromboembolic disease
- Arterial inflammatory disease.

Contraindications:

- Contrast media reaction
- Injection to false lumen of aortic dissection
- End hole catheter malposition
- Inability of the patient to tolerate additional radiographic contrast media

Left anterior oblique or lateral projection: The LAO view is excellent for identifying dissection of the ascending aorta extending up to the neck vessel, optimally delineating the aortic arch; opening of the aortic curvature; and providing clear views of the innominate, common carotid, and left subclavian arteries. The coronary arteries at the root of the aorta are displayed in semilateral projection.

Right anterior oblique projection: The descending thoracic aorta and the ascending aorta may be superimposed across the arch in the AP or LAO projection.

Injection rates and catheter position: Aortography can be performed by use of a minimum flow rate of 15 to 20 ml/sec for total volumes of 40 to 60 ml. Use of multiple side hole catheters reduces the risk of extending or inducing a dissection during contrast medium injection. The catheter should be positioned just above the aortic valve but not close enough to interfere with the valve opening or closing.

PULMONARY ANGIOGRAPHY

Pulmonary angiography provides visualization of vascular abnormalities of the lung vessels (e.g., intraluminal defects representing pulmonary emboli, shunts, stenosis, arteriovenous (AV) malformation, and anomalous connections). It should be preceded by the measurement of pressures of the right side of the heart. Indications of pulmonary angiography include the following:

- Pulmonary embolism
- Peripheral pulmonic stenosis or pulmonary AV fistula
- Anomalous pulmonary venous drainage

- Follow-through for left atrial opacification (suspected atrial myxoma, large thrombi)

Contraindications:

- Allergy to contrast agent
- Pulmonary hypertension with PA systolic pressure (>60 mm Hg)
- Acute RV volume overload (after injection of contrast medium, increased volume may produce RV failure, low cardiac output state, shock, or death)

RADIO CONTRAST MEDIA IN PERCUTANEOUS CORONARY INTERVENTION

Contrast media can be classified as ionic and non-ionic. Most commonly used contrast media are iodine based media. These may be bound either in an non-ionic (organic compound or an ionic compound. Organic agents which covalently bind the iodine have few side effects as they do not dissociate into component molecules. Iodine based contrast media are water soluble. Osmolality is the amount of concentration of solute present in a solvent per Kg. osmolality of blood is 285-295mOsm/Kg. Non-ionic contrast media have lower osmolality and tend to have fewer side effects. Currently available iodinated contrast media based on one(monomer) or two(dimer) triiodinated benzene rings, which can be broadly classified into three groups according to their osmolality, defines as the number of particles dissolved in 1Kg of water.

They are classified based on their osmolality and ioinicity into

- High-osmolar (HO) >1,600 mOsm/1 – no longer recommended.
- Low-osmolar (LO) <850 mOsm/1- ionic and non ionic.
- Iso osmolar (IO) 290 mOsm- non ionic.

Compound	Name	Type	Iodine content(mgI/ml)	Osmolality	
Non ionic	Iopamidol(isovue)	monomer	370	796	Low
Non ionic	Iohexol(omnipaque)	monomer	350	884	Low
Non-ionic	Ioxilan(oxilan)	monomer	350	695	Low
Non-ionic	Iopromide(ultravist)	monomer	370	774	Low
Non-ionic	Iodixanol(visipaque)	dimer	320	290	low

Major adverse outcomes related to the use of current generation contrast media are, contrast induced nephropathy, anaphylactoid reaction and left ventricular failure. The other complications like arrhythmia, anticoagulation and negative ionotrpic effects are no longer encountered with use of non ionic contrasts.

Anaphylaxis

Anaphylaxis is a rapidly progressing life threatening allergic reaction, in which immune system responds to otherwise harmless substance from the environment. Unlike other allergic reactions, however , anaphylaxis can kill. Reaction may begin within minutes or even seconds of exposure and rapidly progress to cause airway construction, skin and intestinal irritation, and altered heart rhythms. In severe cases, it can result in complete airway obstruction, shock and death.

Causes and symptoms: Like the majority of other allergic reactions, anaphylaxis is caused by the release of histamine and other chemicals from mast cells. Mast cells are a type of white blood cells and they are found in large numbers in the tissue that regulate exchange with environment; the airways, digestive system and skin. On their surfaces, mast cells display antibodies called IgE(immunoglobulin type E). These antibodies are designed to detect environmental substances to which the immune system is sensitive. Substance from a genuinely threatening source, such as bacteria or viruses, are called antigens. A substance that most people tolerate well, but to which others have an allergic response, is called an allergen. When IgE antibodies bind with allergens, they cause the mast cells to release histamine and other chemicals, which spill out onto neighboring cells. The interactions of these chemicals with receptors on the surface of blood vessels can causes the vessels to leak fluid into surrounding tissues, causing fluid accumulation, redness and swelling. On the smooth muscle cells of the airways and digestive systems, they cause construction. On nerve endings, they increase sensitivity and cause itching.

Contrast- induced nephropathy (CIN)

CIN has become a leading cause of in-hospital acute renal failure. It has been shown to have a significant adverse impact on the short- and long-term outcome of patients. CIN is defined as increase in serum creatinine over baseline by 48 hr, such as 25% above baseline or an absolute increase of 0.5 mg/dl. The course of CIN generally transpires over a period of about 10 days with a rise in creatine most often with 24 hr, peaking at 3-5 days, and the returning to baseline over the next 5 days.

Risk factors affect CIN

Patient related risk factors	Procedure related risk factors
• **Renal insufficiency** • **Diabetes mellitus with renal insufficiency** • **Age > 75 years**	• **Multiple contrast media usage within 72hrs** • **High volume of contrast media**

• **Volume depletion** • **Hypotension** • **Low cardiac output** • **Class IV CHF** • **Other nephrotoxins** • **Renal transplant** • **Hypoalbunemia(<35g/l)**	• **High osmolality of contrast media**

CORONARY ANGIOGRAPHY

Indications

The American College of Cardiology/American Heart Association (ACC/AHA) Task force has established indications for coronary angiography in patients with known or suspected coronary artery disease.

Class I: There is evidence and/or general agreement that coronary angiography should be performed to risks stratify patients with chronic stable angina in the following settings:

- ∇ Disabling angina symptoms (Canadian Cardiovascular Society (CCS) class III and IV) despite medical therapy
- ∇ High-risk criteria on noninvasive testing indepentendent of the severity of angina
- ∇ Survivors of sudden cardiac death or serious ventricular arrhythmia
- ∇ Symptoms and signs of heart failure.
- ∇ Clinical features suggest that the patient has a high likelyhood of severe coronary artery disease.

Class IIa: The evidence or opinion is in favour of performing coronary angiography to risk stratify patients with chronic stable angina in the following settings:

- ∇ Left ventricular ejection fraction less than 45%, CCS class I or II angina evidence, on noninvasive testing, of ischemia that does not meet high-risk criteria.
- ∇ Noninvasive testing dose not reveal adequate prognostic information.

Class IIb: The evidence or opinion is less well established for performing coronary angiography to risk stratify patients with chronic stable angina in the following settings:

- ∇ Left ventricular ejection fraction greater than 45%, CCS class I or II angina and evidence, on noninvasive testing, of ischemia that does not meet high-risk criteria
- ∇ CCS class III or IV angina that improves to class I or II with medical therapy.
- ∇ CCS class I or II angina but unacceptable side effect to adequate medical therapy.

Class III: there is evidence and/or general agreement that coronary angiography should not be performed to risk stratify patients with chronic stable angina in the following settings:

- ∇ CCS class I or II angina that responds to medical therapy and, on noninvasive testing, shows no evidence of ischemia.
- ∇ Patient preference to avoid revascularization.

Injection Technique

The contrast medium, a viscous, iodinated solution used to opacity the coronary arteries, can be injected by hand through a multivalve manifold the tip of the syringe is kept pointed down so that any small bubbles float up and are not injected into the circulatory system.

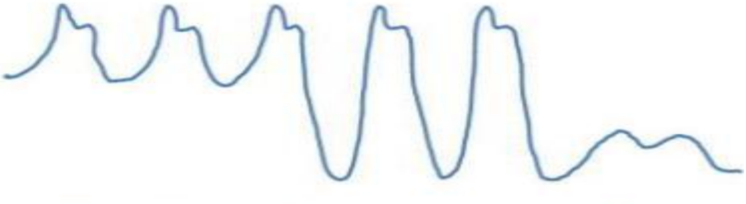
RCA: 6 ml at 3 ml/sec; maximal psi 450.

LCA: 10 ml at 4 ml/sec; maximal psi 450.

Panning Techniques

Most laboratories use X-ray image screen sizes (e.g., 7 inches or less in diameter) that may preclude having the entire coronary artery course visualized without panning over the heart to include the late filling portions of the arterial segments and any collaterals filling. In addition, in most views some degree of panning is necessary to identify regions that are not seen from the initial setup position. Some branches may unexpectedly appear later from collateral filling or other unusual arterial input sources.

Damping and Ventricularization of the Pressure Waveform



A fall in overall catheter tip pressure (damping) or a fall in diastolic pressure only (ventricularization) during catheter engagement in a coronary ostium indicates obstruction of the catheter tip or interference with coronary inflow.

ANGIOGRAPHIC PROJECTONS

Nomenclature

Anteroposterior (AP view): the x-ray beam is travelling from posterior to anterior perpendicularly when the patient is in supine position.

Right Anterior Oblique(RAO view): the image intensifier is towards the right side of the patient and x-ray beam from left to right side of the patient.

Left Anterior Oblique(LAO view): the image intensifier is placed left side and the x-ray beam travels from right to left side of the patient.

Caudal view : the image intensifier will be towards the feet of the patient.

Cranial view: the Image intensifier towards the head side of the patient.

Angiographic views for specific coronary artery segment

Left main - shallow LAO(10^0-15^0)
Proximal LAD - AP caudal(0^0-30^0 to 40^0),LAO caudal(40^0-40^0)
Mid LAD - AP cranial(0^0-45^0),LAO cranial (40^0-20^0), RAO cranial(20^0-40^0)
Distal LAD - AP cranial(0^0-45^0),LAO cranial(40^0-20^0)
Proximal LCx - AP caudal(0^0-30^0),LAO caudal(40^0-40^0)
Obtuse marginal - RAO caudal(20^0-20^0),AP caudal(0^0-30^0)
Distal LCx - LAO cranial(40^0-20^0)
Proximal RCA - LAO caudal(30^0-20^0)
Mid RCA - LAO cranial(40^0-20^0),RAO(30^0),Left lateral
PDA - AP cranial(0-30^0),LAO cranial(40^0-20^0)
PLB - AP cranial(0-30^0)

BYPASS GRAFT ANGIOGRAPHY

Saphenous Vein Grafts: The proximal anastomosis of most aortocoronary SVGs lies on the anterior surface of the aorta, several centimeters above the sinuses of Valsalva. Usually, the location of the various grafts in the relation to one another follows a predictable sequence. Grafts to the left circumflex artery (LCX) are typically placed most superior, followed by grafts to the diagonal branches, LAD, and RCA. Typically grafts to the right coronary artery can be best visualized and cannulated while in the left anterior oblique (LAO) projection; grafts to the left coronary artery (LCA) system are most easily found while in the right anterior oblique (RAO). If graft cannot located, do not assume that the graft is occluded. Other catheters with different angulation may be necessary. If further attempts fail, aortography may be helpful in locating difficult to find grafts.

Internal Mammary Artery Grafts: The LIMA is often anastomosed to the mid-or distal LAD, although it is sometimes attached to the diagonal branches or the LCX instead. It typically arises anteroinferiorly from the left subclavian artery, 1 to 3 cm beyond the vertebral artery. Once engaged, the LIMA graft is injected in at least two projections, paying special attention to the anastomotic site. Forceful injections are discouraged. Straight RAO and LAO projections are most commonly employed. Cranial angulation can be added to either projection to better visualize the distal aspect of the LAD. A cross-table lateral view is sometimes helpful to gain an additional view of the anastomotic site. Selective visualization of the right internal mammary artery (RIMA) is similar to that of the LIMA above, but it is more difficult. A JR4 or LIMA catheter is advanced into the proximal aortic arch past the origin of the innominate artery. The RIMA is often used as a “free” graft, with its proximal anastomosis in the ascending aorta. In this case, the procedure for cannulating the graft is the same as that for saphenous grafts.

LESION QUANTIFICATION

QUANTITATIVE ANGIOGRAPHY

Although visual estimations of coronary stenosis severity are used by virtually all clinicians to guide clinical practice, “eyeball” estimates of present diameter stenosis are limited by

substantial observer variability. The assessment of intermediate coronary lesions (40-70%), having been largely supplanted by physiological measures of stenosis significance, most often fractional flow reserve (FFR). Using hand drawn arterial contours, reference vessel and minimal lumen diameters were measured and were used to evaluate the effect of pharmacological intervention for a number of angiographic plaque regression studies.

LESION COMPLEXITY

Heterogeneity of the composition, distribution and location of atherosclerotic plaque within the native coronary artery results in unique patterns atherosclerotic plaque within the native coronary artery results in unique patterns of stenosis morphology in patients with CAD.

Charact4eristics of ACC/AHA type A,B and C lesions

TYPE A LESIONS:(High success, 85%; low risk	
Discrete (<10mm length)	**Little or no calcification**
Concentric	**Less than totally occlusive**
Readily accessible	**No ostial in location**
Non angulate segment <45^0	**No major branch involvement**
Smooth contour	**Absence of thrombus**
TYPE B LESIONS(moderate success,60 to 85%; moderate risk	
Tubular(10-20 mm length)	**Ostial in location**
Eccentric	**Bifurcation lesions requiring**
Moderate tortuosity of prox. segment	**Double guide wires**
Irregular contour	**Some thrombus present**
Moderately angulated,45-90^0	**Total occlusion<3 months old**
Moderate to heavy calcification	
TYPE C LESIONS(low success, <60%; high risk)	
Diffuse (>2mm length)	**Degenerated vein grafts with friable lesions**

Excessive tortuosity of prox. segment	Total occlusion > 3 months old
Extremely angulated ,>90 ^{0}	Inability to protect major side branches

Characteristics of SCAI type I-IV lesions

SCAI Type I (higher success expected, lower risk).

Does not meet the criteria for ACC/AHA Type C lesion
patent

SCAI Type II Diffuse(<2cm length)

Excessive tortusity of proximal segement
Extremily angulated segments, 90^{0}
Inability to protect major side branches
Degenerated vein grafts with friable lesions.
patent

SCAI Type III

Does not meet the criteria for ACC/AHA Type C lesion
Occluded

SCAI Type IV

Diffuse (>2cm length)
Excessive tortusity of proximal segment
Extremely angulated segments ,>90^{0}
Inability to protect major side branches
Degenerated vein grafts with friable lesions
Occluded more than 3 months.

LESION LENGTH

- **Focal- discrete :** below(10mm)
- **Tubular :** from (10-20 mm)
- **Diffuse - long** (<20mm)

Long lesions increases the procedure and post procedure risk.Needs long stents increases the risk of instent restenosis. More like hood to have overlapped stents and to miss lesion segment. Chance of stent mal-deployment increases. Long lesion indicates large atherosclerotic burden and so more cardiac events.

A TANDEM LESION: is two lesions close to each others with a normal segment between(Like the bullets loaded in a tandem fashion in a gun).

OSTIAL LESIONS: defined as the lesion airising within 3mm from the ostium of the artery. Challenges are: usually fibrotic or calcified and prone to recoil due to the greater thickness of muscular and elastic tissue in the aortic wall(usually dilatation is advisable).

Misinterpreted with ostial spasm(IC nitrates-small cath cuspngram-IVU-CT). more liable to in-stent restenosis. Retrograde dissection is a life threatening condition and requires immediate surgical consultation. Difficult visualization of coronary ostium on cath removal and cuspogram.

THROMBUS BURDEN LESION: there will be more risk of no reflow, distal embolization,thrombus shift to aside branch, thrombus prolapse and lemon seed effect, increase the risk of instent thrombosis. Large intracoronary thrombi are most often noted as intraluminal filling defects during angiography in STEMI and may be treated with a combination of pharmacological agents (e.g., glycoprotein IIb/IIIa inhibitors) and mechanical devices (e.g., rheolytic thrombectomy, manual aspiration catheters).

CALCIFIED LESION: more risk of non dilatation lesion, dissection and perforation.

TOTAL OCCLUSION: diificulty in passage of a guidewire across the occlusion depends on the occlusion duration and on certain lesion morphologic features, such as bridging collaterals, occlusion length of more than 15mm and absence of a " nipple" to guide advancement of the guidewire. Presence of a total occlusion remains one of the major reasons for referral of patients for coronary bypass surgery.

ANGULATED LESION: risk for dissections,inability to deliver the stent, straightening of the vessel that may predispose to late stent fracture.

BIFURCATION LESION: the optimal strategic approach for bifurcation lesions remains controversial. But in general placement of one stent is preferable to stent placement in both the parent vessel and side branch. Risk for side branch occlusion during PCI with plaque shifting is there. It is relates to the relative size of the parent and branch vessel, the location of the disease in the parent vessel, and the stenosis severity in the origin of the side branch.

CORONARY PERFUSION: Perfusion distal to a coronary stenosis can occur anterograde by means of the native vessel, retrograde through collateral, or through a coronary bypass graft. The rate of anterograde coronary flow is influenced by both the severity and complexity of the stenosis and status of the microvasculature.

PERCUTANEOUS CORONARY ARTERY INTERVENTION

Percutaneous Transluminal Coronary Angioplasty include techniques such as balloon angioplasty,stents,blades, burrs and aspiration catheters.

P-Percutaneous: access of the inner body by a needle puncture of the skin, instead of an incision approached to expose the inner body.
C-coronary : the arteries that supply the heart with nutrient rich blood
I-intervention :an action to improve a situation.

History of PCI

PCI was had its origin in the 1960s with dilatation techniques by Dr. Dotter and Judkins. This approach was restricted for clinical application with coronary arteries. In the 1970s, Dr. Gruen zig replaced the dilator with an inflatable balloon used to inflate the

lesion. The first PTCA of a stenotic coronary artery in a human took place in 1977. Balloon angioplasty was the only revascularization techniques used until atherectomy catheters and stents were introduced in the 1990s. the first stents were metal stents and became known as bare metal stents(BMS). In the early 2000s, stents had evolved to include a medication that eluted off of the metal stents to prevent cellular proliferation that restenosed the artery. Over the years , stents have evolved with a change in metal and designs that improve their deiliverability, strength and success at keeping the artery open.

How does PCI work

PCI requires a few basic compounds in order to dilate an artery with a stenotic lesion. The first piece is a guide catheter that allows access, revascularization and delivery of equipment to the artery. The second piece is a guidewire that is advanced across the lesion and allows angioplasty equipment to travel over. The third piece is an interventional catheter (balloon , stent, atherectomy, thrombectomy) that travels to the lesion to revascularize the artery.

The guide catheter is positioned at the ostium of the coronary artery and thin guidewire is advanced across the lesion into the distal aspect of the artery. The tracking it over the guidewire and positioned at the area of lesion. Position is verified with the injection of contrast media to confirm correct position. The catheter is then inflated to expand the luminal area of the artery and then deflated to allow blood to resume flow through the artery.

Indications for PCI:

- Angina pectoris causing sufficient symptoms despite optimal medical therapy
- Mild angina pectoris with objective evidence of ischemia (abnormal stress testing or physiology) and high-grade lesion (>70% diameter narrowing) of a vessel supplying a large area of myocardium.
- Unstable angina
- Acute myocardial infarction (MI) as primary therapy or in patients who have persistent or recurrent ischemia after failed thrombolytic therapy.
- Angina pectoris after coronary artery bypass graft surgery
- Restenosis after successful PCI
- Left ventricular dysfunction with objective evidence of viability of a vessel supplying the myocardium.
- • Arrhythmia secondary to ischemia

Contraindications for PCI:

- Unsuitable coronary anatomy.
- Extremely high-risk coronary anatomy in which closure of vessel would result in patient death.
- Contraindications to coronary artery bypass graft surgery (however, some patients have PCI as their only alternative to revascularization)
- Bleeding diathesis

- Patient noncompliance with dual antiplatelet therapy and unwillingness to follow post-PCI instructions
- Multiple in stent restenosis
- Patient who cannot give informed consent.

Complications to consider

As the arterial wall becomes compressed by the interventional catheters **small tears and disruptions** occur from the shear force of the balloon. This endothelial damage will trigger a healing phase that sends platelets to the site to develop a blood clots. Appropriate anticoagulation therapy will prevent a blood clot from forming that will occlude blood flow at the site of intervention. After the balloon has been inflated , the inner layer may tear and peel away from the arterial wall. This is known as **dissection**. If a dissection is headed distally, then it will contribute to grow just as wind blows in a sail. Dissection planes will need to be stented to avoid the progression of the dissection.

Balloon inflation can cause plaque to become dislodged from the arterial wall and **embolize** down stream potentially occluding a distal branch of the artery. Further intervention may be required to reestablish blood flow to the region.

Abrupt closure of the artery may occur from the elastic recoil nature of the artery. Stretching of the artery with a balloon catheter usually causes the arterial wall to lose its elastic property.

Perforation of the arterial wall is the most catastrophic complications that can occur with interventional equipment. Immediate treatment is required to prevent blood from flowing into the pericardial space. Balloon inflation completely occluded the perforated area is usually performed until subsequent therapy can be performed. The perforation is usually treated with a covered stent. Peri cardiocentesis to drain fluid from the pericardial space is usually performed.

Plaque shifting may occur during inflations. This shifting can cause a narrowing distal to the area treated and may occlude a nearby side branch. If a side branch becomes occluded due to plaque shifting , angioplasty at the region may be required.

PRIMARY ANGIOPLASTY

ST elevation MI accounts for 25 to 40% of acute coronary syndrome events worldwide. ST elevation MI is caused by the total occlusion of an epicardial artery by a thrombus. The nidus for thrombus formation could be an ulcerated plaque or erosion on the endothelial surface of the vessel. Reperfusion strategies have been tailored to re-establish flow in the occluded artery. The two reperfusion strategies available are fibrinolysis and primary PCI. Primary PCI is the catheter base reperfusion modality geared to re-establish flow. Reperfusion therapy has to be administered to all patients with STEMI within the windows period of 12 hours and primary PCI is the preferred mode of reperfusion.

What is door–to–balloon time and door–to–needle time?

Door-to-balloon time (DTB) is the time from first hospital arrival to first attempt at reperfusion with any intracoronary device. Both American College of Cardiology/American Heart Association and the European Society of Cardiology propose a DTB time of less than 90 min as standard. The door to needle time (DTN) is the interval between the patient's arrival at the hospital and the initiation of fibrinolytic therapy. Recommended standard DTN time is less than 30 minutes.

When is an invasive strategy preferred over thrombolysis (fibrinolysis)?

Invasive strategy is preferred over fibrinolysis when skilled PCI laboratory is available where medical contact to balloon dilatation (DTB) can be kept less than 90 minutes. Invasive strategy should be the preferred mode of reperfusion strategy in cases of high risk STEMI with cardiogenic shock and whenever there are contraindications for fibrinolysis like intracranial hemorrhage. If the presentation of MI is late (symptom onset was more than 3 hrs ago) or the diagnosis of ST-elevation myocardial infarction is in doubt after seeing the ECG and history, then preferred strategy should be invasive. The time dependency is critical with fibrinolysis because of the decrease in efficacy of the fibrinolytic agent as coronary thrombi mature over time.

Are there any clinical situations where thrombolysis is preferred over primary angioplasty?

When performed rapidly after presentation in an experienced centre, primary PCI is superior to pharmacological reperfusion therapy. But when invasive strategy is not an option in conditions such as catheterization, preoccupied laboratory, difficulties in vascular access or lack of access to a skilled PCI laboratory; fibrinlolysis should be the revascularization strategy. Fibrinolysis is also preferred in cases of delay to an invasive strategy, for example; prolonged transport to an interventional centre. When the patient presents within 3 hours after the onset of symptoms, fibrinolysis with a bolus thrombolytic over PPCI is acceptable.

Though primary angioplasty is a better reperfusion strategy than fibrinolysis, one should not forget that the time-to-treatment significantly affects the success of the reperfusion strategy. The benefits are **"time-dependent",** hence the rationale for the usage, **"Time is Muscle"**. Each 30 minute delay from symptom onset to reperfusion, increases the relative risk of 1 year mortality by 8%. When performed rapidly after presentation in an experienced centre, primary PCI is superior to pharmacologic reperfusion strategy. Although several studies have reported that referral to a PCI centre is superior to fibrinolysis in a local hospital, such studies were conducted in dedicated health care systems with extremely short transportation and door to balloon time at the PCI centre. Transportation to an invasive centre is definitely not preferred if the anticipated DTB time minus the DTN time is more than 1 hour.

Is there any time limit for primary angioplasty?

Though general consensus is that mortality benefit of PAMI can be obtained up to 12 hours after the onset of symptoms, it is reasonable to consider reperfusion therapy in patients with persistent symptoms and ST segment elevation in surface electrocardiogram beyond 12 hours.

What are the benefits of angioplasty over fibrinolysis?

An overview of short term results of 10 comparisons of the two approaches has shown that, compared to fibrinolysis; primary angioplasty results in a lower mortality (4.4% vs. 6.5%), translating into an absolute benefit of two lives saved per 100 patients treated with angioplasty compared with fibrinolysis. The reduction in the combination of death or non-fatal reinfarction after angioplasty compared with fibrinolysis is even more striking (11.9% vs. 7.2%). With respect to safety, stroke was reduced from 2.0% with fibrinolysis to 0.7% with angioplasty. The higher the risk of the patient, the greater will be the potential of primary angioplasty compared with thrombolysis

CORONARY ATHERECTOMY

Because the atherosclerotic plaque remains in the artery after balloon dilation, physical removal of the plaque from inside the coronary artery may improve procedural and clinical results. Three devices are developed for this purpose, but only the high speed rotablator remains in use today. Directional atherectomy catheter (DCA) is used or peripheral vascular disease, and the transluminal extraction catheter has been retired.

ROTATIONAL ATHERECTOMY (ROTABLATOR)

Utilizes a high speed rotating diamond coated burr to ablate atherosclerotic plaque and restore luminal patency. Rotates 160,000 rpm for smaller burrs and 140,000rpm for burrs bigger than 2 mm. The plaque is ablated into small particles which are removed by the reticula-endothelial system of the body. The resultant lumen is often smooth and free smooth and free of dissection Vs PTCA where there are significant dissections.

The rotation works on two principles:

a. **Differential cutting**: cuts first more rigid, inelastic material and not harming the softer skin
b. **Orthogonal displacement of friction**: reduces the friction between vessel wall and entering device as the principle of turning screw in a wall. Friction develops in a longitudinal direction between the guide wire and the device. High speed rotation changes the friction vector to a circumferential(perpendicular) direction. This will decreases the forward friction facilitates burr advancement through tortuous non-compliant vessels.

INDICATIONS FOR ROTABLATION

a) Calcified lesions, for facilitating stent delivery and expansion.
b) Un-dilatable lesions, (fibrotic) for facilitating stent delivery and expansion.
c) In selected cases of diffuse in-stent restenosis – (for decreasing intimal hyperplasia volume and subsequent balloon slippage and for reducing the need for re-stenting).

CONTRAINDICATIONS

a) Occlusion through which a guidewire will not pass
b) Last remaining vessel with compromised left ventricular function
c) Saphenous vein grafts
d) Angiographic evidence of thrombus prior to treatment with Rotablator system.
e) Angiographic evidence of significant dissection at the treatment site.
f) Lesion length in excess of 25mm.

PRECAUTIONS

a) Appropriate drug therapy including(but not limited to) anticoagulant and vasodilator therapy must be provided to the patient.
b) A temporary pacing lead may be necessary and is particularly recommended during the treatment of lesions in the right coronary and dominant circumflex arteries to resolve electrophysiological aberrations which may occur.
c) There has been limited experience if brachial and radial approach.
d) When the rotablator guidewire and/or catheters are in the body, they should only be manipulated equipment that provides high resolution images.

COMPLICATIONS

a) Bradycardia(RCA lesions)
b) Vasospasm
c) Burr entrapment
d) Intimal splits and medial dissections.
e) In regions of tortuosity or eccentric palque, crater or gutter formation can occur(lesion bias).
f) Heat generation : 2.6^{0} c to 13.9^{0} c may contribute to increased risk of periprocedural myocardial infarction(MI) and restenosis associated with excessive decelerations.

Prevention: smaller burr sizing (burr to artery ratio<0.7) reduces angiographic complications compared with more aggressive sizing.

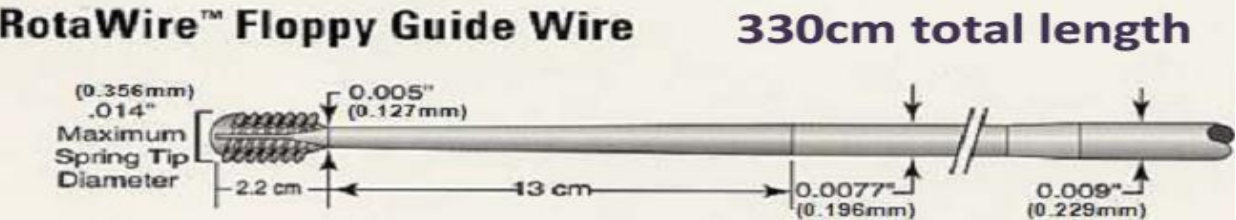
THE ROTABLATOR SYSTEM

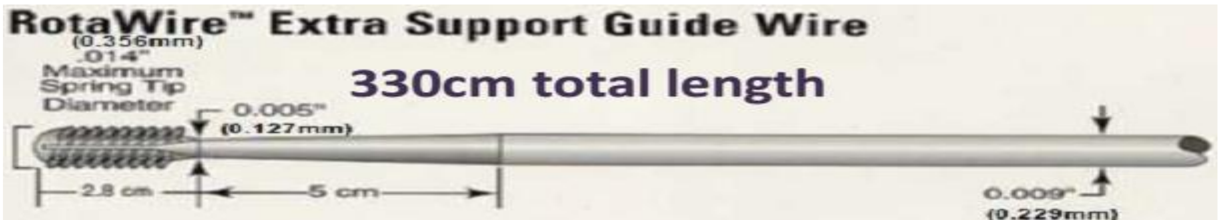
The rotablator works on a compressed air / gas system. Either we can use a higher pressure compressed air / nitrogen cylinder or we can use an online system of air compressor. There should be a pressure of 6.5 to 7.5 bar in the cylinder, otherwise the system is better because it is often difficult to maintain the required pressure in the high pressure gas cylinders and other logistic issues like transportation etc.
In addition to the console and the high pressure gas system, there is the foot pedal for activating the rotablator and the dynaglide. There should be a saline flush continuously flowing to allow smooth rotation and also to cool the rotating parts. The pressure in the saline flush system should be 300 mmHg. Single burr with burr to artery of 0.5 to 0.6. rotational speed of 140,000 to 150,000 rpm. Gradual burr advancement using **"pecking"** motion. Short ablation runs of 15 to 20s. avoidance of deceleration>5000 rpm. Final polishing run.

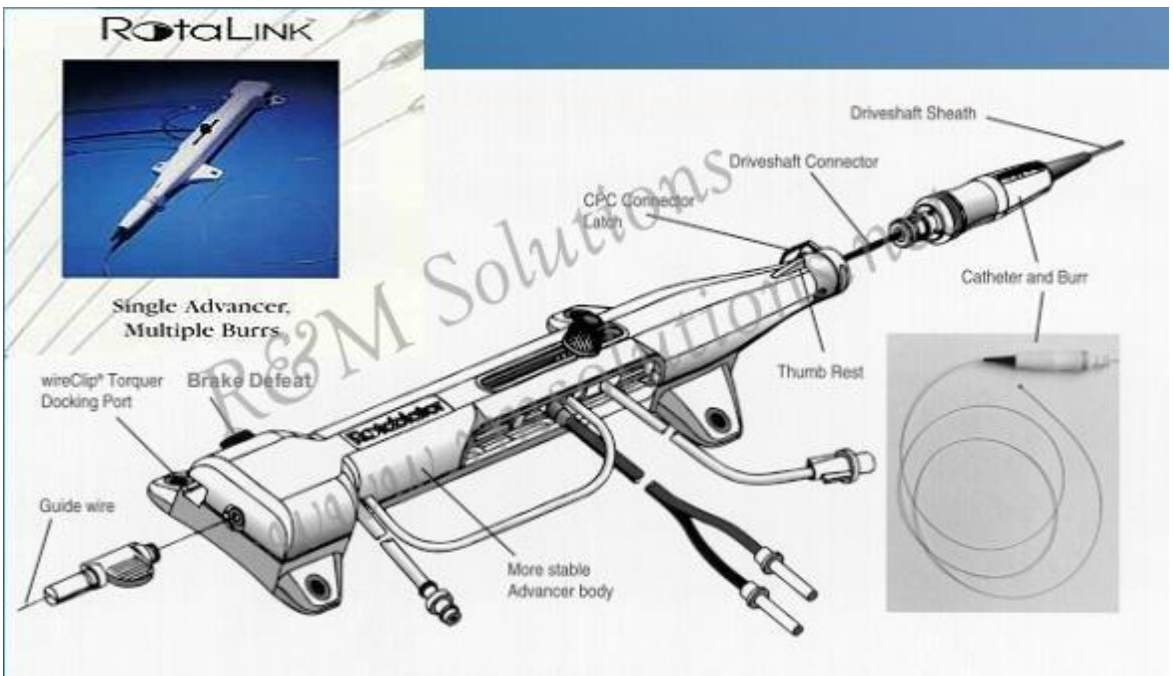
HARDWARE

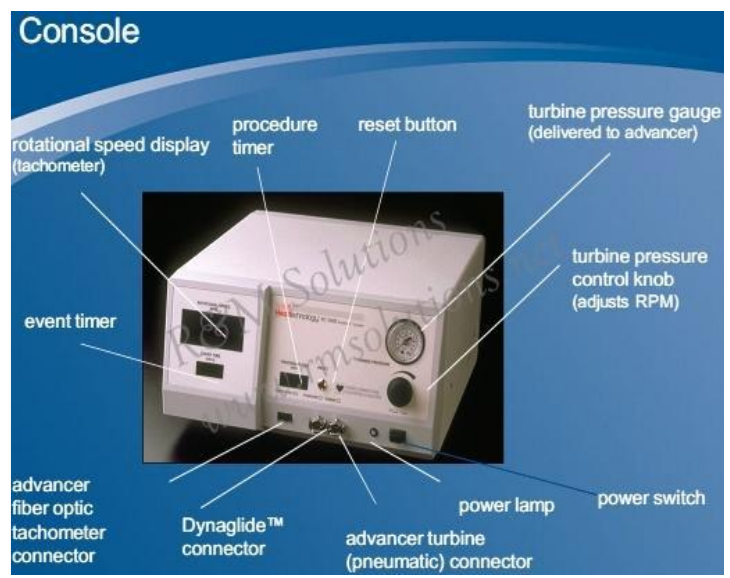
- The distal end of the burr is covered with diamond particulates, ranging from 20-30 microns in size.
- In addition, the tip of the guide wire is made from a radiopaque platinum material.
- The drive shaft is covered in a Teflon casing so that the rotating components will not injure the artery.
- Burr sizes vary in diameter from 1.25mm to 205 mm.

- A motor compresses air transports high pressure nitrogen to the turbine in the advancer body. This turbine derives work out of the high pressure air and uses it as rotational energy to rotate the drive shaft at high speeds.
- The rotational speed of 160,000- 180,000 rpm for 15-45s is recommended.
- Guiding catheters of 6F,7F or 8F are used depending on the burr size.








Console
rotational speed display (tachometer)
procedure timer
reset button
turbine pressure gauge (delivered to advancer)
turbine pressure control knob (adjusts RPM)
event timer
advancer fiber optic tachometer connector
Dynaglide™ connector
advancer turbine (pneumatic) connector
power lamp
power switch

Foot Pedal
Dynaglide button
Dynaglide indicator illuminated on RPM display on console

Air Supply Set Up
Dual Gauge Regulator Byrne
• Monitors gas delivered to console (90 – 110 PSI)
• Monitors gas contained in tank (minimum 500 PSI per case)
H Cylinder Tank
• Compressed Air or Nitrogen
power cord
Dynaglide™ connectors

Disposables
Components
Advancer
Burr catheter
WireClip® Torquer & Guidewires

PHYSIOLOGIC ASSESSMENT OF ANGIOGRAPHICALLY INDETERMINATE CORONARY LESIONS

FRACTIONAL FLOW RESERVE (FFR)

Fractional flow reserve is a measurement of functional severity of a coronary lesion. During coronary angiography, the severity of a lesion is judged by visual inspection and is considered severe or flow limiting, if the vessel diameter narrowing is estimated to be 70% or greater. FFR is measured as the ratio of distal blood flow in a vessel with a coronary lesion to the normal blood flow at the proximal region of the vessel. This measurement is obtained during maximum myocardial hyperemia. FFR contributes to the decision whether a lesion should be treated medically, receive coronary intervention or revascularization.

How does FFR work?

FFR measures the MAP in the coronary artery beyond a lesion. The functional state of the lesion is determined by the heart muscle. A state of myocardial hyperemia is induced to determine if the distal blood flow is no longer sufficient to meet the oxygen demand of the myocytes.

Definition of FFR: $\dfrac{\textbf{Maximum achievable blood flow in stenotic coronary artery}}{\textbf{Maximum blood flow in the same artery without stenosis}}$

- FFR is the ratio of hyperemic myocardial flow in the stenotic territory ($Q_{s\ max}$) to normal hyperemic myocardial flow ($Q_{N\ max}$):

$$FFR = Q_{s\ max} / Q_{N\ max}$$

- Since the flow (Q) is the ratio of the pressure (P) difference across the coronary system divided by its resistance (R), Q can be substituted as following:

$$FFR = [(P_d - P_v)/R_{s\ max}]/[(P_a - P_v)/R_{N\ max}]$$

- Since measurements are obtained under maximal hyperemia, resistances are minimal and therefore equal, and thus they cancel out. In addition P_v is negligible as compare to P_a or P_d, therefore:

$$\textbf{FFR}=P_d/P_a, \quad \textbf{at maxiumm hyperemia}$$

P_d= pressure distal to the lesion.

