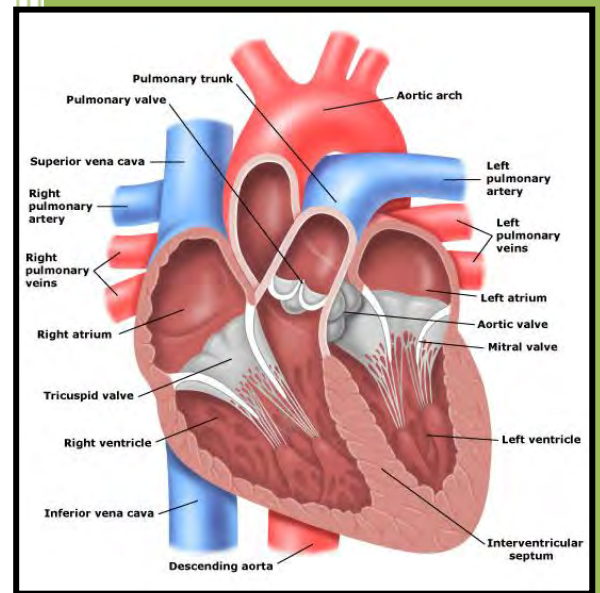


2016

Cardiothoracic Learning  
Package



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[January 2016](#)

## **ACKNOWLEDGEMENTS**

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## **AIM**

The aim of this package is to provide the nurse with adequate theoretical preparation and clinical experiences to safely and competently function in the field of cardiothoracic intensive care.

In order to facilitate the teaching/learning process and cater to the needs of the critical care nurse, it is necessary to consider a number of nursing implications which have been outlined in the program. It is anticipated that this Cardiothoracic Orientation Package will:

- Provide a stimulating and meaningful experience that builds upon the nurses existing knowledge base
- Utilise research findings to improve patient care;
- Utilise innovative teaching/learning approaches;
- Integrate new theoretical concepts with current practises and be
- An open, honest, pragmatic and collaborative learning package.

The care of the critically ill patient involves not only *what the nurse does* but most importantly *what the nurse sees, hears and feels*. With these combinations, antecedents to life threatening conditions are recognised early, prevented and/or managed. Essential nursing care is generic for all types of patients. With initiative and an introductory orientation package to cardiothoracic intensive care, any capable nurse can attain additional knowledge, skills and attitude needed for competence in cardiothoracic intensive care nursing.

## **PROGRAM OVERVIEW**

### **PACKAGE ORGANISATION**

The Cardiothoracic Orientation Package is designed for the participant to build on their own theoretical knowledge base, specifically in the field of Cardiothoracic. Therefore this package utilises a self-directed learning style which shall be supported by clinical teaching at the bedside by the Clinical Nurse Educator, Nurse Unit Manager and experienced Cardiothoracic nurses working in Cardiothoracic ICU. It is anticipated that it shall take the orientee/staff member three to six months to become competent depending on the individual needs, prior intensive care nursing experience and the completion of the allocated worksheets within the package.

The package discusses the following topics:

- Anatomical and physiological overview of the heart
- Types of cardiac surgery,
- Care for patient's post cardiac surgery,
- Post operative complications and cardiac emergencies
- Interpreting cardiac output studies
- Interpreting ECGs
- Thoracic surgery
- Relevant cardiac pharmacology.

The package is to target new staff to Intensive Care and staff who are to rotate through our Cardiothoracic ICU and who have experience working in a General ICU. Preceptors who have experience in the field of Cardiothoracic shall support these staff members. The Cardiothoracic Clinical Nurse Educator and the Unit Manager are responsible for the overall coordination and facilitation of the package.

Resource materials such as appropriate literatures, simulated practice, and worksheets will be made available to encourage self direction. The participants will be required to complete the worksheets that are included in the orientation package and a pulmonary artery catheter competency.

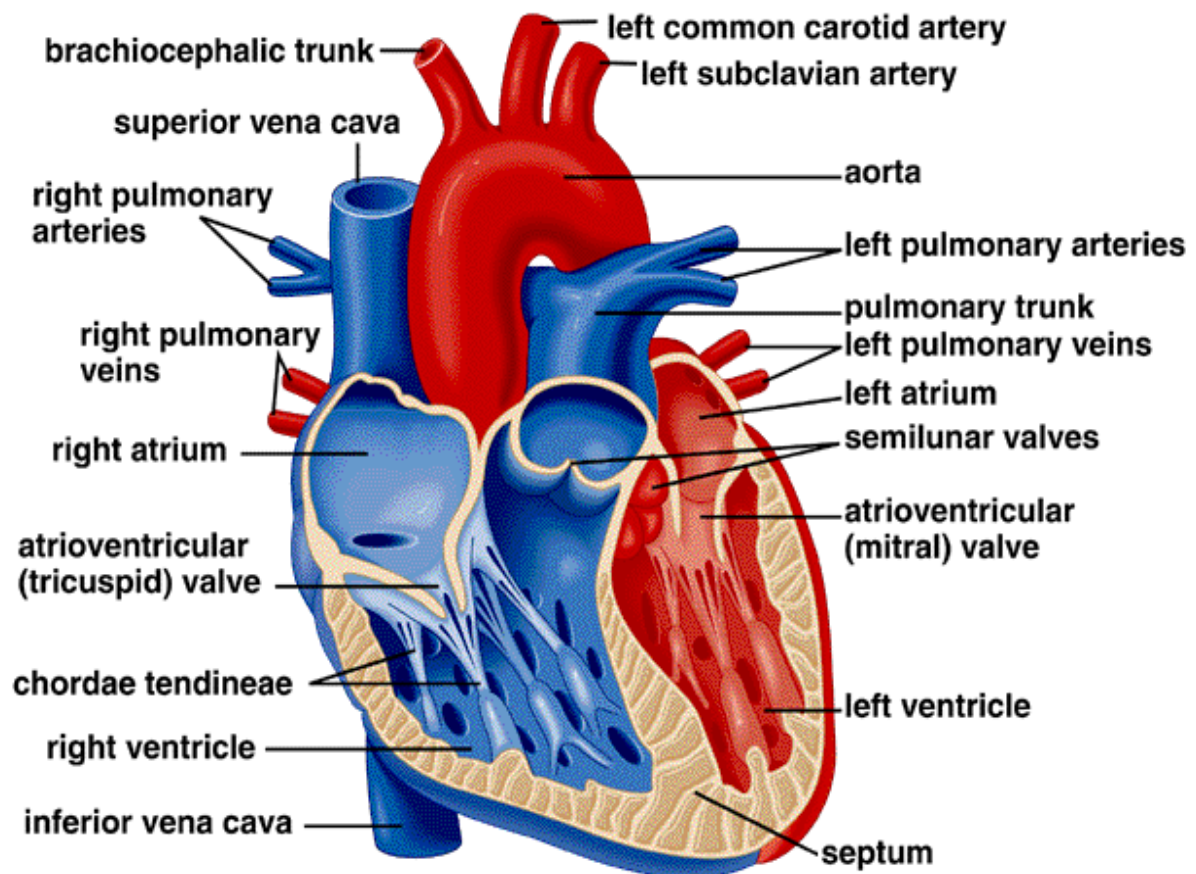
The participants will spend one morning observing a patient undergoing coronary artery bypass graft surgery and/or valve surgery in the operating theatre (This will depend on staff numbers for the day) This will help the orientee to fully understand the anatomical difficulties the surgeon faces, the role of the bypass machine in respect to the heart and lungs and why potential complications may occur.

## CARDIAC ANATOMY AND PHYSIOLOGY

The heart is the centre of the cardiovascular system. This system is the transport system of the body. It is a muscular organ located between the lungs in the mediastinum. The adult heart is about the size of a closed fist. The blood vessels form a network of tubes that carry the blood from the heart to the tissues of the body and then return it to the heart.

Sylvia S. Mader, Inquiry into Life, 8th edition. Copyright © 1997 The McGraw-Hill Companies, Inc. All rights reserved.

### Internal View of Heart



## **STRUCTURE AND FUNCTION OF HEART**

### **Pericardium**

The heart is enclosed in a loose fitting membrane called the pericardial sac which consists of two layers fibrous and serous.

The fibrous layer is the outer layer and is attached to large blood vessels entering and leaving the heart. It is also attached to the diaphragm and to the inside of the sternal wall of the thorax. It is attached to the parietal pleura which prevent the heart from over distending.

The inner or serous layer is thinner and more delicate. It is continuous with the visceral pericardium at the base of the heart and around the large blood vessels.

### **Heart Wall**

The wall of the heart is divided into 3 parts

#### 1. Epicardium

~ Thin transparent outer layer of the wall

~ composed of serous tissue and mesothelium

~ Pericardial sac is between epicardium and pericardium which contains a watery fluid known as pericardial fluid which prevents friction between the membranes as the heart moves

#### 2. Myocardium

~ Middle layer of the heart

~ It is the cardiac muscle tissue which is responsible for the contraction of the heart

~ The muscle fibres are involuntary, and the tissue is arranged in interlacing bundles of fibres

#### 3. Endocardium

~ Is the inner layer of the heart

~ It is a thin layer of endothelium overlying a thin layer of connective tissue pierced by tiny blood vessels and bundles of smooth muscle

~ lines the inside of the myocardium and covers the valves of the heart and tendons that hold them open.

### **Chambers of the heart**

The heart has four chambers

- Right and Left Atrium

~ Are the smaller upper chambers of the heart

~ The left atrium receives blood from the lungs

~ The right atrium receives blood from the rest of the body

~ The atrium allows approx 75% of blood flow directly from the atria into the ventricles prior to the atria contracting. Atrial contraction then adds 25% of filling to the ventricles; this is referred to as atrial kick.

- Right and Left Ventricles

- ~ are the larger lower chambers of the heart and are separated by the interventricular septum
- ~ The right ventricle pumps blood to the lungs
- ~ The left ventricle pumps blood to the rest of the body
- ~ The ventricles have thicker walls than the atria so they can work harder by pumping blood out to the body

### Heart Valves

The heart also has 4 valves. They prevent the blood from flowing backwards.

**Atrioventricular Valves** are the valves that lie between the atria and the ventricles. There are 2:

i) Tricuspid Valve

- ~ Is between the right atrium and the right ventricle
- ~ Consists of three flaps or cusps which are fibrous tissue
- ~ Chordae Tendinae are tiny collagen cords that anchor cusps of the valve to papillary muscle

ii) Mitral Valve

- ~ Lies between the left atrium and left ventricle
- ~ Consists of two cusps

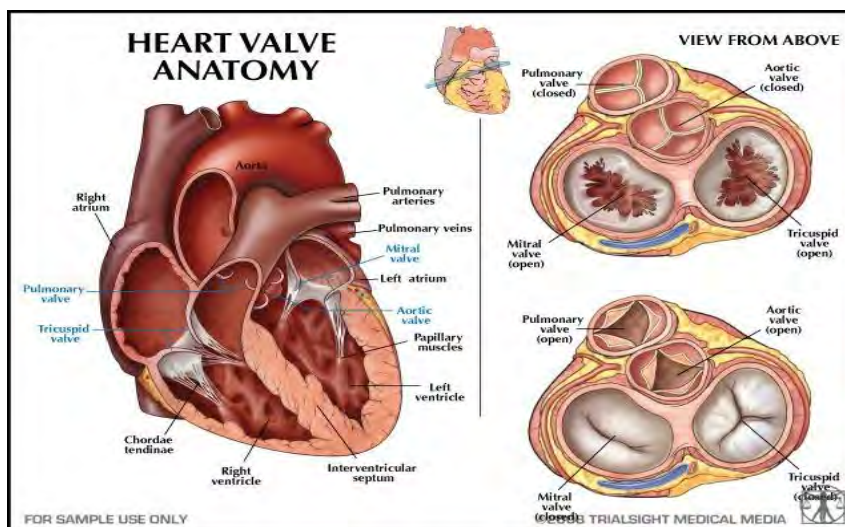
**Semilunar Valves** are the two valves located between the pulmonary artery and the aorta that prevent blood from flowing back into the heart. They are crescent shaped. Both valves consist of three semi lunar cusps. These permit blood flow in one direction from ventricles into the arteries.

iii) Aortic Valve

- ~ found at the base of the aorta and the left ventricle

iv) Pulmonary Valve

- ~lies between the right ventricle and the pulmonary artery



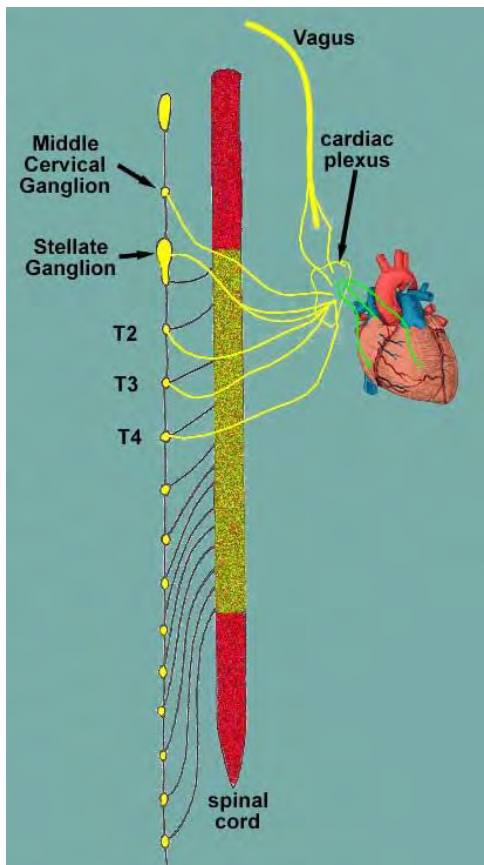


## **CARDIAC PHYSIOLOGY**

The strength and frequency of the heart beat is controlled by the autonomic system. Both parasympathetic and sympathetic parts of the autonomic system are involved in the control of the heart.

- Sympathetic fibers
  - ~ Arise from segments T2- T4 of the spinal cord
  - ~ Then go through the middle cervical and cervico – thoracic ganglia and the first four ganglia of the thoracic sympathetic chain
  - ~ Then pass into the cardiac plexus and into the SA node and cardiac muscle
  - ~ Effect of sympathetic nerves at SA node is to increase heart rate
- Parasympathetic
  - ~ Provided by the vagus nerve
  - ~ Effect of the vagus nerve at the SA node is to decrease the heart rate
  - ~ Also decreases the excitability of junctional tissue at the AV node which slows transmission.

The chambers and walls of the heart can go on contracting and relaxing without any direct stimulus from the nervous system. This is possible as the heart has its own regulating system called the conduction system.

