

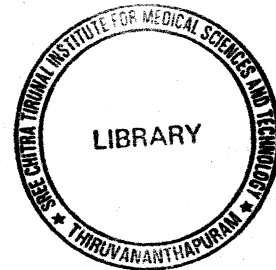
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**SREE CHITRA TIRUNAL INSTITUTE FOR
MEDICAL SCIENCES AND TECHNOLOGY**

THIRUVANANTHAPURAM-695 011

PROJECT REPORT



DIPLOMA IN CARDIAC LAB TECHNOLOGY

DEPARTMENT OF CARDIOLOGY

CANDIDATE NAME: SUNITHA K V

MONTH AND YEAR OF SUBMISSION: NOV 2003

CERTIFICATE

I, *Sunitha.K.V.*.....hereby declare that the projects included in this book were undertaken by me under the supervision Department of Cardiology, SCTIMST, Trivandrum.

Sunitha
22.11.2003
Signature

Forwarded,

The candidate, *Sunilka.K.V.*-----, has carried out the minimum required procedure.

Trivandrum

Date: 22.11.2003

Jaganmohan
Prof. Jaganmohan Tharakan

Professor and Head

Department of Cardiology

SCTIMST

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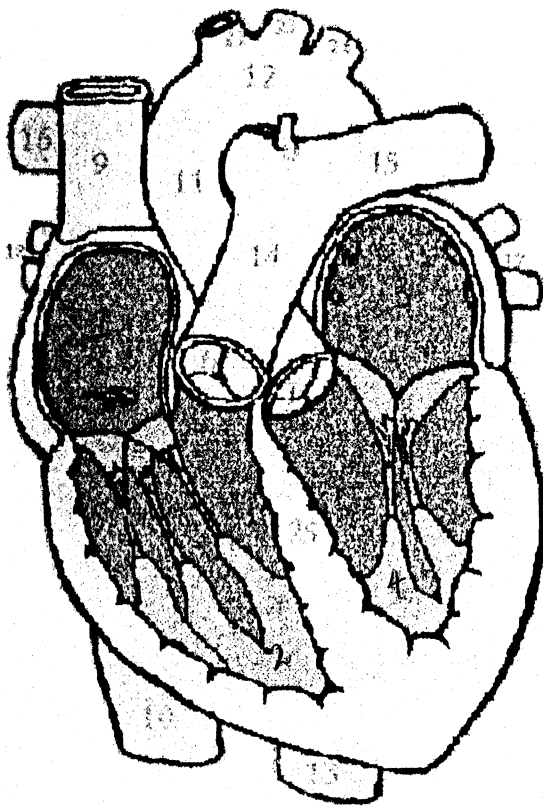
At last I would like to acknowledge my sincere thanks to my seniors and juniors for their co-operation in the work place. I thank all other colleagues, technical and service personal, and all well-wishers who helped me all the way during the last two years of my career.

ABOUT THE COURSE

Diploma in cardiac lab technology is a two-year diploma course offered by the institute under the department of cardiology.

Four seats are available for the course and the selection is based on the national level entrance after the successful completion of graduation in physics. The course offers high standard training in various advanced modalities of invasive interventions, diagnostic and non-diagnostic procedures in cardiology. The course schedule contains the theory classes, practical training and seminar presentation. At the end of course examinations and internal assessment are carried out and Diploma certificate issued.

ANATOMY



1. RIGHT ATRIUM
2. RIGHT VENTRICLE
3. LEFT ATRIUM
4. LEFT VENTRICLE
5. TRICUSPID VALVE, ALSO KNOWN AS THE ATRIOVENTRICULAR (AV) VALVE
6. PULMONARY SEMILUNAR VALVE
7. AORTIC SEMILUNAR VALVE
8. BICUSPID VALVE, ALSO KNOWN AS THE MITRIAL VALVE
9. SUPERIOR VENA CAVA
10. INFERIOR VENA CAVA
11. ASCENDING AORTA
12. ARCH OF AORTA (AORTIC ARCH)
13. DESCENDING THORACIC AORTA
14. TRUNK OF PULMONARY ARTERY (PULMONARY TRUNK)
15. LEFT PULMONARY ARTERY
16. RIGHT PULMONARY ARTERY
17. LEFT PULMONARY VEINS
18. RIGHT PULMONARY VEINS
19. CHORDAE TENDINEAE
20. PAPILLARY MUSCLE
21. LIGAMENTUM ARTERIOSUM

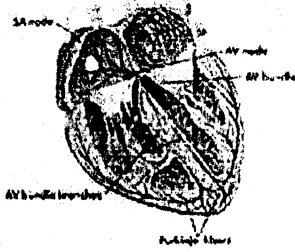
22. BRACHIOCEPHALIC ARTERY
23. LEFT COMMON CAROTID ARTERY
24. LEFT SUBCLAVIAN ARTERY
25. INTERVENTRICULAR SEPTUM

The Anatomy of the Heart

The human heart is a muscular pump. . Unlike most of the other hollow organs, whose muscle layers are composed of smooth muscle, the heart is composed of cardiac muscle. All muscle types function by contraction, which causes the muscle cells to shorten. Skeletal muscle cells, which make up most of the mass of the body, are voluntary and contract when the brain sends signals telling them to react. The smooth muscle surrounding the other hollow organs is involuntary, meaning it does not need to be told to contract. Cardiac muscle is also involuntary. So functionally, cardiac muscle and smooth muscle are similar. Anatomically though, cardiac muscle more closely resembles skeletal muscle. . Smooth muscle is nonstriated. Cardiac muscle could almost be said to be a hybrid between skeletal and smooth muscle. Cardiac muscle does have several unique features. Present in cardiac muscle are intercalated discs, which are connections between two adjacent cardiac cells. Intercalated discs help multiple cardiac muscle cells contract rapidly as a unit. This is important for the heart to function properly. Cardiac muscle also can contract more powerfully when it is stretched slightly. When the ventricles are filled, they are stretched beyond their normal resting capacity. The result is a more powerful contraction, ensuring that the maximum amount of blood can be forced from the ventricles and into the arteries with each stroke. This is most noticeable during exercise, when the heart beats rapidly.

There are four chambers in the heart - two _____ and two _____. The atria (one is called an atrium) are responsible for receiving blood from the veins leading to the heart. When they contract, they pump blood into the ventricles. However, the atria do not really have to work that hard. Most of the blood in the atria will flow into the ventricles even if the atria fail to contract. It is the ventricles that are the real workhorses, for they must force the blood away from the heart with sufficient power to push the blood all the way back to the heart (this is where the property of contracting with more force when stretched comes into play). The muscle in the walls of the ventricles is much thicker than the atria. The walls of the heart are really several spirally wrapped muscle layers. This spiral arrangement results in the blood being wrung from the ventricles during contraction. Between the atria and the ventricles are valves, overlapping layers of tissue that allow blood to flow only in one direction. Valves are also present between the ventricles and the vessels leading from it.

Though the brain can cause the heart to speed up or slow down, it does not control the regular beating of the heart. As noted earlier, the heart is composed of involuntary muscle. The muscle fibers of the heart are also self-excitatory. This means they can initiate contraction themselves without receiving signals from the brain. This has been demonstrated many times in high school classes of the past by removing the heart of a frog or turtle, and then stimulating it to contract. The heart continues to beat with no further outside stimulus, sometimes for hours if bathed in the proper solution. In addition, cardiac muscle fibers also contract for a longer period of time than do skeletal muscles. This longer period of contraction gives the blood time to flow out of the heart chambers.



The heart has two areas that initiate impulses, the SA or sinoatrial node, and the AV or atrioventricular node. The heart also has special muscle fibers called Purkinje fibers that conduct impulses five times more rapidly than surrounding cells. The Purkinje fibers form a pathway for conduction of the impulse that ensures that the heart muscle cells contract in the most efficient pattern. The SA node is located in the wall of the right atrium, near the junction of the atrium and the superior vena cava.

This special region of cardiac muscle contracts on its own about 72 times per minute. In contrast, the muscle in the rest of the atrium contracts on its own only 40 or so times per minute. The muscle in the ventricles contracts on its own only 20 or so times per minute. Since the cells in the SA node contract the most times per minute, and because cardiac muscle cells are connected to each other by intercalated discs, the SA node is the pacemaker of the heart. When the SA node initiates a contraction, Purkinje fibers rapidly conduct the impulse to another site near the bottom of the right atrium and near the center of the heart. This region is the AV node, and slows the impulse briefly.

The impulse then travels to a large bundle of Purkinje fibers called the Bundle of His, where they move quickly to the septum that divides the two ventricles. Here, the Purkinje fibers run in two pathways toward the posterior apex of the heart. At the apex, the paths turn in opposite directions, one running to the right ventricle, and one running to the left. The result is that while the atria are contracting, the impulse is carried quickly to the ventricles. With the AV node holding up the impulse just enough to let the

atria finish their contraction before the ventricles begin to contract, blood can fill the ventricles. And, since the Purkinje fibers have carried the impulse to the apex of the ventricles first, the contraction proceeds from the bottom of the ventricles to the top where the blood leaves the ventricles through the pulmonary arteries and the aorta.



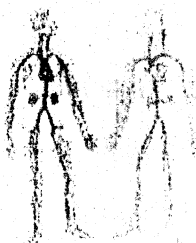
The contraction of the heart and its anatomy cause the distinctive sounds heard when listening to the heart with a stethoscope. The "lub-dub" sound is the sound of the valves in the heart closing. When the atria end their contraction and the ventricles begin to contract, the blood is forced back against the valves between the atria and the ventricles, causing the valves to close. This is the "lub" sound, and signals the beginning of ventricular contraction, known as systole. The "dub" is the sound of the valves closing between the ventricles and their arteries, and signals the beginning of ventricular relaxation, known as diastole.

A physician listening carefully to the heart can detect if the valves are closing completely or not. Instead of a distinctive valve sound, the physician may hear a swishing sound if they are letting blood flow backward. When the swishing is heard tells the physician where the leaky valve is located.



The Pulmonary and Systemic Circuits and the Blood Supply to the Heart.

The heart is responsible for pumping the blood to every cell in the body. It is also responsible for pumping blood to the lungs, where the blood gives up carbon dioxide and takes on oxygen. The heart is able to pump blood to both regions efficiently because there are really two separate circulatory circuits with the heart as the common link. Some authors even refer to the heart as two separate hearts--a right heart in the pulmonary circuit and left heart in the systemic circuit. In the pulmonary circuit, blood leaves the heart through the pulmonary arteries, goes to the lungs, and returns to the heart through the pulmonary veins.

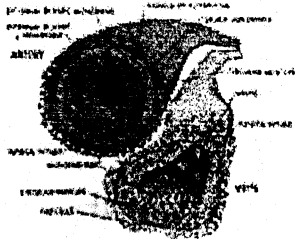


In the systemic circuit, blood leaves the heart through the aorta, goes to all the organs of the body through the systemic arteries, and then returns to the heart through the systemic veins. Thus there are two circuits. Arteries always carry blood away from the heart and veins always carry blood toward the heart. Most of the time, arteries carry oxygenated blood and veins carry deoxygenated blood. There are exceptions. The pulmonary arteries leaving the right ventricle for the lungs carry deoxygenated blood and the pulmonary veins carry oxygenated blood. The blood does not have to travel as far when going from the heart to the lungs as it does from the heart to the toes. It makes sense that the heart would be larger on one side than on the other. The right side of the heart is

distinctly smaller than the left side, and the left ventricle is the largest of the four chambers.

The heart is supplied by its own set of blood vessels. These are the coronary arteries. There are two main ones with two major branches each. They arise from the aorta right after it leaves the heart. The coronary arteries eventually branch into capillary beds that course throughout the heart walls and supply the heart muscle with oxygenated blood. The coronary veins return blood from the heart muscle, but instead of emptying into another larger vein, they empty directly into the right atrium.

The Blood Vessels



Arteries, veins, and capillaries are not anatomically the same. They are not just tubes through which the blood flows. Both arteries and veins have layers of smooth muscle surrounding them. Arteries have a much thicker layer, and many more elastic fibers as well. The largest artery, the aorta leaving the heart, also has cardiac muscle fibers in its walls for the first few inches of its length immediately leaving the heart. Arteries have to expand to accept the blood being forced into them from the heart, and then squeeze this blood on to the veins when the heart relaxes. Arteries have the property of elasticity, meaning that they can expand to accept a volume of blood, then contract and squeeze back to their original size after the pressure is released. A good way to think of them is like a balloon. When you blow into the balloon, it inflates to hold the air. When you release the opening, the balloon squeezes the air

back out. It is the elasticity of the arteries that maintains the pressure on the blood when the heart relaxes, and keeps it flowing forward. If the arteries did not have this property, your blood pressure would be more like 120/0, instead of the 120/80 that is more normal. Arteries branch into arterioles as they get smaller. Arterioles eventually become capillaries, which are very thin and branching.

Capillaries are really more like a web than a branched tube. It is in the capillaries that the exchange between the blood and the cells of the body takes place. Here the blood gives up its carbon dioxide and takes on oxygen. In the special capillaries of the kidneys, the blood gives up many waste products in the formation of urine. Capillary beds are also the sites where white blood cells are able to leave the blood and defend the body against harmful invaders. Capillaries are so small that when you look at blood flowing through them under a microscope, the cells have to pass through in single file. As the capillaries begin to thicken and merge, they become venules. Venules eventually become veins and head back to the heart. Veins do not have as many elastic fibers as arteries. Veins do have valves, which keep the blood from pooling and flowing back to the legs under the influence of gravity. When these valves break down, as often happens in older or inactive people, the blood does flow back and pool in the legs. The result is varicose veins, which often appear as large purplish tubes in the lower legs.



MYOCARDIAL INFARCTION

Acute myocardial infarction (AMI) is the rapid development of myocardial necrosis caused by a critical imbalance between the oxygen supply and demand of the myocardium. This usually results from plaque rupture with thrombus formation in a coronary vessel, resulting in an acute reduction of blood supply to a portion of the myocardium.

Pathophysiology: The most common cause of AMI is narrowing of the epicardial blood vessels due to atheromatous plaques. Plaque rupture with subsequent exposure of the basement membrane results in platelet aggregation, thrombus formation, fibrin accumulation, hemorrhage into the plaque, and varying degrees of vasospasm. This can result in partial or complete occlusion of the vessel and subsequent myocardial ischemia. Total occlusion of the vessel for more than 4-6 hours results in irreversible myocardial necrosis, but reperfusion within this period can salvage the myocardium and reduce morbidity and mortality.

Causes:

- The predominant cause is a rupture of an atherosclerotic plaque with subsequent spasm and clot formation.
- Ventricular hypertrophy (eg, left ventricular hypertrophy [LVH], idiopathic hypertrophic sub aortic stenosis [IHSS], underlying valve disease)

- Hypoxia due to carbon monoxide poisoning or acute pulmonary disorders (Infarcts in this setting usually occur when myocardial demands dramatically are increased relative to blood supply.)
- Emboli to coronary arteries, which may be due to cholesterol or infectious causes
- Coronary artery vasospasm
- Arthritis
- Coronary anomalies, including aneurysms of the coronary arteries
- Cocaine, amphetamines, and ephedrine
 - Increase after load or inotropic effects, which increase myocardial demand
 - Primary vasospasm of the coronary artery
- Risk factors for atherosclerotic plaque formation include the following:
 - Age
 - Being male and younger than 70 years
 - Smoking
 - Hypercholesterolemia and hypertriglyceridemia

- Diabetes mellitus
- Poorly controlled hypertension
- Type A personality
- Family history
- Sedentary lifestyle

ENDOCARDITIS-

Infective endocarditis is defined as an infection of the endocardial surface of the heart, which may include one or more heart valves, the mural endocardium, or a septal defect. Endocarditis can be broken down into the following categories:

- Native valve (acute and sub acute) endocarditis
- Prosthetic valve (early and late) endocarditis
- Endocarditis related to intravenous drug use

Native valve endocarditis (acute and sub acute)

Native valve acute endocarditis usually has an aggressive course. Virulent organisms, such as *Staphylococcus aureus* and group B streptococci, are typically the causative agents of this type of endocarditis. Underlying structural valve disease may not be present.

Sub acute endocarditis usually has a more indolent course than the acute form. Alpha-hemolytic streptococci or enterococci, usually in the setting of underlying structural valve disease, typically are the causative agents of this type of endocarditis.

Prosthetic valve endocarditis (early and late)

Early prosthetic valve endocarditis occurs within 60 days of valve implantation. Staphylococci, gram-negative bacilli, and Candida species are the common infecting organisms.

Late prosthetic valve endocarditis occurs 60 days or more after valve implantation. Staphylococcus epidermidis, alpha-hemolytic streptococci, and enterococci are the common causative organisms.

Endocarditis related to intravenous drug use

Endocarditis in intravenous drug abusers commonly involves the tricuspid valve. S aureus is the most common causative organism.

Pathophysiology: Infective endocarditis generally occurs as a consequence of nonbacterial thrombotic endocarditis, which results from turbulence or trauma to the endothelial surface of the heart. Transient bacteremia then leads to seeding of lesions with adherent bacteria, and infective endocarditis develops.

Pathologic effects due to infection can include local tissue destruction and embolic phenomena. In addition, secondary autoimmune effects, such as immune complex glomerulonephritis and vasculitis, can occur

PERICARDITIS

Pericarditis and cardiac tamponade are clinical problems involving the potential space surrounding the heart or pericardium. Pericarditis is one cause of fluid accumulation in this potential space; cardiac tamponade is the hemodynamic result of fluid accumulation.

The use of limited echocardiography by emergency physicians has enhanced the diagnosis of cardiac tamponade from a variety of causes, including trauma and infectious and noninfectious etiologies.

Pathophysiology: The pericardium (pericardial complex) consists of an outer fibrous layer and an inner serous layer. The fibrous pericardium is a flask-shaped, tough outer sac with attachments to the diaphragm, sternum, and costal cartilage. The serous layer is thin and is adjacent to the surface of the heart. The pericardium serves as a protective barrier from the spread of infection or inflammation from adjacent structures.

The potential space produced by these layers contains approximately 20 cc of fluid with electrolyte and protein profiles similar to plasma. Approximately 120 cc of additional fluid can accumulate in the pericardium without an increase in pressure. Further fluid accumulation can result in marked increases in pericardial pressure, eliciting decreased cardiac output and hypotension (cardiac tamponade). The rapidity of fluid accumulation influences the hemodynamic effect

MYOCARDITIS

Myocarditis is an uncommon disease that is characterized by inflammation of the heart. Subsequent myocardial destruction often leads to a dilated cardiomyopathy.

The acute picture is nonspecific unless overt congestive heart failure develops. Although the causes of myocarditis are numerous, the most common association is an antecedent viral syndrome.

Pathophysiology: Myocarditis is defined as inflammatory changes in the heart muscle and is characterized by an interstitial mononuclear cell infiltrate with attendant myocyte necrosis.

It is not known whether the infiltrate is caused by a direct invasion of the infective agents or by a systemic immune response. In the chronic stage, cytotoxic T lymphocytes infiltrate the myocardium and mediate an autoimmune response with myocardial autoantibody activity directed against cardiac myosin. This autoimmune process persists after the viral particles are no longer detected. Coronary artery thrombus formation, luminal obstruction, ischemia, and dysrhythmias compound the deleterious effects of the inflammatory response.

Myocardial failure

Is characterized by the presence of a weak pulse, pallor, cold extremities, with exercise intolerance and sometimes the development of perennial azotaemia.

PUMP FAILURE

Problems with ejection (systolic dysfunction) include pump failure and outflow obstruction

Pump failure is caused by ischemia, overload, contusion, and inflammation

1. Problems with the pump itself: the heart is a muscle, and if there is damage to the muscle it will not pump effectively. This may be due to inadequate functioning muscle mass, as occurs with ischemia, contusion (bruising in trauma), inflammation (myocarditis) and fibrosis, or to excessive stretch, with excessive fluid administration or valvular incompetence (e.g. aortic regurgitation). With each of these, confirmatory evidence may be available, electro-cardio graphic or echocardiographic evidence of acute ischemia. The patient may give a good history of chest pain or trauma, cardiac enzymes may be positive, and murmurs may be audible. Do not forget the right ventricle: right ventricular contusion or infarction may be much more difficult to diagnose and the treatment is almost the polar opposite of that of left ventricular failure. The pump may be overwhelmed by excessive volume administration, or valvular (aortic or pulmonary) regurgitation.

- 2. Outflow Cardiac outflow obstruction is caused by pulmonary embolism, aortic stenosis, aortic cross clamps

Cardiac outflow obstruction: there are two major sites that cardiac outflow may be blocked: at the level of the aortic valve (aortic stenosis) or within the low pressure (at thus easily occluded)

Pulmonary circulation – pulmonary embolism. The former can be diagnosed on the basis of history, ECG and classic murmur. The latter may be more difficult to diagnose. Useful information includes risk (cancer).

ELECTROCARDIO
GRAPHY

ELECTROCARDIO
GRAPHY

ELECTROCARDIOGRAM

BASIC PRINCIPLE

Electrical activity is a basic characteristic of heart and is the stimulus for cardiac contraction when the heart contracts, electric currents are produced and distributed throughout the body. One can apply two electrodes to any two part of the body to read the current through a recording galvanometer. The graphic representation of these electric currents with respect to time is called an electrocardiogram. William Einthoven introduced the string galvanometer in 1901. In 1933, Frank N Wilson and his associates added unipolar electrocardiography. The electrocardiogram is the most important laboratory test in the diagnosis of various heart diseases, particularly myocardial infarction and is also an extremely useful aid in diagnosis in various non cardiac disorders such as thyroid diseases , renal diseases, pulmonary diseases and various electrolyte imbalances especially hypocalcaemia, hyperkalemia, hypo and hypocalcaemia.

NORMAL ELECTRICAL ACTIVITY OF THE HEART

The normal process of activation begins in the SA node and spreads through the atria in a lateral and downward direction. Since the atria are thin walled structures, little electrical activity results from their depolarization – P wave. An electrode placed on the left side of the body will record an upright P wave, on the right side, a negative P wave.

The wave of depolarization then activates the AV node where there is a 1/10 seconds delay. During this time the electrical activity moves very slowly through the AV node and then in to the ventricles through the

proximal portion of the ventricular conducting system, the bundle of HIS and the bundle of bundle branches, the septum being activated from left to right. All these structures are too small that electrical activity within them is not detected and on the ECG movement of the base line is seen-the isoelectric PR interval.

Activation spreads into the main mass of ventricular muscle from the subendocardial region outwards. Electrocardiographically the ventricles are made up of three muscle groups – right ventricle ,interventricular septum and the left ventricle .The first portion of the ventricular depolarization I the ECG results from septal depolarization from left to right. Since the septum is smaller than the bulk the myocardium, this deflection is relatively small-q wave.

The depolarization then spreads outwards simultaneously through the free ventricular walls from endocrinal to epicardial surface. The thick left ventricular electrical force counteracts the smaller right ventricular force. A large upright deflection R is thus produced in a left-sided electrode. Late activation of an upper part of the right ventricle produces a late negative deflection S. The QRS pattern recorded by a left-sided electrode is mirrored by an RSR pattern in an electrode on the right side of the chest. After the ventricle has been totally depolarized, there is no electrical activity for a brief period until repolarization begins-ST segment.

Repolarization, that is the return of myocardial cells to their resulting negative potential then proceeds from endocardium to epicardium. Ventricular repolarization produces-T wave. The recovery process is much lower than activation and the T wave is generally a broad

deflection in a similar direction as rule to them main wave of QRS complex. After the conclusion of repolarization their again a period of electrical inactivity and the base line of ECG remain isoelectric until the next impulse originate produce the next series.

ECG-lead

An electrocardiography lead is a recording electrode or a pair of recording electrodes at a specified location. In clinical practice, twelve leads are usually used in the diagnostic EKG, although there is no limitation to the number of leads one may select for special purposes.

The leads are usually placed on the wrists and ankles but since the limbs serve as electrical cables, the electrodes behave as if they were at the shoulders and groin. A right leg electrode is used as the ground. There are three of these leads which are usually designated as I, II and III They are all bipolar and detect an electrical potential change in the frontal plane.

Lead I is between the right arm and left arm electrodes, the left arm being positive.

Lead II is between the right arm and left leg electrodes, the left leg being positive.

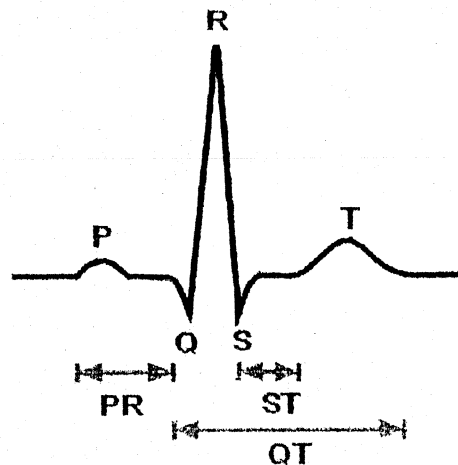
Lead III is between the left arm and left leg electrodes, the left leg again being positive.

A diagrammatic representation of these three leads is termed Einthoven's triangle. The central source of electrical potential in the triangle is the heart. The same three leads that form the standard leads also form the three

unipolar leads known as the augmented leads. These three leads are referred to as aVR (right arm), aVL (left arm) and aVF (left leg) and also record a change in electric potential in the frontal plane. These leads are unipolar in that they measure the electric potential at one point with respect to a null point. This null point is obtained for each lead by adding the potential from the other two leads. These six unipolar leads, each in a different position on the chest, record the electric potential changes in the heart in a cross sectional plane. Each lead records the electrical variations that occur directly under the electrode

.NORMAL ELECTROCARDIOGRAM

As the heart undergoes depolarization and repolarization, the electrical currents that are generated spread not only within the heart, but also throughout the body. This electrical activity is generally measured by an array of electrodes placed on the body surface and the resulting tracing is called an electrocardiogram (ECG, or EKG). A "typical" ECG tracing is shown below. The different waves that comprise the ECG represent the sequence of depolarization and repolarization of the atria and ventricles.



The **P-wave** represents the wave of depolarization that spreads from the SA node throughout the atria and is usually 0.08 to 0.1 seconds (80-100 ms) in duration. The brief isoelectric (zero voltage) period after the P-wave represents the time in which the impulse is traveling within the AV node where the conduction velocity is greatly retarded.

The period of time from the onset of the P-wave to the beginning of the QRS is termed the PR interval and normally ranges from 0.12 to 0.20 seconds. This interval represents the time between the onset of atrial depolarization and the onset of ventricular depolarization. If the PR interval is >0.2 sec, a conduction defect (usually within the AV node) is present (first-degree heart block).

The **QRS complex** represents ventricular depolarization. The duration of the QRS complex is normally 0.06 to 0.1 seconds indicating that ventricular depolarization normally occurs very rapidly. If the QRS complex is prolonged (> 0.1 sec), conduction is impaired within the ventricles. This can occur with bundle branch blocks or whenever a ventricular foci becomes the pacemaker driving the ventricle. Such an ectopic foci nearly always results in impulses being conducted over slower pathways within the heart, thereby increasing the time for depolarization and the duration of the QRS complex.

The isoelectric period (**ST segment**) following the QRS is the time at which the entire ventricle is depolarized and roughly corresponds to the plateau phase of the ventricular action potential. The ST segment is important in the diagnosis of ventricular ischemia or hypoxia because under those conditions, the ST segment can become either depressed or elevated.

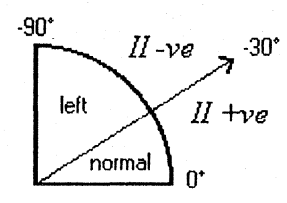
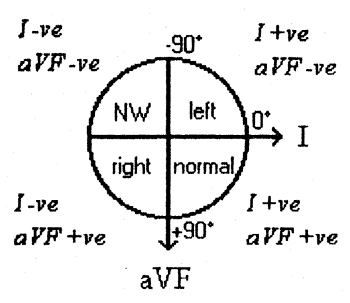
The **T-wave** represents ventricular repolarization and is longer in duration than depolarization (i.e., conduction of the repolarization wave is slower than the wave of depolarization).

The **QT interval** represents the time for both ventricular depolarization and repolarization to occur, and therefore roughly estimates the duration of an average ventricular action potential. This interval can range from 0.2 to 0.4 seconds depending upon heart rate. At high heart rates, ventricular action potentials shorten in duration, which decreases the QT interval. Because prolonged QT intervals can be diagnostic for susceptibility to certain types of arrhythmias, it is important to determine if a given QT interval is excessively long. In practice, the QT interval is expressed as a "corrected QT (**QTc**)" by taking the QT interval and dividing it by the square root of the RR interval (interval between ventricular depolarizations). This allows an assessment of the QT interval that is independent of heart rate. Normal corrected QTc intervals are less than 0.44 seconds.

There is no distinctly visible wave representing atrial repolarization in the ECG because it occurs during, it is masked by the much larger ventricular-generated QRS complex. ventricular depolarization. Because the wave of atrial repolarization is relatively small in amplitude

The electrical axis

Using leads I and aVF the axis can be calculated to within one of the four quadrants at a glance. If the axis is in the "left" quadrant take your second glance at lead II.



both I and aVF +ve = normal axis

both I and aVF -ve = axis in the Northwest Territory

lead I -ve and aVF +ve = right axis deviation

lead I +ve and aVF -ve

lead II +ve = normal axis

lead II -ve = left axis deviation

ELECTROCARDIOGRAPH MACHINE

An ECG electrocardiograph measures the difference in electric potentials between two points on a body and records the temporal difference in waveform

A potential is in the order of $1/1000$ v and is measured by applying detecting electrodes on the body. Waveform recorded on an ECG is of two kinds: the QRS-complex, which shows comparatively rapid change in form, and the P- and T- waveforms, which indicate gradual changes.

These waves are considered to have been formed from sine waves of different frequencies. Fourier analysis of ECG wave shows that they are the composite of various amplitude of sine waves.

CLASSIFICATION BY FUNCTION

Electrocardiographs fall into one of three main types: manual, automatic, and microcomputer. Each type has special features in addition to the basic functions of ECG recording.

The Manual ECG

Conventionally, the manual ECG has been most widely used type although it requires an operator to be on hand all the time for every aspect of its operation.

The Automatic ECG

With the automatic ECG, lead selection, baseline position adjustment, recording sensitivity selection and a number of other functions can be performed automatically.

The Micro-computer ECG

The Micro-computer ECG is the most up-to-date type of the three. A screen is used to display a wide range of information including patient data, electrode positioning and operating confirmation, date, time, and heart rate.

CLASSIFICATION BY TYPE OF RECORDER

There is a wide choice of recorders for ECG equipment. They include photographic, ink injection and thermal head printers. Recorders vary widely in frequency characteristics and suitability for a wide range of recording application including phonocardiogram recording.

In addition, there is also the thermal pen recorder, which is most frequently used at present. It is a direct recorder and can be sub-divided into two types: the edge type and the liner recording type.

CLASSIFICATION BY POWER SUPPLY SYSTEM

The power supply system required to operate an ECG can be either AC or DC. Although an AC power source is most frequently used, there are certain ECGs that use a DC power source either in the form of dry cell batteries or a rechargeable battery, depending on the type of ECG or operating requirements. In addition to single power source ECGs there are also ECGs that can be powered by both an AC or DC power source.

CLASSIFICATION BY THE NUMBER OF CHANNELS

ECGs are classified into two types according to whether they are single-channeled or multi-channeled.

Single-channel ECGs are most frequently used in clinical situations which their performance and ease of operation are satisfactory. A compact light weight ECG is also available for carrying out tests in a non-clinical settings.

Multi-Channel ECGs are used for recording a number of different leads at one time . Since these have the capacity to analyze time phase between leads, they are used in studying the stimuli conduction process.

SIGNIFICANT OE AUTOMATIC ECG ANALYSIS

The application of automatic analysis of ECGs and the results they produce are constantly gaining significance because of the contribution they are making for assisting medical diagnosis.

ECG Storage and Time Series Analysis

Utilization of a computer storage system permits individual repetitive ECGs to be filled and compared, thereby facilitating identification of ECG changes over a period of time.

CONNECTING THE GROUND WIRE

The first, and one of the most important steps to be taken in the preparation of an ECG unit prior to operation, is connection of the GROUND WIRE. Connecting the ground terminal on the ECG unit to the terminal on the wall by means of the ground wire, if a ground AC outlet is not provided.

To ground a bed or shielded room, or when using in conjunction with other ME equipment for a single patient , collect all the ground wires together and

connect them to an appropriate wall ground terminals. This procedure is known as equi-potential grounding.

SETTING THE CONTROLS

The main controls of an ECG and their functions are described below

- Power Switch (POWER)OFF
- Lead Selection (LEAD)1mv (CAL)
- Recording Swiytch (RECORD, CHECK, STOP).....STOP
- Sensitivity Selector (SENSETIVITY).....1
- Pen Position Control (POSITION).....Approximately central
- Stylus Heat Control (STYLUS HEAT).....Approximately centre
- Paper Speed25 mm/sec
- Hum Filter (HUM FILTER).....OFF

Before switching on, make sure that the lead selector is set to 1 mV and the recording switch is off. Connecting and disconnecting the power source should always be carried out by holding the plug and never be done by pulling on the power cord.

Loading the Recording Paper

Loading procedure may differ slightly according to the manual to ensure that the paper is loaded correctly. It is standard practice to feed a small selection of paper through the unit before operation to check that paper moves smoothly.

ADJUSTING THE 1mV TEST WAVEFORM

Adjust the position of the pen so that it is positioned at the center of the recording paper. Press the 1mV SWITCH, record the calibration curve .

1mV Test Waveform

Check the rise in the 1mV test waveform for adjustment or damping after accurately setting pen pressure and temperature. Use the damping adjuster to obtain the correct waveform. An accurate 1mV test waveform is a reliable indication that the ECG unit is functioning correctly. Once this has been achieved, proceed to the next step.

FREQUENCIES

The number of times a type of wave appears per second is called the frequency. A number of frequencies are included in the electrical phenomena produced by the human body. The frequency in ECG is 0.5-200Hz. Approx 1mV

FILTER

The filter is defined as a circuit used to transmit or shut off a required signal simply by changing the frequency characteristic of an electrical circuit.

A Hum filter intercepts only signals of commercial power frequency of 50Hz or 60Hz when it is impossible to eliminate the interference of AC hums. By application of the hum filter, a maximum noise attenuation of about one-tenth can be achieved.

**AMBULATORY
ELECTROCARDIO
GRAPHY**

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ELECTROCARDIO
GRAPHY**

AMBULATORY ELECTROCARDIOGRAM

Ambulatory electrocardiography is also called Holter monitoring, ambulatory ECG or ambulatory EKG. In it, a patient wears a small recorder called a Holter monitor as he or she goes about normal daily life. The machine makes a graphic record of the heart's electrical currents.

Ambulatory EKG is mainly used to document and describe abnormal electrical activity in the heart. This can be random, spontaneous, sleep-related or caused by emotion or stress. Capturing and relating symptoms with rhythm disturbances (changes in the normal electrical pattern of the electrocardiogram) during activity requires recording or observing the heart's electrical activity during that time. This must be done continuously over time as a person goes about normal daily activities. There are two basic types of recording devices. Continuous recorders are most often used for 24-48 hours. Intermittent recorders are used for weeks to months to provide brief, intermittent recordings.

ELECTRODE PLACEMENT

Holter monitor system utilizes a bipolar electrode system. This consists of three electrodes the exploring (colored red), indifferent (colored white) and ground electrode (green). Two basic electrode-positioning systems are used, although any suitable modification is acceptable. The general application is a bipolar modification of lead V4 or V5. In this system, the exploring electrode is placed over the fifth rib in the midclavicular line; the indifferent electrode is placed high over the sternum, and the ground electrode over the fifth rib in the right midclavicular line

Before attaching the electrodes the skin should be shaved and defatted with acetone, and antiperspirant should be applied and allowed to dry. After the

leads are securely applied, loops of the connecting wire from each lead should be tapped into the patients to protect against sudden tension on the wire disconnected a lead. The lead system is then connected to a conventional ECG to verify the atrility of the lead morphology and baseline steadiness. The lead contains fresh battery supply and recodable flash card. The time the monitor activated is recorded in the patient's diary and patients can be dismissed. The monitor can be carried over the shoulder or connected to a belt.

LOCATION OF ELECTRODES

| Channel | Polarity | Location |
|-------------|------------|---|
| Ch -1 V5 | -Ve White | Rt manubrial border of sternum |
| | +Ve Red | Lt anterior auxiliary line rib |
| Ch-2 V1 | -ve Black | Lt manubrial border of sternum |
| | +Ve brown | 2cm right to the Xiph process on the rib margin |
| Ch-3 V3 | -Ve blue | Centered on manubrium |
| | +Ve Orange | Lt mid clavicular line, rib |
| Ground | Green | Lower rt rib margin of the bone |

INSTRUCTIONS TO PATIENT

- 1 Patient must be told to keep the recorder and electrodes dry
- 2 He must be advised not to touch or displace the electrode during the recording period because this may produce artifact in the recording
- 3 Patient should be presented with the patient diary and instructed to keep a careful recording of the daily activities symptoms and the time of their occurrence
- 4 He should be reminded not to forget to return the diary when he returns the recorder
- 5 Patient should be given a list of types of activities to record such as exercise, walking stairs and other daily activities.