P_a=pressure proximal to the lesion

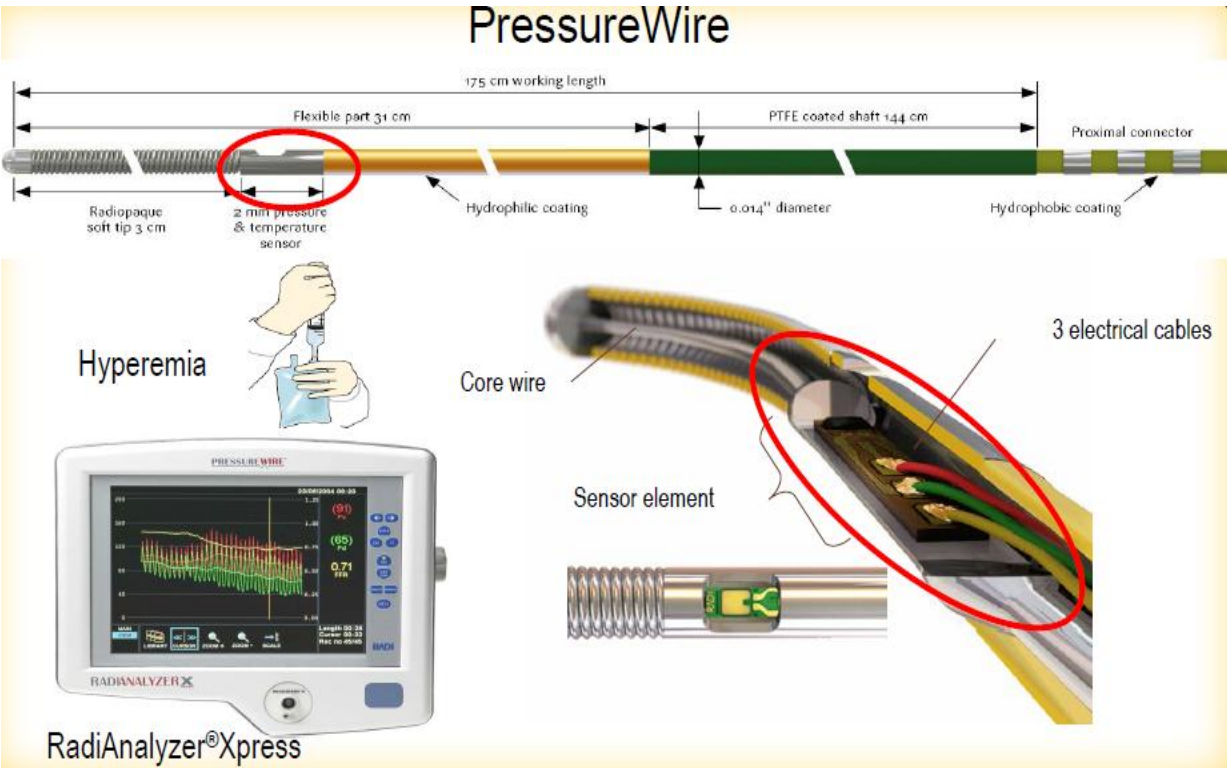
The mean pressure measured by the pressure wire is divided by the mean pressure measured by the catheter.

FFR 1.0	**-**	**normal blood flow**
FFR > 0.75	**-**	**ischemia is very unlikely**
FFR<0.75	**-**	**specific indication for ischemia**

How does FFR performed?

FFR is performed very much like an interventional procedure. The patient is given anticoagulation medication to prevent blood clot formation on the wire while it remains in the coronary. Appropriate size catheters are chosen especially without side holes for optimal hemodynamic monitoring for FFR.

A. The FFR wire, a 0.014" wire with a pressure transducer near the tip, is zeroed and then advanced through the catheter to the ostium of the coronary.



B. Equalize the wire pressure with the catheter pressure.
C. Advance the wire in to the coronary past the lesion where the transducer is positioned beyond the lesion, baseline FFR is calculated.

If the baseline FFR is greater than 0.75 , then a hyperemic agent is administered(Adensoine) and FFR is calculated.

Adenosine administration

Adenosine can be administered IC or IV to induce maximum myocardial hyperemia. Intra coronary injections are usually performed while IV infusions are used with ostial lesions of the Left Main or RCA.

Adenosine induces maximum hyperemia by causing endothelial relaxation of smooth muscle found inside the arterial wall. An artery with a lesion will not allow the increased demand of blood flow to the distal myocardium.

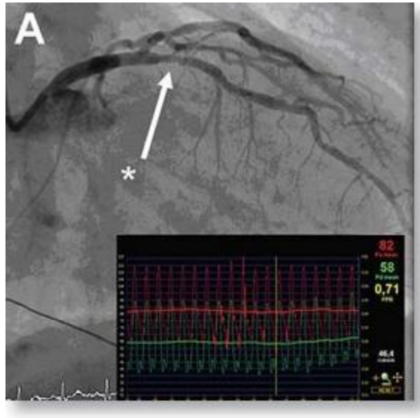
To achieve maximum hyperemia, adenosine is usually given in bolus of 15-30 mcg for RCA and 20-40 mcg for LCA. IV infusions of Adenosine is given in a dose of 140 mcg/kg/min over 3-4min. the IV infusion has been the gold standard of administration in order to achieve maximum hyperemia, but is also associated with more systemic adverse effects.

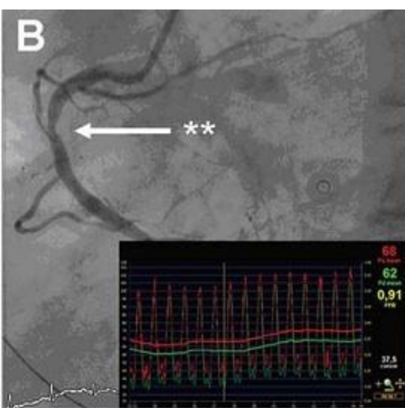
Effects: adenosine is used as a rapid bolus to treat SVT by inducing heart block at the AV node. Careful consideration should be used with IC doses for RCA. Asystole may be noted with higher doses injected into the right coronary with a return to the patients baseline rhythm.FFR values depends on the demands of collaterals if present.

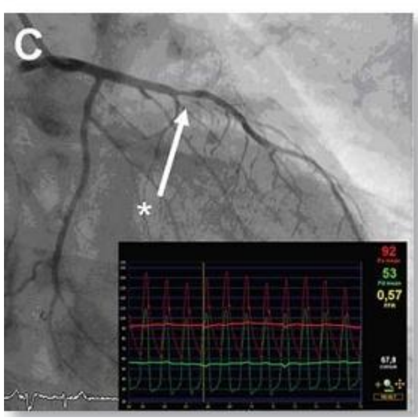
Several caveats should be considered when interpreting FFR values. These can be classified into three main categories:

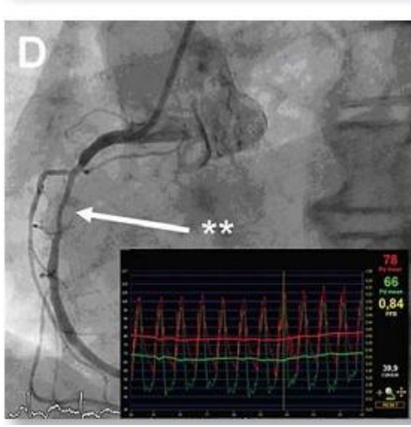
- Technical issues: These may be include the use of guide catheters with side holes or pressure wire drift.
- Suboptimal hyperaemia: This problem commonly arises from intracoronary administration of adenosine. It is not addressed by escalating bolus dose of adenosine.
- Physiological interpretations: with few exceptions, the FFR is an accurate measure of the hemodynamic significance of a stenosis. In the presence of multi-vessel coronary artery disease, an alternative culprit lesion should be considered if an angiographically “significant” stenosis turned out not to be so on FFR measurement.

Alternative explanations could include the presence of collateral, infracted myocardium, and small territory involvement.









(A, B) Two lesions in one patient from the FAME trial, both categorized as 50–70% stenosis by angiography. FFR demonstrated only lesion A was functionally significant (≤ 0.80). (C, D) Two lesions in one patient from the FAME trial, both categorized as 70–90% stenosis by angiography. FFR demonstrated only lesion C was functionally significant and so was stented.

FFR is the best choice for assessing functional significance of lesions and identifying what to treat.

Situations where measured FFR is spuriously elevated (i.e., falsely negative) include the presence of severe left ventricular hypertrophy, during the context of a ST-elevation myocardial infarction (STEMI) and coronary artery spasm as cause of the patient's symptoms. Conversely, the measured FFR may be spuriously low (i.e., falsely positive) when the patient is grossly fluid-overloaded. In these situations, the interventional cardiologist would have to consider the measured FFR value in the context of the physiological states that may affect the FFR value.

INTRAVASCULAR ULTRASOUND (IVUS)

Intravascular ultrasound (IVUS) is a tool with many applications. It identifies and better characterizes plaques which are not visualized by angiography, which essentially a lumenogram.

Principle of IVUS

The IVUS probe is essentially designed as a catheter with a miniaturized ultrasound probe at its distal tip which has a piezoelectric crystal capable of emitting ultrasonic waves in the

20-50 MHz range. The reflected ultrasound waves are interpreted by a computer based algorithm and are used to recreate two dimensional cross-sectional tomographic views of the vessel wall. These images can be used to calculate cross-sectional area, diameters of the vessel wall at various points of the vessel of interest and thus help in assessing the true severity of the atherosclerotic lesion together with a fair idea of its underlying morphology.

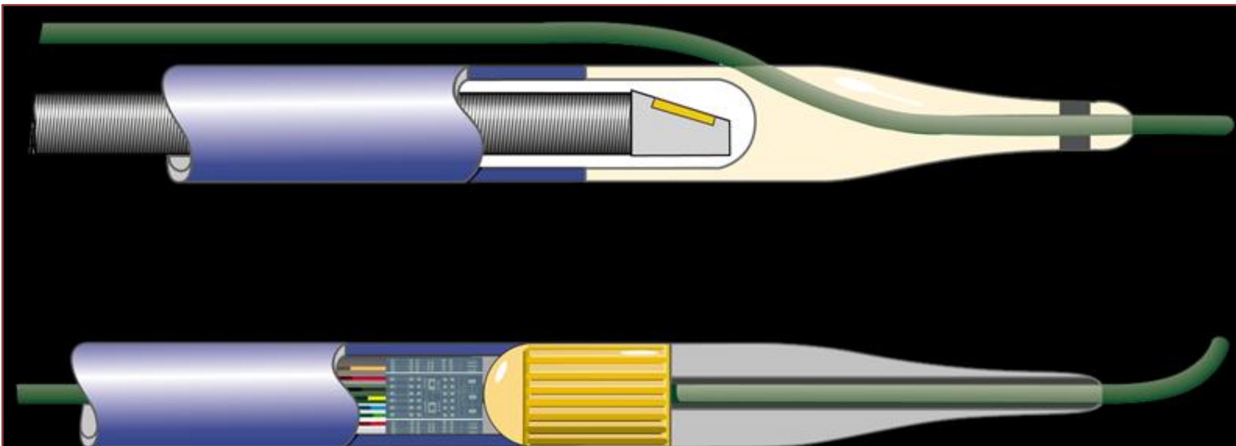
Basic Setup of IVUS

Consider of two basic components:

1. Catheter with miniaturized ultrasound probe
2. Console and electronics for reconstructing and interpreting the image.

The ultrasound probe in use have a frequency between 20 MHz and 50 MHz, which gives them an axial resolution of 150 µm and come in different sizes varying from 2.6 to 3.5 French which can be placed through 6F guide catheter. There two available transducer designs-mechanically rotated devices and phased array/solid state transducers. Piezoelectric transducer at its tip which after electrical excitation generates sound waves which interacts with the vessel wall, and the structures within providing detailed information. **Mechanical rotating element catheters:** single transducers at the tip of the catheter at 1800rpm. **Phased array/solid state catheters :** array of fixed transducers at their tip, which are electronically sequentially excited allowing to generate rotational cross-sectional images.

Mechanical Transducer – 40 MHz Atlantis Pro (Bos Sci)



Solid-State Transducer – 20 MHz Eagle Eye (Volcano)

Indications:

- Evaluation of lesion severity at a location difficult to image by angiography in a patient with a positive functional study and a suspected flow-limiting stenosis
- Assessment of a suboptimal angiographic result after PCI
- Diagnostic and management of coronary disease after cardiac transplantation
- Assessment of the adequacy of deployments of coronary stent, including the stent apposition and determination of the minimal luminal diameter within the stent

• Determination of plaque location and circumferential distribution

• Determination of the mechanism of stent restenosis (inadequate expansion versus neointimal proliferation) and to enable selection of appropriate therapy (plaque ablation versus repeat balloon expansion)

• Pre interventional assessment of lesion characteristics as a means of selecting an optimal revascularization device.

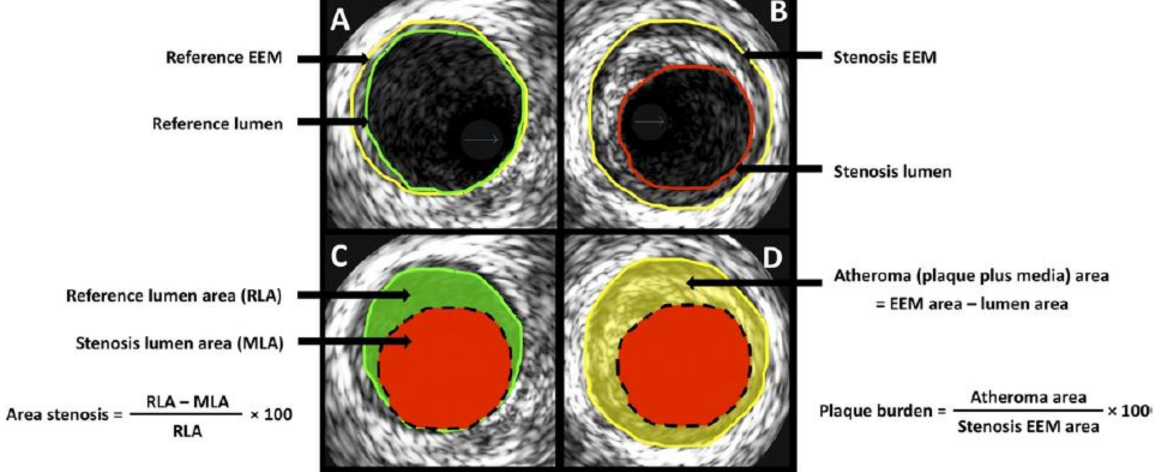
Examination Technique

The ultrasound transducer which has a radiopaque marker is positioned beyond the point of interest and a pullback examination is done. Pullback can be manual or motorized (0.5 mm/sec). The latter has smoother image acquisition. Side branches useful landmarks to facilitate interpretation and comparisons in sequential examinations.

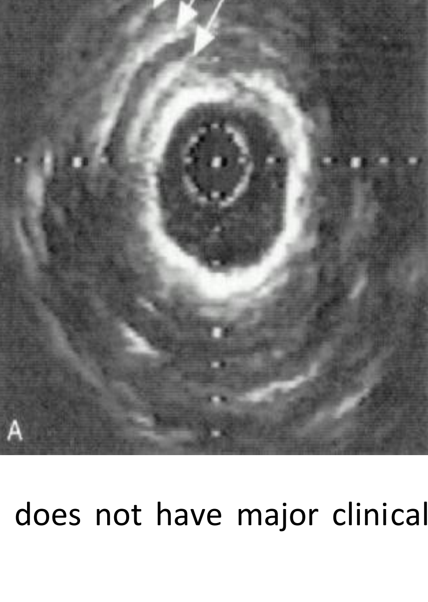
The coronary artery is essentially a trilaminar structure, the normal intima gives a thin bright echo and is the innermost structure nearest to the transducer. The media which comprises muscle cells gives a dark signal just underlying the intima. The adventitia is the outermost structure and has an onion skin appearance. The onset of atheroma formation leads to thickening of the intima.

Plaque characterization

Plaque can be broadly classified based on their appearance as – soft, fibrotic or calcific. The soft plaque has an echogenic signal which is lighter than the adventitia. The fibrous plaque on the other hand throws a brighter signal which is equal or brighter than the adventitia. Majority of the plaque observed in IVUS are fibrotic. The calcified plaque is the brightest of the three and gives reverberating negative shadows. A soft plaque with a dark signal suggesting the presence of a necrotic core, one see this plaque in its ruptured form called the "ruptured plaque". With the use of IVUS, it is easy to calculate the minimal lumen area (MLA) and plaque burden of concentric or eccentric lesions at single/multiple points.



Calcific plaque

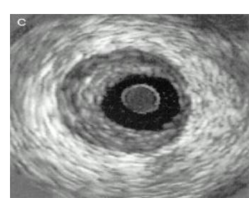


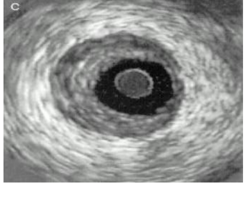
- Calcific plaque is the simplest tissue type to identify
- Bright reflection of intense signal attenuation.
- “ghost arcsor” reverberations.
- Calcification is seen in 60-80% of target lesions using IVUS compared to 30-40% by angiography.
- 180^0 of vascular circumference must be calcified before it can be visualized by angiography.
- Sometimes, shadowing with no bright reflections occurs in calcified lesions.
- IVUS imaging of calcium is angle dependent, and the calcific plaque itself is imaged only when the beam is perpendicular.
- Acoustic shadowing can occur in the absence of calcium in the presence of dense fibrous tissue.
- Therefore it is correct to refer to lesions with shadowing on IVUS as fibrocalcific. This distinction does not have major clinical implication.

Fibrous Plaque

- Plaques with echogenicity that is < bright than calcium, but higher than that from muscle or fat tissue.
- In general, brightness of fibrous tissue is similar to that of adventitia.
- No reverberations.
-

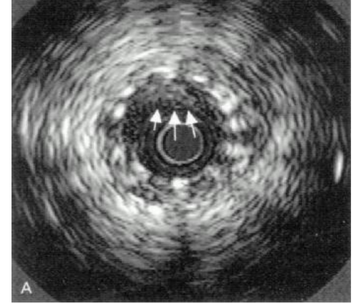
fatty plaque





- Radiolucent, and has a soft grey-scale appearance on IVUS.
- Radiolucent areas within fibrous plaques reflect accumulation of lipid.
- Shadowing from a heavily fibrotic plaque can be mistaken for lipid.

Thrombus



One of the most difficult tissue types to identify by IVUS

- Sparking pattern on real time IVUS imaging.
- Presence of michrochannels, echogenicity< 50% of the surrounding adventitia and deep Ca are clues to the correct diagnosis of thrombus.

Optical Coherence Tomography(OCT)

OCT is an emerging technology capable of generating images with micron scale resolution. It is the optical analogue of pulse- echo IVUS, where electromagnetic waves using a light source as opposed to acoustic(sound) waves are used to create the image.

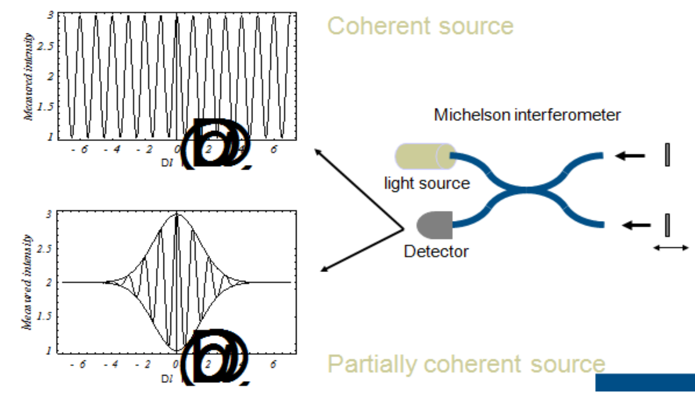
Principle

The light source used OCT imaging is in the Near-infrared range, around 1,300nm wavelength, selected to achieve both penetration is limited to 1 to 3 mm as compared with 4 to 8 mm achieved by IVUS. Cross -sectional images are generated by measuring the echo time delay and intensity of light that is reflected or backscattered from internal structure in tissue. The echo time delay cannot be measured directly is much faster than that of sound

Michelson type interferometer

The interferometer splits the emitted light source into a reference and sample beam; the reference beam is directed to a reference mirror at known distance, the sample beam is directed to the structure of interest. The backward light from the sample is interested with reflected light from the reference arm and their interference fringes are detected by a photodetector.

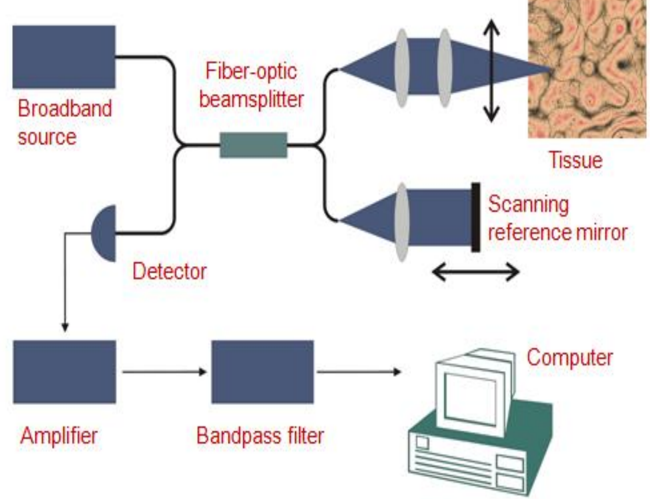
When the back scattered optical intensity of the two arms(interference signal) is measured and compared, the optical properties of the tissue can be deduced. The intensity of the back-reflected light can be measures and quantified digitally in grey scale, enabling the creation of a digital image.



How does it works?

The probe rotates with frequency of 100 rounds/s, which allows acquisition of 50000 axial lines of the signal/s. the contrast may be injected either manually or using an automatic injector. The setting for the automatic injection is as : flow speed=4ml/s, volume=14ml, pressure=400 psi. the presence of blood in the OCT catheter or in the lumen of the vessel disqualifies acquired images from the analysis. The guiding catheter used for OCT imaging should not contain side holes and should provide a stable support. The pullback of the OCT probe starts automatically following the contrast injection. When the pullback is finished, the OCT probe returns to its initial position and the catheter may be

removed from the vessel. Intracoronary OCT is performed by introducing the small (2.7 French) imaging catheter over a guide wire (0.014 inch) distally into the coronary artery using standard guide catheters (6F or larger). A motorized pullback is performed to scan the coronary artery segment The pullback speed is typically 20 mm/sec with a frame rate of 100 frames per second or higher.Since blood scatters the OCT signal, it is temporarily cleared by an injection of x-ray contrast medium during the duration of the OCT pullback (typical flush rate 3.0ml/s).The principle safety considerations relate to the possible induction of ischemia due to the need of blood displacement for image acquisition. Current OCT systems allow for very fast data within a few seconds and therefore are unlikely to lead to significant ischemia.Patients should be anticoagulated during the OCT procedure Intracoronary NTG to minimize the potential for catheter-induced vasospasm.



Hardware

DOC Controls Optis

1 Emergency shutoff – stops rotation, pullback and scanning

2 Enable scanning; second push will start pullback in manual trigger mode

3 Toggle between Live and Standby

4 Unload catheter

The present OCT system that available in India is from St. Jude Medical and it has a console that can be connected to rapid exchange imaging catheter through a Drive Motor & Optical Controller (DOC) for motorized automatic pull through. The console has two monitors with user interface for feeding patient data, reviewing the archive, manual and automated

measurements and for zooming and switching the planes of the port for connecting the sterile single use imaging catheter. The imaging catheter consists of an optical fiber core mounted in a rapid exchange catheter system measuring 135 cm in length and 2.7F maximum cross sectional diameter that is compatible with a guide catheter of 6F or more. The distal end of the catheter has two radio-opaque markers 20 mm apart that facilitate. angiographic measurement of the segment to be imaged target. The optical lens in its forward position is just proximal to the proximal marker up to 50 mm or 75 mm with the present generation catheters. The time for image acquisition is about 3 seconds and only transient interruption of blood flow is required. In the newer catheters an additional radio-opaque marker is placed over the optical fiber for angiographic visualization of the automated pull through which helps to correlate the image in relation to angiography. During pull through the imaging the core rotates rapidly and acquire continuous lines of images (1024 pixels/line). One complete (360 degree) spin of the optical fiber acquires 500 image lines and constitutes one frame. Axial resolution is along the line of acquisition (10 μ), lateral resolution is between the line in the same frame (25 μ) and longitudinal resolution between different frames. As the core is simultaneously pulled back during rotational acquisition a cylinder of vessel is imaged in a spiral manner which can be displayed axially, longitudinally and in 3 dimensions also.

Normal vessel: all the three layers can be seen clearly as a trilaminar appearance. The intima is signal rich and bright, the media is relatively signal poor and homogenously dark whereas the adventitia is heterogeneously signal rich. The internal and external elastic lamina can also be seen in some cases as small bright lines at the borders.

Fibrous plaque: homogenous bright signal rich finely textured area with low attenuation, i.e. deeper structures will also be seen easily through the plaque.

Lipid plaque: lipid core produces a higher attenuation and hence the deeper structures beyond a lipid rich plaque may not be visible and the margins are not well defined.

Calcified plaque: A calcified plaque has low attenuation and low back scatter hence appears as a het erogenous dark area (signal poor) with well-defined margins.

Red thrombus: An intraluminal filling defect with high back scatter from the surface with the darker core and poor visualization of deeper structures due to high attenuation.

White thrombus: irregular intraluminal filling defect with high back scatter and low attenuation, hence deeper structures would be visible.

Thin cap fibro-atheroma (TCFA): similar characteristics of a lipid rich plaque with an intimal cap are less than 65 μ. Accumulation of macrophages can also be seen as signal rich linear areas.

Evaluation of stents: the leading edges of metallic stents have high attenuation and back-scatter hence produces bright short arcs with dark shadows behind due to attenuation. The struts of bio-resorbable scaffolds (BVS) produce rectangular box like appearances with brighter outer border and a darker central area.

Stent apposition, coverage, tissue prolapse, neointimal hyperplasia and neo atherosclerosis: Immediately after deployment of a stent, the stent artifacts can be seen at the luminal surface. If there is a separation of stent struts from the vessel wall that is not across a side branch it qualifies for incomplete stent apposition. Mal apposition can be considered only when the distance from the endoluminal surface of the strut to the vessel wall is higher than the sum of the metal and the polymer thickness together. With the formation of neointima the stent struts gets buried and the artifacts are seen starting deeper from the vessel wall and any amount of neointima is considered coverage and is more than 90% complete by 6 months after any DES. Percentage of neointima is calculated by the fraction of the difference between the stent area and the lumen area to the stent area. If this is more than 50% it is called significant neointimal hyperplasia. Accumulation of lipids within the neointima leads to neo atherosclerosis which can rupture and present as very late stent thrombosis. There can be structures seen protruding through the stent struts on OCT imaging which may be thrombus or tissue prolapsed.

Limitations of OCT

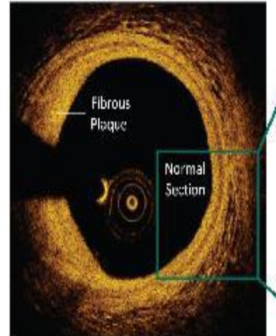
- Penetration
- Inability to measure plaque burden
- Need for a bloodless field and contrast flush.
- Artefacts,
- Absence of large scale clinical trials.
- Ostial lesions

Physical characteristics of OCT vs IVUS.

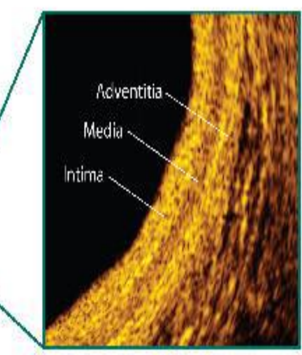
	TD-OCT[a]	FD-OCT[b]	IVUS[c]
Energy source	Near-infrared light	Near-infrared light	Ultrasound (20–45 MHz)
Wave-length	1.3 µm	1.3 µm	35–80 µm
Resolution	15 µm (axial) 40 µm (lateral)	15 µm (axial)	100–200 µm (axial) 200–300 µm (lateral)
Frame rate	15–20 frames/s	100 frames/s	30 frames/s
Pull-back rate	1–3 mm/s	2 cm/s	0.5–1 mm/s
Max. scan diameter	7 mm	10 mm	15 mm
Tissue penetration	1.5–2 mm	2 mm	10 mm

IMAGE GUIDE

Coronary Anatomy by OCT



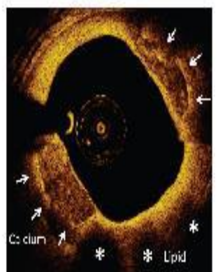
Coronary Artery Anatomy



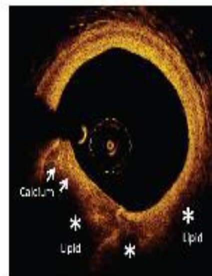
Zoom-in View Normal Section

Coronary Plaque Characterization

Fibrous Plaque

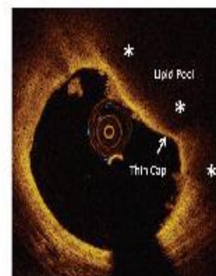


Calcium & Lipid Plaques



Mixed Plaque

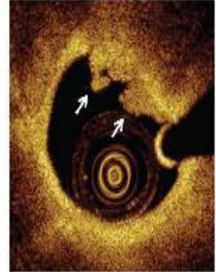
Cholesterol Crystal



Thin Cap Fibroatheroma

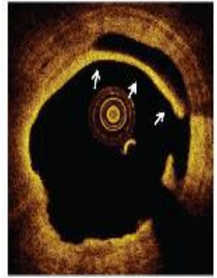
Acute Coronary Syndrome

Red Thrombus

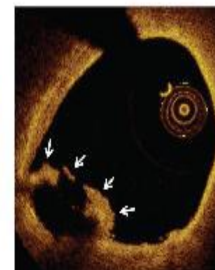


White Thrombus

DES Late Stent Thrombosis



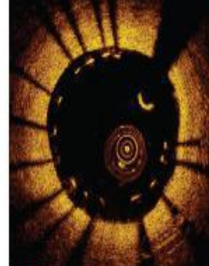
Spontaneous Intimal Dissection



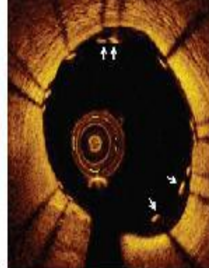
Ruptured Plaque

Stent Optimization

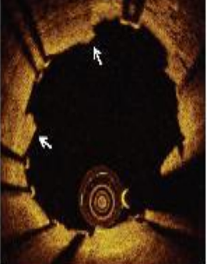
Proper Stent Apposition



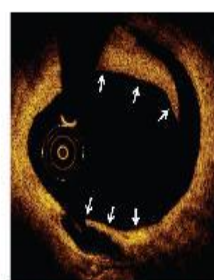
Undersized Stent



Malapposed Stent Struts



Tissue Prolapse

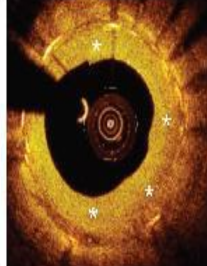


Distal Stent Edge Dissection

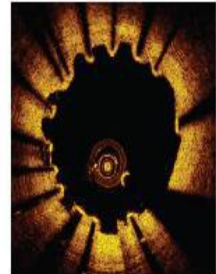
Post-Stenting Follow-Up

Newly Implanted BVS

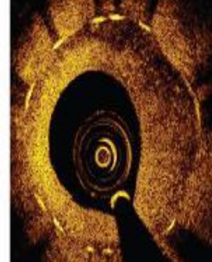
Optimal Strut Coverage



Neointimal Proliferation



DES Non-healing & Positive Remodeling



DES Neo-atherosclerosis

TRANSLUMINAL ALCOHOL SEPTAL ABLATION FOR HYPERTROHIC OBSTRUCTIVE CARDIOMYOPATHY

Criteria for alcohol septal ablation for HOCM include (1) refractory symptoms on maximal medical therapy, (2) a septal thickness of 1.8 cm or more, (3) an outflow tract gradient greater than 40mmHg at rest and greater than 60 mm Hg with provocation, and (4) the gradient localized to the septal obstruction. Other considerations should be moderate or mild mitral regurgitation with no organic abnormalities of the mitral valve and minimal coronary artery disease responsible for the symptoms.

Complete hemodynamic and angiographic study should precede alcohol induced septal ablation. Right and left femoral arteries and veins are cannulated. A 5F pigtail catheter is positioned in the left ventricle, pacemaker is positioned in the right ventricle for prophylactic pacing if complete heart block induced. After the catheters are positioned, coronary angiography identifies the large septal artery originating in the proximal left anterior descending artery. The echocardiographic technician performs imaging of the LV septum and LV outflow tract gradient. A 0.014-inch angioplasty guidewire is used to cannulate the large first septal artery. An over the wire balloon catheter is inflated in the septal artery. The guidewire removed, a small amount of contrast material is injected into the septal balloon to (1) ensure no reflux of contrast material or, later, alcohol and (2) opacify the septal artery and subbranch distribution. Confirmation of correct septal branch occlusion, 1 to 2 ml of 98% denatured alcohol is delivered slowly over 3 minutes into the septal artery followed by a 5-minute observation period. Complete heart block may occur with the need for temporary pacing. After the observation period the balloon catheter is aspirated and then deflated. Suction is kept on the catheter lumen, the catheter is withdrawn from the LAD, and angiography is repeated. Final hemodynamic values are again measured. In most cases, the LV outflow gradient is abolished; 10% to 20% of patients may need permanent ventricular pacing.

PERCUTANEOUS TRANSLUMINAL MITRAL VALVULOPLASTY (PBMV)

Balloon valvotomy is a non-surgical procedure performed in the cardiac catheterization laboratory by a cardiologist and a specialized team of nurses and technicians. Balloon valvotomy is used to increase the opening of a narrowed (stenotic) valve.

BALLOON MITRAL VALVOTOMY (BMV)

Percutaneous balloon mitral valvuloplasty (BMV) was first reported by Inoue and associates' in 1984. Since then, BMV has become an alternative to surgery in the treatment of symptomatic mitral valve stenosis. Although BMV was initially restricted to young adults without severe valvular calcification. It was soon extended to the general adult population, including some elderly patients with calcific mitral stenosis. The mechanism of BMV is

similar to that of surgical commissurotomy, in that both methods relieve valvular stenosis by splitting the fused mitral commissures.
A catheter with a special balloon is passed from the right femoral vein, up the inferior vena cava and into the right atrium. The interatrial septum is punctured and the catheter passed into the left atrium using a "trans-septal technique". The balloon is sub-divided into 3 segments and is dilated in 3 stages. 1st the distal portion (lying in the left ventricle) is inflated and pulled against the valve cusps. Second the proximal portion is dilated, in order to fix the centre segment at the valve orifice. Finally the central section is inflated. This should take no longer than 30 seconds since full inflation obstructs the valve and causes congestion, leading to circulatory arrest and flash pulmonary edema.

Indications:

- In asymptomatic patients, intervention is recommended in moderate to severe MS and pulmonary hypertension (pulmonary artery systolic pressure >50 mmHg at rest or >60 mmHg with exercise).
- When intervention is indicated in patients with rheumatic MS, is preferred to surgery if the valve morphology is favorable and the patient does not have left atrial thrombus or moderate to severe (3+ to 4+) mitral regurgitation. Valve repair is performed if possible and preferred over valve replacement which has higher preoperative mortality and morbidity. Valve repair includes both open commissurotomy and placement of an annuloplasty ring after direct visualization of the valve.
- The decision of whether valvuloplasty is superior to surgery depends on age (<60 favors valvuloplasty), and Cath/ECHO findings (e.g. LVEDP, degree of mobility, thickening and calcification). The average end result for the mitral valve surface area with both strategies is about 2 cm^2.
- Mitral stenosis is amenable to percutaneous mitral valvuloplasty if the echocardiography demonstrates: Thickening confined to valve tips, good mobility of anterior mitral valve leaflet, little chordal involvement, no more than trivial mitral regurgitation, no left atrial thrombus, and no commissural calcification.
- Symptoms of shortness of breath and valve area or less than 1.5 cm2 are indications for commissurotomy.

Contraindications

- Mitral regurgitation +2
- Left atrium thrombus
- Severe calcifications of the mitral valve.

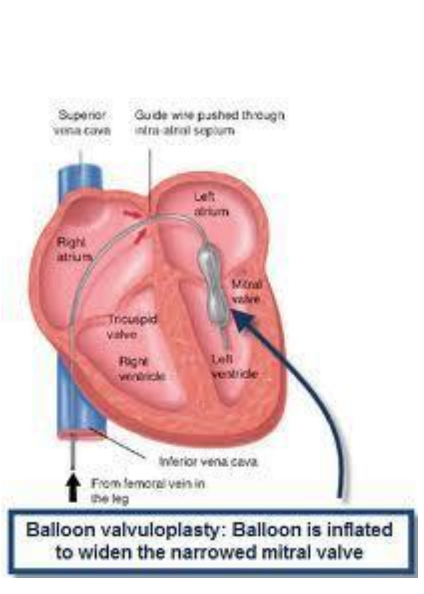
Selection of Appropriate Balloon Size

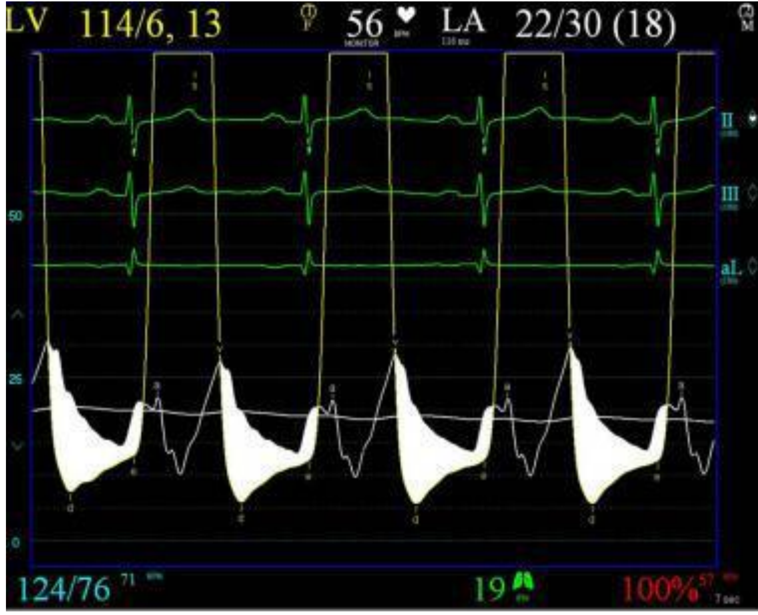
The Inoue Balloon:

The Inoue balloon is constructed of two layers of latex in between there is a nylon mesh.