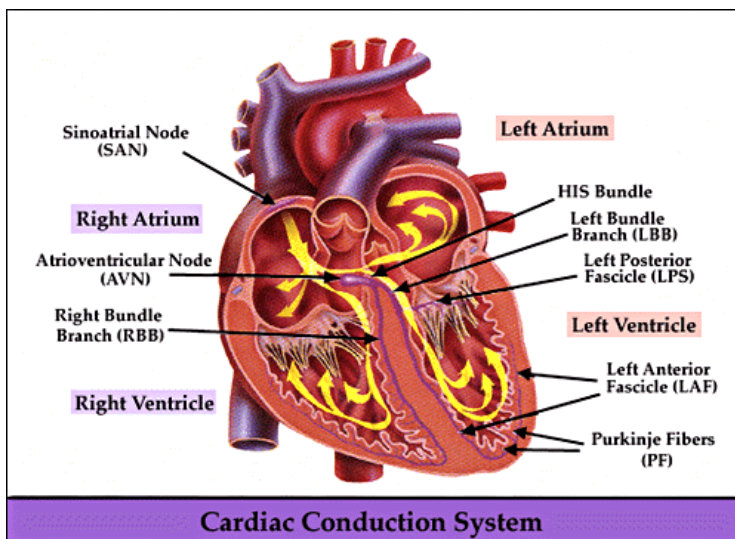


Diagrammatic view of the origin of the sympathetic fibres to the cardiac plexus. (Norman, 1999)

**Conduction System**

The conduction system is composed of specialised muscle tissue that generates and distributes the electrical impulses which stimulate the cardiac muscle fibres to contract. These tissues are the

- Sinoatrial node (SA node)
  - ~ Located in the right atrial wall inferior to the superior vena cava
  - ~ Initiates each cardiac cycle so it sets the basic pace for the heart rate
  - ~ Intrinsic heart rate is 60-100bpm
  - ~ Once SA node initiates an electrical impulse, it then spreads to both atria causing them to contract and at the same time depolarising the AV node
- Atrioventricular node (AV node)
  - ~ Lies just above the insertion of the tricuspid leaflet, anterior to the ostium of the coronary sinus
  - ~ Intrinsic heart rate 40-60bpm
  - ~ One of the last portions of the atria to be depolarised
  - ~ Permits the impulse to reach the ventricles via the interventricular septum
- Atrioventricular bundle (bundle of HIS)
  - ~ From AV node a tract of conducting Purkinje cells form to make up the His bundle
  - ~ Intrinsic rate is 30-40bpm
- Right and left bundle branches and the Purkinje fibres
  - ~ The bundle of His then continues down both sides of the septum and divides into the right and left bundle branches
  - ~ The bundle of His distributes the charge over the surfaces of the ventricles
  - ~ The right bundle activates the right ventricle and the left bundle the left ventricle
  - ~ The Purkinje fibres that emerge from the bundle branches stimulate the actual contraction of the ventricles
  - ~ Intrinsic rate of 15-30bpm



Cardiac conduction system:  
(Marquette, KH 1996)

**CARDIAC MUSCLE PHYSIOLOGY: ELECTROCHEMICAL MECHANISM**  
**THE SODIUM POTASSIUM PUMP**

The heart is composed of three major types of cardiac muscle

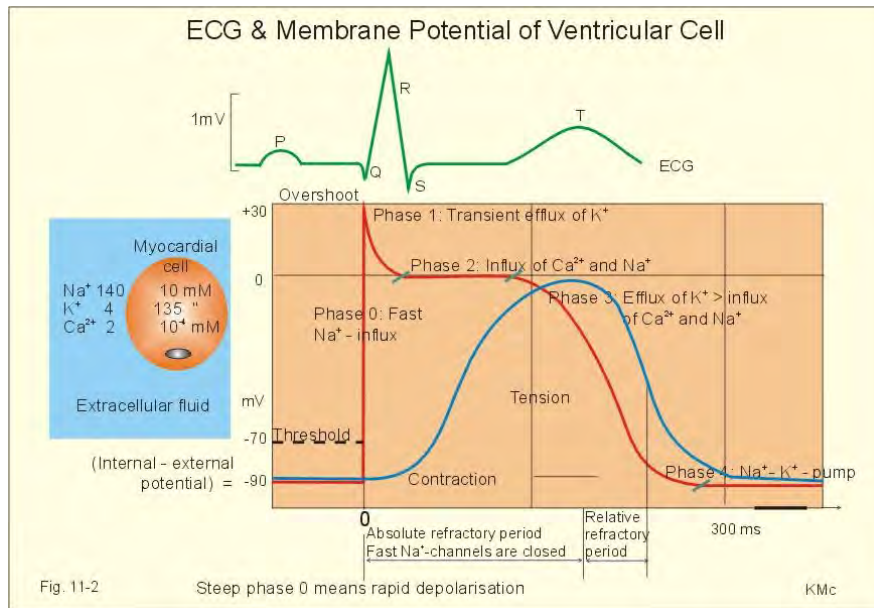
- ~ Atrial muscle
- ~ Ventricular muscle
- ~ Specialised excitatory and conductive muscle fibres
- The atrial and ventricular contractions are much longer than skeletal muscle.
- The specialised excitatory and conductive muscles fibres contract only feebly as they contain few contractile fibrils; instead, they exhibit rhythmicity and varying rates of conduction, providing an excitatory system that controls the rhythmical beating of the heart. (Guyton & Hall 10<sup>th</sup> Ed p.96)

**Cardiac muscle fibres:**

- ~ are arranged in latticework formation
- ~ are striated like skeletal muscle
- ~ have myofils that contain actin and myosin filaments like skeletal muscle
- ~ But are different from skeletal muscle in that they have cell membranes that are called intercalated discs, that separate individual cardiac muscle cells from one another
- ~ Thus, cardiac muscle is a syncytium of many heart muscle cells, in which the cardiac cells are so interconnected that when one of these cells become excited, the action potential spreads to all of them, spreading from cell to cell throughout the latticework interconnections.
- ~ Therefore, ions can move with ease in the intracellular fluid without hindrance. (Guyton & Hall. 10<sup>th</sup> Ed p.96)

**Action Potentials**

- Any process that decreases the size of the resting membrane potential (depolarisation), tends to activate the fast Na<sup>+</sup> channels
- The threshold potential for release of the action potential is a rise of 25mV from -90mV
- The cardiac action potential is an all or none response which is divided into 5 parts
  - Phase 0 – Fast depolarisation
    - ~ rapid entry of Na<sup>+</sup> into the cells
    - ~ causes phase 0 of atrial, ventricular and Purkinje action potentials
  - Phase 1 –
    - ~ is early repolarisation from the upstroke related to the K<sup>+</sup> outflux
  - Phase 2-
    - ~ plateau of the action potential where slow Ca<sup>2+</sup> and Na<sup>+</sup> channels remain open for a long period
    - ~ The net influx of Ca<sup>2+</sup> and Na<sup>+</sup> is balanced by the net efflux of K<sup>+</sup>
    - ~ Calcium activates the muscle contraction process
    - ~ When slow calcium –sodium channels close at the end of the plateau the voltage gated K<sup>+</sup> channels are activated and K<sup>+</sup> increases rapidly
  - Phase 3 - Terminal repolarisation
    - ~ With all K<sup>+</sup> channels open large amounts of K<sup>+</sup> diffuse out the ventricular fibres
    - ~ The equilibrium potential for K<sup>+</sup> (-94mV) and the resting membrane potential is approached
  - Phase 4 –
    - ~ Recognised by resting membrane potential of -90mV
    - ~ Na<sup>+</sup> - K<sup>+</sup> pump restores ionic concentrations by exchanging Na<sup>+</sup> for K<sup>+</sup> in the ratio of 3:2
  - Phase 5 –
    - ~ Covers the relative refractory period and the T- wave on ECG
    - ~ The long absolute refractory period of ventricular cells covers the whole shortening phase of the contraction (Blue curve in diagram below).
    - ~ In the absolute refractory period all the fast Na<sup>+</sup> channels are inactivated (Poul-Erik Paulev, 2000. Chapter 11)



**Diagram:**  
Recordings of ECG, intraventricular membrane potential (red curve) and contraction (blue curve) of one heart cycle in a ventricular fibre. Textbook in Medical Physiology and Pathophysiology 2000, Chapter 11

## CARDIAC CYCLE

~ is the term referring to all or any of the events related to the flow of blood pressure that occurs from one heartbeat to the next. (Guyton A.C & Hall J.E. 2006)

~ The cardiac output is dependent on an orderly pumping action of the heart, which is achieved through a cardiac cycle.

~ In a normal heart beat, the two atria contract (systole) simultaneously while the two ventricles relax (diastole). Then when the two ventricles contract, the two atria relax. This cycle can be broken up into three phases;

- Atrial systole,
- Ventricular systole and
- Ventricular filling.

### Atrial Systole

~ Is the contraction of the heart muscle of the left and right atria

~ Electrical systole is the electrical activity that stimulates the myocardium to make the atria contract

~ As atria contract the blood pressure in each atrium increases forcing additional blood into the ventricles

~ The additional flow of blood is known as atrial kick

~ 70% of blood flows passively down into the ventricles

~ Atrial kick is absent if there is loss of the normal electrical conduction such as AF, and heart blocks

~ Atrial contraction follows the P wave of the ECG

**Ventricular Systole**

- ~ Is the contraction of the muscles of the left and right ventricles
- ~ As the ventricular pressure rises, the AV valves close, mitral before tricuspid and this can be heard as the first heart sound.
- ~ An additional 0.02-0.03secs is required for the ventricle to build up sufficient pressure to push the aortic and pulmonary valves open against the pressures in the aorta and pulmonary artery.
- ~ During this time, contraction is occurring in the ventricles with no emptying, and is known as **isovolumetric contraction**
- ~ Ventricular ejection begins when the intraventricular pressure exceeds that of the aorta and pulmonary arteries and the semi lunar valves open, aortic before pulmonary.
- ~ Immediately blood flows out of the ventricles, 70% occurs in the first third period of ejection (rapid ejection) and 30% during next two thirds (slow ejection)
- ~ At the end of this period the ventricles relax and the pressure falls below that of the aorta and the pulmonary arteries.
- ~ The semi lunar valves now close and this constitutes the second heart sound.
- ~ Then follows a pause, called the **isovolumetric relaxation period**, where all the valves in the heart are closed and the intraventricular pressures fall.
- ~ The opening of the AV valves terminates this when the pressure in the atria exceeds that in the ventricles. ~ The stored atrial blood then flows into the ventricles.
- ~ Ventricular depolarisation and subsequent contraction occur at 0.12-0.20 seconds (P-R interval on ECG) after similar events in the atrium.

**Ventricular Filling**

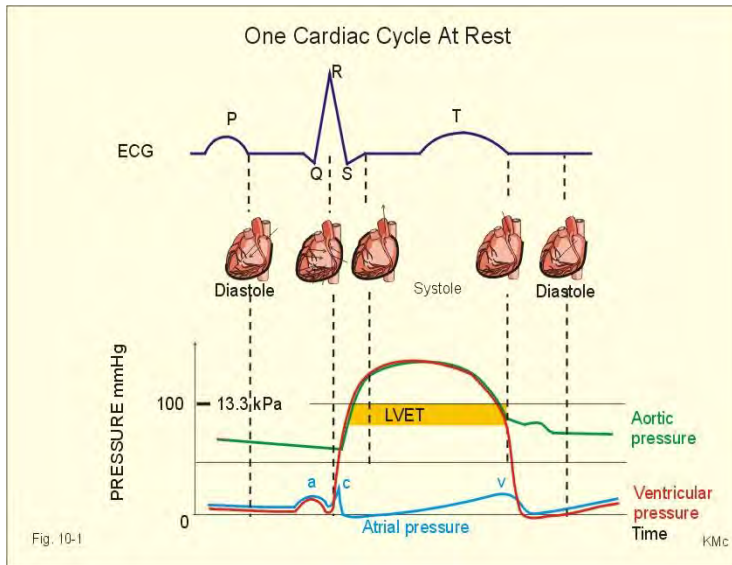
- ~ The initial filling of the ventricles is very rapid and the AV valves are wide open. However, in mid-diastole the valves move toward the closed position and then open again widely during atrial systole.

**End-Diastolic Volume, End-Systolic Volume and Stroke Volume Output**

- ~ End - Diastolic volume is the filling in each ventricle (110-120mL) at the end of diastole.
- ~ Stroke volume output is the volume left (70mL) as the ventricles empty during systole.
- ~ The remaining volume in each ventricle 40-50ml is called the end-systolic volume
- ~ The fraction of the End-diastolic volume that is ejected is called the Ejection Fraction, usually equal to about 60%

**Cardiac cycle**

Cardiac cycle describes volume, pressure and electric phenomena in the left ventricle as a function of time and one heart beat

**Electromechanical events in cardiac cycle**

Textbook in Medical Physiology and Pathophysiology 2000 Chapter 10

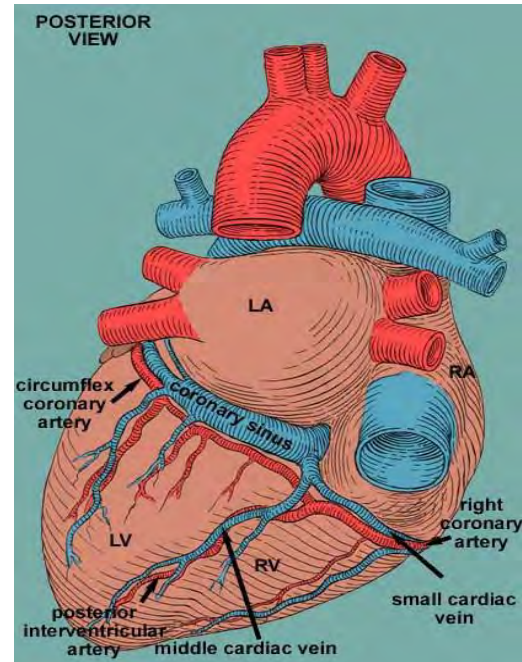
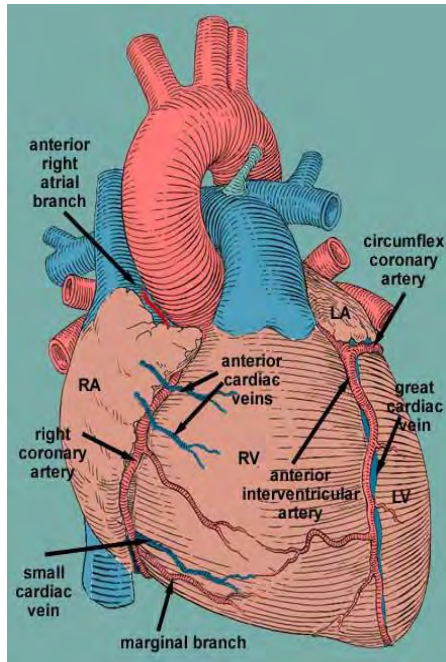
**Green curve**: the aortic pressure

**Blue curve**: atrial pressure curve with a, c, & v waves

**Red curve**: Intraventricular pressure

## CORONARY CIRCULATION

Coronary circulation is the circulation of blood in the arteries and veins of the heart muscle. The heart muscle is supplied by the coronary arteries which are direct branches of the ascending aorta. The vessels that deliver oxygenated blood to the myocardium are coronary arteries and the vessels that remove de-oxygenated blood are coronary veins.