INDICATION FOR HOLTER MONITOR ELECTROCARDIOGRAM

A. Diagnosis of cardiac arrhythmias

Extra systoles

Tachy arrhythmias

Brady arrhythmias

Conduction disturbances

WPW syndrome

B. Evaluations of various syndromes to correlate with actual arrhythmias and patient activity

1 Syncope

2 Palpitations

3 Irregular pulses

4 Dyspnea

C. Diagnosis of myocardial ischemia

1A typical ischemia

D. Evaluation of anti arrhythmic drug therapy

1Efficiency of drugs

2Toxicity of drugs

E. Evaluation of artificial pacemaker function

1Assessment of normally functioning pacemaker

2Diagnosis of malfunction of pacemaker

G Miscellaneous

1 Follow up of MI

2Intermittent bundle branch block

3Evaluation of various drugs

VALUE OF HOLTER MONITOR ECG VERSES EXERCISE ECG

| | Holter Monitor ECG | Exercise ECG |
|---|--------------------|---------------------------|
| Cardiac arrhythmias | Excellent | Good |
| Myocardial Ischemia | Good | Excellent |
| Evaluation of symptoms (Dizziness, syncope, palpitations etc) | Excellent | Excellent (Chest Pain) |
| Evaluation of drug efficiency | Good | Very good |
| Evaluation of artificial pacemaker | | No value |

Departmental Equipment (Holter monitor)

Make : Delmar Avionics

Recorder

Digicoder: Solid state recoder Model : 483

Three channel recorder upto fortyeight hours at sampling rate of either 128SPS or 25SPS. ECG is recorded on the flash memory card.

Specifications.

Recording method: Digital, microprocessor controlled, 16 bit CPU.

Memory type : removable, PCMCIA types

Hard disk : 105 MB

Flash memory : 40 MH

Frequency response : 0.05 Hz to 50Hz at 128 samples per second.

Input amplitude : (+/-) 2.5 mV Maximum, normal mode(+/-) in half gain mode

Input impedance : 10Mega Ohm minimum at 10 Hz.

CMRR :80 dB minimum at 50/60 Hz.

Sample rate : Analog to digital 128SPS or 256 SPS

User interface: LCD display, 2 line , 16-characters

Analog out put:Three channel ECG , 1 m.v

Power:One nine volt alkaline battery

Weight:333 gram

TREADMILL TEST

TREADMILL TEST

TREAD MILL TEST

The stress test is one of the popular objectives aiding to the diagnosis of angina pectoris. Modern stress testing is based on the empirical discovery that exercises in patients with coronary disease produce ST segment depression. This discovery might be credited to Bous Field who recorded st segment depression. In the 3 standard ECG leads, during spontaneous attack of angina in 1918, changes as being due to the decrease in blood flow to the heart and the changes return to normal after pain had subsided and also the administration of nitroglycerine. Modern stress testing might be dated from 1956 when Bruce reported a work test performed on Treadmill. Many of the protocols of treadmill stress testing have been based on the extension of the principle Bruce established that time. Shortly before this Astrand and Rhyimming had documented that maximum oxygen uptake or aerobic capacity could be predicted by the heart rate at the sub maximal exercise. Thus the groundwork is necessary to establish the progressive exercise list as a physiological exercise tolerance had been laid.

Conventional exercise today usually done with a treadmill is being supplemented by nuclear technique such as thallium scintigraphy and blood pool nuclear ventriculogram as well as the estimation of wall motion by electrocardiograms and physiological stress test using dipyridamole, dobutamine and adenosine. These techniques combined with conventional testing improve the diagnostic certainty and often help to localize the diseased vessel.

PROTOCOLS

Although different investigators have designed numerous multistage exercise protocols none of these are ideal for an individual. In some exercise protocols speed changes with fixed grade, where as in some others grade is increased with constant speed. In Bruce, change in both grade and speed increases workload. For progressive increment of workload at least 3 min interval is preferable so that steady state blood pressure and heart rate response can be achieved. Metabolic Equivalence (METs), is multiple of basal metabolic rates, is commonly used to express the workload. One MET is defined as 3.5ml of oxygen consumed per kilogram per minute. While correlating cardiovascular function capacity with METs, functional class 3 patients become symptom related at 3-4 mets, class 2 at 5-6 mets, and class 1 should be able to perform beyond 7 or 8 mets.

Different protocols are-

| | |
|------------------|------------|
| 1 Bruce | 7 Ellestad |
| 2 Modified Bruce | 8 Mc Henry |
| 3 Naughton | |
| 4 Cornell | |
| 5 Balke | |
| 6 Modified Balke | |

Blood pressure is measured during each stage. The heart rate and the ecg is continuously monitored. During the recovery phase, the patient is asked to lie down and the measurements are repeated until the blood pressure, heart rate and st changes are stored to normal levels.

Bruce Protocol

| phase | Duration(min) | Speed(mph) | Gradient(%) | Met's |
|-------|---------------|------------|-------------|-------|
| 1 | 3 | 1.7 | 10 | 5 |
| 2 | 3 | 2.5 | 12 | 7 |
| 3 | 3 | 3.4 | 14 | 10 |
| 4 | 3 | 4.2 | 16 | 13 |
| 5 | 3 | 5 | 18 | 16 |
| 6 | 3 | 5.5 | 20 | 19 |
| 7 | 3 | 6 | 22 | 22 |

Modified Bruce Protocol

| Phase | Duration(min) | Speed(mph) | Gradient(%) | Met's |
|-------|---------------|------------|-------------|-------|
| 1 | 3 | 1.7 | 0 | 1.7 |
| 2 | 3 | 1.7 | 5 | 2.8 |
| 3 | 3 | 1.7 | 10 | 5.4 |
| 4 | 3 | 2.5 | 12 | 7 |
| 5 | 3 | 3.4 | 14 | 10 |
| 6 | 3 | 4.2 | 16 | 13 |
| 7 | 3 | 5 | 18 | 17 |

EQUIPMENT NECESSARY IN TMT LAB

1. Treadmill with specially designed electrodes and cables.
2. Single to three channel continuous ECG monitor and recorder.
3. Sphygmomanometer.
4. DC defibrillator
5. Airways, oral and tracheal
6. Oxygenator, intermittent positive pressure capability.
7. Bag-Valve –Mask hand respirator
8. Cut down tray with syringes & needles, intravenous stand
9. Stethoscope
10. Laryngoscope

COMMONLY USED DRUGS

1. Antiarrhythmic drugs
2. Cardiac glycosides
3. Atropine
4. Nitroglycerin
5. Morphine
6. Sodium bicarbonate solution
7. Dextrose, 5% in water

INDICATIONS FOR TMT TEST

1. Confirmation of the diagnosis of CAD
2. Assessment of possible for chest pain of unknown cause
3. Assessment of nature of arrhythmia in relation to exercise
4. Early detection of hypertension

5. Evaluation of functional capacity of patients with CAD
6. Evaluation of efficacy of medical or surgical therapy
7. Evaluation of prognosis in patients with previous MI
8. Rehabilitation of cardiac and non cardiac patients
9. Assessment of functional capacity-

CONTRAINDICATIONS

1. Patients with acute MI
2. Patients suffering from acute myocarditis or pericarditis
3. Patients exhibiting signs of unstable progressive angina

Patients with rapid ventricular or atrial arrhythmias , second or third degree heart block & known left main disease

4. Acutely ill patients such as those with infection, hyperthyroidism or severe anemia
5. Patients with locomotion problems
6. severe AS
7. Acute pulmonary embolism

INDICATION TO TERMINATE TMT

Absolute indications

1. Patient's request
2. Reduction of blood pressure and heart rate during increase in work loads
3. Significant symptoms or signs: severe chest pain, ataxia, vertigo, visual or gait disturbances, pallor, cyanosis

4. Serious arrhythmias: grouped (3 or more) VPC's, VT, VF
5. Acute MI
6. Mal functioning equipments in the lab

Relative indications

1. Less serious symptoms: significant chest pain, dizziness, fatigue or dyspnea
2. Marked (2mm or more) horizontal or down sloping ST segment depression or marked (2mm or more) horizontal or upsloping ST segment elevation
3. Marked hypertension (systolic bp above 220mm of hg or diastolic bp above 110mm of hg)
4. Failure of bp to rise during increasing work load (sys: bp rise less than 20mm of hg during the first three stages)
5. Frequent VPC's or multifocal VPC's
6. Persisting SVT's

PATIENT PREPARATION

_____ The patient should be instructed not to eat or smoke for 2-3 hours before the test. The patient must not take any vasodilating drugs or beta blocking agents for at least 24 hours before the performance of the test

A complete chest shaving is needed in case of male patients. A detailed explanation of the procedure should be given, outlining risk and possible complications. Patient should be told how to perform the test and the testing procedure should be demonstrated.

PROCEDURE

The exercise test consists of walking on a motor driven treadmill. ECG will be recorded while the patient is supine, standing as well as resting blood pressure must be obtained before the exercise. Several patterns of graded exercise are in common use. We normally use the Bruce protocol. The workload is increased by changing both speed and grade. At least 3 min intervals per stage are preferable so that steady state BP and heart rate response are obtained. MET refers to the resting VO₂ for an adult 1 MET is equivalent to 3.5ml/min/kg of body weight.

Department equipment

Centra (Marquette)

This device uses computerized ECG analysis program, which can be used as a tooling ECG tracing & interpretation

Important module- Acquisition module: A device that straps around a patient's waist serves as an interface between the patient and workstation.

- The work station advances from phase and /or stage according to the protocol settings
- The work station controls the treadmill belt speed and grade

CARDIAC ARRHYTHMIAS

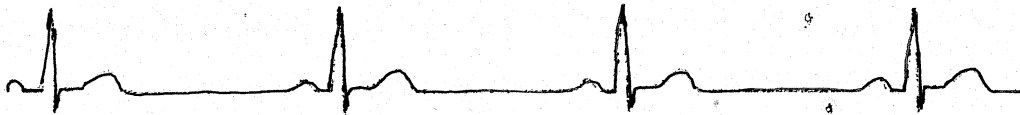
CARDIAC ARRHYTHMIAS

Introduction to Cardiac Arrhythmias

Arrhythmia is a generalized term used to denote disturbances in the heart's rhythm. A regular rhythm and PR interval duration has a range of 0.12 sec - 0.20 sec, characterizes normal sinus rhythm. Evaluating the EKG in a systematic manner can recognize Arrhythmias.

Sinus Bradycardia

Sinus bradycardia occurs when the heart's rate is slower than 60 beats per minute. The sinus bradycardia rhythm is similar to normal sinus rhythm, except that the RR interval is longer. A QRS complex in a ratio of 1:1 follows each P wave. The PR interval is often slightly prolonged and occasionally, the P-waves might be abnormally wide.



The symptoms of sinus bradycardia include dyspnea, dizziness, and extreme fatigue. It may be accompanied by an increase in stroke volume due to greater end diastolic pressure (preload). The pulse volume may be greater due to a greater stroke volume and an increased diastolic run-off time.

Sinus bradycardia may occur due to any of the following:

- a. Increase in parasympathetic (vagal) tone, due to training in athletes.

This is a normal response. The heart rate increases with exercise or atropine.

- b. Parasympathetic (vagal) stimulation, for instance, with carotid sinus stimulation. Stimulation of carotid sinus baroreceptors results in increased parasympathetic stimulation that decreases the heart rate.
- c. Sick sinus syndrome or sinoatrial (SA) node disease. These are rhythm disorders that occur if the SA node loses its ability to initiate or increase the heart rate. If the SA node is unable to properly function due to sick sinus syndrome, the AV node (or ventricular tissue if the AV node is also not functioning) take over the initiation of the heart beat, but at a rate that is slower than the sinus rhythm.
- d. Heart block, which occurs when the signal from the SA node is slowed or stopped at the AV node or in the ventricular conducting system. Heart block is described as first, second, or third degree. The decrease in the heart rate depends on the degree of heart block.
- e. Acute myocardial infarctions.
- f. Drugs like digitalis and beta-blockers.

Sinus Tachycardia

Sinus tachycardia occurs when the sinus rhythm is faster than 100

complex in a ratio of 1:1. At very rapid rates, the P-waves might become superimposed on the preceding T waves such that the P waves are obscured by T waves

Sinus tachycardia may be accompanied by a decrease in stroke volume because the ventricles do not have enough time to fill (after Atrial systole) before ventricular contraction.. The pulse pressure may decrease due to a lower stroke volume and decreased time for diastolic run-off.

Sinus tachycardia results from increased automaticity of the SA node, for instance, due to increased sympathetic stimulation of the heart, fever or cardiac toxicity

Atrial Flutter

Atrial flutter occurs when the atria are stimulated to contract at 200-350 beats per minute usually because electrical impulses are traveling in a circular fashion around and around the atria. Often the impulses are traveling around an obstacle like the mitral valve, tricuspid valve or the openings of the superior or inferior vena cavae.

The atrial flutter waves, known as F waves, are observed. F waves are larger than normal P waves and they have a saw-toothed waveform. Not every atrial flutter wave results in a QRS complex (ventricular depolarization) because the AV node acts as a filter. Some flutter waves reach the AV node when it is refractory and thus are not propagated to the

ventricles. The ventricular rate is usually regular but slower than the atrial rate. A whole number fixed ratio of flutter waves to QRS complexes can be observed, for instance 2:1, 3:1 or 4:1.

Atrial flutter is usually associated with mitral valve disease, pulmonary embolism, thoracic surgery, hypoxia, electrolyte disturbances and hypercalcaemia. Atrial flutter results in poor atrial pumping since some parts of the atria are relaxing while other parts are contracting. Cardiac output decreases because the ventricles do not sufficiently fill (as they would normally) before ventricular contraction. Ablation of some of the heart tissue to stop impulses from traveling around can be used to treat this condition

Atrial Fibrillation

Atrial fibrillation occurs when the atria depolarize repeatedly and in an irregular uncontrolled manner usually at Atrial rate greater than 350 beats per minute. As a result, there is no concerted contraction of the atria. No P-waves are observed in the EKG due to the chaotic Atrial depolarization. The chaotic atrial depolarization waves penetrate the AV node in an irregular manner, resulting in irregular ventricular contractions. The QRS complexes have normal shape, due to normal ventricular conduction. However the RR intervals vary from beat to beat. The ventricular rate may increase to greater than 150 beats per minute if uncontrolled.

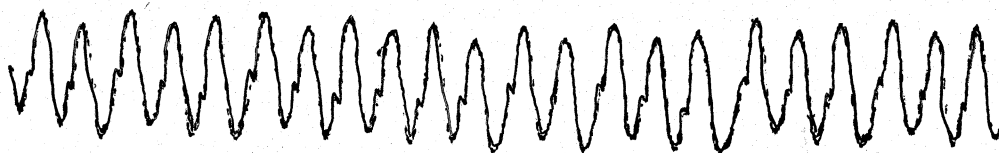


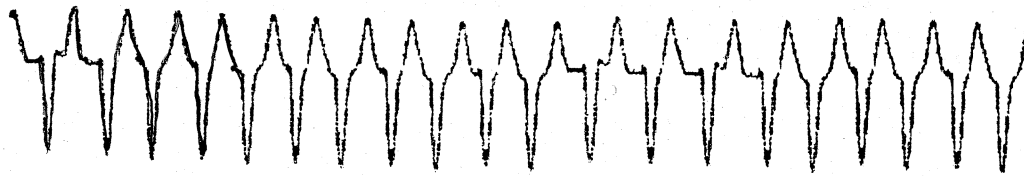
The irregular ventricular contractions cause the systolic arterial pressure to vary from beat to beat as ventricular filling time changes. The pulse pressure also may vary from beat to beat because the diastolic runoff time varies from beat to beat.

Atrial fibrillation often involves micro reentry. Atrial fibrillation is most common in individuals with Atrial enlargement, often associated with valve diseases, sick sinus syndrome, pericarditis, lung disease and congenital heart defects. The incidents of Atrial fibrillation increase with age and are slightly more frequent in men than women.

Ventricular Tachycardia (VT)

Ventricular tachycardia occurs when electrical impulses originating either from the ventricles cause rapid ventricular depolarization (140-250 beats per minute). Since the impulse originates from the ventricles, the QRS complexes are wide and bizarre. Ventricular impulses can be sometimes conducted backwards to the atria. in which case, P-waves may be inverted. Otherwise, regular normal P waves (60-100 beats per minute) may be present but not associated with QRS complexes (AV dissociation). The RR intervals are usually regular.





Ventricular tachycardia is often due to some form of heart disease. Ventricular tachycardia can occur rarely in response to exercise or anxiety. In this case, the electrical impulses and rhythmic beats is similar is a normal beat but at a much faster rate.

During ventricular tachycardia pumping blood is less efficient because the rapid ventricular contractions prevent the ventricles from filling adequately with blood. As a result, less blood is pumped to the body. The reduced blood flow to the body causes weakness, dizziness, and fainting. If left untreated, ventricular tachycardia may lead to a more life-threatening condition. Note, because of the decreased diastolic time, coronary blood flow is decreased, increasing the chances of a myocardial infarction.

Ventricular Fibrillation

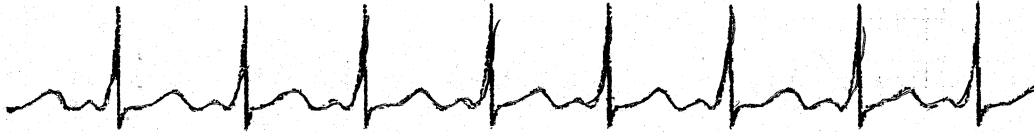
Ventricular fibrillation occurs when parts of the ventricles depolarize repeatedly in an erratic, uncoordinated manner. The EKG in ventricular fibrillation shows random, apparently unrelated waves. Usually, there is no recognizable QRS complex.

Ventricular fibrillation is almost invariably fatal because the uncoordinated contractions of ventricular myocardium result in ineffective pumping and little or no blood flow to the body. There is lack of a pulse and pulse pressure and the patients lose unconsciousness rapidly. When the patient has no pulse and respiration the patient is said to be in cardiac arrest. A person in cardiac arrest must receive CPR immediately.

Electrical defibrillation, by passage of current at high voltage, may be successful in restoration of a normal regular rhythm. The electrical current stimulates each myocardial cell to depolarize simultaneously. Following synchronous repolarization of all ventricular cells, the SA node assumes the role of pacemaker and the ventricular myocardial cells can resume the essentially simultaneous depolarization of normal sinus rhythm. Ventricular fibrillation is associated with drug toxicity, electrocution, drowning and myocardial infarction

Wolff-Parkinson-White Syndrome

Normally, the AV node is the only conduction pathway for impulses from the atria to the ventricles. Wolff-Parkinson-White syndrome is characterized by the presence of an accessory atrioventricular pathway located between the wall of the right or left atria and the ventricles, known as the Bundle of Kent. This pathway allows the impulse to bypass the AV node and activate the ventricles prematurely. Consequently, an initial slur to the QRS complex, known as a delta wave may be observed. The QRS complexes are wide, more than 0.11 sec, indicating that the impulse did not travel through the normal conducting system. The PR is shortened, to less than 0.12 sec, because the delay at the AV node is bypassed.



The accessory pathway can cause a reentry circuit to be established. Reentry is initiated by a premature atrial or ventricular beat coupled with a unidirectional block in one of the pathways (because the normal impulse gets to pathway when it is refractory after the premature beat). The result is continuous impulse conduction. Reentry causes two kinds of tachycardia.

1. Orthodromic AV reentrant tachycardia, which occurs when the impulse is conducted through the AV node with retrograde return to the atria via the Bundle of Kent. The heart rate is usually 140-250 BPM. The QRS complexes are narrow and delta waves are not observed.
2. Antidromic AV reentrant tachycardia, which occurs when the impulse is conducted through the Bundle of Kent with retrograde return to the atria via the AV node. The QRS complexes are wide.

Wolff-Parkinson-White syndrome is commonly associated with congenital heart abnormalities like Tetralogy of Fallot, coarctation of the aorta, tricuspid atresia and transposition of the great vessels. In severe cases, treatment would involve surgical removal or ablation of one of the pathways.

CONVENTIONAL PLACEMENT OF ECG CHEST LEADS

The chest leads (V1-V2) show the electrical current of the heart as detected by electrodes placed at different position on the chest wall. The precordial leads at different positions leads used today are also unipolar leads in that they measure the voltage in any one location relative to zero potential. The chest leads are recorded simply by means of electrodes at six designated locations on the chest wall.

Lead V1 placed on the fourth intercostal space just to the right of the sternum.

Lead V2 placed on the fourth intercostal space just to the left sternum

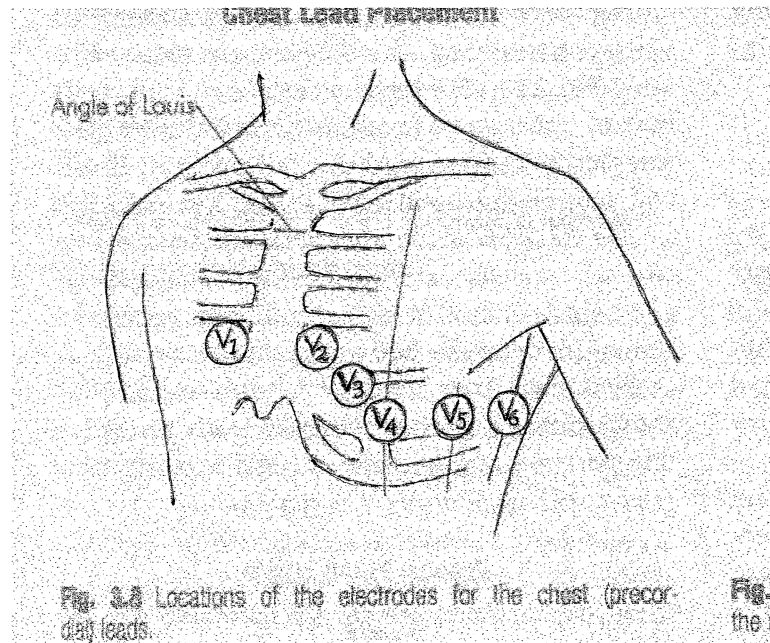
Lead V3 midway between leads V2and V4

LeadV4 placed on the midclavicular line in the fifth interspace.

LeadV5 placed on the anterior auxiliary line at the same level as lead V4

LeadV6 placed on the midaxillary line at the same level as lead V4

The chest leads, like the six extremity leads, can be represented digramatically. Like the other leads each chest lead has a positive and negative pole. The positive pole of each chest lead points anteriorly toward the front of the chest. The negative pole of each chest lead points posterior toward the back



Lewis Lead

This is the chest a right arm lead which is taken with the right arm lead (+ve pole) placed over the left chest wall and left arm lead (-ve pole) placed either on the right arm or the right chest and recorded from lead1 on the ecg machine. This later lead offer particular advantage i.e., the high voltages are recorded frequently making atrial activity more distinct.

ECHOCARDIOGRAPHY

INTRODUCTION

Echocardiography is a unique noninvasive method for imaging the living heart. It is based on detection of echoes produced by a beam of ultrasound (very high frequency sound) pulses transmitted into the heart.

From its introduction in 1954 to the mid 1970's, most echocardiography studies employed a technique called M-mode, in which the ultrasound beam is aimed manually at selected cardiac structures to give a graphic recording of their positions and movements. M-mode recordings permit measurement of cardiac dimensions and detailed analysis of complex motion patterns depending on transducer angulations. They also facilitate analysis of time relationships with other physiological variables such as ECG, heart sounds, and pulse tracings, which can be recorded simultaneously.

A more recent development uses electromechanical or electronic techniques to scan the ultrasound beam rapidly across the heart to produce two-dimensional tomographic images of selected cardiac sections. This gives more information than M-mode about the shape of the heart and also shows the spatial relationships of its structures during the cardiac cycle.

A comprehensive echocardiographic examination, utilizing both M-mode and two dimensional recordings, therefore provides a great deal of information about cardiac anatomy and physiology, the clinical value of which has established echocardiography as a major diagnostic tool.

TRANSDUCERS

Transducers are device capable of converting one form of energy into another. The piezoelectric material converts electrical energy into sound energy fall within this large group of device known as transducers.

The most important feature of the transducers is its characteristic frequency. This is determined by the thickness of the piezo electric element. When the thickness of the element is exactly one half the wavelength the reflected and transmitted stress at each surface reinforce each other and the transducer resonate with maximum displacement amplitude. The frequency that corresponds to half wavelength thickness is called fundamental resonant frequency of the transducer.

Transducer size must be considered because it contributes to the shape of the ultrasonic field. When the area through which the ultrasonic beam can be directed to deeper structures is limited, use of a smaller transducer may be necessary.

CHOISE OF APPROPRIATE TRANSDUCER

Increasing transducer frequency improves axial resolution. The type of transducer one uses depends upon the patient being examined. In adult echocardiography a 2.0 or 2.5megahertz transducer is the one, which is used most frequently. This particular frequency represents a good compromise between penetration and resolution. High frequency transducers have excellent resolution but poor penetration. Low frequency transducers are reverse.

In infants and young children, the problem of penetration is significantly less.

In these patients a higher frequency transducer is usually preferable. A transducer with a frequency of 3.5 or 5.0 MHz is the most useful transducers. The size of transducers is an important factor. A large transducer has a longer near field and thus more parallel, relatively narrow beam because the diverging far field occurs farther from transducers. On the other hand larger transducers are difficult to angulated. For routine adult echocardiography a 1/2 inch diameter transducer is most useful. In young children and infants a 1/4 inch transducer is preferred size. The most common transducer used for pediatric echocardiography would be 1/4 inch transducer with a frequency of either 3.5 or 5 MHz.

The Doppler Principle and the Study of Cardiac Flow

The Frequency of Sound Waves

Conventional two-dimensional echocardiographic systems emit high frequency bursts of sound (ultrasound) into the tissues. In standard echocardiographic imaging a given pulse of ultrasound is transmitted into the body and then reflected back from the various tissues. Since the speed of sound in tissue is known (approximately 1540 m/sec), a standard ultrasound imaging system can wait for a given time for the transmitted pulse to travel to a target (time X) and then back (time 2X) and the given target will be received and recorded. In complex two-dimensional imaging systems this alternating process is repeated in a

variety of directions thousands of times each second. The best ultrasound images are made when the target is perpendicular (or secular) to the sound waves.

Frequency is a fundamental characteristic of any wave phenomenon, including sound, and refers to the number of waves that pass a given point in one second (Fig. 1.4). It is

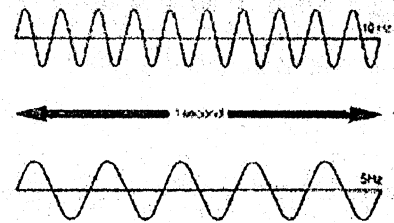


Fig. 1.4

usually described in units of cycles per second or Hertz (Hz). Thus, the top of the illustration in Figure 1.4 shows an example of a waveform of 10 Hz while the one below is 5 Hz. Ultrasound is emitted in waveforms of a known frequency.

Doppler echocardiography, on the other hand, depends entirely on measurement of the relative change in the returned ultrasound frequency when compared to the transmitted frequency. Depending on the relative changes of the returning frequencies, Doppler echocardiographic systems measure these characteristics of disturbed flow: direction, velocity and turbulence. This enables examiners to differentiate between normal and abnormal flow patterns and, in some cases, to quantitate those characteristics that are helpful in determining the severity of abnormal flow states.

Most readers understand frequencies in relationship to the

pitch of audible sound. The relationship between pitch and frequency is simple: the pitch of any given sound is proportional to its frequency. As sound wave frequency increases, pitch gets higher; and as frequency decreases, pitch declines.

Doppler systems are totally dependent on the changes in the frequency of the transmitted ultrasound that result from the encounter of the wave front with moving red blood cells. Figure

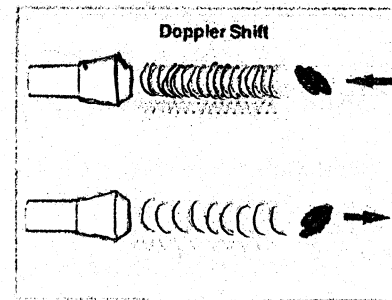


Fig. 1.5

1.5 shows a transducer on the left that is emitting a given frequency of ultrasound toward the right and into the tissues. The transmitted sound waves encounter a group of red cells moving toward the transducer and are reflected back at a frequency higher than that at which they were sent producing a positive Doppler shift. The opposite effect occurs when a given frequency sent into the tissues encounters red cells moving away. The result is the return of a frequency lower than that transmitted, and the Doppler shift is negative.

The use of Doppler echocardiography

In a clinical setting, DE is primarily used for confirming a diagnosis of valvular incompetence based on auscultatory findings, for mapping the area of the jet or regurgitant blood in order to estimate the severity of the condition, and for measuring the velocity of blood shunting in order to determine the pressure gradient.

In a research setting it is a very useful, non-invasive method of estimating stroke volume and cardiac output.

DE allows identification of the timing of blood flow during the cardiac cycle if an ECG is recorded simultaneously. It enables to determine the direction of blood flow. This is usually sufficient information to confirm the diagnosis. Using pulsed-wave Doppler echocardiography this can be combined with spatial information from the 2DE image so that the location of blood flow within the heart can be documented.

The duration of the jet may not appear to last throughout the period during which regurgitation takes place. This may be because the jet moves out of the line of the ultrasound beam during the cardiac cycle. In addition, the heart swings towards the apex during systole so the position of the sample volume relative to the valve and chamber alters during the cardiac cycle.

Pulsed-wave Doppler mapping - Satisfactory 2DE and M-mode views can be obtained from parasternal views because the chambers lie perpendicular to the line of the beam and therefore return strong

signals. When using DE to measure blood flow, the line of the beam should be as close to parallel with blood flow as possible, and this is seldom achievable. A second problem is that many jets of regurgitant blood flow, particularly mitral regurgitation, do not flow in predictable patterns and can be difficult to detect. Auscultation is probably just as accurate a method of identifying regurgitant blood flow. PWD mapping is a useful method of assessing the severity of valvular disease.

For evaluation of MR, a left parasternal long-axis view is obtained, and the transducer are then moved slightly ventral and angled steeply dorsal so that the LA is in the far field and the MV is as near perpendicular to the valve as possible. An angle of around 45° to the valve is usually achievable. The sample volume is then placed behind the valve and moved along the atrial side of the valve until a regurgitant jet is detected. The area of the origin of the jet is mapped out if possible, and then the sample volume is moved gradually further and further into the atrium. The process should be repeated with the image plane slightly altered, remembering that the jet and the atrium are three-dimensional. Using color-flow Doppler, the process is far less time-consuming, but multiple image planes should be examined. Jets of MR often run up the walls of the atrium, or some run straight into it.

For examination of tricuspid regurgitation (TR), a right parasternal long-axis view optimized for the LV outflow tract is obtained. The transducer is then tilted slightly dorsal and the sample volume is

placed on the atrial side on the tricuspid valve. Jets usually run down the atrial wall where it borders the aorta.

Mapping of jets of AR is less helpful than for AV valve regurgitation. It is difficult to get a good angle on the valve and the whole of the LV at the same time. A tilted left parasternal long-axis view with the transducer aimed cranially and dorsally is best.

Jets of regurgitant flow which are detected at or close to the valve in early systole (AV valves) or diastole (semi lunar valves) can be regarded as normal. There is no clear cut-off point between normality and abnormality, particularly in the case of tricuspid regurgitation. As a general rule, significant jets are much more extensive and are more easily detected. They may be graded from mild to severe on the grounds of the extent of the jet. This is an estimate rather than a quantitative method because other factors may influence the size of the jet, including the control settings of the ultrasound machine and the quality of the Doppler unit.

Estimation of pressure gradients

Measurement of the velocity of a jet allows the pressure gradient between the two chambers between which the jet is flowing to be estimated. For this, simplified version of the Bernoulli equation is used. If the pressure within one chamber is known, the pressure within another can be estimated if a regurgitant jet of blood flows between them. This is seldom helpful in the case of assessment of the severity of valvular regurgitation.

The principles of echocardiography

2DE and M-mode echocardiography

Ultrasound is high-frequency sound, which is produced when a piezoelectric crystal, mounted in a transducer, is stimulated by an electrical current. The sound waves are too high in frequency to be audible. They are thought to be harmless to tissue at the intensities used in diagnostic imaging. The passage of sound waves depends on the acoustic impedance of body tissues. Sound is reflected by interfaces between materials of different acoustic impedance.

The majority of the ultrasound waves pass through structures on to other structures lying further from the surface, but reflected sound returns to strike the crystal, deforming it and producing electric signals which correspond to the degree of deformation. This electrical information is transformed by electronics in the ultrasound machine so that it can be displayed on a cathode-ray tube as pulses of light. Because the speed of sound within the body is relatively constant, the depth of the tissue interface can be known and reflected echoes are displayed on the screen on a depth scale.

In echocardiography, sound is directed into the body and is reflected by interfaces between tissues of different acoustic impedance such as myocardium, valves and blood. Blood reflects little sound so it appears relatively black (hypo echoic, or anechoic) compared with the myocardium which reflects more of the ultrasound and therefore

appears relatively white hyper echoic or echo The endocardium and valves are the most echogenic structures. Ultra does not pass through air or bone. Because the heart is surrounded by lung over the majority of its surface and is contained within the bony cage of the thoracic cavity, the ultrasound beam must be aimed through gaps (which are known as acoustic windows), in order to produce images of the heart.

Frequencies of 2-10 MHz are used in diagnostic ultrasound. The lower end of this range is used in transducers designed for equine echocardiography because low frequency sound penetrates a greater depth of tissue. A trade-off in the use of low frequency transducers is that there is a loss of image detail because the increased wavelength results in reduced resolution.

M-mode echocardiography and two-dimensional echocardiography (2DE) are the two techniques which are usually used to produce images of the heart in real-time. In M-mode echocardiography the crystal is stationary and the beam produced is a pencil-beam of sound. The signal is produced almost continuously. Echoes are displayed on the screen on the Y-axis, with time displayed on the X-axis. This produces an almost continuous image of the position of the cardiac structures, which are in the line of the beam.

Echocardiography has become more popular since the advent of 2DE, which produces an image of cardiac structure. 2DE images are also referred to as B-mode images (brightness mode). Originally, static B-mode images were probut improvements in technology allowed the image to be rapidly updated resulting in a real-time image. 2DE

images are more easily understood than Mtraces because of the greater spatial detail.

A 2DE image can be produced in one of three ways. The first and simplest is to have multiple crystals mounted in line to produce a 'curtain' of sound. This method is known as linear array and is used in many rectal probes in current use in veterinary medicine. The problem with the use of this technology for echo-cardiography is that it is difficult to aim the curtain of sound through the acoustic windows, so some of the heart is likely to be obscured from view and only a limited number of views can be obtained. This prevents a complete echo investigation, although it may allow detection of gross abnormalities such as a pericardial effusion, very poor myocardial contractility, or large vegetations on the heart valves.

To be able to examine the heart in a large number of image planes, a point source is required with a sector or 'fan' of sound produced by sweeping the sound in an arc. Transducers, which produce this arc of sound, are called sector scan the sector can be produced by rotating or oscillating the crystal mechanically; this type of transducer is called a mechanical sector scanner. Alternatively the sector can be produced by using an array of crystals, which are electronically stimulated in sequence to produce a fan-shaped beam. These are known as phased-array transducers. Because the beam has to be swept through an arc, a finite time is taken to produce each sector image. The arc is then repeated and the image is updated. The quality of the image therefore depends on how many lines of data are displayed per arc and how

often the image is updated. The rate at which the image is updated is known as the frame rate.

One of the main problems in equine echocardiography is the great depth, which is required for the whole heart to be displayed on the screen. Because the transducer has to wait longer for the sound to be reflected back from tissue interfaces at greater depth, the whole arc takes much longer to produce than those with less depth, and the frame rate is relatively low. Fortunately, the sign of this is offset to some extent by the slow resting heart rate in the horse, which means that the number of frames per cardiac cycle is much the same in equine echocardiography as it is in human echocardiography.

Doppler echocardiography

The principles of Doppler echocardiography (DE) are somewhat more complex than those of ultrasound imaging. DE has been used in equine medicine since the late 1980s and has proved particularly valuable in evaluation of congenital heart disease and acquired valvular heart disease. It is used to provide information about blood flow, following evaluation of the structure and the size of the heart using 2DE and M-mode studies. DE is also very useful in a research setting for assessment of cardiac function. A thorough DE examination requires rigorous technique and is extremely time-consuming. Equipment for DE is still relatively costly and the technique is likely to remain limited to referral centers for the foreseeable future. Recently, color-coded DE has become available to a few institutions. This technique reduces the time required for

Doppler examinations and makes them easier to perform, but has the same physical limitations.

The Doppler principle is evident to anyone standing by a railway line or a busy road as traffic moves past: the pitch of the sound of an engine drops as the vehicles pass. This is because sound waves are compressed as a source of sound moves towards an observer and are therefore higher in frequency than those emitted when the source is traveling away from the observer. The principle also applies to sound, which is reflected off a moving target. The change in frequency of the sound is proportional to the velocity of the target in relation to the source of the sound.

DE involves the emission of ultrasound waves of known velocity, which are reflected from interfaces and return to the transducer, in the same way as for echocardiographic imaging. The equipment detects changes in the frequency of the reflected sound in comparison to the emitted sound. In DE, the most important interfaces, which reflect the sound, are red blood cells (RBCs). Thus, if the emitted ultrasound is reflected off RBCs moving towards the transducer the reflected sound will be of a higher frequency (and shorter wavelength) than the emitted sound. The change in frequency (frequency shift) is proportional to the velocity of the cells towards the transducer. A computer inside the Doppler unit calculates the velocity of the moving blood from the Doppler shift equation. The calculated velocity is displayed on a velocity/time graph with blood flow towards the transducer displayed above a baseline and flow away displayed below it. This form of display is known as spectral

Doppler. The standard form of DE, in which the transducer emits and receives sound waves simultaneously, is known as continuous-wave (CW) Doppler.