The Accura Balloon:

The accura double lumen PTMC bloon dilation catheter has 11F, 80 cm shaft and three stages of expandable balloon. In our hospital we are using the accura balloon.
Selection of the appropriate balloon size is one of the most critical factors for accomplishing this procedure (i.e., for releasing the stenotic mitral orifice without causing extensive damage to the commissures, leaflets, and subvalvular apparatus leading to excessive mitral regurgitation). For selecting the appropriate balloon size, some researchers have advocated methods to select balloon size with the patient’s height or body surface area as a reference. **A simple equation to obtain the reference size (height [cm]/10+10) has been proposed.** The obvious point should be made, however, that the relationship of one’s height to the diameter of the mitral valve orifice is not necessarily linear. Furthermore, the operator must remember that annular calcification will affect the size of the mitral valve orifice regardless of the patient’s physical constitution. To avoid undesired extensive injury to the mitral valve apparatus, we select the balloon size by directly measuring the mitral annular diameter using 2-dimensional echocardiography. The mitral annular diameter can be measured on the apical 4- or 2-chamber view during mid- to end-systole. Measuring mitral annular diameter at different planes gives different values owing to the noncircular, nonplanar shape of the annulus. Theoretically, a diameter close to the distance between 2 commissures (probably annular diameter on the apical 2-chamber view) could serve as the reference size for the Inoue balloon. However, the minimal mitral annular diameter should be adopted to avoid the risk of tearing the leaflets.





Selection for percutaneous trans venous mitral commissurotomy

Balloon diameter(mm)	Balloon dilating area(cm^2)	Patient height cm
26 to 30	7.07	>180
24 to 28	6.16	>160
22 to 26	5.13	<160

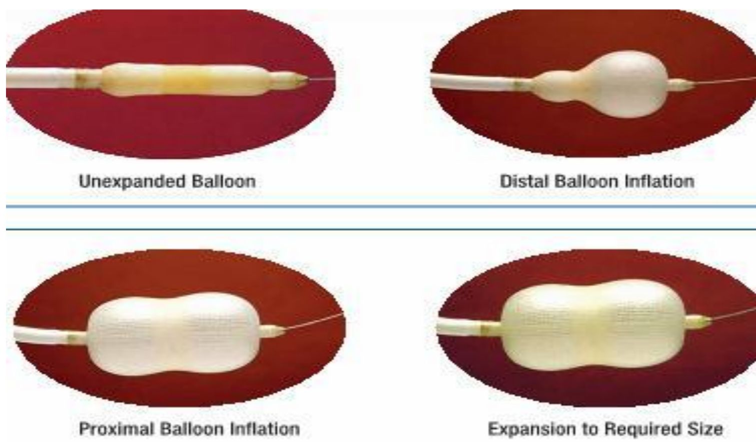
Balloon Preparation

Once the diagnosis of mitral stenosis is confirmed after successful trans septal puncture, the balloon catheter can be prepared. The balloon catheter comes packaged with all the components necessary for the dilatation procedure. These include:

- A balloon-stretching metal tube
- A calibrated inflation syringe specifically matched to each balloon
- A rigid 12F to 14F plastic dilator
- A 0.025-inch spring-tipped exchange guidewire
- A stylet for manipulating the balloon across the mitral valve after it has been placed in the left atrium
- The Inoue balloon catheter
- Calipers for measuring the balloon diameter and confirming its inflated size

The balloon catheter lumen is flushed with saline. Dilute contrast (saline:contrast 2:1 or 3:1) is injected through the vent lumen to purge air from the inflate/deflate channel to the balloon and the stopcock is closed on that lumen. The precalibrated balloon inflation syringe is filled to the calibration corresponding to the smallest inflated 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 5 seconds so that the nylon mesh may be slowly stretched without risking mesh 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 the precalibrated inflation syringe. If the balloon does not inflate to the desired diameter, small amounts of contrast are added or subtracted to achieve proper calibration.

The syringe is then filled to the calibration corresponding to the maximum nominal inflated size. The balloon may be tested to ensure that the maximum size calibration is also correct. In practice, this calibration step is often omitted. The next step in balloon preparation is to elongate the balloon catheter along its long axis, causing it to become

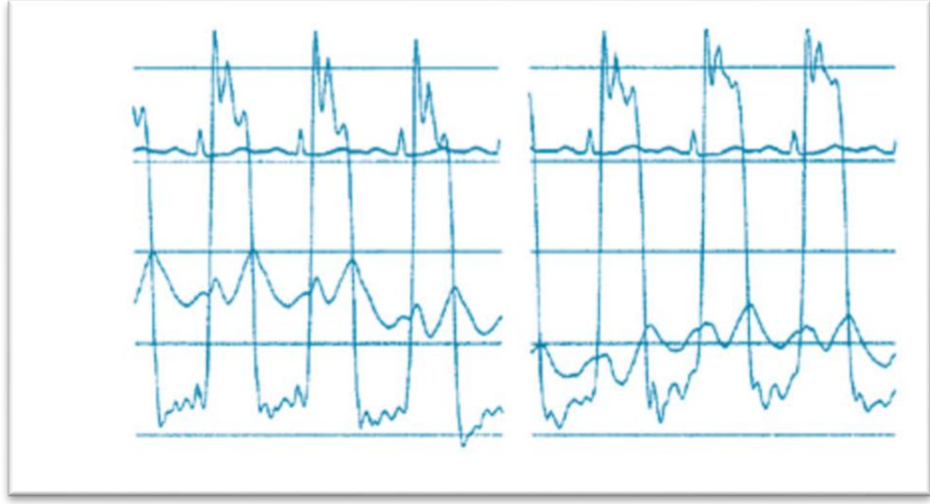


more slender. A metal tube (balloon-stretching tube) is inserted into the center lumen of the balloon over the guidewire and advanced until it locks into the metal hub at the proximal end of the balloon catheter. The balloon and stretching tube are then advanced into the balloon catheter shaft until they engage the plastic slot on the balloon catheter Luer lock. This leaves the balloon in its elongated, slenderized form to ease not only percutaneous insertion but also delivery across the interatrial septum.

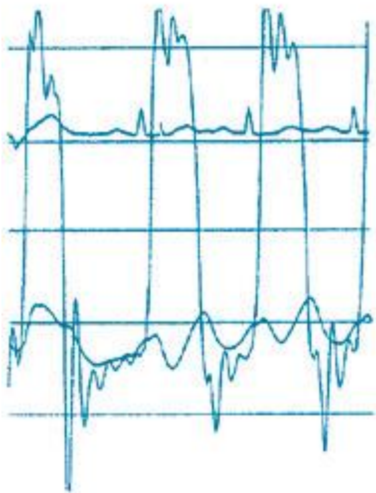
Stepwise Balloon Inflation

If a transmitral gradient persists and no significant increase in mitral regurgitation has occurred, another balloon inflation is performed at aninflated diameter 1 mm greater than the preceding inflation. This sequence is repeated until either an increase in mitral regurgitation or a sufficient decrease in the transmitral gradient occurs.Balloons can be overinflated by 1 to 2 mm diameter by using an additional 1 to 2 mL inflation volume above the maximal calibrated balloon size. If sufficient reduction in gradient is not achieved after maximal or supermaximal inflation, a larger balloon size can be used.

prevalvulotomy



Post balloon dilatation



HARDWARES USED DURING BMV

Trans-septal puncture is an essential step in percutaneous mitral valvotomy (PMV) through the antegrade route. This technique was developed by Ross, Braunwald and Marrow at the National Heart Institute. To perform a septal puncture use a mullins sheath or dilator (7 F), brokenbrough needle, three way stop cock with saline and contrast.

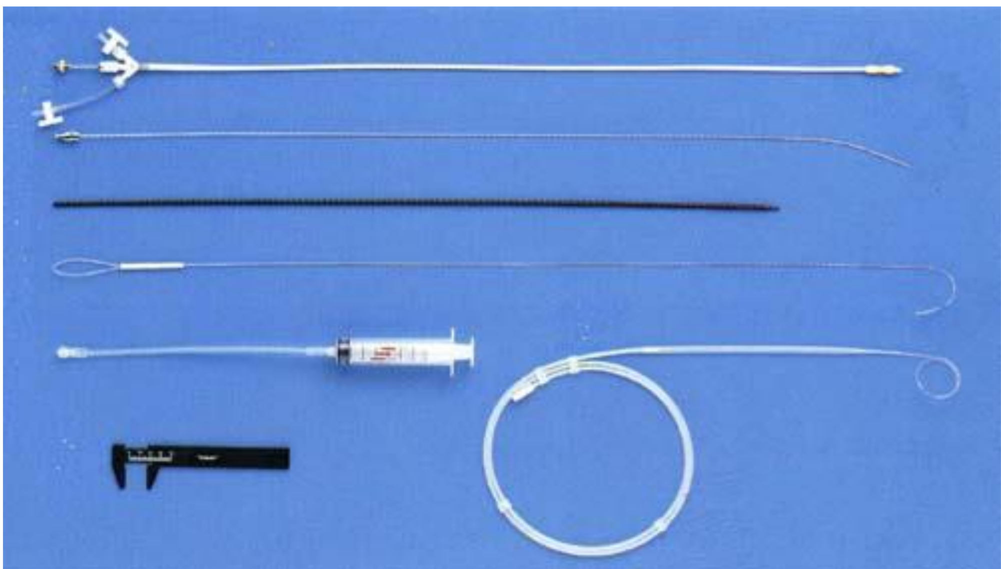
Mullins sheath and Dilator:

The cathether has a 270^0 curve at the end that is straightened by the dilator.

Desription	Size	
	Outer Diameter	Length
Balloon stretching tube	1.2mm	80cm
Dilator	14Fr.	70cm
Guidewire	.025"	175cm
Stylet	.038"	80cm
Ruler	—	—

Brockenbrough Needle:

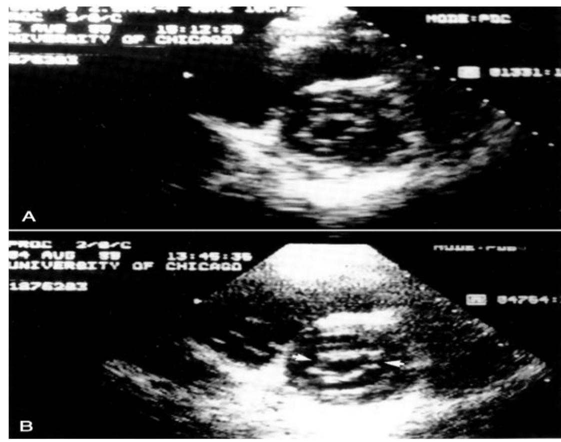
This needle is an 18-gauge hollow tube that tapers distally to 22-gauge. The proximal end has a flange with an arrow that points to the position of the needle tip. The interventional cardiologist gains access to the mitral valve by making a puncture in the interatrial septum during cardiac catheterization. Inflation and rapid deflation of a single balloon or a double-balloon opens the stenotic valve. This mechanism is similar to that of surgical commissurotomy.



PROCEDURE

- Trans venous trans septal technique is most commonly used with the Accura balloon system.
- Fossa ovalis lies usually at 1-7 o'clock but this orientation can be distorted in the presence of mitral stenosis where the interatrial septum becomes more flat, horizontal and lower.
- For the femoral vein approach a 70 cm Brockenbrough needle should be used or an 8 Fr Mullins sheath and advanced under fluoroscopic guidance with pressure monitoring.
- The latter is necessary to monitor for puncture into adjacent structures such as aorta.
- Further catheter manipulation may be necessary to direct the catheter into the left ventricle through the mitral valve rather than towards one of the pulmonary veins.
- The Mullins sheath is exchanged for a solid-core coiled 0.025 inch guidewire over which a 14 Fr dilator is placed.
- This is exchanged for the Accura balloon (24-30 mm) which inflates in three stages allowing for balloon self-positioning with the last inflation resulting in commissural splitting.

A transthoracic echocardiography should be done to measure the mitral valve area and assess the severity of regurgitation as a complication of the procedure. PMBV should be stopped if adequate valve area has been achieved or if the severity of mitral regurgitation has been increased.



Short-axis echocardiogram illustrating commissural splitting following balloon dilatation. A, Fishmouth orifice of the mitral valve. B, Bilateral commissural splitting, indicated by solid white arrows.

Balloon Aortic Valvuloplasty

Balloon aortic valvuloplasty (BAV) showed great promise as an alternative to surgical aortic valve replacement when the procedure was initially described in the early 1980s. Balloon dilatation of the aortic valve results in an immediate increase in aortic valve area with the expected fall in the transvalvular pressure gradient and a rise in cardiac output. Most patients have immediate clinical improvement and this is accomplished with a percutaneous procedure resulting in substantially less morbidity than valve replacement surgery. Unfortunately it was quickly discovered that the durability of these results is short-lived. Disappointment with the clinical results of this procedure over a 1- to 2-year follow-up resulted in a pendulum-like movement away from the performance of BAV. With the introduction of transaortic valve implantation methods, the use of BAV is now an integral procedural step.

Indications

There are currently six clinical situations in which BAV is us.

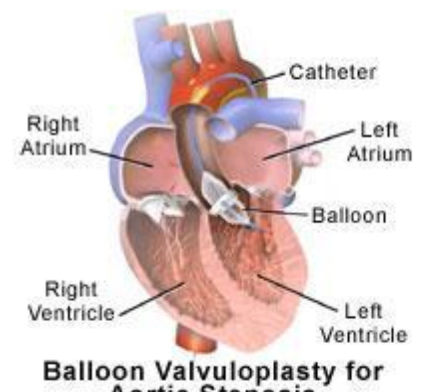
1. Cardiogenic shock. Patients who present with aortic stenosis and Cardiogenic shock may be stabilized for the short term.
2. Congestive heart failure preceding aortic valve replacement. Among patients with severe left ventricular dysfunction or shock in whom aortic valve replacement is planned, balloon dilatation may be performed to allow improvement in left ventricular performance before surgery. Prerenal azotemia associated with their medical therapy may improve after aortic valve prolapse.
3. Preparation for major noncardiac surgery. Patients found to have aortic stenosis during the evaluation for major noncardiac surgery may undergo valvuloplasty. This is especially useful for patients with malignancies.

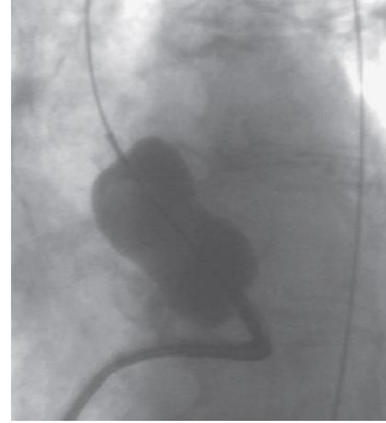
4. Preparation for hospice transfer. Hospital-bound patients with severe aortic stenosis who are not candidates for valve replacement surgery may undergo balloon dilatation with successful short-term improvement. This is useful for patients who are dependent on intravenous pressors and in an intensive care unit. Although valvuloplasty does not improve their long-term prognosis, it may allow them to be transferred to a regular floor or discharged from the hospital so that they may have a better quality of life, at least in the short term.
5. Diagnostic test in low gradient, low output aortic stenosis. There is a group of patients in whom balloon valvuloplasty may be performed as a diagnostic test. This is useful when the valve area is between 0.8 and 1.0 cm2 with low cardiac output and a low transvalvular pressure gradient. In this group of patients, the severity of valvular stenosis is especially difficult to ascertain. Poor ventricular function has made therapy in this group difficult. In the past, valve replacement could be performed and, if the patient had improvement in left ventricular function, then survival was good. Unfortunately, for those patients who did not show improvement in left ventricular performance, perioperative mortality was very high. Balloon dilatation may be performed and serial echocardiography used to monitor changes in left ventricular function. If symptoms and left ventricular performance improve with opening of the aortic valve using valvuloplasty, later valve replacement surgery can be undertaken with a high expectation of long-term success.

PROCEDURE

Diagnostic right and left heart catheterization was performed on all patients. Heparin was administrated in all patients 10 to 70 U/kg after 9- to 13-F sheath insertion in the femoral artery. BAV was performed according to standard techniques via the retrograde femoral approach in all patients except 1. In that individual, axillary artery access was employed because of severe peripheral vascular disease. Equalization of pressures was documented before entering the left ventricle. The gradient was confirmed with pullback gradient measurements. Peak and mean gradients were measured, and AVA was calculated with the Gorlin formula. The sinotubular junction is the site prone to rupture during a BAV procedure. We analyzed the minimal diameter of the sinotubular junction by aortogram in the left anterior oblique with a marker pigtail and by echocardiography. The chosen balloon size was 3 to 5 mm under this measurement. To stabilize the balloon position across the valve, the heart was paced at a high rate (180 to 200 beats/min) until the blood pressure fell to <50 mm Hg before inflation. Pacing was continued until the balloon was fully deflated. Additional BAV procedures were performed subsequently in cases in which the mean gradients did not decrease significantly (∼30% to 40%), and a larger balloon was employed in selected cases in which the initial balloon size failed to significantly decrease gradients (∼30% to 40%). At the end of the procedure, measurements of cardiac output and pressure gradients and calculations were repeated. After BAV, an aortogram was

performed to assess aortic regurgitation. Arterial puncture sites were closed with closure devices (6-F
Perclose or 12-F Prostar [Abbott] or 8-F Angio-Seal [St. Jude Medical, St. Paul, Minnesota]). If the device failed, manual compression was applied. Serious adverse events were defined as intraprocedural death, stroke, coronary occlusion or dissection, moderate-to-severe aortic regurgitation, profound hypotension requiring resuscitation and intubation or cardioversion, tamponade, permanent pacemaker requirement, and vascular complication requiring intervention.





Complications

The major complications of aortic balloon valvuloplasty are ventricular perforation from the balloon or guidewires used in the left ventricle, and femoral artery complications related to the large sheath size that is necessary for the retrograde technique. Cardiac tamponade from catheter perforation has been reported in about 1% of cases. Vascular surgery for femoral arterial complications is required in as many as 5% of patients. This has been dramatically reduced in our recent experience using suture closure in association with retrograde aortic valvuloplasty or with the antegrade approach. We have also been able to “preclose” 14F venous punctures with suture closure with good success. Since the balloon catheter abrades the ventricular septum during balloon inflations, bundle branch block may occur and requires pacing in some cases. Rarely, permanent pacemaker implantation is necessary. It is critical to place a temporary pacemaker prior to balloon dilatation in patients who have bundle branch block or high grades of heart block preprocedurally. Severe aortic regurgitation.

BALLOON PULMONARY VALVULOPLASTY

BPV is a safe, effective and reliable treatment for patients with congenital valvular PS. The pulmonary balloon valvuloplasty is the treatment of choice for patients with symptomatic pulmonary stenosis.

INDICATIONS

- Patients with exertional dyspnea, angina, syncope, or presyncope.
- Asymptomatic patients with normal cardiac output (estimated clinically or determined by catheterization) and transvalvular peak systolic pressure gradient more than 30 mmHg.

- Tetrology of fallot

TECNIQUE OF PULMONARY BALLOON VALVULOPLASTY

Vascular access via femoral vein, right ventricular (RV) angiography was done with a Berman balloon or NIH catheter initially. Hemodynamic data including RV pressure and pulmonary artery (PA) pressure were documented during catheterization with Swan-Ganz catheter. BPV was performed with a long J tipped exchange guide wire (260 cm) was used to advance the balloon to the pulmonary valve site. Single balloon technique was performed via femoral vein, with the balloon sized about 25% greater the annulus diameter. Usually, repeated balloon dilatation 2-3 times was performed and each inflation-deflation time was no more than 30 seconds.

RISKS

- Damage of tricuspid
- Perforation of right ventricular outflow tract
- Embolization

DEVICE CLOSURE FOR ATRIAL SEPTAL DEFECT

An atrial septal defect (ASD) is a communication or opening between the atria that results in shunting of blood between the two chambers. There are 4 anatomic types:

- Ostium primum - low in atrial septum, may involve a cleft mitral valve.
- Ostium secundum - center of the atrial septum. Most common type of ASD.
- Sinus venosus - high in the atrial septum. Associated with P-TAPVR.
- Coronary sinus - large opening between the coronary sinus and left

SYMPTOMS OF ASD

Severity of symptoms often depends on the size of the hole. Large ASDs may cause fatigue, shortness of breath, pulmonary hypertension, arrhythmia and/or an enlarged heart.

PROCEDURE

This procedure is performed in the cardiac catheterization lab. The patient will be given an anesthetic which may be general (GA) or local (LA) depending on the technique used. Once anaesthetized, an imaging probe (TEE) will be passed into your gullet (oesophagus) for accurate sizing of the ASD and to assist during deployment of the device. A multipurpose was used to perform hemodynamic measurement will be inserted via a vein in the groin and navigated until it reaches the heart. Sometimes, the catheter is positioned at different chambers of your heart to measure the pressure and oxygen content prior to device closure. In certain circumstances, balloon sizing of the ASD may be required. The appropriate size device is connected onto a cable, put into a special delivery tube, advanced through your ASD and carefully deployed. Your doctor will study the device's position and stability before releasing the device. The catheter and imaging probe are removed and the procedure is completed.

COMPLICATIONS

- Air embolism, LA disk deformation, Arrhythmia, AV block, Device embolization, Cardiac perforation.

TRANS CATHETER VALVE THRAPIES

TRANSCATHETER AORTIC VALVE REPALCEMENT(TAVR)

Age-related calcific aortic valve degeneration is the most common cause of aortic stenosis (AS) in adults and its prevalence steadily increases with age. Survival in patients with AS dramatically decreases with onset of symptoms (angina, syncope, congestive heart failure) with an average of 1 to 3 years and the poorest survival seen in patients with failing left ventricles. Aortic valve replacement is a proven treatment to prolong survival in patients with severe AS.

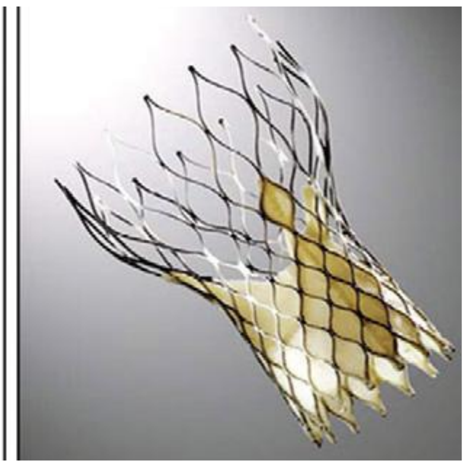
Indications

Aortic valve replacement (AVR) is indicated in all symptomatic patients with severe AS (2006 ACC/AHA Valvular Heart Disease Guidelines, Class I recommendation, Level of Evidence: B). Despite this recommendation, a significant number of elderly patients (up to one third) do not undergo AVR because of comorbidities, including advanced age and the associated increased morbidity and mortality with surgery.

- The patient has senile degenerative aortic valve stenosis with echocardiographically derived criteria: mean gradient >40 mm Hg, jet velocity >4.0 m/sec, or an initial aortic valve area of <0.8 cm2
- Aortic annular diameter 18–24 mm
- NYHA functional class II or greater symptoms
- Patients must have comorbidities such that he or she has a minimum STS score of >10.(society of thoracic surgoens-STS).

Transcatheter Valves

There are two catheter-implantable valves commercially available overseas. They are the balloon-expandable Edwards SAPIEN valve and the self-expanding Medtronic CoreValve . The Edwards SAPIEN valve was studied in the PARTNER trial, which completed randomized enrollment in fall 2009. The Medtronic CoreValve randomized trial started enrollment in December 2010. The initial international experience and the results of PARTNER cohort A and B suggest that outcomes compare favorably with conventional valve surgery in selected patients. In November 2011, the Edwards SAPIEN valve was FDA approved and became commercially available in the United States for PARTNER cohort B (inoperable) patients.



Left: Edwards SAPIEN transcatheter heart valve, which consists of a stainless steel stent with bovine pericardial tissue leaflets sewn onto the balloon-expandable stent. Right:Medtronic CoreValve transcatheter aortic valve system, which consists of a nitinol-based frame with porcine pericardial tissue leaflets affixed to the self- expanding stent..

Patient Selection

A thorough clinical evaluation of the patient is performed with special attention to vascular access and potential sources of complications. The evaluation includes history and physical examination, laboratories, chest radiograph, electrocardiogram, transthoracic echocardiography, pulmonary function testing, carotid ultrasound, right and left cardiac catheterization, coronary angiography, and computed tomography angiography (CTA) of the chest, abdomen, and pelvis. The CTA is performed to assess aortoiliac patency, dimensions, and tortuosity. Occlusive peripheral arterial disease, small vessels (<7 mm), and excessive tortuosity are characteristics that would preclude a transfemoral approach since the delivery systems for the SAPIEN valve are currently 22F and 24F sheaths for the 23-mm and the 26-mm valves, respectively. The second-generation Edwards valve, SAPIEN XT, is comprised of a cobalt chromium frame and allows for an 18F delivery system. Patients with unfavorable aortoiliac characteristics can have TAVR via a transapical approach with the Edwards valve. The advantage of the CoreValve system is the smaller 18F profile of its delivery system, but CoreValve can be delivered only in a retrograde fashion. If femoral access is not available, CoreValve may be delivered from subclavian and carotid approaches.

Hardware required

- Temporary pacemaker set
- Peri cardiocentasis tray.
- 35mm*120cm single loop snare.
- 6 Fr coronary guide catheter.
- 14 Fr and 16 Fr introducer sheath.

Preparation of the Catheter

- Attach a 10ml syringe filled with saline to the stopcock on the first flush port and flush. Use the micro knob and macro slider on the handle to open and close the catheter (figure 1).

- Attach a 10ml syringe filled with saline to the third flush port on the handle on the catheter and flush (figure 2).

- Fill a loading bath with cold, sterile saline (0^0C to 8^0C) and place the CLS components in the bath.

Bioprosthetsis Rinsing procedure

- Fill each of 3 rinsing bowls with approximately 500ml of saline at temperature between 15^0C to 25^0 C.
- Immerse the entire bioprosthesis in a sterile rinsing bowl.
- Gently agitate the bioprosthesis by hand for 2minutes to remove the glutaraldehyde from the bioprosthesis. Repeat the above steps to ensure complete removal of glutaraldehyde.

Bioprosthesis Loading Procedure

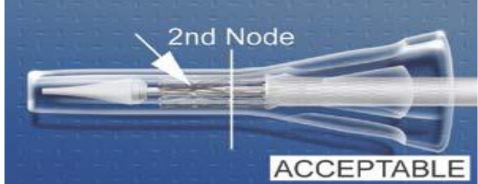
- Advance the outflow tube over the catheter shaft toward the handle.(figure3)

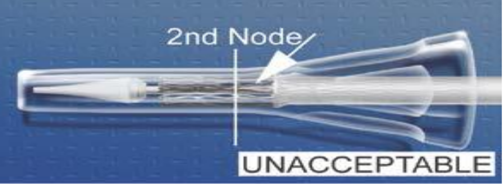
- Gently squeeze the outflow part of the cold bioprosthesis frame and insert it into the outflow cone. After fully inserted,secure the outflow cap.

- Advance the inflow tube with distal catheter tip into the outflow cap.
- Carefully withdraw the inflow tube and attach exposed frame loop to the catheter tabs.
- Advance the radiopaque marker band of capsule prior to advancing the capsule.

- Advance the inflow cone away from the bioprosthesis over the outflow tube. Ensure that the bioprosthesis frame axis is coaxial with inflow cone axis.

- If a crease or infold greater than 2nd node long, do not use the bioprosthesis.





- Slowly advance the capsule over the bioprosthesis until the capsule contacts the catheter tip.
- If the micro knob has fully advanced the capsule and a small gap remains between the end of the capsule and the catheter tip, stabilize the handle with one hand; position the other hand on the blue catheter shaft and gently advance the capsule manually to close the gap between the capsule and the catheter tip. Remove the outflow cone and the tube from the catheter.

Access: Bilateral femoral artery access and one femoral vein for temporary pacemaker implantation.

PROCEDURE

The procedure is typically performed in a cardiac catheterization laboratory or hybrid operating room suite. The implant procedure involves the cooperation of a multidisciplinary team, including cardiac surgery, interventional cardiology, echocardiography, and anesthesia. A surgical team and cardiopulmonary bypass machine are on stand-by in case bailout surgery is required. Preprocedure planning includes identificatssion of the implant side from the CTA scan and measurement of the aortic annulus to choose valve size, which will determine the size of the delivery system. Transesophageal echocardiogram (TEE) is used to size the annulus and to assist in valve positioning. Placement of the probe can be done once the patient is adequately sedated. A 23-mm valve is used for annular dimensions of 18 to 21 mm and a 26-mm valve is used for annular dimensions of 22 to 24 mm. Femoral arterial and venous accesses are obtained on the non- implant side, and 6F sheaths are placed. These will be used for the temporary pacemaker (rapid pacing) and pigtail catheter (aortography), but they also provide rapid access in case the patient needs to be placed on emergency cardiopulmonary bypass.

Pulmonary artery catheterization is done via jugular vein access. Due to the large delivery systems, surgical femoral artery cutdown and repair are best performed on the implant side. After cutdown, an 8F sheath is placed in the artery and a 6F sheath in the vein. Successful arterial closure with a Prostar XL closure device or two Perclose closure devices has been performed, making the procedure truly percutaneous. Weight-based heparin is administered intravenously once access has been obtained. Target activated clotting time (ACT) is 250 to 300 seconds. A temporary pacemaker wire (used for rapid ventricular pacing during balloon inflations) is positioned in the right ventricle through the venous sheath on the non-implant side. A pigtail catheter is advanced to the ascending aorta and supravalvular aortography is performed in the left anterior oblique cranial and right anterior oblique caudal projections. The projection that best lays out all three aortic valve leaflets in a single plane is chosen for valve positioning.

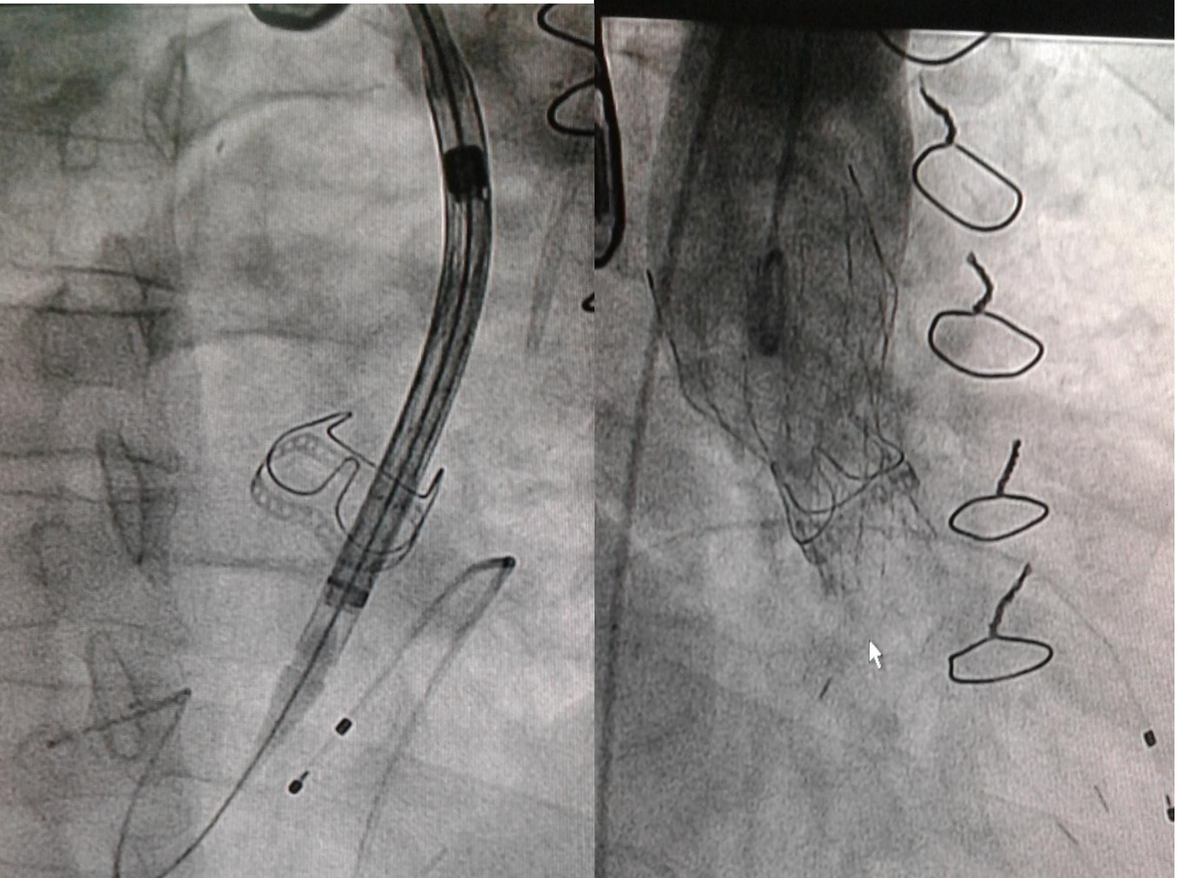
The implant-side arterial access must be serially dilated with hydrophilic-coated dilators over a stiff 0.038-inch guidewire. From 8F, the artery is serially dilated with 10F to 24F dilators. The delivery sheaths for the 23-mm and 26-mm valves are 22F and 24F, respectively. The delivery sheath is advanced over the wire and positioned in the abdominal aorta.

The stenotic aortic valve is crossed using standard technique. After crossing the valve with the straight wire, the catheter is advanced into the left ventricle (LV) and the straight wire is exchanged for an Amplatz super stiff (1-cm tip) guidewire with a pigtail curve. A double-lumen pigtail catheter is advanced over this wire and positioned in the LV. Baseline LV-aortic gradient is measured. The stiff wire is then advanced into the left ventricle, and the pigtail catheter is exchanged for the valvuloplasty balloon. The balloon (typically 20 × 40 mm) is positioned across the aortic valve, rapid ventricular pacing at 180 bpm is initiated, and the balloon is inflated once the systolic blood pressure has fallen below 50 mm Hg (5-sec inflation). The balloon catheter is removed with the stiff wire remaining in the LV.

Next, the RetroFlex 3 delivery prosthetic valve system with the crimped SAPIEN valve is advanced through the delivery sheath over the stiff wire. The delivery system has a flexing mechanism that assists in steering through the aortic arch and minimizes trauma on the outer curvature of the arch. When crossing the native aortic valve,

the delivery system is fully retroflexed, which gives a central and coaxial orientation of the prosthesis to the native valve. The delivery catheter also has a nose cone, which facilitates crossing of the native valve. After the valve has been crossed, the retroflex catheter is retracted to fully expose the delivery balloon. The valve is positioned using fluoroscopy, aortography, and TEE. Approximately 60% of the valve assembly should be on the ventricular side of the aligned sinuses. Once positioning is confirmed, rapid pacing is performed and the delivery balloon is inflated when the blood pressure falls below 50 mm Hg. The balloon is inflated for 5 seconds, then deflated.

After stent valve deployment, aortography is repeated to assess for paravalvular leak, and if significant, repeat balloon inflations may be necessary. The double-lumen pigtail catheter is again placed and final LV-aortic gradients are measured.



Complications From TAVR

- Arterial dissection/ perforation
- Myocardial ischemia
- Coronary obstruction by the valved stent
- Cardiogenic shock
- Stroke
- Bradyarrhythmia
- Paravalvular leak.

TRANSCATHETER PULMONARY VALVE REPLACEMENT(TPVR)

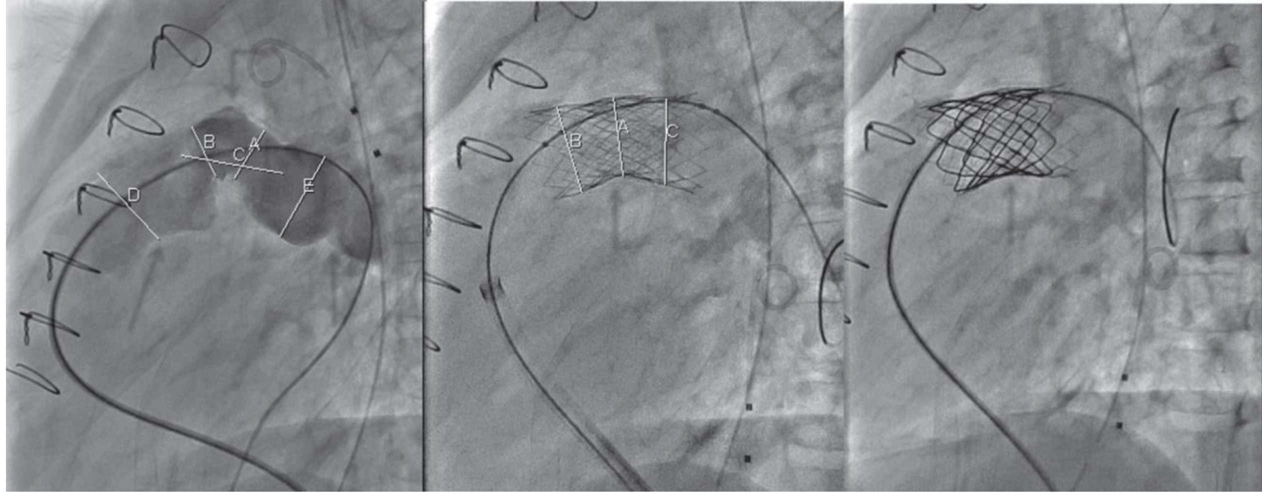
Indications

- Tetralogy of Fallot who have had prior surgery but now have a regurgitation or stenosis pulmonary valve, especially if they have undergone a prior surgical replacement.
- Patients with other underlying diagnoses who have a surgically implanted pulmonary valve(such as a homograft or a bioprosthetic valve) that is no longer working well; including patients with truncus arteriosus, patients aortic valve disease who have had a Ross procedure, and some patients with pulmonary stenosis or atresia.