Anterior and Posterior views of coronary circulation: (Norman 1999)

### CORONARY ARTERIES

The heart is supplied by two major coronary arteries, the right coronary artery and the left coronary artery.

#### LEFT CORONARY ARTERY

Arises from the left posterior aortic sinus as the Left main coronary artery and passes behind the base of the pulmonary artery. The left main coronary artery divides into the **Left Anterior Descending artery (LAD)** or the intraventricular artery and the **Left Circumflex artery (Cx)**.

The LAD artery runs in the anterior interventricular groove and continues up to the apex of the heart. It supplies the anterior part of the septum with septal branches and the anterior wall of the left ventricle with diagonal branches. The LAD supplies most of the left ventricle and the AV bundle.

The diagonal branches come off the LAD and run laterally to supply the antero-lateral wall of the left ventricle. There can be one or more diagonal branches: D1, D2

The LAD is the artery most often involved in coronary occlusions and therefore the artery that is mostly bypassed in cardiac surgery.

## **CIRCUMFLEX**

The circumflex (Cx) lies in the left AV groove between the left atrium and the left ventricle and supplies the vessels of the lateral wall of the left ventricle. These vessels are known as obtuse marginals (M1, M2 etc) because they supply the lateral margin of the left ventricle and branch off with an obtuse angle. The circumflex artery also supplies branches to the left atrium.

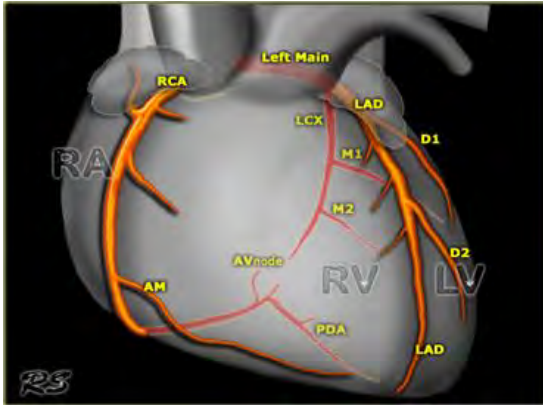


Illustration showing overview of coronary arteries  
([Smithuis & Willems 2008](#))

Left coronary artery (LCA)

- ~ Left anterior descending (LAD)
- ~ Diagonal branches (D1,D2)

Circumflex (Cx)

- ~ Marginal branches (M1,M2)

Right coronary artery

- ~ Acute marginal branch (AM)
- ~ AV node branch
- ~ Posterior descending artery (PDA)

## **RIGHT CORONARY ARTERY**

The right coronary artery arises from the anterior aortic sinus and courses through the right atrioventricular groove between the right atrium and right ventricle to the inferior part of the septum.

The **pulmonary conus branch** is the first branch of the right coronary artery and crosses the pulmonary trunk at the level of the pulmonary valve. It is an important collateral pathway to the LAD artery. The atrial branches are small with the exception of those supplying the cardiac conduction system.

The atrioventricular node artery passes upwards near the point of intersection of the atrioventricular groove and the posterior interventricular groove (the **crux of the heart**). Multiple muscular branches arise from the right coronary artery to supply the right ventricle. Since most of these cross the acute margin of the heart they are referred to as the **acute marginal branches**. They are important collateral pathways to the posterior interventricular groove and the distal LAD.

The branch to the posterior interventricular groove is called either the **Posterior Descending Artery (PDA)** or the Posterior Intraventricular Artery (PIVA)



**DOMINANCE**

Is a term used to indicate which coronary artery supplies the **atrioventricular node, the posterior descending artery and the posterior surface of the left ventricle**. In 80% of the population the right coronary artery is dominant

However, the left coronary artery still supplies the major portion of the left ventricle. About 20% of the population has either left dominance or a balanced system in which the right coronary supplies the AV node and a small PDA while the distal circumflex artery supplies the posterior surface of the left ventricle.

**VEINS OF THE HEART**

The coronary sinus is situated in the left atrioventricular groove and is formed by the junction of the great cardiac vein and the posterior vein of the left ventricle. It empties into the right atrium between the opening of the inferior vena cava and the tricuspid valve.

The great cardiac vein begins at the apex of the heart and ascends in the anterior interventricular groove to reach the atrioventricular groove. It curves to the left and receives tributaries from the left atrium and the left marginal vein from the antero-lateral surface of the left ventricle. On reaching the back of the heart the great cardiac vein ends in the coronary sinus.

The posterior vein of the left ventricle drains the diaphragmatic surface of the left ventricle a little to the left of the middle cardiac vein, which lies in the posterior interventricular groove. The small cardiac vein drains the right atrioventricular groove and enters the right extremity of the coronary sinus. Often the small cardiac vein receives the right marginal vein, which drains the posterior surface of the right atrium and right ventricle. The anterior cardiac veins drain the anterior surface of the right ventricle and empties into the right atrium.

The major importance of the coronary veins is that they may occasionally be confused with a late appearing artery and they may impede surgical access to the coronary artery in the left side of the atrioventricular groove.

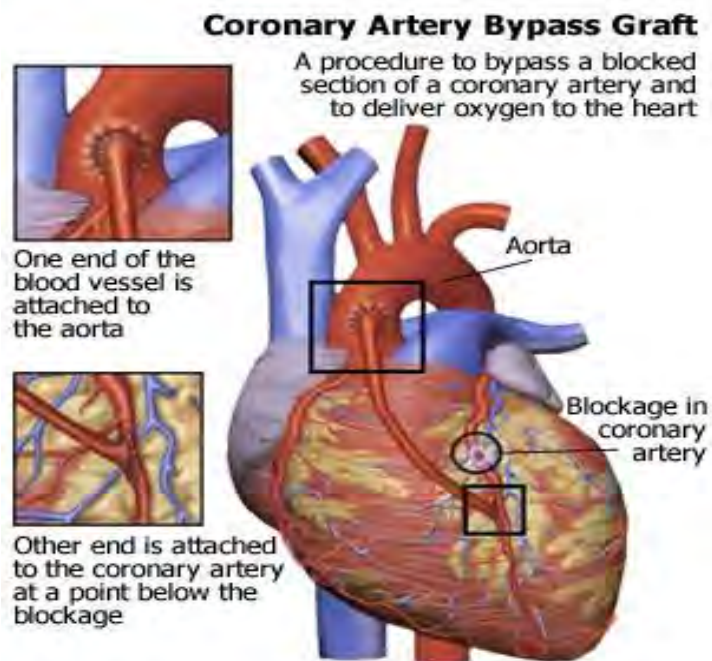
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## TYPES OF CARDIAC SURGERY

### CORONARY ARTERY BYPASS GRAFT SURGERY

Coronary bypass graft surgery is a procedure where arteries or veins are grafted onto the patient's coronary artery to bypass atherosclerotic narrowing's and improve the blood supply to the coronary circulation. This creates a new pathway of blood flow that ensures the delivery of oxygen and nutrients to the heart muscle.

Bypass grafting is possible due to the atherosclerotic plaques being situated distally in the proximal portions of the major coronary artery branches. These lie on the epicardial surface of the heart. The obstruction in the coronary artery is bypassed by anastomosing one end of the vein graft to the aorta and the other end to the coronary artery just past the obstruction. Saphenous Vein Grafts (SVGs) can be simple with an end-to-end anastomosis to the aorta and the coronary artery. Alternatively they can be a skip or sequential with an end-to-side anastomosis to the aorta, a side-to-side anastomosis to one coronary artery and an end-to-side anastomosis to another coronary artery.



(Kumar 2007)

**BYPASS PROCEDURE: CAGS AND VALVES**

- ~ The chest is opened via median sternotomy
- ~ The pericardium is opened and retracted
- ~ Patient is anticoagulated
- ~ The grafts are harvested from saphenous veins, internal mammaries or radial arteries
- ~ The surgeon sutures cannulae into the heart and the perfusionist starts cardiopulmonary bypass (CPB).
- ~ The aorta is cross clamped and cardioplegia is inserted into the aorta and coronary arteries to cause cardiac arrest (see below)
- ~ Then the grafting process begins where one end of the graft is sewn to the aorta and the other onto the coronary artery beyond the blockage
- ~ If surgery is for a valve replacement the diseased valve is repaired or replaced
- ~ The heart is restarted and CPB is slowly discontinued
- ~ Protamine is given to reverse the effects of heparin
- ~ Pacing wires are inserted into the myocardium
- ~ Intercostal drains are inserted into the pericardium, mediastinum and pleural space
- ~ The sternum is wired together and then the skin is closed

**Cardioplegia**

Is a solution that is injected into the aorta during heart surgery to accomplish asystole. It is used to protect the myocardium from damage as a non beating, cold heart uses less oxygen.

An iced solution of potassium, magnesium, glucose and other ingredients is introduced into coronary circulation via cannulae.

The cold fluid ensures that the heart cools down to 15-20°C which causes a decrease in metabolism of the heart and the high concentration of potassium and magnesium causes the heart to stop which ensures that the heart does not use up energy stores (ATP-adenosine triphosphate)

When the solution is introduced into the aortic root it is called antegrade cardioplegia. When it is introduced into the coronary sinus it is called retrograde cardioplegia

**Cardiopulmonary Bypass machine**

CPB is a procedure used in cardiac surgery where a machine takes over the function of the heart and lungs to maintain the circulation of the blood to vital organs while the surgeon operates on a still heart.

The CPB machine has three components, a pump, an oxygenator and plastic circuit. Venous blood is drained into the oxygenator by a cannula placed in the right atrium or vena cava. The oxygenator takes over the function of the lungs by oxygenating the blood and removing carbon dioxide. It also cools the blood when the patient is going on bypass and re-warms blood as patient is coming off bypass.

Oxygenated blood is then returned via a cannula in the ascending aorta to maintain blood supply to the brain, kidneys and other organs.

Blood flow rates are adjusted to maintain a MAP of approx 70mmhg and a cardiac index of 2-3l/min/m<sup>2</sup>.

Patient is given heparin to prevent clotting and the blood viscosity is reduced by decreasing the haemocrit using a crystalloid solution.

**CARDIOPULMONARY BYPASS MACHINE**

Accessed June 2010  
Cardiothoracic Surgeons of Grand Traverse 2007

**Off Bypass**

Off bypass coronary bypass surgery (OPCAB) is when grafting is performed without the bypass machine. This can be achieved by using a stabilising device or “octopus” which is applied to the area of the heart to keep it still while the grafting is being performed.



Stabilising device



Stabilising device in place

Accessed June 2010  
Heart Surgeons.com  
Mid Atlantic Surgical Associates 2009

**CONDUITS**

The choice of conduits used depends on the surgeon and the suitability of the conduits. Usually the Left Internal Mammary Artery (LIMA), Right Internal mammary Artery (RIMA), saphenous veins from the legs and radial arteries are used for grafting.

**INTERNAL MAMMARY ARTERY (IMA)**

Can use either the left or the right and it originates as a branch off the subclavian artery which descends down the anterior chest wall just lateral to the sternum behind the costal cartilage, around the 4th -6th intercostal space.

- ~Once it is located it is dissected away and is flipped over and usually grafted to the LAD.
- ~IMAs are longer lasting than veins as already connected to the arterial tree and need only to be connected at one end
- ~IMAs size is very close to the coronary arteries diameter
- ~Flow may be less turbulent due to IMA and coronary artery geometry
- ~IMA grafts have no valves or varicosities
- ~Aortic anastomosis is not required
- ~No leg incision if only the IMA is used
- ~IMAs are sensitive to handling and may go into spasm

**SAPHENOUS VEIN GRAFTS**

Usually the greater saphenous vein of either the right or left leg is used for grafting.

- ~Veins that are used have their valves removed or are turned around so that the valves do not occlude blood flow in the graft
- ~Veins are pliable enough to allow easy suturing
- ~Usually can be harvested as free grafts of sufficient length to bypass stenosed arteries.
- ~Graft closure is more likely in
  - grafts with poor distal runoff and low flows
  - grafts to coronary arteries of small calibre
  - or arteries supplying muscle that is heavily scarred

**Alternative Conduits Used**

- ~ Radial artery
- ~ Brachiocephalic Vein
- ~ Gastroepiploic artery
- ~ Inferior epigastric artery

The last three are rarely used due to difficult mobilisation

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## **VALVULAR HEART SURGERY**

### **VALVULAR HEART DISEASE**

Heart valve disease is generally classified as either valvular stenosis or valvular regurgitation.

#### **Valvular Stenosis**

~ Is when the valve openings become too narrow due to stiff or fused leaflets, which makes the heart work hard to pump blood through the valves

~ This causes an impedance of blood flow behind the valve which leads to an increase in pressure in the chambers behind the valve.

#### **Valvular Regurgitation**

~ Also called valvular insufficiency, incompetence or a “leaky valve”

~ Is when the valves become incompetent allowing the blood to leak across the valve when they are supposed to close

~ This causes an increase in blood volume in the chambers which results in dilatation of the chamber

- Valvular stenosis and regurgitation in any of the cardiac chambers can produce permanent weakening of the cardiac muscle and eventually lead to heart failure.

Both of these cause turbulence of blood flow which can be heard as a “heart murmur” when listening to the heart with a stethoscope

### **MITRAL STENOSIS**

~ The mitral valve separates the left atrium from the left ventricle

~ Stenosis occurs due to rheumatic heart disease, bacterial endocarditis or calcification.

~ The disease process causes fusion of the commissures and fibrotic contraction of valve leaflets, commissures and chordae tendinae.

~ Forward blood flow is impeded as the valve orifice becomes smaller leading to pooling of blood in the left atrium and eventually the lungs

### **MITRAL REGURGITATION**

~ May result from rheumatic endocarditis, left ventricular dilatation, ischaemic damage to the subvalvular apparatus or mitral valve prolapse.

~ Regurgitation of blood through the incompetent valve during systole causes increased volume and pressure in the left atrium with resultant left atrial enlargement.

~ Left ventricular hypertrophy and dilatation develop, as the ventricle has to pump more forcefully to maintain adequate forward flow.

### **Manifestations**

~ Fatigue

~ Exertional dyspnoea

- ~ Pulmonary congestion
- ~ Pulmonary oedema
- ~ Palpitations
- ~ Pulmonary hypertension
- ~ Atrial Fibrillation, atrial flutter due to atrial dilatation

### **AORTIC STENOSIS**

- ~ Aortic valve separates the left ventricle from the aorta
- ~ Stenosis may develop as a result of rheumatic heart disease, idiopathic calcification, or valvular abnormality usually congenital bicuspid valve.
- ~ It causes fusion of the commissures and fibrous contractures of the cusps, obstructing left ventricle outflow therefore increasing pressure in the left ventricle
- ~ The left ventricle hypertrophies with diastolic filling being impaired leading to left sided heart failure.
- ~ It is also associated with an increased incidence of ventricular arrhythmias, conduction disturbances and sudden cardiac death.