A critical feature of the Doppler shift equation is the effect on the angle of the beam to blood flow. If the cells are moving in a direction oblique to the line of the ultrasound beam, the velocity calculated will be an underestimate of their true velocity unless this angle is known and can be included in the calculation. Unfortunately, the exact angle is seldom known. Estimating it and using the angle correction software which is available on many machines usually only adds to the potential inaccuracy of the method. This means that, at all times, every effort should be made to keep the line of the ultrasound beam parallel to blood flow. In practice, angles under 15° either side of parallel are acceptable because the error will be less than 4%.

Initially, DE was performed independently of echocardiographic imaging. Later, it became possible to display Doppler information at the same time as an image, a technique known as duplex imaging. This allows the echocardiologist to guide the position of the Doppler beam within the heart. Another advance was the development of pulsed-wave Doppler (PWD). With standard CW Doppler, the frequency shift in the reflected ultrasound can come from anywhere along the course of the beam. However, it is often helpful to obtain information about blood flow in specific parts of the cardiac chambers and great vessels. PWD sends a pulse of known, short duration, and then records returning echoes for a limited period some time later. By limiting the period during which returned echoes are detected, the

distance from the transducer of the targets, which return the echoes, is known. The 'gated' period can be displayed on the screen as a small box known as the sample volume. This corresponds to the area of the heart from which the Doppler signals are received. The time taken for the PWD sound wave to reach the near end of the sample volume can be termed T1. The time taken for it to reach the far end of the sample volume can be termed T2. The machine starts to listen to returning echoes after $T1 \times 2$ and stops after $T2 \times 2$. The sample volume can be guided into specific; areas of interest such as the atrial side of atrioventricular (AV) valves to detect regurgitant blood flow, or the right ventricular side of a ventricular septal defect (VSD) to detect blood flowing through the defect. Thus DE can be used to detect blood flow, to identify the direction of flow, and to calculate the velocity of flow.

One of the problems with the use of PWD is that the rate at which the pulses of sound are produced is limited by the fact that a new pulse cannot be sent until the preceding one has been received. The number of pulses, which can be sent per second, is known as the pulse repetition frequency (PRF). The greater the depth of the sample volume, the lower the PRF. The PRF becomes a limiting factor when measuring the frequency shift associated with high velocity jets. This is because of a physical factor known as the Nyquist principle. This states that frequency shift (and therefore velocity) can only be measured accurately when the sampling rate is twice or more than twice that of the frequency shift being measured. When the Nyquist limit is exceeded, the flow of blood will be displayed in the opposite

direction to its true direction (aliased), which can be confusing. Aliasing is a particular problem in horses because the depth of many areas of interest means that PRF is relatively low, while the velocity of jets associated with valvular regurgitation or a VSD are usually quite high. For this reason, quantitative data about high-velocity jets is best derived from CW Doppler, which is not affected by aliasing.

When a PWD sample volume is placed in an area of laminar flow, for example the flow, which is usually found in the pulmonary artery, a clear line will be shown on the velocity/time graph indicating that the majority of the blood cells are moving at the same speed. Where this clear line is seen it is described as an 'envelope'. A line can be drawn around the envelope, and the area under the line is the velocity/time integral (VTI). The VTI is directly proportional to the stroke volume ejected through the valve and, if the area of the vessel is accurately measured and the VTI is the maximum, which can be recorded from the site (i.e. the ultrasound beam is parallel to the line of blood flow), the stroke volume can be calculated. Cardiac output is then simply calculated by multiplying the stroke volume by the heart rate.

The clarity of the envelope of a PWD signal depends on the range of blood flow velocities within a sample volume. When the sample volume is placed in an area of turbulent flow associated with a regurgitant jet, which is causing a cardiac murmur, a range of RBC velocities will be seen because blood is flowing in different directions relative to the transducer. It may even be displayed either side of the baseline.

The principles of physics can be put to good use by clinicians. An example is the use of DE to estimate pressure gradients, a task which previously required invasive catheterization techniques. Once accurate measurements of the frequency shift associated with a jet of blood flowing between two chambers have been made, it is possible to estimate the pressure gradient between these chambers using a derivative of the Bernoulli equation. A simplified version of this equation is suitable for use in most instances. An example of the use of this principle is estimation of the pressure gradient between the left ventricle (LV) and right ventricle (RV) in an animal with a VSD. This is estimated from CW Doppler measurement of the velocity of blood shunting through the defect. If the RV pressure is raised then the defect is likely to be of clinical significance and may affect the ~~heart's~~ athletic ability.

Colour-flow Doppler mapping

Colour-flow DE (color-coded DE) is a form of PWD, which is subject to exactly the same physical limitations as standard PWD. The direction of flow in a large number of individual sample volumes is measured and color-coded. Usually, flow away from the transducer is coded blue; flow towards the transducer is coded red. The color scale is usually displayed on the side of the screen. The tone of the color depends on the velocity of flow and its brightness on the intensity of the signal (i.e. on the number of RBCs reflecting sound). The color-coded pixels are displayed in a sector superimposed on a 2DE image so that the location of the flow within the heart is known.

Blood Flow Patterns

Blood flow through the heart and great vessels has certain characteristics

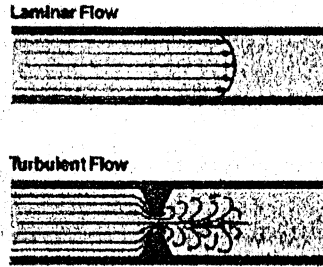


Fig. 1.2

that can be

measured using

Doppler instruments designed for medical use. For the purpose of understanding flow patterns in the heart, it is important to recognize the difference between laminar flow and turbulent (or disturbed) flow. Laminar flow is flow that occurs along smooth parallel lines in a vessel so that all the red cells in an area are moving at approximately the same speed and in the same direction (Fig. 1.2). Due to friction, flow is always slightly slower near the walls of a vessel. With the pulsations of the heart, the red cells generally accelerate and decelerate at approximately the same speed. Flow in most of the cardiovascular system, including the heart and great vessels, is normally laminar and rarely exceeds the maximum velocity of 1.5 m/sec.

In contrast, turbulent or disturbed flow is present when there is some obstruction that results in a disruption of the normal laminar pattern. This causes the orderly movement of red blood cells to become disorganized and produces various whirls and eddies of differing

velocities and directions. Obstruction to flow usually also results in some increase in velocity. Thus, turbulent flow is characterized by disordered directions of flow in combination with many different red cell velocities. If the obstruction is significant, some of the red blood cells may be moving at higher velocities than normal and may reach speeds of 7 m/sec. Turbulent flow is usually an abnormal finding and is considered indicative of some underlying cardiovascular pathology.

Abnormal flows are therefore generally characterized by turbulence and any increase in

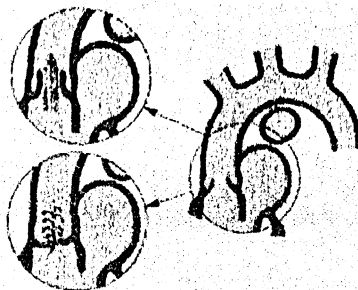
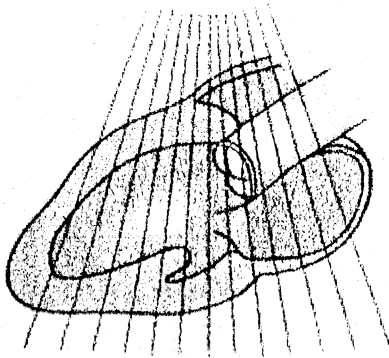


Fig 1.3

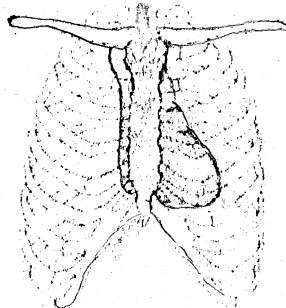
velocity. As an example, consider blood flow in the ascending aorta during systole. If the aorta and aortic valve are normal, then this flow is laminar. However, the presence of a valvular stenosis will induce a turbulent flow pattern. Figure 1.3 shows that a narrowed aortic valve orifice interrupts the parallel lines of normal laminar flow and produces turbulent flow. The resulting jet of blood creates a short segment within the proximal aorta with complex flow and velocity characteristics.

ECHO CARDIOGRAPHIC VIEWS

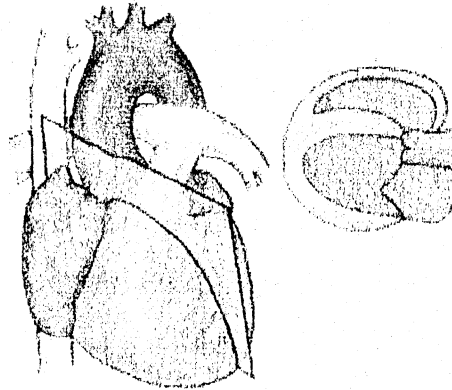
Principles



Sound is emitted from the echo transducer and the time taken for the reflected sound (echo) to return is proportional to the distance traveled. The computer maps the echoes on a line & then takes another sample on a different line thus building up a 2D picture approx 50 times a second. The frequencies commonly used (2-10MHz) are much higher than the audible range of 2-20KHz. Higher frequencies allow better resolution but tissue penetration is poorer.

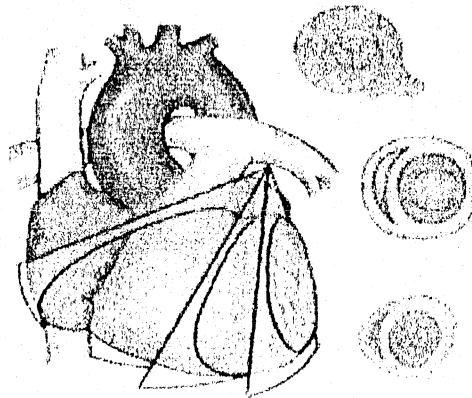


Parasternal Long Axis



The transducer is placed in just to the left of the mid to upper sternal border. The right ventricular outflow region, the ventricular septum and the left atrium and ventricle are well visualized. This is one of the best views to obtain an M Mode and hence information on cardiac function.

Parasternal Short Axis



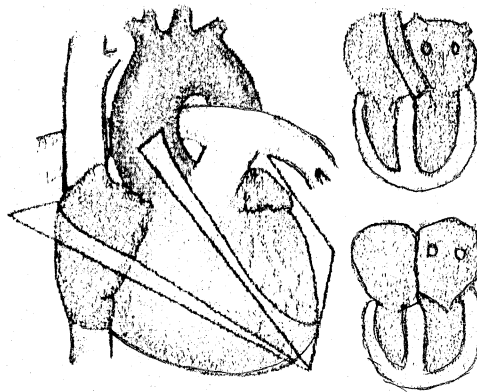
From the parasternal long axis view the transducer is rotated 90° to point towards the left shoulder. The aorta and coronary artery origins are seen well in cross section. The PA is also seen along with the branches and PDA .

In children all parts of the heart can be imaged either through rib spaces, sub costal area or suprasternal notch (echo windows). The anatomy, wall and cavity sizes easily determined.

2D Echo

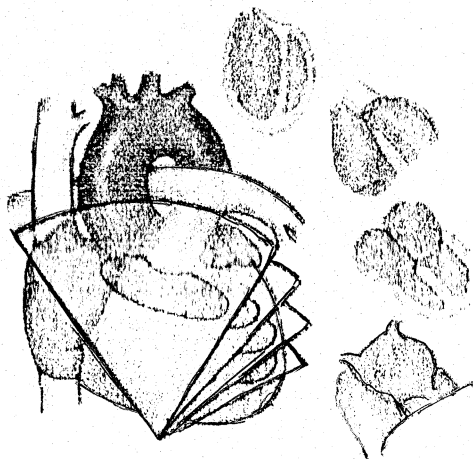
The 2D pictures are taken from the various echo windows and give "standard views" to build up a complete picture of the cardiac anatomy. Not all chambers are visible in every view.

APICAL FOUR CHAMBER VIEW



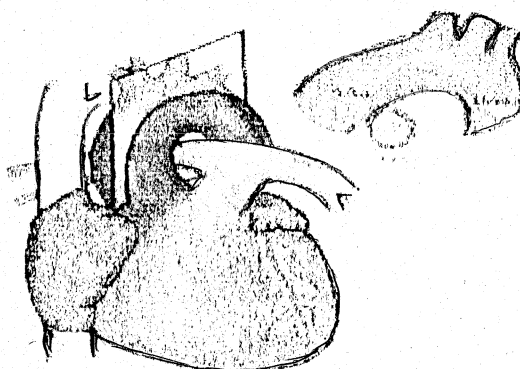
Transducer is held at the apex of the heart and angled towards the right shoulder. The 4 chambers are readily seen and both the mitral and tricuspid valves. If the transducer is angled anteriorly then the aorta is also visualized. This view is shows the ventricular septum well to look for septal defects.

Sub costal View



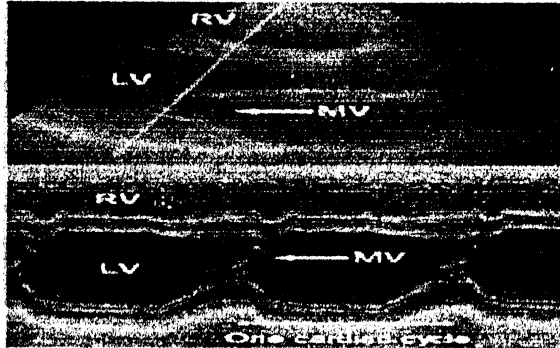
This is a good view to see lovely images - especially in babies as no ribs or lung tissue obscures the view. The Atrial septum is particularly well seen. Unfortunately the transducer is the furthest from the heart and in older children and adults the distance may be too great to allow detailed imaging.

Arch View



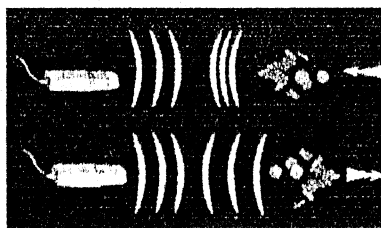
This is obtained by sliding the transducer towards the upper sternal edge and suprasternal notch. It allows the ascending aorta, arch and neck vessels to be imaged.

M Mode



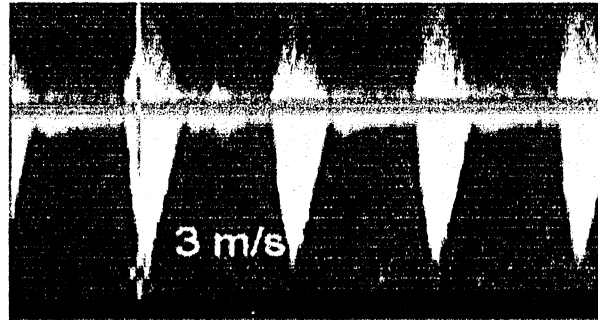
Used to measure accurately chamber size at various times in cardiac cycle. Consider it as if several pencils were attached on the line seen on the 2D image & a piece of paper drawn rapidly across - the line moves according to the cardiac motion of that specific point in the heart. The M Mode measures the interventricular wall thickness in systole (IVSs & IVSd) and diastole, the LV posterior wall thickness in systole and diastole (LVPWd & LVPWs) and the LV cavity in systole and diastole (LVDs & LVDd) which allows calculation of the shortening fraction (FS) by dividing the difference between LVDd & LVDs into the LVDd and is a measure of LV contractility.

Doppler



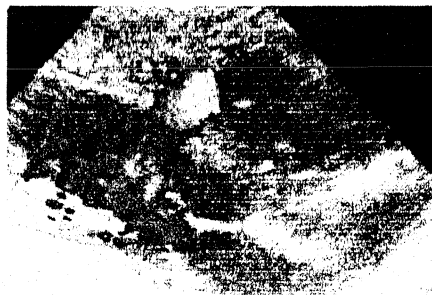
Sound waves at a constant frequency are emitted from the transducer when an electric current is passed through a crystal. They are reflected back by the red

blood cells-if the blood is traveling towards transducer the wavelength is compressed (upper diag), the converse if blood is traveling away (lower diag).



The velocity of blood flow is calculated from the wavelength difference. The velocity can be converted into a pressure difference by the Bernoulli equation - this allows gradients across valves or narrowed arteries to be measured accurately. The pressure drop = $4V^2$.

Colour Doppler



Over the area selected by the sonographer the machine analyses the Doppler flows using the pulsed wave principle, codes the results using color (by convention red towards the probe, blue away) and overlays the color map on the 2D image. This allows rapid assessment of blood flow direction and an assessment of its velocity.

Transesophageal Echocardiography



PFO demonstrated on TEE

This is performed under sedation in adults but the majority of children prefer a general anesthetic. The TEE probe is placed down the oesophagus and lies immediately behind the left atrium. As the probe is thus very near to the cardiac structures and there are no other structures in the way the image quality is excellent. It is particularly beneficial in those in whom precordial images are poor – especially the adult congenital population. It is much more sensitive than transthoracic echocardiography in the investigation of endocarditis.

TRANSESOPHAGEAL ECHOCARDIOGRAPHY

Introduction

Transesophageal echocardiography was introduced by frozen et al (1976), shorter time after the clinical acceptance of echocardiography. All TEE transducers essentially consists of an ultrasound transducer mounted at the tip of a flexible endoscope's that has a maximum shaft diameter of 10-11mm and a total shaft length of 70-120cm.the guidance control allows the tip at least 90degree of antro posterior flexion and up to 70 degree of lateral mobility in each direction. This mobility's are effected mechanically two wheels on the endoscopes handle with the possibility of locking the steering controls. Most current generation probes use an operating frequency around 5MHZ and are compromised of a phased-array transducer containing 48-68 elements .The ultrasound elements are mounted at the distal flexible tip so as to provide transverse plane images of the heart, ie, their scan planes is at an angle 90 degree to the shaft of endoscopes.

Recently, biplane and omni plane transesophageal probes have been constructed in an attempt to overcome the lack of versatile associated with imaging structures only in the transverse plane.

Diagnostic applications

Esophageal echocardiography has been suggested to be useful as an alternative diagnostic procedure in cases in which TTE is technically in adequate and the clinical question is important to resolve. TEE is more commonly employed in obese patient, those with lung disease or those with sternal epigastria wounds. Diagnostic question for which this technique has been reported to be of particular value include the detection of intra cardiac thrombi particularly in LA appendage, evaluation of patient with a suspected

cardiac embolic stroke, visualization of small bacterial vegetation and endocarditic associated abscesses and the diagnosis of aortic dissection. Also useful in detecting septal and papillary muscle rupture following acuteMI, prosthetic valve dysfunction and specific features of nature and post operative congenital heart disease.

The primary reported reason for performing TEE has been as a pre procedure screen for atrial thrombi in-patient undergoing PTMC and to rule out a cardiac source of embolus .

Patient preparation

Patient must fast for 4 to 6hrs before undergoing a TEE study. It is important to carefully explain the procedure to the patient and to obtain a detailed history of any gastroesophageal related symptoms and prior endoscopy. Drug allergy and medication history are also important.

Dentures and oral prosthesis must be removed before the examination, intravenous access established and an oxygen delivery system, defibrillator, airway bite guard, suction and sphygmomanometer must be immediately available. Cardiac rhythm should be continuously monitored. a 12-lead ecg should be immediately available for emergencies.

Topical anesthesia is generally applied to the hypo pharynx by 4% xylocaine spray 10% cetacaine spray or by having the patient gargle and swallow 2% viscous xylocaine. Peak anaesthetic effect occurs in 2 to 5 minutes and the effect persists for 30 to 45 minutes. As a result nothing should be by mouth for at least 30 minutes after the procedure.

CONTRAINDICATION

TEE is contraindicated in patient with esophageal pathology such as structure, varices, tumors, diverticula and scleroderma, severe atlanto axial

joint disease that prohibits flexing of the neck, prior radiation to the chest and perforated chest.

TRANSESOPHAGEAL TWO-DIMENSIONAL ECHOCARDIOGRAPHY

A basic examination consists of a single plane examination of the heart and great vessels.

The terms single plane, transverse plane, horizontal plane, and imaging at 0 degrees are synonymous.

Basic Tee Examination Sequence

| Step | View |
|-------------|--|
| 1 | Distal AO arch & descending upper abdominal AO |
| 2 | Basal short axis scan of great vessels |
| 3 | Basal short axis scan of aortic valve |
| 4 | Long axis scan of left ventricular outflow tract |
| 5 | Long axis scan of mitral valve and left ventricle |
| 6 | Basal short axis scan of left atrial appendage |
| 7 | Imaging of interatrial septum |
| 8 | Long axis tricuspid valve and RV at the level of AML |
| 9 | Long axis of tricuspid valve and right ventricle at level of coronary sinus |
| 10 | Tran gastric short axis mid-chamber scan of right and left ventricles |

CONTRAST ECHOCARDIOGRAPHY

Echocardiographic contrast agents are used to understand blood flow in the heart in a variety of modes, including color and power Doppler, power angio and coded harmonic angio. The goal: helping you make more confident diagnoses, particularly in assessing wall motion in difficult to image patients and in contrast application such as left ventricular opacification (LVO).

Contrast agents travel with in the blood in both the heart chambers and myocardial tissue. These contrast agents typically consist of a gas core encapsulated by a shell.

The scattered ultrasound signals during 2D harmonic imaging modes from these contrast agents increase the blood flow ultrasound signal detected by three orders of magnitude. With your ultrasound system in power Doppler or coded harmonic angio, the ultrasound transmitted beam may actually burst the shells and scatter the gas cores, creating an even stronger ultrasound signal. Typically the increased ultrasound signal during 2D harmonic imaging modes is used to opacify the left ventricular blood volume, allowing better visualization of the left ventricular wall motion. The effect of the additional signal, seen from the agents in power Doppler modes, may be used by researchers to visualize blood flow in the myocardial tissue.

Additional ECG triggering techniques are used to eliminate the bubbles in one frame and visualize the power Doppler from the agents in another frame, helping clinicians further research myocardial perfusion.

DOBUTAMINE STRESS ECHOCARDIOGRAM

Dobutamine stress echo is a noninvasive test used to evaluate coronary artery disease in patients are unable to exercise on atreadmill.dobutamine is a medication that increases heart rate and blood pressure similar to the effect of exercise. The rise in heart increases the oxygen demand of the heart and helps to determine if the heart muscle is enough blood and oxygen. The test includes an echocardiogram done at rest again at peak heart rate. This procedure uses sound waves to produce an image of the internal structure of the heart. in order to produce an image of the heart muscle ,gel is applied to the patients chest area and a transducer is moved over the chest. Electrodes are placed on the chest to record an ECG.,which monitors the heart rate and rhythm. An IV line will be started and dobutamine will be administered by anurse.the cardiologist will observe for any symptoms ,irregular heart hrhythm,an in appropriate heart rate or bp response.

This test will help the doctor evaluate the patients cardiac condition related to the following:

- *how well the heart muscle and valves are working and how they function under stress
- *The size of the hearts pumping chambers(ventricles)
- *abnormal heart function: coronary artery disease or inadequate coronary blood supply.

Preparation of Dobutamine

50mg in 50ml of dextrose is the dobutamine preparation.

So 1ml contains 1mg of dobutamine.

Ie,1000micrograms in 1ml

Dose for 1kg is 5micrograms/min

For a50kg adult, dose=50*5

= 250micrograms/min

ie,250*60micrograms/hr=15000micrograms/hr

1ml contains 1000micro grams

dose for an adult of 50 kg is 15000/1000=15ml/hr

ECHO PAC

The echo PAC network is a complete network solution for acquiring , storing, and managing digital ultrasound data. The echo PAC network is standards- based- DICOM, windows (R) NT (R), and HL 7. That means it is fully compatible with GE Vingmed System5 (R) and other DICOM conformant ultrasound devices. And because it is fully scaleable, you can start with just a desktop workstation today, and add review stations , scanners and mask storage devices as your requirements evolve .

Image review

Echo PAC Optimizes image review by :

- Preserving the raw image data, for excellent image quality and high frame rate.
- Permitting through serial study comparison of images from DICOM 3.0 – complaint ultrasound equipment or agilent's TIFF format, to improve patient follow up.

Image Analysis

With echo PACC, full digital quantitative analysis of raw image data is possible. Included is an extensive quantification and post processing tool set to help increase your diagnostic confidence.

- Contrast research capabilities for automatic or manual image subtraction
- Anatomical m-mode allowing the clinicians to acquire correct M-mode planes, regardless of the scan plane
- Densitometry package to quantify ,color, or contrast power angio images
- Multi-segmental quantification research tool set for contrast or tissue velocity imaging.

Echo PAC's echo patients archive

- Let's you perform all critical post exam analysis off line for your day-to -day clinical routine.
- Virtually eliminates the need for patient recall
- Gives instant access to previous exam results
- Allows researcher's to analyze all raw digital image data off line

Departmental equipment

System five-System five is a Doppler ultrasound system for applications such as adult pediatric & neonatal cardiac peripheral vascular imaging

Probe frequencies:

| | |
|-------------------------------|-----------------------|
| Adult transducer | :2.5MHz (1.7-3.6 MHz) |
| Pediatric Transducer | :5MHz(5-8MHz) |
| Continuous Doppler transducer | :2MHz |
| TEE transducer | 5MHZ |

CFM 800 A

Probe frequencies

| | |
|-------------------------------|----------------|
| Adult probe | :2.5 /3.25 MHz |
| Pediatric | :5MHZ |
| Continuous Doppler transducer | :2MHz |

Sonos 1000

Probe frequencies

| | |
|-------------------------------|--------------|
| Adult | :2.5/2 MHz |
| Pediatric | :3.5/2.7 MHz |
| Continuous Doppler transducer | : 1.9 MHz |

CATHETERIZATION

LAB

CATHERINATION

LAB

CARDIAC CATHETERIZATION LAB

Cardiac catheterization is a combined haemodynamic and angiographic procedure undertaken for diagnostic or therapeutic purpose. The cardiac catheterization team should be aware of the equipment used for cardiac angiographies, radiographic principles, and programs for radiographic quality assurance, radiation protection and characteristics of various intravascular contrast agents.

The angiographic room

The modern cardiac catheterization laboratory consists of a patient support table; equipment for monitoring of intracardiac pressures and electrocardiographic activity and a floor or ceiling supported gantry that allows variable angulations of the x-ray beam. If desired a second complete imaging chain may be used to provide simultaneous viewing of cardiac structures from a separate angle. Such big plane imaging is generally restricted to laboratories that study a high percentage of congenital cases.

The purpose behind this support equipment is to allow precise positioning of a radiographic imaging chain, relative to the patient in either left or right anterior oblique (rotation) or cranial or caudal (skew) angulations. The image chain consists of a generator / cine pulse system, an x-ray tube, an image intensifier, an optical distributor, 35mm cine camera, and a television camera and a monitor. As such an image chain can provide both live fluoroscopy to facilitate placement of cardiac

catheters and cine angiographic film exposure that permanently captures details about the anatomic and functional state of cardiac chambers, great vessels and the coronary circulations.

To house this bulky and expensive equipment and support personal, the room should have a floor space of at least of 500 ft square with a ceiling height of at least of 10 ft (3mm). The walls should be shielded with 1mm of lead up to a height of 7 ft to provide radiation protection for personal in the surrounding work areas, and any observations windows should be lead- treated glass to provide similar radiation shielding. The bulky components constitute the generator and its associative electronics (i.e. the racks.)May be placed in a ventilated closets along the walls of the rooms but must be placed so that the high voltage cable runs are short and racks themselves are easily accessible to service personal for diagnostic and repair articles

X-ray generator

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

To be useful in cardiac study ,the generator must be combined with a cine pulse system ,which converts the generator output into the brief pulses that are required to eliminate motion induced blurring of the rapidly moving

coronary arteries. The cine pulse system must be capable of handling the 60 to 100 kw power output of the generator if that power is to be delivered effectively to the x-ray tube. the automatic exposure control of the generator compensates for changes in the transmission of x-rays to the image intensifier as the beam is panned through structures of differing attenuation.

THE X-RAY TUBE

The x-ray tube consists of an evacuated glass tube or metal housing that contains tungsten filament and an anode disc, which rotates at more than 10,000rpm during cine angiography. electrons are emitted by thermionic emission and are accelerated towards the anode by electric field supplied by the generator. The rapid interaction with the tungsten atoms in the anode results in the emission of x-ray photons. Two focal spots are included in the x-ray tube.

THE IMAGE INTENSIFIER

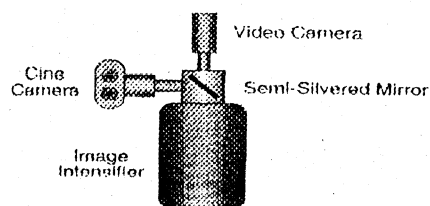
The image intensifier converts an x-ray image into a visible light image. The image intensifier consists of a large glass vacuum bottle coated internal with a fluorescent phosphor at each end, cesium ionized at the input and zinc cadmium sulfide at the output. Image intensifier increase the brightness of the image by thousands fold. The image intensifier contains an electrostatic lens that focuses the electrons during their flight. The qualities of image intensifier are central to the performance of the image chain. Other qualities such as gain,

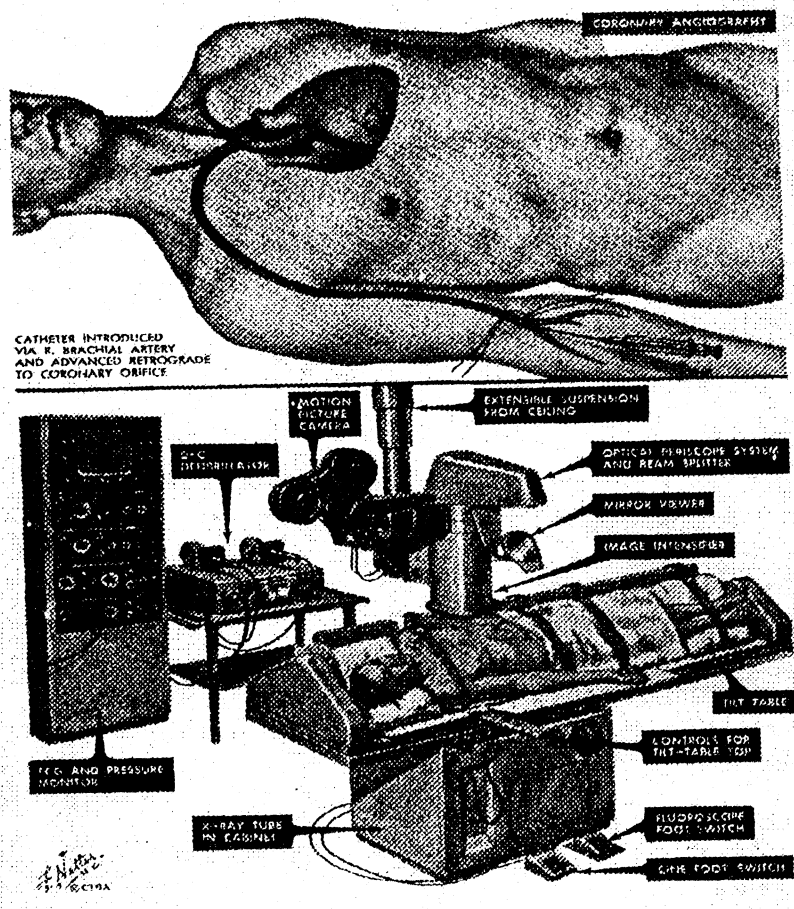
quantum detection efficiency, spatial resolution and contrast tend to be mutually exclusive.

CINE CAMERA AND ASSOCIATED OPTICS

Angiographic images are recorded on 35-mm film. Film advancement within the camera triggers the generator to produce each cine angiographic x-ray pulse. The camera views the output phosphor of the image intensifier through an optical system consisting of matched collimator and camera lenses. The focal length of the optical system determines the framing mode—the way in which the round output phosphor is represented on the rectangular cine frame.

To improve the quality of the simultaneously obtained television images to facilitate precise catheter manipulation and allow online evaluation of results during interventional procedures, the television camera is mounted along with the 35-cine camera on a distributor.





DIGITAL ANGIOGRAPHY

Digital imaging uses the voltage of analog video signal produced during image acquisition, which is proportional to the brightness of the original image. an ads samples the video signals and converts the voltage level into a series of discrete numbers. the number of possible grey levels for each pixel is determined by the number of bits available for the analogue -to digital conversion. in cardiac digital angiography ,this is

typically 8 bits ,which in binary numbers corresponds to 256 possible grey levels.

The horizontal and vertical sampling rate of ADC determines the matrix size of the image. Most current system generates 512* 512 samples, each refereed to as a picture element, or pixel. cadrdiac digital angiography thus consists of 512*512 matrix, the digital system will generate 3.4 pixels /mm for a 15-mm image image intensifier.

Cardiac digital angiography requires large data storage capacity. An 8 sec coronary injection requires about 60 MB (30-frames/sec) and a full angiographic study may require up to 1GB. In current interventional practice, images are stored temporarily on computer hard disc for immediate replay and review.

ADVANTAGES

It is possible to acquire useful arterial images with much smaller amount of contrast than with film angiography and to store and analyse images in a more quantitative fashion. In addition, the digital approach allows the performance of a number of manipulations on the stored images. The contrast image can be amplified or enhanced. One image can subtract from another. Digital image manipulations allow many views that are not possible with film radiographs.

DIGITAL ARCHIVING (cine replacement)

Digital angiography is attractive as a long-term storage medium for archiving cardiac catheterization studies. Digital information allows production of multiple images with no image degradation. Digital data can be transmitted electronically for remote examination and consultation. The economic cost of storing patient study may be reduced relative to current film expertise.

Vascular tracing: A procedure called vascular tracing or road mapping often provided in digital angiography system.

Digital fluoroscopy aids: many digital angiography systems utilize functions of the image processor during fluoroscopy, when subtraction angiography is not performed. One of these functions is image noise reduction during fluoroscopy by integrating several successive frames. Another is last image hold, which allows the operator to retain the last image displaced on the monitor each time the fluoroscopic foot switch is released.

Quantitative coronary angiography

The degree of coronary stenosis is quantified from the cineangiogram and, in clinical practice, is usually visual estimation of the percentage of diameter narrowing using the presumed proximal normal arterial segment and the ratio of the normal diameter to the normal diameters. Quantitative methodology uses digital clippers or automated or manual edge detection systems.

DIGITAL IMAGE PROCESSING

POST PROCESSING

For the purpose of image enhancement operation are performed at the viewer's option after the basic image is formed as post processing. Three-post processing is common.

1. NOISE SMOOTHENING –FILTER.

Noise smoothening is an attempt to decrease the visual prominence of noise so that low contrast object of moderate to large size may be better appreciated. All methods of noise smoothening will reduce resolution. Averaging it with the closest neighbours reduces the statistical fluctuations in each pixel.

2. EDGE ENHANCEMENT

IT is intended to increase the visibility of small structures with moderate to high contrast. It is accomplished by suppressing information regarding large structures.

Unfortunately noise is also more in edge-enhanced image.

A low-pass filtered image contains only the information about large structures and low noise, while the original image contain additional information about the edge and noise. The subtraction of low –pass filtered image from the original image yields an image in which edge and small structures remains.

3. INFORMATION EXTRACTION

From the acquired original images some information like percentage of stenosis length of the vessel segment, diameter of the vessel etc. can be measured. Also information like ejection fraction and ventricular function can be accessed.

4.REMASKING

If one sequence the initial mask image is inadequate because of patient motion, improper technique or any other reason, later images may be used as mask images.

5. RE-REGISTRATION OR PIXEL SHIFT

If patient moves in between acquisition the image obtain will not registered exactly in the same pixels of image matrix. This type of artifact can be eliminated by re-registration of mask. Shifting the mask by one or more pixels so that superimposition of images is again obtained does this.

DEPARTMENTAL EQUIPMENTS

INTEGRIS H5000F (PHILIPS)

The floor –mounted monoplane Integris 5000 f is the dedicated system of cardiac procedures. It brings excellent digital imaging performance to cardiac suite. The large diameter of the poly diagnostic G stand and patient sensing of body guard allow high rotation and angulations speed of up 25 degree per second. New fully integrated, all digital imaging chain is based on CCD technology optimized for complex cardiovascular applications. It provides high speed, high resolution imaging with true 1024 matrix.

Specifications.

Environmental requirements

Ambient temperature : 10-35 degree Celsius

Humidity : 20-80%
Mains : 440v+/_10%,50and 60
Hz,3 phase.

X-ray generator.

Microprocessor controlled 100 Kw high frequency
converter generator.

Voltage range : 40kv to 150 KV
Max.current : 1000mA at 100 kV,800mA at
125 kV

X-ray tube (MRC) 0508

Power : 0.5/0.8,45/85kW
Anode heat storage capacity : 2400KHU anode
Continuous heat dissipation : 3500w
TV chain XTV 16, CCD camera with proprietary digital

output

Examination light
Light intensity : 30000Lux
Focusable light field size : 14-25cm
Lamp type : halogen 22.8/24v 50w

Automatic wedge filter

Two semi transparent wedge shaped filters automatically
or manually adjusted.

Digital acquisition

Direct link with XTV 16 image chain
12 programs for digital dynamic acquisition
Frame speeds (frames/sec) at 512* 512* image
resolution.

Frame speed

50Hz : 12.5,25,30
60 Hz : 15,30 ,60
11 sizes : 9,7,5 inches

BV 300(PHILIPS)

BV300 is a mobile diagnostic X-ray image acquisition
and viewing system.

- 9" or 12" triple-mode field of view.
- Fixed or rotating anode X-ray tube technology.
- 200,2000,100000 image capacity.

A unique combination of CCD camera and anamorphic lens
that delivers high quality image details.

SEIMENS COROSKOPE- BIPLANE

Coroskope-digitron is biplane cath system with DSA and cine angiographic support. Equipped with seimns polvdors 100 –high frequency X-ray generator with microprocessor control.