Procedure

Access is usually obtained via the femoral vein; however, the procedure may also performed via the internal jugular vein. A 7F and a 5F sheath are used for venous and arterial access, respectively. After obtaining access, the patient is given intravenous heparin, to achieve an activated clotting time(ACT) of >200s, preferably > 250s.

Standard right heart catheterization is performed to assess the preprocedural saturations, pressures and the pressure gradient across the dysfunctional conduit. Angiography is then performed as follows: straight lateral and frontal with 20^0 cranial. Selective coronary angiography is performed simultaneously with a noncompliant angioplasty balloon in the RVOT conduit. Adequate assessment of the RVOT size, anatomy, and RV function is of paramount importance to unsure appropriate valve delivery is feasible.



The bare metal stent is usually deployed on BiB catheter over a stiff guide wire placed preferably in the left pulmonary artery. Prior to valve insertion, the valve stent is crimped symmetrically using a specialized crimping device onto a 30mm long presized balloon cat heter. The valve delivered across the presented outflow tract over a very stiff guide wire; Meier wire or Lunderquist wire. Multiple angiograms are performed prior to balloon inflation to ensure proper position of the stent.
Post procedural imaging with angiography and/or echocardiography is performed to evaluate the site and function of the implanted valve. Echocardiography enables evaluation of the pressure gradient by Doppler and the degree of valve regurgitation or perivalvular leak.
Hemostasis is achieved after the procedure with either Perclose sutures, which should have been inserted prior to inserting the large sheath or simply by suturing tehcnique.

Complications

- Valve migration.
- Homograft Rupture
- Guide wire injury to a distal branch pulmonary artery
- Damage to the tricuspid valve
- Arrhythmia
- The “Hammock” effect- in which the valve does not oppose the stent leading to stenosis.

CONGENITAL PROCEDURES

BALLOON ATRIAL SEPTOSTOMY

Balloon atrial septostomy (BAS) was first described by Rashkind and Miller in 1966 and remains an important interventional procedure in the palliation of certain forms of congenital heart disease (CHD). Creating an atrial septal defect in patients with transposition of the great arteries (d-TGA) will enhance bidirectional mixing of the pulmonary and systemic venous blood, hence improving oxygen saturation.

INDICATIONS

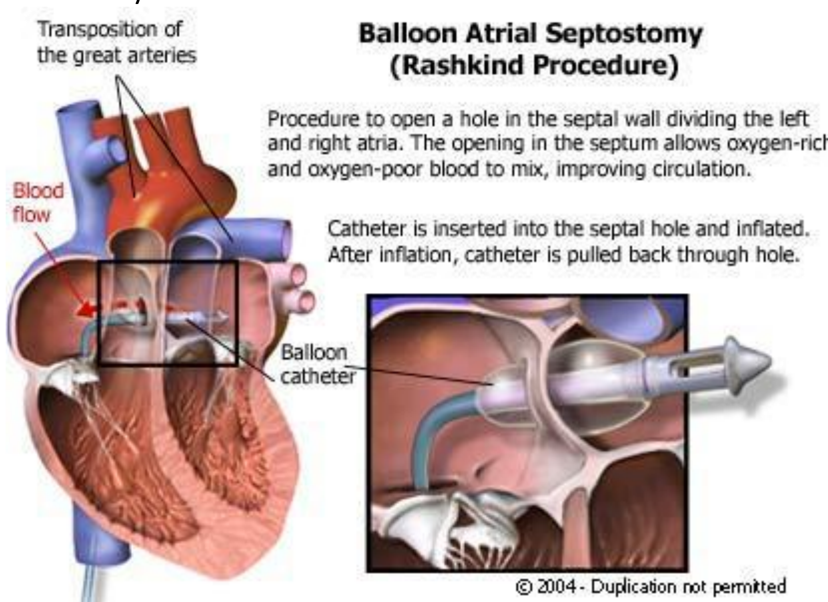
- D-TGA
- Hypoplastic left heart syndrome
- Double outlet right ventricle

CATHETER DESCRIPTION

A variety of catheters are in use for balloon atrial septostomy. The most commonly used catheter at the present time, the 5F Miller balloon atrial septostomy catheter, needs a 7F to 8F introducer. The low profile, dual lumen Z-5 atrioseptostomy catheter, comes in 4F or 5F sizes, depending on the balloon diameter, and requires a 5 or 6F introducer, respectively. The Z-5 catheter is advanced over a guide wire, as opposed to the Miller catheter.

PROCEDURE

The access can be achieved via the femoral or the umbilical vein. The femoral vein is punctured in the typical way, a 4 or 5F introducer is placed in the vessel, and then exchanged for a 7F or 8F introducer, when the Miller catheter is used. The balloon catheter is advanced into the right atrium. Then the foramen ovale is crossed and the catheter is positioned in the left atrium. Correct position is documented via biplane fluoroscopy or two-dimensional echocardiography. In the anteroposterior fluroscopic projection, it is sometimes advantageous to position the catheter tip in the orifice of the left upper pulmonary vein, to confirm its left atrial location. The balloon is quickly inflated with 3-4 ml of dilute radiopaque solution, locked, and then sharply withdrawn into the right atrium down to the junction of the inferior caval vein. The catheter is then advanced into the body of the right atrium, so as not to obstruct inferior caval return, and then deflated rapidly. This maneuver may be repeated two to three times. The size of the interatrial communication can be measured directly by echocardiography at the table. The efficacy is usually immediately obvious, from the rise in systemic arterial saturation.



DEVICE CLOSURE OF VENTRICULAR SEPTAL DEFECT

A ventricular septal defect (VSD) is a communication (or multiple communications) between the right and left ventricles. VSD's are classified by their location in the ventricular septum. There are 4 anatomic types:

- Perimembranous - upper portion of septum (most common).
- Subpulmonary - below pulmonary valve.
- Muscular - muscle portion of ventricular septum. Usually low in the ventricular septum. Multiple muscular defects may be referred to as 'swiss cheese' defects.
- Atrioventricular canal - located beneath the tricuspid valve. Also called an inlet VSD.

Device Implantation Technique

Different from the atrial septum, the right ventricular trabeculae make crossing a muscular VSD difficult from a venous approach. Therefore a double catheter approach has been widely used for the muscular VSD closure. The procedure is done under general

endotracheal anesthesia and TEE. Access is obtained in the femoral vein, the femoral artery and the right internal jugular vein. The patients are fully heparinized. Routine right and left heart catheterization is performed to assess the degree of shunting and to evaluate the pulmonary vascular resistance. Axial angiography is performed to define the location, size and number of VSD. However, for catheter closure of multiple muscular VSDs, TEE guidance should be routine. The VSD is measured. The appropriate device size is chosen to be equal to or 1-2 mm larger than the VSD size as assessed by TEE or TTE.

The next step in the closure sequence is placement of a long sheath (6-8 French) across the VSD. This can be accomplished in a variety of ways. The most common approach used for mid muscular VSDs is to advance a curved end-hole catheter (Judkins right, or Cobra) into the VSD from the left ventricular side. An exchange length 0.035" Amplatz wire is then advanced through the VSD and the right ventricle into the pulmonary artery. This wire is snared in the pulmonary artery or in the right atrium using the Amplatz gooseneck snare and is exteriorized out the right internal jugular sheath. This provides a stable rail to allow advancement of the 6-8 French long sheath across the VSD. The sheath is preferentially advanced from the jugular approach to limit the sheath size in the artery. The sheath can then be advanced from a retrograde approach through the femoral artery. On occasions to eliminate kinking of the sheath in the aortic arch or the septum area, a 0.018" glide wire is left inside the sheath while advancing the delivery cable and device. Once the device reaches the tip of the sheath, the wire is removed prior to deployment of the ventricular disk. In some patients with larger VSDs, the catheter could cross from the right ventricular side. If the VSD is crossed and a catheter can be placed in the body of the left ventricle the stiff wire is advanced into the left ventricle and the sheath is advanced through the right internal jugular vein into the left ventricle. Once the sheath is in proper position the appropriate sized VSD device is then screwed onto the delivery cable and pulled into the loader under water. The loader is then flushed with saline through the side arm of the valve supplied with the delivery system to prevent any air embolism. The loader is placed into the proximal end of the long sheath and the device is advanced with short pushes of the delivery cable to the distal tip of the sheath. The cable should be advanced without rotation to prevent premature unscrewing of the device. The device is then slowly advanced out the sheath to allow the distal disc to expand. In the cavity of the LV, repeat small injections using the pigtail catheter positioned in the LV after each step is of paramount importance. These injections are used for optimal device positioning. The device and sheath are then retracted against the septum with gentle tension and the sheath is retracted to open the waist of the device in the VSD and open the proximal disc against the opposite side of the septum. The device position is then assessed using TEE. If device position is satisfactory, the pin vise is then fixed onto the delivery cable and the device is released with counter clockwise rotation.

PATENT DUCTUS ARTERIOSUS CLOSURE

A patent ductus arteriosus (PDA) is a blood vessel connecting the aorta with the pulmonary artery. This channel is important prior to birth to allow oxygen rich blood from the mother to circulate throughout the fetus's body.

INDICATIONS

Severity of symptoms often depends on the size of the PDA. Small PDAs may cause no symptoms and are sometimes only detected by the doctor hearing a heart murmur through a stethoscope. Medium to large PDAs may cause fatigue, poor growth and eventually lead to heart failure. 2, 3 All sizes of PDAs may increase a patient's risk for a bacterial infection.

DEVICE DESCRIPTION

The Amplatzer Duct Occluder (AGA Medical Corporation, Golden Valley, MN) is a self-expanding and self-centering device, made from 0.0004 to 0.0005inch Nitinol wire mesh. It is mushroom-shaped with a low profile and consists of a flat retention disk and a cylindrical main body, into which polyester fibers are sewn. Platinum marker bands are laser welded to each end and a steel sleeve with a female thread is welded into the marker band . The retention disk is 4 mm larger than the main body, which itself has a conical structure. The delivery system consists of a delivery cable, a Mullins-type sheath, loader and a pin vise. The device comes in different sizes, requiring sheath sizes from 5 to 7 F for delivery. The size of device chosen is generally such that the diameter of the pulmonary end of the device is at least 2 mm larger than the narrowest diameter of the duct. Device sizes are categorised according to the diameters of the aortic and pulmonary ends of the device. The devices are all 7mm long. The device can be delivered through sheath sizes ranging from 5F (for devices upto 8/6) to 7F.

Implantation technique

The procedure can be performed under conscious sedation in older patients, or using general anesthesia. A single dose of intravenous antibiotic is administered (usually a cephalosporin), as is standard practice for all interventional implantation procedures. The femoral vein and artery are cannulated percutaneously.

The size and configuration of the duct are determined by descending aortic angiography, using a 4F or 5F pigtail catheter. The minimum diameter, the diameter of the aortic ampulla and the length of the duct are measured. An end hole catheter (usually 5F) is passed through the duct from the pulmonary side into the descending aorta, if necessary with the help of an 0.035" guidewire. An end-hole 5F multipurpose catheter has been advanced anterogradely from the femoral vein and through the duct into the descending. The end hole catheter has been exchanged over a exchange wire for a 6F delivery sheath, with the tip of the sheath in the descending aorta. The appropriate ADO device is chosen, such that the diameter of the pulmonary end is at least 2 mm larger than the narrowest

diameter of the duct, and immersed into saline solution. The delivery cable is passed through the loader and the device is screwed on clockwise to the tip. The delivery cable for the ADO has been passed through the loading sheath. After attachment of the ADO to the delivery cable, the cable is gently withdrawn to allow the device to pass into the loading sheath. Thereafter, the whole system is immersed into saline again. The device is now pulled into the loader and the loader is introduced into the delivery sheath. The ADO is completely within the loading sheath, and the entire system is flushed with saline to remove any air bubbles. The device is advanced through the delivery sheath into the descending aorta. An aortogram is performed to confirm correct device position, taking care in particular to ensure that the retention disc is sitting entirely on the rim of the ductal ampulla, is not obstructing the descending aorta, and has not partially prolapsed into the body of the duct. If the position is correct, the device is released by rotating the delivery cable in an anticlockwise direction (as indicated by the arrow on the vise) with the pin vise.

PATENT FORAMEN OVALE CLOSURE (PFO)

PFO is one of the most common congenital defects. PFO as a factor in cerebrovascular accident, migraine and transient ischemic attacks.

PROCEDURE

Use a wedge catheter (end-hole catheter) of 7F diameter. The inner lumen of the catheter admits a stiff wire. In order to provide stiffness to the catheter, an Amplatzer super-stiff wire through it (advanced to the tip of the catheter-not outside of the catheter). Then cross the PFO while coming from the inferior vena cava, a gentle clock-wise torque while advancing the catheter is helpful in crossing almost all defects. The primary trick is that the interventionalist should be able to propagate the torque while advancing the catheter in. Then gone to the SVC and pulled the catheter tip in the right atrium to cross the PFO. Sometime, a multi-purpose or Judkin's right coronary artery catheter can be used. A Terumo guide wire is very helpful in teasing the catheter through the PFO. Once the catheter is across the PFO, a wire is advanced through it into the left upper pulmonary vein. Once, the wire is in the left upper pulmonary vein, the catheter is removed and the delivery sheath advanced over the wire. The sheath may or may not be advanced into the pulmonary vein. Its position can be easily checked with echocardiography. The dilator and the wire are removed. The sheath is allowed to bleed back and then flushed with saline. The device is advanced through the delivery sheath and under fluoroscopy and echocardiography, the device is deployed. Since the PFO is a slit-like opening, it is important that the right atrial disc overlaps the limbus. If there is no right atrial disc overlap, the risk of device embolization to the left atrial side increases.

PDA STENTING

The stenting can be done through femoral access, axillary access, jugular access. The access taken based on the origin of PDA. ‘Vertical’ ductus arteriosus generally is not

amenable to stenting via the retrograde, femoral artery route as it is very difficult to engage, axillary access is taken for this. Heparin is given before the procedure.

A 5F Judkin's Right guide catheter us manueuvered into the ampulla and a hand injection is done under fluoroscopy to visualize the duct. A 0.014 coronary wire is used to cross the ductus and enter into the pulmonary artery. It is important to have a Y-connector secured at the hub of the catheter to prevent blood loss. The choice wire short, hydrophilic and stiffer wire for tracking the balloon across the ductus. The wire is passed to LPA. Based on the angiogram the length and diameter is measured. Stent size is taken based on this length and size of PDA and a bare metal stent is taken. Stent is advanced through the guide wire towards the ampulla, it is necessary to ensure that the guide wire remains as straight as possible. Once the tip of the stent is across the narrowest part of the ductus contrast injections are repeated in the same projections and fine adjustments are made to position the stent. Once the position is statisfied, inflate rapidly and deflate rapidly. The stent is inflated only on the nominal pressure, because over inflation makes it to produce thrombosis. It is important to look for branch pulmonary artery stenosis and ensure both the pulmonary and the aortic ends of the ductus are covered.

COMPLICATIONS

- Acute thrombosis
- Spasm of ductus arteriosus
- Migration of the stent

MAPCA COILING(Major Aorta-Pulmonary Collateral Arteries)

INDICATION FOR COILING

MAPCA are required to be closed before surgery of TOF and for hemostatis.

PROCEDURE

The arterial access is taken and usually femoral access is taken. A 5fr pigtail is advanced and placed in DTA and a DTA angiogram is taken. The collaterals are visualized. A 5fr RCA or MPA is taken to hook up MAPCA and the diameter of MAPCA is measured. The coil is taken based on the diameter we measured. The appropriate coil is taken it is advanced through the catheter and diploid. The DTA angiogram is taken after the procedure to check the residual flow.

DEVICE CLOSURE OF RUPTURED SINUS OF VALSALVA (RSOV)

Rupture of aneurysm of the sinus of Valsalva (RSOV) is an uncommon condition with a wide spectrum of presentation, ranging from an asymptomatic murmur to cardiogenic shock or even sudden cardiac death.

TECHNIQUE

All the patients underwent a detailed clinical evaluation and echocardiographic assessment, to determine their hemodynamic status, the origin and exit site of the RSOV,

presence of associated lesions such as VSD and aortic regurgitation (AR), and evaluation of biventricular contractility. After assessing the suitability for device closure, informed consent was obtained. The procedure wasdone under general anesthesia with transesophageal echocardiographic (TEE) and fluoroscopic guidance. All the patients were heparinized (100 IU/kg) after obtaining vascular access (femoral artery and vein).

Detailed TEE evaluation was done prior to proceeding with the hemodynamic study and angiography. This included assessment of the maximum diameter of the aortic end of the RSOV, the minimum diameter and the length of the windsock, and the distance of the aortic end of the RSOV from the coronary ostium. Presence of AR and VSD were also ruled out. An aortogram was done to confirm the TEE findings pertaining to the RSOV and to assess the degree of AR. A left ventricle angiogram was done to exclude any VSD. The RSOV was crossed with a 6F Judkin Right catheter and a 0.035" × 260 cm straight tipped Terumo wire from the aortic side. The wire was manipulated into the superior vena cava (SVC) and snared through the right femoral vein with a 10 mm Goose Neck Snare, to form an arteriovenous loop. The delivery sheath was passed from the venous end and pushed over the wire across the RSOV. The device was loaded into the sheath. The aortic retention disc was opened into the ascending aorta and the entire system was pulled back till it anchored at the aortic end of the RSOV. At this point, the other end of the device was delivered by stabilizing the loading cable and pulling back the sheath. The entire maneuver was performed under fluoroscopic and transesophageal echo guidance. Once it was found to be optimum, the device was released.

PATENT DUCTUS ARTERIOSUS

For decades cardiologists have sought an effective transcatheter method of closing the PDA. Coil occlusion of the patent ductus is simple and effective. It requires only a 4F or 5F catheter and is relatively inexpensive. Since first described in 1992, coil occlusion of the restrictive PDA has rapidly become the treatment of choice at many institutions. It provides effective therapy for the large majority (more than 90%) of restrictive PDAs when the minimum angiographic diameter is less than 4 mm. Coil embolization has also been described for the larger but still restrictive PDA with a minimum diameter of 4 to 7 mm. The coil occlusion technique is not appropriate for the nonrestrictive PDA, and its use in the clinically silent PDA has also been questioned. Coil occlusion of PDA can be performed transarterially or transvenously and may require implantation of one or more coils. The use of a snare catheter to hold the pulmonary artery end of the coil during transarterial delivery may facilitate successful PDA occlusion. Follow-up data have shown that tiny residual shunts noted immediately after coil implantation often resolve spontaneously. A recent retrospective study has found that hospital charges are substantially lower for coil occlusion than surgical ligation even when charges associated with surgery for residual PDA after coil occlusion are taken into account. Complications related to PDA coil occlusion include a persistent residual shunt in 5% to 10% of cases, embolization of a coil to the

pulmonary artery or rarely to a systemic artery requiring catheter retrieval, occasional femoral artery injury following cannulation with a 4F to 5F catheter, and very rarely hemolysis associated with a residual shunt. Important left pulmonary artery stenosis, coarctation, clinical thromboembolism, endarteritis, or late recanalizations have not been reported after PDA coil occlusion.

COARCTATION OF AORTA AND STENTING

It is not uncommon for newborns and young infants with severe coarctation of aorta to present with cardiogenic shock, metabolic acidosis and end organ dysfunction when the ductus arteriosus closes.

PROCEDURE

After explaining the details of the procedure, complications and alternative options, an informed consent was taken from parents. Conscious sedation was given. A 4F pediatric introducer sheath was inserted in one of the femoral arteries and 100 U/kg of heparin administered. A 4F right coronary catheter with 0.018 Terumo wire was used to cross the coarctation segment. No difficulty was encountered during attempts to cross the segment. Pressure gradients were recorded across the coarctation segment. An aortic angiogram straddling coarctation segment was done using a 4 F pigtail in both posterior-anterior and lateral views. Isthmus and transverse arch measurements were made. The balloon diameter for dilatation was determined by the echocardiographic or angiographic (whichever was larger) measurement of the isthmus. The narrowest coarctation diameter was not used to determine balloon size. Balloon was upsized if residual gradients were present, but did not exceed distal transverse arch dimension. Coronary and renal stent balloons were used for dilatation using an indeflator with a pressure gauge. The balloon dilatation was graded and the nominal inflation pressures recommended for the balloons were not exceeded. Pressure gradients were recorded across the dilated/stented segment and repeat angiograms were done to assess the efficacy of the procedure. The decision to stent the coarctation segment depended on the aortic arch anatomy, degree of arch hypoplasia and associated cardiac lesions. The criteria for stenting were not predefined and not uniform. In general stent was used when the isthmus was hypoplastic. Stent was deployed primarily and not after balloon dilatation. Balloon dilatation alone was preferred when the coarctation was thought to be discrete with a good sized isthmus. Essentially any premounted baremetal renal or coronary stent of appropriate diameter (4-5 mm) with a length of 8-13 mm was used. All stents were deployed at the recommended pressure. After balloon dilation or stent deployment final angiograms and pullback gradients were obtained.

ENDOMYOCARDIAL BIOPSY

Endomyocardial biopsy is an uncommon procedure in the catheterization laboratory and used mostly for monitoring of cardiac transplant rejection.

INDICATIONS

Monitoring cardiac transplant rejection and determined anthracycline cardiotoxicity, myocarditis, restrictive or infiltrative cardiomyopathy.

CONTRAINDICATIONS

Anticoagulation and anatomic abnormalities.

BIOPSY DEVICES

There are two basic types of bioptomes are stiff shaft devices and floppy shaft devices that are positioned with the aid of a long sheath. The femoral sheath dilator is 94cm long, and the long sheath is 85cm long. Biopsy sheaths come in 5 and 7cm curves.

CARDIAC TAMPONADE

Cardiac tamponade is pressure on the heart that occurs when blood or fluid builds up in the space between the heart muscle (myocardium) and the outer covering sac of the heart (pericardium).

Causes

In this condition, blood or fluid collects in the pericardium, the sac surrounding the heart. This prevents the heart ventricles from expanding fully. The excess pressure from the fluid prevents the heart from working properly. As a result, the body does not get enough blood. Cardiac tamponade can occur due to: Dissecting aortic aneurysm (thoracic), end stage lung cancer, Heart attack (acute MI), Heart surgery, Pericarditis caused by bacteria or viral infections, wounds to the heart. Other possible causes include: Heart tumors Hypothyroidism, Kidney failure, Leukemia, Placement of central lines Radiation therapy to the chest, recent invasive heart procedures, Recent open heart surgery.

SYMPTOMS

Anxiety, restlessness, sharp chest pain that is felt in the neck, shoulder, back, or abdomen. Chest pain that gets worse with deep breathing or coughing, problems breathing, Discomfort, sometimes relieved by sitting upright or leaning forward, Fainting, light-headedness. Pale, gray, or blue skin, Palpitations, Rapid breathing, Swelling of the abdomen or other areas. Other symptoms that may occur with this disorder: Dizziness, Drowsiness, Weak or absent pulse.

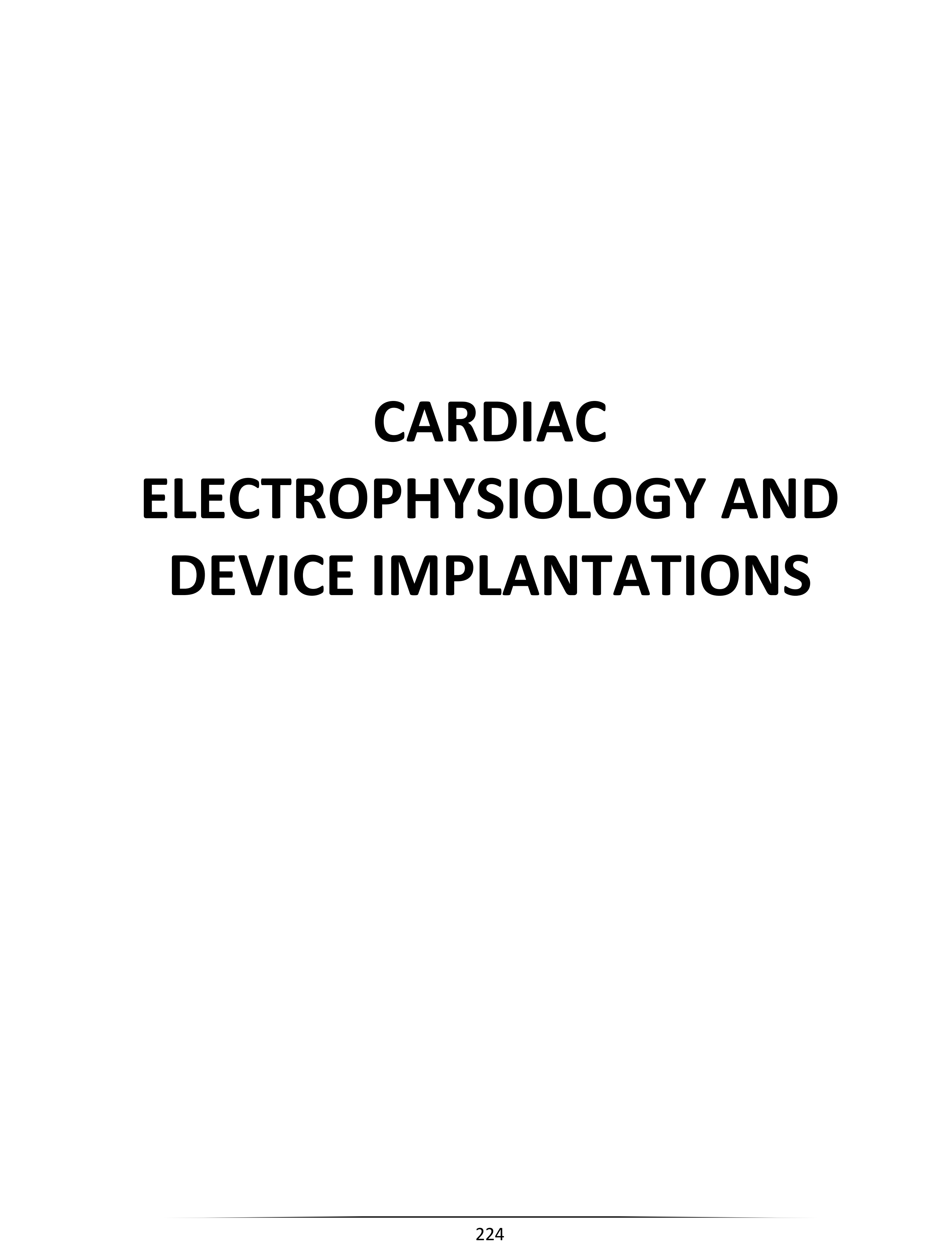
PROCEDURE

Peri cardiocentesis is usually proceeding by echocardiographic confirmation of pericardial fluid. The patient will be given a mild sedative. An IV (intravenous) line is inserted in your hand or arm in case fluids or medications are needed. The operator will use a local anesthetic to numb an area on your chest. A needle will be inserted and then a 6F pigtail a catheter (a thin plastic tube) will be threaded into the pericardial sac around your heart. Then will drain the fluid that has collected around your heart. When the fluid has been removed, the catheter may be removed. Sometimes it is left in place for more drainage.

COMPLICATIONS

Peri cardiocentesis is usually fairly safe, especially when imaging is used to guide the needle. But there is a risk that the procedure may:

- Induce an irregular heart rhythm
- Cause cardiac arrest
- Cause a heart attack
- Puncture the heart, liver, lung, stomach



CARDIAC ELECTROPHYSIOLOGY AND DEVICE IMPLANTATIONS

CARDIAC ELECTROPHYSIOLOGY

INDRODUCTION

The use of cardiac electrophysiology procedures includes diagnostic and interventional treatment procedures. In general, diagnostic EP studies are performed to determine an arrhythmia diagnosis or EP mechanism of known arrhythmia. Interventional or therapeutic EP studies consist of endocardial catheter ablation of supraventricular and ventricular arrhythmias.

SET-UP OF THE ELECTROPHYSIOLOGY LAB

Setting up an electrophysiology laboratory for cardiac ablation requires specifics in the layout of laboratory, and equipments requirements. Conventional electrophysiology studies and ablation procedures should be performed with adequately trained personnel. One or two physicians are responsible for catheter manipulation and ablation. For most EP procedures, conscious sedation is preferred to allow for assessment of symptoms and minimization of risks associated with anaesthesia. Support for complications related to EP studies and ablations should be readily available, including cardiac and vascular surgery and neurologic imaging modalities.

JUNCTION BOX

The junction boxes receive the intracardiac signals from the catheters and provide an interface in to the physiologic recorder. Multiple switches within the junction box are designated to a recording and stimulation channel which can be selected through the recording apparatus. The junction boxes are mounted at the foot of the patient table and connected to the physiologic recorder, which is kept as close as possible. This helps to minimize noise on the channels as well as reduce floor cutter.

RECORDING APPARATUS

The physiologic recorder records, displays and stores intracardiac and surface recording. It consists of filters, amplifiers, display screens, and recording software. From the junction box, the physiologic signals are typically low in amplitude and require amplification recorder. These signals are typically low in amplitude and require amplification prior to displaying and recording. The recording system amplifies and filters each input channel separately, with most currents systems supporting up to 64 or more channels. The amplifiers have the ability to automatically or manually adjust gain control. The amplifiers should be mounted as close to the patient table as possible. This will reduce the cable length of the intracardiac connections and surface ECG, which minimize the signal noise. The amplifiers then connected to the main physiologic recorder through a floor channel, which, ideally, should run separately from electric power cables. Filters are used to eliminate unnecessary signals that distort electrograms. High pass filters eliminate signals

below a given frequency and low pass filters eliminate signals above a given frequency. Most intracardiac electrograms are clearly identified when the signals is filtered between a high pass of 40 Hz and a low pass of 500 Hz. Several pages can be simultaneously recorded and one of these typically includes a 12-lead ECG. The page displayed during studies typically shows several intracardiac electrograms with 3 to 4 surface ECG leads which allow for axis determination, activation timing, and P, QRS morphology. Pressure channels if used, for simultaneous hemodynamic monitoring.

STIMULATOR

A programmable stimulator is necessary to obtain electrophysiologic data beyond measurement of conduction intervals. Stimulators are capable of various modes of pacing, including rapid pacing, delivery of single or multiple extra stimuli following a paced drive train, and delivery of timed extra stimuli following sensed beats. Stimulators should be capable of delivering variable currents, ranging from 0.1 to 10 mA. With satisfactory positioning of catheters, current thresholds under 2mA can usually be achieved in both the atrium and ventricle. Higher outputs are seen with diseased myocardium, within the coronary sinus, and with the use of anti arrhythmic medications. Output is usually set at twice the diastolic threshold. Most stimulators have the ability to pace through more than one channel; however one channel generally suffices for all studies unless dual chamber pacing is required.

CARDIOVERTER

A primary and back up defibrillator should be available throughout all EP studies. Current defibrillators deliver energy in a biphasic waveform which offers enhanced defibrillation success. Defibrillation pads are attached to the patient and electrically grounded. ECG can be recorded through the defibrillation pads separate from the data acquisition system.

RADIOFREQUENCY ABLATION

Radiofrequency ablation uses alternating current delivered between the catheter tips and grounding source to deliver energy to tissue, resulting in necrosis. Radiofrequency generators deliver current with a frequency generators deliver current with a frequency between 300 and 750 kHz, with generation of heat occurring as a result of resistive and conductive heating. Monitoring of time, power, and impedance is necessary to ensure safe and effective ablation lesions. Through the generator, limits on impedance and temperature are programmed and the desired power level is set.

MAPPING SYSTEMS

Cardiac mapping is the process by which arrhythmias are characterized and localized. Conventional mapping involves acquiring electrogram data from fixed and moving catheters and creating mental activation maps with fluoroscopic two dimensional images. More sophisticated mapping techniques provide three dimensional anatomic localization of the catheter to assist in mapping and ablation. These technologies involve the acquisition of multiple electrogram location to provide a high resolution activation, voltage

or propagation map. In addition to correlating local electrogram to 3D cardiac structures, these newer mapping techniques reduce the radiation exposure to the patient and physician. The most widely used the electro anatomic mapping by creating electrical fields between opposing pairs of patch electrodes located on the patient's chest. Six patches are placed on the body to create three orthogonal axes with the heart located centrally. A transthrosic electrical field is created through each pair of opposing patch electrodes and the mapping catheter delivers this signal for processing.

INDICATIONS

SINUS NODE DISEASE

For most patients with sinus node disease, the decision regarding whether to implant a pacemaker can be made on the basis of the history and ambulatory ECG monitoring; electrophysiology testing of the sinus node is not necessary. However, in some patients persistent symptoms may be associated with only mild ECG abnormalities. In these cases determination of sinus node recovery time may be helpful, as a markedly prolonged sinus node recovery time is an indication for implantation of a permanent pacemaker. However, a normal sinus node recovery time does not exclude symptomatic sick sinus syndrome, and the significance of a mildly abnormal sinus node recovery time is established.

ATRIOVENTRICULAR BLOCK

For most patients with atrioventricular block the decision regarding whether to implant a pacemaker can be made on the basis history and the surface ECG without intracardiac recording. Patients who have symptoms require a pacemaker. Patient who have no symptoms require pacemakers only for high-grade infranodal block. The mobitzII pattern during second degree block and the rate and morphologic characteristics of the escape rhythm during complete heart block are quite accurate in predicting the level of block. However, for some patients the diagnosis is not clear from the surface ECG. Specifically, intracardiac recording may be helpful in patients with AV Wenckebach block associated with bundle branch block, apparent Mobitz II pattern in the setting of a narrow QRS segment, and apparent Mobitz II pattern with frequent junctional extrasystoles, suggesting concealed junctional extrasystoles mimicking Mobitz II pattern.

INTRAVENTRICULAR CONDUCTION DELAY

Patients who are free of symptoms who have bifasicular block have demonstrated slow progression to high- degree block and do not require specific therapy. Patients with symptomatic bifasicular block may have intermittend complete heart block that has escaped detection on monitoring. For these patients, demonstration during EPS of pacing induced infranodal block or of a markedly prolonged HV interval is an indication for pacemaker implantation. A normal study does not exclude the possibility of intermittent heart block but makes it much less likely. For some of these patients with bifasicular block and symptoms, EPS will reveal causes for syncope other than heart block.

SUPRAVENTRICULAR TACHYCARDIA

Patient with supraventricular tachycardia (SVT) that is only mildly symptomatic may be treated with empiric drug therapy, if treated at all. EPS is needed for patients with rapid tachycardia associated with severe symptoms patients with recurrent symptomatic bouts of tachycardia refractory to empiric therapy. In such cases EPS allow selection of drug therapy, it allows the selection of nonmedical forms of therapy such as specific antitachycardia pacemaker and antitachycardia surgery.

WOLFF – PARKINSON – WHITE SYNDROME

Patients with WPW syndrome constituted a significant portion of patients with SVT requiring study. Because of the complex interactions of drugs on the normal and anomalous pathways, prefer to study all patients with WPW in whom drug therapy is being started for tachycardias. Clearly, all patients with a very rapid ventricular response to atrial fibrillation require EPS. Patient with WPW who are free of symptoms may also have the potential for very rapid conduction during atrial fibrillation, but the risk of this appears to be quite small.

DIFFERENTIAL DIAGNOSIS OF WIDE QRS TACHYCARDIA

EPS is the only definitive way to differentiate SVT with aberrancy from VT. In most patients, the tachycardia in question can be reproduced in the EP lab. Careful documentation of the relationship of the His bundle and the atrial electrogram allows a definitive diagnosis of the nature of the tachycardia.

VENTRICULAR TACHYCARDIA

EPS is indicated for all patients with recurrent, sustained VT. This arrhythmia can be reproduced in the electrophysiology laboratory in 90% of patients using the technique of programmed stimulation. Moreover, drug testing during EPS predicted the clinical response to medications, with the possible exception of Amiodarone. EPS is mandatory if a pacemaker, the automatic implantable cardioverter, or surgical therapy is being considered.

SYNCOPE

For most patients with syncope, a careful history, physical examination, and ECG will establish a diagnosis. When syncope remains unexplained after non invasive evaluation, and particularly when syncope is recurrent, EPS is indicated.

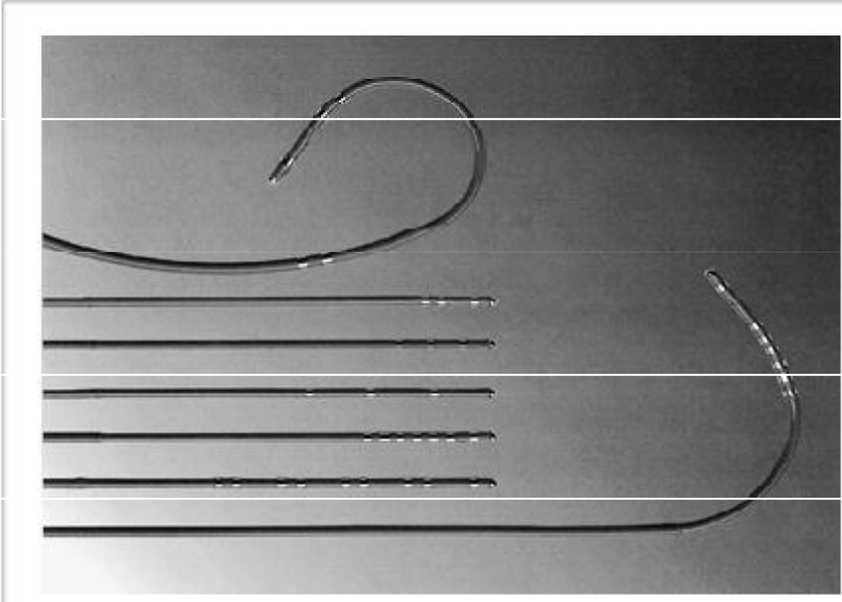
ELECTRODE CATHETERS

A variety of catheters is currently available with at least two ring electrodes that can be used for bipolar stimulation and or recording. The catheter construction may be of the Woven Dacron variety or of the newer extruded synthetic materials such as polyurethane. As a general all-purpose catheter, we prefer the woven Dacron catheters because of their greater durability and physical properties. These catheters come with a variable number of electrodes, electrode spacing, and curves to provide a range of options for different

purposes. Although they have superior torque characteristics, their greatest advantage is that they are stiff enough to maintain a shape and yet they soften at body temperature so that they are not too stiff for forming loops and bends in the vascular system to adapt a variety of uses. The catheters made of synthetic materials cannot be manipulated and change shapes within the body, so they are less desirable.