### **AORTIC REGURGITATION**

- ~ Often due to rheumatic endocarditis or can be secondary to ascending aortic aneurysms, aortic dissection or Marfan's syndrome.
- ~ The left ventricle is subjected to increased volume and pressure as a portion of each stroke volume is regurgitated during diastole.
- ~ The left ventricle hypertrophies and SVR is decreased to maintain an adequate forward flow. Over a period of time the left ventricle dilates and loses the ability to contract effectively.
- ~ Ejection fraction decreases and symptoms of left sided heart failure are seen.
- ~ Therefore early valve repair/replacement is indicated in these patients

### **Manifestations**

- ~ Exertional dyspnoea
- ~ Orthopnoea
- ~ Paroxysmal nocturnal dyspnoea
- ~ Syncope
- ~ Angina
- ~ Widened pulse pressure
- ~ Low diastolic blood pressure

### **TRICUSPID STENOSIS**

- ~ Tricuspid valve separates the right atrium from the right ventricle
- ~ When stenosis develops there is an increase in pressure in the right atrium which leads to an increase in pressure throughout the body

~ This causes oedema of the legs, abdomen and liver

### **TRICUSPID REGURGITATION**

~ Is usually seen in patients who have pulmonary hypertension that may also be due to mitral valve disease and sometimes with aortic valve disease.

~ Tricuspid regurgitation results in right sided heart failure due to right ventricular enlargement.

~ It is commonly seen in IV drug abusers as the valve is infected by venous contamination.



Aortic Valve Replacement: Surgical sutures are placed then valve is lowered into the patient's aorta

### **VALVE REPLACEMENT**

~ Was first performed in 1960 by Harken and Starr using a caged ball prosthesis.

~ The patient is put on CPB and the surgeon opens the heart through the left atrium and exposes the diseased valve

~ If the valve is partially damaged it is repaired and the rim or "annulus" of the valve is supported with a "ring"

~ If the valve cannot be repaired it is removed and replaced with a tissue or mechanical valve

~ The valve is tested to make sure it opens and closes and the atrium is closed

~ Then the clamp on the aorta is removed and all the air is expelled from the heart

~ The patient is then weaned from CPB

(Mid-Atlantic Surgical Associates 2008)

### **TYPES OF CARDIAC VALVULAR PROSTHESIS**

#### **Mechanical**

~ Made from ceramic

~ Excellent durability

~ Require anticoagulation for life due to high risk of thromboembolism

~ May cause some haemolysis

Patients that are likely to receive mechanical valves are:



- 
- ~ Children
  - ~ Young adults excluding women of child bearing age
  - ~ Previous tissue valve recipients
  - ~ Patients with renal failure
  - ~ Patients with small valvular annulus
  - ~ High pre- operative risk
  - ~ Other indications for anticoagulation eg atrial fibrillation
  - ~ Patients requiring aortic root replacements

### **Tissue valves**

- ~ Made from cow or pigs hearts
- ~ Allograft or homograft are obtained from the human heart, when transplant of the heart is unsuitable but the cardiac valves are satisfactory
- ~ May not require anticoagulation
- ~ Usually wear out after 10-15 years

Patients that are likely to receive tissue valves are:

- ~ Elderly patients where chronic durability is less important
- ~ Patients in whom chronic anticoagulation is not advised, such as
  - history of major bleeding episodes
  - women of child bearing age
  - non compliance with medical treatments
  - life style with high risk of trauma
  - advanced age with a potential for dosage error or falling
- ~ Patients with increased risk of thromboembolism
- ~ Patients living in remote areas where regular blood testing is not available.



**Aortic Valve replacement:** Prosthetic aortic valve is in place inside the transacted aorta. The cardiac surgeon is tying sutures that will hold the valve in place

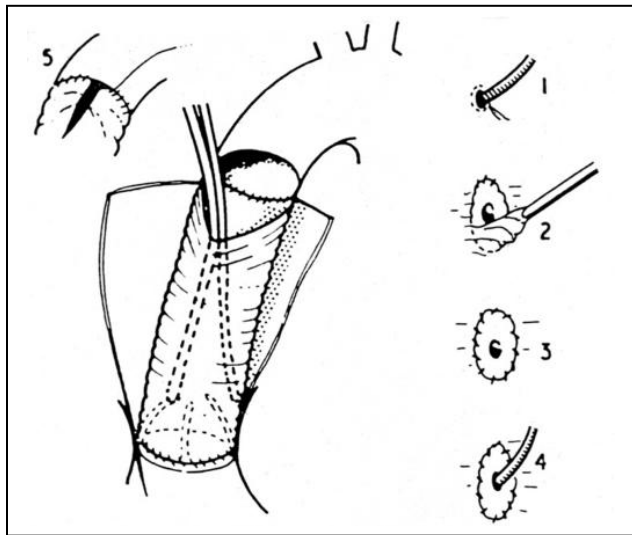
**BENTALLS PROCEDURE OR AORTIC ARCH REPLACEMENT**

A Bentalls procedure is a graft replacement of the aortic valve, aortic root and ascending aorta, with re-implantation of the coronary arteries into the graft.

~ Used to treat aortic valve and ascending aortic disease, including lesions associated with Marfans syndrome

~ First described in 1968 by Hugh Bentall, M.D and De Bono, M.D. The original description of the technique involves direct implantation of coronary arteries into the tube graft and tidily wrapping the rest of the aorta around the prosthesis.

~ Numerous modifications of the technique have been suggested since. (Marko Turina 2003)



**Diagram shows** Combined prostheses in situ.

Insets 1 to 4 show:

- Details of holes fashioned in the sidewall of the Teflon tube to reincorporate the coronary ostia within the lumen of the new ascending aorta.
- Inset 5 shows the vertical slit in the prosthesis.

(Marko Turina 2003)

**AORTIC DISSECTION**

Aortic dissection is a tear in the wall of the aorta that causes blood to flow between the layers of the wall of the aorta and forces the layers apart.

This creates a false channel or double lumen within the aortic wall which can vary in the extent of dissection.

This is a medical emergency and it can quickly lead to death if the dissection tears the aorta completely open.

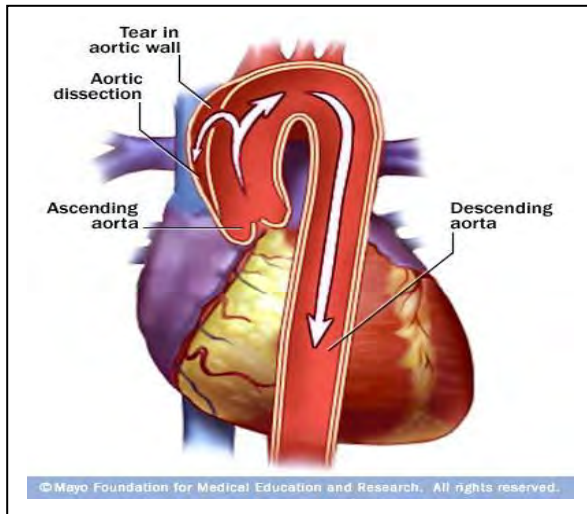
~ The aorta has three layers:

- intima – has direct contact with flow of blood and made up of endothelial cells
- media – muscular layer and made up of smooth muscle cells and elastic fibers
- adventitia – outermost layer made up of connective tissue

~ In aortic dissection blood penetrates the intima and enters the media layer

~ The force of the blood that enters the media causes the tear to extend

~ This can continue along the length of the aorta forwards or backwards

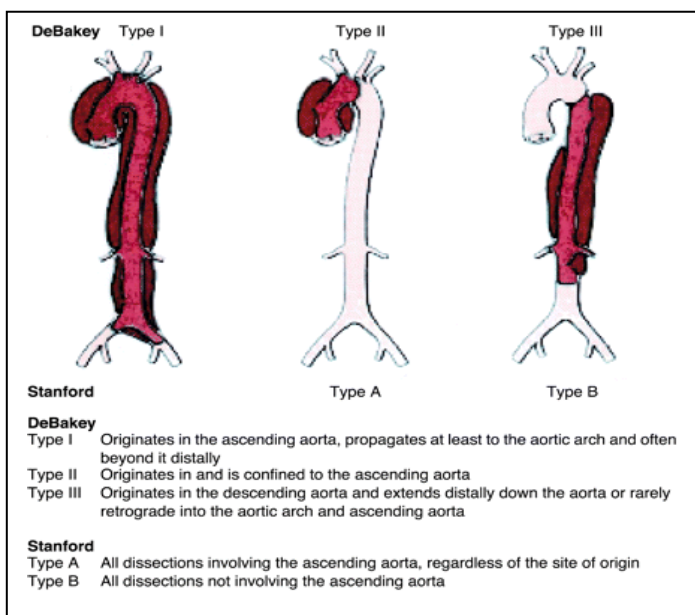


Aortic dissection occurs when a partial tear in the main artery of your heart (aorta) causes a separation (dissection) of the layers of the aortic wall.

### Classification of Dissections

There are two common classifications of dissection which are based on the anatomy of the dissection

- **DeBakey**
  - ~ categorises the dissection based on where the original intimal tear is located and the extent of the dissection
  - Type 1 - Originates in the ascending aorta continues to the aortic arch and sometimes beyond it distally
  - Type 2 - Originates in and is confined to the ascending aorta
  - Type 3 - Originates in the descending aorta rarely extends proximally but will extend distally
- **Stanford**
  - ~ divided into two groups A and B depending on whether the ascending aorta is involved
  - A - Type 1 and 2 DeBakey
  - B - Type 3 DeBakey



Classification of Aortic Dissection  
(Vijay S.Ramanath MD et al. May 2009)

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**CAUSES AND CONTRIBUTING FACTORS OF AORTIC DISSECTION**

- Congenital cardiovascular disease
- Bicuspid aortic valve disease or coarctation of the aorta
- Connective tissue disease such as Marfan's syndrome or Ehlers-Danlos syndrome
- Hypertension
- Cocaine use
- Pregnancy
- Thoracic trauma
- Recent invasive medical or surgical procedures

**KEY PHYSICAL FINDINGS WITH AORTIC DISSECTION**

- Hypertension
- Restlessness
- Unequal blood pressure in both arms
- Absence of/changes in peripheral pulses
- Pallor
- New diastolic murmur of aortic insufficiency
- Signs of shock indicative of rapid progression or possible rupture of dissection
  - ~ Tachycardia
  - ~ Hypotension
  - ~ Oliguria
  - ~ Diaphoresis

**PHYSICAL FINDINGS OF AORTIC DISSECTION ACCORDING TO ANATOMICAL LOCATION**

- Brachiocephalic Artery Obstruction
  - Changes in peripheral pulses
- Pericardial Effusion
  - S3 heart sound, pericardial rubs
- Ascending aortic dissection
  - Aortic insufficiency
- Coronary Artery Obstruction
  - Chest pain, ECG changes.
- Renal Artery Obstruction
  - Decrease in urine output
- Carotid Artery Obstruction
  - Loss of consciousness, syncope, dizziness, paralysis.

- Pericardial Tamponade  
Increased jugular venous pressure, narrowed pulse pressure, distant heart sounds.
- Superior Mesenteric Artery Obstruction  
Increased bowel sounds, melena, abdominal pain.
- Iliac Artery Obstruction  
Change in peripheral pulses.

## **POST-OPERATIVE CONSIDERATIONS FOR THORACIC AORTIC SURGERY**

### **Blood Pressure Control**

~ BP is tightly maintained usually using a combination of beta-blockade and Sodium Nitroprusside infusion. This is done to protect the graft anastomosis.

~ Life-long control of hypertension is needed

### **Bleeding and Ischaemic organ injury**

~ Can be due to extensive blood loss during theatre, prolonged CPB time, and excessive haemodilution or coagulation defects.

### **Stroke or Generalized Cerebral Dysfunction**

~ is secondary to embolisation of air or particulate matter.

### **Infection of the Vascular Prosthesis**

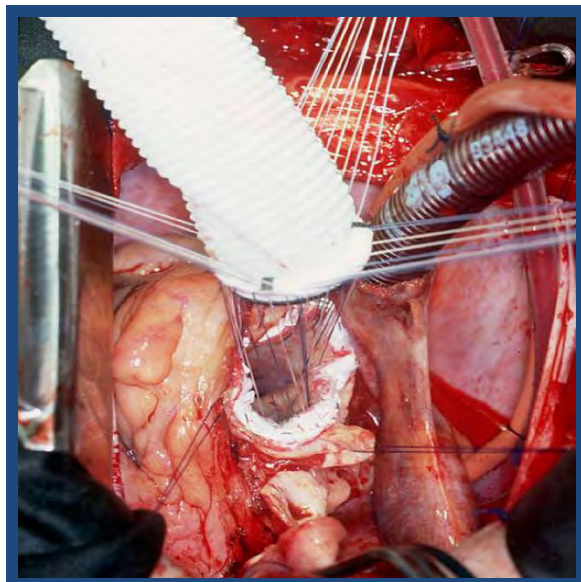
~ The infection usually produces a false aneurysm at the suture line

~ High risk patients for infection are

- Diabetics
- Pre-operative use of steroids
- Post -op sepsis or
- Infection elsewhere in the body.

Composite graft implantation using Teflon felt buttressing in a patient with Marfan disease

(Marko Turina 2003)



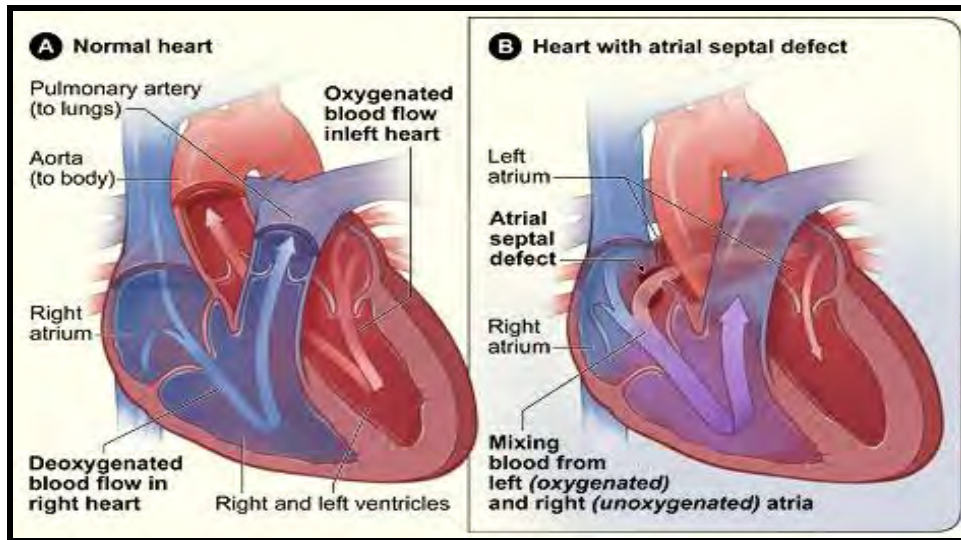
## ASDs AND VSDs

### ATRIAL SEPTAL DEFECTS

An atrial septal defect is a hole in the wall that separates the atria of the heart.

This allows blood to flow from one atria to the other, usually from left to right side. This causes extra blood flow in the right atrium, right ventricle and the lungs.

ASD is the second most common congenital heart defect



National Institute of Health 2010

Diagram A: shows the normal anatomy and blood flow of the interior of the heart.