GE Advantax DLX-LCV

A single plane system equipped with high rated grid . controlled tube with air –cooling system. Focal spots available are 1.1, 0.06,0.03,0.030b, 0.020b and 0.15b. Maximum KVP and mA are 120 and 1000 respectively. System is provided with cine attachment. Other facilities are last Image Hold(LIH) auto injection of contrast, edge enhancement , noise filtering, road map etc, image processing include different subtraction mode , pixel shifts, gray scale inversion, remasking, image filter etc.

DICOM

INTRODUCTION

The **Digital Imaging and Communications in Medicine (DICOM)** Standard is a detailed specification that describes a means of formatting and exchanging images and associated information. The standard applies to the operation of the interface, which is used to transfer data in and out of an imaging device. DICOM relies on computer industry standard network connections, and media devices that address the communication and storage of digital images from diagnostic modalities such as CT, MR, PET, Nuclear Medicine,

Ultrasound, X-ray, CR, digitized film, video capture and HIS/RIS information. It also supports the connection of networked printers, such as laser imagers (cameras). DICOM is the result of an alliance of potential users of the standard (members of the American College of Cardiology and American College of Radiology) with the companies that manufacture medical equipment (members of the National Electrical Manufacturer's Association – NEMA). The DICOM effort really began in 1984, and it was originally called the ACR/NEMA standard. Now, DICOM has been embraced by other worldwide standards organizations outside of Cardiology and Radiology. For example, DICOM has been adopted by the Committee European de Normalization (CCEN TC 251) and the Japanese Industry Association for Radiation Apparatus (JIRA).

The DICOM standard has now been implemented in an increasing number of medical products from various vendors. The rapid adoption of DICOM by the medical imaging industry is opening new opportunities for health care organizations to increase the quality and cost effectiveness of patient care.

THE NAME – DICOM

Version 3.0 of the ACC/ACR-NEMA standard is called the Digital Imaging and Communications in Medicine

(DICOM) Standard to reflect the contribution of other international organizations as well as the standard's ability to expand beyond support of cardiology and radiology images, to include images from endoscopy, surgery and pathology.

DICOM REVIEW STATION

In general, a DICOM Review Station is to the cine-projector as the CD-R disc is to the interchange aspect of cine film. In order to describe the concept of a DICOM Review Station, let us make an analogy to both 35 mm cine film and the cine film projector. There are 5 basic tasks that the Cardiologist is faced with when using cine film. These tasks include:

Acquisition- The X-ray Cardiac Angiographer performs an X-ray exam (diagnostic or interventional) on a patient and acquires the exam to 35 mm cine film.

Film Developing- The cine film must be processed so that it can be used for "review".

Review- The Cardiologist must load the film into a 35 mm cine-projector. The cine-projector provides the basic image display functionalities required to diagnostically assess the clinical severity of most morphological defects. The Physician can then view the entire exam by controlling the cine-projector (e.g., fast forward, reverse, pause, etc.). The cine-projector does

enable the cardiologist to perform basic image processing such as zoom, roam, or brightness adjustment.

Interchange- The Cardiologist can send the film across town, across the state, across the country or around the world for review on any 35 mm projector. This exchange process enables physicians to communicate with other people (i.e., physicians, patients, etc.)

Archive- After the patient has been treated or discharged; the cine film (exam) can then be archived longer-term storage (e.g., a warehouse).

So what is the analogy between (cine projectors/cine film) and (DICOM Review Stations/CD-R)? The basic DICOM Review Station provides the exact functionality of a 35 mm projector. A primary difference, however, is that a DICOM Review Station can (although not mandatory) provide random access to the digital acquisition sequences (runs) corresponding to a specific acquisition run. In addition, the benefits of "digital" technology enable the DICOM Review Station to provide the same image processing capabilities as those found on high-end digital X-ray imaging systems. For example, the image sequences can be transferred from the CD-R interchange into the DICOM Review Station. The DICOM Review Station can provide edge enhancement filtering or qualitative analysis

productivity, image quality, and clinical care. So, is a DICOM Review Station like a cine-projector? Yes... but it can be much, much more! Loading Images into a DICOM Review Station. When a User receives a DICOM CD, the following sequence of events are required to view the images:

- Load the DICOM CD into the DICOM Review Station Drive,
- Select the Sequences to be viewed,
- Import the Sequences from the DICOM CD into Review Station

NOTE: The images are stored in a loss less compressed format on the DICOM CD; therefore the DICOM Review Station must decompress them. This can be done by specialized hardware or by software. Import performance is limited by both the CD-ROM (or CD-R) drive performance as well as the decompression approach used by the Review Station.

CD-R (COMPACT DISC-RECORDABLE)

The Ad Hoc Group selected the CD-R (Compact Disc – Recordable) as the standard medium for exchange of cardiac digital angiographic examinations. The standardization process has resulted in a sufficiently detailed definition of how images

are stored on a compact disc to permit the manufacturers of imaging equipment to read and write and digitally-recorded study, regardless of the source of the original image data.

KEY FACTS ABOUT THIS MEDIUM INCLUDE

- ❖ In 1996, the standard speed CD-R drive is 4X (600 KB/second) performance. Most CD-R drives are backwards compatible and can provide read and write rates at 1X (150 KB/second) and 2X (300 KB/second) speeds.

- ❖ Continuing investment in research and development in CD technology will guarantee substantial improvement in performance in coming years, resulting in increased storage capacity and data transfer rates.

- ❖ CD-R is a “write once” non-erasable medium, physically robust (after writing), unaffected by electromagnetic fields, and relatively insensitive to dust and scratching. Digital recording on CD-R is virtually error free (error rate is less than one false bit in 1,000,000,000,000 bit, whew!).

- ❖ CD-R storage capacity is 650 MBs, which permits recording of upto 4,800 frames per disc at a 512 x 512 x 8 bit resolution utilizing 2:1 loss less compression. As is evident from the above statistics, this capacity is sufficient to record at least 99 % of all cardiovascular examinations on a single disc. Standard CD-R media are sold and referenced in minutes of storage capacity, as this technology is derivative of the audio CD-ROM disc. A 650 MB CD-R is a 74-minute disc.

The Ad Hoc Group recognizes that current CD-R technology lacks sufficient speed to enable direct viewing of angiogram at 30 frames per second. The Committee did not consider the lack of real-time retrieval to represent a great disadvantage for an interchange medium. The CD-R disc permits random access to any exam sequences. Thus, the relatively low retrieval rate is partially compensated by rapid access to any sequence of interest. Furthermore, review stations with CD-ROM drives can be equipped with image buffers allowing perfecting of the entire examination followed by real-time review as require. CD media and drive technologies are still in a state of evolution towards higher performance. Traditionally, write and read data rates have been referenced to the single-speed performance of 150 K Bytes per second. Currently, the most technically advanced drives have six to eight times this performance (.9 MB/sec to 1.2 MB/sec, respectively). An 8 X drive, for example, enables images to be stored and retrieved at a rate of approximately 10 frames per

second, therefore requiring approximately 3 minutes and 42 seconds for an average study containing 22 is a 74 minute disc.

The Ad Hoc Group recognizes that current CD-R technology lacks sufficient speed to enable direct viewing of angiogram at 30 frames per second. The committee did not consider the lack of real-time retrieval to represent a great disadvantage for an interchanging medium. The CD-R disc permits random access to any exam sequence. Thus the relatively low retrieval rate is partially compensated by rapid access to any sequence of interest. Furthermore, review stations with CD-ROM drives can be equipped with image buffers allowing perfecting of the entire examination followed by real time review as required.

CARDIAC
CATHERIZATION

INTRODUCTION

Cardiac catheterization is the general name for a group of procedures in which long, thin tubes called catheters are placed in the heart and its adjacent blood vessels. catheters allows measurement of pressures, injection of fluids like contrast agents. The general type of cardiac catheterization and angiography in adults are described in the following subsections.

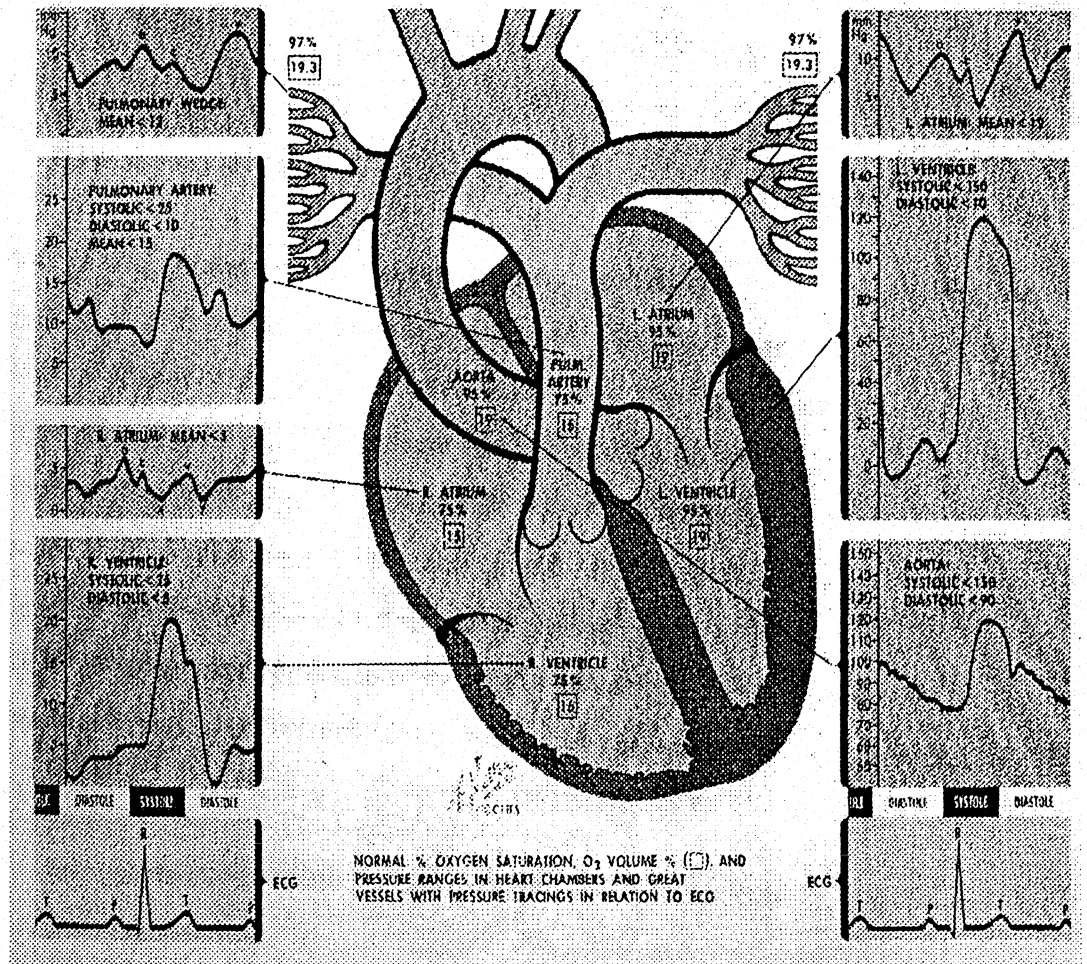
LEFT HEART AND CORONARY ARTERIOGRAPHY

Left heart and coronary Angiography is a study of the left ventricle, which is main pumping chamber of the heart, and of arteries that supply oxygen and nutrients to the left ventricle (coronary arteries). The study gives some information about the aortic valve and mitral valve .it is commonly used in a person who has had heart attack or who is at a risk for having a heart attack. This test requires that catheters, which are hollow, be placed into an artery (femoral artery) or brachial artery and then to be moved in the aorta just above the left ventricle or into the left ventricle after pressures are measured contrast is injected and moving pictures are recorded.

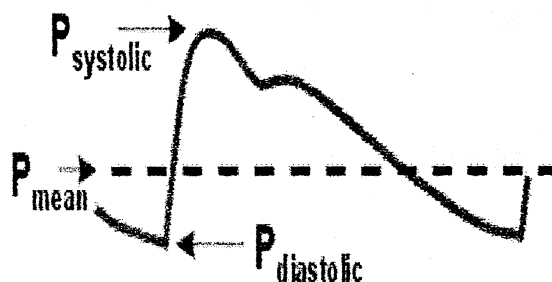
Measurement of pressure during cardiac catheterization

Pressures are measured in the left ventricle and in the aorta .the end diastolic left ventricular pressure reflects the state of hydration of the patient, the diastolic and systolic function of the ventricle .It is below normal when the patient is dehydrated, it is above normal when the patient has too much fluid in the blood stream has diastolic dysfunction.

The difference in the pressure between the LV and aorta is the “gradient” across the aortic valve. (Rarely, the ventricular muscle below the aortic valve can also create a gradient. This occurs in a condition known as “idiopathic hypertrophic sub aortic stenosis”. If the gradient is very high, the patient has aortic stenosis.



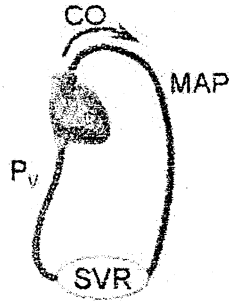
Arterial Blood Pressure



Ejection of blood into the aorta by the left ventricle results in a characteristic aortic pressure pulse. The peak of the aortic pressure pulse is termed the systolic pressure (P_{systolic}), and the lowest pressure in the aorta is termed the diastolic pressure ($P_{\text{diastolic}}$). The difference between the systolic and diastolic pressures is the aortic pulse pressure. The mean aortic pressure (MAP) is the average pressure (geometric mean) during the aortic pulse cycle.

As the aortic pressure pulse travels down the aorta and into distributing arteries, there are characteristic changes in the systolic and diastolic pressures, as well as in the mean pressure. As the pressure pulse moves away from the heart, the systolic pressure rises and the diastolic pressure falls. There is also a small decline in mean arterial pressure as the pressure pulse travels down distributing arteries due to the resistance of the arteries. Therefore, when arterial pressure is measured using a sphygmomanometer on the upper arm, the pressure measurements represent the pressure within the brachial artery, which will be slightly different than the pressure measured in the aorta or the pressure measure in other distributing arteries

Mean Arterial Pressure



As blood is pumped out of the heart into the resistance network of the systemic circulation, pressure is generated. In reality, this pressure is pulsatile because of the cardiac output is intermittent. If we were to assume that the cardiac output were continuous (i.e., non-pulsatile), then the mean arterial pressure is determined by the cardiac output(CO), systemic vascular resistance(SVR), and central venous pressure(CVP) according to the following relationship which is based upon the relationship between flow,pressure,resistance: $MAP = (CO \cdot SVR) + CVP$

Because CVP is usually at or near 0 mmHg, this relationship is often simplified to: $MAP = CO \cdot SVR$

Therefore, changes in either CO or SVR will affect MAP. If CO and SVR change reciprocally and proportionately, then MAP will not change. Although cardiac output is pulsatile rather than continuous, the above relationship is still a valid approximation. In practice, MAP is not determined by knowing the CO and SVR, but rather by direct or indirect measurements of arterial pressure. From the aortic pressure trace over time, the shape of the pressure trace yields a mean pressure value (geometric mean) that is less

than the arithmetic average of the systolic and diastolic pressures

$$MAP \cong P_{dias} + \frac{1}{3}(P_{sys} - P_{dias})$$

At high heart rates, however, MAP is more closely approximated by the arithmetic average of systolic and diastolic pressure because of the change in shape of the arterial pressure pulse (it becomes narrower). Therefore, to determine mean arterial pressure with absolute accuracy, analog electronic circuitry or digital techniques need to be employed to arrive at the mean value.

Venous Pressure

Venous pressure is a term that represents the average blood pressure within the venous compartment. We sometimes use the term "central venous pressure" (CVP) to describe the pressure in the thoracic vena cava near the right atrium. CVP is influenced by a number of factors, including cardiac output, respiratory activity, contraction of skeletal muscles (particularly legs and abdomen), and hydrostatic forces. All of these factors, however, ultimately affect CVP (ΔP_v) by changing either venous blood volume (ΔV) or venous compliance (C_v)

$$\Delta P_v \propto \frac{\Delta V}{C_v}$$

Therefore, an increase in venous volume will increase P_v by an amount determined by C_v . Furthermore, a decrease in venous compliance, as occurs during sympathetic activation of veins, will increase P_v .

RIGHT ATRIUM

RA: 0 — 7 mmHg (higher in ventilated patients)

PULMONARY ARTERY

PA Systolic: 20 — 30 mmHg (same as LV systolic)

PA Diastolic: 6 — 10 mmHg

PA : < 20 mmHg

RIGHT VENTRICLE

RV Systolic: 20 — 30 mmHg

RVEDP: 0 — 7 mmHg (approx equal to RA)

PAWP

PAWP : 4 — 12 mmHg (higher in ventilated patients)

LEFT ATRIUM

LA : 4 — 12 mmHg (similar to PAWP)

LEFT VENTRICLE

LV Systolic: 100 — 140 mmHg

LVEDP: 4 — 12 mmHg (similar to ;PAWP:LA)

AORTIC PRESSURES

SystolicBP: 100 — 140 mmHg

[dependent on DBP & Pulse Pressure]

DiastolicBP: 60 — 80 mmHg

[dependent on Systemic vascularresistance]

Pulse Pressure: 40 — 60 mmHg

BP : 70 — 90 mmHg

$$BP = \frac{SBP+2DBP}{3}$$

$$= DBP + \frac{1}{3}PP$$

$$BP = CO \times SVR$$

CORONARY AND CARDIAC ANGIOGRAPHY

The process of taking pictures of the blood vessels using X-ray imaging and injections of the X-ray dye is called Angiography. If there are no blockages in the coronary arteries, the dye flows smoothly from the opening of each artery through all the branches until the all branches become too small to see. If there is partial blockage, they appear as irregularities in the column of the dye that fills the artery. Complete blockages, or occlusions results in dye not being able to flow along branches that otherwise would be seen.

When dye is injected into left ventricle or other cardiac chambers, it fills the chamber fully and then is ejected and replaced with ordinary blood that has no dye in it.

The x-ray movies that are taken while the chamber is full of blood show how the walls of chamber move, and whether there are abnormal masses with in the chamber.

The person undergoing left heart cathertization feels a singing and some burning as the numbing medicine (local anesthetic) is injected into the skin, and some times some temporary deep pain as the introducer (through which the catheters are passed into the artery), is placed in the artery. The person may feel some fluttering as the catheter is positioned within the heart itself, may feel anginal pain when dye is

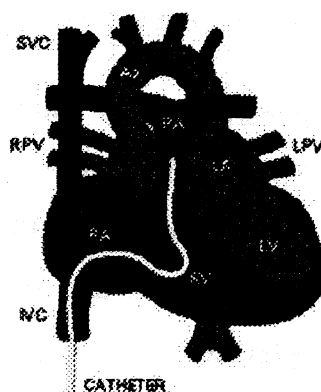
Injected into the coronary arteries, and will almost certainly feel a hot flash as dye is injected rapidly into the left ventricle during the ventriculogram.

The risk of left heart catheterization are moderate, and arise from four principle steps: insertion of a catheter into the artery, positioning of catheters in the heart, leaving the catheters in heart for minutes at a time, and use of x-ray dye. The risks include bleeding and or obstruction of the artery; puncture of the heart with the catheter, dissection of the aorta or a coronary artery with the catheter. the risk can cause death, myocardial infarction and stroke.

More minor risk can create the need for emergency surgical repair, usually of the arterial puncture. The contrast agents can cause allergic reaction, cause damage the kidneys. As with many procedure that is not free of risk, it should be performed only when its estimated benefit exceeds the estimated risks.

RIGHT HEART CATHETERIZATION

Right heart catheterization into the heart through a vein in the arm, the neck, or groin. The veins are the brachial, subclavian, internal jugular and femoral veins respectively. This procedure allows measurement of the rate of blood flow and pressure gradient across the tricuspid and pulmonic valves, and the pressure in the right atrium. When pacemaker electrodes or a biopptome are placed in the heart instead of hollow catheter, the procedure is called cardiac electro physiology and cardiac endocardial biopsy respectively.



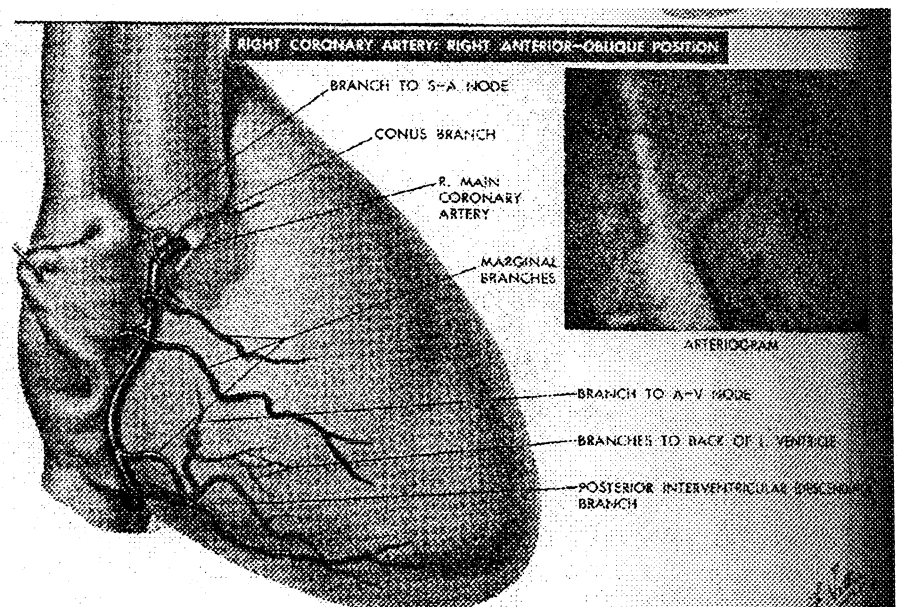
Why we are doing right heart catheterization?

When a person has an enlarged heart for which there is no other explanation sometimes having Atrial septal defect or ventricular septal defect. The concept behind the use of catheterization is that blood on the right side of the heart, which is on its way to the lungs, should have relatively little oxygen. Blood on the left side of the heart, which comes from the lungs. Should have a lot of oxygen. The amount of oxygen in the blood should not change much from place to place on either side of the heart by measuring oxygen saturation at various place on both sides of heart abnormal changes in the oxygen content can be detected. If there is a left to right shunt, there will be an increase in oxygen content starting at the shunt continuing into the lungs. In the case of right to left shunt, there

will be a decrease in oxygen content starting at the level of the shunt and continuing into aorta.

Pressures measured on the right side of the heart are also useful for estimating the risk and benefits of surgery to repair intracardiac shunts and valves, and for transplanting hearts.

The risk of right heart catheterization is small, and arises from two principal steps: Insertion of catheter into the vein, particularly when the vein is in the shoulder or the neck, and positioning of the catheter into the heart. The risks include pneumothorax and myocardial puncture with pericardial effusion and tamponade.



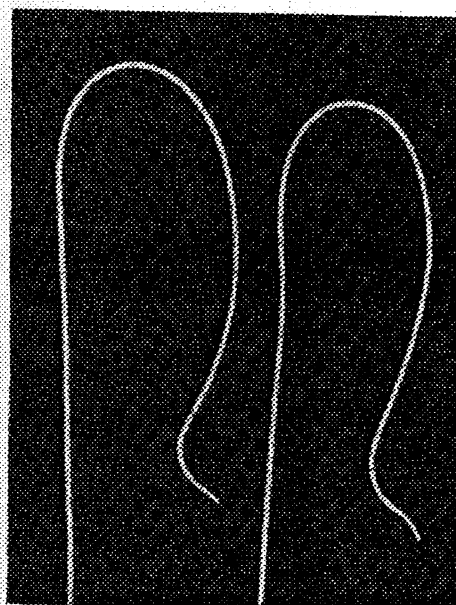


Figure 9.35. Amplatz right coronary catheters: A, ARI. B, ARII.

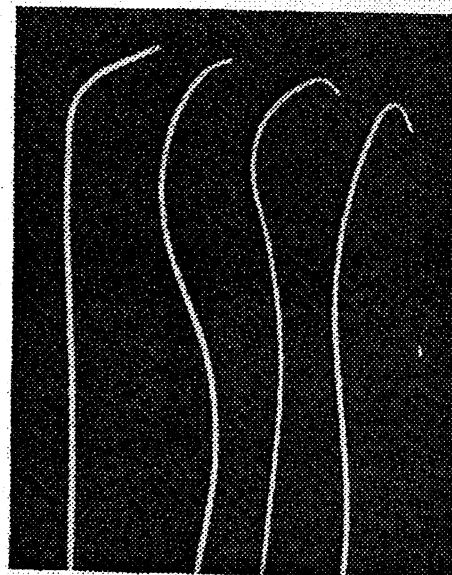


Figure 9.36. Multipurpose and bypass graft catheters. A, Multipurpose. B, Right bypass. C, Left bypass. D, Internal mammary artery.

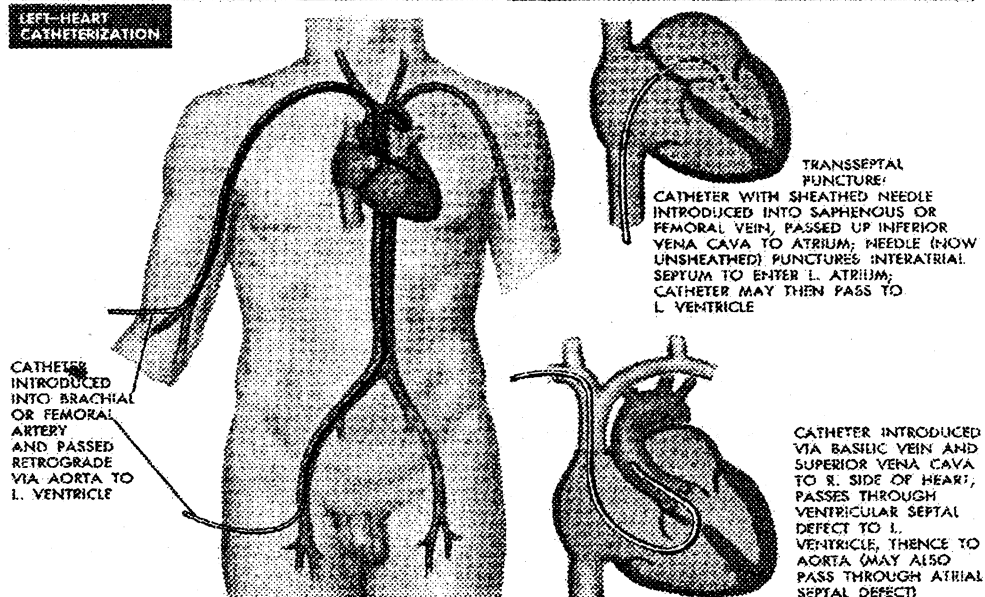
LEFT HEART CATHETERIZATION

Right heart catheterization allows measurement of the rate of blood flow (cardiac output), the pressure gradient across the tricuspid and pulmonic valves, and the pressure in the right atrium, right ventricle, pulmonary artery, and the left atrium (via indirect pulmonary capillary wedge pressure'). Left heart catheterization allows measurement of pressure gradient across the aortic valve. Simultaneous right and left heart catheterization allows measurement of pressure gradient across the mitral valve, and oxygen saturations (which measure the amount of oxygen in a sample of blood taken from various parts of the heart and great vessel) can be measured simultaneously both sides of the heart.

These additional pieces of information are important for several reasons.

When a valve is suspected of being too tight, it is useful clinically to estimate the "valve area", which is the size of the opening in the valve when it is opened as far as it will go. The valve area cannot be estimated from only the gradient because the gradient depends on the area of the opened valve ("cardiac output").

It is important to note that valve areas can also be estimated by echocardiography, which can be performed with much less risk than can cardiac catheterization. If echocardiography estimates the valve as being not too bad, the senior cardiologist on the case should make final decision. The reasons behind these rules of thumb are (1) echocardiography can be wrong in its estimates of valve gradients, (2) catheterization carries a small but definite risk of morbidity and mortality, and (3) valve replacement surgery carries a larger risk of morbidity and mortality.

LEFT-HEART
CATHETERIZATION

CARDIAC BIOPSY

A "biopsy" is a small piece of tissue that is removed from the body to diagnose illness. Cardiac biopsy is performed mostly in people who have had heart transplants in order to see whether the body is rejecting the new heart. It is sometimes performed in people who have unusual symptoms to see whether they might have rare diseases such as viral myocarditis or cardiac amyloidosis.

A “ cardiac biopsy” is the removal of a small piece of heart tissue (myocardium) using a small pincer-like device called “biptome”. The tissue is usually removed from the interventricular septum. The biopsy process starts with placing the biptome into a large vein, usually the right internal jugular vein in the neck or right femoral vein in the groin. Using fluoroscopy to make sure the biptomes goes where it supposed to go , the biptome tip is positioned to right ventricle against the septum. Its jaws are open, the tip is pushed gently against the septum and the jaws are then closed around a bit of muscle about the size of a few grains of rice. The biptome is then removed from the body, and the piece of tissue is removed from the biptome using sterile technique. The process is repeated until about three to five satisfactory pieces of heart muscle are removed.

The risks of procedure are those of introducing a needle into a large vein (bleeding, infection, blood clot, puncture of the lung or near by artery when the needle goes into the shoulder or neck), and the risk of making a hole in the heart with the biptome.the last risk is quite small but there is always the possibility of emergency heart surgery if a large hole is made that won't close on its own.

CATHETERS

A catheter must have

1. Axial control-Ability to directly transmit forces from the end of the catheter to the tip.
2. Body- Segment of catheter between the tip and hub.
3. Contrast medium delivery-Ability to deliver high contrast material flow rates within a specified injection pressure range.
4. Flexibility-Ability of a section of a catheter to bend on contact with a resistant surface.
5. Internal diameter- Diameter of the internal lumen of the catheter, which determines a guide, wires can accommodate and expected contrast medium delivery.
6. Maneuverability- Ability to advance a catheter around a sharp bends or through tortuous vascular segments.
7. Memory- Ability to recover and maintain a specific configuration after insertion and guide wire removal.
8. Pliability- Ability to bend and shaped.
9. Pressure monitoring characteristics-Ability to accurately transmit pressure from catheter tip to pressure transducer.
10. Pushability- Ability to directly transmit force applied to the hub of the catheter longitudinally to the tip.
11. Radiopacity-Ability to visualize the catheter under fluoroscopic control.
12. Softness-Ability to easily bend.
13. Stability- - Ability of a catheter to remain in position, a function of stiffness, memory and a matching of catheter to anatomy.
14. Strength- ability to withstand high-pressure injections.
15. Back-up support- Ability to remain in position despite resistance.
16. Torque control-Ability to directly transmit rotational forces from the end of the catheter to the tip.
17. Trackability-Ability of a catheter to follow a guide wires along its course through the vascular anatomy.

CATHETER CONSTRUCTION

Cardiac catheters composed of many layers. In most multilayered designs, one tube is stretched over another to form a bond.

Most multilayered catheters consist of an inner tube of Teflon, over which is a layer of nylon Woven Dacron or stainless steel braiding. A tube of polyethylene or polyurethane is then heated and extruded over the two inner layers, to bond firmly as a third or external layer of catheter.

The inner layer provides a smooth surface through which guide wire and contrast agents may pass, and should be nonthrombogenic. The thickness of the filaments and density of wire or nylon braiding in the middle layer, and the type of plastic used determine the stiffness and torque control of the catheter.

External layers determine the catheter performance. It must be coated with radiopaque material, such as barium, bismuth. Sometime it may soften the catheter material and produce fine pitting of the surface of the catheter and leads to the thrombogenicity. to overcome this, the material may be incorporated into the middle layers or apply nonthrombogenic material like silicon.

Polyethylene and polyurethane have different character to the catheter. Polyethylene is relatively resistant to the softening effect of radiopaque material, but can be softened by heating to allow reshaping for special tip configuration. Polyethylene must be gas sterilized because of its heat instability. Polyurethane is less resistant to heating, is a soft material due to more random molecular arrangement. Many companies design "High flow" catheters. The high flow design incorporates a thinner catheter wall and large internal lumen for a given external catheter. This allows for higher contrast flow rates at a specific injection pressure. It provides fairly good torque response, but sacrifices flexibility and pliability and lead to catheter kinking during advancement or rotation.

CATHETERS SIZES

Catheters for angiographic use are sized by external and internal diameter and length. The internal diameter is specified either by actual diameter in thousandth of an inch or mm, or by the maximum diameter guide wire which can be passed through the catheter. External diameter is expressed in French sizes, which are obtained by multiplying the actual in mm by 3.0

French = diameter (mm) * 3.

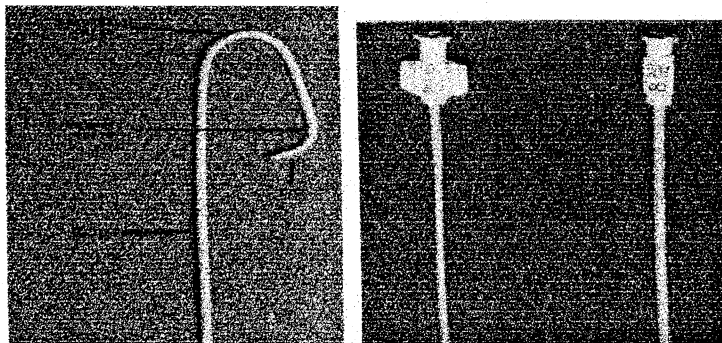
1" = 25.4mm

1mm = 3F = 0.039"

1F = 0.013 "

| French | Size mm |
|--------|------------|
| 1F | 1/3 |
| 2F | 2/3 |
| 3 F | 3/3 |
| 9F | 3mm |

French sizes from 5-8 are currently used for diagnostic angiography. Catheters vary in length, depending on configuration and purpose, and on the route of insertion. Most frequently used pigtail catheters are 100 cm in length and Judkins catheters are 100cm in length, brachial catheters have 80 or 100cm.



CATHETERS OF RIGHT HEART CATHETERISATION

Balloon flotation catheters

Introduction of the balloon flotation catheter for clinical use by Swan and Ganz. The use of a flow directed catheter for right heart catheterization without fluoroscopic control has significantly advanced management of patients in critical care areas. Most balloon flotation catheters are made from PVC in a multiple extrusion, multilumen

construction. The balloon is usually composed of latex and has inflated volumes ranging from 0.8 to 2.2cc. Catheters vary in length and diameter from 60 cm 5Fr for pediatric use to 110cm 7Fr for use in adults. The catheter has minimum of lumen for balloon inflation and additional lumen according to their use. A berman angiographic catheter has its second lumen for injection of radiographic contrast and has no end hole, but it has multiple side holes to help prevent catheter recoil during rapid injection of contrast agents.

The Cournand catheter: it is a woven Dacron, polyethylene or polyurethane, single end hole catheter, designed specifically for right heart catheterization .it can be used for selective blood sampling, and with its singly end hole it can be used for obtaining pulmonary capillary wedge pressure measurements.

The Goodale-Lubine catheter: It is similar to cournand catheter, except that it has two side holes close to the tip in addition to its end hole making it very useful for blood sampling.

NIH Catheters-it has no end hole but having side holes and is used for angiography



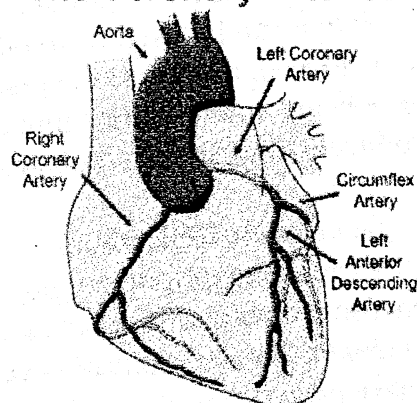
pigtail

Lehman

NIH

Gensini

The Coronary Arteries



CORONARY ANGIOGRAPHIC CATHETERS

Coronary angiographic catheters can be grouped into several series;

Sones, amplatz, multipurpose, Judkins, and Bypass graft.

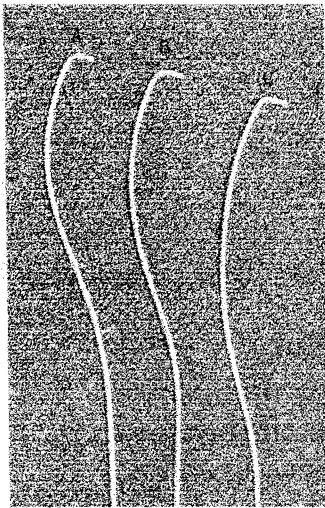
Sones catheters-It is initially designed for insertion via brachial artery cut down and for percutaneous brachial insertion via sheath system. Original construction was based on Woven- Dacron, but polyurethane and polyethylene also used.

Judkins catheters: the principle advantage of Judkins catheters lies in their ability to naturally seek the coronary orifice when advanced into the respective sinus of valsalva. there are two types Judkins left coronary and right coronary catheters. The judkins catheters have specially designed curves and tapered end holes and are available in different French sizes and curves. The left coronary catheters have 3 curves, primary, secondary, and tertiary. The performed curves are designed on the principle that the proximal and transverse portions of the aortic arch, ascending aorta, and left coronary ostium lie in approximately in same plane.

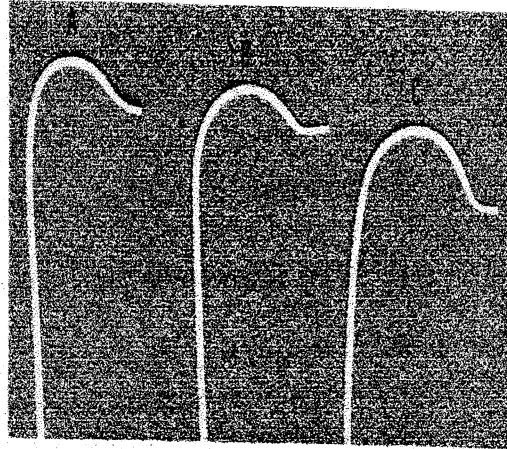
Amplatz catheters- these are particularly used in situations in which Judkins catheters are not suited to given anatomic variation, particularly

when the coronary artery originates from high in the sinus of valsava. Amplatz size 1 is for the smallest aortic root, 2 for normal, 3 for large roots. Advantage of Amplatz catheters is @it can be used from either the femoral or the brachial artery. @ Attempts to seek a coronary ostium require less operator training. @ They have only an end hole; thus immediate dampening of the pressure waveform is produced when the tip is obstructed.