The advantages of the synthetic catheters are that they are cheaper and can be made smaller than the woven Dacron types. Currently, most electrode catheters are size 3 to 8 French. The smaller sizes are used in children. In adult patients, sizes 5 to 7 French catheters are routinely used. Other diagnostic catheters are routinely used. Other diagnostic catheters have deflectable tip. These are useful to reach and record specific sites. In general, a quadripolar polar catheter suffices for recording and stimulation of standard sites in the right atrium, right ventricle, and for recording his bundle electrogram. Standard catheters 24 poles that can be deflectable to map large and or specific areas of the atrium. Of particular note are catheters shaped in the form of a halo to record from around the tricuspid ring.

Ablation catheters generally have a larger tip (4mm) so that the energy is more efficiently distributed. They have flexible tips, are capable of independent tip rotation, flexion and extension, and have different curvatures that are suitable application with small or large hearts. Some catheters have a cooled tip, one through which saline is infused to allow for enhanced tissue heating without superficial charring. Ablation catheters deliver RF energy through tips that are typically 4-5 mm in length but may be as long as 10 mm.



CATHETER PLACEMENT

Most electrophysiologic catheters are placed via the femoral vein. The atrial catheter may be positioned at the junction of the superior vena cava and right atrium or in the right atrial append is positioned by prolapsing the catheter the catheter across the tricuspid

valve into the right ventricle in the AP fluoroscopic position, where the tricuspid valve is seen in a profile and the His bundle.
The right ventricular catheter may be positioned in either the apex or the outflow tract. We prefer the use of the deflectable tip catheter for the right ventricular recording and pacing because it is easier to manoeuvre it to position. Positioning of coronary sinus catheters may be accomplished either from the right femoral position or from the left subclavian or jugular approach. Coronary sinus is located in the medial posterior septum of the right atrium and in the RAO view lies posterior to the inferior margin of the tricuspid valve.
Placement of catheter in the left ventricle may be accomplished by either the retrograde transaortic or trans septal approach. Occasional epicardial mapping needs to be done. In paediatric patients, due to limited access less number of catheters may be used and occasional oesophageal catheters for atrial signal recording may be used.

PATIENT PREPARATION

Obtain informed consent. Explain to the patient the nature of the procedure and the risks involved. If the patient has ventricular arrhythmias, explain the possibility of inducing VF in the laboratory. We explain to that the occurrence of a ventricular arrhythmia in the laboratory is much preferable to its occurrence at home and that in the laboratory we are prepared to treat this arrhythmia immediately. Most patients undergoing these studies are aware of the serious nature of their arrhythmias and are able to accept this risk without undue anxiety. Discontinue all antiarrhythmic drugs at least 4 half – lives before the study. Avoid premedication if possible because of the electrophysiologic effect may influence the study. The patient may take nothing for 6 hours before the procedure in case emergency cardioversion is necessary.

INTRACARDIAC ELECTROGRAMS

Intracardiac electrograms (IEGMs) and surface electrocardiograms (ECG) both record cardiac electricity activity. However, there are some differences that one must understand for appropriate interpretation of intracardiac electrograms. The surface ECG, which is recorded outside of the body on the surface, provides information about the electrical activity of the entire heart. Intracardiac electrograms are recorded inside the heart by intracardiac multipolar catheters and display local electrical activity near the recording electrodes of the catheter (near field) as well as more remote significant cardiac electrical activity (far field). The IEGMs are usually filtered differently from the ECG to minimize noise and interference. The paper recording speed is also generally faster than the standard 25mm/s 12-lead ECG speed, 100 or 200 mm/s more frequently. The intervals measured during an EP study are generally expressed in milliseconds rather than beat per minute. To obtain the cycle length in ms, the following formula is used:

Cycle length in ms = 60,000/rate in bpm

Conversely, the rate is obtained as the Rate in bpm = 60,000/cycle length in ms. Like surface ECG, electrical potentials can be recorded between two electrodes within the heart (bipolar) or one electrode in the heart (unipolar), the other being outside.

BASIC INTERVALS

The A-H interval represents conduction time from the low right atrium at the interatrial septum through the A interval is at best only an approximation of A measurement should therefore, be taken from the earliest reproducible rapid deflection of the atrial electrogram in the His bundle recording to the onset of the his deflection. This time estimate the conduction time across the AV node and is generally between 50 and 120 ms.

The H-V interval represents conduction time from the proximal His bundle to the ventricular myocardium. The measurements of the interval is taken from the beginning of the his bundle deflection to the earliest onset of ventricular activation recorded from multiple-surface ECG leads or the ventricular electrogram in the his bundle recording. Reported normal values in adults range from 25 to 55 msec. They are shorter in children.

METHODS OF PACING

EXTRASTIMULUS TESTING

Typically, a drive train of 6 to 8 paced beats s1 is performed in the heart structure at a stable cycle length between 600 and 300 m, followed by a premature beat s2 with a coupling interval starting from 500 to 400 ms. The drive train is repeated with a progressive decrease of the coupling interval by 10 to 20 ms until the structure becomes refractory and is no longer captured. If needed, double (s2s3) or more extra stimuli can be delivered by repeating the same sequence with an s1 drive train sequence, an s1-s2 conducted beat and a decremental scanning interval of s3. The minimum coupling interval is commonly 200 ms because of the important risk of inducing arrhythmias with shorter coupling intervals.

INCREMENTAL PACING

- Incremental pacing begins at a stable cycle length slightly below that of the sinus rhythm. The pacing cycle length is then shortened by 10 to 50 ms, a few beats are observed at the new pacing cycle length and the cycle length is again decreased by the same amount. This sequence is repeated to achieve the goal of incremental pacing, determination of the Wenckebach's cycle length in the AV node. Of note, the term 'incremental pacing' refers t a rate in beats per minute instead of a cycle length in ms.

OTHER METHODS OF PACING

- BURST PACING; delivering a set number of pulses at a constant cycle length per sequence. The sequence can be repeated at progressively shorter cycle lengths. This method can be used in sinus rhythm to try to induce an arrhythmia or during a tachycardia to penetrate the circuit's arrhythmia. In this latter case, burst pacing cycle length is initially slightly shorter than the tachycardia or at a programmed

percentage of the cycle length of the detected tachycardia. **Ramp pacing** is almost the same future, except that within a pacing sequence, each subsequent paced beat is decremented by a set amount.

- LONG SHORT PACING; a sequence with alternatively a long interval between two paced beats, followed by a short interval. It can be used to provoke arrhythmias or pattern of conduction block.

REFRACTORY PERIODS

- The refractoriness of a cardiac tissue can be defined by the response of that tissue to the introduction of premature stimuli. These periods are measured during an S1drive train and a progressive S2 decrement stimulus. When the refractory periods of a cardiac structure are measured, the input interval will be related to the upstream depolarized structure cycle length and the output to the downstream depolarized structure cycle length.
- The Relative refractory period (RRP) is the longest coupling interval of a premature impulse that results in prolonged conduction of the premature impulse relative to that of the basic drive. The output interval starts to be longer than the input interval.
- The Effective refractory period (ERP) of a cardiac tissue is the longest coupling interval between the basic drive and the premature impulse that fails to propagate through that tissue. Longest input interval that fails to propagate, therefore no output.
- The Functional refractory period (FRP) of a cardiac tissue is the minimum interval between two consecutively conducted impulses through that tissue. I.e., shortest output interval possible that can be elicited by any input interval.

BASELINE RECORDINGS

Baseline recording during a typical electrophysiological study include surface ECG to time events from the body's surface, and IEGMs, all of which are recorded simultaneously. Electrical activation of the atrium is followed by that of the ventricle with a delay which occurs in the atrioventricular node.

The intracardiac recordings obtained from the electrodes are sharper unlike the wave forms of the surface ECG. These electrograms fall within the P wave or the QRS complex depending on the spread of activation within the chamber in which they are placed. Typical electrogram of the high right atrium electrode will show a sharp deflection in the early part of the P wave. Recording from the His electrode shows an A(Atrial) signals, a His signal and a V(ventricular) signal. As the coronary sinus is in the left AV groove, a coronary sinus catheter records both the left atrial and left ventricular electrograms. RV catheters records ventricular signals from the right ventricle. Activation of the left atrium at the CS ostium (CSP) electrode is normally earlier than at the CS distal (CSD) electrode. Catheters records the signal from all the four chambers directly or indirectly and the His bundle.

After baseline measurements are recorded, pacing is performed via intracardiac electrode catheters. Different chambers can be paced and the activation patterns noted.

ASSESMENT OF SINUS NODAL DISEASE

Sinus nodal (SN) function is assessed by sino-atrial conduction time and sinus nodal recovery time. Measuring sino-atrial conduction time (SACT) involves placement of a catheter near the sinus node from which progressively premature atrial extrastimuli are introduced after every eight to tenth beat of either a stable sinus rhythm, or atrial pacing for 8 beats is done at a rate just lower than the sinus rate. The difference of time of the recovery beat post pacing versus that of the sinus cycle length gives us 2 times the SACT value. Interval up to 250 msec is considered normal.

The sinus node recovery time (SNRT) is another method of assessing SN function and is performed by placing a catheter near the sinus node and pacing for at least 30 seconds at a fixed cycle length. This is repeated at progressively shorter cycle lengths. Pacing rates up to 200beats/minute may be employed. The maximum SNRT is the longest pause from the last pacing stimulus to the first spontaneously occurring sinus beat at any paced cycle length. As the sinus cycle length (SCL) affects the SNRT, it is often normalized or corrected. The corrected sinus node recovery time (CSNRT) is determined by subtracting the SCL from the SNRT. Normal values considered are SNRT/SCL<150 percent and CSNRT<550 milliseconds. A normal study however does not rule out clinical sinus nodal disease.

ASSESSMENT OF AV NODAL DISEASE

During sinus rhythm higher than normal AH and HV intervals may represent abnormality in the conduction system. His bundle electrogram duration reflects conduction through the short length of compact His bundle that penetrates the fibrous septum. This interval is normally short, 15 to 25 msec. Fractionation and prolongation, or even splitting of the His bundle potential, is seen with disturbances of His bundle conduction.

The atrium then is paced at progressive faster rates and Wenchebach point is noted. Programmed atrial extra stimuli can be used to determine the effective refractory period of the AV node, which should be <450 milliseconds. If the block occurs in the AH segment at >450 msec, in the absence of other abnormalities, there still is no clearly demonstrated risk of progression to AV block, however having a block in the HV segment suggests His Purkinje disease and pacemaker implantation is advised. Atropine and procainamide can be given if required to stress conduction system.

VENTRICULAR PACING

Normally during ventricular pacing the impulse travels retrograde into the atrium through the AV node. VA dissociation is referred when while pacing the ventricle, no impulse travels back to the atrium. Ventricular pacing is then done at decreasing cycle length till VA conduction is blocked. Thereafter ventricular extra stimuli are given at decreasing interval till a VA block is obtained. During ventricular pacing atrial activation pattern whether central (His A is earliest) or eccentric (His A is later that A in RA or LA recording

catheter) is seen. Lengthening of VA interval with decrement in the stimulus interval suggests decremental conduction. Central and decremental conduction generally suggests conduction through the nodal tissue.
Eccentric and non decremental conduction suggests presence of retrograde conducting accessory pathway. Multiple (3 to 5) ventricular extra stimuli may be needed to induce ventricular arrhythmia some times. VA interval during RV apical and RV base pacing and VA interval of broad and narrow QRS during para- Hisian pacing is useful to detect presence of septal located bypass tract.

VENTRICULAR INCREMENTAL PACING

When retrograde VA conduction is present, the ventricular cycle length is progressively shortened until the ventricle does not conduct to the atrium in a one to one relation. This cycle length is called the AV node retrograde Wenchebach cycle length. The classic pattern of Wenckebach is the most frequently observed with progressive prolongation of the VA interval prior to block. The His purkinje system is more likely to adapt its refractoriness when the pacing cycle length increases progressively during incremental pacing than during extra stimulus testing.

ATRIAL PACING

Atrial pacing from either the right atrium or the left atrium as clinically indicates is done to see for antegrade conduction characteristics through the AV node. As discussed prior Wenchebach point is noted. Most of the time a SVT is induced at Wenchebach point. In presence of WPW syndrome, there can be eccentric activation with increasing preexcitation which can be easily appreciated on a 12 lead ECG during the study. Atrial extra stimuli are then given (single or multiple). Atrial extra stimuli may show an AH jump (where in there is AH prolongation of more than 50 msec with a decrement of 10 msec on atrial extra stimuli interval), induce a tachycardia or bring out pre excitation with or without decremental antegrade conduction with a pre excited QRS complex.

ATRIAL INCREMENTAL PACING

The atrial cycle length is progressively shortened until the atrium does not conduct to the ventricle in a one to one relation. This cycle length is called the AV node antegrade Wenchebach cycle length.

TACHYCARDIA STUDY

Once tachycardia is induced relationship of the atrium and ventricular signals are noted. 1:1 AV activation generally suggest AVNRT, AVRT and atrial tachycardia but rarely ventricular tachycardia. A>V suggests atrial tachycardia and rarely AVNRT but never an AVRT or VT. V> A suggests ventricular tachycardia. During SVT atrial activation patterns helps to diagnose the nature of tachycardia.

MAPPING FOR LOCALIZING SITE OF ABLATION

In many cases, catheter ablation immediately follows diagnostic electrophysiologic study. Mapping refers to careful movement of a mapping or ablation catheter in the area of

interest, probing for the site at which radiofrequency ablation will be successful at curing the arrhythmia.

ACTIVATION MAPPING

Where the activation signal in the mapping catheters shows earlier signals as compared to the surface P/QRS suggests the area to be near the origin of the arrhythmia and suggests an appropriate site for treatment.

PACEMAPPING

In a patient especially with ventricular tachycardia, pacing at the same cycle length as the tachycardia from the mapping catheter and comparing the 12 lead ECG obtained during pacing with that of tachycardia is called pace-mapping. If there is a 12/12 match is suggests the origin of the tachycardia to be nearby and is especially helpful for treatment of idiopathic ventricular outflow tachycardias.

ENTRAINMENT

Pacing during tachycardia from the site of interest, when results in similar activation pattern to that of tachycardia, with difference in return cycle length of<20 msec from that of the cycle length of the arrhythmia, suggests the site of slow conduction and possible site for ablation. It is also called concealed entrainment. Presence of abnormal diastolic potentials, fractionated potentials or areas of continues activity may suggest areas of slow conduction and possible sites of ablation.

RADIOFREQUENCY CATHETER ABLATION

RF energy is a form of electrical energy that is produced by high frequency alternating current. As the current passes through tissue, heat is generated. RF current used during endocardial catheter ablation is alternating current with a 500,000 to 750,000-Hz frequency range. The current passes from the electrode tip to a large surface area skin patch. The current is typically applied for 10 to 60 seconds at a time using 45 to 55 W. Catheter delivery of RF energy causes tissue heating in a small area around the electrode. The typical lesion is 3*4*5 mm. Alternate forms of energy for lesion generation are currently under development, including cryoablation, ultrasound and laser and microwave energy sources.

After a diagnosis is made, an ablating catheter is positioned at target area; catheter can be steered and has four to six electrodes 2 to 5 mm apart. The catheter tip is 4 to 8 mm long and serves as the electrode through which RF current is applied. The target area is located using fluoroscopy and by observing the electrogram patterns recorded by the distal mapping electrode pair.

POST ABLATION PROTOCOLS

To confirm the success of ablation all pacing protocols need to be repeated after successful treatment of the arrhythmia with and without isoprenalin, atropine and adenosine whenever required.

COMPLICATIONS

Complications related to the cardiac catheterization can be largely avoided by good technique and careful patient selection. Patients with left main artery stenosis, unstable angina, and critical aortic stenosis are the absolute contra indications. Other complications are Local vascular complications, perforation and vasovagel reactions.

Radiofrequency ablation shares all the vascular risks and potential complications of EP studies. However, some unique complications may be associated with this technique. These include post ablation chest pain, pericarditis, AV block and radiation burns after prolonged procedures.

PACEMAKERS

INDRODUCTION

Early pacemakers were single chamber devices designed to pace only in the ventricle, and the only programmable parameters were pacing rate and output. With the introduction of dual- chamber pacemakers with the capacity of pacing the atria and the ventricles, the number of programmable parameters increased dramatically. Antitachycardia devices were developed to terminate supraventricular and ventricular tachyarrhythmias using pacing techniques, cardio version, or defibrillation.

INDICATIONS

The primary indication for a pacemaker is symptomatic brady arrhythmias. In occasional instances, severe asymptomatic brady arrhythmias are indications for pacemaker implantation.

The most common indications for permanent pacemakers can be classified as follows;

- **Acquired atrioventricular block**
  Complete heart block, infranodal, persistent or intermittent. Mobitz type II second degree AV block, persistent or intermittent. AV nodal block, Mobitz type I, only when associated with symptoms. Atrial flutter or fibrillation with a 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. Tachycardia – bradycardia syndrome, if drugs necessary to control the tachycardia produce **symptomatic bradycardia**.
- Hypersensitive carotid syndrome with syncope resulting from bradycardia.

CONDRAINDICATIONS

There are very few contraindications to permanent pacemaker implantation. Implantations are contraindicated in patients with active infections; these patients should be managed with medications or temporary pacemaker until the infection is resolved.

RISKS

The risks associated with permanent pacemakers are generally small. They include:

- Catheter dislodgement, lead fracture, or other causes of pacemaker system failure
- Pacemaker syndrome and pacemaker mediated tachycardia
- Infection or erosion of the pulse generator.
- Cardiac perforation with very rare instances of tamponade
- Thrombosis of the superior vena cava or right atrium

EQUIPMENT FOR PERMENANT PACEMAKER IMPLANTATION

The following equipment is necessary for implantation of a permanent pacemaker.

- Pulse generator.
- Ventricular and atrial leads.
- 18 – gauge vascular needle.
- 0.035 inches J – tip safety guidewire.
- Vein dilator with a peel away sheath.
- Pacemaker system analyzer to measure pacing thresholds and endocardial signals
- Pacemaker programmer
- Slandered surgical instruments and suture materials.
- Back up equipment; external pacemaker, defibrillator, emergency cart with drugs, airways, suction equipment, and emergency sets for tube thoracotomy, open thoracotomy and peri cardiocentesis.

PATIENT PREPARATION

Prepare the patient for pacemaker implantation as for any other major surgery. Inform the patient regarding the procedure, the risks, and what to expect. Obtain informed consent from the patient and from the relative, and what to expect. Keep in fasting for 6 to 8 hours before the procedure. Review result of blood count, electrolyte coagulation studies, chest radiograph and ECG. Administer a mild sedative half hour before the procedure. Establish an iv line before the procedure.

Maintain continues ECG monitoring. Select the implant site. Both the left and right pectorial areas can be utilized. Any areas with abnormalities should be avoided. Prepare surgical field. Strict attention to aseptic techniques mandatory for all pacemaker insertions. Do not apply skin electrodes in the pacemaker field.

PACEMAKERS AND LEADS

A pacemaker system for permanent pacing consists of a pacemaker and one or two leads implanted in the right atrium or right ventricle or both. The pacemaker contains a battery,

the power source and a pulse generator, the electronic unit controlling the behavior of the pacemaker.

BATTERY

The power source is usually constant –voltage **lithium- iodine battery**, which has a predictable voltage behavior over time; the battery voltage remains relatively constant throughout most of its discharge. The potentials delivered by brady pacemakers are monophasic. Battery current drain is highly dependent on pacemaker programming. A pacemaker with a battery life of 6 years under nominal pacing parameters may reach its replacement time at 2 years at one extreme or at more than 10 years at the other extreme. Expected battery longevity for dual chamber pacemakers is 5-10 years; for a single chamber pacemaker it is 7-12 years.

PACEMAKER LEADS

Pacemaker leads are unipolar or bipolar. In a unipolar system a single electrode (the cathode) is located at the lead tip, and the pacemaker can act as the anode. Bipolar systems have both electrodes near the distal end of the lead, the tip electrodes acting as the cathode and the more proximal ring electrode as the anode.

Bipolar leads have some advantage over unipolar leads. Bipolar leads reduce the risk of myopotential over sensing, far field sensing, cross talk and local skeletal muscle stimulation and they allow programmable switching between bipolar and unipolar configuration. The most important disadvantage of bipolar leads is that they have lower long term reliability than unipolar leads.

Most modern leads have a small tip surface area with porous surface, promoting low thresholds, low current drain and good sensing. Steroid eluting leads have a little reservoir of glucocorticoids, which diminishes the inflammatory reaction at the electrode-endocardial interface and thus improves acute and chronic pacing thresholds and sensing.

THRESHOLD

The pacing threshold is defined as **the minimum stimulus amplitude** at any given pulse width that is required to achieve myocardial depolarization consistently outside the heart’s refractory period. Threshold can be measured by increasing stimulus amplitude until capture occurs or by decreasing amplitude until loss of capture occurs. At pacing rates below 150 bpm, there is no significant difference between thresholds measured by these two methods. The thresholds depend on the pulse duration according to the strength – duration relationship. At implantation, an atrial threshold of <1.5v and a ventricular threshold of <1.0V at pulse width 0.5ms should be obtained. In most patients, the threshold rises during the first 2-4 weeks after implantation.

SENSING, SENSITIVITY AND IMPEDENCE

Sensing is determined by the peak to peak amplitude in millivolts of the intracardiac electrogram. The amplitude of the ventricular R wave electrogram is larger than the size of the atrial P wave electrogram. At implantation, an atrial electrogram >1.5mVand a

ventricular electrogram of a >6mV should be obtained. After implantation, the amplitude of the electrogram usually declines during the first week, then increases and reaches a chronic value slightly lower than that noted at implantation.

The slew rate defined as the change in intracardiac electrogram voltage over time (dV/dt), should be >0.5V/s in both atrium and ventricle. Both the amplitude of the electrogram and the slew rate are important determinants for whether an electrical signal will be sensed by the pacemaker. In general, there are no significant differences in sensing amplitudes or slew rates between leads with passive and active fixation.

Immediately after lead implantation, the sensed electrogram is distorted by an injury current –an ST elevation after the initial R-wave spike in the ventricular electrogram or an elevation after the P wave spike in the atrial electrogram. This injury current is thought to result from damage to the endocardial surface produced by contact with or pressure from the lead, and thus reflects good endocardial contact. The injury current disappears after a few days.

Sensitivity is the level that an intracardiac electrogram has to exceed in order to be sensed by the pacemaker. Sensitivity is programmable and acts as a filter for noise, ensuring that small are not sensed by the pacemaker and interpreted as cardiac electrograms. Sensitivity should be programmed after measurement of the intracardiac electrogram. Sensitivity should not exceed half that of the sensed electrogram. Sensitivity must be larger than unipolar leads than with bipolar leads because noise signals are larger in unipolar leads. Impedance can be defined as the sum of all forces opposing the flow of current in an electric circuit. Impedance is measured in ohms. In Pacemaker systems, the lead impedance is determined by the resistance of the conducting lead, the tissue between the electrodes, and the tissue- electrode interface. Normal lead impedance is 250-1200 ohm at output 5.0V. Usually, lead impedance remains stable or decreases slightly after implantation. Very low impedance may indicate failure of lead insulation, whereas high impedance accompanied by a high pacing threshold indicates lead fracture.

PROGRAMMABILITY OF PACEMAKERS

Programmability is the features by which the electronically controlled performance of a pacemaker can be noninvasively altered. This alteration is permanent until the pacemaker is otherwise reprogrammed. Programming is achieved by using an external programmer to transmit preselected messages in the form of binary code, via either radiofrequency waves or a pulsed electromagnetic field, to the implanted pulse generator. Features that can be programmed noninvasively include stimulus output, rate, amplifier sensitivity, escape rate, hysteresis, lower and upper rate limits, AV interval, refractory period and mode.

The voltage, current and pulse duration constitute the pulse generator output. The voltages of two batteries in series are added so it is a battery source of about 5.6v. The automatic rate is the interval between consecutive atrial or ventricular paced stimuli. This setting is programmable between 30 and 150 bpm.

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.

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, i.e. the pacemaker does not begin firing until the patient's rate drops below 60 beats per minute.

The lower rate limit is the lowest rate at which a pulse generator is programmed to track to the spontaneous atrial rate. The upper limit is the fastest rate at which a pulse generator is programmed to track the spontaneous atrial rate on 1:1 basis. The AV interval should be programmed within the physiologic rate 0.12 to 0.2 seconds for a maximum cardiac output.

REFRACTORY PERIOD

The refractory period is the period during which a pulse generator a pulse generator is unresponsive to an input signal. The pacing refractory period follows a paced complex, and the sensing refractory period follows a sensed spontaneous complex.

MODES OF PACEMAKERS

The current nomenclature used to describe the expected function of a pacemaker was established by members of the North American Society of pacing and electrophysiology and the British pacing and the electrophysiology group and is designated the NBG code for pacing nomenclature. The code describes the expected function of the device according to the site of the pacing electrodes and the mode of pacing.

The first letter describes the chamber that is paced: A, atrium: V, ventricle, D dual, O none. The second letter describes the chamber where intrinsic electrical activity is sensed: A, atrium V, ventricle D dual O, none. The third letter describes the pacemaker's response to sensing of intrinsic electrical activity. I inhibited, T triggered, D dual, O none. The fourth letter denotes the presence or absence of rate modulation, R rate modulation O none. The fifth letter specifies the location or absence of multisite pacing, which includes either bi atrial or biventricular pacing or more than one stimulation site in a single chamber. The most commonly used pacing modes are VVI and DDD.

PACEMAKER IMPLANTATION

Most frequently in order to place a lead for permanent pacing, the cephalic vein is located and cannulated in the delto pectorial groove under local anesthesia. Less commonly, the internal or external jugular vein is used. Under fluoroscopic imaging the electrode can be guided into the apex of the right ventricle. Satisfactory thresholds are documented by measurement with a pacing system analyzer rather than with a temporary pacemaker. The minimum voltage necessary to capture the ventricle is determined. The electrical resistance in ohm can be calculated. This value reflects the resistance of the lead and the

electrode – myocardial interface. The amplitude of the patient's intrinsically generated QRS pattern is then measured.
The leads are attached to the pulse generator and a pocket is fashioned by separating the pectorils muscle fascia from the overlying subcutaneous tissue. The subcutaneous and skin are closed in layers. Endocardial fixation of leads can be either passive or active. Passive fixation is obtained using leads with tines or wings, which are trapped in the trabeculae of the right atrium or the right ventricle. Active fixation is obtained by screwing a tip –screw into the atrial or ventricular myocardium. Active fixation reduces the frequency of atrial lead dislodgement and is therefore recommended as standard in the right atrium. In the right ventricle leads with active fixation can be implanted in localities other than the apex, such as the outflow tract or the septum. Atrial pacing leads are being implanted with increasing frequency. This kind of lead is almost always implanted simultaneously with a ventricular lead, but positioned in the atrial appendage. If the atrial J lead is used, the lead is advanced with the stylet in place to the lower right atrium near the tricuspid valve. The stylet should be withdrawn and the tip positioned anteriorly as the lead assumes its J shape. The lead then can be pulled back slightly to engage the right atrial appendage. The tip should be stable in the right atrial appendage with gentle rotational movement of the lead and with deep breaths by the patient.
A type of introducer with a peel away sheath is used often for permanent Trans venous lead placement. A subclavian vein puncture is performed, a guide wire is passed into the subclavian vein, and a dilator and a sheath are advanced over the guide wire into the subclavian vein. The guide wire and dilator are then removed, and the sheath is left in the vein for passage of the pacing lead. The lead can be advanced to the heart through the sheath, and sometimes both atrial and ventricular leads are placed through a single sheath. The sheath then can be split apart and discarded to allow connection of the lead to the generator.

LEAD AND GENERATOR CONNECTION

The attachment of the electrode to the generator is an important step in pacemaker implantation, and problems with the connection are relatively common sources of pacemaker malfunction. The exposed metal portion of the connector is inserted into the pulse generator. The insertion must be complete, and the metal wire and insulating material should fit snugly in to the socket. The fit is tight to avoid fluid intrusion into the connection. A metal set screw is screwed down to complete the connection. Finally an insulating plastic plug is placed over the metal set screw to avoid fluid instruction that could lead to a short circuit in the system or corrosion of the connection. Modifications of this type attachment are present in various pacemaker models.

COMPLICATIONS

- Early failure to capture and sense
- Late failure to pace

- Pacemaker syndrome
- Pacemaker – mediated tachycardia
- Infection
- Myocardial perforation and tamponade
- Thrombosis and embolism
- Erosion of the pulse generator

PACEMAKER FOLLOW – UP

Patient should have a thorough evaluation at 2 to 4 weeks after implantation. The history should document relief of pre pacing symptoms. A physical examination should be performed, with special attention to the pacemaker pocket. Pacemaker function should be checked by a rhythm strip with magnet if necessary and accurate records should be kept off the pacemaker analysis. Any necessary adjustment can be made by programming. Many physicians set the pacemaker at a high output for the first 6 weeks to avoid exit block as the lead matures. After this time period the output can be decreased to preserve battery life. Most physicians check the pacemaker by one or the other modalities approximately every two months and increase this frequency to once monthly as the end of the pacemaker life approaches.

IMPLANTABLE CARDIOVERTER DEFIBRILLATOR

INDRODUCTION

An implantable cardioverter-defibrillator (ICD) or automated implantable cardioverter defibrillator (AICD) is a device implantable inside the body, able to perform cardio version, defibrillation and pacing of the heart. The device is therefore capable of correcting most life-threatening cardiac arrhythmias. The ICD is the firstline treatment and prophylactic therapy for patients at risk for sudden cardiac death due to ventricular fibrillation and ventricular tachycardia. Current devices can be programmed to detect abnormal heart rhythms and deliver therapy via programmable antitachycardia pacing in addition to low-energy and high-energy shocks.

INDICATIONS

- With LVEF ≤ 35% due to prior MI who are at least 40 days post-MI and are in NYHA Functional Class II or III
- With LV dysfunction due to prior MI who are at least 40 days post-MI, have an LVEF ≤ 30%, and are in NYHA Functional Class I
- With non ischemic DCM who have an LVEF ≤ 35% and who are in NYHA.Functional Class II or III
- With non sustained VT due to prior MI, LVEF < 40%, and inducible VF or sustained VT at electrophysiological study.
- With structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable.

- With syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiological study.

GENERATOR

Today, ICD generators are similar in size to the original pacemakers and can now be placed within the chest wall. The largest components of the generator are the capacitor and the battery. ICD are usually placed in the pectorial region, allowing the procedure to be performed in the catheterization laboratory.

The generator consists of two major components. One operates at low current, low voltage and low energy, and yields signal amplification, electrogram storage, arrhythmia sensing and detection, and decision making when therapy criteria are fulfilled. The other component operates at high current, high voltage, and high energy. The battery used in the ICD is usually lithium silver vanadium oxide batteries providing 6.4V or 3.2V, which allows rapid discharge into a capacitor for the frequent rapid shocks that, may be required of the ICD device. Longevity is currently often estimated to be 4 years, depending on the frequency of use.

LEADS

The lead would be placed in the right ventricle and would provide continuous sensing of the ventricular rate. The current systems incorporate both therapy and rate sensing function to a single transvenous endocardial lead for simplicity. The bipolar lead can pace in the ventricle and sense the ventricle rate, and the shocking portion of the lead can provide a larger electric charge between the coil and generator.

Current leads are generally dual coil; the generator and the proximal coil have the same polarity. Electricity flows in a three- dimensional configuration from the distal coil to both the proximal coil and generator. This greater dispersion of the electrical field increases the likelihood of depolarizing the entire ventricular myocardium at once leading to successful defibrillation.

SENSING

Sensing in an ICD is much more complicated issue than in a pacemaker. In the ICD the sensing circuit must be dynamic. It must sense and count QRS complexes appropriately and avoid double counting by sensing the T wave, which can lead to inappropriate electrical therapy. The device must in addition be able to become more sensitive after sensing the QRS complexes so that if ventricular fibrillation occurs, it can sense the defibrillatory waves. The device also must be programmable to deal with sensing.

TACHYCARDIA DETECTION

The fundamental way that the ICD identifies the presence of a sustained ventricular tachyarrhythmia is by detecting that the heart rate has exceeded a critical value as measured in the ventricle. Because most episodes of sustained VT exhibit a rate in excess of 150 beats per minute, the device can be programmed to initiate therapy when this rate

is reached. Programming decision regarding tiered therapy must take into account the hemodynamic status of the patient during the tachyarrhythmia episode.
For antitachycardia pacing, additional detection criteria may be programmed to enhance the certaintity that a ventricular tachyarrhythmia is present rather than a supraventricular tachyarrhythmia, often AF, such detection enhancements include the identification of cycle length stability, the abruptness of onset of the tachyarrhythmia, morphology analysis, and the duration of sustained rate. The morphology of ECG as measured by the device may also be used to differentiate supraventricular tachycardia versus ventricular tachycardia.

THERAPY TIERS

Because VF often manifests as rates in excess of 240 bpm, the ICD may be programmed to respond to such rates by defibrillation. With tiered therapy devices, rate ranges can be programmed that determine the type of therapy to be delivered. Thus with rates of 150 to 240 bpm, antitachycardia pacing or low energy cardio version can be used for rates greater than 240 bpm defibrillations may be theprogrammed response.

BRADYCARDIA PACING

Often patients successfully converted out of VT or VF is found to have marked sinus bradycardia immediately post conversion, posing another threat to hemodynamic stability. Therefore modern ICD devices often have the option of VVI, DDD, DDDR, pacing. After a shock, the threshold required to pace may increases. Therefore, as a precaution, a higher output can be programmed for several beats after the shock.

ANTITACHYCARDIA PACING

Many ICDs are implanted as the first – line therapy for recurrent sustained monomorphic VT. It has been shown that a sustained VT can be reliably terminated by antitachycardia stimulation techniques. Delivery of antitachycardia pacing, either a **burst** or a **ramp** of stimuli, is coupled to the last sensed tachycardia beat that fulfilled detection algorithm criteria. The number of stimuli within a burst, the coupling interval and the burst cycle length are programmable. In general, more than one burst attempt is recommended for VT therapy. A scanning mode is a pacing modality in which the burst cycle length of subsequently firing bursts progressively shortens or lengthens. A ramp mode means that the cycle length within one burst shortens or lengthens from stimulus to stimulus. A combination of scanning and ramp pacing may also be applied.

LOW ENERGY CARDIOVERSION

Low level cardio version is occasionally the first line treatment for VT with rates greater than 150 to 180 and less than 240 bpm not responsive to tachypacing. The tiered ICD delivers the cardioversion synchronized with the QRS complex, thus avoiding the vulnerable period of the T wave and risking initiation of VF. The amount of energy may be programmed to a minimum of 0.1J. Significant reduction in pain perception may be noted

below 2J, whereas no perceptible differences may be observed between 5 to 34J in many patients.
More efficient cardioversion has been affected by improvement in lead design, lead arrangement, and the shape of the pulse waveform. If antitachycardia pacing or cardioversion fails to terminate the tachycardia after a programmed time interval or if the tachycardia accelerates, defibrillation therapy will be initiated.

DEFIBRILLATION

Defibrillation is the first line therapy when VF is the presenting rhythm. For defibrillation, electric current delivers does not need to be synchronized with a given portion of the ECG complex. After the patient has been in VF for a programmed interval, shocks that may range between 50 and 850 V or 0.1 and 38 J will be delivered. If the first shock fails to terminate the rhythm, subsequent shocks will be delivered, usually up to five or six, depending on the manufacturer and what has been programmed.

COMPLICATIONS

- Perioperative mortality and morbidity
- Infection
- Frequent ICD discharges
- Sudden cardiac death despite ICD therapy
- Quality of life and driving restrictions

PATIENT FOLOW UP

The fundamental objective of a follow up program for ICD patients is continues lifelong patient care, which must be distinguishing from pure device management. Compliance with follow-up visits is an important element in evaluating appropriate ICD function. Follow up must be performed in an organized program.

CARDIAC RESYNCHRONIZATION THERAPY

INDICATIONS

Basically the current indications are that the patient has a left ventricular ejection fraction of equal to or less than 35%. The source of the cardiomyopathy can be ischemic or non ischemic. The QRS duration should be greater than or equal to 120 msec. The patient should be in the AHA class III or IV congestive heart failure ranking and should be on optimal pharmacologic therapy for congestive heart failure. CRT has been advocated in patients with RBBB and heart failure and patients with LBBB and heart failure.

IMPLANTATION

Patient undergoing CRT has a right atrial lead and ventricular lead placement. In the case of the AF, the atrial lead is not implanted. The more complex portion of the implantation is to place a lead through the coronary sinus and down a vein on the posterolateral side of the heart. The lead is an inactive with no tines or the ability to be secured with screw in leads. The curve at the end is built into the lead itself. There are different types of curves

available, but the general principle is that the curve is designed to cause pressure of the pacemaker tip against the myocardium within the narrow vein. The lateral threshold may be higher than a typical threshold bur the difference is fairly minimal. The sensing is done through the right ventricular lead.