Diagram B: shows a heart with an atrial septal defect, which allows oxygen-rich blood from the left atrium to mix with oxygen-poor blood from the right atrium.

### **Types of ASD**

Three major types of ASD exist, based on the location of the defect on the septum:

- **Secundum.** This defect is in the middle of the septum. It is the most common form of ASD. About 7 out of every 10 babies born with ASD have this type. This type often closes on its own, unless it is large.
- **Primum.** This defect is in the lower part of the septum. It also involves an incomplete or partial atrioventricular septal defect, and the valves that separate the upper and lower heart chambers are not normal. About 2 out of every 10 babies born with ASD have primum defects. This type of defect does not close on its own.
- **Sinus venosus.** This defect is in the upper part of the septum near where a large vein (the superior vena cava) brings blue blood from the upper body to the right atrium. It is rare, accounting for only about 1 out of every 10 cases of ASD. Children with sinus venosus defects usually have an associated condition called partial anomalous pulmonary venous return, in which one or more of the veins carrying red blood from the lungs return to the wrong chamber of the heart. This type of defect does not close on its own. (National Institute of health 2010)

**Effects of ASD**

- ~ Enlargement of right ventricle
- ~ Right heart overload
- ~ Arrhythmias
- ~ Stroke
- ~ Pulmonary hypertension

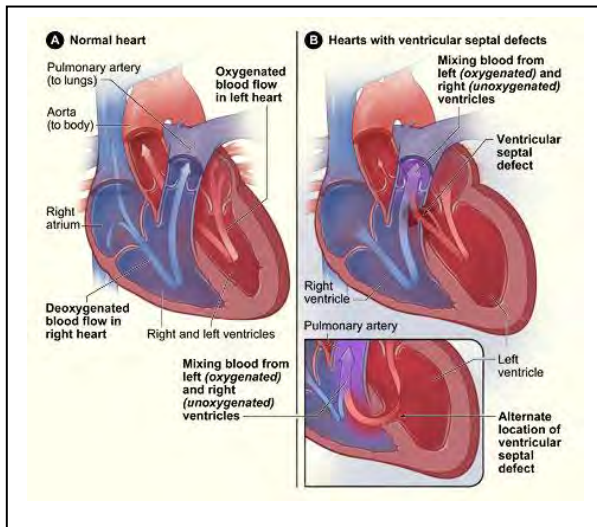
**Treatment**

- ~ Surgery for ASDs involves opening of at least one atria and closing the defect with a patch

**VENTRICULAR SEPTAL DEFECTS**

VSD is a hole or defect in the wall that separates the ventricles of the heart. This wall is called the ventricular septum. The defect causes blood to flow directly between the ventricles which allows oxygenated blood to mix with deoxygenated blood.

VSD is also a congenital heart defect.



National Institute of Health 2010

Diagram A: shows the normal anatomy and blood flow of the interior of the heart.

Diagram B: shows two common locations of ventricular septal defects. The defect allows oxygen-rich blood from the left ventricle to mix with oxygen-poor blood in the right ventricle

**Types of VSD**

Doctors classify VSDs based on the:

- Size of the defect.
- Location of the defect.
- Number of defects.
- Presence or absence of a ventricular septal aneurysm—a thin flap of tissue on the septum. It is harmless and can help a VSD close on its own. (National Institute of Health 2010)

VSDs are found in different parts of the septum.

- Membranous VSDs are located near the heart valves. They can close at any time if a ventricular septal aneurysm is present.
- Muscular VSDs are found in the lower part of the septum. They are surrounded by muscle, and most close on their own during early childhood.
- Inlet VSDs are located close to where blood enters the heart. They are less common than membranous and muscular VSDs.
- Outlet VSDs are found in the part of the ventricle where the blood leaves the heart. This is the rarest type of VSD.

**Effects of VSD**

- ~ Congestive heart failure
- ~ Failure to thrive in children
- ~ Bacterial endocarditis
- ~ Arrhythmias
- ~ Pulmonary hypertension

**Treatment**

- ~ Can be conservative with some closing by themselves
- ~ Surgical closure of hole



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## **POST OP CARE OF CARDIAC PATIENTS**

THIS IS A GUIDE ONLY FOR THE CARE OF THE STABLE / ROUTINE CARDIOTHORACIC PATIENT  
Adapted from Care of Post-op Cardiac Patient Guideline 2015

### **PREPARATION OF BED AREA:**

~Set up bed according to "Setting up bed for Cardiothoracic (CT) Patient" list found in ward clerk area

~Set up Bed area:

- Ventilator checked, set up with appropriate settings and ready to connect
- Emergency equipment checked and working
- 1Litre 4% glucose + 0.18% sodium chloride+20mmol MgSO<sub>4</sub> (maintenance fluid)
- Cardiac Output module and cable
- 12 lead ECG cable
- Suction canisters and tubing x2
- Pathology tubes, for EUC,FBC,LFT,CMP,COAG,CKMB,Troponin
- Paperwork: flowchart, medication chart, pathology result chart
- Protamine 100mg, sodium nitroprusside 100mg
- Chest clamps
- 0.9% sodium chloride for fluid boluses
- Notify ICU doctor when 20min call received

### **ASSESSMENT AND IMMEDIATE POST OP CARE**

There are usually two nurses an ICU Doctor, Anaesthetist and Cardiothoracic surgeon at the bedside to receive a full clinical handover on the patient.

Priority of nursing care will always be

- Airway
- Breathing
- Circulation

The role of the nurse is as follows:

#### Nurse 1

- Connect ECG and Spo<sub>2</sub> modules and cables to bedside monitor
- Zero and level pressure transducers and modules
- Monitor and manage HR, BP, CVP, PAP, and Spo<sub>2</sub>
- Receive handover from anaesthetist and OT nurse
- Check alarms are on and limits set appropriately
- Check all infusions are correctly labelled and running via appropriate lumens and at desired rates
- Connect maintenance fluids give protamine 100mg if required (see pharmacology guidelines)
- Check Pulmonary artery catheter
  - ~ Check position of PA catheter as per black markings at insertion site (usually between 50-60cms) and record on flowchart
  - ~ Check catheter is wedging and secure catheter to patient's chest
  - ~ Check that PA trace on monitor is a pulmonary artery trace

- Check Pacemaker
  - ~ Check pacing wires are secured in the connector block and the lead pins are tight in the pacemaker
  - ~ Change pacemaker from Biotronic to Medtronic
  - ~ Check that the leads are connected to the correct chamber, atrial to right of the patient's sternum, ventricular to left of the patient's sternum
  - ~ Check pacemaker settings are on
    - Mode DDD,
    - Atrial Output 10v
    - Atrial sensitivity 0.2mv
    - Ventricular output 10v
    - Ventricular sensitivity 2.0mv
    - Thresholds can be done later as long as patient is stable
    - If underlying rhythm 12 lead ECG if patient stable
- 12 lead ECG
- Cardiac output studies

### Nurse 2

- Ensure ventilator is off standby
- Connect patient to ventilator after confirming ventilator settings with Anaesthetist and ICU Doctor
- Assess patient airway, ensure ETT secure and check cuff pressure
- Assess patients breathing, auscultate chest, attach ETCO<sub>2</sub> monitor
- Check ETT position and cuff pressure
- Attach suction to UWSD to 20cmH<sub>2</sub>o, check drainage, bubbling and oscillation
- Attend to NG aspirate, insert anti reflux valve and place on free drainage
- Warming device e.g. Bair hugger if temp less than 35°C
- Attend to pathology- ABG, UEC, LFT, CMP, FBC, COAG, CKMB, Troponin
- CXR
- Document observations
- Family in to visit when appropriate



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## **POST OPERATIVE OBSERVATIONS AND CARE**

All observations are attended hourly until extubation then second hourly

### **Airway**

- Assess and document position of ETT at teeth and on CXR
- Check cuff pressure each shift
- Assess patient ventilator settings which are routinely set at:
  - Mode SIMV / Pressure Support
  - FiO<sub>2</sub> 50% (Titrated to saturation above 95%)
  - TV 6 - 8m L/kg
  - RR 12bpm
  - PEEP 5cmH<sub>2</sub>O
  - PS 10
  - I. Time 1.7secs
  - Rise Time 0.2secs
  - Flow Trigger 1 L/min
- Check and set all ventilator alarms
  - ~ Airway pressure limit – 10cmH<sub>2</sub>O above pts airway pressure
  - ~ Respiratory rate – 10bpm above patient's respiratory rate
  - ~ Apnoea – alarm on, 20secs
- Check ABG on return from OT and when clinically indicated
- Perform suctioning as clinically indicated. Avoid suctioning in the first 4hrs due to increased intra thoracic pressures which could dislodge grafts
- All patients that are haemodynamically stable with no bleeding can be considered for extubation 4-6 hours post operative

### **Circulation:**

All observations attended hourly until extubated then second hourly

#### **A. Heart Rate**

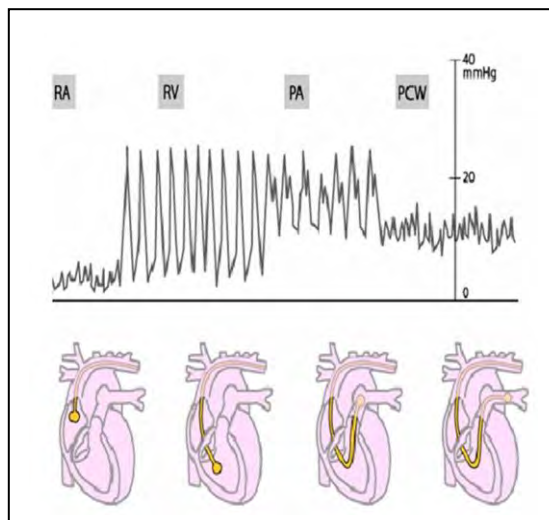
- 12 lead ECG post op, when rhythm changes and when ST segment alarms sound. If stable then daily @ 0600hrs
- Maintain heart rate 70 -90 bpm
- Check ST segment settings and ST alarms are turned on
- Monitor for arrhythmias and manage according to ICU protocols and ARC Resuscitation guidelines (see cardiac pharmacology below)
- Monitor electrolytes and replace as indicated keeping MgSO<sub>4</sub> >1.0mmol/L and K<sup>+</sup> >4.0mmol/L
- If patient is paced check connections, rate, mode, AV delay, sensitivity and output thresholds. Document these settings on pacemaker observation chart CR 155
- If patient is paced and not haemodynamically compromised an ECG can be done by turning the rate on the pacemaker down slowly then doing a 12 lead ECG to determine the patient's underlying rhythm

**B. Blood Pressure**

- Monitor systolic, diastolic and MAP via arterial line
- Maintain MAP between 70 – 90 mmhg
- Use fluid boluses, inotropes and vasopressors as indicated by patients clinical status and cardiac output studies
- May consider using anti-hypertensives or sodium nitroprusside if MAP persistently > 90mmhg
- GTN 50mg in 500mls 5% glucose at 10mls /hr used to prevent vasospasm of new grafts ( Not indicated for control of BP)

**C. Pulmonary Artery Catheter (PAC)**

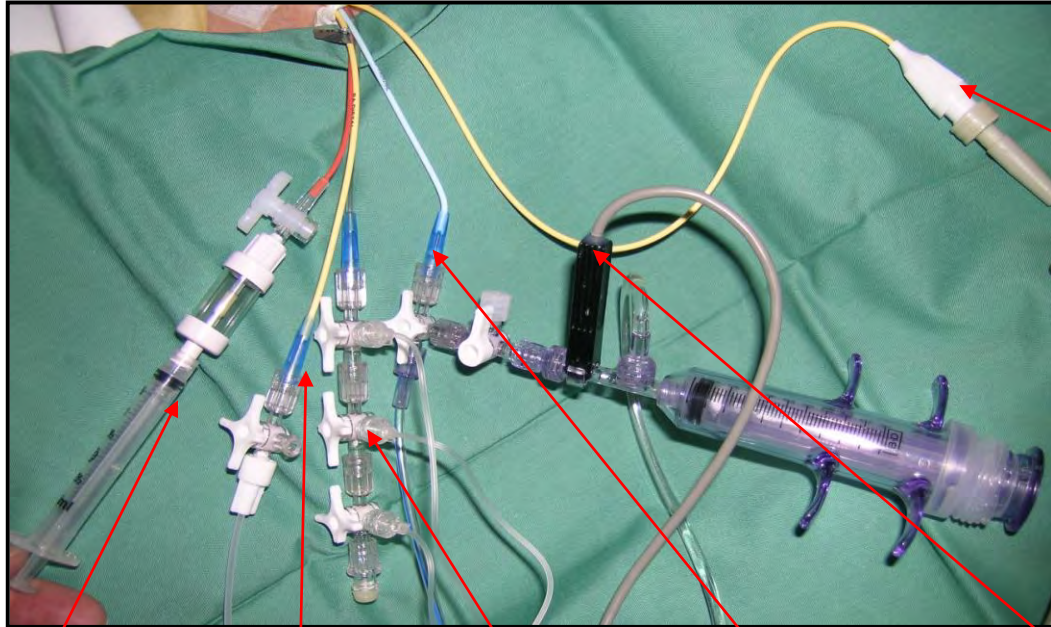
- Check position of PA catheter at insertion site and on CXR ( 1-2cm left of mediastinal border)
  - PA alarms always on: set systolic alarm 10mmhg above pts systolic and diastolic to detect catheter wedging
  - Maintain normal pulmonary pressures according to each patient as those with valve surgery or stenosis or pulmonary disease may be elevated
  - Perform PAWP as clinically indicated, to assess patients filling status
  - Perform cardiac output studies on return from OT as a baseline.
  - Repeat when there is a change in clinical status, inotropic requirements and fluid status to assess contractility, systemic vascular resistance and stroke volume
  - Secure PAC to patients chest with clear occlusive dressing
  - Regularly observe trace to ensure the catheter has not migrated and become wedged
  - If trace spontaneously wedges or will not wedge only staff who are accredited are able to “float” PAC into position
  - For further PAC information see Pulmonary Artery catheter learning package
- THE PULMONARY ARTERY CATHETER MUST BE TRANSDUCED AND MONITORED AT ALL TIMES



Characteristic intracardiac pressure waveforms of pulmonary artery catheter during passage through the heart

Anaesthesia UK 2010

## PULMONARY ARTERY CATHETER



Temperature cable for core temp

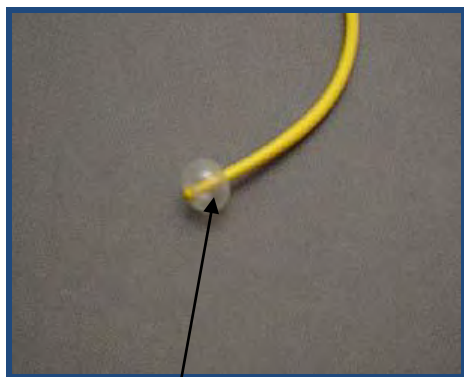
Wedging syringe: When not performing wedge procedure should be left with no air in syringe