Multipurpose catheter-It is single catheter technique; can be used from the femoral approach and for both ventriculography and angiography. It is made up of polyurethane or woven Dacron with stainless steel incorporated. the tip is more flexible tip than that of Gensini. Torque control is very good and can be manipulated into either coronary ostia or left ventricle.



Judkins right coronary



Amplatz-AL1, AL2, AL3

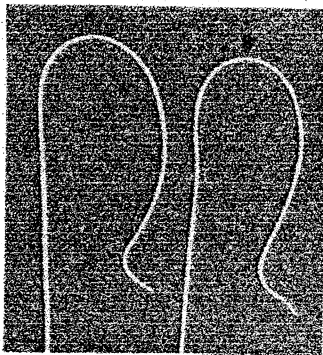


Figure 9.35. Amplatz right coronary catheters: A, ARI; B, ARII.

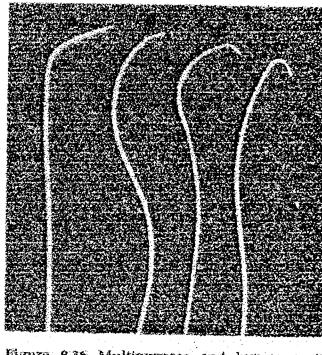
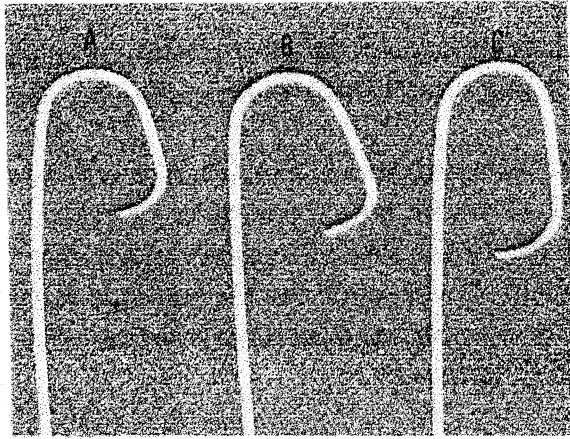
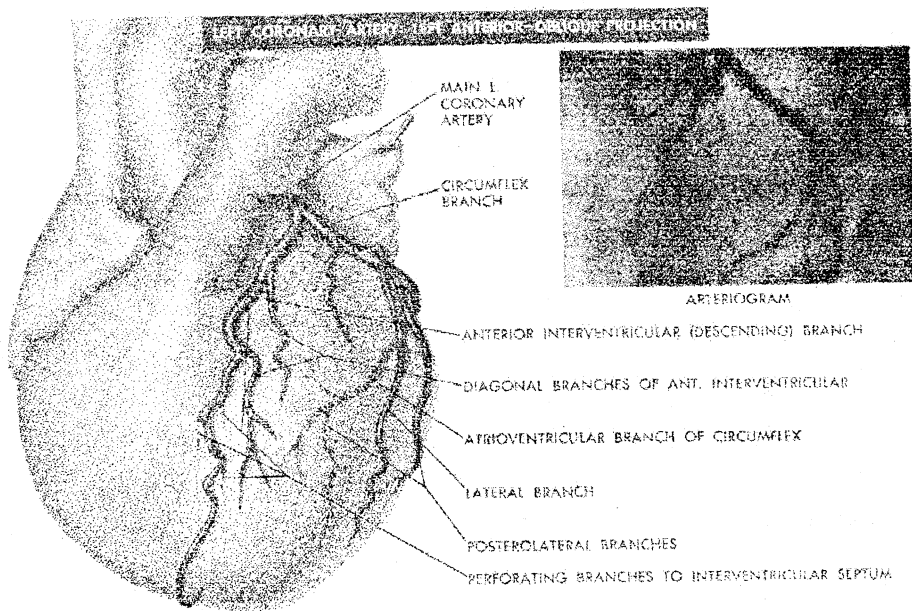


Figure 9.36. Multipurpose and bypass graft catheters. A, Multipurpose; B, Right bypass; C, Left bypass; D, Internal mammary artery.



Left coronary catheters



GUIDE WIRES

Guide wires have very much importance in cardiac catheterization. The discussion of guide wires is going to its basic construction, size, length and shape.

Spinning a thin strand of round wire around a metal tube or rod usually makes the coil. The coil is advanced over a core or mandrel and fixed at the ends by a soldered bond. At the flexible end or tip, a safety ribbon is soldered and runs the length of the guide wire adjacent to the core. A gradually tapered core allows for a smoother transition from the very flexible end or tip to the stiffer body of the wire.

Coating may be applied as a bath or spray to the assembled wire and then bake-dried in an oven to ensure adequate bonding. Wire coatings provide for lubricity and reduced thrombogenicity. Despite Teflon coating, the irregular surface of coiled guide wires continues to predispose to thrombus formation. Stainless steel wires are currently used predominantly for venous access, and not for catheter guidance or manipulation in the arterial system. The wire designed by Terumo Corporation consisting of an elastics alloy ore coated with a polyurethane jacket and a hydrophilic coating. This gives the excellent torque control and reducing thrombogenicity.

Guide wires are available in lengths from 35-260 cm, and may be cut into any length. The shorter length is used for the introduction of venous access. Guide wires most frequently used in diagnostic angiography are either 0.035 or 0.038 inch external diameter. The 0.035-inch wire allows advancement of a catheter over it with less internal friction than the 0.038-inch wire. 0.038 inch wire is used for advancing through fibrotic subcutaneous tissue or calcified arterial wall or stenotic valve. The 0.025 wires is reserved for smaller diameter catheters or when there is a difficulty in advancing a 0.035 inch wire. Diameters such as 0.016, 0.018, 0.025, are available also.

ACCESSORY EQUIPMENTS

1. ADAPTERS
2. STOPCOCKS

3. MANIFOLDS
4. TOUHY-BORST
5. MICRO PUPNCTURE NEEDLES
 - SINGLE WALL
 - SELDINGER TYPE
 - MODIFIED COURNAND TYPE
6. DILATORS
7. SHEATHS

ADAPTERS

Provides a means of connection between instruments, classified as male and female; the female adapter can only be connected to a male connector and a male adapter to a female connector

STOPCOCKS

Valve attachments to control the passage of fluid (blood, contrast, parenteral fluids) that open and close with the turn of a lever.

MANIFOLDS

A series of stopcocks placed in either a Y formation or a linear formation. The linear formation allows for the discreet regulation of individual stopcocks

TOUHY-BORST

It is an adapter that provides a continuous flush in coaxial systems between the two systems.

MICROPUNCTURE NEEDLES

Used to reduce complications in patients when they are at a risk for bleeding, or to prevent accidental puncture to nearby nerves or vital organs. They are also useful in arteries that are prone to spasm or in patients with grafts or poorly palpable arteries.

One piece or single walled needles are multipurpose needles that consist of a beveled needle with a base plate; there is no stylet. Two-piece or double walled needles consist of an outer cannula with an inner stylet and obturator. Examples include that two-piece seldinger or modified Potts needle.

Three-piece or sheath needles consist of a cannula, stylet and sheath. The stylet is in the cannula, and this system is inside the sheath. Examples include the three-piece potts Cournand.

NEEDLES

Single wall
Seldinger type
Modified courante type

PERCUTANEOUS ARTERIAL NEEDLES

Arterial needles generally are 2 1/8 " long and come in a variety of gauge sizes. The 16 to 20 gauge needles are generally used, with the 18 gauge being the standard. All needles used in angiography are thin-walled in comparison to hypodermic needles, which are not. This allows the passage of the guide wire through the needle. A variety of needles are also available for special interventional procedures. Percutaneous arterial needles are available in the variety of designs.

DILATORS

Dilators are thick walled plastic tubing with a tapered end that provides a tract from the skin surface to the vessel, assuring smooth catheter entry. The dilator is most commonly used when a guide wire is in a vessel but catheter placement is difficult, when a vessel is heavily scared, when plaque is present at the puncture site, or in the presence of a graft. In the case of dilatation of the graft, the interventionist will find it necessary to over dilate, by going up one French size.

SHEATHS

Sheaths provide smoother and safer catheter introduction during procedures that require multiple catheter exchanges or arduous manipulation of the catheter. Sheaths usually come with safety features such as a side port for heparin and or contrast installation and for obtaining pressure measurements. There should also be a haemostasis valve to prevent air aspiration and blood backflow. Sheath also come with a interlocking dilator to aid in the insertion of the sheath, once the sheath dilator is in the artery, the interlocking hub can be detached and the dilator portion can be removed.

CONTRAST MEDIA

IONATED CONTRAST MEDIA

When we talk about contrast in radiography it is the difference in optical density between two points of the radiograph. The difference in density (contrast) between bone, muscles, fat and gas forms the basis of the plain radiograph. But these plain films have certain limitations like if we take abdominal radiograph, the outline of the kidney is visible because it is surrounded by perinephric fat but the pelvic calceal pattern is not visible.

Similarly adjoining fat and bone does not delineate the fine structures of many organs, and the detail is not visible because of lack of contrast. Artificial contrast media is employed to delineate or enhance the details from the organs. Contrast agents are used in three basic ways in diagnostic imaging; direct injection into a vascular lumen, intravenous administration to evaluate distribution of various compartments and intravenous or oral administration.

Contrast media may be divided into two groups – positive and negative. Positive contrast media are those, which have high atomic weight and provide positive contrast. E.g., Iodine and barium sulphate (BaSO_4) suspension. Negative contrast media have low atomic weight and provides negative contrast. E.g., air, CO_2 , O_2 etc.

IODINATED RADIOGRAPHIC CONTRAST MEDIA

Iodinated contrast media are the one, which is most commonly used, in daily radiographic practice. The use of iodinated water-soluble

contrast agent is not based on their pharmacological action but on their distribution and elimination from the body. Therapeutic agents are given to effect biological

and chemical changes, whereas contrast media are used only for organ or tissue enhancement. So the ideal contrast agent for angiography and urography is one that would provide excellent X-ray opacity without disturbing normal physiological function or exerting toxic effects. This agent should not penetrate the cell membranes or cross the intact blood brain barrier. It would be excreted rapidly in unchanged form.

HISTORICAL DEVELOPMENTS

The first report of opacification of urinary tract by renal excretion rather than by retrograde introduction of contrast agent appears in 1923, when Osborne et. al. took advantage of the fact that intravenously injected 10 % sodium iodide solution which was used in the treatment of syphilis was excreted in the urine.

Binz and Rath in Berlin synthesized a number of pyridine neutral to detoxify the iodine. One of these selectman neutral was excreted in the urine but the images were poor.

Moses Swick suggested some modification to the molecules and in 1928-29 the first urogram with the compound uroselectron (Iopax) were performed. This was a monoiodinated compound, which was further developed into iodinated compounds Uroselectron B (Neoiopax) and diodone (Diodrast, Umbradil) and in 1952, the first tri-iodinated compound, sodium acetrizate (Urokon) was introduced in clinical

radiology. Sodium acetrizoate was based on a six carbon ring structure, tri-iodo benzoic acid, and was the precursor of all modern water soluble contrast agents.

In 1955, a much safer derivative became available – Diatrizoate (Urographin, Hypaque). This was a fully substituted benzoic acid derivative and an acetamide group at the previously unsubstituted position 5 of the benzene ring. Isomerization of diatrizoate and substitution at position 5 of:

N-methyl carbamyl produced of the iothalamate ion (Conray) in 1962.

Modern hyper osmolar contrast media are distinguished by difference at position 5 of the anion and by the cation sodium and / meglumine.

Solubility of contrast media is achieved by using contrast media salts, which has high solubility in water. To make a contrast media salt, the Hydrogen (H^+) is removed and replaced by a different cat ion, i.e. a particle, which also carries positive electrical charges.

Two cat ions are in common uses are sodium (Na^+) and meglumine (Mgl^+) a short form of Methyl glucosamine.

Ionic contrast media dissociates into ions when in solution. With the solution, which we are talking about now, the positively charged particle is either the Na^+ or Mgl^+ (meglumine). The one with the negative charge is the left over acid group along with the attached benzene ring, its iodine and other substituents. This negative ion is responsible and necessary for contrast.

Major problem with this contrast media are electricity and the number of charged particles.

Electricity:- If we inject a substance, which has electrical charges it, may exert electrical effects such as heart arrhythmias because of calcium ion binding.

The next disturbing fact about this agents were its dissociation into two particles, the side effect of contrast medium are related to the number of particles in the solution. The higher the number of particle in a given volume, the more pronounces the side effects. The number of particles in a solution can be measured. If there are many particles agents are said to have a higher osmolality.

Most of the authors suggest that the higher the osmolality the more pronounce will the side effects. Consequently, if we could reduce the osmolality, we should be able to reduce the side effects. The above mentioned contrast media molecule containing three iodine at the benzene ring gives us two particles in solution. That means the iodine to particle ratio is 3:2. Two particles give us the contrast effect of three iodine atoms. So the next challenge was to reduce the number of particles but without changing the iodine concentration.

Two solutions to this problem were formed; one was the synthesis of dimeric contrast medium (Dimer means two benzene rings are coupled, in contrast to a monomer, which contains only one benzene ring).

In the dimeric substances (Ioxaglate), two iodine-carrying benzene rings were connected by bridge. However, only one of them had the "salt" forming acid group. This means that when we dissolve this substance.

We still get two particles, however, the iodine to particle ratio is 6:2. In other words, for the same number of particles, we have as many iodine atoms. Or stated in the other way, the same number of iodine atoms we need only half the number of particles in solution. Consequently, such a contrast agent has less osmolality for the same amount of iodine. The resulting molecule is however also larger, which increases the viscosity of the contrast medium. This dimeric substance has less osmolality related side effects. There are however still electrical charges, i.e. it is still an ionic contrast media.

The other way to solve the problem turned out to be better. The idea was to take away the acid i.e., the salt forming group and replace it. The constituent taking its place had to, however allows for sufficient water solubility of the compound.

Research led to contrast media, which no longer fell apart into ions, the non-ionic contrast media like Iopromide.

The iodine to particle ratio with this substance is 3:1. Consequently, a similar contrast solution with same iodine contents has a much lower osmolality. Besides having less osmolality these non-ionic substances have an additional advantage. They no longer carry electrical charges. These agents are better tolerated and have much fewer side effects than ionic contrast media.

CM research did not stop with development of “non-ionics”. They still had a higher osmolality than blood; reduction in osmolality was attempted by combining the two rings of non-dissociating type mentioned before, in order to make a dimeric non-ionic CM. That was IOTRALAN. Which has iodine to particle ratio of 6:1 i.e., still lower osmolality. This can be used in an iso osmolal conc., and is currently the best tolerated CM.

PROPERTIES

OSMOLARITY AND OSMOLALITY

OSMOLARITY

A one molar solution contains one mole {i.e., about $6 \times (10 \times 23)$ particles} of solute in one litre of solution, for CM that means, one litre of CM contains one mole of particles. The actual water content of conc., CM solution is about 0.7 litre of CM.

As one litre of water may contain a different number of water molecules, Depending on the temperature, osmolarity is influenced by temperature. For this reason, not osmolarity, but osmolality is used for CM.

A one molal Solution contains one moles particle in one Kg. of water. A Kg. is Kg., and is unaffected of temperature.

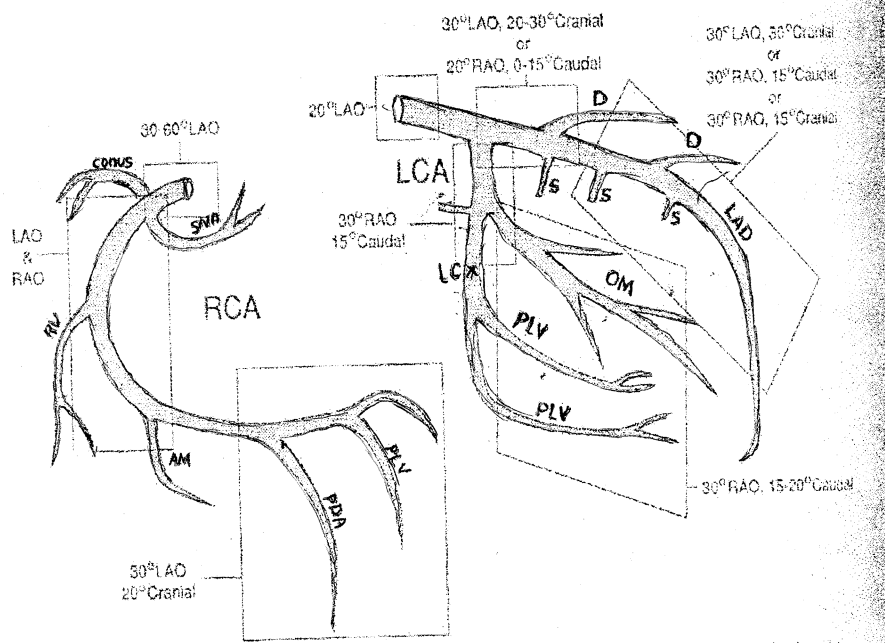
OSMOLALITY OF CM AND BODY FLUIDS

Osmolality of blood, or fluid inside cells is about 300 (270 – 320) mille iosmol / kg. The osmolality of a CM naturally depends on its conc.

And as we have heard, on the type of CM. Basically, the higher its iodine content, the higher its osmolality, if we consider the same type of CM,

I.e., ionic monomers, non-ionic monomers, or non-ionic dimer.

INTERVENTIONAL
PROCEDURES

242 / *Diagnostic Cardiac Catheterization Techniques*

DIAGNOSTIC CARDIAC CATHETRIZATION OF CORONARIES

PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY (PTCA)

INTRODUCTION

Andreas Gruentzig in 1977, performed the first PTCA. Over the first 20 years, improvements in the equipments and technique have result in dramatic growth of PTCA as a successful method of coronary visualization. PTCA was shown to be more effective than medical therapy in relief of angina in single vessel coronary disease. . PTCA is accomplished with a small balloon

catheter inserted into an artery in the groin or arm, and advanced to the narrowing in the coronary artery. The balloon is then inflated to enlarge the narrowing in the artery. When successful, PTCA can relieve chest pain of angina, improve the prognosis of patients with unstable angina, and minimize or stop a heart attack without having the patient undergo open-heart coronary artery bypass graft (CABG) surgery.

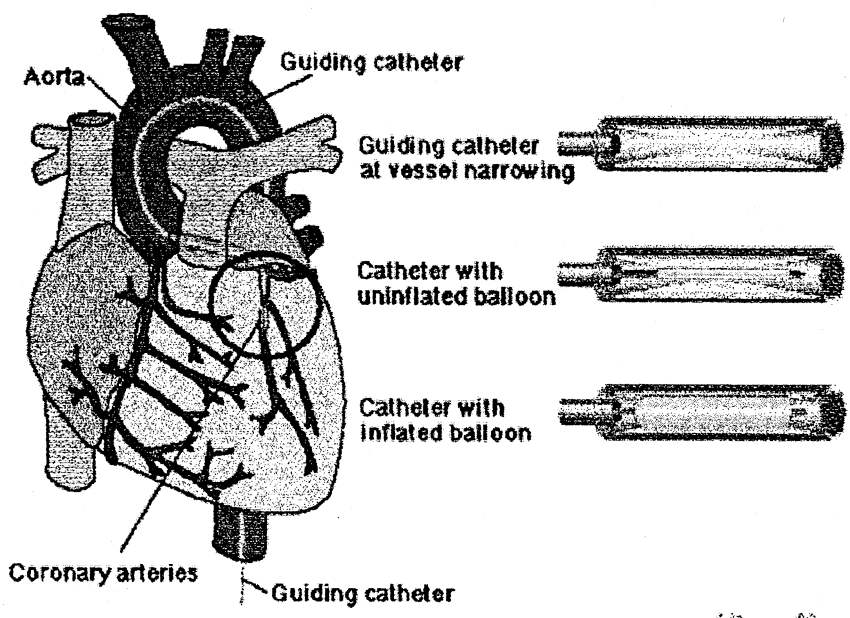
P percutaneous means access to the blood vessel is made through the skin

Transluminal means the procedure is performed within the blood vessel

C coronary specifies that the coronary artery be being treated

Angioplasty means, "to reshape" the blood vessel (with balloon inflation) also referred to as "balloon treatment" because special balloons are used to open up obstructed arteries.





Attwell

BALLOON ANGIOPLASTY

OVERVIEW OF THE METHOD

Under -fluoroscopy, a guiding catheter is inserted into the femoral or brachial artery and is advanced into the ostium of the narrowed artery of the heart. The balloon catheter is loaded with a thin, steerable guide wire (0,014-0.018in. diameter). The balloon is loaded into the guiding catheter. The guide wire is advanced into the coronary artery and positioned across the stenotic area. Tracking the balloon over the guide wire and positioned across the stenotic area and inflated there. This process produces clefts in the atheromatous lesion, compression and distribution of its contents and stretching of the media. In most cases this results in a substantial increase in the atrial lumen.

Mechanism of angioplasty

Disruption of plaque and the arterial wall

The inflated balloon exerts pressure against the plaque, and the arterial wall, causing fracturing and splitting. Concentric lesion fracture and split at its thinnest and weakest points. Eccentric lesions split at the junction of the plaque and arterial wall. Dissection or separation of the plaque from the medial wall releases the splinting effect that is caused by the lesion and results in a larger lumen. This is the major mechanism of balloon angioplasty.

INDICATIONS FOR PTCA

1. Angina pectoris causing sufficient disability to warrant coronary artery bypass graft surgery in spite of optimal medical therapy.
2. Mild angina pectoris with objective evidence of ischemia and coronary lesion in a vessel supplying large area of myocardium.
3. Unstable angina.
4. Acute myocardial infarction in-patient who have contraindication to thrombolytic therapy or who have evidence of persistent or recurrent ischemia despite thrombolytic therapy.
5. Angina pectoris after coronary artery bypass graft surgery.
6. Symptomatic restenosis after successful PTCA

CONTRA INDICATION TO PTCA

1. Unsuitable coronary anatomy eg: left main
2. High-risk coronary anatomy in which closure of vessel would result in patient death.
3. Contraindications to coronary bypass graft surgery.
4. Bleeding diathesis (low platelet count, coagulopathy etc)
5. Patient noncompliance with procedure and post PTCA instructions.
6. Multiple PTCA restenosis.

COMPLICATIONS OF PTCA

1. death (<1%)
2. Myocardial infarction.
3. Emergency coronary artery bypass grafting (<5%)
 4. All complications that can occur during diagnostic cardiac catheterization can also occur during PTCA

- a) Access site bleeding, especially with larger sheaths and prolonged anticoagulation.
- b) Contrast-media reactions.
- c. cerebral vascular accident, myocardial infarction, and so on.

5. Vascular injury.

HARDWARES USED FOR PTCA

1. Guiding catheter
2. The balloon catheter
3. Coronary guide wire
4. Accessory equipment

1. Guiding Catheter

A special large lumen catheter is used to guide the coronary balloon system or interventional device to the vessel of the lesion to be dilated. A guiding catheter serves major three functions during angioplasty. It provides balloon catheter delivery and guidance, back up support for balloon advancement, and pressure monitoring. A guiding catheter provides a method for the delivery of the balloon catheter to the coronary ostium. If the guiding catheter is not seated coaxially, it may not be possible to advance the balloon to the stenotic area. Support or back up for balloon catheter advancement is achieved after cannulating the guide catheter in the coronary ostium. Inadequate back up support will result in failure to cross a lesion and an unsuccessful procedure. The guiding catheter measures aortic pressure during the procedure.

The balloon catheter

There are three types of angioplasty balloon catheters

1. Over the wire
2. Monorail (rapid -exchange)
3. Perfusion balloon catheters

Over- the wire balloon catheter

This balloon catheter has a central lumen through out the length of the catheter for the guide wire and another separate lumen for balloon inflation. These balloons are approximately 145-155cm long and can be used with guide wires of various dimensions. In this system, the guide wire and the balloon catheter move independently. The major advantage is the ability o maintains artery access with the guide wire beyond the lesion while

exchanging one balloon catheter for another. But it has very low balloon profile and it must need very good catheter back up support.

Rapid-exchange balloon catheters

Rapid exchange monorail balloon catheters were developed to improve exchanging angioplasty balloon catheters by single operators. These catheters have only a variable length of the catheters shaft containing two lumens. One lumen runs the entire length of the catheter and is used for balloon inflation the other lumen, which extends only a portion of the catheter shaft., houses the guide wire. Because only a limited portion of the balloon requires dual lumens, rapid exchange catheters are smaller, improving contrast visualization of the artery. But it includes the need for excellent guiding catheter support and difficulty with simultaneous manipulation of the guide wire, balloon catheter.

Perfusion balloon catheter

The perfusion catheter is variant of the conventional monorail balloon catheter system with multiple side hole proximal and distal to the balloon, communicating directly with the central lumen of the catheter, allowing atrial blood to flow through the proximal side holes, down the center lumen and out side of the distal side hole with the balloon inflated in the lesion. This balloon catheter permits perfusion of the myocardium during balloon inflation. A perfusion catheter is selected to reduce severe angina, hypotension from compromised ventricular function, or when prolonged inflation are required. Its disadvantages are related to bulky size, it makes it difficult to track.

Angioplasty guide wires

Coronary guide wires were very small caliber, steerable wire advances into the coronary artery or branches beyond the lesion to be dilated. The characteristics are determined by the core and tip. The shorter the distance between the center of core and the distal wedge tip, the more rigid and torquable the wire will be. Larger guide wires have better torquability and steerability and provide more support, while small diameter wires are more trackable. The main characteristics of guide wires are stiffness, steerability, flexibility, malleability and radiopacity.

Accessory equipments.

1. adjustable haemostatic and rotating Y-connector valve

The Y-connector is attached to the guide catheters to permit the introduction of the balloon catheter into the guide while allowing injection of contrast agent through the guide catheter connector is an accessory device that minimizes the bleeding through the guiding catheter while balloon catheters is inserted when attached to the balloon catheter, pressure around a guide wire can be measured.

2. Balloon inflation device.

Disposable syringe device delivers pressure to inflate the balloon on the angioplastic catheter. The pressure gauge displays the exact pressure. The normal balloon inflation pressure is around 4 to 12 atm.

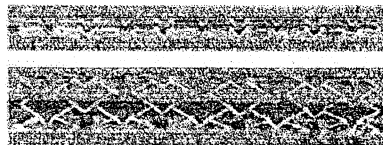
3. Guide torque device

A small cylindrical pin wise clamp slides over the proximal end of the guide wire, permitting the operator to perform the fine manipulation of the guide wire.

3. Guide wire introducer

A very thin needle like tube with a tapered conical opening helps the guide wires to be inserted into balloon catheter and through Y-adaptors.

STENTS



stent shown in the delivery state (above) and in the expanded installed state (below)

Stents are tiny mesh like tubes made from stainless steel. They are placed permanently inside an artery to hold it open after balloon angioplasty. The actual procedure for the placement of the stent is the same as an angioplasty with the addition of the stent placement. A stent may be used to keep an artery open that has closed or partially closed after a previous angioplasty to improve the flow of blood. In some case, stents are used when blocked vein bypass grafts are opened through angioplasty.

They are 'deployed' in the artery by either expansion by a balloon or by a unique 'self expanding' delivery design. They serve as a scaffold to prop the inside of the artery (the lumen) open, which increases blood flow to the heart muscle. They are permanently deployed devices that stay in the artery. They

ultimately become covered with cells and in essence become part of the artery over time.

Symptoms Experienced during the procedure of stenting

- ❖ A slight burning or stinging from the medicine used to numb the catheter insertion site.
- ❖ Chest pain may occur as the balloon catheter is being inflated, but it is temporary.

After the procedure

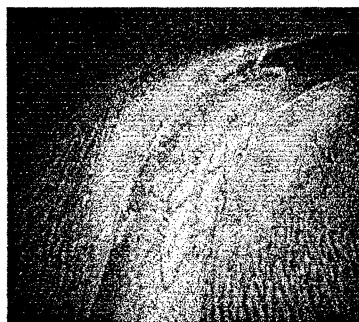
- ❖ The sheath is usually left in place until blood-thinning medications are discontinued and clotting time returns to normal.
- ❖ A band –aid or pressure dressing will be placed over the area where the catheter was inserted.
- ❖ The patient will be admitted to a special care unit (ICCU) to be closely observed.
- ❖ The insertion site will be checked frequently for signs of bleeding.

Signs and symptoms to be reported immediately

- ❖ Discomfort or sudden pain at the site of insertion
- ❖ Bleeding
- ❖ Any discomfort in chest, shortness of breath, weakness or dizziness.

DRUG ELUTING STENTS

Restenosis remains the main drawback to the use of coronary artery stents. While radiation for in-stent restenosis is a promising approach, there are drawbacks to therapy and it doesn't always work. Much more useful would be a means of preventing the restenosis in the first place.



Coated and Drug-Eluting Stents

More and more, the solution moved away from the purely mechanical devices of the 90's and toward pharmacologic advances that were being made. If interventional medicine, using the body's circulatory system as a "highway" to deliver therapy, worked with devices, it could also work with medicines. Physicians and companies began testing a variety of drugs that were known to interrupt the biological processes that caused restenosis. Stents were coated with these drugs, sometimes imbedded in a thin polymer for time-release, and clinical trials were begun.

One immunosuppressant drug named Sirolimus is manufactured by Wyeth-Ayerst (it is found in the soil of Easter Island) and Johnson & Johnson / Cordis has coated their CYPHER™ Stent with it.

The second large clinical trial recently reporting Boston Scientific TAXUS II, utilizing Boston Scientific's stent, coated with a polymer, which elutes a chemotherapy agent Paclitaxel, manufactured by Angiotech of Canada. Paclitaxel is a version of Taxol. The SIRIUS II results showed a restenosis rate of 2.3% and 4.3% for the slow and moderate-release cohorts respectively. Paclitaxel was also used in the Cook ELUTES trial and the Guidant DELIVER trial, both of which have reported very similar positive results. The technology seems to work! (Note: Recent longer term results from the Guidant DELIVER trial, while positive, did not show a great enough difference between their coated stent and their bare-metal stent, thus effectively ending Guidant's Paclitaxel stent program. The Guidant stent was coated with the drug, whereas the Boston Scientific stent embeds the drug in a polymer, which releases it slowly over time. It may be that this difference in drug-delivery is crucial.)

"Stent Wars"

The status and availability of drug-eluting stents are the subject of many legal disputes and other factors, which we have labeled "Stent Wars". Since marketing analysts predict that drug-eluting stents are so successful clinically that they will double the current world market for stents to \$5 billion annually, it is easy to

understand the flurry of activity among and between all of the competing device manufacturers.

Currently three coated stents, the Cordis CYPHER™, the Cook V-Flex Plus and the Boston Scientific TAXUS™ paclitaxel-eluting stent system, have received the CE Mark and are available in Europe. Additionally, the Cordis CYPHER™ has just become the first coated stent to receive U.S. FDA approval. Boston Scientific hopes to gain FDA approval for marketing its stent in the U.S. late in 2003. Nine-month data for this stent's effectiveness will be announced at the 2003 Transcatheter Cardiovascular Therapeutics (TCT) meeting in September.

IDENTIFYING VULNERABLE PLAQUES

Not all coronary lesions are equal. Some atherosclerotic plaques are relatively stable, and relatively unlikely to cause heart attacks. Other plaques are particularly vulnerable to rupture, which triggers the clotting phenomenon that produces sudden blockage of the coronary artery and thus heart attacks.

Unfortunately, when a partial blockage is viewed on a standard catheterization, there is no way to distinguish stable and vulnerable plaques. Therapy, therefore, is directed towards the degree of blockage, and not whether a plaque is actually more prone to rupture. MRI techniques are now being actively developed to help distinguish between stable and vulnerable plaques. This technique will allow doctors to target their therapy more effectively to the very lesions that pose the highest risk.

ANGIOGENESIS

Angiogenesis is the growth of new blood vessels. Scientists have long known that naturally occurring proteins called Growth Factors can stimulate blood vessel growth. Early trials have used infusions of GF into coronary arteries to stimulate new blood vessels in areas of the heart where the coronary arteries have become blocked. These trials have been promising.

The gene, which codes for a form of the GF protein, was delivered to the heart by infusing the gene in an adenovirus "package". Viruses are essentially packages of DNA coated with protein. When a virus infects a cell, it uses the cell's protein factory to "express" the DNA borne by the virus.

Thus the infected cell begins making proteins coded by the viral DNA. This trial showed that patients receiving the gene therapy showed evidence of significantly improved coronary artery blood flow.

While much work needs to be done to assure the safety and effectiveness of this approach, it is likely that gene therapy to stimulate angiogenesis will become a viable, routine treatment for patients with severe coronary artery disease within the next several years.

ROTATIONAL ANGIOPLASTY (ROTABLATOR)

The rotational angioplasty system is a catheter based angioplasty device utilizing a diamond-coated elliptical burr at the tip of the flexible drive shaft. Tracking coaxially over a guide wire and rotating at up to 190,000 RPM, the burr ablates plaque into fine particles that are disposed by the body's reticuloendothelial system.

INDICATIONS FOR USE

PERCUTANEOUS ROTATIONAL ANGIOPLASTY is a sole therapy or adjunctive balloon angioplasty, is indicated in patients with coronary artery disease who are acceptable candidates for CABG and who meet in of the following selection criteria.

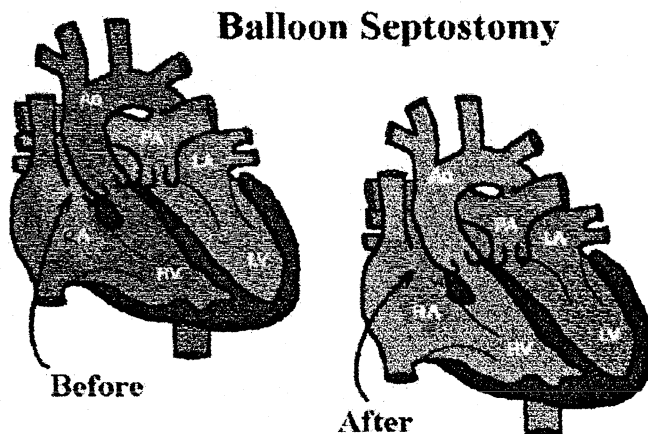
- Single vessel atherosclerotic coronary artery disease with a stenosis that can be passed with a guide wire.
- Multiple vessel coronary artery disease that in the physician's judgment does not pose undue risk to the patient.
- Certain who have had prior PTCA, and who have a re stenosis the native vessel.
- Native vessel atherosclerotic CAD that less than 25mm in length.

CONTRAINDICATIONS

- Occlusions through which a guide wire will not pass.
- Last remaining vessel with compromised ventricular function.
- Saphenous vein grafts
- Angiographic evidence of thrombus prior to treatment with rotational angioplasty. Such a patient may be treated with thrombolytics (urokinase). When thrombus has been resolved for two or four weeks, lesion may be treated with the rotational angioplasty.
- Angiographic evidences of significant dissection at the treatment site.

THE BALLOON ATRIAL SEPTOSTOMY

Balloon atrial septostomy is the standard initial therapy for the infants with d-transposition of great arteries in this congenital anomaly; the pulmonary and the systemic circuits run in parallel rather in series, resulting in severe hypoxia and acidosis shortly after birth. In this setting septostomy allows bi-directional mixing at the atrial level, resulting in an immediate rise in arterial oxygen saturation with alienation of acidosis. The 4F or 5F Rash kind septostomy catheters are introduced through the femoral vein and the tip of the catheter is advanced in the mid LA, using biplane fluoroscopy. The balloon is held against the atrial septum and inflated rapidly. The catheter is advanced 1 or 2 mm before being pulled briskly to the IVC/RA junction, advanced to the RA and then rapidly deflated. This sequence is usually repeated at least twice to ensure that an adequate atrial septal opening has been created. A successful outcome is associated with a rapid rise in the arterial blood of oxygen saturation, evidence of bi-directional shunting in the atrial level, and abolition of inter atrial pressure gradient. Complications are extremely rare, but tears of pulmonary veins and atrial walls have occurred.



PERCUTANEOUS BALLOON ANGIOPLASTY OF COARTATION OF AORTA

Percutaneous balloon angioplasty of coartation was first described in 1982 and has since been used in large number of patients with native coartation and postoperative recoartation. Coartations have been dilated

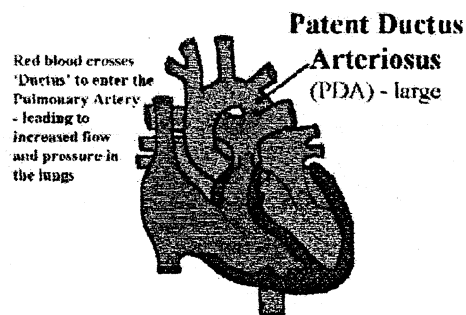
using Antegrade approach, but the retrograde femoral arterial approach is preferred for most patients. Right and left heart hemodynamics is measured and careful pull back is performed to localize gradients. Biplane aortograms are performed and diameters of the narrowest area of coarctation and the normal proximal and distal aorta are measured. The balloon dilatation catheter is advanced over the exchange wire, centered across the coarctation, inflated until the waist disappears and deflated. An aortogram is performed following dilatation to determine diameter of the narrowing and to detect tears, aneurysms, or dissections.