LEAD EXTRACTION

The lead extractions are probably one of the most challenging procedures. The indication for transvenous lead extraction can be categorized into two groups, patients related and lead related. Patients related indications are infection, ineffective therapy, perforation, migration, embolization, induction of arrhythmias, venous thrombosis, unrelenting pain, device interactions, device upgrades. Lead related indications include lead recalls, lead failure, and lead interactions.

TECHNIQUES OF LEAD EXTRACTION

- Control of the lead body and tip, which could be achieved by binding of its elements with the application of uniform force on the entire length of the lead, to remove it in once piece with minimal disruption
- Locking stylets provide tensile strength to the lead all the way to the tip electrode.
- Control disruption of the fibrous tissue using counter pressure
- Bracing the cardiac wall using countertraction, which involves opposing the traction placed on the lead by bracing the myocardium with the overlying blunt sheath. This focuses the traction force perpendicular to the heart wall and limits the counter pressure to the scar tissue immediately surrounding the lead tip.

DIRECT CURRENT CARDIOVERSION AND DEFIBRILLATION

Cardioversion is the procedure by which direct current (DC) that has been synchronized to discharge with the QRS complex of the ECG is delivered across the heart for the purpose of converting an atrial or ventricular arrhythmia to normal sinus rhythm. Defibrillation is non synchronized cardioversion and is used when QRS complex and T waves are not distinguishable or present, as in ventricular flutter or ventricular fibrillation.

THE VULNERABLE PERIOD

The heart is particularly susceptible to ventricular fibrillation during the so called vulnerable period, an interval that begins and ends with the T wave but that is at its peak for about 30 msec just before the apex of the T wave of the ECG. At this point the current required to elicit ventricular fibrillation is at its lowest. In the ischemic heart the stimulus required to cause fibrillation is much less than it is in the normal heart.

TYPES OF DEFIBRILLATORS

DAMPED SINE WAVE

The damped sine wave defibrillator consists of a capacitor, inductor, and electrodes. The energy is stored in the capacitor and is discharged through the inductor to the electrodes

and then directly through the heart or across the thorax and through the heart. The energy delivered to the heart is less than the stored energy because of the resistance in the inductor, the internal resistance of the defibrillator, and the transthoracic impedance, which varies considerable among humans. Thus the energy stored in a defibrillator can be accurately measured, as can the energy delivered into a test load, but the actual energy current delivered to the heart remains variable and not directly measured.

TRAPEZIODAL WAVE

Defibrillators using the trapezoidal wave have been available for more than 10 years. Definitive data regarding their advantages and disadvantages are lacking. Maximal output from these defibrillators is 400 J, and the threshold current needed for defibrillation is approximately the same as for the damped sine wave. It has larger electrodes than do other defibrillation.

INDICATION

- Atrial flutter or fibrillation of recent onset that cannot be converted to a sinus rhythm after the elimination or control of initiating or predisposing factors and after the use of antiarrhythmic medications.
- Supraventricular tachycardia that does not respond to vagal manoeuvres or antiarrhythmic therapy and is not caused by digitalis intoxication
- Ventricular tachycardia that is refractory to antiarrhythmic drug therapy or is associated with hemodynamic instability
- Ventricular flutter or ventricular fibrillation

CONTRAINDICATIONS

Supraventricular tachycardia caused by digitalis toxicity is an absolute contraindication for synchronized cardioversion. It is relatively contraindicated either a low chance of success or high chance of recurrence of the arrhythmia, appreciable risk of precipitating a potentially more dangerous tachyarrhythmia, or risk of precipitating or unmasking of bradycardia or asystole.

SYNCHRONIZING THE CARDIOVERTER

Connect the ECG to the oscilloscope of the cardioverter and monitor the ECG in the lead. Test the synchronous discharge of the cardioverter to be sure that discharge occurs during or slightly after the R wave of the ECG. The risk of provoking VF is the main danger in cardioversion is the procedure is done without proper synchronization.

PADDLE POSITION

Correct paddle electrode placement is important; improper placement reduces intracardiac current flow and is an important cause of failure to cardiovert or defibrillate. The paddle may be applied to the chest in one of three ways; anteroapical, anteroposterior, apical posterior. All three positions are probably equally effective.

For patients with dextrocardia, the position of electrodes should be a mirror image to reflect the change in the heart's position. In patients with heart malposition or chest wall deformity, allowances for paddle position should be made to permit positioning the heart between the paddles.

CHOICE OF ENERGY SETTING

The initial setting of cardioversion in an adult may be 10 to 100 J delivered, depending on the arrhythmia. In VT 10 J may be a large enough charge for successful cardioversion, whereas 100 J is almost always effective. Atrial flutter may be converted to sinus rhythm at energy levels of 20 to 25 J. Very low energy levels may convert atrial flutter to atrial fibrillation and thus are not recommended. Most SVT responds to verapamil or vagal manuevers. When cardioversion is needed, a 50 to 100 J charge is almost always successful in converting it to sinus rhythm. Atrial fibrillation usually requires from 100 to 150 J delivered energy and sometimes over 200J. In case of possible or suspected digitalis excess or subclinical digitalis toxicity, it may be safer to start all electic cardioversion. The first defidrillatory attempt should be with 200J delivered energy, nonsynchronized. Rapidly follow with 200 to 300j and 360 as needed. If refibrillation occurs, repeat the shock at the previously successful energy level.

DEFIBRILLATION PROCEDURE

- Turn on the defibrillator
- Apply the adhesive pads in proper electrode position
- Set energy level to 120 to 200 J for biphasic and 360 for monopahasic defibrillators
- Charge capacitors, it make several seconds
- Ensure proper electrode placement on the chest.
- Scan the area to ensure that no personnel are in contact directly or indirectly with the patient. State firmly "all clear"
- Check rhythm, if patient remains in VF, deliver shock by depressing discharge button
- Repeat assessment procedure

TEMPORARY PACEMAKERS

A temporary pacemaker consists of a transvenous catheter electrode attached to an external pulse generator. Temporary pacemakers are used when the need for pacing is immediate. Temporary pacemakers may be followed by permanent pacers if the need for pacing persist or may simply be removed if the need for pacing is self- limited.

INDICATIONS

- Complete heart block with slow ventricular escape
- Symptomatic sinus bradycardia, asystole, or prolonged sinus pauses

- Acute anterior myocardial infarction with complete heart block, Mobitz type II AV block, or new bifascicular block
- Selected tachyarrhythmias such as bradycardia-induced or drug induced torsades de pointes, atrial flutter, or recurrent sustained VT
- Malfunction of implanted pacemaker

TEMPORARY PACING CATHETER

The most commonly used pacing catheter is a 6F bipolar catheter, which is relatively stiff and requires manipulation under fluoroscopy. Once properly positioned, it tends to be quite stable. Bipolar catheters have two wires terminating in two electrodes located 1 to 2 cm apart at the tip of the catheter. The distal stimulating electrode is negative and the proximal electrode is positive. Unipolar catheters have one wire terminating in a distal electrode (cathode), which comes in direct contact with myocardium.

PROCEDURE

Under fluoroscopic guidance, advance the pacing catheter to the right atrium, then through the tricuspid valve and into the right ventricle. Then advance catheter into the right ventricular apex. After achieving a satisfactory right ventricular position, connect the pacing catheter, using a sterile connecting cable, to the external pulse generator. Test the pacing threshold by turning on the pulse generator at a moderately high output and at a rate higher than the patient's intrinsic rate. After satisfactory capture is achieved, gradually reduce the output until capture is lost. This milliampere level is the patient's pacing threshold. This is less than 1mA. Repeat the step to be sure that the threshold is consistent. The output should be set at 3 to 4 times this level for consistent capture. Set the pacing rate usually 70 to 80 bpm, although higher or lower rates may be preferred in certain circumstances.

During insertion of the pacing lead, atrial or ventricular arrhythmias may as a result of excessive catheter manipulations. Failure to capture may often be corrected by increasing the output of the pacemaker. Failure to sense can often be corrected by adjusting the sensitivity of the unit. If these manuevers are not sufficient, then the pacing catheter requires repositioning for failure to pace. Cover the insertion site with antibiotic ointment and sterile dressing. Asses pacemaker function again.

COMPLICATIONS IN INTERVENTIONAL PROCEDURES

HEMATOMA

Bleeding after cardiac catheterization may manifest as a hematoma. Clinically, hematomas present as pain or focal discomfort, discoloration, and bruising, and rarely present as femoral nerve compression with resultant quadriceps weakness. Hemostasis should be achieved with manual pressure or a mechanical compression device before leaving the patient's bedside

RETROPERITONEAL HEMATOMA

A retroperitoneal hematoma is usually associated with arterial puncture above the inguinal ligament. Multiple punctures, and puncture through the posterior wall of the artery, will dramatically increase the risk of bleeding. Since all the bleeding may be internal, the patient often present with unexplained hypotension and tachycardia. While flank pain and bruising may be seen in some patients, an unexplained fall in hematocrit may be the only finding in others.

PSEUDOANEURYSM

A pseudoaneurysm is defined as an arterial wall disruption with resultant extra luminal flow into a chamber contained by adjacent tissue. Arterial endothelium does not comprise the interior of the pseudoaneurysm. The incidence of pseudoaneurysm is between 0.3% to 0.5% of cardiac catheterization. A pseudoaneurysm may manifest as pain, a bruit, a pulsatile mass, an expanding hematoma, and leg weakness. The majority arise from the common femoral artery. Risk factors include multiple arterial punctures, superficial femoral artery puncture, larger sheath size, hypertension, and the use of antithrombotic therapy.

ARTERIOVENOUS FISTULA

Arteriovenous fistulas develop when the needle tract crosses both the femoral artery and vein and is then dilated during sheath insertion. Risk factors include an arterial puncture below the common femoral artery, larger arterial sheath size, older age and prolonged anticoagulation or fibronolytic therapy. An arteriovenous fistula is clinically characterized by a continuous bruit at the site of catheter insertion. In addition an expanding groin hematoma, decreased or absent lower extremity pulses and a pulsatile mass in the groin may be appreciated. Diagnosis of an AVF confirmed by Doppler ultrasound.

CARDIAC TAMPONADE

Cardiac tamponade may occur after temporary pacemaker placement with an unsuspected perforation of the right ventricle in an anticoagulated patient, or may occur rapidly if there is coronary perforation from guide wire, balloon catheter, or new device manipulation. Tamponade must be identified in patients undergoing coronary angioplasty as an unsuspected cause of hypotension.

DEATH

Death as a complication of diagnostic catheterization has declined progressively over the last 30 years. There has been a progressive reduction in the overall mortality of diagnostic cardiac catheterization; patients with severe left main coronary disease remain at increased risk. Patients have significant left main disease should always begin with careful catheter entry into the left coronary ostium to facilitate early recognition of ostial left main diseases through catheter pressure damping or performance of immediate test. Patients with cardiogenic shock in the setting of acute myocardial infarction or severe chronic left ventricular dysfunction also have a several fold increased risk of procedural mortality.

Interventional procedures tend to carry higher mortality than purely diagnostic catheterizations because they involve the use of more aggressive catheters, super selective cannulation of diseased coronary arteries and brief interruption of coronary or even systemic flow.

CEREBROVASCULAR COMPLICATIONS

Cerebrovascular accidents are uncommon but potentially devastating complications of diagnostic cardiac catheterization. Every invasive cardiologist should be familiar with potential etiologies, preventive strategies, and treatment for catheterization related stroke, and should develop the routine habit of speaking with the patients directly at the end of the procedure. If patient is less alert, has slurred speech, and either visual, sensory, or motor symptoms during or after left heart procedure, there should be a low threshold for performing a screening neurologic exam or obtaining an urgent stroke neurologic consultation. The risk of stroke is somewhat higher with coronary interventions, as expected based on the use of guiding catheters, multiple equipment's exchange in the aortic root, aggressive anticoagulation, and longer procedure times. Although cerebral hemorrhage must always be excluded, the main cause of catheterization related strokes seem to be embolic. So beyond paying careful attention to flushing and injection technique, there can be no excuse, however for contributory technical malfeasance such as sloppy catheter flushing, introduction of air bubbles during contrast injection. In addition to aortic root sources, embolic material may also originate in the cardiac chambers, thrombotic coronary arteries, or the surface of cardiac valves. One should thus avoid placing the pigtail catheter fully out to the left ventricular apex in patients with suspected aneurism or recent myocardial infarction, since either condition may be associated with potentially dislodge able mural thrombus.

ARRHYTHMIAS

Various cardiac arrhythmias (tachycardia or bradycardia) or conduction disturbance may occur during the course of diagnostic or therapeutic cardiac catheterization. Most, likely VPCs during catheter entry into the right or left ventricle are devoid of clinical consequence. Others, like asystole or ventricular fibrillation, pose immediate risk. Finally some rhythm disturbances are well tolerated in most patients.

VENTRICULAR ARRHYTHMIAS

Ventricular ectopy or even brief runs of ventricular tachycardia are not uncommon during passage of catheters into the right or left ventricle. Even balloon flotation right heart catheterization may cause such brief runs to ventricular tachycardia or ventricular fibrillation. Ventricular tachycardia and ventricular fibrillation may result from catheter manipulation, the most common is intracoronary injection into the right coronary artery, it can still occur if the contrast injection prolonged or performed with a partially damped catheter pressure.

ATRIAL ARRHYTHMIAS

Atrial extra systoles are common during catheter advancement from the right atrium to the superior vena cava, or during looping of the catheter in the right atrium to facilitate passage in a patient with enlargement of the right sided heart chambers. These extra systoles usually subside once the catheter is repositioned, but they may progress to atrial flutter or atrial fibrillation. Atrial flutter or fibrillation are generally benign during catheterization, but may cause clinical sequelae if the ventricular response is rapid or if the atrial kick causes hypotension in a patient with mitral stenosis, hypertrophic cardiomyopathy, or diastolic left ventricular dysfunction.

BRADYARRHYTHMIAS

Transient slowing of the heart rate occurs commonly during coronary angiography, particularly, at the end of a right coronary artery injection performed using a high osmolar ionic contrast agent. Vasovagal reactions, in which bradycardia is associated with hypotension, nausea, yawning, and sweating, should be suspected when bradycardia is more prolonged. Conduction disturbances bundle branch block or complete AV block are an uncommon but potentially serious cause of bradycardia during cardiac catheterization. When complete heart block develops, atropine is rarely helpful in the setting of inadequate junctional escape and hemodynamic deterioration, but should be given anyway, since it has few adverse effects.

PERFORATION

Perforation of the cardiac chambers, coronary arteries, or the intrathoracic great vessels is fortunately a rare event in diagnostic catheterization. Right atrial perforation involved trans-septal catheterization. The right ventricle was the most common site for perforation in the remaining diagnostic procedures, related to the use of stiff catheters. When cardiac perforation does occur, it is usually heralded by bradycardia and hypotension owing to vagal stimulation. In the modern interventional laboratory, however the most common cause of temponade is perforation or rupture of a coronary artery. Some perforation, particularly those limited to deep injury into the vessel wall with localized perivascular contrast staining, can simply be observed. In contrast, free perforation may lead to the development of frank tamponade within seconds to minute. Perforation of the great vessels is extremely rare. The aorta is sufficiently elastic to resist perforation, except in the case of weakening by ascending aorta dissection or aneurysm. Aortic puncture may occur, however, during attempted transseptal puncture with too anterior a needle orientation. Ascending aortic dissection can also result from proximal coronary dissection. If the dissection remains localized angiographically, and is confined to the first few centimetres of the aortic root, it can usually be managed medically and will resolve within weeks. Rupture of the pulmonary artery is also rare, but care must be taken not to use stiff tip guidewires in these thinner walled vessels. Perforation of the branch pulmonary arteries has been reported when balloon floatation catheters are inflated while positioned in a distal branch.

INFECTION AND PYROGEN REACTIONS

Cardiac catheterization is an inherently sterile procedure, infection is extremely unusual. Recommended technique includes shaving and cleaning the catheter introduction site with Providone- iodine, use of a nonporous drape, and adequate operator clothing. When performing a repeat procedure within 2 weeks of an initial diagnostic procedure, the controlateral groin should be used since an increased infection rate has been reported with early reuse of the same groin site. Full sterile precautions are also strongly recommended for the femoral approach when the procedure is prolonged, when the sheath will remain in place for any period, when a stent or permanent pacemaker being implanted, or when a vascular graft is punctured.

ALLERGIC AND ANAPHYLACTIOD REACTIONS

Cardiac catheterization may precipitate allergic or anaphylactiod reactions to three materials: (a) local anaesthetic, (b) iodinated contrast agent, or (c) protamine sulphate. True allergies to local anesthetic do occur, but are more common with older ester agents than with newer amide agents. The most common allergic reactions are triggered by iodinated contrast agents, reaction to protamine sulphate, a biologic product derived from salmon eggs, can also occur. Another allergic reaction that should be considered- even though it is rarely seen in the cardiac catheterization laboratory is heparin induced thrombocytopenia.

RENAL DYSFUNCTION

Temporary or permanent renal dysfunction is a serious potential complication of cardiac angiography. The potential mechanism of contrast-induced nephropathy includes vasomotor instability, increased glomerular permeability to protein, direct tubular obstruction. The main defence against contrast induced nephropathy is limitation of total contrast volume. In patients with reduced renal function and especially with diabetes, extra attention must be paid to limiting unnecessary angiographic views and multiple contrast puffs during interventional wire and device placement. Another cause of renal failure following cardiac catheterization is systemic cholesterol embolization.

HYPOTENSION

Reduction in arterial blood pressure is one of the most common problems seen during catheterization. This reduction represents the final common manifestation of variety of conditions including the following: hypovolemia, owing to inadequate prehydration, blood loss, or excessive contrast –induced dieresis, reduction in cardiac output, owing to ischemia, tamponade, arrhythmia, or valvular regurgitation, inappropriate systemic arteriolar vasodilation, owing to vasovagal, excessive nitrate administration or a vasodilator drugs such as dopamine or dobutamine.

VOLUME OVERLOAD

Patients in the cardiac catheterization laboratory are prone to volume overload owing to the administration of hypertonic contrast agents, myocardial depression or ischemia

induced by contrast, poor baseline left ventricular function, as well as their supine position an attempts to volume load patients at risk for contrast induced renal dysfunction.

CORONARY DISSECTION

The most common cause of prolonged ischemia during angioplasty is coronary dissection. Although coronary dissection may be detected by characteristic angiographic imaging, at times, the dissection cannot be differentiated from thrombus formation. Classification of coronary artery dissection followed the national heart blood and lung institute system A-F, where type A is minor radiolucencies within the coronary lumen due to contrast injection with no persistence of dye; type B is a parallel track or double or double lumen impression of radiolucent area during contrast injection with no persistence, type C is extra luminal cap with persistence of contrast after coronary angiography , type D is spiral luminal filling defects in multiple areas of the vessel that persist, and type F is type A-E dissection that lead to impaired flow or abrupt closure.

INTRACORONARY THROMBUS

Presumed intracoronary thrombus is also treated with prolonged balloon inflations and frequently intracoronary thrombolytics. It should be noted , however that routine use of intracoronary thrombolytics has not been found to reduce and may potentially increase, the risk of ischemic events.

CORONARY ARTERY EMBOLISM

Occasionally coronary air embolisms will occur in the performance of procedure catheter exchange, balloon extraction and reinsertion. Air embolism may occur under the following conditions:

- Incomplete aspiration of guiding catheter upon introduction into the circulation
- Balloon leakage or rupture
- Prolonged negative suction of self venting balloon catheters when exposed to room air
- Introduction of balloon catheters into the guide
- Removal of balloon catheters from deeply seated guiding catheter
- Structural failure of catheter
- Injection of air due to bubble in contrast injection line and or syringe
- Vacuum air accumulation on use of tracker exchange system.

GUIDE CATHETER OBSTRUCTION

Ischemia due to guide catheter ostial occlusion is treated by removal of the guide catheter or use of a catheter with side holes. Balloon catheters with relatively larger shaft sizes may create ischemia when they are inserted into relatively smaller arteries. This ischemia persists despite the balloon deflation. The solution is to remove the balloon into the guiding catheter after each inflation.

CORONARY SPASM

Coronary vasospasm occurs frequently doing angioplasty, and intra coronary nitroglycerin can readily reverse vasospastic tendencies. Some patients may require continuous

intravenous nitroglycerin to remove this potentially complicating factor. Coronary vasospasm should be suspected in every case of reduced flow and excluded by the administration of intracoronary nitroglycerin.

CATHETER KINKING

Catheter rotation during a trans radial procedure is much more difficult to perform than via the femoral approach because of the friction generated by the size of the Vessel. Catheter advancement around loops and in calcified segments must be monitored (the tip must rotate with the rest of the catheter!) by x-ray and pressure measurement. Indeed, catheter kinking, which can also occur via the transfemoral approach, may have worse consequences. The catheter must not be withdrawn before being straightened up by rotating it in the opposite direction and by inserting a hydrophilic wire which should be advanced in a larger artery.

INTRA-AORTIC BALLOON COUNTER PULSATION

Intra Aortic Balloon Counter pulsation (IABP)

Intra aortic balloon pump is a mechanical device that increases myocardial oxygen perfusion along with increasing cardiac output, which in turn increases the coronary blood flow and myocardial oxygen delivery.

Indications:

- Cardiogenic shock
- Hemodynamic support during or after PCI.
- Primary angioplasty.
- High risk patients including unprotected LMCA disease, LV dysfunction.
- Prior surgical repair for hemodynamically significant MR or post MI VSD as they are prone to rapid cardiogenic shock.
- Unstable angina.
- Improving hemodynamics and facilitating wearing from cardio pulmonary bypass in CABG cases.
- Bridge to cardiac transplantation.

Absolute contra indication.

- Severe Aortic Regurgitation.
- Aortic dissection.
- Severe aorto iliac occlusive disease.

Relative contra indication

- Prosthetic aortic vascular graft.
- Aortic aneurysm .
- Aorto femoral grafts.

HARDWARE

There are two separate containers which contain all the hardware required for IABP insertion

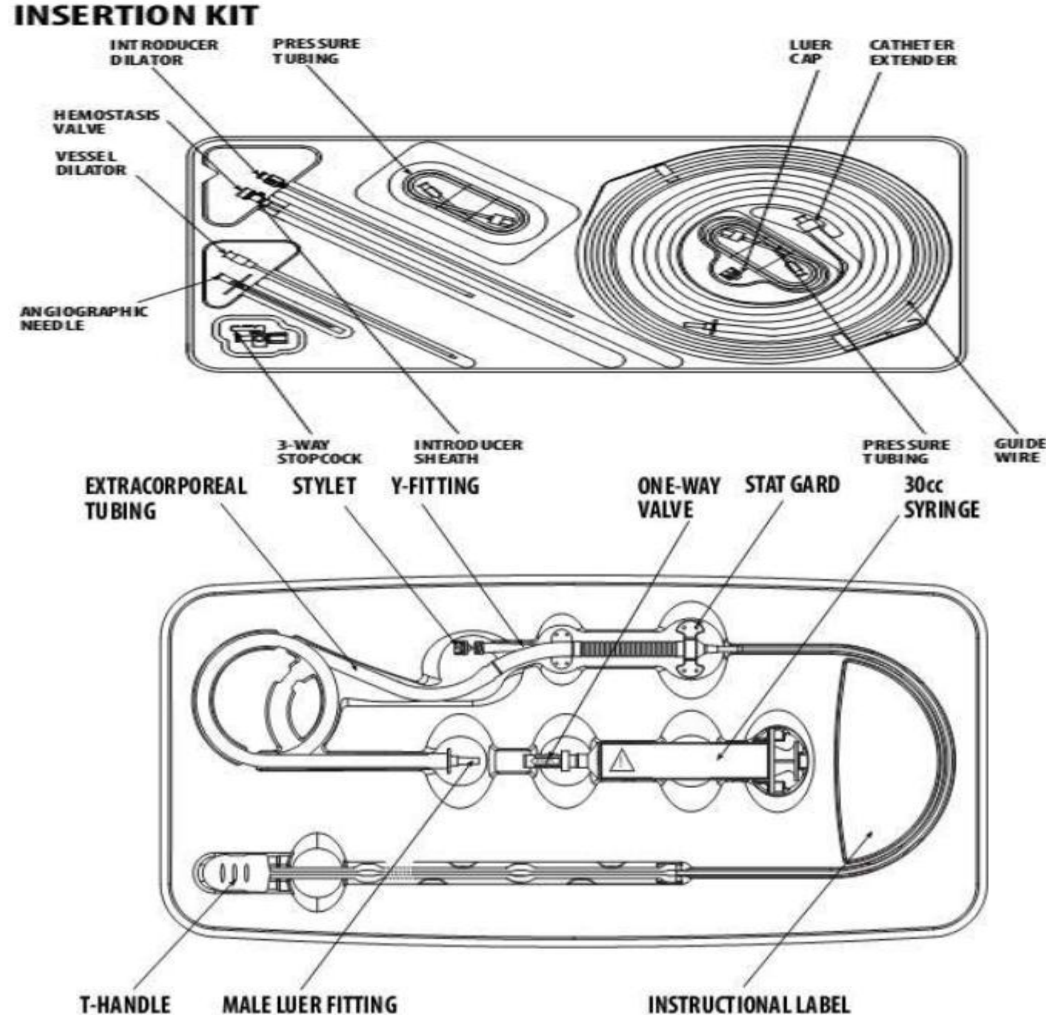
1. **Insertion kit**
   a. It contains all the instrumentation required for arterial access
   b. In also contains the catheter extender and pressure tubing required for connecting IABP to the console.
2. **Intra aortic balloon**

a) This kit contains the balloon with stylet, one way valve and a 30 cc luer lock syringe.

IABP catheter

- ~10-20 cm long polyurethane bladder
- 25cc to 50cc capacity
- Optimal 85% of aorta occluded (not 100%)
- The shaft of the balloon catheter contains 2 lumens: one allows for gas exchange from console to balloon;

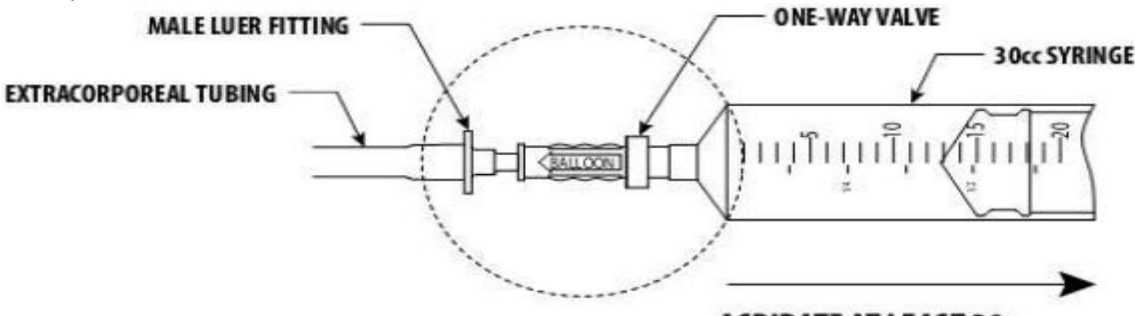
- The second is used for catheter delivery over a guidewire and for monitoring of central aortic pressure after installation



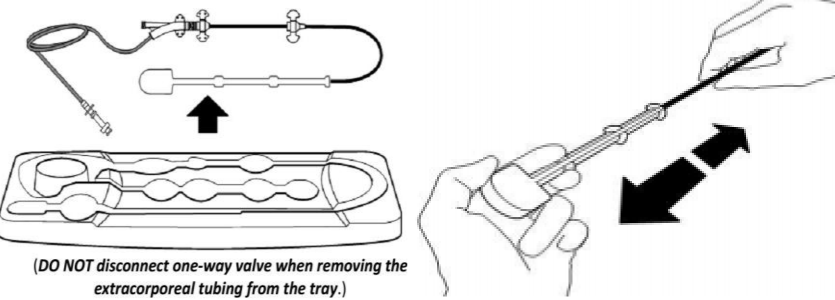
STEP BY STEP (STANDARD FLUOROSCOPIC WITH SHEATH)

1. Connect the IABP to supply line. Check Helium gas status.
2. Connect ECG- to the IABP console. (good ECG – must for trigger).
3. Ensure that appropriate size IABP balloon and IABP insertion kit (supplied in separate boxes) is available.
4. Femoral access – followed by insertion of the supplied sheath (usually 8 or 9.5 French depending on size)
5. Insert the 0.030 inch supplied J-shaped guide wire to the level of the aortic arch
6. Once access is secured and sheath is on place prepare the balloon
7. The shaft of the balloon catheter contains 2 lumens: one allows for gas exchange from console to balloon; the second is used for catheter delivery over a guidewire and for monitoring of central aortic pressure after installation.

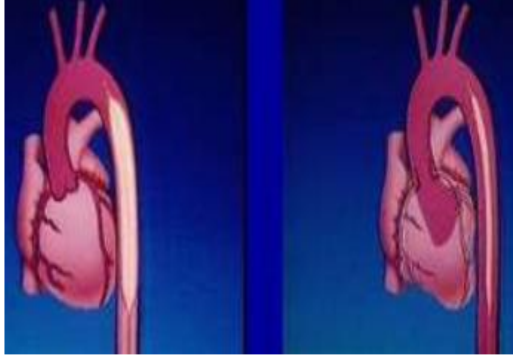
8. Before removing balloon catheter from box, use the supplied 30cc syringe, slowly aspirate at least 30cc.

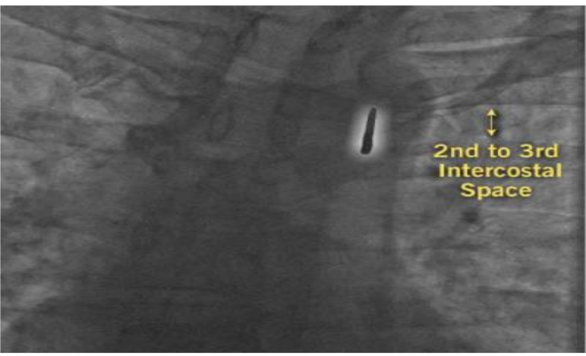


9. Remove the syringe while leaving the one-way valve place.
10. Carefully remove the extracorporeal tubing, Y-fitting and IABP catheter handle from the tray – (do not disconnect one-way valve when removing the extracorporeal tubing from the tray)
11. Do not remove the T-handle or packaging stylet until immediately prior to insertion.
12. Maintain a vacuum on the IAB catheter through insertion. Do not remove the one-way valve.
13. IABP central lumen is flushed with saline
14. Remove the IAB catheter from the T-handle by pulling straight out to avoid damaging it.

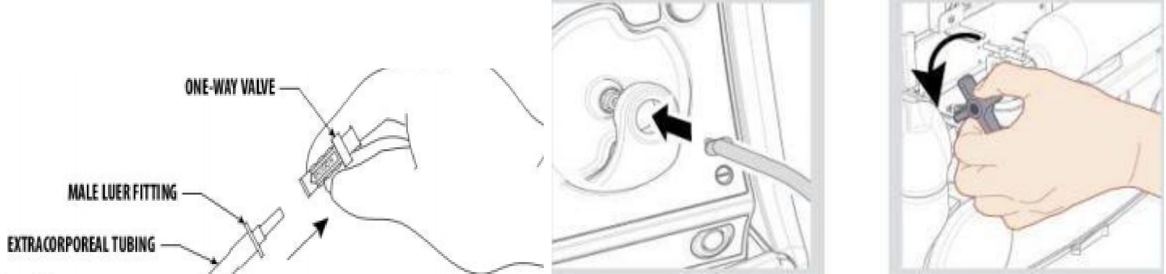


15. Insert a balloon (remove stylet) only over the guidewire.
16. Advance in small steps of 1 to 2 cm at a time and stop if any resistance.
17. The tip of the IABP catheter should lie 1-2 centimetres distal (below) to the origin of the left subclavian artery
18. This corresponds to 2 and 3 rd intercostals space on fluoro.





19. The proximal end of balloon should be above the renal arteries.
20. After IAB insertion has been completed, remove the one-way valve from the male luer of the extracorporeal tubing.



21. Connect to the console via a long extender connector (also supplied) and the system is purged with helium.
22. The central lumen of the catheter is connected to pressure tubing and a pressure transducer to allow for monitoring of central aortic pressure.
23. Heparin should be given as a bolus and continuous infusion to maintain a partial thromboplastin time (PTT) of 60-80 seconds or an activated clotting time (ACT) of 1.5-2.0 times normal.
24. The ECG and pressure tracing should be clear on the IABP console
25. The balloon catheter is secured to the skin by sutures. Although not ideal IABP catheter may also be inserted sheathlessly and without fluoroscopy. If fluoroscopy is not available, measure the tip of the IAB from the sterna angle of Louis to the umbilicus and then obliquely to the femoral insertion site for catheter insertion length. X-ray chest must be taken immediately to confirm balloon position.

Initial set-up

1) Once connected properly the console would show ECG and pressure waveforms.
2) Elevate flush bag at least 3' (91.44 cm) above transducer
3) A 3 cc/hour continuous flow through inner lumen is recommended
4) By default the system will be in standby mode. Start pumping by pressing START on the console (automatic mode).

Pressing the START key

- Automatically purges and fills IAB
- Automatically select most appropriate lead and trigger
- Automatically sets inflation and deflation timing

Auto operation mode

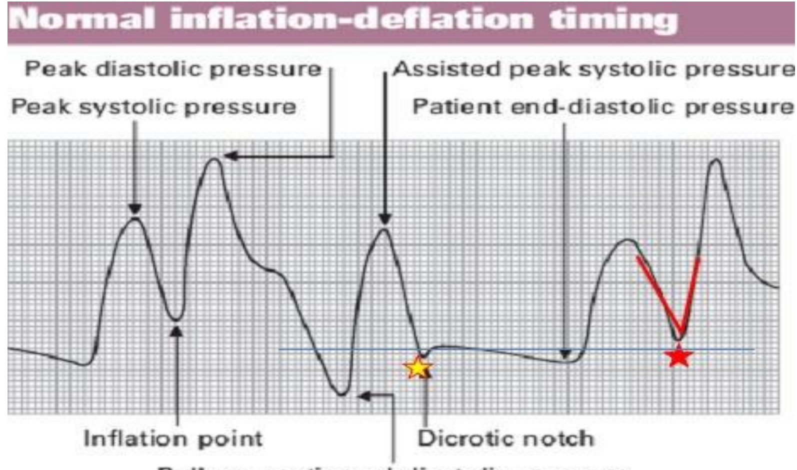
- Automatic lead and trigger selection
- Automatic and continuous inflation and deflation timing management – user has ability to fine-tune deflation timing.
- Automatic management of irregular rhythms

Triggering and timing

- Correct timing of balloon inflation and deflation during the cardiac cycle is vital to ensure optimal effects of counter pulsation whilst minimizing potentially harmful effects related to mistiming.
- Most commonly (and by default) the ECG waveform is used to trigger balloon inflation and deflation.
- The arterial pressure waveform is an alternative technique that may be useful if either the ECG trace is poor or there are cardiac arrhythmias.
- Modern machines allow either method to be easily selected.
- The balloon starts to inflate at the onset of diastole. This corresponds to the middle of T wave on the ECG waveform and the dicrotic notch of the arterial pressure trace.
- As aortic valve closure has occurred balloon inflation causes a sharp upstroke on the arterial pressure waveform followed by a tall peak which represents the assisted diastolic pressure.
- Deflation occurs at the onset of systole immediately before opening of the aortic valve. This corresponds with the peak of the R wave on the ECG trace and the point just before the upstroke of systole on the arterial pressure trace.
- As the balloon deflates, the assisted aortic end-diastolic pressure dips down to create the second deep wave, usually U shaped on the arterial pressure waveform

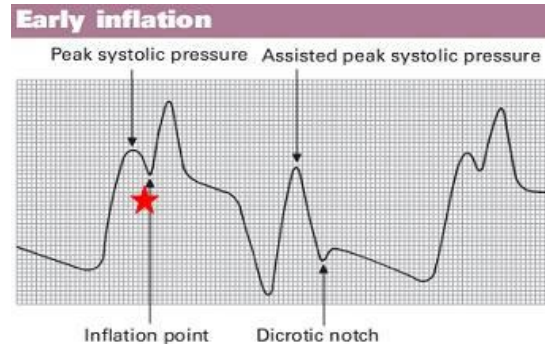
Troubleshoot – timing

Analyse the wave form periodically to check if IABP is working well. The following points on the waveform are noted.



A: Unassisted End Diastolic pressure
B: Unassisted Systole
C: IAB Inflation
D: Diastolic Augmentation

E: Assisted End Diastolic Pressure
F: Assisted Systole



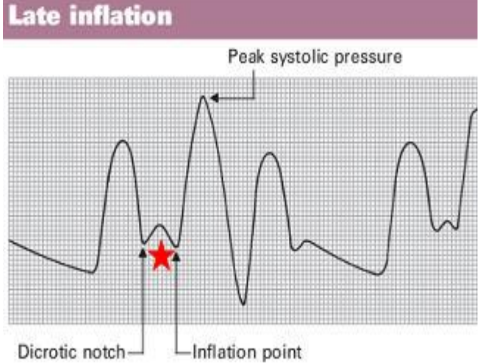
Timing error – early inflation of IAB (prior to aortic valve closure)

Waveform characteristics:

- Inflation IAB to dicrotic notch
- Diastolic augmentation encroaches onto systole (may be unable to distinguish).