PA port: for transducing pulmonary artery pressures

Medication port: only used for sedation when no other access

CVP port with cardiac output syringe attached

Temperature cable: measures temperature of cardiac output solution



Inflated balloon

**D. Chest Drains**

- ICC should be secured to the patient and all connections secured with brown tape
- Connect ICC to the UWSD with suction of  $-20\text{cmH}_2\text{O}$
- Observe for oscillation, bubbling and drainage
- Drainage should not exceed 100mls/hr if so inform ICU medical team and CT team
- Assess drainage for clots, if drainage ceases and patient haemodynamically unstable patient maybe tamponading. Inform ICU and CT teams
- Can be removed Day 1 provided drainage is less than 100mls over 4 hours

**E. Fluid Input**

- Maintenance fluid of 4% glucose & 0.18% sodium chloride solution & 20mmol  $\text{MgSO}_4$  at 1mL/kg/hr
- GTN (50mg/500mLs 5% glucose) at 10mLs/hr used to prevent vasospasm of the new grafts. This is weaned at 0500hrs on Day 1- 8mLs/hr at 0500hrs, 5mLs/hr at 0600hrs, 3mLs/hr at 0700hrs and off at 0800hrs
- Morphine or fentanyl infusion for analgesia, reduce rate on return from OT and titrate according to pain score
- Propofol infusion, decrease rate on return from OT.
- Hourly total of all infusions
- Administration of colloids is dependant on:
  - CVP, PAP, PAWP, CI
  - BP
  - Temperature
  - Left Ventricular function
  - Urine output

**F. Fluid Output**

- Measure and document hourly urine output should be greater than 1/2 ml/kg/hr, if not report to ICU Doctor
- NGT on free drainage, removed on extubation
- Hourly ICC output
- Perform hourly total of all fluid loss

**G. Analgesia**

- Analgesia commenced in ICU. Fentanyl 500mcg/ 50mls 0.9% sodium chloride
- Assess patients pain and sedation score and aim for score of 0-1 (RASS scale) to wake, wean and extubate
- Administer adequate analgesia to keep patient comfortable and to enable adequate deep breathing and coughing
- Once patient extubated commence PCA and oral analgesia usually panadol or endone as ordered by ICU and pain team

**H. Diet**

- NBM till extubated then ice to suck
- Day1 patient commence on clear fluid with 1500mL fluid restriction
- Day 2 commenced on low saturated fat no added salt diet and maintain fluid restriction

**I. Positioning**

- Position patient head up at least 30 degrees after CXR

- Patient may be turned 4 hours post op if stable and no bleeding
- Pressure area care 2-4<sup>th</sup> hourly, including heels
- Patient must support sternal wound when moving, coughing by folding arms across chest and limit arm movements to maintain sternal stability

#### **J. Wound Care**

- Sternal wound and graft sites dressed with hydro colloid dressing and remains intact unless oozing blood
- Bandages and drains on leg graft sites removed day1 and TEDS applied
- Dressings to CVC and PA sheath sites to remain dry and intact
- ICC drains dressing to remain dry and intact. Occlusive dressing applied to ICC sites post drain removal
- Pacing wires to be wrapped in gauze if not in use

#### **K. Intravenous Lines**

- Arterial, pulmonary artery catheter, CVC, Large bore cannulas
- All lines to be dry and intact and dress PRN second daily
- PAC medication line only used for sedation. Not to be used for inotropes
- Large bore CVC for blood products and fluid boluses
- Side arm off PAC sheath for maintenance fluid

#### **L. Medications**

- Routine post op infusions include GTN and morphine
- Protamine 100mg in 100mLs maintenance fluid over 1 hour given to all on pump patients to reverse heparin
- Antibiotics – cephazolin 1g 8<sup>th</sup> hourly until drains removed. Vancomycin 1g twice daily if in hospital patient or from another hospital
- Anticoagulation – aspirin 150mg given in evening of Day 0 for off bypass patients. On bypass patients commenced on heparin and aspirin Day 1. Warfrin commenced on valve patients Day 1 or as per surgeon
- Vasodilators – Sodium Nitroprusside (SNP) used if MAP consistently over 90mmhg to prevent dislodgment of grafts.(see cardiac pharmacology at end of package)
- Glycerin Trinitrate (GTN) 50mg in 500mLs glucose @ 10mLs/hr used to prevent vasospasm, not for BP control
- Vasopressor – noradrenaline 4mg in 50mLs glucose to maintain MAP above 70mmhg.
- Milronone is used commonly as a inodilator to increase cardiac output in patients with impaired ventricles to give a cardiac output greater than 2.0L

#### **M. Hypothermia**

- Causes post op shivering, a decrease in myocardial contractility, ECG changes, vasoconstriction and diuresis.
- Re warm patient slowly as warming too quickly may cause vasodilatation and decrease in SVR.
- Pethidine 25mg may be given to stop shivering

#### **N. Pathology**

- ABG, FBC, Coag, UEC, CKMB,Troponin done immediately post-op and results corrected as needed
- Troponin levels should be attended 8<sup>th</sup> hourly on Day 1& Day 2.

- Maintain K<sup>+</sup> above 4.0mmol/L to prevent arrhythmias and MgSO<sub>4</sub> above 1.0mmol/L to prevent atrial fibrillation
- 6-8 hourly bloods Day 1 then daily if stable
- BSL second hourly if on insulin infusion
- Mixed venous blood gas sample taken from PA lumen to assess the adequacy of the patients tissue oxygenation. Can be an early indicator that patients condition is deteriorating. Should be greater than 65% saturation

**Table 1** Limits of mixed venous oxygen saturation

SvO <sub>2</sub> >75%	Normal extraction O <sub>2</sub> supply >O <sub>2</sub> demand
75% >SvO <sub>2</sub> >50%	Compensatory extraction Increasing O <sub>2</sub> demand or decreasing O <sub>2</sub> supply
50% >SvO <sub>2</sub> >30%	Exhaustion of extraction Beginning of lactic acidosis O <sub>2</sub> supply <O <sub>2</sub> demand
30% >SvO <sub>2</sub> >25%	Severe lactic acidosis
SvO <sub>2</sub> <25%	Cellular death

Critical Care Medicine 2009**O. Physiotherapy**

- Suctioning not attended until patient 4hrs post op to prevent unnecessary coughing which may dislodge new grafts
- Suctioning then done PRN
- Patients who are haemodynamically stable and have no bleeding can be considered for extubation 4-6 hrs post-op
- Once extubated deep breathing and coughing is encouraged 2<sup>nd</sup> hourly to re-expand the lungs and prevent atelectasis
- Always encourage patient to splint sternum with towel when coughing and moving to prevent sternal breakdown
- Education on not using arms to lift themselves
- Sit out of bed and mobilise on Day1
- Teds to both legs



**POST OPERATIVE CARE****DAY 1 & DAY 2****Observations**

- On day 1, provided the patient is stable and after patient is decannulated, second hourly observations are recorded on the ICU flowchart or the ward observation chart and are as follows:
  - BP (mean, S/D)
  - HR and rhythm
  - Temperature and
  - SpO<sup>2</sup>
- Hourly recordings of urine output; blood loss/chest drainage; blood/colloid replacement; and auto-transfusion, are recorded on the ICU flowchart.

**Fluids**

- Day 1- IV fluids continue at patient's body weight.
- GTN is ceased as per pharmacology guideline
- Analgesia is changed to PCA, plus oral
- If still in ICU at midnight of Day 1, the IV maintenance fluid is ceased or decreased to 10mls/hr.
- This decrease in IV fluids is implemented only if the patient is tolerating oral fluids and their urine output is adequate.
- A fluid restriction of 1500mls is imposed on these patients for the first two days.
- Oral intake should be encouraged ensuring if oral intake increases then IV intake must be decreased.

**Chest Drains**

- Usually removed on day 1, provided the drainage is less than 100mls over a four hour period and the patient is sitting upright to promote chest drainage.
- Drains removed with 2 RNs - one pulling out the drain, the other tying the purse string suture. An occlusive dressing applied to site
- CXRs must be reviewed post drain removal by the Medical Officer.

**Decannulation**

- Routinely performed on day 1 after the Intensivist and Surgical rounds, providing the patient is haemodynamically stable
- Arterial line: removed prior to transfer to ward (day 1 or day 2) if ABGs are satisfactory and after all routine bloods have been taken.

- Pulmonary Artery Catheter/ is removed Day 1, providing the patient no longer requires monitoring. The introducer sheath and is also removed
- Triple lumen catheter: removed before the patient goes to ward unless otherwise instructed by ICU Doctor
- Left peripheral line: removed day 1 if not required for IV access.
- Right peripheral line is removed if the GTN infusion has been weaned and ceased. The analgesia infusion can be moved to the PAC sheath and piggy backed with the maintenance fluids.
- The PAC **must be TRANSDUCED and MONITORED at all times.**
- If patient has only the sheath in with an infusion going then ensure that a peripheral line is left in for IV access.
- IDC removed Day 2, 0600hrs

### **Wounds**

- Dressing to chest to remain intact with comfeel unless oozing
- Bandages to legs to be removed and re-dressed with comfeel if oozing
- Teds to both legs if patient ambulant. If patient in bed for prolonged periods use calf compressors
- Pacing wires wrapped in gauze if not in use. Removed day 2-5

### **General**

- Education of patient on the importance of physiotherapy to re-expand lungs, supporting chest when coughing and moving
- Pressure area care if patient in bed or sitting in chair
- Pt to sit out of bed Day1
- Clear fluids day 1, light diet day2, 1500mL fluid restriction

## INTERPRETING CARDIAC OUTPUT STUDIES

To understand whether to fill or use inotropes when your patient has a low blood pressure is dependant on your interpretation of the patient's haemodynamic status.

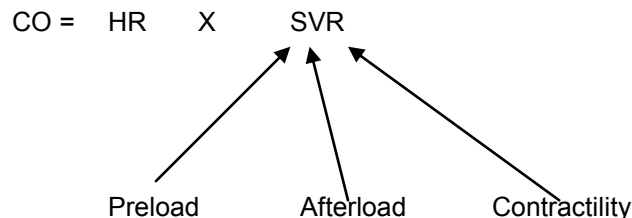
The most important haemodynamic indicator in the early postoperative period is cardiac output. No one parameter, however, should be considered or treated in isolation. Rather, all are evaluated in combination to determine appropriate therapeutic interventions. The goal is to maintain adequate systemic perfusion to protect cerebral, myocardial, and visceral function.

**Cardiac Output (CO)** ~ is defined as the amount of blood ejected from the ventricle per minute.  
~ Normal value is 3 -5 l/min  
~ Determinants are Heart Rate x Stroke volume (mls/beat)

**Cardiac Index (CI)** ~ Determined by HR, SV, Height and Weight  
~ This is a more accurate determinant of heart function as it takes into account the patient's body surface area (m<sup>2</sup>)

Heart Rate and Systemic Vascular Resistance can be manipulated to increase Cardiac Index

- The heart rate can be increased by initiating temporary epicardial pacing
- The optimal heart rate balances coronary blood flow (which takes place mainly during diastole) with cardiac output and is usually between 80 and 100 beats per minute. Normal sinus rhythm ensures atrioventricular synchrony and maximises cardiac efficiency (Salenger et al 2003)
- Stroke volume can be manipulated by fluids, inotropes vasopressors and dilators



*(Hudak, C. M., Gallo, B. M., Benz, J. J. 1990)*

**Stroke volume** is the amount of blood which is ejected from the heart with each beat. It is determined by three factors.

- ~ **Preload** of the ventricle
- ~ **Afterload** of the ventricle
- ~**Myocardial contractility**

### Preload

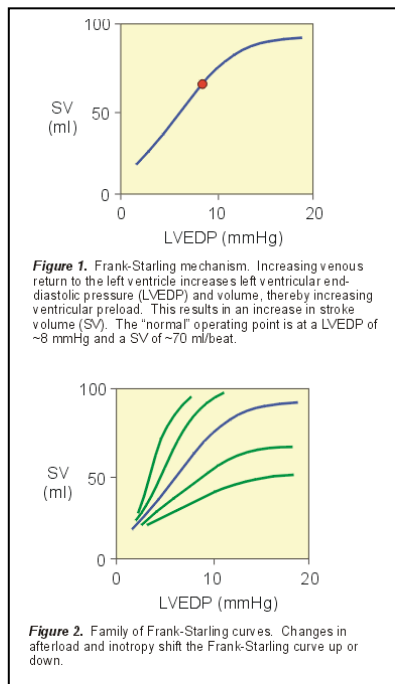
~ Is the pressure or stretching of the ventricle. It is the end-diastolic volume in the ventricle and serves as an estimation of average diastolic fibre length.

“The heart will pump what it receives” Starling’s law of the heart

The Frank-Starling mechanism describes the ability of the heart to change its force of contraction (and hence stroke volume) in response to changes in venous return. In other words, if the end diastolic volume increases, there is a corresponding increase in stroke volume.

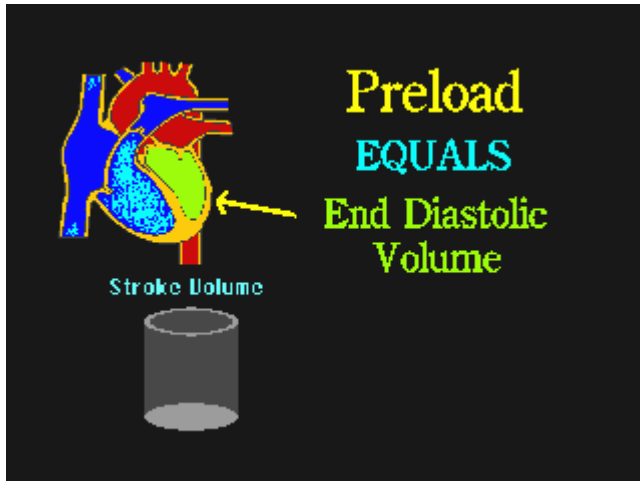
The Frank-Starling mechanism can be explained on the basis of preload. As the heart fills with more blood than usual, there is an increase in the load experienced by each muscle fibre. This stretches the muscle fibres, increasing the affinity of troponin C to  $Ca^{2+}$  ions causing a greater number of cross-bridges to form within the muscle fibres. This increases the contractile force of the cardiac muscle, resulting in increased stroke volume.

Frank Starling curves can be used as an indicator of muscle contractility (inotropy). However, there is no single Frank-Starling curve on which the ventricle operates, but rather a family of curves, each of which is defined by the afterload and inotropic state of the heart. Increased afterload or decreased inotropy shifts the curve down and to the right. Decreased afterload and increased inotropy shifts the curve up and to the left.



### Cardiovascular Physiology Concepts

Richard E Klabunde 2007

**PRELOAD = VOLUME COMING IN**

Increases with ~ hypervolaemia  
~ regurgitation of valves

Decreases in ~ hypovolaemia  
~ vasodilatation  
~ post op bleeding

Preload reflects the volume status of the patient and is measured by the PAWP via the thermodilution catheter or PA Catheter.

The preload that provides optimal cardiac output varies from each patient and is dependant on ventricular size.