EMBOLISATION OF PATENT DUCTUS ARTERIOSUS

Patent Ductus Arteriosus (PDA) is one of the best known of all congenital cardiac anomalies; it was the first to be treated via surgery. It represents about 8% of all congenital heart disease, and is twice as frequent in females as in males. The incidence is considerably higher in prematurely born infants, particularly those with respiratory distress.

PDA anatomy varies considerably in both size and configuration. The PDA diameter arbitrarily refers to its narrowest segments, which is smaller than 4 mm in 78 % of cases. To be effective, an occlusion device must adapt to the diameter, length and shape of the ductus.

PDA's have been classified into five types. The most common in one large series is type A funnel shaped ductus with a localized narrowing at the pulmonary artery junction. Type B, the next most common (18%) includes funnel shaped PDA's with an aortic ampulla. The remaining include type C (tubular shape), type D (Oval shape with both aortic and pulmonary ampullae) and the type E (other bizarre forms).



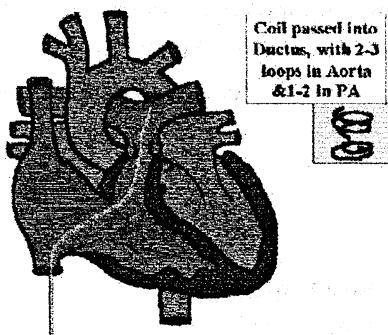
PDA COIL EMBOLISATION

Recently coils have been used to routinely close small PDAs. Using a retrograde approach from the femoral artery, the catheter is advanced through the PDA in to the pulmonary artery.

Approximately a third is advanced out of the catheter, and then the catheter is pulled back until the loops are at the pulmonary end of the duct. The catheter is then withdrawn over the coil is delivered on the aortic side of the PDA the coil is chosen to be twice the diameter of the narrowest part other the PDA and to have sufficient length to form four lops as it coils. Coils also can be delivered from the femoral vein.

Multiple coils can be delivered simultaneously with minimal risk of migration.

Coil Occlusion of PDA



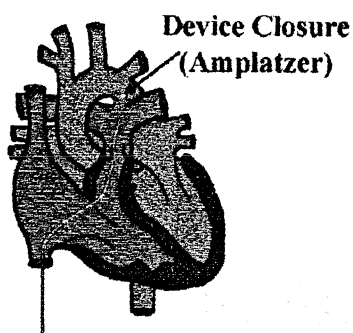
PDA DEVICE CLOSURE

The Amplatzer Duct Occluder can generally be used for all five types of PDAs. In many cases, it can also be used to close calcified or short, window-type PDAs. Both are difficult to close successfully with surgical ligation.

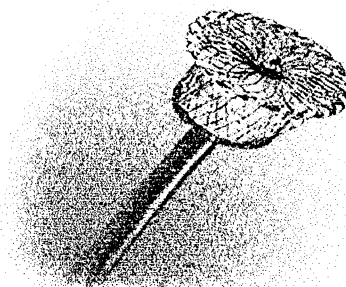
In the case of large communications, PDA closure is usually required for hemodynamic reasons to decrease left to right shunting, and thereby prevents pulmonary hypertension and cardiac failure. Closure is also indicated, however, for prevention of bacterial endocarditis.

Although the risk of endocarditis with small residual shunts remains unknown, continued bacterial endocarditis prophylaxis is generally advocated as long as flow persists, or a murmur can be heard. Interestingly, one recent experimental study in a swine model showed no susceptibility to direct infection as long as near complete transcatheter closure of the shunt was accomplished. Nevertheless, the current consensus is that every PDA with a murmur should undergo closure, and that closure should be complete, although these closures are often performed surgically, a variety of transcatheter occlusion devices have also been used. Such techniques offer a simpler, less invasive alternative to traditional surgical methods.

Most current PDA device closures, however, present such drawbacks as technical complexity, uncontrolled deployment, non-retrievable designs, and risk of misplacement or embolization. The Amplatzer Duct Occluder was designed specifically to address such drawbacks.



The Duct Occluder



CLOSURE OF ATRIAL SEPTAL DEFECTS

Atrial Septal Defects (ASDs) are congenital abnormalities characterized by structural deficiency of the atrial septum. ASDs account

for 10% of all congenital heart disease, with a 3:2 female/male ratio the most frequent type of atrial septal defect is in the ostium secundum (fossa ovalis) location.

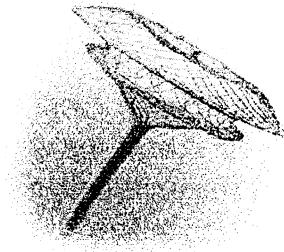
The physiological consequences of an atrial septal defect depend on the magnitude and duration of the shunt, and on the response of the pulmonary vascular bed, in large defects with significant left to right shunts, the right atrium and right ventricle are volume overloaded, and the augmented volume is ejected into a low-resistance pulmonary vascular bed. Pulmonary vascular occlusive disease and pulmonary arterial hypertension may then develop in adulthood.

Survival is limited in young adults who develop progressive pulmonary hypertension. Even these patients, however, may live to reach their 40s. In their 50s, these patients experience increasingly frequent atrial arrhythmias, which represent a common precipitating cause of heart failure patients diagnosed as having secundum ASDs with a significant shunt (defined as pulmonary blood flow to systemic blood flow ratio (Q_p/Q_s) of >1.5) are operated upon ideally before 5 years of age or whenever a diagnosis is made in later years. Left to right shunt has in the past been measured through cardiac catheterization with oximetry. With the advent of two dimensional echocardiography and Doppler color flow mapping, it is now possible to visualize the anatomy of the defect and estimate the Q_p/Q_s ratio from Doppler indices.

Cardiac surgery requires cardiopulmonary bypass equipment to achieve closure through a right atriotomy. Small defects are sutured shut, and larger ones are patched with pericardium, polyester, or Gore-Tex. The mortality reported in 1968 was as high as 12.5% this has been progressively reduced to 6%, to 3.3% and currently to less than 1%. The presence of significant residual shunts has been reported in as many as 17% of patients undergoing repeat catheterization, and in the current era only 2% of patients undergo repeat cardiac surgery for residual shunts.

A variety of devices for transcatheter closure ASDs have been developed in the past few decades. Until now, however, none of them have been entirely satisfactory. The Amplatzer Septal Occluder may offer a number of advantages over other septal occlusion devices.

The Septal Occluder

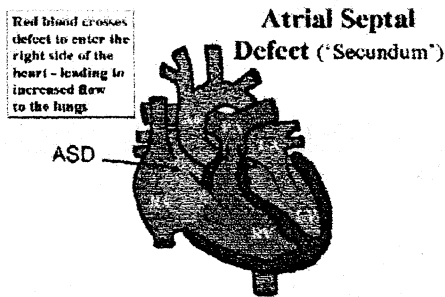


DEVICE CLOSURE OF ASD USING THE AMPLATZER SEPTAL OCCLUDER

DESCRIPTION: - The AMPLATZER Septal Occluder is a self-expandable, double disc made from a NITINON wire mesh. The two discs are lined together but a short connecting waist corresponding to the size of ASD. In order to increase its closing ability, the discs and the waist are filled with three polyester patches. The polyester patches are securely sewn to each disc by a polyester thread. The device is securely screwed on to a delivery cable and loaded into an introduced sheath. The device is advanced and the distal disc is released in the left atrium. It is pulled against the septum, which can be felt and observed by transesophageal echocardiography. The right atrial disc is then deployed in the right atrium by pulling back the sheath and the device is disconnected by turning the delivery cable in the direction indicated on the plastic vise (provided).

INTENDED USE

The Amplatzer Septal Occluder is a percutaneous, transcatheter, atrial septal defect closure device designed for the occlusion of secundum atrial septal defects and patent foramen ovale in patients with a history of stroke or transient ischemic attacks (TIAs).



INDICATIONS

SECUNDUM ATRIAL SEPTAL DEFECTS

1. Secundum atrial septal defect
2. Diameter <40mm
3. Dilatation or RV with evidence or RV volume overload
4. Left to right shunting
5. A distal of >5mm from the margins of the defect of the coronary sinus, AV valves and right upper lobe pulmonary vein.

PATENT FORAMEN OVALE (PFO) WITH PARADOXIAL CEREBRAL EMBOLISM (PCE)

Patients with patent foramen ovule (PFO) with a history of stroke of Transient Ischemic Attacks (TIAs) diagnosed by transesophageal echocardiography with contrast studies during the vasalva maneuver, and patients with patent foramen ovule with associated aneurysm of the septum.

CONTRA INDICATIONS

1. Associated congenital cardiac anomalies, which require cardiac surgery (VSD, PDA ETC).
2. Ostium primum atrial septal defects
3. Sinus venous atrials septal defects
4. Anomalous pulmonary venous drainage (partial or complete)

5. Severe pulmonary arterial hypertension / bi-directional or right to left shunting.

PATENT FORAMEN OVALE WITH PARADOXICAL CEREBRAL EMBOLISM

1. PFO without aneurysm of the fossa ovalis
2. Demonstrable thrombus in the pelvic vein or inferior vena cava
3. Carotid angiogram and or Doppler evidence of carotid disease
4. A left heart source of embolism identified on Transthoracic and / or transesophageal echocardiography
5. Atrial fibrillation

GENERAL EXCLUSION CRITERIA

1. Inferior vena cava thrombosis / pelvic vein thrombosis leading to total occlusion
2. Sepsis (local / generalized)
3. History of repeated pulmonary infection
4. Any type of serious infection <1 month prior to procedure
5. Malignancy where life expectancy is <3 years
6. Demonstrated intracardiac thrombi on echocardiography (especially left atrial or left atrial appendage thrombi)
7. In patients with a history of allergy to iodinated contrast agents (eg. Anaphylaxis), the procedure will be performed under TE ultrasound guidance
8. Previous surgery for esophageal atresia
9. Inability to obtain informed consent
10. Children less than one year age

PROCEDURE

1. Following percutaneous puncture of the femoral vein, standard right heart catheterization performed
2. The left atrium is catheterized and an angiogram is performed using a 45 degree LAO position and with cranial angulations by injecting contrast medium into the left atrium or into the right upper lobe pulmonary vein in order to demonstrate the atrial communication

3. An exchange "J" guide wire (2cms) is introduced into the left atrium and a balloon catheter is inserted over the exchange wire into the left atrium
4. The balloon catheter is inflated with various increments of carbon dioxide or contrast medium and pulled across the atrial communication. There should be only a slight deformity of the sizing balloon to determine the stretched diameter. Sizing of the defect can be repeated several times, which is very important for the appropriate selection of the occlusion device.
5. The sizing balloon is removed and re inflated with the same amount of carbon dioxide or contrast medium and passed through various openings of the supplied sizing plate to determine the stretched diameter. The same size occlusion device or 1 mm larger is selected. The device therefore will be stenting the defect.
6. The delivery cable is passed through the loader and the occlusion device is screwed to the tip of the delivery cable using clockwise rotation clockwise rotation can be achieved right handed.
7. Once securely attached, the device and the loader are immersed in saline solution and the device is pulled into the loader
8. The dilator is inserted into the delivery sheath a secured by tightening the Towhee borsht adapter. The dilator should protrude beyond the sheath such that its tips reach the inferior Vena Cava. Depending on the size of the patient, this length can be adjusted.
9. The sheath in the groin is removed and the dilator-delivery sheath assembly is introduced. Once the delivery sheath has reached the inferior cent Cava, the dilator is removed and the sheath is flushed. The sheath is then advanced over the guide wire through the communication into the left atrium. Correct position of the delivery sheath is verified by the test injection of contrast medium. Once in good position the exchange wire is removed and the sheath is flushed with saline
10. The loader is now introduced through the Touhy Boast adaptor. (if using the 8Fr deliver system or larger, the Touhy Boast adapter must be removed and the loader introduced into the 8 Fr. Sheath). The device is then delivered into the sheath by using the delivery cable without rotations
11. Under fluoroscopic and ultrasonic guidance, the left atrial disc is deployed and pulled gently against the atrial septum, which can be felt and observed by ultra sonography. By gentle tension on the delivery

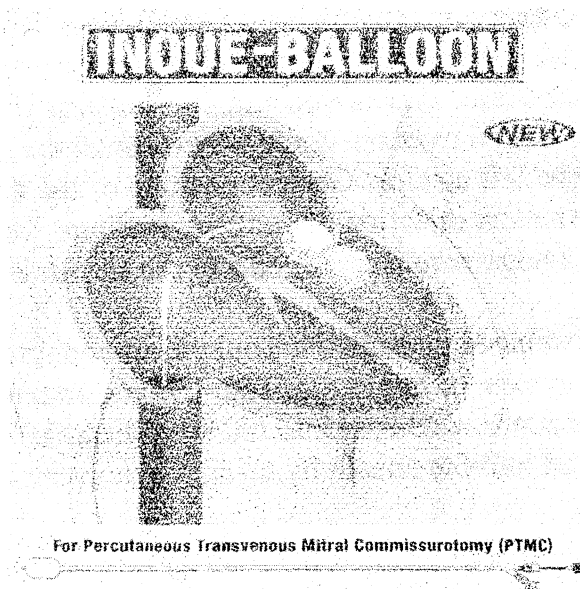
cable, the sheath is pulled back and the right atrial disk is deployed. The sheath is pulled back about 15 cm. Back bleeding will occur. The Touhy Boast adaptor is re-attached and the system is flushed with saline solution. To and fro motion with the delivery cable assures secure position across the atrial septal defect, which can be observed by ultrasound. DO NOT PULL OR PUSH TOO HARD.

12. Attach the plastic vise to the delivery cable, tighten the screw and unscrew counterclockwise as indicated by the arrow on the vise. In the unlikely event that this should not be possible, advance the sheath against the right atrial disc to secure that device, which facilitates detachment.
13. Record procedure information on the appropriate forms.

BALLOON MITRAL VALVULOPLASTY

Balloon mitral valvuloplasty for the treatment of mitral stenosis has been studied extensively over the past 10 years and when successful has been found to yield both marked immediate hemodynamic improvement and sustained clinical benefit.

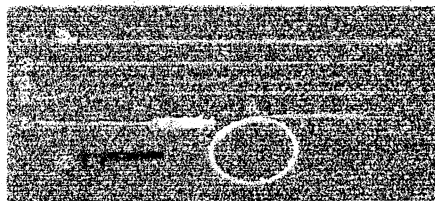
PERCUTANEOUS TRANSVENOUS MITRAL COMMISSUROTOMY (PTMC)



- | | |
|---|---------------------------------|
| 2. Balloon Stretching tube, 19G, 80cm | Elongation of the Balloon |
| 3. Dilator, 14F tapered, 70 cm | Dilatation of the insertion |
| 4. Guide wire, 0.025 stainless steel with Spring coil, 175 cm | Puncture site and atrial septum |
| 5. Stylet, 0.038" stainless steel, 80 cm | Guide for catheter and dilator |
| 6. Syringe | Directing balloon to valve |
| 7. Caliper (ruler) | Inflation of the balloon |
| | Measurement of balloon Diameter |

The New INQUE-BALLOON is Safe, Simple and Easy to Operate

| Description | Use |
|----------------------------|---|
| 1) Dilator | Dilatation of inserted valve |
| 2) Balloon stretching tube | Elongation of balloon |
| 3) Dilator | Extension of insertion orifice |
| 4) Guide wire | Guiding the balloon, catheter and dilator |
| 5) Syringe | Inflation of balloon |
| 6) Caliper | Measurement of balloon diameter |



INOUE-BALLOON

| Cat. No. | Balloon Diameter (Max) | Catheter Size | | Patient Weight |
|-------------------|------------------------|----------------|--------|----------------|
| | | Outer Diameter | Length | |
| PTMC-20N, IMS-20N | 30mm | 19F | 70cm | 5-150cm |
| PTMC-28N, IMS-28N | 28mm | 17F | 70cm | 5-150cm |
| PTMC-30N, IMS-30N | 28mm | 17F | 70cm | 5-150cm |
| PTMC-31N, IMS-31N | 24mm | 15F | 70cm | 5-150cm |
| PTMC-25N, IMS-25N | 22mm | 15F | 70cm | 40-70cm |
| PTMC-22N, IMS-22N | 20mm | 14F | 70cm | 40-70cm |

① IMS-20N, IMS-28N, IMS-30N, IMS-31N, IMS-25N, IMS-22N contains catheter coil wire and springs only.
 ② The IBC without the vent tube has been coded with the letter "N" following the catalogue number eg PTMC-28N.
 ● Package: 5pcs / case ● EOQ: 500

Individually supplied as follows

| Cat. No. | Description | Outer Diameter | Size | Length |
|----------|-------------------------|-----------------|------|--------|
| IMS-2 | Balloon stretching tube | 1.1mm | 19F | 81cm |
| IMS-3 | Dilator | 1.1mm | 17F | 70cm |
| IMS-4 | Guide wire | 0.64mm (0.025") | 17F | 175cm |
| IMS-5 | Stylet | 0.97mm (0.038") | 17F | 80cm |
| IMS-1 | Ruler | | | 62cm |

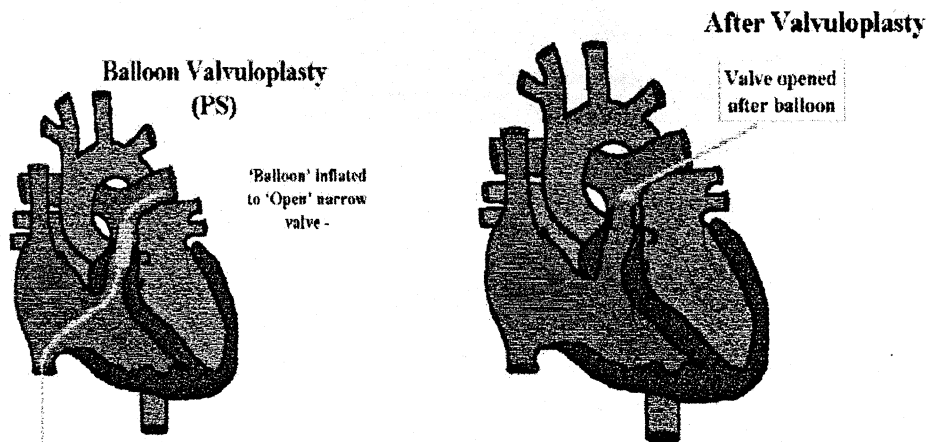
① The new type of balloon stretching tube (IMS-2), dilator (IMS-3) and stylet (IMS-5) are exclusively for use with this product (no vent type). Do not use these for the former type of the IBC (with a vent tube). Do not use the former type of balloon stretching tube (IMS-1), dilator (IMS-3) and stylet (IMS-5) for this product (no vent tube type).
 ● Package: 50pcs / case ● EOQ: 500

BALLOON PULMONARY VALVOTOMY

In 1797 Semb and coworkers reported the first successful use of balloon catheters in the critically ill patient with pulmonary valve stenosis.

Pulmonary stenosis can now be easily treated by percutaneous balloon valvuloplasty. The technique is similar to mitral valvuloplasty. Femoral venous access is obtained. Guide wire placement across the pulmonic valve is followed by a balloon dilatation. Success is determined by the reduction of pulmonary gradient and reduced RV pressure.

Balloon catheters are specifically chosen to dilate with the balloon size 20 to 40 % larger than the annular diameter. In patients with annular diameter is less than 25 mm, a single balloon is used, and two balloons are used for patients with larger diameters. The annular diameter is subsequently measured at the valve hinge points from the lateral view.



AUTOMATIC PRESSURE INJECTORS

The automatic power injector most commonly used today allows for specific volume of contrast to safely be delivered at a precise flow rate, regardless of the variables. Using the electromechanical injector, the parameters can be set at a control panel, directing the amount (mL) of contrast media to be delivered over a set at a control panel, directing the example, 15 mL/sec for 4 seconds would be total volume of 60 mL. The components of the pressure include syringe removable, usually sterile disposable type heating device reduce viscosity of the contrast media High Pressure Mechanism Electromechanical system with a motor drive connected to a screw, that drives the plunger piston plate and transmit motion to the syringe control panel allows for programming and display of the injection parameters, i.e. Volume flow rate, PSI injection delay, film delay, film delay, linear rise, reset/abort button, visible and audible warning alarms.

Functions of an automatic injector include:

Flow rate – the rate at which the contrast media is going to flow. Variable introduce into flow rate include catheter length, diameter and the number of side holes, as well as contrast viscosity.

Volume – the desired amount of contrast you want to deliver Linear rise – the time it take to reach the desired flow PSI (pounds per square inch)- the pressure at which you inject the contrast media. The pressure should never be higher than the catheter can accept or higher that the vessel can tolerate. The smaller the vessel, the lower the pressure.

Injection delay – the filming will start and the injection, according to the delay set. Filming delay-the injection will start first, and then the filming will begin.

Automatic pressure injectors are equipped with multiple safety features to prevent inadvertent damage to the patient or damage to the catheter.

Function monitoring devices: A flashing red light, audible warning or a printed message to notify the operator that a setting has been omitted or that there is one mechanical malfunction.

Volume limiting device: The maximum limit on the amount of contrast administered is set on the control panel, but a backstop lever is located on the head of the injector. Pressure limiting device : Sets maximum pressure (PSI) to be induced, prevents catheter breakage or recoil and vessel damage, ensure safe use with low pressure catheters.

Accelerator regulators: Allows the drive motor to propel over an exact duration of time, to guard against recoil.

Rate rise control: Allows gradual rise in (PSI) instead of an instant surge or pressure all at once.

Additionally, the cath lab technician must take care to properly load the syringe. With the syringe pointing up straight up, clear all air bubbles from the syringe. Automatic injectors have loading and unloading

buttons to draw up and empty contrast. When hooking up to the catheter, point the injector head down. In the event any air bubbles are in the syringe, they would rise away from the catheter. A high-pressure connector is usually used to connect to the catheter. This allows for better visualization of any air connection is made, aspirate back blood and check for any air bubbles in the connection tube. Care should be taken not to aspirate too much blood, as this will dilute the contrast and could possible cause clot formation in the injector syringe. Before the injection begins, the operator and technician should confirm the parameters selected. Once the injection beings, the technician must be prepared to terminate the injection in the event of an emergency.

INJECTORS IN USE

A. Angiomat illumina

This is a digital injection system

Syringe size: 150 ml

Fill rate, forward reverse: 0.25 – 25 ml/sec

Flow rate: 0.1 to 40 ml/s in 0.1 ml/s increments up to 9.9 ml/s, 1.0 ml/s increments thereafter.

Volume: 0.1 to volume is syringe in 0.1 ml increments up to 9.9 ml, 1 ml increments thereafter.

Pressure limit: * 75 to 1200 PSI increments

X-ray delay: 0 to 300 sec.

Air detection system and warning system: looks for air in the neck during the enable process.

B. ANGIOMAT 6000

Volume range: 0.1ml – 9.9 ml in 0.1 ml increments

1ml-150 ml in 1ml increment.

Flow rate range: 0.01ml – 40 ml/sec in 0.01 ml increments.

Duration range: 0.10/255 sec in 0.01 sec increment.

Pressure limit: 127 – 1200 psi in 1-psi increment.

PSHYSIOLOGICAL RECORDERS

In addition to observing and recording images of the heart during catheterization, it is also necessary to observe and record the ECG and various blood pressures with in the cardiovascular system. A reliable electrocardiographic and pressure monitoring system is essential for the safety of the patient and collection of diagnostic information. The typical monitor in the cardiac catheterization laboratory is a multichannel unit that can process, display and record ECG signals and pressure tracings. The transducer and amplifier convert the mechanical energy of the pulsatile column of fluid in the catheter to an electrical signal that displayed on the monitor or recorder.

OXIMETER

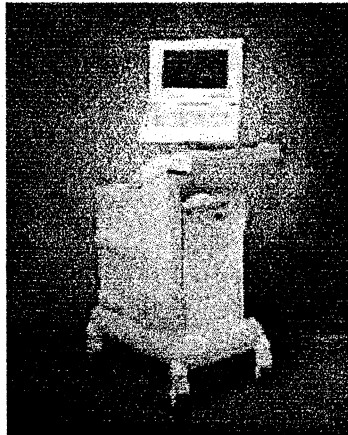
Oximetry refers to the determination of the percentage of oxygen saturation of the circulating atrial blood. The amount of oxygen that delivered to the tissue is determined by the flow rate (cardiac out put), the

oxygen saturation of the atrial blood (SaO₂), and the hemoglobin concentration of the blood.

Oxygen transport = Cardiac output * Atrial oxygen saturation * hemoglobin * 1.34

Oxymetry is the important method to determine the intracardiac shunt. Measurement of the degree of saturation of the blood can be made by spectrophotometric method (transmission oximetry) or using reflection oximetry (scattering of light by using erythrocytes).

INTRA AORTIC BALLOON PUMPING



Augmentation of diastolic pressures was initially studied in 1951, when Kantrowitz noted the resultant increased coronary blood flow during diastole. This discovery was the basis for the development of intra aortic balloon (IAB) catheter by Mouloupoulos and Koff in 1962. Kantrowitz clinically used "Phase shift pumping" with the use of IAB and the pump in 1967 patients with irreversible stroke secondary to myocardial infarction. Since then numerous investigators have studied other physiological aspect of

IAB pumping and have expanded its application to a larger number of clinical situations.

A polyurethane balloon (40 to 60 cc adult size) is mounted on a vascular catheter, inserted into the femoral artery and positioned in the descending aorta just distal to the left subclavian artery. The balloon catheter is connected to a pump console that shuttles helium or carbon-dioxide into the balloon during diastole to inflate it. During isovolumetric contraction, the gas is rapidly withdrawn to deflate the balloon (counter pulsation).

Inflation during diastole augments the diastolic pressure and displaces the blood in the aorta distally and proximally toward the heart and into the coronary arteries and may increase coronary blood flow, although clinical documentation of increased flow have been inconsistent. The major advantage seems to be systolic deflation, which lowers the intra aortic volume and pressures and reduces both after load and myocardial oxygen consumption (MVO₂). These physiologic responses improve the patients cardiac output and coronary circulation and temporarily improve hemodynamics. In general counter pulsation can augment cardiac output by 15%. Frequently, this is sufficient to stabilize the patient's hemodynamic status, which might otherwise rapidly deteriorate.

Until 1979, all IAB catheters were inserted via a surgical cut down, generally of the femoral artery. Since then the development of a percutaneous IAN catheter has allowed quicker and perhaps safer insertion

and has resulted in more expeditious institution of therapy and expansion of clinical application.

The system 97 uses a safety disk drive for adult IAB's. The safety disk is compatible with Data scope balloon catheter extenders, allowing for a range of balloons covering 34 to 50 cc of volume for adults. An adapter for the pediatric safety chamber must be used for pumping pediatric IAB's. A range of 2.5 – 20 cc can be accommodated.

In the STANDBY mode, the operator-selected period of IAB inflation is highlighted at all times on the invasive blood pressure waveform by an intensified brightness on the display screen. When the system is in the ASSIST mode, the pressure trace is completely highlighted. Holding the Verify key when in the Assist mode highlights only the augmented portion of the pressure trace.

An integral, thermal array printer supplies hard copy printouts of ECG, invasive blood pressure, balloon pressure (optional), and trend data along with alphanumeric printout of lead configuration, scale information, pressure data and heart rate. The recorder is capable of printing up to two traces at the same time.

The system 97 contains a RS-232 data connector (9-pin serial data). Status of all monitor module and pump module controls, system variables alarm parameters and status and digitized waveforms (ECG, invasive blood

pressure and optional balloon wave form) are available for direct PC interfacing. All displayed patient data is accessible at this connector.

On selected configurations, the system 97 also incorporates an integral internal modem, which provides the same data as that on the RS-232 data connector. The internal modem is used for remote clinical troubleshooting.

Internal power up diagnostics is provided to indicate failures of specific components and or subsystems via an error message on the display screen. The diagnostics are run each time the IABP is powered up. The series 90 trainers is available as an optional accessory for clinical training. It provides stimulated QRS complexes and arterial pressures waveforms that interact with the system 97 pump controls. The system 97 recognizes the presence of the trainer and the system TRA/NER message displays on the screen.

MEDICAL INDICATIONS FOR COUNTER PULSATION

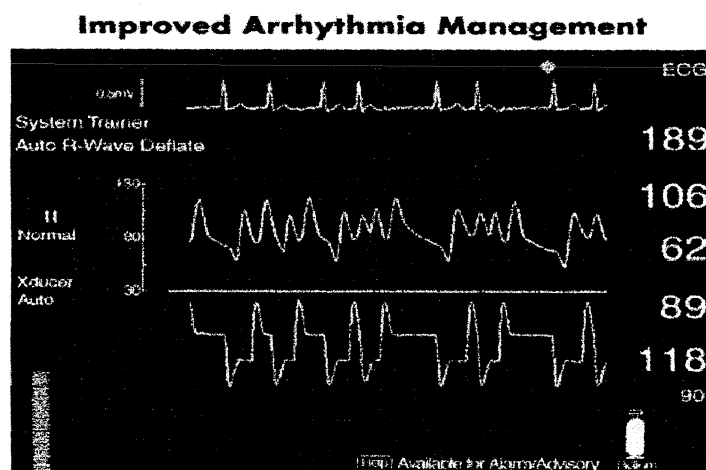
1. Unstable angina refractory to aggressive medical therapy
2. Acute MI with cardiogenic shock
3. Post infarction recurrent angina
4. Papillary muscle dysfunction with acute mitral regurgitation or post infarction ventricular septal rupture.
5. Recurrent intractable ventricular arrhythmias secondary to myocardial infarction
6. In conjunction with PTCA and angiography in unstable patients

7. Terminal cardiomyopathy in patients awaiting cardiac transplant or implantation of an artificial heart
8. Cardiac arrest, in selected cases

SURGICAL INDICATIONS FOR COUNTERPULSATIONS

1. Unsuccessful discontinuation of cardiopulmonary bypass
2. Post cardiopulmonary bypass left ventricular failure and some instances of right ventricular failure.
3. Major non cardiac surgery in patients with advanced LV dysfunction

Probably the most successful use of counter pulsation is an adjunct to preoperative and postoperative measurements of cardiovascular surgical patients with impaired LV function. Elective intraoperative augmentation in high-risk patients has been shown to diminish mortality.



CONTRA INDICATIONS

ABSOLUTE CONTRA INDICATIONS TO COUNTERPULSATION

1. Aortic dissecting aneurysm
2. Severe aortic insufficiency
3. Major coagulopathies, systemic thrombolytic therapy
4. Underlying brain death, advanced or terminal neoplastic disease, or a case in which aggressive life prolonging treatment is not indicated

Relative contra indications to the conventional method of IAB catheter insertion include severe aortic and peripheral vascular arteriosclerosis.

RISKS

1. Ischemic extremities
2. Thrombosis or emboli
3. Arterial perforation
4. Bleeding
5. Infection
6. Aortic dissection
7. Thrombocytopenia

PSHYSIOLOGY OF COUNTER PULSATION

Diagram of normal cardiac pressure flow sequences as compared with the counter pulsed pressure flow sequence. Counter pulsed diastole

mechanically boosting volume flow retrograde to the aortic arch, heightening diastolic pressure and possible coronary perfusion.

INTRA VASCULAR ULTRA SOUNE (IVUS)

HISTORY AND DEVELOPMENT

- Cieszynski in 1956 built an ultrasonic catheter for intra cardiac investigation
- Segall and co-workers made the first device for intraventricular measurement using two crystals
- Eggleton developed four crystal device fro producing cardiac cross sections
- Bom *et at.* In 1969 designed a 32-element phased array catheter working at 5.6MHz.

IVS CATHETER SYSTEMS

Rotational and

Array-based

- US crystal with a center frequency between 20-30 MHz.
- Ability to image close to or the outer wall of the catheter
- Diameter 1-2.5mm and usable length 90-100 cm
- Flexibility – Especially distally, particular for coronary arteries
- Part or the entire catheter should be trackable over the guide wire.

ROTATIONAL CATHETER SYSTEM

- A single crystal mounted on the distal end of the wire. The wire is rotated at high speed while the crystal is excited.
- The ultrasonic waves fall perpendicular to the long axis of the vessel
- Non linear rotation of the catheter will lead to distortion in image quality
- Angulations of the shaft of the catheter due to vessel tortuosity is a problem
- Movement of the catheter within the vessel during data acquisition may lead to image distortion
- The power cable to the crystal also has to spin

ROTATING ACOUSTIC MIRROR CATHETER

- Crystal is fixed at the end of the central wire
- U S waves is aimed retrograde
- Reflected by a rotating acoustic mirror

ARRAY BASED CATHETERS

- Many crystals / a sheet of piezo-electric material oriented around periphery of the catheter
- Each crystal is individually wired or a mulplexer is used to reduce wiring load
- Multiple wires increase stiffness of the catheter
- Cross-talk between the cables may occur leads to bad image quality

- Small cables call for micro soldering techniques

IMAGE METHOD

- The crystal is made to resonate to produce waves of 20-30 MHz
- The waves transmitted out to the tissue interface and reflected back to produce signals
- In rotational catheter the crystal is stimulated frequently during the spin cycle.
- In array based catheter, one or more crystal is fired simultaneously to obtain the data

Incorporation of guide wire is trivial for the array-based catheter technology as these are usually constructed around a central lumen

In rotating type catheter, the guide wire runs adjacent of he catheter past the ultrasound tip, then it runs through a short channel within the distal and of the catheter (monorail principle)

IMAGE DISPLAY

- The data from the catheter is displayed on a monochrome analog display screen or be processed to enhance the image
- The information returning from the catheter is usually displayed as a cross-sectional slice corresponding to an image perpendicular to the long axis of the vessel
- In single crystal catheter the data does not to be processed significantly to produce reasonable cross-sectiona images

- Helps to assess lesion location, present stenosis, length of the lesion and extent of calcification etc. and so useful during angioplasty.
- IVUS offers particular advantage in patients with reduced renal function by reducing the amount of contrast media
- Ultrasound crystals have been mounted within the coronary dilatation balloons allowing monitoring of the immediate result of dilatation
- Areas of calcification, mild stenosis, branch vessel etc. can be identified.

2. ATHERECTOMY – IVUS COMBINATION

- Permits online assessment of the efficiency of plaque removal and thus procedure time is reduced

3. LASER-IVUS COMBINATION

- A system using high-resolution ultrasound for real time guidance and control of laser energy delivery is being developed.
- The laser is directed parallel to the US beam

4. DOPPLER – IVUS COMBINATION

Good quality, high fidelity velocity signals have been recorded from any sites within coronary circulation during coronary arteriography and balloon angioplasty.

5. IVUS DURING STENT PLACEMENT

- Studies conducted at the Miami vascular institute, IVUS was used to assess the degree of stent infiltration against arterial wall after deployment
- Stent status are clearly identified by the i.e. Regularly spaced highly echogenic appearance
- IVUS is helpful in evaluating the extent if intimal dissection and in particular the effectiveness of stenting is eliminating the adverse effects of dissection

FORWARD LOOKING IVUS

- The crystal is directed forward
- In totally occluded vessels the distal lumen is not demonstrated with contrast injection
- The ability of visualize the lumen on the far side of the obstruction would enhance the operators confidence.
- Very helpful for an interventionalist who try to cross a tight lesion.

LIMITATIONS OF IVUS

- The current limitations are the catheter and image processing technology
- Rotating transducer catheters may be minniaturisable but will have difficulty when used in a tortuous anatomy
- Array-based catheters may not suffer this difficulties but may not be easily miniaturized as each crystal needs individual wiring
- For obtaining high-quality real time images a high speed state of the art computing is necessary
- The technology is very expensive

FUTURE TRENDS

With increased complex processing power and an appreciation of the true three-dimensional shape of the vessel, it may be possible to demonstrate the effect of stenosis is different sites within complex geometry like coronary artery.

ELECTROPHYSIOLOGY

ELECTRO PHYSIOLOGY

INTRODUCTION

The modern era of intracardiac electrocardiography began in the late 1960s with the demonstration by Scherlag et al that the His bundle potential could be reliably recorded in humans. The introduction of programmed stimulation by Wellens and Dunner greatly expanded the potential of the conduction systems and abnormalities. Attention passed to the Wolff Parkinson White (WPW) syndrome and other supra ventricular Tachycardia arrhythmia's. Recently ventricular Tachycardia (VT) and the problem of sudden cardiac death have become the major foci. During the evolution electro physiologic study had developed from a research tool to a practical procedure.

SA NODE

INDICATIONS

Sinus node disease

Atrioventricular block

Interventricular conduction delay

Supraventricular Tachycardia

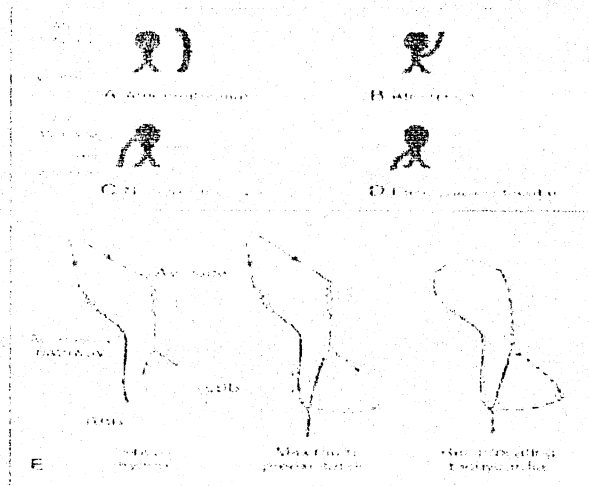
Wolff-Parkinson-White syndrome

Differential diagnosis of wide QRS Tachycardia

Ventricular Tachycardia

Out-of hospital cardiac arrest

Syncope



Cardiac anatomy in relation to EPS

CONTRA INDICATIONS

EPS is contraindicated with (1) when acute factors make the finding unrepresentative of the patient's usual state and (2) when the patient's underlying cardiac disease makes it likely that induced arrhythmias will be extremely difficult to terminate and carry a high risk death.