Physiologic effects:

- Potential premature closure of aortic valve
- Potential increase in LVEDV/LVEDP/PCWP
- Increased left ventricular wall stress or after load
- Aortic regurgitation
- Increased MVO_2 demand



Timing error – late inflation (inflation of IAB markedly after closure of aortic valve)

Waveform characteristics:

- Inflation of IAB after dicrotic notch
- Absence of sharp "V"
- Sub-optimal diastolic

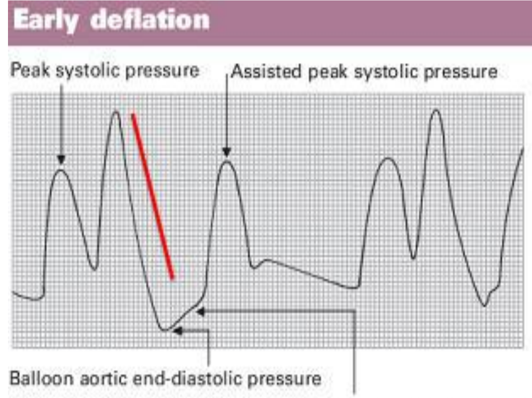
Physiologic effects:

- Sub-optimal coronary artery perfusion

Timing error – early deflation (premature deflation of IAB during diastolic phase)

Waveform characteristics:

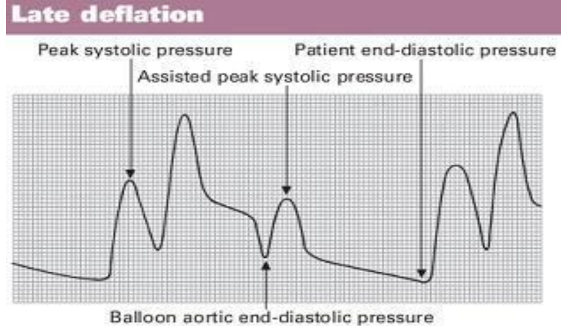
- Deflation of IAB is seen as a sharp drop following diastolic augmentation
- Assisted end diastolic pressure may be equal to or less than unassisted end diastolic pressure
- Sub-optimal diastolic augmentation
- Assisted systolic pressure may rise



Physiologic effects:

- Sub-optimal coronary perfusion
- Potential for retrograde coronary and carotid blood flow, which may result in angina
- Sub-optimal after load reduction
- Increased MVO_2 demand

Timing error – late deflation (deflation of IAB after aortic valve has opened)



Waveform characteristics:

- Assisted end diastolic pressure may be equal to or higher than unassisted End diastolic pressure
- Rate of rise assisted systole is prolonged
- Diastolic augmentation may appear wide

Physiologic effect:

- Afterload reduction is essentially absent
- Increased MVO_2 consumption due to left ventricle ejecting against a greater resistance and a prolonged isovolumetric contraction phase
- IAB may impede left ventricular ejection and increase after load

Complications

- Vascular limb ischemia, hematoma around the access site, bleeding.
- Infections
- Balloon rupture – due to calcified aortic plaque.
- Helium gas embolization.
- Inappropriate use of adequate size balloons.
- Hemolytic anemia and thrombocytopenia.

Mechanism

Computer controlled mechanism of inflating a cylindrical polyethylene balloon, that is placed 1cm below the left subclavian artery and 2cm above the carina, during diastole. The machine is linked to the ECG ang pressure transducer.

Helium is used to inflate the IABP balloon as it is to a low viscous gas, providing quick to and fro motion of gas through the long connecting tubes from console to the balloon and ahs a lower risk of embolism than air if the balloon ruptures.

Appropriate sizing of the balloon is selected on the basis of height of the patient.

- 34cc- 152-163cm
- 40cc-164-183cm
- 50cc-height > 183cm.

The diameter of the balloon should not exceed 80-90% of the diameter of descending aorta.

The IABP balloon catheter is inserted percutaneously into the femoral artery through an introducer sheath using modified Seldinger technique in the cardiac cath lab.

The outer lumen of the catheter is used for delivery of helium gas to the balloon and inner lumen can be used for monitoring pressures.

The console is programmed to identify a trigger of balloon inflation and deflation. Commonly used are ECG and systemic arterial pressure waveform. The balloon inflates with the onset of diastole and deflates at the onset of LV systole. The balloon is set to inflate after the aortic valve closure(corresponds to dichrotic notch in the aortic waveform) and deflate before the upstroke on arterial pressure waveform and thus the diastolic augmentation of pressure is achieved. With early or late deflation and inflation, proper augmentation of the pressure a cannot be achieved.

Removing IABP catheter.

Weaning is achieving when the iotropic requirements are minimal, gradually reducing the ratio of augmented to non augmented beats from 1:1 to 1:2 or less.

CARDIAC EMERGENCY DRUG

DRUG	DOSE AND DELIVERY	ACTION
Anticoagulant		
Unfractioned heparin (UFH)	50-70 iu/kg IV, followed by small boluses to keep ACT>250 sec or >200sec if a Gp IIb/IIa inhibitor is given	Most commonly used thrombin inhibitor Dependant on antithrombin III Stop original clot from expanding and prevent additional clots from forming Small biovailability
Low molecular weight heparin (LWMH) (Enoxaparin)	1 mg/kg subcutaneously	Combined inhibition of factor IIa and Xa Increased anti-factor Xa activity High biovailability More consistent anticoagulation
Bivalirudin (Angiomax	IV bolus of 0.75 mg/kg followed by an infusion of 1.75 mg/kg/ hour.	Direct thrombin inhibitor Does not depend on antithrombin Can inhibit clot bound thrombin Stable anticoagulation level Short acting Dose not cause thrombocytopenia

		Increased incidence of acute stent Thrombosis
Antiplatelet Agents		
Aspirin	160-325 mg tablet	Slows platelet aggregation, reduces further arterial occlusion or reocclusion, and reduces chance of recurrence
II b/IIIa inhibitors		
Tirofiban (Aggrastat)	Acute coronary syndrome 0.4 mcg/kg per min IV for 30 min,then 0.1 mcg/kg per min IV infusion	Antiplatelet drug Tirofiban is indicated to reduce the rate of thrombotic cardiovascular events (combined endpoint of death, myocardial infarction, or refractory ischemia/repeat cardiac procedure) in patients with non-ST elevation acute coronary syndrome (NSTEACS).
Arrhythmia and Bradycardia		
Atropine sulfate	0.5-1.0 mg IV every 3-5 min as	For vasovagal or symptomatic sinus

	needed	bradycardia Increases the heart rate through anticholinergic effect
Atrial fibrillation or flutter		
Dofetlide	Single infusion of 8mcg/kg over 30 min	For atrial fibrillation and flutter
Supraventricular tachycardia		
Adenosine	6 mg by rapid I.V. push, followed by 1 0 to 20mL of normal saline solution (NSS) flush; may repeat a 1 2-mg dose twice, followed by NSS flush	Depresses the sinoatrial and atrioventricular node activity, slowing the heart rate
Ventricular		
Amidarone	1 50-mg bolus given I.V. over 1 0 minutes, followed by continuous I.V. infusion at 1mg/min for 6 hours, then 0.5mg/minute for	For ventricular fibrillation

	1 8 hours	
Beta-blockers		
Atenolol, Metoprolol	5 mg IV bolus over 5 minutes; may repeat Atenolol dose once and Metoprolol dose twice	Reduce catecholamins leading to slower heart rate and lower blood pressure
Calcium channel blocker		
Diltiazem	5 to 20 mg by I.V. push over 2 to 5 minutes, followed by I.V. infusion or additional 20 to 25 mg by I.V. push after 1 5 minutes	Lengthens cardiac cycle, slowing the heart rate
Verapamil	2.5-5.0 mg IV bolus over 2 min. Second dose: 5 mg bolus every 15 min to total dose of 30 mg	Slowing the heart rate
Conscious sedation		
Fenatanyl	25-50 mcg intravenously.	Monitor vital sign , oximetry, and

	Repeat as needed every 5 min	state of consciousness as per conscious sedation guidelines
Morphine sulphate	Morphine sulphate	Monitor vital sign , oximetry, and state of consciousness as per conscious sedation guidelines Reduce ventricular preload and cardiac O_2 requirements
Contrast nephropathy		
Hydration	Normal saline 1 ml/kg per hr for 12 hr pre and 12 hr post contrast exposure Alternative normal saline 3 mg/kg over 1 hr preprocedure, then 1 ml/kg per hr 6 hr postprocedure Alternatively sodium bicarbonate (154mEq/L) in	For prevention of contrast induced nephropathy

	D5W	
Diuretic		
Furosemide (Lasix)	IV infusion: 0.5-1.0 mg/kg given over 1 to 2 min If no response,double dose to 2.0 mg/kg, slowly over 1 to 2 min	It is a loop diuretic (water pill) that prevents your body from absorbing too much salt, allowing the salt to instead be passed in your urine.
Inotrope		
Dobutamine (Dobutrex	IV infusion: Dilute 500 mg (20ml) in 250 ml D5W Usual infusion rate is 2-20 mcg/kg per min	It works by increasing the strength and force of the heartbeat, causing more blood to circulate through the body
Dopamine	5 to 1 5 mcg/kg/minute by IV infusion	Stimulates dopamine receptors and increase cardiac output, with increase in minimal oxygen consumption; causes peripheral vasoconstriction

Digoxin	IV infusion: 0.25 mg/ml or 0.1 mg/ml supplied in 1 or 2 ml ampule Loading dose of 10-15 mcg/kg lean body weight – therapeutic effect with minimum toxicity	For rate control in atrial fibrillation and flutter. Maintenance dose is affected by body size and renal function
Pressure agents		
Vasopressine	Dose for cardiac arrest: 40 U IV push *1 Wait for 10 minutes before initiating epinephrine protocol For refractory hypotension: 20 U in 250 ml D5W. Infuse at 0.01-0.10U/min	For cardiac arrest (option to epinephrine) Vasopressin helps prevent loss of water from the body by reducing urine output and helping the kidneys reabsorb water into the body. Vasopressin also raises blood pressure by narrowing blood vessels

Coronary		
Nitoglycerine	Dilute 200 mcg/ml Administer 100-200 mcg intracoronary	Epicardial vasodilation or treatment of coronary spasm
Adenosine	Dilute 10 mcg/ml For RCA 18-24 mcg through guiding catheter or selectively into distal coronary For LCA 24-36 mcg through guiding catheter or selectively into distal coronary Alternatively, 140-180 mcg /kg per min peripheral intravenous infusion for 3 min	For measurement of fractional flow reserve (FFR

DEPARTMENTAL EQUIPMENTS

PHILIPS ALLURA XPER FD 10

The Allura Xper FD10/10 combines exceptional speed and performance of the geometry with superb fl at detector image quality. Whether your focus is Interventional Cardiology, Paediatric Cardiology or Electrophysiology (EP), Philips has developed special features and protocols for the Allura Xper FD10/10. Bi-plane viewing power and safety The Allura Xper FD10/10 brings fl at detector technology to biplane viewing. This system delivers superb image quality in both the frontal and lateral plane, enabling cardiologists to view them side-by-side. The Allura XperFD10/10 saves valuable time when capturing accurate 3Dinformation while also reducing x-ray dose and contrast medium.

• **DYNAMIC FLAT DETECTOR:** Philips' 14-bit virtually distortion-free dynamic flat detector offers 184 micron pixels for higher resolution and a DQE(O) of 75% that provides better image quality, especially for low dose fluoroscopy. The compact design with a very large field of view of 25 cm (10 in.) is the optimal size for dedicated cardiology and EP applications. It also offers a refresh light that provides temporal virtually artefact-free imaging by "blanking" the detector, thereby eliminating image glow during dynamic studies.

• **G SHAPED GANTRY:** The compact, motorized, floor-mounted G-arm provides excellent patient accessibility from all sides. The large diameter of the G-arm allows virtually all cardiac Projections, even with obese patients. Two projection scan be stored and recalled for faster positioning.

• **XPER TABLE:** The Xper table is a dedicated cardiovascular table with a free floating tabletop. This table has very high patient loadability and can make a large longitudinal floating movement.

• **PAN HANDLE**: The PAN handle is a tabletop float control extension, which can be attached to any side of the table.

• **ACQUISITION:** The acquisition page contains information on the current selected patient. The page shows a full overview of all acquired runs and allows you to do QA. The history file of the patient can be reviewed at the touch of a button.

• **ARCHIVE:** Clinical studies can be transferred to an optional Xcelera DICOM Recorder (IDR) or to a PACS, like the Xcelera PACS. The archive process - including multiple destinations, archive formats, and background transfer can be completely automated

• **DSA:** The Digital Subtraction Angiography (DSA) option extends the vascular applicational functionality of the Allura Xper system. DSA features real-time digital subtraction at low frame speeds of 0.5, 1, 2, 3, or 6 frames per second. The DSA programs can be selected via the Xper settings.

• **FLUOROSCOPY:** Three fluoro modes are available at table side and these can be programmed via Xper settings. Each mode can be programmed with different composition of X-ray dose rate, digital processing, and filter settings.

• **X GENERATOR:** X-ray generation consists of the following elements: Xray generator, X-ray tube, collimator (including Spectra Beam, Beam filtration), and dose protection mechanism. The complete dose protection mechanism is part of the Dose Wise program.

• **MONITORS:** The system is delivered standard with four black and white 18- inch LCD monitors in the examination room. One 19-inch LCD color monitor and two 18-inch black and white LCD monitors are standard in the control room.

• **STENT BOOST:** Stent Boost is a simple, quick, and cost-effective tool to enhance visualization of stents in the coronary arteries. It is the superimposition of multiple digital images uses markers on the balloon or stent delivery to better visualize objects in the direct environment of the markers.

PHILIPS INTEGRIS H 5000 F

The floor mounted monoplane integris H 5000 F is the dedicated system of cardiac procedures. It brings excellent digital imaging performance to cardiac suit. The larger diameter of the polydiagnost G stand and patient sensing of bodyguard allow high rotation and angulations speeds of up 25 degree per sec. 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.

X RAY GENERATORS

Microprocessors controlled 100 kW high frequency converter generator
Voltage range: 40 kV to 150 kV
Max current : 1000mA at 100kV, 800 mA at 125 kV
X ray tube : MRC 0508
Power : 0.5/0.8, 45/85kW
Anode heat storage capacity: 2400kHU anode
Continues heat dissipation : 5300W
TV chain XTV 60, CCD camera with proprietary digital output
Light intensity : 30000 lux
Focusable light field size : 14 – 25 cm
Digital acquisition
Frame speed (frame per second) at 512*512 image resolution.

SIEMENS AXIOM ARTIS CATHLAB SPECIFICATIONS

FEATURES

- Floor-mounted C-arm stand with flexible positioning options and fast programmable movements
- Compact and slim line C-arm design
- Dyna vision rotational angio feasibility

- Catheter table with free-floating tabletop, optionally with tilt and motorized table stepping
- In the examination room: Complete ergonomic system control in the examination room with integrated generator and imaging system control on a color touch-screen interface
- syngo operation in the control room using mouse and keyboard, and color or black and white flat display monitors
- Full-size CCD camera system
- Pulsed fluoroscopy is standard on all basic systems
- Expanded CARE applications for maximum radiation protection
- Full DICOM functionality for patient data acquisition as well as for documentation and archiving
- Integration of Siemens cath lab information and recording system
- Axiom senses hemodynamic system
- High-frequency generator with automatic dose-rate control
- Choice of dual or triple-focus high-performance liquid-bearing or ball bearing **X-ray tube**
- Single joystick for all patient-angle oriented C-arm and I.I. movements

IMAGE PROCESSING:

- Modes range from single frame to series display with frame rates from 0.5 up to 60 frames/second.
- Pulsed fluoroscopy standard
- Integration with CATHCOR LX cath lab information and recording system.
- Storing for fluoroscopic images also during fluoroscopy.

MONITORS:

- TWO ceiling suspended: high resolution digital CCD.

GENERATOR:

- High frequency with integrated, fully automatic dose rate control.
- 100 kW at 100 kV
- SID tracking

TABLE TYPE:

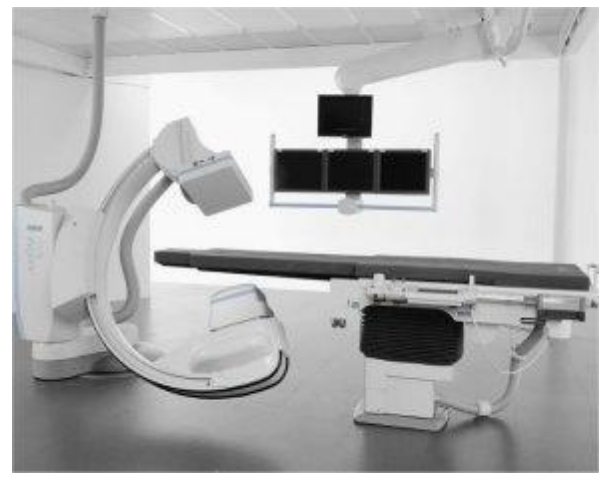
- Carbon fiber floor-mounted catheter table with free-moving tabletop; no tilt.
- Narrow with recess at head end.
- Rotation +/- 120 degrees.
- Maximum patient weight 100kg.

OTHER FEATURES

- Integration with CATHCOR LX cath lab information and recording system.
- CD recorder.
- DICOM print.
- UPS for imaging system.
- Intercom.

BARD EP PRO

The lab system Pro EP recording System is performance- driven software has deliver increased flexibility for data review and export. Patient review Mode provides significant improvements to work flow management by allowing the physician or nurse the clinical lab system PRO EP recording system as a workstation for case review, study annotation, and report generation. New CLEARSIGN Amplifier delivers excellent signal quality with unique adaptive noise filtering and up to 160 intracardiac channels. The new report generator seamlessly creates an informative patient record by automatically capturing user- defined study log information into a customizable template. Waveform and fluoroscopy images can also be included to deliver the most complete picture of the patient's diagnosis and treatment. New advanced patient data archiving offers increased archiving confidence with improved status updates and data verification.



The lab system PRO review workstation delivers EP study data. The EP Review Workstations puts the capabilities of the recording system at your finger tips and allows access to your EP study data from any location, in or out of the lab. New CLEARSIGN Amplifier delivers excellent signal quality and intuitive design for improved efficiency and accuracy during your EP procedure.

CS300 IABP WITH INTELLISENSE

The New CS300 with IntelliSense combines fibre-optic speed with automatic in vivo calibration. The result is faster time to therapy, faster signal acquisition, and faster adaptation to rate and rhythm changes.

Faster Time to Therapy

- Faster inflation and deflation speed
- CS300 has true one-button start-up
- Automatically calibrates the fibre-optic pressure sensor in the patient and recalibrates every 2 hours or sooner should patient or environmental conditions change

- Automatically evaluates and selects the best lead and trigger source
- Automatically sets optimal timing
- Automatically adjusts to changes in patient conditions without clinician intervention

The CS300 Can Be Used With:

- The SENSATION 7Fr. fibre-optic IAB catheter
- Conventional fluid-filled IAB catheters
- Pediatric IAB catheters

Fibre-optic pressure signal transmission to a patient monitor: An electrically isolated, low-level pressure output from the CS300 enables clinicians to send the fibre-optic arterial pressure waveform directly to a patient monitor by simply attaching an appropriate cable. Unique automatic in vivo calibration: MAQUET has the ONLY fibre-optic IABP and catheter system that automatically calibrates in the patient after insertion and automatically recalibrates in vivo every 2 hours or sooner should patient or environmental conditions change.

ST. JUDE ILUMIEN OCT SYSTEM

The basic principles of OCT consist of a single mode optic fiber that transmits and receives infrared light waves at a bandwidth of 1250-1350 nm which is the ideal spectral range for imaging biologic tissue. The OCT started as a research tool it is graduating to become an important clinical tool particularly to view the edoluminal structure of the blood vessels. The present OCT system that is available in India is from St Jude Medical.

ILUMIEN Therapy Guidance System is intended for the imaging of coronary arteries and is indicated in patients who are candidates for transluminal interventional procedures. ILUMIEN System will further acquire radio frequency signal outputs from both a distal intracoronary pressure transducer and a proximal aortic pressure transducer to determine the physiological parameter, Fractional Flow Reserve (FFR). The physician may use the FFR parameter, along with knowledge of patient history, medical expertise and clinical judgment to determine if therapeutic intervention is indicated.

PRODUCT BENEFITS

- Combine the functional and anatomical modalities of Fractional Flow Reserve (FFR) and Optical Coherence Tomography (OCT)
- Move between FFR and OCT modalities with one touch
- Wireless FFR setup in one or multiple labs
- Easy on-screen guide simplifies Ilumien FFR and OCT procedures with a new seamless user interface
- The flexibility and cost efficiency of a mobile unit
- Integrated case storage and easy patient ID retrieval.
- Ability to perform FFR measurements with optional wireless Wi-Fi Box™, PressureWire Aeris and cables

Figure 1-1: ILUMIEN System - Physician Side

GENERAL PRODUCT INFORMATION

The compact, mobile console contains all the working elements of the Ilumien Therapy Guidance System, including:

- Ilumien proprietary imaging engine, custom PC software and wireless receivers to perform FFR measurements.
- Two monitors (17” and 19”) plus remote video output for multiple sightlines
- Keyboard or mouse click control of system and catheter
- 22x CD/DVD±RW dual layer DVD-RAM drive for faster image management
- Large hard drive for ample data storage
- Integrated drive-motor and optical controller (DOC)

TECHNICAL SPECIFICATION

- **Ilumien:** Size mm/in: 1430 (h) x 471 (w) x 683 (d)/56.3 (h) x 18.5 (w) x 27 (d)
- **Dragonfly** Usable length: 135 cm ,Outer diameter: 2.7 F (distal), Wire lumen: 0.014”
- **PressureWire Aeris:** Operating pressure: -30 to +300mmHg Accuracy: ±1 mmHg plus ±1% of reading (-30 to 50mmHg) ±3% of reading (50 to300mmHg).
- **AO pressure via Wi-Fi Box to Ilumien:** Operating pressure: -30 to +300mmHg
- Accuracy: ±1 mmHg or ±1% of reading whichever greatest **AO pressure via Wi-Fi Box to hemodynamic recording system:** Direct galvanic connection Max Pressure shift: <2mmHg
- **Radio specification:** Frequency range: 2.4000-2.4835GHz Type: Frequency hopping spread spectrum (FHSS) Range: 0-4m Delay time: <20ms

IMAGING PARAMETRES

- Maximum frame rate: 100 fps
- Nominal pullback speed: 20 mm/sec
- Scan diameter: 10 mm
- Axial resolution: 15 µm

IVUS – VOLCANO

Volcano Corporation develops, designed to facilitate endovascular procedures, and enhances the diagnosis of vascular and structural heart disease and guide optimal therapies. The intravascular ultrasound (IVUS) product line includes ultrasound consoles that can be integrated directly into virtually and modern cath lab single use disposable imaging catheters unique to system. IVUS offerings are used by clinicians to measure the stage and severity of disease present in cardiac and peripheral vessels. IVUS is also used post-stent placement procedures to confirm adequate expansion of the stent and full opposition to the vessel wall. Volcano IVUS offers unique features, including both phased array and rotational IVUS imaging catheters and advanced functionally options, such as IVUS tissue characterization technology.

FEATURES ARE:

- Automated border rendering: automatically generate borders and measurements in grayscale or VH mode to highlight lumen and vessel boundaries
- Advanced border editing tools: additional border editing tools allow for free draw adjustments or placements of dots to mark desired border location
- Automatic Measurements: automatically calculated key documentation endpoints including MLD, CSA, and plaque burden
- VH IVUS: proprietary plaque composition technology can be seen on cross section and longitudinal views
- Target assist: quickly identifies IVUS frame with most severe narrowing and automatically displays lesion length
- Rapid Review: cine animation of consecutive static frames for improved image interpretation, newly enhanced to allow concurrent boarder rendering and measurement during playback
- Chroma Flow: proprietary blood motion technology highlights flow in orange to assist in conforming proper stent apposition and stent CSA
- Digital Archiving & DICOM Service: supports offline archiving to local hard drive, to DVD and to DICOM network via DICOM store. Also supports DICOM work list management
- Expanded settings options Powerful new settings options allows you to customize your configurations to accelerate and simplify your network.

QUANTIEN WIRELESS FFR

The QUANTIEN integrated FFR solution gives you a wireless patient environment, removing the clutter of cables and clearing the table for what’s most important the Patient ID is quickly loaded from your hospital scheduling system, and the results are exported back to your archive, all with a minimum of manual interaction. Place the QUANTIEN main unit anywhere in the lab, freeing up valuable table space and allowing control by either sterile or nonsterile-dressed staff. Regardless of where you chose to place QUANTIEN, its video screen can be displayed on existing lab monitors for convenient viewing right next to your angiography and hemodynamic screens. Integrating QUANTIEN into your cath lab environment is quick and easy — no need to install cables through the floor or ceiling. QUANTIEN lets you go wireless without taking down the lab.

SECURE WIRELESS

Fast, robust and secure wireless communication using advanced radio technology from St. Jude Medical. Well proven in cath labs around the world. The Quantien integrated FFR platform is compatible with Pressure Wire CertusTM and Pressure Wire Aeris from St. Jude Medical.

AUTOMATIC PRESSURE INJECTORS

The automatic pressure injectors most commonly used today allows for specific volume of contrast to safely be delivered at a precise flow rate.

ANGIOMATE ILLUMINA

- **DIGITAL POWER HEAD DISPLAY:** The LED display automatically “flips” as the power head is rotated ensuring proper orientation for viewing. Indicates programmed protocol and volume remaining in the syringe.
- **FILL CONTROL BAR:** Allows easy, one finger operation. Speed control of ram for Syringe filling, pull-back, or infusion.
- **SYRINGES:** The latex free and transparent syringes provide a crystal clear view of the contrast medium. Compatible with pre-filled syringes.
- **AIR DETECTION DEVICE:** The optional Air Detection and Warning System (ADAWS) detects empty syringes and air bolus.
- **TOUCH SCREEN:** The pressure sensitive touch screen display makes the injection setup direct and easy. Protocol manager to store and recall user defined protocols.
- **MULTIMODALITY:** With just a touch of a button the injector switches between Angio and CT modes.

IE 33 X MATRIX ECHOCARDIOGRAPHY SYSTEM

Combining unprecedented 2D and 3D image quality in the same transducer and a host of easy-to-use quantification, clinical performance, and information management tools, the new iE33 xMATRIX echo system addresses the clinical needs of managing patients with cardiac disease, including hear failure, valvular disease, and congenital heart disease.

X5-1 xMATRIX ARRAY

The X5-1 transducer helps remove the barriers to 3D imaging, giving clinicians the power to choose 2D, 3D or combination imaging without disrupting workflow. Combining Xmatrix array technology with PureWave crystal technology for consistently excellent image quality, the X5-1 supports 3D, 2D, color flow, M-mode, PW/CW Doppler, Tissue Doppler imaging and contrast-enhanced exams.

- 3,040 elements with microbeam forming
- 1 to 5 MHz extended operating frequency range
- 2D, Live 3D volume, high volume rate (HVR), one-beat, two-beat and four-beat
- Live volume, color flow, Live 3D color, PW/CW Doppler, M-mode, color Mmode, contrast, Tissue Doppler imaging, Live xPlane imaging
- iRotate – Electronically rotatable scan angle from 0 to 180 degrees
- Adult, contrast LVO, pediatric CHD, cardiology coronary
- Ergonomic design for reduced user fatigue
- Solid state reliability and no complex liquid cooling required
- Philips green label approved transducer
- Dimensions: 9.2 x 3.9 x 2.9 cm (3.6 x1.5 x 1.1 in) LWD

LIVE 3D TEE TRANDSUCER

On the EPIQ 7, iE33 xMATRIX and CX50 xMATRIX systems, the X7-2t transducer combines xMATRIX array and PureWave crystal technologies in a fully functional TEE probe for exceptional Live 3D Echo performance during transesophageal exams and interventional procedures.

- 2D and 3D matrix array
- 7 to 2 MHz extended operating frequency range
- 2D, color flow, PW Doppler, CW Doppler, M-mode, Live xPlane imaging, Live 3D Echo, Live 3D zoom, dvanced XRES, triggered full volume, triggered 3D color
- Harmonic imaging
- Dynamic elevational focusing

- Electrocautery suppression
- Electronic rotatable array from 0 to 180 degrees
- TEE applications for patients >30 kg/66 lb

2D TEE TRANDSUSER

On the EPIQ 5, CX50 and Sparq systems, the X7-2t transducer combines xMATRIX and PureWave crystal technologies in a TEE probe for remarkable 2D transesophageal imaging.

- 2D matrix array
- 7 to 2 MHz extended operating frequency range
- 2D, color flow, PW Doppler, CW Doppler, M-mode, advanced XRES
- Harmonic imaging
- Dynamic elevational focusing
- Electrocautery suppression
- Electronic rotatable array from 0 to 180 degrees
- TEE applications for patients >30 kg/66 lb

An optional transducer adapter is available for purchase that enables the CX50 an Sparq X7-2t transducer to be used in both 2D and 3D modes on an iE33 system.

HD 15 PURE WAVE ULTRASOUND SYSTEM

Philips exclusive PureWave crystal technology is clinically proven to improve penetration in difficult-to-image patients. PureWave crystals are the result of a manufacturing technique that creates a near-perfect atomic level arrangement. The uniformity and lack of grain boundaries help transfer energy with up to 85% greater precision and efficiency over conventional materials. Their extended bandwidth covers the frequency range of two transducers, with improved Doppler performance and highly sensitive harmonic imaging. PureWave crystals have virtually perfect uniformity for greater bandwidth and twice the efficiency of conventional ceramic materials. The result is excellent imaging and Doppler performance.

SOPHISTICATED TECHNOLOGIES.SIMPLE TO US

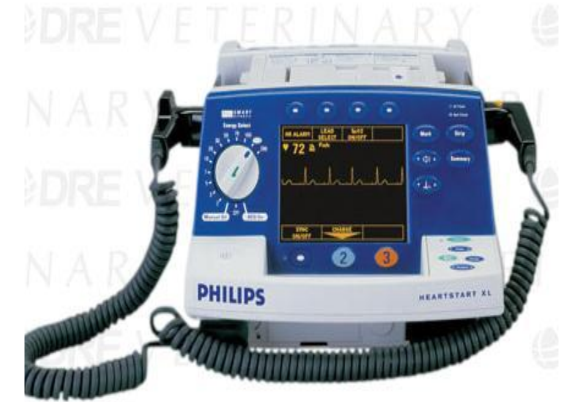
For advanced imaging and greater diagnostic confidence, rapid 3D volume renditions and dynamic 4D displays are presented with incredible clarity and detail resolution. This is a result of the HD15's next generation architecture with new high resolution analog to-digital conversion (A/D) technology and 4X parallel processing, leading to incredible contrast resolution as well as outstanding frame rates and 4D volume rates. For superb image sharpness and uniformity, Microfine EX focusing provides dynamic receive lens tuning with five times more focal points than previous generation systems. For images with less noise and fewer artifacts, SonoCT is true real-time compound imaging that merges up to nine lines of sight to create one amazing image with better clarity of curved and irregular borders. To attain extraordinary clarity and edge definition advanced XRES adaptive image processing uses proprietary algorithms originally developed for Philips MR to display images that are essentially free from noise, without reducing frame rates. To quickly

achieve optimal image quality in 2D, color and Doppler exams, Iscan image optimization is a one-button push that automatically adjusts.

PHILIPS HD 15 FEATURES

- New high resolution A/D technology
- 4 times Parallel Processing
- Microfine EX focusing
- SonoCT real-time compound imaging
- Advanced XRES adaptive image processing
- PureWave crystal technology

PHILIPS HEART START XL BIPHASIC DEFIBRILLATOR WITH ECG MONITORING



- Fast charging. Goes from 0 to 360 joules in under 5 seconds
- Battery Indicator. Low battery LED message lets you know when battery is running
- Back up AC power. Can be plugged into a standard AC wall socket.
- True 1-2-3 operation
- Event summery. Documents and stored up to 28 events, plus even stores data after the unit is turned off
- Shock advisory option. Lets you know whether a rhythm is shockable or not and guides you through the process of defibrillation
- Advisory event summary. Included with shock advisory, this features documents and stores up to 50 ECG strips and 200 events in shock advisory mode
- Large CRT display. User friendly messages and monitoring information appear on a bright 5 inch screen
- Leads off indicator, paddle contact indicator
- Uncluttered front panel
- Monitoring, an ECG bedside monitor when a separate unit isn’t available
- Annotating recorder, Hands free defibrillation
- Built in pacing option, Integrated pulse oximator option
- Autogain, adjustable heart rate alarms
- Easy loading thermal array recorder
- Built in pediatric paddles and internal paddles.
- 5 wire ECG option
- Light weight

GE MAC 500 PORTABLE ECG MACHINE

Portable 12 lead electrocardiograph, designed for acquisition, with or without analysis. For those facilities needing minimal ECG requirements.

SPECIFICATIONS

- **Dimensions**: Size: 290 x 80 x 200 (width x height x depth in mm).Weight: 2 kg Including battery
- **Power Supply**: From main or built-in battery lead-acid 12 V, 1.2 A hr
- **Languages:** There are 17 operational languages (German, English, French, Italian, Spanish, Swedish, Norwegian, Danish, Dutch, Czech, Finnish Hungarian, Macedonian, Bulgarian, Polish, Portuguese, and Russian)
- **Recording :**Thermal dot printer process, writing width 80 mm
- **Paper:** Paper width 90 mm
- **Paper speed:** 5-25-50 mm/s
- **Display :**Alphanumeric LCD 2 x 16 characters; Adjustable display contrast Zeroing; Automatic setting in the optimal recording range Anti-Drift- System
- **Electrode check:** Audible indicator and display on LCD; monitoring of individual electrodes Heart rate
- **Display**: Range 30-300 beats/min
- **Heart rate control:** Definable upper and lower limits; alarm when outside defined range
- **Mains operation:** Battery charging from built-in charger Voltage independent mains Operation /universal power supply; nominal input range: 100...240 V, 49...65 Hz

MORTARA ELI 250

The Mortara ELI 250 ECG is durable, practical and flexible, able to monitor and interpret electro cardio graph exams to determine heart rates. It is compact and light weight, the electrocardiograph provides comprehensive functionality with A4 paper. It is easy to use and portable device. Features of Mortara ELI 250 are

- High resolution power display
- Real time preview of real time 12 lead ECG
- Post acquisition review of acquired ECG
- By using the algorithm about the gender, adult and pediatric criteria provides a second opinion for resting ECG
- Bidirectional communication between USB, memory stick, LAN , wireless mortara

- Internal storage up to 60 digital ECG records. Optional expended storage up to digital ECG records
- 1000Hz sampling rate ELI 250 automatically selects and displays the best 10 seconds of ECG signal form five minute segment of internal full disclosure reducing clinical review time and eliminating the need of repeat ECG

AMBULATORY HOLTER MONITORS

SEER MC AMBULATORY DIGITAL ANALYSIS RECORDER

The SEER MC Recorder provides ambulatory ECG signal, as well as automated analysis of recorded data. Results of the automated analysis intended to assist the physician in the interpretation of the recorded data. Features of the SEER MC ambulatory recorder are

- It can collect and store up to 48 hours
- It have 2 to bipolar channels of the ECG data
- On demand it can acquire 12 lead ECG
- Ambulatory Holter recording provides an opportunity to assess the cardiac function while patient undergoing routine daily activities
- SEER MC have an event button when record ECG, it creates a patient marked event
- ECG acquired in the ambulatory environment will include more artifact.
- SEER MC stored each 10 seconds 12 lead ECG as a sample that makes up a trend; also
- acquire these samples so that they can be trended over time. It have an LCD display from which can get pacemaker spike detect display, AM/PM display, Heart beat display, numerical display.
- Hard ware's of the SEER MC are the ambulatory recorder, light weight color coded ambulatory cable, 4AA alkaline battery and a removable ECG data card of memory of 64 Mb up to 48 hours

SEER MC SETTINGS

- Paced detect mode off
- AECG gain unity gain(1x)
- AECG high frequency 55 Hz
- AECG low frequency- 0.5 Hz
- Pace pulse frequency response 3.0to 15KHz

PHILIPS Digitrak XT Holter monitoring System

- Simple navigation bottons with tactile response for fast setup. On-screen lead map and EASI lead placement support artifact- free hookup. The automatic power on self-test allows for reliable recording. The plug and play Dual Docking Station allows fast ECG downloads- often in less than seconds.
- ESAI derived 12 lead readings are standard on all recorders. It provides pacemaker detection with sensitivity adjustment.
- Lightweight design is easy to wear. The swivel belt clip and pouch make it easy to wear.
- The DigiTrak XT records up to 7 days on a single AAA battery. It has a water resistant case and includes a 2 year warranty. The dual docking station accommodates both DIgiTrak XT and DigiTrak Plus recorders.
- The lightweight recorder has a stresmlined from with a swivel belt clip for patient comfort and privacy. Its water resistant case means it will be not affected if used in water.

Ninhon Kohden CardioFax M EKG Machine

Easy operation and efficient workflow are the foundations of Nihon Kohden's Cardiofax M electrocardiogram. With its large 7inch color display, data can be viewed easily and allows ECG preview before recording. Cardiofax M supports paper-based and paperless workflows, enabling both waveform and analysis results to be transferred to a PC for review.

Nihon Kohden's breakthrough technology supports clinicians in testing more pateints with enhanced information available from analysis.

- 12-channel recording.
- Meet IEC60601-2-51 new international standard for digital electrocardiographs specifying accuracy and digital filter performance.
- Vivid color LCD screen

atLarge memory capacity: 40 ECG files in internal memory, 3000 ECG files on an SD memory card.

- Data transfer to PC by USB, SD memory card or LAN
- ECG data can be reviewed and managed on a Windows PC with optional ECG viewer software.

GE T2100 Treadmill System

Patient Exercise test Interpretations algorithm highlights critical values for efficient and timely diagnosis. Finite Residual Filter and Cubic Spline provide ECG baseline correction

and artifact resolution without sacrificing critical ST measurements. Automatic Arrhythmia Detection assists in documenting arrhythmias that occur during stress testing. Multiple networking options are available

- 60inch wide walking tread
- Two modes for stopping
- Handrails
- Emergency stop
- Speeds as high as 13.5 mph
- Grades as high as 25%
- GE T2100 horsepower motor.
- Self- calibrating.
- Digital control.

BIO MEDICAL WASTE

Biomedical waste is any kind of waste containing infectious materials. It may also include waste associated with generation of biomedical waste that visually appears to be of medical or laboratory origin as well research laboratory waste containing biomolecules or organisms that are restricted from environmental release. Discarded sharps are considered bio medical waste whether they are contaminated or not, due to the possibility of being contaminated with blood and their propensity to cause injury when not properly contained and disposed of. Biomedical waste is a type of biowaste.

Biomedical waste may be solid or liquid. Examples of infectious waste include discarded blood ,sharps, unwanted microbiological cultures and stocks, identifiable body parts(including those as a result of amputation), other human or animal tissue, used bandages and dressings, discarded gloves, other medical supplies that may have been in contact with blood and body fluids and laboratory waste that exhibits the characteristics described above. Waste sharps include potentially contaminated used(and unused discarded) needles, scalpels, lancets and other devices capable of penetrating skin.