**Afterload**

~ is the impedance to left ventricular contraction, is assessed by measuring systemic vascular resistance (SVR). It is the degree of constriction or dilatation of the arterial circulation.

**AFTERLOAD = RESISTANCE L) VENTRICLE MUST OVERCOME TO CIRCULATE BLOOD**

Increases with ~ hypothermia  
~ history of hypertension  
~ vasoconstriction  
~ aortic valve stenosis  
~ increase in SVR  
~ ventricular dilatation

~ High afterload increases myocardial work load and oxygen demand and decreases cardiac output

**Contractility**

~ Is the ability of the myocardial muscle fibres to shorten independent of preload and afterload. It is the ability of the heart to contract and the force at which it does so.

~ The force of contraction is determined by the concentration of calcium ions in the cells

- Increase contractility by flooding cell with more calcium (beta agonist) or by keeping more calcium in the cell and not letting it escape.

~ Mechanism that regulates cardiac output is

- The autonomic nervous system by altering the heart rate, contractility, preload and afterload.
- The parasympathetic nervous system slows the heart rate
- The sympathetic nervous system innervates the conduction system of the heart, the arterioles and veins
- Stimulation produces an increase in heart rate, contractility, preload (venous constriction) and afterload (arterial vasoconstriction).

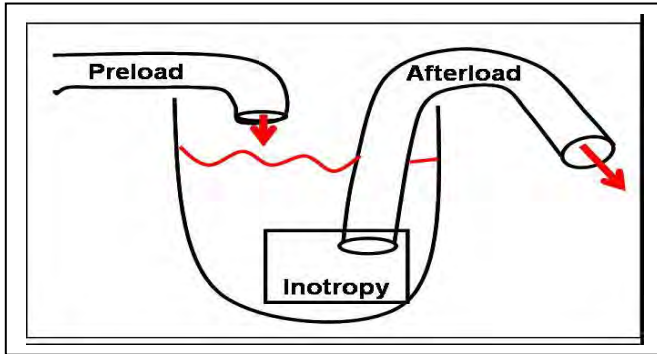
~ Ejection fraction is often used to evaluate the ability of the heart to contract

- Ejection fraction is the fraction of blood pumped out of the ventricles with each heart beat
- Normal value for a healthy person is 55-65%
- End-diastolic volume (EDV) is the volume of blood within a ventricle immediately before a contraction.
- End-systolic volume the volume of blood left in a ventricle at the end of contraction.
- Stroke volume(SV) is the difference between end-diastolic and end-systolic volumes
- Ejection fraction ( $E_f$ ) is the fraction of the end-diastolic volume that is ejected with each beat; that is, it is stroke volume (SV) divided by end-diastolic volume (EDV) (Richard E Klabunde 2007)

**~ Poor contractility may be due to:**

- Surgical manipulation of the myocardium
- Post - Cardiopulmonary bypass myocardial depression
- Cardioplegia
- Ischaemia during the aortic cross-clamping
- Myocardial infarction
- Changes in the ventricular muscle as a result of valve disease

~ Myocardial contractility is enhanced if necessary by using inotrope pharmacological agents, such as milronone, dobutamine and levosimendan. The choice of agent is individualised to the specific clinical situation.



**Summary**

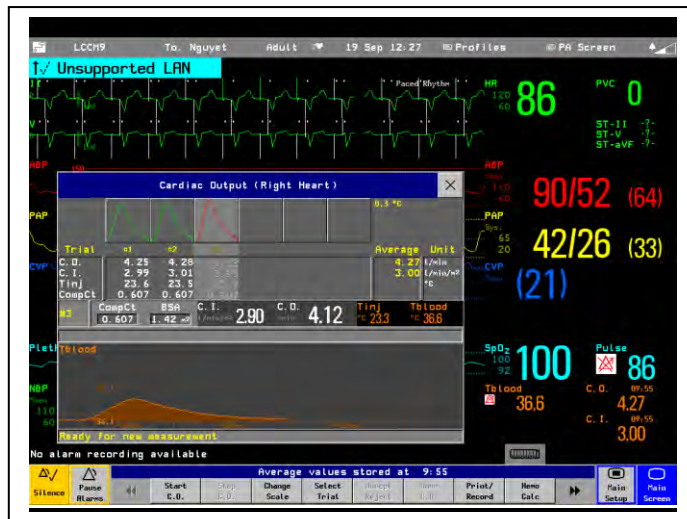
**Preload** = tap filling the heart. The faster it turned on = more constriction  $\uparrow$  preload

**Contractility** / Inotropy = Pump

**Afterload** = diameter of hose or resistance

**Performing cardiac output studies**

- The PA catheter is useful for obtaining advanced hemodynamic values
- A bolus of 10mls of dextrose fluid is injected into the proximal (CVP) lumen using a cardiac output set. This is done three times to get an average cardiac output
- As the fluid (mixed with blood) passes through the right atrium, right ventricle and then into the pulmonary artery, it's temperature changes
- The temperature probe on the end of the PA catheter measures this temperature change and calculates the patient's cardiac output

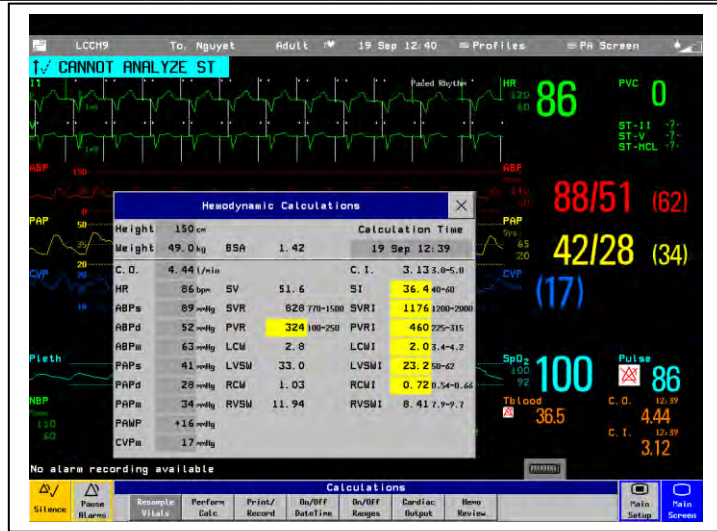


**Cardiac output screen on Phillips Monitor**

- Cardiac output studies can obtain the following advanced hemodynamic

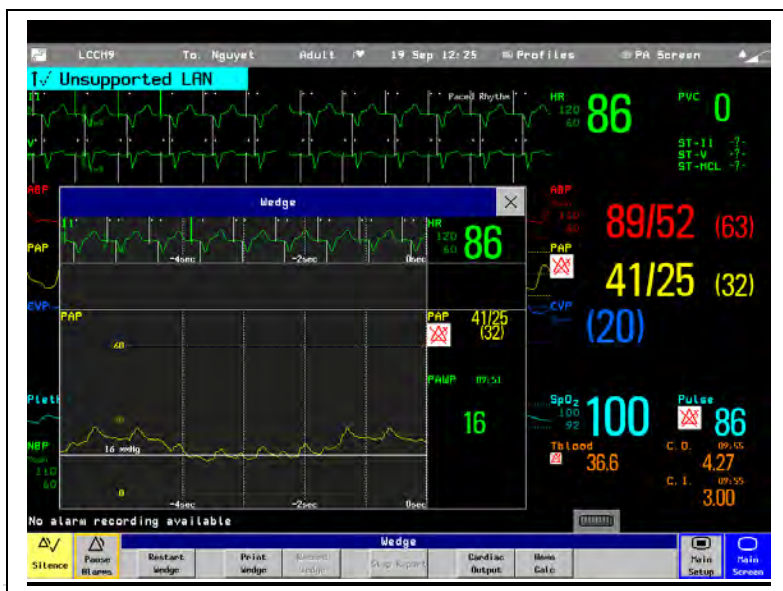
values:

- Cardiac index
- Systemic vascular resistance (SVR)
- Pulmonary vascular resistance (PVR)
- Systemic vascular resistance index (SVRI)
- Pulmonary vascular resistance index (PVRI)
- Pulmonary artery wedge pressure (PAWP)



Haemodynamic calculation screen on Phillips monitor

- These values as well as a clinical assessment are useful in determining the fluid and inotrope requirements of the patient:  
 For example a
  - ~ Low cardiac index can be managed by
    - ~ ↑ HR
    - ~ manipulate contractility with milrinone, dobutamine
    - ~ manipulate SVR
  - ~ Systemic vascular resistance (SVR) is
    - ~ How dilated or constricted the patient
    - ~ Low SVR can be managed by filling, inotropes
    - ~ High SVR can be managed by antihypertensives, sodium nitroprusside
  - ~ PAWP
    - ~ Wedge pressure is reflection of the filling of left ventricle
    - ~ Normal value depends on size of pts ventricle. A hypertrophied ventricle will need more filling than a normal size ventricle



Pulmonary artery wedge trace on Phillips monitor



## ECG INTERPRETATION

Electrocardiography is a record of the electrical activity of the heart.

- As the action potentials move through the heart they generate a positive current (depolarisation) that is immediately followed by a wave of negative current (repolarisation)
- It is these currents that the ECG detects and records

### ~ Electrode Placement

- Placement of the leads can give you positive or negative deflections and shows different areas of the heart
- 'LEAD' in ECG refers to the tracing of the voltage difference between two of the electrodes and is actually what is produced by the ECG machine

### ~Unipolar vs. bipolar leads

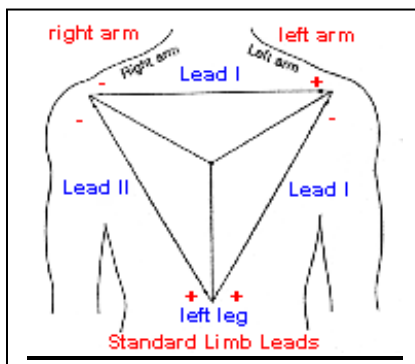
There are two types of leads: unipolar and bipolar.

~ Bipolar leads have one positive and one negative pole. In a 12-lead ECG, the limb leads (I, II and III) are bipolar leads.

~ Unipolar leads also have two poles, as a voltage is measured; however, the negative pole is a composite pole made up of signals from lots of other electrodes. In a 12-lead ECG, all leads besides the limb leads are unipolar (aVR, aVL, aVF, V<sub>1</sub>, V<sub>2</sub>, V<sub>3</sub>, V<sub>4</sub>, V<sub>5</sub>, and V<sub>6</sub>).

### ~ Types of Leads

- Standard limb leads
- Augmented leads
- Precordial leads



### Einthoven's Triangle

#### Standard Limb Leads:

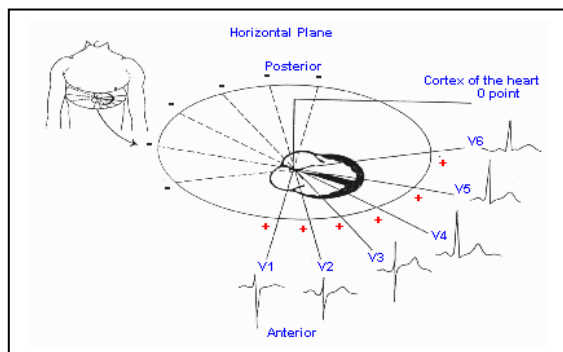
- Form the points as what is known as Einthoven's triangle
- Lead I: The positive lead is above the left breast or on the left arm and the negative lead is on the right arm. Records the difference of potential between the Left arm and Right arm.
- Lead II: The positive lead is on the left abdomen or left thigh and the negative lead is also on the right arm. Records the difference of potential between the left leg and the right arm
- Lead III: The positive lead is also on the left abdomen or left lower lateral leg but the negative lead is on the left arm. Records the difference of potential between the left leg and the right arm

### Augmented Leads

- The four limb leads go on the four extremities as follows:
- RA – on right arm avoiding bony prominences: aVR has positive (white) electrode on RA
- LA – on the left arm: aVL has positive (black) electrode on LA
- RL – on right leg avoiding bony prominences
- LL – on left leg: aVF has positive (red) electrode on LL
- Lead aVR faces the heart from the right shoulder and is oriented to the cavity of the heart.
- Lead aVL faces the heart from the left shoulder and is oriented to the Left Ventricle.
- Lead aVF face the heart from the left hip and is oriented to the inferior surface of the Left Ventricle.

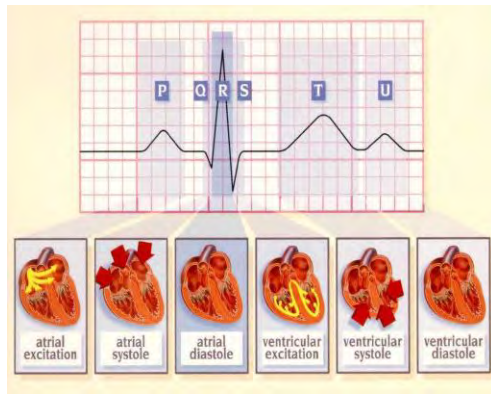
### Precordial Leads

- Six Precordial Electrode Placement: are directly on the chest
- Records potential in the horizontal plane.
- Each lead is positive.
- The major forces of depolarisation move from right to left.
- V1 and V2 are negative deflections.
- V3, V4, V5 and V6 become more positive (peak positive is V3 or V4).
- V1 - fourth intercostal, right sternal border.
- V2 - fourth intercostal, left sternal border.
- V3 - equal distance between V2 and V4.
- V4 - fifth intercostal left mid clavicular line.
- V5 - anterior axillary line, same level with V4.
- V6 - mid axillary line, same level with V4 and V5.