RISKS

Risks of EPS include those related to cardiac catheterization (thrombolisation, cardiac perforation and tamponade) and infection. Risks specifically associated with electric stimulation include induction of atrial fibrillation VT requiring cardio version and VF.

EQUIPMENTS

The basic equipments required for EPS is described below

FLUOROSCOPY UNIT – Adequate imaging can be obtained with catheterization laboratory equipment or portable unit.

PROGRAMMABLE STIMULATOR – The stimulator must be electrically isolated and allow introduction of one to three precisely timed premature stimuli during both paced and spontaneous rhythm. It should allow selection of the pacing output, interval between stimuli, interval between premature stimuli, and length of pause after each stimulation sequence. It must allow a rapid change for placing modes used to induce arrhythmias to pacing model used to terminate arrhythmias.

MULTI CHANNEL PHYSIOLOGICAL RECORDER

The physiological recorder should allow the simultaneous filtering, amplification and recording of surface ECG and intra cardiac signals. The recorder should be connected to a switch box that allows selection of intracardiac signals. Recording must be possible at paper speed ranging from 50-200 mm/sec. The recorder should generate time lines to allow accurate measurement.

MULTI CHANNEL TAPE RECORDER

A tape record allows the retrieval of any information not recorded during the study and allows replay of intracardiac events to display these for illustrative purposes

RESUSCITATION EQUIPMENT

All standard resuscitation equipments should available in the EP laboratory. Defibrillation pads should apply to the patient's chest at the beginning of procedures so that defibrillation without skin burns can be applied without delay.

INTRACARDIAC ELECTRODE CATHETERS

A catheter with a curved tip with three electrodes spaced 1 cm apart is commonly used for recording the His potential. For atrial and ventricular recording and stimulation, the standard catheter is a quadripolar catheter, with four electrodes spaced 10 cm apart. The distal pair of the electrodes can be used for stimulation while the proximal pair is simultaneously used for recording.

INTRODUCER KITS AND CUT DOWN TRAY

These are necessary for venous or arterial access.

TECHNIQUE FOR PROGRAMMED STIMULATIONS TYPE OF STIMULI

Standard EPS uses bipolar, cathodal, square wave stimuli of 15 to 2 msec. Duration and twice the diastolic threshold.

The threshold is determined by pacing at a high output, gradually decreasing the output during pacing, and noting the output at which capture becomes inconsistent. If the threshold is greater than 2 MA, one should consider repositioning the catheter.

INCREMENTAL PACING

The stimulator allows the administration of stimuli at any fixed rate desired or at any interval desired. The stimulator is synchronized to the native QRS complex, set for the desired interval, and activated. Continuous pacing at a fixed rate occurs until pacing is terminated. The pacing can then be resumed at an increased rate. Incremental pacing is used in evaluating sinus node function, evaluating atrioventricular conductions, including supraventricular and ventricular conduction, arrhythmias, and terminating supraventricular and ventricular arrhythmias.

PREMATURE STIMULATION

Basic drive versus native rhythm – the stimulator allows the introduction of precisely timed premature stimuli during sinus rhythm, during paced rhythms, and during Tachycardia. Paced rhythms ensure less variability and allow testing at a variety of rates, however, extra stimuli during the patients'

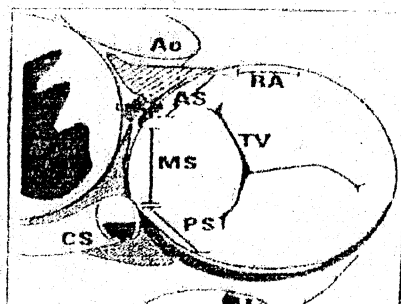
native rhythm may provide more physiologic information because many electrophysiologic properties vary with heart rate, it is important to perform premature stimulation at multiple cycle lengths to maximize the induction of Tachycardia.

In addition, premature stimuli are induced during any sustained observed in the electrophysiology laboratory. Single premature stimuli measuring the refractory period- in general, extra stimuli are administered after every eight beats of basic drive. The extra stimulus is administered later in the cycle and progressively brought closer and closer to the last beat of the basic drive We utilize 20 msec decrements until a coupling interval of 400 msec and 10 msec decrements thereafter. A pause of 2-3 second is allowed between each sequence of basic drive and extra stimulus. The extra stimulus is moved toward the preceding beat until capture does not occur. The interval at which the capture does not occur defines the effective refractory period. The beats of basic drive are from called s1 and the premature stimulus is often referred to as s2/.

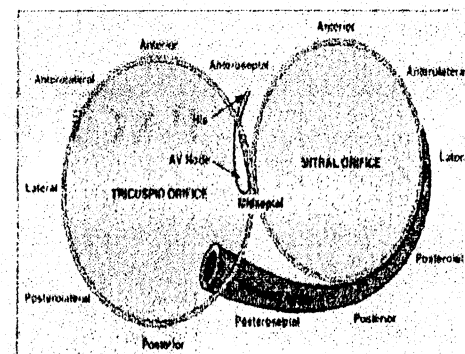
Double and multiple stimulus – the extra stimulus technique has been expanded to include more than one premature stimulus.

In administering a second premature stimulus (s3), the first premature stimulus (s2) is set at 10 msec beyond the refractory period. S3 is administered late in the cycle and gradually moved s2 in 10 msec decrements until it no longer captures. This procedure can be repeated for a third extra stimulus (s4) and so on. Premature stimulus is used in measuring refractory periods, initiating SVT and VT, defining the mechanism of SVT and VT and terminating SVT and VT.

ABLATIVE THERAPY



KENT BUNDLE



overview

There has been great interest in the use of electric charges administered through electrode cathodes to treat certain arrhythmic conditions. This technique was first applied for His bundle ablation in patients with intractable atrial arrhythmias. Unfortunately this technique does render the patient dependent on pacemaker. Complications do include VF, pericardial tamponade, and a small incidence of sudden cardiac death in the months following ablation.

A similar technique has been attempted in patients with accessory pathways. While preliminary reports are encouraging for posterior septal accessory pathways, cardiac perforation and tamponade have been reported in patients with left sided accessory pathways approached through the coronary sinus. The technique is limited by the difficulties of catheter mapping to identify the site of origin arrhythmia and further studies are awaited.

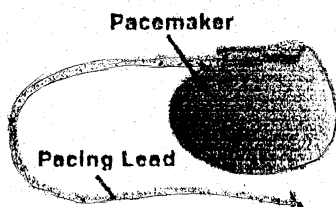
PACEMAKERS

PERMANENT PACEMAKERS

INTRODUCTION

A pacemaker is a small electronic device that regulates the heart beats by sending electrical signals to the heart. The pacemaker device consists of two parts. The battery units; lead, which carries electrical messages back and forth between the heart and the pacemaker. The device is much compatible than previous pacemakers.

PERMANENT PACEMAKERS



INDICATIONS:

The primary indications for a pacemaker are symptomatic Brady arrhythmias. In occasional instances, severe asymptomatic Brady arrhythmias are indications for pacemaker implantations.

The most common indications for permanent pacemaker are as given below:

1. Acquired Atrioventricular block
2. Atrioventricular block associated with mi
3. Sick sinus syndrome
4. Hypertensive carotid syndrome with syncope resulting from bradycardia.

Indications for permanent pacemaker in AV Block

Sick Sinus Syndrome with symptomatic Bradycardia

Second Degree AV Block with symptomatic Bradycardia

Third Degree AV Block with one associated condition

Sick Sinus Syndrome with rate <40 bpm Symptom association with Bradycardia unclear

Asymptomatic third degree AV Block

Asymptomatic Type II second-degree AV Block

Asymptomatic Type I AV Block at His level

First-degree AV Block and pacemaker syndrome symptoms

CONTRAINDICATIONS

There are very few contraindications to permanent pacemaker implantation. Implantations are contraindicated in patients with active infections; the patient should be managed with medications or a temporary pacemaker until the infection is resolved.

RISKS

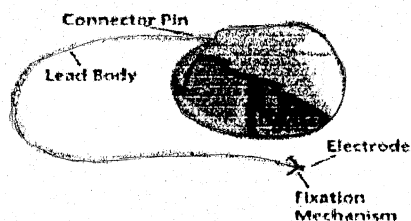
1. Catheter dislodgement, lead fracture, or other causes of pacemaker system failure.
2. Pacemaker syndrome and pacemaker-mediated tachycardia.
3. Infection or erosion of the pulse generator.
4. Cardiac perforations or thrombosis of svc or right atrium.

EQUIPMENT FOR PERMANENT PACEMAKER

IMPLANTATION:

1. Ventricular leads.
2. Atrial leads.
3. 18-gauge vascular needles.
4. j-tip safety guide wire.

5. Vein dilator with a peel away sheath
6. Well grounded ecg
7. Pacemaker system analysis (PSA) to measure thresholds and endocrinal signals.
8. Pacemaker programmer.
9. Fluoroscopy.
10. Standard surgical instruments and suture materials.



PULSE GENERATOR

The pulse generator consists of electric circuitry, based on digital chips and a power source for generating the electric stimuli. Lithium batteries, which lasts a minimum of 6-8 years, are used almost exclusively as the pacer source. The entire source is hermetically sealed in stainless steel or titanium housing to isolates the contents from the biologic environments.

PACING LEADS

Both unipolar and bipolar leads are commonly used. In the unipolar system the cathode is at the distal tip of the catheter, and the body of the pacemaker is the anode. In the bipolar systems the distal tip of

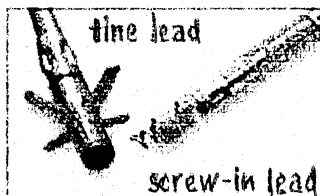
the electrode is cathode; a ring electrode, usually 1 cm proximal to the tip, is the anode. Bipolar systems are less susceptible to myopotential inhibition and less prone to cause pectoral stimulation.

VENTRICULAR LEADS

Ventricular leads, with silicon rubber or polyurethane insulations and are removable stylets are thin flexible and resilient. Lead fixation devices such as fins and tins reduce the incidence of dislodgement and are favoured by most pacemaker physicians. Fins and tins intertwine with right ventricular trabeculae. Active fixation device may be preferable in some patients with markedly dilated right ventricle.

ATRIAL LEADS

The most commonly used atrial lead has J configuration, which allows entry into the atrial appendage. Tine helps to anchor this lead. Active fixation lead has been used to attach lateral atrium and are particularly useful when the atrial appendage have been removed surgically.



PACEMAKER IDENTIFICATION CODE

Pacing systems have been classified according to a universal code. The first letter represents the chamber paced; the second letter, the chamber sensed; the third letter, the pacing mode; the fourth letter programmability; and the fifth letter, the anti Tachycardia functions.

The most commonly used single chamber pacemakers are VVI pacemakers, which have programmable output rate and sensitivity. Pacing and sensing occur only in the ventricle. Intensive ventricular activity will inhibit pulse generator output.

SELECTION OF INDIVIDUALIZED PACING SYSTEM

Selection of proper pacing pulse generator to match the particular clinical situation is of utmost importance. A few principles of pacemaker selection should be emphasized. When the patient with chronic atrial fibrillation requires a pacemaker, it generally should be a VVI pacemaker. VVI pacemaker is also adequate for electrically inactive patients and the patients in whom the pacemaker is largely a standby device, such as those with rare sinus pauses.

PROGRAMMABILITY OF PACEMAKERS

Programmability is the feature by which the electronically controlled performance of a pacemaker can be non-invasively altered. This alteration is permanent and the pacemakers are otherwise reprogrammed.

Programming is achieved by using an external programmer to transmit pre-selected messages in the form of binary code, via, either radio frequency waves or a pulsed electromagnetic field, to the implanted pulse generator, which then assumes its new function. To prevent accidental reprogramming by external time – varying magnetic fields, most of the microprocessor codes required an electronic password before the code can be altered. Some model transmit a message from the pulse generator to the programmer, indicating that the instructions have been revived, accepted acted on; otherwise, confirmation of the success of programming is obtained on the ECG testing.

Features that can be non-invasively including stimulus output (pulse duration, current or voltage), rate, amplifier sensitivity, escape rate, hysteresis, lower and upper rate limits. AV interval, refractory periods and mode.

Output – The voltage (V), current (mA), and pulse duration constitute the pulse generator output; each may be programmable, depending on the model. The output may be increased to manage periods of threshold rise, such as may occur 1-4 weeks after implant, after cardioversion or defibrillation, during myocardial infarction, with electrolyte imbalance, or because of drug therapy.

The output may be reduced to improve battery longevity by providing the minimal level of stimulation necessary for reliable pacing. If

diaphragmatic stimulation is a problem, symptoms may be achieved by a reduction in output. Reducing the output to a sub threshold level allows the underlying rhythms to emerge and allows evaluation of conduction disorders and diagnosis of myocardial infarction. Reducing the output until capture is lost may be used to evaluate the myocardial stimulation threshold.

Rate – The automatic rate is the interval between consecutive atrial and ventricular paced stimuli. This setting is programmable between 30 and 150 pulse/ minute. Rate may be decreased to minimize angina related to the faster paced rate, alternate symptoms of the pacemaker syndrome by permitting the emergence of the underlying sinus rhythm, or reduce the patients' awareness of the paced rhythm. The rate may be increased to provide a faster rate commensurate with increased metabolic needs and suppress Tachycardias.

Sensitivity – Sensitivity is 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 slow rate. The capability to recognize slow rate allows the sensing circuit to differentiate between the QRS and the T wave.

Escape rate and hysteresis – The escape rate is the interval between the last sensed beat and first pacemaker beat to follow. Hysteresis occurs when the escape interval is longer than the programmed interval. Even though the pulse generator may be programmed to pace at 72 pulse/minute, it will not begin pacing until the rate falls below 60 beats/minute, giving the patient a greater opportunity for conducted sinus beats.

Lower and upper rate limits – Lower and upper rate limits are programmable features of the dual chamber units that permit rate responsive pacing. The lower rate limit is the lowest rate at which the pulse generator is programmed to track the spontaneous atrial beat. If the atrial or ventricular rate falls below the programmed lower rate limit, the generator will pace either chamber at the standby rate. The upper rate limit is the fastest rate at which a pulse generator is programmed to track the spontaneous atrial rate on a 1:1 base. The upper rate is set to prevent any excessively rapid ventricular response to rapid supraventricular rhythms.

Refractory period – The refractory period is the period during which the pulse generator is unresponsive to an output signal. The pacing refractory period follows a paced complex, and a sensing refractory period follows a sensed spontaneous complex. The ventricular pacemaker refractory during which signal at the ventricular input is ignored. The atrial pacemaker refractory period is that time during which signals at the atrial input are ignored.

Blanking period – The blanking period is a very short ventricular refractory period that is initiated in the ventricles by an atrial pacemaker pulse for the purpose of preventing the ventricular electrode from sensing the atrial pulse (cross talk) and being inhibited by it. If this interval is too long, it could result in delivery of the ventricular pacing pulse at the same time as an unsensed spontaneous QRS. If too short, cross talk could result in ventricular asystole.

Mode – Several modes of function are programmable in the DDD pacemaker. For example, DDD mode can be changed to VVI, if atrial fibrillation develops.

AV interval – The AV interval should be programmed within the physiologic range (0.12 to 0.2 seconds) for maximal cardiac output. In some patients, AV interval may be programmed to allow AV conduction to occur, with the patients' QRS inhibiting the ventricular output channel. On this way, only one output pulse is used and battery life is preserved. In selected patients, the AV interval may be altered to prevent Tachycardia.

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.

TECHNICAL ASPECTS OF PACEMAKER IMPLANTATION

Determination of the Pacing Threshold

This is crucial to optimize pacemaker longevity and is determined at the time of implantation using a testing device (PSA) with circuitry similar to that of the Implantable pulse generator. Most Implantable pulse generators are constant voltage source; the leading edge of the voltage pulse remains constant regardless of the impedance. The threshold should be determined in volts at a given pulse with of 0.5 to 0.6 m/sec. to measured the threshold, the PSA is set at 5v and pulse with 0.5 m/sec (usually the normal parameters of the Implantable device). The pacing rate is increased until consistent pacing capture is achieved. The voltage is then slowly reduced until loss of capture occurs out side the myocardial refractory period. The lowest voltage at a given pulse with capable of causing consistent capture out side the myocardial refractory period defines the stimulation threshold.

The impedance according to ohms law determines the current delivered to the myocardium. Normal lead impedance, ranging from 250 to 1000 ohms, typically is 500 to 700 ohms at a normal output of 5v. A low output voltage and /or short pulse with enhances battery longevity.

Lead Impedance

Lead impedance normally remains constant or falls slightly over time. Lead fracture elevates both the voltage threshold and the lead impedance (> 1000 ohms) while with insulation defect the voltage threshold may be low or normal and the lead impedance will be low (< 250 ohms). A high voltage threshold due to lead displacement or an excessive tissue reaction around the electrode (exit block) is associated with normal lead impedance.

Changing in the pacing threshold

With conventional leads, this variable rises shortly after implantation because edema and inflammation separate the tip from the myocardium.

Sensing

A pacemaker senses the potential difference between two electrodes used for pacing. A bipolar system, the bipolar electro gram should be recorded to determine adequate electrode position for sensing. The amplitude and slew rate (dv/dt) of the electro gram must exceed the sensitivity of the pulse generator to ensure reliable sensing.

Signal Amplitude

The ventricular signal is often 6 to 15 mv, a range that exceeds the sensitivity threshold of the pulse generator. The ventricular electro gram should measure at least 5 to 6 mv and the atrial electro gram at least 1.5 to 2 mv. A low electrographic signal may require repositioning of the lead. Following initial current injury (ST – evaluation) that reflect good endocardial contact and disappears after few days.

Lead Design

Generally, bipolar leads are preferred because of the greater signal to noise ratio, less sensitivity to extraneous interference (skeletal myopotentials, less frequent cross talk (atrial stimulus sensed by the ventricular lead in a dual chamber system). Adhering leads utilize either passive fixation with tines or fines to enhance entanglement in trabeculae or active fixation with myocardial penetration by grasping screws or small jaws. Active fixation leads are particularly useful in right ventricular

dilatation, in tricuspid insufficiency, and when pacing of the RVOT is needed. Porous electrode with a small surface area yield a low pacing threshold and yet provide a greater surface area for improved sensing.

TEMPORARY PACEMAKER IMPLANTATION

A temporary pacemaker consists of transvenous catheter electrode attached to an external pulse generator. Temporary pacemakers are used, when the need for pacing is immediate.

INDICATIONS

Complete heart block with slow ventricular escape.

Symptomatic sinus bradycardia, asystole, or prolonged sinus pauses.

Acute anterior myocardial infraction with complete heart block.

Acute inferior MI with CHB and poorly tolerated rates, Hypotension, congestive heart failure, or ventricular arrhythmias.

INITIATING PACING

After achieving a satisfactory right ventricular position, connect the pacing catheter using sterile connecting cable to external pulse generator.

Test the pacing threshold by turning on the pulse generator at moderately high out put (5 mA) and at a rate higher than patients intrinsic rate. After satisfactory capture is achieved, gradually reduce the output until capture is lost. This milli ampere level is the patient pacing threshold (i.e., the lowest energy at which consistent capture is achieved). This is usually less than 1

mA. The output should be set at 3 to 4 times this level for consistent capture.

Test the sensing function, if the patient has an underlying rhythm. Do this by placing the system in demand mode and decrease the rate until it is suppressed by that patient's rhythm.

Set the pacing rate – usually 70 to 80 beats/min.

Test the lead for stability by having the patient breath deeply and cough while observing the ECG for continued capture.

AUTOMATIC IMPLANTABLE CARDIOVERTER/ DEFIBRILLATOR

The development of the concept and implantation of the automatic implantable defibrillation (AID) and subsequently the AICD has largely been the work of Dr. Michel Mirowski and his coworkers at Sinai Hospital in Battinmore. Their work began in the late 1960s and met much initial professional skepticism. Most thought that the energy needed for successful defibrillation was more than could be stored in miniaturized device.

However, in early canine work Mirowski showed that the actual energy requirement for direct defibrillation was only in the range of 30 to 50 Joules. He also demonstrated that to restore sinus rhythm it was only necessary to depolarize a critical cell mass, not the entire heart.

AICD GENERATOR

The AICD generator consists of a sensing device, batteries, and energy storage capacitors. It is 11.2 x 7.1 x 2.5 cm, weighs 292 g and is housed in a titanium case. The header is made of epoxy.

The device senses arrhythmias on the basis of two independent algorithms: heart rate and the probability density function (PDF) – both of which must be satisfied for the device to discharge. The rate detection circuit senses an averaged heart rate, which remains above a preset value (usually 155 beats/minute) long enough to satisfy the sensing circuit. However, using rate criteria alone, the device cannot distinguish supraventricular Tachycardia (SVT) from VT

The PDF diagnoses VT or VF based on the amount of time the QRS spends away from isoelectric baseline.

The ECG waveform in sinus rhythm or SVT without aberration spends most of its time on the isoelectric baseline. Wide complex VT or coarse VF are away from the baseline high percentage of sense time and produce a sinusoidal waveform that easily satisfies the morphologic sensing criteria.

The AICD usually requires 5 to 20 seconds to sense VT or VF and then 5 to 15 seconds to charge its energy storage capacitors. It then delivers a 23 to 28 J shock, and if necessary, a second, third and fourth counter shocks of 28 to 37 J each after additional detection and charging periods of 10 to 35 seconds. If the arrhythmia persists after the fourth shock, the unit will not recycle. This feature prevents multiple shocks in misdiagnosed rhythms. High-energy units delivering 28 to 37 J for all four shocks in the sequence are also available.

ELECTRODES

The electrode for ACID comprises three types of leads – a superior vena caval spring lead, a trans venous bipolar electrode, and the ventricular patch lead. The superior vena caval spring lead 100 cm long and has a 10-cm² surface area, its lead wire is colour coded, black. Rate sensing is either by a transvenous lead placed in the right ventricular apex or by a bipolar epicardial screw-in pair of leads on the left ventricle.

The ventricular patch lead is either 10 cm² (small patch) or 20 cm² (large patch) and is colour coded, red. The patch is made of titanium mesh and is covered with a silicone on its shiny side. Its mesh side is placed on the pericardium or myocardium during surgery.

TERMINOLOGY

REDIRECT

Redirect is the feature by which a charge can be directed away from the patient and into the device, if necessary. During EP testing, ventricular rhythms often terminate after the unit has sensed the rhythm but before it discharges. Placing the magnet quickly over the upper portion of the device for only 2 to 3 seconds directs the charge internally rather than to the patient.

A redirect is signaled by the rapid series of beeping tones. The magnet must be removed after the beeps are heard. Otherwise, overhanging would damage the capacitor.

DEFIBRILLATION THRESHOLD

The defibrillation threshold (DFT) is the minimum amount of energy required for successful, reliable termination of an induced rhythm. It is measured at the time of implantation with the use of an external Cardioverter/defibrillator device (ECD).

SENSE TIME

Sense time (detect time) is the time from the onset of the arrhythmia to the initiation of the charging of capacitors. Its duration is 2.5 to 5 seconds for VF and 5 to 20 seconds for VT.

CHARGE TIME

The charge time of the capacitor is under 10 seconds unless the battery is near its end of life. If a charge time greater than 10 seconds is detected, the magnet test should be repeated one or two times because charge times may be prolonged, if the capacitor has not been charged for a long period to time.

INDICATIONS

Survivors of sudden cardiac death not associated with acute MI, whose inducible arrhythmias are not controlled by pharmacologically are candidates for AICD implantation. The one-year mortality without the AICD for this group of patients is 35 % to 40 % despite empiric arrhythmic therapy.

The AICD is also indicated for patients with more than one cardiac arrest whose arrhythmia cannot be induced during EP testing. Patients with sustained VT who have had a clinical recurrence on conventional

antiarrhythmic medications or who have failed while taking a drug known to be ineffective during EP studies benefit from AICD implantation.

Patients with long QT syndrome who have been resuscitated from sudden cardiac arrest also candidates for AICD implantation.

CONTRAINDICATIONS

Patients with frequent non-sustained or sustained VT that cannot be controlled with Antiarrhythmic drugs may not be candidates of frequent triggering of the device.

Patients with uncontrolled congestive heart failure, in which there is legitimate concern that AICD implantation will not produce substantial prolongation of life, are not candidates for AICD implantation.

Patients with extreme physiologic uncertainty about the device are not candidates for AICD implantation.

Centers not having adequately trained personnel and operating room equipment to allow full EP studies and dedication to long term patient follow up should not implant this device.

RADIATION HAZARDS

RADIATION HAZARDS AND PROTECTION

CURRENT RECOMENDATIONS OF ICRP

Radiological protection is concerned with protecting man against deleterious effects of ionizing radiations. The international commission on radiological protection (ICRP) was established in 1928, with the name of the international

X-ray and radium protection committee. Followed by a decision of second international congress of the radiology, to deal with the protection of the man against ionizing radiation. In 1950, it was restricted and re-named as international committee on radiological protection (ICRP) . The present recommendation of 20mSv (2R) per day in 1934 for radiation workers.

UNITS OF RADIATION AND QUANTITIES USED IN RADIATION PROTECTION

Following are the quantities and units used in radiation protection as specified by ICRP and ICRU.

| QUANTITY | SI Unit | SPECIAL UNIT | CONVERSION OF UNIT |
|-----------------|---------------|--------------|-----------------------------|
| Activity | Becquerel(Bq) | Curie(Ca) | 1Ci = 3.71x10 ¹⁰ |
| Exposure | AirKerma(Gy) | Roentgen(R) | 1R=0.0087 |
| Absorbed dose | Gray(Gy) | Rad | 1Rad=1cGy |
| Equivelent Dose | Sievert (Sv) | Rem | 1rem=10mSv |

TYPES OF RADIATION EXPOSURES:

Radiation exposures are categorised as the following:

Occupational exposure-may be defined as radiation exposure occurred at work regardless of source of radiation. Exposure due to natural source such as potassium 40 in the body cosmic rays at ground level and radio nuclides in the workplace and work with materials containing natural radio nuclides.

The exposures from natural sources considered as part of occupational exposures in the following cases. Operation in work places where the regulatory agencies has declared that radon needs attention

Operation with and storage of material not usually regarded as radio nuclides, but which contain significant traces of natural radio nuclides and which have been identified with the regulatory agencies.

Operation of jet air craft

Medical exposure-it is the radiation exposure incurred by individuals as part of their medical diagnosis or treatment and exposures incurred at the their on risk by individuals helping patients undergo diagnosis treatment

Public exposures-it is composed of all exposures other than occupational and medical. The largest component of public exposure due to natural sources.

RECOMMENDED VALUES OF DOSE LIMITS

| APPLICATION | OCCUPATIONAL DOSE LIMIT | PUBLIC DOSE LIMIT |
|---|-------------------------|-------------------|
| Effective Dose Average over a period of five years | 20mSv per year | 1mSv in a year |
| Annual equivalent dose | 150mSv | |
| In the lenses of eye . | 500mSv 500mSv | 15mSv 50mSv |
| In the skin | | |
| In the hand and feet | | |

The limits apply to sum of relevant dose from external exposures in the specified period, and the 50 year committed dose from the intakes in the same period. The above dose limits are suggested with the further provision that the effective dose should not exceed 50mSv in any single year. For members of the public, a higher value of effective dose could be allowed in single year provided that the average value over a period of five years Does not exceed 1 mSv / year.

THE OCCUPATIONAL EXPOSURE FOR WOMEN

The basis of the control of occupational exposure of women who are not pregnant is the same as that for men. Once pregnancy of the women

radiation worker has been confirmed, the concept should be protected by applying a supplementary equivalent dose limit to the surface of the women's abdomen (lower trunk) of 2 mSv for the remainder of pregnancy. And , by limiting the intake's of radionuclides to 1/20 of the annual limits of intakes (ALI).

MEDICAL EXPOSURE OF PREGNANT WOMEN

Diagnostic and therapeutic procedures causing exposures of the abdomen of women likely to be pregnant should be avoided unless there are strong clinical indications. The exposure of the embryo in the first three weeks following consumptions is not likely to result in deterministic or stochastic effects in the live born child. A pregnant patient is likely to know, or at least suspect, that she is pregnant after one missed menstruation, so the necessary information on possible pregnancy and should, be obtained form the patient herself. If the most recent expected menstruation has been missed and there is no other relevant information, the women should be assumed to be pregnant.

CARDIAC DRUGS

CARDIAC DRUGS

There are many different types of cardiac drugs that treat heart disease. A wide variety of cardiac drugs are used to:

- * reduce or prevent the pain of an angina attack
- * lower blood pressure
- * help regulate the heart rhythm
- * help prevent the formation of blood clots
- * help the pumping action of the heart
- * lower blood fats and
- * help prevent future heart attacks

1 .Adenosine (ATP)

Antiarrhythmic, first line drug used for termination of Supraventricular Tachycardia (SVT) involving the AV node or the accessory pathways (WPW).It can also block the AV node transiently to facilitate the interpretation of the surface ECG.

Dose: 10mg-20mg IV push

Side effects: flushing, chest discomfort, and asthmatic attack

Contra: asthma, COAD/COPD, heart blocks

2.Aldactone

A diuretic to treat heart failure, fluid retention due to cirrhosis of liver.

Recent stud (RALES) showed that it is useful for heart failure patients.

Dose: 25mg Om - 50mg tds

3.Alteplase tPA (t-PA)

Thrombolytic, used to lyses the clot inside the coronary vessels in acute myocardial infarction, can also be used for pulmonary embolism

Dose: 10mg bolus then 50,20,20mg q1h. Accelerated: 5mg bolus then 50mg over 30 minutes, 35mg over 1 hour

Side effects: bleeding

Contra: recent surgical operation, recent GI bleeding, cerebral hemorrhage, pregnancy, bleeding tendency, severe hypertension

4.Amlodipine

Calcium Channel Blocker (CCB), 2nd generation. Use for hypertension, ischemic heart disease and angina.

Dose: 5-10mg per day

Side effect: dizziness, headache, peripheral edema, and congestive heart failure

5.Amiodarone

Class III anti-arrhythmic. Used for terminating and preventing supraventricular arrhythmias (SVT) including Atrial fibrillation and ventricular arrhythmias (VT)

Dose: loading 5mg/kg then 15mg/kg/day, tail off

Side effect: pulmonary fibrosis, visual disturbance, thyroid dysfunction, heart failure, rash, GI upset, many drug interactions (digoxin, anti-arrhythmias, warfarin)

6. Aspirin

Analgesic, used also for reducing risk of myocardial infarction and risk of death after infarction or angina. Also used for reducing risk of thromboembolism in high risk patients

Dose: 80-325mg P.O.

Side effect: GI upset, bleeding, ulcer

Contra: bleeding tendency

7. Atenolol

Beta blocker. Used for treatment of hypertension, ischemic heart disease, angina, post myocardial infarction, heart failure

Dose: 50-200mg/day

Side effect: heart failure, heart block, and bradycardia, reduce exercise tolerance, prolong QT

Contra: asthma, COAD/COPD, heart block, severe heart failure, peripheral vascular disease

8. Atropine

Anticholinergic. Treatment of bradycardia and heart blocks

Dose: 0.6-2.0mg (max) IV

Side effect: dry mouth, urinary retention, and confusion

Contra: myocardial ischemia, asthma, ileus, glaucoma,

9.Abciximab (Reopro)

A new Glycoprotein IIb/IIIa receptor antagonist. Use for complicated PTCA/PTCS procedures, also studied for use in unstable angina and acute myocardial infarction.

Dose: bolus + infusion

Side effects: allergic reactions, bleeding tendency

10.Captopril

Angiotensin Converting Enzyme Inhibitor (ACEI). Used for treatment of hypertension, heart failure and post myocardial infarction remodelling

Dose: 25-150mg tds PO

Side effect: impair renal function, cough, headache, anemia

Contra: hyperkalemia, renal artery stenosis, pregnancy, outflow obstruction

11.Carvedilol

Alpha & Beta-blocker with vasodilator activity. Used for treatment of congestive heart failure. Start at low dose and titrate up slowly in 2 weekly time. New studies showed that it reduce mortality in Class II-IV heart failure patients.

Side effect: asthmatic attack, bradycardia, worsening of heart failure

12.Chlorothiazide

diuretic. Used for treatment of hypertension, heart failure

Dose: 0.5-1g PO/IV

Side effects: hypotension, hypokalemia, impaired renal function

13.Clofibrate

Fibric acid derivative. treatment of hyperlipideamia

Dose: 2G PO

Side effect: nausea, diarrhoea, myopathy

14.Clopidrogel

A new anti-platelet (acts on ADP receptor) with similar action to ticlodipine. Use for angina, PTCA/S procedures and strokes. New studies showed that it may be useful for unstable angina and myocardial infarction.

Side effects: bleeding tendency, allergic reaction, GI upset, TTP.

15.Digoxin

Digitalis. Use for the control of ventricular rate in atrial fibrillation, heart failure and PAF

Dose: 0.0625-0.3125mg /day

Side effect: atrial and ventricular arrhythmias, heart blocks, GI upset

Contra: ventricular fibrillation

16. Dipyridamole

Antiplatelet. Use for prevention of thromboembolic disease, cardiac valvular replacement, stenting.

Dose: 75-100mg BD PO

Side effect: hypotension

17. Dobutamine

inotropic agent. Use for blood pressure support, hypotension.

Dose: 2-40ug/kg/min iv

Side effect: increase heart rate, myocardial ischemia, increase risk of arrhythmia

Contra: hypertrophic heart disease, severe aortic stenosis

18. Dopamine

inotropic agent. Use for blood pressure support, hypotension, renal vascular perfusion (low dose)

Dose: 2-50ug/kg/min iv

Side effect: increase heart rate, myocardial ischemia, increase risk of arrhythmia

Contra: pheochromocytoma, tachyarrhythmia

19.Enalapril

ACEI. see Captopril

Dose: 2.5-40mg/day

Side effect & Contra: see captopril

20.Epinephrine

Vasopressor. treatment of hypotension and shock, ventricular fibrillation, asystole, cardiac arrest, bradycardia, anaphylactic shock

Dose: 1-20ug/min

Side effect: sweating, headache, increase heart rate, peripheral vasoconstriction with necrosis of tissue

21.Furosemide

Loop diuretics. Treatment of hypertension and heart failure

Dose: 20-600mg/day

Side effect: GI upset, sensitivity, hypotension, hypokalemia

22.Heparin

Anticoagulant. Treatment of deep vein thrombosis, pulmonary embolism, acute myocardial infarction, unstable angina, peripheral vessel embolism.

Dose: 5,000U iv bolus and 500-2,000U/hour. Adjusted by APTT time.

5,000-20,000U S.C. for prophylaxis

Side effect: bleeding, low platelets, osteoporosis

Contra: severe thrombocytopenia, bleeding tendency

23 (b)Heparin (low molecular weight)

Anticoagulant. Prophylaxis of deep vein thrombosis, pulmonary embolism. Also used after PTCA/S.

Dose: 0.2-1.0 ml S.C. every 12 hours

Side effect: same as heparin

24.Hydralazine

direct vasodilator. Treatment of malignant hypertension, heart failure, pre-eclampsia, eclampsia

Dose: 10-400mg/day PO, iv 10mg bolus with 200-600mg/day maintainance

Side effect: edema, hypotension

Contra: myocardial ischemia, severe mitral valve disease

25.Isosorbide dinitrate

Nitrate. treatment of angina and ischemic heart disease

Dose: 5-20mg PO

Side effect: headache, hypotension, tolerance to drug, flushing

Contra: hypertrophic cardiomyopathy, severe anaemia^s

* Drug expires if container opened for more than 3 months

26 ADRENALINE

Indication : drug of choice in anaphylactic shock

Action : It acts on alpha and adrenergic receptors sites. It inhibits the inhibition of the intestinal muscle contraction (involuntary muscle) and enhances contraction of voluntary muscles.

Dosages : 0.1 – 0.2 mg through intravenous route.

27 SODIUM BICARBONATE

Indication : Metabolic acidosis.

Action : Decrease concentration of hydrogen ion and increases PH.

Dosages : By intravenous route. Dosages = Body weight in Kg x deficit of HCO_3^- x 0.3

28. CALCIUM SULPHATE

Indication : Given when there is Hypocalcemia and cardiac emergencies.

Action : Stimulates myocardial excitability and increases contractility.

Dosages: By slowly through intravenous route 0.5 to 1ml of 10% solution with cardiac monitoring.

29. MORPHINE SULPHATE

Indication : To relieve pain and preanaesthetic drug used in the postoperative period for sedation

30. DIAZEPAM

Indication : Pre-operative medication to reduce anxiety and tension.

Action : It acts on the thalamus and hypothalamus to induce calming effect.

Dosage : 0.3 mg/kg body weight/day through oral route.

31. NITROGLYCERIN

Indication : Hypertensive crisis.

Action : Peripheral vasodilator , acts on arterial venous smooth muscle.

Dosages : 50 mg is dissolved in 250 ml of 5% dextrose; given intravenously at the rate of 0.5-5 micrograms/kg body weight/min.