Biomedical waste is generated from biological and medical sources and activities, such as the diagonosis, prevention or treatment of diseases. Common generation(or producers) of biomedical services, medical research laboratories), waste with these characteristics may alternatively be called medical or clinical waste.

Biomedical waste is distinct from normal trash or general waste, and differs from other types of hazardous chemicals and radioactive materials. While such wastes are normally

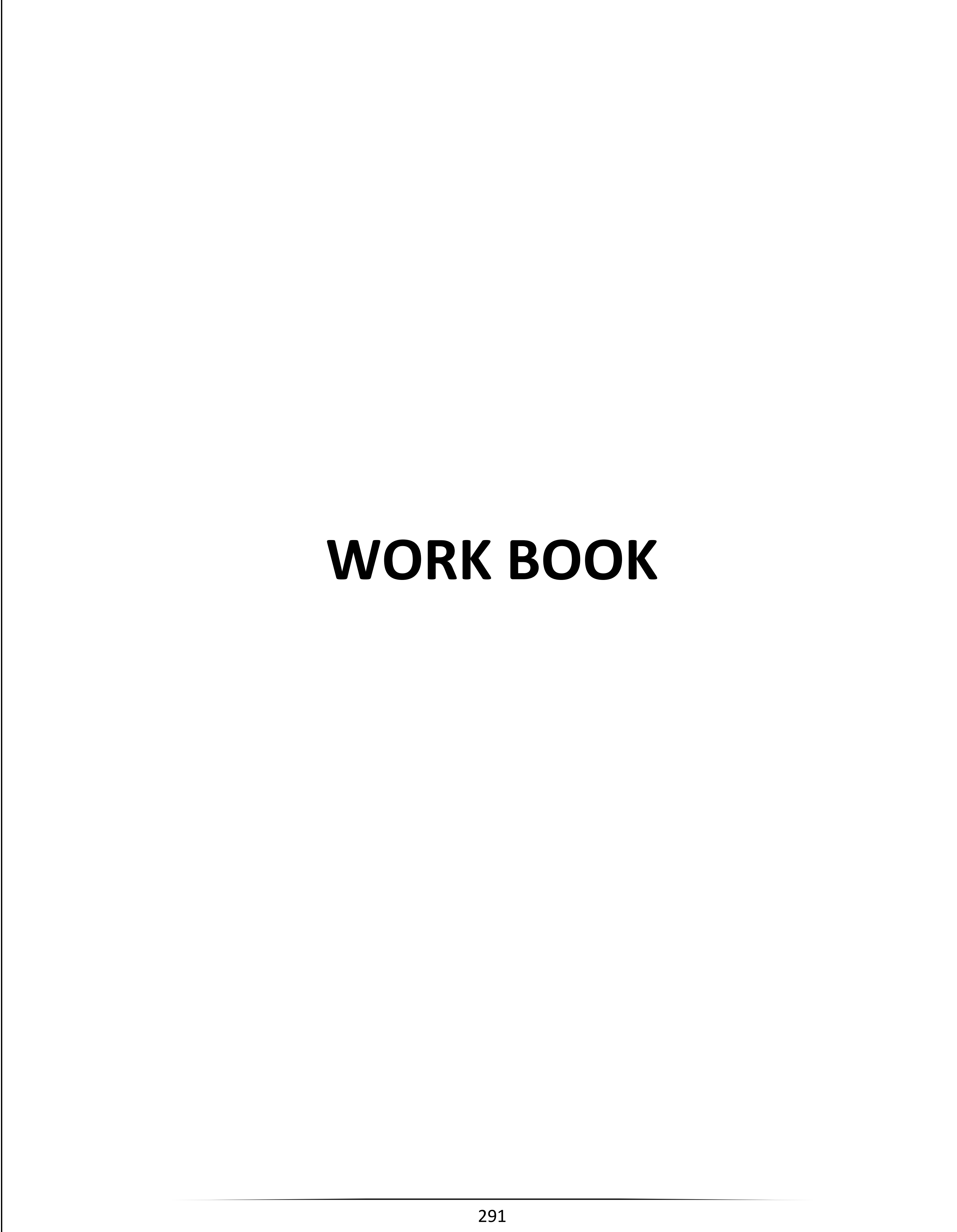
not infectous, they require proper disposal. Some wastes are considered multihazardous, such as tissue samples preserved in formalin.

Risk to human health

Disposal of this waste is an enviromental concern, as many medical wastes are classified as infectious or biohazardous and could potentially lead to the spread of infectious disease. The most common danger for humans is the infection which also affects other living organisms in the region. Dily exposure to the waste leads to accumulation of harmful substances or microbes in the person's body.

COLOUR CODING AND TYPE OF CONTAINER FOR BIOMEDICAL WASTE

COLOUR	WASTE DESCRIPTION
YELLOW*	Human tissues, organs, body parts, items contaminated by blood/body fluids, soiled cotton & dressing, soiled plaster casts etc.
RED*	Catheters, tubes, cannulae, syringes, plastic IV bottles & sets, used gloves, infected plastics, specimen containers, lab waste, microbiology cultures, used or discarded bags of blood/blood products, vaccines etc.
BLUE*	Glass items, needles, syringes, scalpels, blades, used and unused sharps etc.
BLACK*	Discarded medicines, discarded cytotoxic drugs etc.
GREEN	General waste, non-infected plastic materials & papers, disposables, cardboards, metal containers, office waste, food waste etc.



WORK BOOK

ECG PERFOMED

Sl,NO	Name	Hos.No
1	Dhanya	410175
2	Renuka	275107
3	Sobana kumary	211411
4	Surendran sreekandan	413120
5	Chinnamma	9013
6	Jaseena R S	407681
7	Paul K Rajeev	9601120
8	James Varghese	8700032
9	Abdul haji	412297
10	Simon	414646
11	Swamidas	414999
12	Alexander	413515
13	Indira	240776
14	Vilasini	410721
15	Somini Bhai	414921
16	Sony	321032
17	Karthika0	293897
18	Meenakumari	414552
19	Anandakrishnan	412193
20	Paul K Rajeev	411751
21	Priya	411751
22	Arifa	211016
23	Aneesh Babu	402522
24	Selvaraj	275499
25	Babukuttan pillai	398594
26	Babu M	411150
27	Abraham Varghese	396812
28	Sudhakaran Nair	392048
29	Mariamma Thomas	310707
30	Chinna thai	414348
31	Amina ebnezeer	414398
32	Vandana	391491
33	Baby M	410128
34	Nufaila	410692
35	Padmanabhan	2518
36	Shameena	409143
37	Nizamudheen	410602

38	Ramalekshmi	412362
39	Sasi	239000
40	Ammini	9307241
41	Krishnan Nair	362323
42	Shahul Hameed	413903
43	Liji	413948
44	Bilal	395141
45	Preman kesavan	413532
46	Ajith raj R	410267
47	Manoj	413607
48	Muhammed Sinan	413721
49	Meenakshi sundaran	367553
50	Allirani B	321114
51	Sujith	410769
52	Saninudheen	409360
53	Thoufeed	409396
54	Raju R	269696
55	Jamaludheen Rawther	316742
56	Moideenkutty	410345
57	Ganesh	414267
58	Abubekker	255805
59	Malathi devi	368671
60	Karthika	414856
61	Lathika devi	398273
62	Manikandan	9907305
63	Sunitha M S	414138
64	Devu K	250782
65	Joseph Abraham	266102
66	Krishna Ammal	411037
67	Bhaskaran Nair	334688
68	Christhudas	244481
69	Sreekandan Nair	411490
70	John jayasingh	8904433
71	Cicily Johny	250967
72	Sellappan	411948
73	Maiyan pillai	415496
74	Durai	412094
75	Kochuthresia	415449
76	Indira	412662
77	Noby	413412
78	Manoj	229399

79	Vineetha	401678
80	Girija	246534
81	Velappan	369085
82	Manikandan	12719
83	Sofiya	339424
84	Vidhya Sagar	411510
85	Nivitha	410970
86	Radhika	241495
87	Sarath Chandran	403898
88	Vinesh	411568
89	Alwin das	393469
90	Anju Unnithan	264937
91	Muhammed Haneefa	406597
92	Chandrasekharan	397507
93	Akhila	220964
94	Roskutty Jacob	244690
95	George Jacob	297831
96	Saji P Chacko	374944
97	Radha	9407406
98	Amal Prasad	404705
99	Shibu Babu	415880
100	Sudalai mani	403271

HOLTER PERFOMED

Sl,NO	Name	Hos.No
1	Vinesh	410175
2	Vineetha	275107
3	Vilasini	211411
4	Vidhya Sagar	413120
5	Velappan	9013
6	Vandana	407681
7	Thoufeed	9601120
8	Swamidas	8700032
9	Surendran sreekandan	412297
10	Sunitha M S	414646
11	Sujith	414999
12	Sudhakaran Nair	413515
13	Sudalai mani	240776
14	Sreekandan Nair	410721
15	Sony	414921
16	Somini Bhai	321032

17	Sofiya	293897
18	Sobana kumary	414552
19	Simon	412193
20	Shibu Babu	411751
21	Shameena	411751
22	Shahul Hameed	211016
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24	Sellappan	275499
25	Sasi	398594
26	Sarath Chandran	411150
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28	Saji P Chacko	392048
29	Roskutty Jacob	310707
30	Renuka	414348
31	Ramalekshmi	414398
32	Raju R	391491
33	Radhika	410128
34	Radha	410692
35	Priya	2518
36	Preman kesavan	409143
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38	Paul K Rajeev	412362
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43	Nivitha	413948
44	Muhammed Sinan	395141
45	Muhammed Haneefa	413532
46	Moideenkutty	410267
47	Meenakumari	413607
48	Meenakshi sundaran	413721
49	Mariamma Thomas	367553
50	Manoj	321114

LIST OF ECHO ASSISTED

SL.NO.	HNO	NAME	AGE1	SEX	DATE
1	424,990	SHIVANI S PILLAI.	1	F	02-mar-2017
2	386,941	SRUTHI S.	9	F	02-mar-2017
3	386,426	JUNAITHA BEEVI S.	47	F	02-mar-2017
4	387,373	JAYAKUMARI B.S.	39	F	02-mar-2017
5	418,180	MOLY I T.	49	F	02-mar-2017

6	402,596	RAJEEV T.	4	M	04-oct-2017
7	396,150	AKASH S.	6	M	03-aug-2017
8	322,082	ADWAITH J.K.	7	M	03-may-2017
9	392,944	THEERTHA REGHURAJ	7	F	03-may-2017
10	403,344	YALINI S.	2	F	01-jun-2017
11	437,846	MURUGAVALLI	31	F	09-oct-2017
12	408,707	RESHMA K.J.	23	F	25-oct-2017
13	393,226	RISA ROSE KURIACHAN	3	F	03-may-2017
14	430,746	REMYA S.	25	F	02-may-2017
15	432,799	SREENATH B.V	40	M	02-aug-2017
16	397,822	ROSHAN ANTONY	7	M	03-aug-2017
17	343,769	JAYA W	27	F	12-oct-2017
18	400,305	AMBIKA M	41	F	12-oct-2017
19	365,330	JOB K S	46	M	12-oct-2017
20	439,537	REGHUNADHAN ASARI S.	69	M	07-oct-2017
21	407,094	ANUSREE S.	8	F	03-aug-2017
22	438,689	THULASI S NAIR.	48	M	09-oct-2017
23	426,611	BRAMANAYAGAM M.	6	M	01-jun-2017
24	429,095	PREETHA K P.	32	F	13-jun-2017
25	402,722	HARSHA PRAKASH	11	F	26-oct-2017
26	439,449	DUAA FATHIMA S.	0	F	04-oct-2017
27	402,635	ANSHAD MOIDU	8	M	04-oct-2017
28	392,405	JENIN JOHN JIBIN	3	M	03-may-2017
29	408,042	JAKSON J. CHACKO	14	M	25-oct-2017
30	440,341	DEVIDAYA M.	0	F	19-oct-2017
31	267,062	SHIBILA S.	18	F	26-oct-2017
32	437,224	SANTHA M A.	62	F	26-oct-2017
33	436,787	SARATH V S.	30	M	26-oct-2017
34	307,901	POOJA J.	7	F	02-aug-2017
35	392,571	AJIMON T.	33	M	03-may-2017
36	431,274	SHREYAS S.	3	M	05-oct-2017
37	437,499	RAMESH S.	45	M	05-oct-2017
38	424,362	ALBERT LEON	26	M	03-aug-2017
39	423,933	B/O SAJITHA	1	M	11-oct-2017
40	435,673	ANANTHA NARAYANAN	1	M	11-oct-2017

LIST OF TEE ASSISTED

SL.NO	HOS.NO	NAME	AGE	SEX	DIAGNOSIS
1	322,082	ADWAITH J.K.	7	M	OS ASD
2	392,944	THEERTHA REGHURAJ	7	F	OS ASD
3	437,846	MURUGAVALLI	31	F	RHD,SEV MS
4	408,707	RESHMA K.J.	23	F	OS ASD
5	432,799	SREENATH B.V	40	M	OS ASD
6	397,822	ROSHAN ANTONY	7	M	OS ASD

7	207910	PASUMATHY		F	RHD,SEV MS
8	294035	MUNIYA SAMY		M	RHD,SEV MS
9	434993	PRIYADARSANA KUMAR		M	PFO
10	250396	DEEPA V		F	RHD,SEV M

LIST OF TMT PERFORMED

SL.NO.	HNO	NAME	AGE1	SEX
1	431,046	RAMACHANDRAN NAIR S.	59	M
2	209,605	CHANDRAN.T.A.	75	M
3	431,281	NADESAN D.	68	M
4	433,603	SEEMANDINI AMMA S.	60	F
5	337882	SHEEJA R.I	47	F
6	433890	HARI KUMAR K G.	56	M
7	436,488	MARY THOMAS	59	F
8	433,769	RAVEENDRAN NAIR C.	66	M
9	431,460	BAIJU SIMON	50	M
10	439294	EMMANUEL M Y	66	M

LIST OF CORONARY ANGIOGRAM PERFORMED

SI.NO.	HNO	CATHNO	NAME	AGE1	SEX	DOP	DIAG.
1	259,610	70,624	RAJAMMA.P.	80	F	01-mar-2017	CAD
2	427,561	70,647	THANKACHAN N.	67	M	03-mar-2017	CAD
3	421,219	70,671	RACHEL JAMES	57	F	06-mar-2017	CAD
4	427,961	70,682	MAJITHA A.	39	F	07-mar-2017	CAD
5	427,463	70,693	RAJENDRAN N.	68	M	08-mar-2017	CAD
6	429,623	70,789	BENAZIR BEEVI A.	44	F	17-mar-2017	CAD
7	428,652	70,806	RAGHAVAN ACHARY N.	65	M	20-mar-2017	CAD
8	246,593	70,849	DINESH B.	63	M	23-mar-2017	CAD
9	428,926	70,868	PUSHPANGADAN P.	70	M	27-mar-2017	CAD
10	425,845	70,896	USHA KUMARI D.	45	F	28-mar-2017	CAD
11	429,820	70,908	APPUKUTTAN NAIR N.	82	M	30-mar-2017	CAD
12	312,997	70,918	SABU KOSHY (FR)	59	M	31-mar-2017	CAD
13	302,186	70,941	NAJUMUDEEN M.	56	M	03-apr-2017	CAD
14	429,763	70,943	RAJAN N.	64	M	04-apr-2017	CAD
15	428,571	71,004	SARADAMMA K.	82	F	10-apr-2017	CAD
16	429,753	71,031	RAMESH KUMAR K.	53	M	12-apr-2017	CAD
17	430,921	71,038	KARTHIKEYAN NAIR R.	72	M	15-apr-2017	CAD
18	430,454	71,053	RAJMOHAN V.	53	M	18-apr-2017	CAD
19	429,904	71,062	PHILOMINA.	65	F	19-apr-2017	CAD
20	429,950	71,074	RADHAMONY AMMA S.	61	F	20-apr-2017	CAD
21	430,870	71,091	ABDUL RASHEED.	52	M	21-apr-2017	CAD
22	424,953	71,111	SATHEESAN R.	67	M	24-apr-2017	CAD

23	389,219	71,112	MOHANAN B.	64	M	25-apr-2017	CAD
24	294,582	71,130	PHILIP V P (DR)	63	M	26-apr-2017	CAD
25	428,418	71,134	GHOSH S	57	M	27-apr-2017	CAD
26	429,915	71,160	IBRAHIMKUTTY E.	62	M	28-apr-2017	CAD
27	429,818	71,180	SREEDHARAN PILLAI G.	67	M	02-may-2017	CAD
28	431,731	71,192	SASIDHARAN NAIR N.	71	M	02-may-2017	CAD
29	350,611	71,201	KOCHUKRISHNAN NAIR V.	73	M	03-may-2017	CAD
30	431,722	71,212	SUKUMARAN S.	60	M	04-may-2017	CAD
31	430,864	71,220	SANTHOSH KUMAR C R.	53	M	05-may-2017	CAD
32	427,312	71,241	KOCHU CHERUKKAN K.	72	M	08-may-2017	CAD
33	430,555	71,243	VINOD E K.	45	M	09-may-2017	CAD
34	431,203	71,255	THOMAS T J.	55	M	11-may-2017	CAD
35	431,243	71,274	RAJKUMAR D.	48	M	12-may-2017	CAD
36	406,406	71,280	MANIYAN R	67	M	15-may-2017	CAD
37	432,180	71,284	MOHANADAS K	64	M	16-may-2017	CAD
38	430,790	71,307	VEERANAN V.	53	M	17-may-2017	CAD
39	400,852	71,322	BALAN E.V.	64	M	18-may-2017	CAD
40	274,898	71,335	MOHAMMED HANIFA M.	43	M	19-may-2017	CAD
41	431,220	71,349	BADARUDEEN A.	58	M	22-may-2017	CAD
42	430,793	71,371	SURESH N	46	M	24-may-2017	CAD
43	9,702,386	71,373	DAMODARAN.O.	70	M	25-may-2017	CAD
44	431,539	71,392	JALEELA BEEVI S.	65	F	29-may-2017	CAD
45	292,096	71,421	RABEL SILUVAI	68	M	30-may-2017	CAD
46	433,240	71,432	SREENADHAN S	34	M	31-may-2017	CAD
47	433,376	71,445	BHADRAKUMARAN NAIR K S.	57	M	01-jun-2017	CAD
48	433,392	71,456	HUSSAINSHA K K.	60	M	02-jun-2017	CAD
49	397,949	71,458	VIJAYAPPAN M.	67	M	03-jun-2017	CAD
50	432,245	71,469	SURESHKUMAR.S	50	M	05-jun-2017	CAD
51	431,816	71,465	SHAJAHAN M.	63	M	06-jun-2017	CAD
52	388,104	71,485	VENKITASUBRAMANIAN P.R.	72	M	07-jun-2017	CAD
53	433,590	71,502	MANIYAN D.	70	M	08-jun-2017	CAD
54	432,486	71,518	INDHUMATHI M.	49	F	09-jun-2017	CAD
55	432,992	71,532	AYOOBKUTTY M.	47	M	12-jun-2017	CAD
56	9,605,934	71,570	THAHA K	54	M	15-jun-2017	CAD
57	432,582	71,572	RADHAKRISHNAN M P.	65	M	16-jun-2017	CAD
58	270,266	71,600	KONRAJ T.	64	M	19-jun-2017	CAD
59	417,868	71,597	RAMAKRISHNAN G.	45	M	19-jun-2017	CAD
60	433,858	71,615	GIRIRAJAN D	77	M	21-jun-2017	CAD
61	433,843	71,631	HARI KUMAR V.	60	M	22-jun-2017	CAD
62	433,815	71,694	LEENA RAJMOHAN	55	F	30-jun-2017	CAD
63	9,702,025	71,755	MANIAN ASARY.P.	70	M	07-jul-2017	CAD
64	413,625	71,770	CHANDRA SEKHARAN K.	65	M	10-jul-2017	CAD
65	433,228	71,775	AJITHA P.	43	F	11-jul-2017	CAD
66	434,981	71,808	INDIRA V.	51	F	13-jul-2017	CAD

67	9,501,336	71,820	KRISHNAN NAIR.N.	80	M	14-jul-2017	CAD
68	433,908	71,828	SAMUEL JOYKUTTY	58	M	17-jul-2017	CAD
69	433,218	71,849	PANKAJAKSHY AMMA K M.	72	F	19-jul-2017	CAD
70	433,817	71,869	BABY K.	59	M	20-jul-2017	CAD
71	433,934	71,867	RAGHUVARAN R.	55	M	20-jul-2017	CAD
72	433,748	71,866	GEORGE P THOMAS.	70	M	20-jul-2017	CAD
73	435,365	71,877	JOHN S.	70	M	20-jul-2017	CAD
74	434,509	71,879	JAYA S.	59	F	21-jul-2017	CAD
75	431,580	71,880	CHANDRAN P V.	56	M	21-jul-2017	CAD
76	434,271	71,882	SUSEELA S.	58	F	21-jul-2017	CAD
77	436,088	71,907	SUSEELA T.	70	F	25-jul-2017	CAD
78	436,183	71,957	ASHARAF. E	65	M	31-jul-2017	CAD
79	433,972	72,110	AMBIKA DEVI. G	56	F	16-aug-2017	CAD
80	428,549	72,120	CHANDRAN CHETTIYAR N	52	M	17-aug-2017	CAD
81	396,580	72,133	JANAKI P.	54	F	18-aug-2017	CAD
82	436,132	72,138	JAYAKUMARI J.	42	F	21-aug-2017	CAD
83	431,205	72,150	SAINUDHEEN KUNJU M.	70	M	22-aug-2017	CAD
84	211,778	72,199	MAYA NANDAKUMAR A.(DR)	54	F	25-aug-2017	CAD
85	339,019	72,222	RAMADAS V.	60	M	28-aug-2017	CAD
86	435,689	72,235	JANAKI M.	51	F	29-aug-2017	CAD
87	386,598	72,261	SAINUDEEN KUNJU A.	75	M	31-aug-2017	CAD
88	437,963	72,274	JEEJA P J.	42	F	01-sep-2017	CAD
89	428,591	72,334	SREEKUMARI P S.	60	F	13-sep-2017	CAD
90	208,554	72,351	BHASKARAN.M.	67	M	14-sep-2017	CAD
91	437,045	72,357	BALU V.	50	M	15-sep-2017	CAD
92	433,964	72,365	SUNEER S.	45	M	18-sep-2017	CAD
93	435,808	72,387	MAHESWARI E.	34	F	19-sep-2017	CAD
94	435,735	72,401	ABDUL AZIZ.	59	M	20-sep-2017	CAD
95	435,383	72,417	AMINA P.	53	F	22-sep-2017	CAD
96	438,224	72,425	SURENDRAN P.	61	M	25-sep-2017	CAD
97	436,424	72,438	NABEESA BEEVI.	66	F	26-sep-2017	CAD
98	8,705,423	72,457	SUBHODHINI V K	45	F	27-sep-2017	CAD
99	437,817	72,462	JITHIN MOHAN	32	M	28-sep-2017	CAD
100	436,699	72,482	SABU S.	48	M	03-oct-2017	CAD
101	437,221	72,505	DILEEP KUMAR C.	49	M	04-oct-2017	CAD
102	436,450	72,517	BASHEER RAVUTHAR.	55	M	05-oct-2017	CAD
103	437,270	72,526	KRISHNAN N .	75	M	06-oct-2017	CAD
104	438,351	72,534	SANTHA B.	63	F	09-oct-2017	CAD
105	405,281	72,561	SOBHA P NAIR	38	F	11-oct-2017	CAD
106	417,106	72,582	UDAYAN V.	68	M	12-oct-2017	CAD
107	349,486	72,594	SATYA P P	50	F	13-oct-2017	CAD
108	428,620	72,612	TONY VARGHESE	51	M	16-oct-2017	CAD
109	439,846	72,642	PARVATHY M.	68	F	19-oct-2017	CAD
110	436,599	72,644	KRISHNANKUTTY V.	70	M	20-oct-2017	CAD
111	411,372	72,675	NISHA FIROS KHAN	36	F	23-oct-2017	CAD
112	9,601,469	72,687	NARAYANAN NAIR.K.	84	M	24-oct-2017	CAD

113	437,632	72,697	VELUMANI GURUSAMY.	50	F	25-oct-2017	CAD
114	436,858	72,705	BASHEERKUTTY A.	57	M	26-oct-2017	CAD
115	436,697	72,732	NANDAKUMAR D.	47	F	27-oct-2017	CAD
116	437,704	72,749	LATHA JAYAN.	48	F	30-oct-2017	CAD
117	313,376	72,760	ABDUL ASSIS.A.K	52	M	31-oct-2017	CAD

LIST OF CATH ASSISTED

Sl.No.	HNO	NAME	AGE1	SEX	DOP	DIAG
1	412,970	RASHA FATHIMA C.K.	8	F	31-mar-2017	AVCD
2	411,061	SAINANDA V. S.	10	F	22-mar-2017	DORV
3	311,468	AYANA ALEX	14	F	08-jun-2017	SCIMITAR SYNDROME
4	434,956	SREEKUTTY	0	F	30-aug-2017	OS-ASD,
5	429,343	SANTHAMMA B.	52	F	23-aug-2017	OS ASD
6	231,350	HARSHA DILEEP	13	F	23-aug-2017	PULMONARY ATRESIA,
7	327,919	SUBUHANA A.	6	F	07-sep-2017	DORV
8	246,955	AKSHAY A.M.	12	M	07-sep-2017	D-TGA
9	319,022	ARCHANA G S	7	F	07-sep-2017	TRICUSPID ATRESIA,TGA
10	429,001	KAVITHA M.	23	F	27-apr-2017	VSD,SEV PAH
12	429,891	MOHAMED MUNEER.	38	M	26-may-2017	RHD - S/P AVR
13	436,361	GOODWIN S S.	7	M	23-aug-2017	MOD AVVR,LONG SEG PA,
16	426,067	B/O SHAHINA	1	F	08-jun-2017	DORV
19	414,212	FIRDOUS R.	4	M	27-apr-2017	PAPVC
20	431,355	KOKILA P.	13	F	27-jul-2017	DORV,VSD WITH SEV PS

LIST OF PTCA ASSISTED

sl.No.	HNO	NAME	AGE	SEX	DOP	DIAG
1	416,276	ABDUL RAHUMAN M A.	70	M	01-mar-2017	CAD, ACS-RVMI+IWMI
2	426,773	VISWANATHAN NAIR K.	65	M	20-mar-2017	CAD, UA
3	264,104	JOSE ABRAHAM	75	M	20-mar-2017	CAD, ACS-NSTEMI
4	268,409	GODFREY R E.	74	M	25-mar-2017	CAD

5	428,799	RAVINDRAN D.	58	M	27-mar-2017	CAD, ASMI-LYSED WITH STK
6	249,188	GIREESH kUMAR G.	50	M	07-apr-2017	CAD, OLD AWMI
7	428,152	VIJAYAMMA L.	65	F	21-apr-2017	CAD, CSA
8	429,822	ABDUL RASAK K.	51	M	24-apr-2017	CAD, ACS-IWMI
9	431,142	AYYAPPAN THAMPI M.	71	M	25-apr-2017	CAD, ACS-AWSTEMI
10	302,186	NAJUMUDEEN M.	56	M	02-may-2017	CAD, AWMI-(30/09/2009)
11	430,461	MOHANAN P.	52	M	02-may-2017	CAD , ACS -NSTEMI 22-3-2017
12	430,557	RADHAKRISHNAN P.	56	M	08-may-2017	CAD - AWMI (20 MARCH 2017)
13	424,351	RAVEENDRAN NAIR T.K.	73	M	09-may-2017	CAD, CRITICAL LEFT MAIN OSTIAL DI
14	428,864	GOPALAKRISHNAN B.	58	M	12-may-2017	CAD
15	431,336	JAGADAMMA K.	53	F	16-may-2017	CSA II-III
16	11,945	NIRAMALA.S.	66	F	17-may-2017	CAD, H/O ?NSTEMI
17	431,423	JOHN B.	79	M	18-may-2017	CAD. ACS 2004.
18	432,654	ANZAL K.	23	M	19-may-2017	CAD, ACS-IW STEMI
19	431,175	CHANDRIKA C.	63	F	22-may-2017	ATYPICAL CHEST PAIN
20	437,615	BALAKRISHNA PILLAI	76	M	29-aug-2017	CAD/ACS-NSTEMI

LIST OF DEVICE CLOSURES ASSISTED

SL.NO.	HNO	NAME	AGE1	SEX	DOP	PROCEDURE
1	402,596	RAJEEV T.	4	M	04-oct-2017	ASDDC
2	408,707	RESHMA K.J.	23	F	25-oct-2017	ASDDC
3	400,305	AMBIKA M	41	F	12-oct-2017	ASDDC
4	365,330	JOB K S	46	M	12-oct-2017	ASDDC
5	402,635	ANSHAD MOIDU	8	M	04-oct-2017	ASDDC
6	408,042	JAKSON J. CHACKO	14	M	25-oct-2017	ASDDC
7	437,224	SANTHA M A.	62	F	26-oct-2017	ASDDC
8	436,787	SARATH V S.	30	M	26-oct-2017	ASDDC
9	431,274	SHREYAS S.	3	M	05-oct-2017	ASDDC
10	437,499	RAMESH S.	45	M	05-oct-2017	ASDDC

SL.NO.	HNO	NAME	AGE1	SEX	DOP	PROCEDURE
1	424,990	SHIVANI S PILLAI.	1	F	02-mar-2017	PDADC
2	386,941	SRUTHI S.	9	F	02-mar-2017	PDADC
3	429,269	ASHA A.	1	F	06-apr-2017	PDADC
4	429,029	ASHLE JANE V J.	2	F	06-apr-2017	PDADC
5	343,769	JAYA W	27	F	12-oct-2017	PDADC
6	402,722	HARSHA PRAKASH	11	F	26-oct-2017	PDADC
7	439,449	DUAA FATHIMA S.	0	F	04-oct-2017	PDADC
8	440,341	DEVIDAYA M.	0	F	19-oct-2017	PDADC
9	267,062	SHIBILA S.	18	F	26-oct-2017	PDADC
10	423,933	B/O SAJITHA	1	M	11-oct-2017	PDADC

SL.NO	HNO	NAME	AGE1	SEX	DOP	PROCEDURE	DIAG
1	362,599	AADHI POORNA PORSELVAN .J	4	M	22-jun-2017	VSDDC	5.5 MM MUSCULAR VSD
2	17,981	SURESH KUMAR.N.	43	M	31-may-2017	VSDDC	ASD+VSD WITH PAH
3	439,537	REGHUNADHAN ASARI S.	69	M	07-oct-2017	VSDDC	
4	307,901	POOJA J.	7	F	02-aug-2017	VSDDC	3.5MM SA-VSD

LIST OF BALLOON ATRIAL SEPTOSTOMY ASSISTED

SL.NO	HNO	NAME	AGE1	SEX	DOP	CM_PROCEDURE
1	428,113	ADITHYA B N.	0	F	07-mar-2017	BAS
2	435,511	B/O PRINCY.	0	M	13-jul-2017	BAS
3	439,506	MUHAMMED AKHNAS.	0	M	04-oct-2017	BAS
4	415,692	ADHRIT KRISHNA	1	M	03-aug-2017	BAS
5	439,324	B/O SHAKKIRA.	0	M	27-sep-2017	BAS
6	434,138	B/O REMILA	0	M	24-jun-2017	BAS
7	439,440	DHANNUIN.S.R	0	M	29-sep-2017	BAS
8	438,146	B/O FATHIMA SUHARA	0	F	01-sep-2017	BAS
9	440,139	B/O ASWATHY	0	F	12-oct-2017	BAS
10	435,289	JOANNA SHAJI	0	F	09-jul-2017	BAS

LIST OF BALLOON PULMONARY VALVOTOMY ASSISTED

SL.NO	HNO	NAME	AGE1	SEX	DOP
1	9,504,508	RAJESH.M.P	22	M	14-mar-2017
2	433,850	B/O FAHIDA	0	M	24-aug-2017

3	290,611	SARAVANAN S.	9	M	20-apr-2017
4	411,546	ARYAN G NAIR	2	M	25-may-2017
5	397,749	DARSAN R.K.	3	M	14-sep-2017
6	438,616	ALIYA FATHIMA S.	0	F	14-sep-2017
7	438,383	SIDDHI.A.S	1	F	25-oct-2017
8	433,654	B/O TISSY	0	F	16-jun-2017
9	439,598	B/O BINDU.	0	M	05-oct-2017
10	430,805	MUHAMMED.K	1	M	24-may-2017

LIST OF BALLOON AORTIC VALVOTOMY ASSISTED

SL.NO	HNO	NAME	AGE1	SEX	DOP
1	429,241	B/O JINCY	0	F	09-mar-2017
2	387,959	DAMODHARAN K.	82	M	11-may-2017
3	437,509	MUHAMMED SIDAN C P.	0	M	23-aug-2017
4	360,195	ALOK RANJAN TOSH	9	M	25-may-2017
5	239,399	MARIAMMA GEORGE	69	F	20-apr-2017
6	433,968	NIJO MATHAI	18	M	30-jun-2017
7	439,515	SUBASH S.	12	M	04-oct-2017

LIST OF BALLOON MITRAL VALVOTOMY ASSISTED

SL.NO	HNO	NAME	AGE1	SEX	DOP
1	409,959	SUBAIDA E.P.	51	F	06-mar-2017
2	437,846	MURUGAVALLI	31	F	09-oct-2017
3	436,676	MUTHUPETCHI A.	40	F	14-aug-2017
4	435,380	NASILA BEEVI U.	35	F	14-aug-2017
5	429,474	SOFIYA A.	39	F	05-apr-2017
6	438,689	THULASI S NAIR.	48	M	09-oct-2017
7	266,871	SOUMYA K.SOMAN	31	F	10-apr-2017
8	416,329	MARY JOHN JOSE	38	F	10-apr-2017
9	233,407	MANJUSHA.H.	41	F	16-oct-2017
10	438,442	AYYAPPAN R.	33	M	16-oct-2017

LIST OF BALLOON COARCATATION OF AORTA ASSISTED

SL.NO.	HNO	NAME	AGE1	SEX	DOP
1	9,401,813	ARCHA GOURI.J	23	F	01-jun-2017
2	9,004,295	BOBY P.A.	54	F	21-sep-2017
3	440,216	ANJANA HARI	0	F	19-oct-2017
4	437,870	MAYA PRASANNAN	35	F	19-oct-2017

LIST OF PERMANENT PACEMAKER IMPLANTATION ASSISTED

SL.NO.	HNO	NAME	AGE1	SEX	DOP
1	428,308	ABDUL AZEEZ M.	63	M	03-apr-2017
2	279,965	ADIYAPPAN M.	46	M	01-mar-2017
3	440,838	SREEKANTAN NAIR K G.	75	M	27-oct-2017
4	9,808,547	VILASINI BAI.K.	61	F	03-jul-2017
5	417,204	SREENISELVAM S.	15	F	03-oct-2017
6	440,543	BALAKRISHNA PILLAI KM.	88	M	23-oct-2017
7	431,742	LEDWIN DHAS S	22	M	02-may-2017
8	437,878	SAINABA M A.	42	F	24-oct-2017
9	9,407,406	RADHA.L	48	F	06-oct-2017
10	257,097	MADHAVAN PILLAI.G	82	M	21-oct-2017

LIST OF CARDIAC RESYNCRONISATION THERAPY/ICD ASSISTED

SL.NO.	HNO	NAME	AGE1	SEX	DOP	PROCEDURE
1	189,786	ALEX PAUL	59	M	07-mar-2017	CRT
2	428,725	ABBAS T.	44	M	04-mar-2017	CRT
3	348,819	THAJUDEEN K J	49	M	07-mar-2017	CRT
4	429,891	MOHAMED MUNEER.	38	M	20-jul-2017	CRT
5	428,801	HARIS BABU K.	56	M	11-may-2017	CRT

SL.NO.	HNO	NAME	AGE1	SEX	DOP	PROCEDURE
1	438,480	SULAIKHA HASSAN.	73	F	06-oct-2017	ICD
2	424,125	VIJAYAN N.A.	72	M	06-apr-2017	ICD
3	435,290	SARALA S.	63	F	14-jul-2017	ICD
4	431,868	ALIYARUKUTTY M.	66	M	08-may-2017	ICD
5	282,422	YOOSUF T.K	50	M	11-jul-2017	ICD

LIST OF ELECTROPHYSIOLOGICAL PROCEDURES ASSISTED

SL.NO.	HNO	NAME	AGE1	SEX	DOP	PROCEDURE
1	427,593	MATHIYAS P.	60	M	01-mar-2017	EPS+RFA
2	435,371	KRISHNA PRIYA ANIL.	10	F	01-aug-2017	EPS+RFA
3	435,192	ANJANA M.	10	F	01-aug-2017	EPS+RFA
4	439,260	PUSHPA VARGESE.	51	F	06-oct-2017	EPS+RFA
5	316,402	RATHEESH KUMAR M	49	M	02-jun-2017	EPS+RFA
6	435,047	BIBILA S.	32	F	02-sep-2017	EPS+RFA
7	370,527	SOURAV K S	12	M	02-may-2017	EPS+RFA
8	431,180	FIDA SHERIN C.K.	15	F	02-may-2017	EPS+RFA
9	435,849	VINEESH P K.	35	M	31-oct-2017	EPS+RFA
10	282,419	MOHANAN G	50	M	02-jun-2017	EPS+RFA

LIST OF SPECIAL PROCEDURES ASSISTED

SL.NO.	HNO	NAME	AGE1	SEX	DOP	PROCEDURE
1	413,094	YASODHA C.	70	F	29-mar-2017	TAVR
2	422,129	PHILIP K S.	65	M	30-mar-2017	TAVR
3	414,526	ATHEL JAISON.	14	M	28-jul-2017	TPVR
4	8,808,243	JOWHAR.K.	43	M	28-jul-2017	TPVR
5	437275	DEVI SASI	30	F	24-aug-2017	RSOV DC