PROCEDURES ASSISTED

EXPERIENCE IN ECG LAB

| NUMBER | PATIENT | HOSP NO |
|--------|--------------|---------|
| 1 | DINEESH | 198407 |
| 2 | SAROJINI | 204265 |
| 3 | LATHA | 204253 |
| 4 | ABRHAM | 204286 |
| 5 | BIJU | 9501854 |
| 6 | BEERAN SAHIB | 9309 |
| 7 | GEORGE | 204292 |
| 8 | JEGADAMMA | 203479 |
| 9 | CHANDRA | 8802456 |
| 10 | MOHANAN | 204308 |
| 11 | LEELA | 9102327 |
| 12 | RAJEEV | 1247 |
| 13 | JINCY | 204326 |
| 14 | SIVA | 204274 |
| 15 | SEETHA | 204302 |
| 16 | DIVYA | 9010096 |
| 17 | RENJINI | 8804754 |
| 18 | SAPNA | 9604563 |
| 19 | SUBHA | 204301 |
| 20 | FARSANA | 204319 |
| 21 | HAMSA | 201390 |
| 22 | PHILIP | 204351 |
| 23 | SABITHA | 9806188 |
| 24 | SAVITHRY | 204370 |
| 25 | SOPHYA | 196856 |
| 26 | KAREEM | 204394 |
| 27 | JOHN | 204400 |
| 28 | SUNIL | 9402372 |
| 29 | ABIN | 9501535 |
| 30 | AKHIL | 9001317 |
| 31 | GEETHU | 9701182 |
| 32 | AMALA | 9807251 |
| 33 | SUMA | 9300520 |
| 34 | ARUN | 196722 |
| 35 | BISMY | 9708574 |
| 36 | NIHAR | 204452 |
| 37 | ANOOP | 204457 |
| 38 | SAJITHA | 181547 |
| 39 | NEETHU | 204463 |
| 40 | JOY | 204407 |
| 41 | TINU | 9400170 |
| 42 | NITYA | 204487 |
| 43 | GIRIJA | 6424 |
| 44 | MANEESH | 8904878 |
| 45 | ANU | 9006051 |

| | | |
|----|----------------|---------|
| 46 | LATHIKA | 9002513 |
| 47 | LEELA | 204291 |
| 48 | RAVI | 204574 |
| 49 | MRUGAN | 204241 |
| 50 | PRIYA | 199678 |
| 51 | OMANA | 9309589 |
| 52 | MAJEED | 6957 |
| 53 | MINI | 9204098 |
| 54 | VENU | 207697 |
| 55 | SARALA | 207134 |
| 56 | FEBIN | 207704 |
| 57 | MARY | 9704244 |
| 58 | RADHIKA | 207707 |
| 59 | GOKUL | 207701 |
| 60 | NAVEEN | 207610 |
| 61 | RESHMA | 9707671 |
| 62 | SONIA | 207721 |
| 63 | AJEENA | 9107533 |
| 64 | RASIA | 207728 |
| 65 | PHILIP | 21565 |
| 66 | PAREED | 9502736 |
| 67 | DEVI | 207774 |
| 68 | KRISHNA PILLAI | 207676 |
| 69 | ABDUL KAREEM | 203482 |
| 70 | SUDHA | 8905897 |
| 71 | ELSY | 9902606 |
| 72 | AMBIKA | 207764 |
| 73 | BINU | 8805531 |
| 74 | MARIA | 9109319 |
| 75 | SUMATHI | 8872 |
| 76 | MOHANAN | 9804814 |
| 77 | VIJAYAMMA | 207839 |
| 78 | SHEEJA | 207829 |
| 79 | MADHAVAN | 207843 |
| 80 | MANJU | 9405697 |
| 81 | JOSEPH | 207837 |
| 82 | GOPALAKRISHNAN | 207811 |
| 83 | ABHILASH | 6688 |
| 84 | SAHADEVAN | 205700 |
| 85 | JYOTHI | 192615 |
| 86 | SREEDEVI | 200483 |
| 87 | RAJU | 207804 |
| 88 | VIJAYAN | 9300367 |
| 89 | RUKIA | 8800560 |
| 90 | ANU | 9606051 |
| 91 | SAHEER | 207868 |
| 92 | VASUDEVAN | 198762 |
| 93 | SUDHARMAN | 198932 |
| 94 | RAVEENDRAN | 207881 |
| 95 | THOMAS | 205181 |

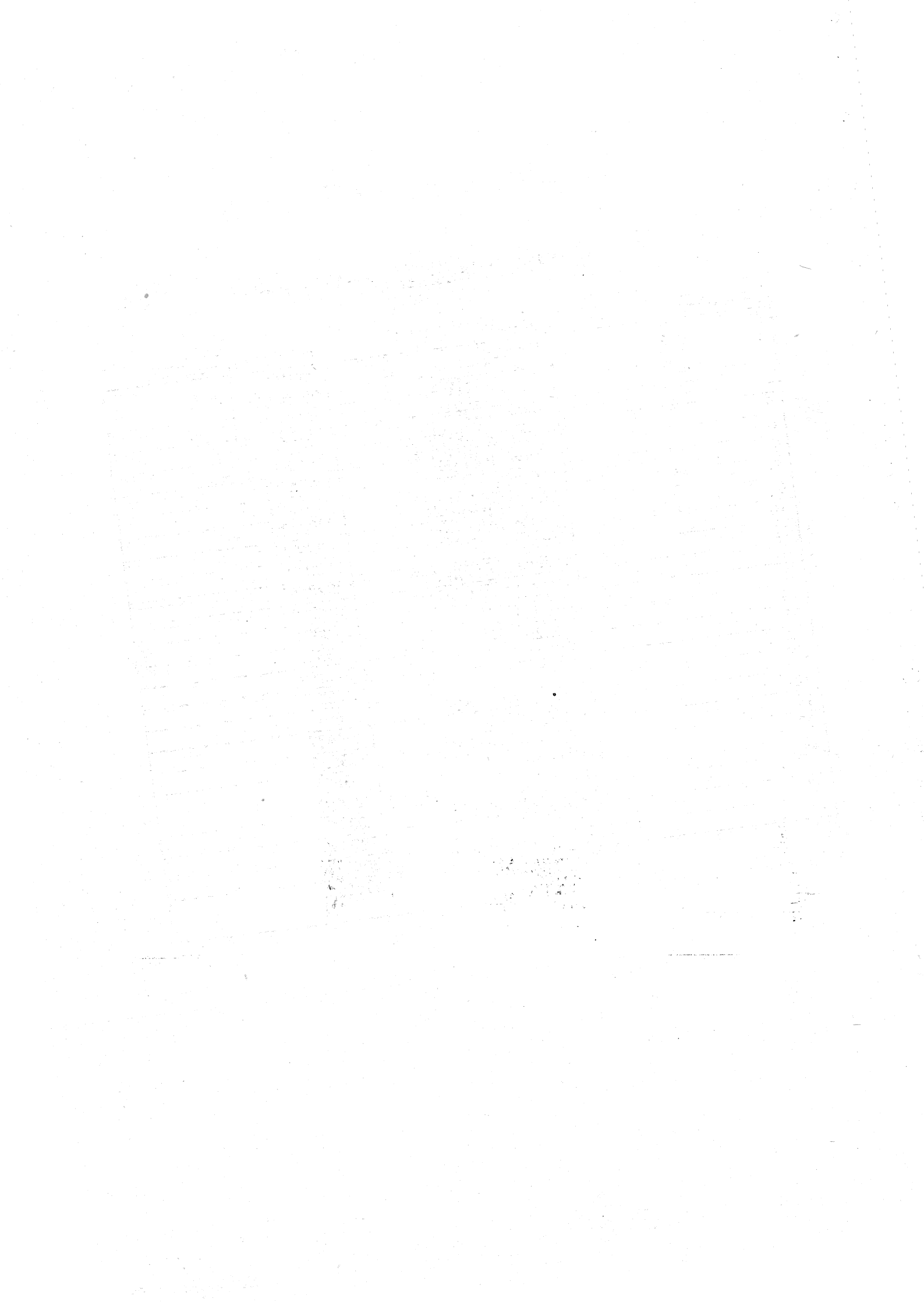
1870

| Year | 1870 | 1871 | 1872 | 1873 | 1874 | 1875 | 1876 | 1877 | 1878 | 1879 | 1880 |
|------------------------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|-----------|
| Population | 1,000,000 | 1,050,000 | 1,100,000 | 1,150,000 | 1,200,000 | 1,250,000 | 1,300,000 | 1,350,000 | 1,400,000 | 1,450,000 | 1,500,000 |
| Area (sq. miles) | 100,000 | 100,000 | 100,000 | 100,000 | 100,000 | 100,000 | 100,000 | 100,000 | 100,000 | 100,000 | 100,000 |
| Density (per sq. mile) | 10 | 10.5 | 11 | 11.5 | 12 | 12.5 | 13 | 13.5 | 14 | 14.5 | 15 |

| | | |
|-----|--------|---------|
| 96 | MANU | 207891 |
| 97 | SURESH | 13247 |
| 98 | JOHN | 8709067 |
| 99 | DAISY | 203211 |
| 100 | SUMA | 207912 |

EXPERIENCE IN HOLTER LAB

| NAME | NAME | HOSPITAL NUMBER |
|------|------------|-----------------|
| 1 | REETHAMMA | 4670 |
| 2 | SAVITHRY | 212369 |
| 3 | PARUKUTTY | 211846 |
| 4 | BASHEER | 187590 |
| 5 | SUKUMARAN | 211879 |
| 6 | SATHY AMMA | 211993 |
| 7 | VIMALA | 211992 |
| 8 | SINI | 212569 |
| 9 | VARGHESE | 950988 |
| 10 | RADHA | 198397 |
| 11 | AYYAPPAN | 9908112 |
| 12 | AJI THAMPI | 207654 |
| 13 | ANI | 9006912 |
| 14 | SINDHU | 212731 |
| 15 | RADHA | 9908198 |

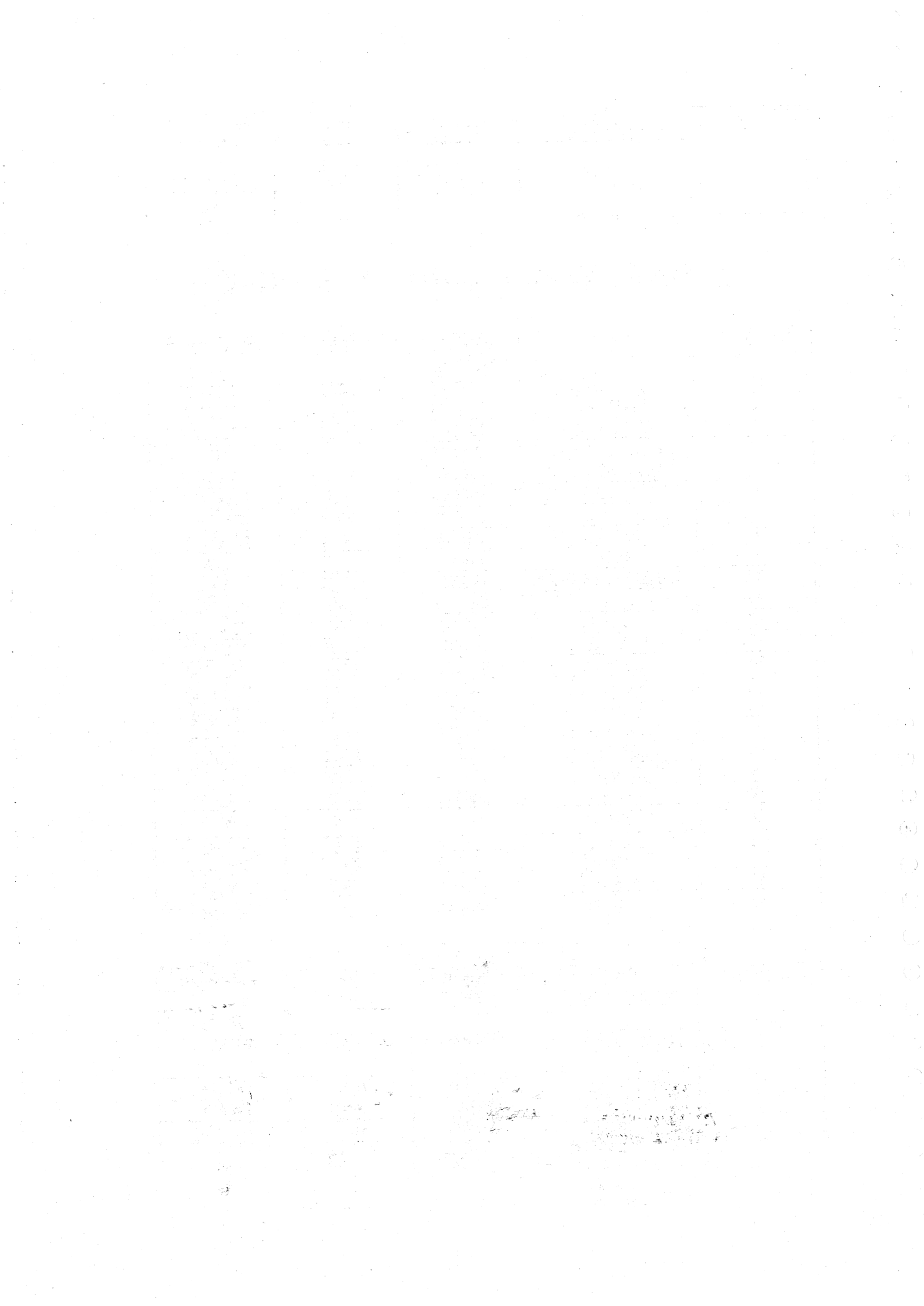


EXPERIENCE IN TMT LAB

| NUMBER | NAME | HOSPITAL NUMBER |
|--------|-----------------|-----------------|
| 1 | RAJAN | 9001592 |
| 2 | ABDUL SAMAD | 9900635 |
| 3 | DEVARAJAN | 202037 |
| 4 | SATHIYAN | 201856 |
| 5 | KRISHNANKUTTY | 9702919 |
| 6 | VISWARAJAN | 9900972 |
| 7 | MERCY | 9607354 |
| 8 | MOHANKUMAR | 9908289 |
| 9 | THYAGARAJAN | 9308424 |
| 10 | VENU | 203214 |
| 11 | ABDULREHUMAN | 202855 |
| 12 | OMANAKUTTAN | 203545 |
| 13 | KRISHNAN | 9709914 |
| 14 | MOHANAN | 203326 |
| 15 | VENU | 203888 |
| 16 | IYER | 188323 |
| 17 | SUSEELAN NAIR | 186918 |
| 18 | TGNATIOUS | 203487 |
| 19 | APPUKUTTAN NAIR | 203830 |
| 20 | AJITHA | 203835 |
| 21 | RUKIYA | 203652 |
| 22 | VISALA MENON | 203904 |
| 23 | SASIKUMAR | 197296 |
| 24 | SUKUMARAN NAIR | 204111 |
| 25 | CHINNAMMA | 204186 |

EXPERIENCE IN ECHOCARDIOGRAPHY

| NUMBER | NAME | HOS: NO | AGE/SEX | DIAGNOSIS |
|--------|---------------|---------|---------|-----------|
| 1 | MANU | 9306978 | 13/M | VSD, PS |
| 2 | RATHY DEVI | 372 | 45/F | MS |
| 3 | RAMESH | 201208 | 1/M | ASD |
| 4 | SAJITHA | 201195 | 3/F | TOF |
| 5 | SUNDARESWAREY | 201185 | 11/F | AR, MS |
| 6 | ABHILASH | 9106990 | 10/M | PDA |
| 7 | LILLY | 201213 | 21/F | ASD |
| 8 | SAHALA | 201250 | 1/F | VSD |
| 9 | REJANI | 201255 | 29/F | MS |
| 10 | AYYAPAN | 9701556 | 48/M | AS |
| 11 | MILTUS | 9408147 | 33/M | MS |
| 12 | ANANTHU | 191452 | 6/M | ASD |
| 13 | THANKAMMA | 201252 | 49/F | AS |
| 14 | SREENATHAN | 201291 | 1/M | PDA |
| 15 | ALPHIN | 201299 | 1/M | PDA |
| 16 | MOHD: FAISAL | 9902169 | 5/M | TOF |
| 17 | SELVARAJ | 201325 | 22/M | AR |
| 18 | SASI | 9508221 | 41/M | MR |
| 19 | VIJAYAKUMARI | 16489 | 34/F | MS |
| 20 | ANAND | 9607851 | 5/M | ASD |
| 21 | RAKESH | 35756 | 13/M | VSD, PAH |
| 22 | KUNJALA | 201110 | 63/F | PFO |
| 23 | SUNIL KUMAR | 201315 | 30/M | AS, AR |
| 24 | SADANANDAN | 9909539 | 66/M | RWMA |
| 25 | HAJIRA | 9603984 | 12/F | PO ICR |
| 26 | SOBHANA | 751 | 36/F | ASD PS |
| 27 | ABDULLA | 201417 | 32/M | PAH PR |
| 28 | RILWANA | 9802193 | 3/F | TOF |
| 29 | KHALID | 9300831 | 42/M | MR |
| 30 | SUDHA | 201435 | 54/F | RWMA |
| 31 | SARA | 8908054 | 45/F | MS |
| 32 | ARJUN | 9800267 | 4/M | TOF |
| 33 | NOBERT | 201467 | 42/M | PAH |
| 34 | SHIHAB | 9500633 | 52/M | SAM |
| 35 | PRABHAKARAN | 201459 | 60/M | RWMA |
| 36 | NUSRAT | 9006945 | 10/F | PDA PAH |
| 37 | VISHNUPRIYA | 180820 | 1/F | VSD PAH |
| 38 | SULOCHANA | 9900677 | 29/F | RHD MS |
| 39 | AKSHAYA | 9902086 | 3/F | SV PS |
| 40 | HABEEB | 970675 | 5/M | RHD AR MR |
| 41 | SATHU | 9905433 | 19/M | VSD, DORV |
| 42 | B/O SINDHU | 201545 | 0/M | DTGA |
| 43 | SUFAIJA | 9507723 | 9/M | DORV |
| 44 | SINDHU | 201741 | 25/F | TGA VSD |
| 45 | GOMATHY | 8906886 | 40/F | POST CMV |
| 46 | ARCHANA | 9905435 | 3/F | POST BDG |



| | | | | |
|----|-----------|---------|------|------------------|
| 47 | ANTO | 9107676 | 28/M | POST COA |
| 48 | SANTHAMMA | 200080 | 38/F | BVEMF |
| 49 | JOSE | 9707798 | 4/M | DTGA |
| 50 | SAHAYA | 199164 | 9/F | DORV, VSD, PS |

TRANSESOPHAGEAL ECHOCARDIOGRAPHY

| NUMBER | NAME | HOSPITAL NUMBER | AGE/SEX | DIAGNOSIS |
|--------|---------------|-----------------|---------|------------|
| 1 | MAHESWARI | 199015 | 25/F | RHD MS |
| 2 | FATHIMA | 199188 | 45/F | RHD MS |
| 3 | PRASANNA | 196944 | 20/M | RHD MS |
| 4 | BERA SAMAJAN | 199585 | 41/M | ASD |
| 5 | VIJAYAKUMARI | 196593 | 35/M | LA CLOT |
| 6 | VIJAYAKUMARI | 198660 | 50/F | RHD MS |
| 7 | ANIL | 199705 | 20/M | R/O PFO |
| 8 | VASUDEVAN | 31604 | 47/M | RHD MS |
| 9 | REMA | 8906055 | 64/F | ASD |
| 10 | RADHAKRISHNAN | 199276 | 49/M | ASD |
| 11 | LEELAVATHY | 9804312 | 54/F | RHD MS |
| 12 | KALA | 199812 | 37/F | RHD MS |
| 13 | SAINABA | 198487 | 35/F | RHD MS |
| 14 | DURGA DEVI | 950930 | 16/F | SA ASD |
| 15 | SOPHIYA | 200141 | 24/F | RHD MS |
| 16 | SABITHA | 199886 | 23/F | R/O ASD |
| 17 | SAROJARAJ | 199886 | 37/M | RHD MS |
| 18 | SANTHOSH | 8800707 | 17/M | ASD |
| 19 | SERMAKANI | 200453 | 35/M | R/O PFO |
| 20 | INDIRA | 200517 | 28/F | ASD |
| 21 | MALATHI | 91004 | 30/F | ASD |
| 22 | AYYAPPANKUTTY | 180054 | 35/M | P.O DVR |
| 23 | SIRUMANI | 199411 | 45/M | RHD MS |
| 24 | REENA | 34712 | 24/F | VSD |
| 25 | MEERA | 950918 | 47/F | VEGETATION |

DOBUTAMINE

| NUMBER | NAME | HOS.NUMBER | AGE/SEX | DIAGNOSIS |
|--------|---------------------|------------|---------|----------------|
| 1 | MOHAN DAS | 188064 | 69/M | LV FUNCTION |
| 2 | KUTTAPPAN PILLAI | 180219 | 65/M | LVF |
| 3 | JAMEELA | 216902 | 42/F | CAD |
| 4 | KUTTIAMMA | 216293 | 53/F | CAD |
| 5 | PRABHAKARAN | 213028 | 57/M | LVF |

EXPERIENCE IN CATH LAB

[The page contains extremely faint and illegible text, likely due to low contrast or poor scan quality. The text is arranged in approximately 25 horizontal lines across the page.]

CORONARY ANGIOGRAM

| NUMBER | NAME | AGE/SEX | HOS: NO |
|--------|-----------------|---------|---------|
| 1 | CHANDRASEKHARAN | 46/M | 982350 |
| 2 | KOMALAM | 39/F | 206038 |
| 3 | KUMARI | 63/F | 206277 |
| 4 | VISWAMBARAN | 52/M | 206291 |
| 5 | ZEERA | 54/F | 206026 |
| 6 | KUTTYKRISHNAN | 53/M | 199964 |
| 7 | KHADER | 40/M | 206227 |
| 8 | DIVAKARAN | 58/M | 202378 |
| 9 | VIJAYARAGHAVAN | 60/M | 206371 |
| 10 | UMMER | 56/M | 20582 |
| 11 | JOHN | 64/M | 204400 |
| 12 | SALEEM | 42/M | 206308 |
| 13 | INDIRA | 50/F | 206076 |
| 14 | VELLAPPAN | 49/M | 206369 |
| 15 | PADMINI | 58/F | 9400885 |
| 16 | RAJESEKHARAN | 46/M | 198832 |
| 17 | SREEKUMAR | 44/M | 206379 |
| 18 | AMINA | 45/F | 206435 |
| 19 | HARIHARAN | 64/M | 206414 |
| 20 | KARIM | 50/M | 206208 |
| 21 | GEORGE | 69/M | 206312 |
| 22 | PANICKER R T | 61/M | 206205 |
| 23 | SUDHA | 40/F | 8905897 |
| 24 | SAHADEVAN | 62/M | 205700 |
| 25 | ASEESA | 55/F | 206539 |
| 26 | SIVASANKARAN | 62/M | 206512 |
| 27 | BEERAN | 48/M | 203523 |
| 28 | SUBAIDA | 55/F | 206494 |
| 29 | JOHN | 65/M | 206559 |
| 30 | KRISHNAPILLAI | 80/M | 206567 |
| 31 | JAMES | 58/M | 206373 |
| 32 | JANAKI | 64/M | 206579 |
| 33 | RAJAPPAN | 59/M | 9007015 |
| 34 | REMLA | 54/F | 206665 |
| 35 | YUSUF | 60/M | 206654 |
| 36 | GEORGE | 59/M | 206802 |
| 37 | RADHAMMA | 51/F | 191622 |
| 38 | KOMALAVALLY | 61/F | 206842 |
| 39 | VELU | 50/M | 8800223 |
| 40 | NIZAM | 38/M | 203179 |
| 41 | SANTHAMMA | 48/F | 205815 |
| 42 | SREEKUMAR | 40/M | 206717 |
| 43 | SOBHANA | 30/F | 5857 |
| 44 | VIJAYAN | 48/M | 201859 |
| 45 | LEELAMMA | 60/F | 207828 |
| 46 | JOSEPH | 54/M | 206763 |
| 47 | RAMACHANDRAN | 48/M | 205482 |
| 48 | RUBY | 62/F | 206450 |
| 49 | NABEESA | 60/F | 207106 |
| 50 | DLIT ID | 50/M | 5526 |

1957

| DATE | DESCRIPTION | AMOUNT | BALANCE |
|-------|-------------|--------|---------|
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PTCA

| NUMBER | NAME | AGE/SEX | HOS: NO |
|--------|--------------|---------|---------|
| 1 | NARAYANAN | 53/M | 9500803 |
| 2 | GOPINATHAN | 65/M | 188016 |
| 3 | RAJAN | 54/M | 205368 |
| 4 | BEERAN | 65/M | 206350 |
| 5 | GEORGE | 63/M | 205472 |
| 6 | PREMCHANDRAN | 57/M | 206470 |
| 7 | RAMACHANDRAN | 68/M | 206445 |
| 8 | MAMMEN | 55/M | 206693 |
| 9 | EDWARD | 45/M | 206692 |
| 10 | SUSEELA | 66/F | 9809856 |
| 11 | NIZAMUDEEN | 48/M | 206841 |
| 12 | CHELLATHAMBI | 50/M | 206437 |
| 13 | IBRAHIM | 41/M | 205731 |
| 14 | ARUNKUMAR | 24/M | 207011 |
| 15 | PHILIPSE | 71/M | 206245 |
| 16 | AYSHA | 57/F | 205943 |
| 17 | BALACHANDRAN | 62/M | 206279 |
| 18 | RAJU | 58/M | 207270 |
| 19 | ABRAHAM | 60/M | 207129 |
| 20 | JAMES | 58/M | 188780 |
| 21 | THOM,AS | 64/M | 206808 |
| 22 | RAMAKRISHNA | 52/M | 206577 |
| 23 | THILAKAM | 62/F | 206492 |
| 24 | G R NAIR | 55/M | 2041431 |
| 25 | VENUGOPALAN | 58/M | 207503 |

PTMC

| NUMBER | NAME | HOS.NO | AGE/SEX |
|--------|--------------|---------|---------|
| 1 | SAFEERA K | 9003504 | 24/F |
| 2 | SARITHA | 205827 | 16/F |
| 3 | LAKSHMY | 206688 | 40/M |
| 4 | SOBHANA | 5857 | 30/F |
| 5 | RADHIKA | 202664 | 21/F |
| 6 | JAISY | 200135 | 25/F |
| 7 | MARY JOSEPH | 206106 | 10/F |
| 8 | ESAKIAMMAL | 206247 | 40/F |
| 9 | ANTONY | 206422 | 36/M |
| 10 | GEETHANGALY | 206899 | 54/F |
| 11 | PRASANNA | 195563 | 41/F |
| 12 | ANITHA | 206848 | 21/F |
| 13 | ALAKAMMAL | 207321 | 27/F |
| 14 | BINU | 207166 | 28/M |
| 15 | KUNJAMMA K | 9003684 | 41/F |
| 16 | GANESAN | 207298 | 35/M |
| 17 | PREMALATHA | 200255 | 26/F |
| 18 | RADHIKA | 200063 | 15/F |
| 19 | SIVARAMAN | 207367 | 29/M |
| 20 | OMANAMMA | 205712 | 50/F |
| 21 | KADEEJA | 207394 | 37/F |
| 22 | PASUMATHY | 207910 | 38/F |
| 23 | AMALAPUSHPAM | 207246 | 48/F |
| 24 | SEENATH | 208238 | 30/F |
| 25 | JANAKI | 207037 | 40/F |

CATH

| NUMBER | NAME | HOS. NO | AGE/SEX | DIAGNOSIS |
|--------|--------------|---------|---------|------------------------|
| 1 | B/O HASEENA | 205742 | 1/M | PDA, PAH |
| 2 | BENJO | 185938 | 5/M | SV |
| 3 | VINEETH | 8908005 | 12/M | SV, TA, PA |
| 4 | SURENDRAN | 9001738 | 38/M | VSD, PAH |
| 5 | B/O ESTHER | 206393 | 2M/F | TAPVC |
| 6 | BRINDA S | 205825 | 22/F | ASD |
| 7 | VALSAMMA | 206643 | 47/F | ASD, PAH |
| 8 | MINU | 9404634 | 8/F | VSD, TA |
| 9 | SWAMINATHAN | 206806 | 14/M | VSD, PA |
| 10 | SAJU | 204477 | 26/M | ASD, PAH |
| 11 | SALY | 206483 | 12/M | COA |
| 12 | ASHRAF | 206065 | 26/M | COA |
| 13 | PHILIP | 5626 | 59/M | CAD, MVP, MR |
| 14 | ARUNAGIRY | 207069 | 46/M | CAD, AS, MS |
| 15 | NIDHEESH | 9500665 | 6/M | DORV, VSD, PA |
| 16 | NISHAD | 207002 | 14/M | VSD, PDA |
| 17 | THANKSAPANDY | 206697 | 6/M | DORV, VSD |
| 18 | NOUSHIJA | 9909405 | 3/F | DTGA, VSD, PS |
| 19 | ABHILASH | 9106990 | 12/M | VSD |
| 20 | GRACE | 203000 | 4/F | TOF |
| 21 | AKASH | 204900 | 1/M | TOF |
| 22 | CHANDRAN | 207444 | 40/M | HOCM |
| 23 | INDU | 207446 | 30/F | RSOV |
| 24 | SANGILIPANDI | 204287 | 6/M | DORV |
| 25 | SAROJINIAMMA | 8800043 | 65/F | BVEMF |
| 26 | SHAMOL | 207865 | 7M/F | TAPVCASD |
| 27 | ASHIK | 181937 | 2/M | PA, PDA, MAPCA |
| 28 | ROBIN | 9806959 | 4/M | ASD, COMM. AV VALVE |
| 29 | RAHUL | 195304 | 2/M | DORV, VSD, PDA |
| 30 | B/O SREEKALA | 208211 | 8M/F | ALKAPA |

PDA COILING

| NUMBER | NAME | HOS.NO | AGE/SEX |
|--------|-------------|--------|---------|
| 1 | THOMAS BABY | 196420 | 9/M |
| 2 | NEENU.S | 206322 | 5/F |
| 3 | ARYA | 203520 | 2/F |
| 4 | ANITHA.J | 198514 | 4/F |
| 5 | RAJESWARI | 207039 | 18/F |
| 6 | ROJA | 200926 | 26/F |
| 7 | ABHHIRAM | 204969 | 1/M |
| 8 | ALTAZ | 192548 | 2/M |
| 9 | LEKSHMI | 204631 | 28/F |
| 10 | ANASWARA | 205515 | 5/F |
| 11 | SILPA | 205278 | 1/F |
| 12 | VICTORIA | 202358 | 3/F |
| 13 | HAREESHNATH | 206006 | 2/M |
| 14 | GIFTY | 208731 | 5/F |
| 15 | SREEKUTTY | 183940 | 7/F |
| 16 | JAMSHEED | 205484 | 15/M |
| 17 | RIYAS | 191296 | 4/F |
| 18 | ASWATHY | 206141 | 2/F |
| 19 | JALIAN | 206436 | 4/M |
| 20 | LIJU | 207982 | 4/M |

ASD DEVICE CLOSURE

| NUMBER | NAME | HOS NO. | AGE/SEX |
|--------|------------|---------|---------|
| 1 | BRINDA | 205825 | 22/F |
| 2 | SAVITTHRI | 201613 | 55/F |
| 3 | HEMA HENRY | 204244 | 12/F |
| 4 | SUSHAMA | 206082 | 33/F |
| 5 | ARAVIND | 207616 | 5/M |
| 6 | GOKUL | 198237 | 6/M |
| 7 | FARSANA | 187822 | 6/F |
| 8 | REMYA.R | 213573 | 10/F |
| 9 | ABHISHEK | 210865 | 10/M |
| 10 | SUJITH | 198956 | 13/M |

BAS,BPV,BAV

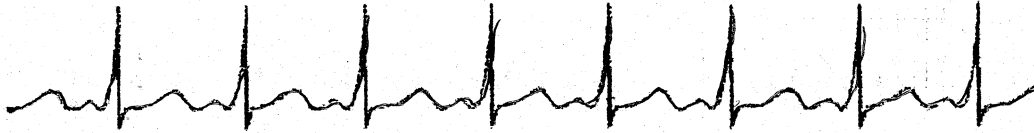
| NUMBER | NAME | HOS.NO. | AGE/SEX | PROCEDURE |
|--------|------------------|---------|---------|-----------|
| 1 | THANSI | 206677 | 2M/F | BAS |
| 2 | B/O SELVAKANI | 207123 | 11M/M | BAS |
| 3 | B/O SUMINA | 207102 | 1M/F | BAS |
| 4 | NADIYA | 207194 | 1M/F | BAS |
| 5 | B/OAJITHA | 207552 | 1M/M | BAS |
| 6 | NISHRA | 208342 | 20/F | BPV |
| 7 | GOPU.G | 9908164 | 14/M | BPV |
| 8 | UNNIKUTTAN | 204317 | 6/M | BPV |
| 9 | RARICHAN | 8706818 | 19/M | BPV |
| 10 | AYAPPAN | 9205279 | 37/M | BPV |
| 11 | MANJU.M.T | 209053 | 15/F | BAV |
| 12 | KUMAR | 9603622 | 16/M | BAV |
| 13 | SELVAN | 211495 | 18/M | BAV |
| 14 | MUSTHAFA | 215007 | 18/M | BAV |
| 15 | MAHALAKSHMI | 217219 | 17/F | BAV |

PACEMAKER IMPLANTATION

| NUMBER | NAME | HOS.NO | AGE/SEX | MODE |
|--------|--------------|---------|---------|------|
| 1 | KUHILEKSHMI | 4607 | 78/F | AAI |
| 2 | DEVU.K.K | 9102024 | 69/F | AAI |
| 3 | KAMALAKSHY | 213476 | 80/F | AAI |
| 4 | SULOCHANA | 208807 | 37/F | VVI |
| 5 | EMMIAMMAL | 213899 | 70/F | VVI |
| 6 | RASEENABEEVI | 214029 | 65/F | VVI |
| 7 | INDIRA | 216512 | 70/F | DDD |
| 8 | EASOMATHEW | 216552 | 58/M | DDD |
| 9 | KUNJURAMAN | 2047 | 40/M | DDD |
| 10 | IBRAHIM | 219144 | 79/M | VVI |
| 11 | DOLLY | 217372 | 55/F | AAI |
| 12 | BABY | 210734 | 45/F | VVI |
| 13 | IBRAHIM | 219144 | 79/M | VVI |
| 14 | PADMANABHAN | 219350 | 80/M | DDD |
| 15 | LATHA | 2848 | 38/F | AAI |

ELECTROPHYSIOLOGY + RFA

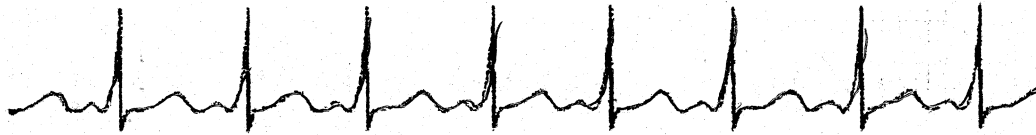
| NUMBER | NAME | HOS.NO | AGE/SEX | DIAGNOSIS |
|--------|----------------|---------|---------|-----------|
| 1 | RAMACHANDRAN | 1526 | 63/M | AVNRT |
| 2 | AMINA | 213355 | 60/F | AVNRT |
| 3 | CHERIYAPUSHPAM | 214012 | 51/F | SVT |
| 4 | GOPINATHAN K | 216822 | 54/M | AVNRT |
| 5 | ABDULRAZACK | 217102 | 24/M | PSVT |
| 6 | SUJATHA | 217166 | 55/F | AF |
| 7 | SAM PHILIP | 217141 | 30/M | VT |
| 8 | AISHA K | 217831 | 70/F | AVNRT |
| 9 | ANILKUMAR | 217743 | 26/M | WPW |
| 10 | SASIKUMAR | 9908513 | 51/M | VT |



The accessory pathway can cause a reentry circuit to be established. Reentry is initiated by a premature atrial or ventricular beat coupled with a unidirectional block in one of the pathways (because the normal impulse gets to pathway when it is refractory after the premature beat). The result is continuous impulse conduction. Reentry causes two kinds of tachycardia.

1. Orthodromic AV reentrant tachycardia, which occurs when the impulse is conducted through the AV node with retrograde return to the atria via the Bundle of Kent. The heart rate is usually 140-250 BPM. The QRS complexes are narrow and delta waves are not observed.
2. Antidromic AV reentrant tachycardia, which occurs when the impulse is conducted through the Bundle of Kent with retrograde return to the atria via the AV node. The QRS complexes are wide.

Wolff-Parkinson-White syndrome is commonly associated with congenital heart abnormalities like Tetralogy of Fallot, coarctation of the aorta, tricuspid atresia and transposition of the great vessels. In severe cases, treatment would involve surgical removal or ablation of one of the pathways.



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Wolff-Parkinson-White syndrome is commonly associated with congenital heart abnormalities like Tetralogy of Fallot, coarctation of the aorta, tricuspid atresia and transposition of the great vessels. In severe cases, treatment would involve surgical removal or ablation of one of the pathways.

CONVENTIONAL PLACEMENT OF ECG CHEST LEADS

The chest leads (V1-V2) show the electrical current of the heart as detected by electrodes placed at different position on the chest wall. The precordial leads at different positions leads used today are also unipolar leads in that they measure the voltage in any one location relative to zero potential. The chest leads are recorded simply by means of electrodes at six designated locations on the chest wall.

Lead V1 placed on the fourth intercostal space just to the right of the sternum.

Lead V2 placed on the fourth intercostal space just to the left sternum

Lead V3 midway between leads V2 and V4

Lead V4 placed on the midclavicular line in the fifth interspace.

Lead V5 placed on the anterior auxiliary line at the same level as lead V4

Lead V6 placed on the midaxillary line at the same level as lead V4

The chest leads, like the six extremity leads, can be represented digramatically. Like the other leads each chest lead has a positive and negative pole. The positive pole of each chest lead points anteriorly toward the front of the chest. The negative pole of each chest lead points posterior toward the back