The precordial views make up a cross section view of the heart in a transverse horizontal plane projecting a view across the AV Node

(Mitch Medler E.M.T Paramedic. Accessed 2010)

**Electrical Representation****ECG PAPER**

- ~ Time is measured on the horizontal axis: 25mm/sec paper speed is standard
- ~ Each small square is 1mm in length & represents 0.04sec in time
- ~ Each large square is 5mm in length and represents 0.2secs in time. Five large squares = 1sec
- ~ Amplitude is measured on the vertical axis: standard is 1mv = 10mm which is two large squares: compares waveform voltage

**P wave**

- ~ represents atria excitation or contraction
- ~ Small, rounded & no taller than 2.5mm or wider than 0.11 sec (just under 3 small boxes)
- ~ Only one before each QRS

**QRS complex**

- ~ Ventricular excitation
- ~ Shape depends on which lead you are looking at
- ~ Width represents interventricular conduction time
- ~ Should be less than 0.12 sec (3 small boxes)
- ~ The *Q wave* is the first downward deflection
- ~ The *R wave* is the positive upward deflection
- ~ The *S wave* follows the *R wave* and is a downward negative deflection
- ~ Positioning of the leads affects the amplitude

**T wave**

- ~ Upright & no taller than 5mm in limb leads or 10 mm in chest leads
- ~ Represents ventricular recovery (repolarisation)
- ~ Duration is 0.1-0.2 secs
- ~ Should be slightly round and smooth
- ~ Flattening or inversion is generally indicative of myocardial ischemia
- ~ Peaked *T waves* may be due to hyperkalaemia

**Normal PR Interval**

- ~ 0.12 - 0.20 secs (3-5 small boxes)
- ~ This is measured from the beginning of the p wave to the beginning of the *QRS complex*
- ~ PR segment should be isoelectric
- ~ It includes the conduction time from the beginning of atrial excitation to the beginning of ventricular excitation

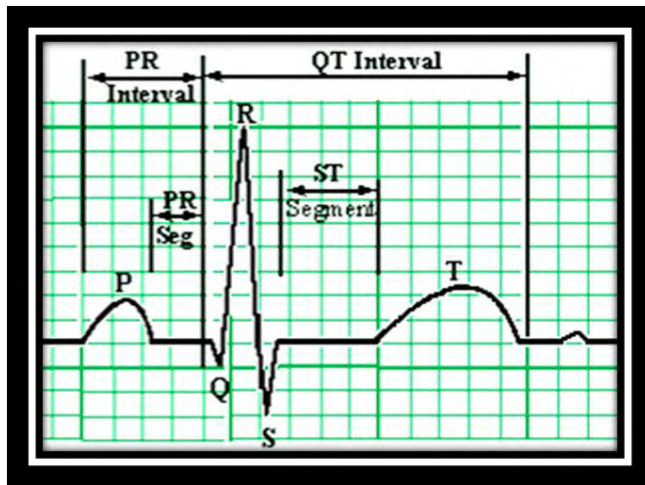
**ST Segment:**

- ~ Measured from the end of the QRS complex to the onset of the
- ~ Normally isoelectric
- ~ Normal duration is 0.24 - 0.32
- ~ ST segment depression occurs with myocardial ischaemia and elevation with acute myocardial infarction

**ECG Complex****QT Interval:**

- ~ measured from the beginning of the *QRS complex* to the end of the *T wave*
- ~ Lengthening of the interval is indicative of myocardial damage coronary ischaemia or conduction abnormalities
- ~ Normal duration is 0.35-0.42 seconds

Diagram showing segments and intervals of an ECG trace



(GMCT Rhythm Interpretation power point)

**INTERPRETATION OF MYOCARDIAL INFARCTION AND ISCHAEMIA**

Electrical current of a healthy heart should flow from negative to positive. If there is damage, injury or infarct to an area of the heart the ECG will record an abnormal flow of the electrical current. If there is an infarcted area of the heart the electrical flow will go opposite to where it is expected to flow.

**Ischemia**

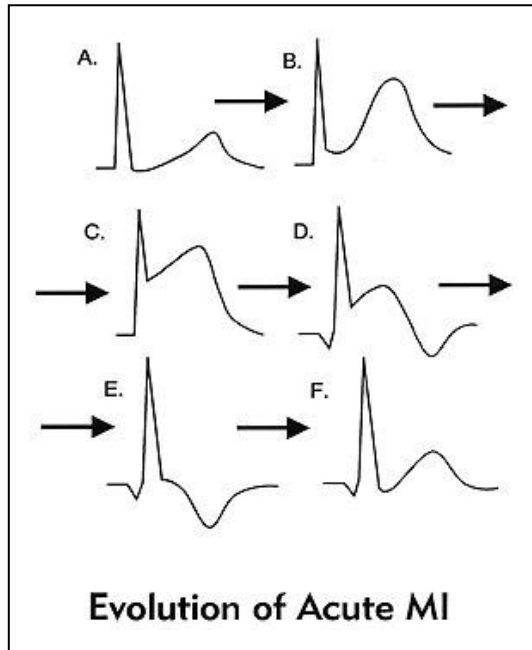
- ~ Inverted T waves

**Injury**

- ~ Elevated ST segment
- ~ signifies an acute process
- ~ More elevated the more current the injury

**Infarction**

- ~ Significant Q waves, first negative deflection
- ~ Greater than 1mm wide and 1/3 height of the entire complex

**INTERPRETATION OF MYOCARDIAL INFARCTION****Sequence of changes in acute MI**

A) Shows the normal QRS complex in a lead.

B & C) Within **hours** of the clinical onset of an MI, there is **ST segment elevation**. At this stage no QRS or T wave changes have occurred. This indicates myocardial damage only, not definitive evidence of infarction.

D) Within **days**, the R wave voltage falls and abnormal Q waves appear. This is sufficient evidence of an infarction. In addition, T wave inversion will also have appeared but the ST segment elevation may be less obvious than before.

E) Within **one or more weeks**, the ST segment changes revert completely to normal. The R wave voltage remains low and the abnormal Q waves persist. Deep, symmetrical T wave inversion may develop at this stage.

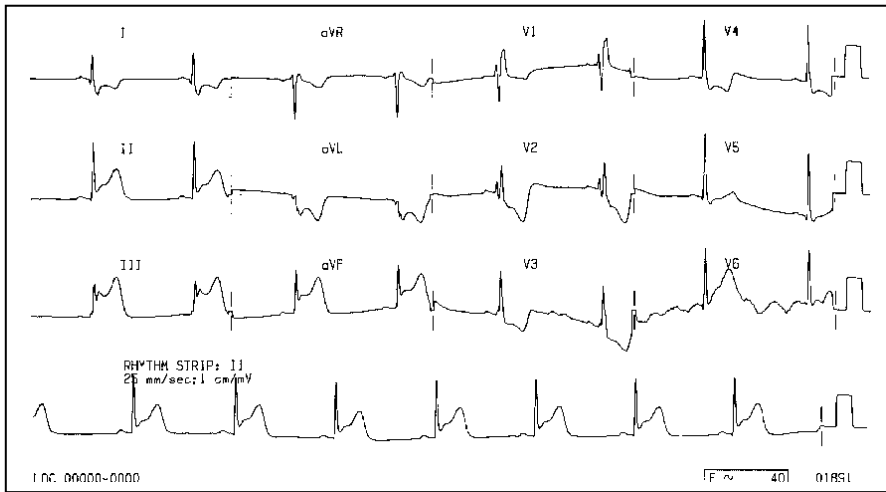
F) **Months** after the MI, the T waves may gradually return to normal. The abnormal Q waves and reduced R wave voltage persist.

Occasionally, all evidence of infarction may be lost with the passing of time; this is due to shrinkage of scar tissue.

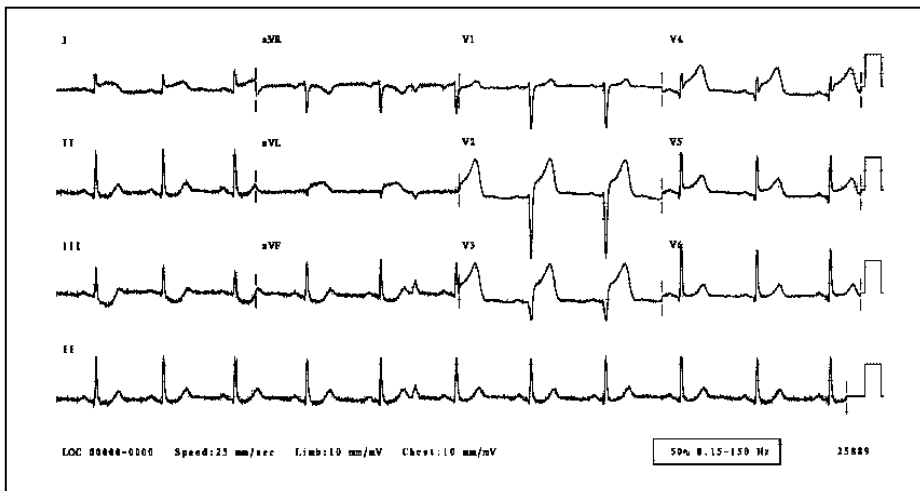
Anaesthesia UK 2010

**AREAS OF INFARCTION**

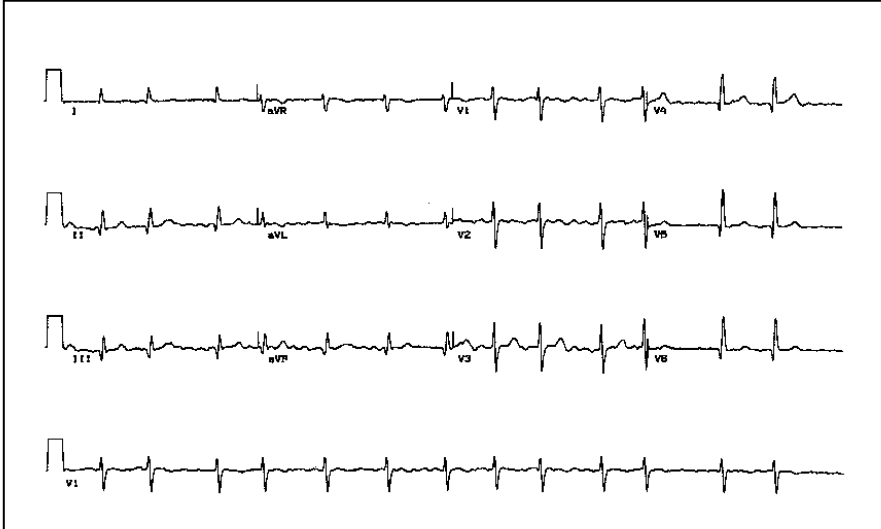
<b>AREA</b>	<b>LEADS</b>
<b>Inferior – R.C.A</b>	II, III, aVF
<b>Anterior – L.A.D</b>	V1 to V4
<b>Anterolateral – L.C.A &amp; circumflex</b>	V1 to V6 & I, aVL
<b>Lateral - circumflex</b>	V5, V6, I & aVL
<b>Posterior – R.C.A &amp;/or Circumflex</b>	V1, V2 ( mirror image)

**ECG INTERPRETATION (continued)****EXAMPLES:**ECG Library 2009**Acute inferior myocardial infarction**

- ST elevation in the inferior leads II, III and aVF
- reciprocal ST depression in the anterior leads

ECG Library 2009**Acute anterior myocardial infarction**

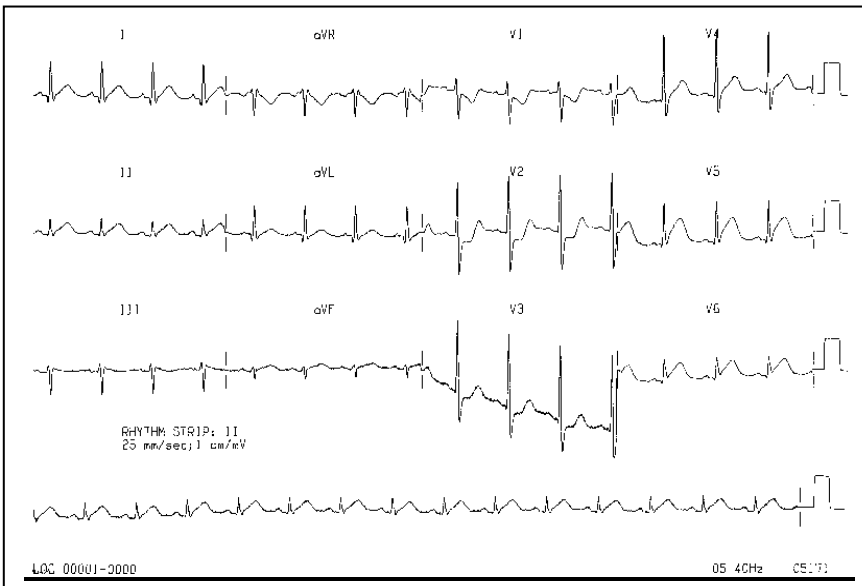
- ST elevation in the anterior leads V1 - 6, I and aVL
- reciprocal ST depression in the inferior leads



ECG Library 2009

Old inferior myocardial infarction

- a Q wave in lead III wider than 1 mm (1 small square)
- a Q wave in lead aVF wider than 0.5 mm and
- a Q wave of any size in lead II



ECG Library 2009

Acute posterior myocardial infarction

- (hyperacute) the mirror image of acute injury in leads V1 - 3
- (fully evolved) tall R wave, tall upright T wave in leads V1 -3
- usually associated with inferior and/or lateral wall MI

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## POST-OP COMPLICATIONS and CARDIAC EMERGENCIES

### Left Ventricular Dysfunction / Cardiogenic Shock / Low Cardiac Output:

#### ~ Definition

The inability of the left ventricle to sustain adequate tissue perfusion.

#### ~ Clinical findings

- Cardiac index < 2.0l/m<sup>2</sup>
- Systemic vascular resistance > 2100dynesseccm<sup>-5</sup>
- Atrial pressure >20mmHg
- Urine output <20ml/hr
- Cold, clammy extremities
- Hypotension
- Tachycardia
- Persistent obtundation

However the effects of anaesthesia, hypothermia and osmotic diuresis that occur after bypass may mask some of the early symptoms in post-operative period.

#### ~ Treatment

- Volume replacement
- Inotropic support
- Pacing
- IABP
- All of these manipulate the patient's heart rate, preload, afterload, and contractility of the heart.

### Bleeding:

#### ~ Definition

Characterised by chest drain output of > 100mls /hour

#### ~ Causes

- Post bypass coagulopathies
  - ~ Contact of the blood between the non- physiological surfaces of the bypass machine causes a decrease in the number of platelets, platelet survival and function.
  - ~ Haemodilution also reduces the level of clotting factors by approx 50%.
- Graft rupture / leaking
- Inadequate heparin reversal
- Thrombolytic therapy, platelet inhibitors used for stenting
- Impaired platelet function
- Prolonged hypothermia
- Hypertension immediately post- op
- Loosening of clips or sutures

#### ~ Sites of bleeding

- IMAs, cannulation sites, vascular anatomises, pleural or pericardial fat
- Sternal wire sites, coronary veins, edge of pericardium ( Salenger et al 2003)



**~ Signs**

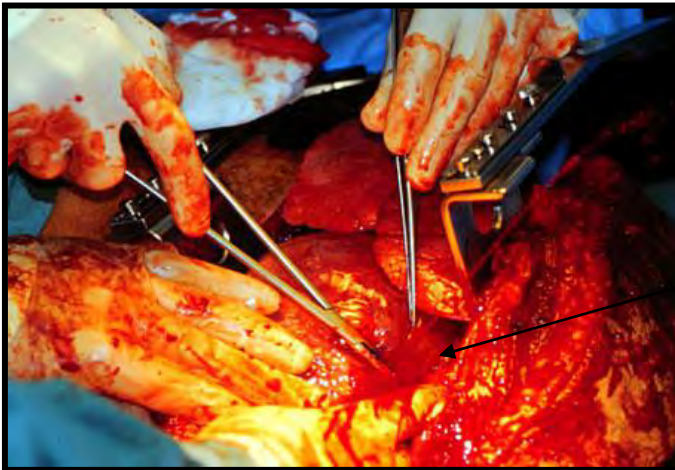
- Increase in drain output
- Tachycardia
- Hypotension
- Decrease cardiac output
- Decrease in filling pressures
- Widened mediastinum on CXR

**~ Nursing responsibilities**

- Hourly checks on chest drain, output, appearance, consistency, temperature
- If greater than 100mls/hr - inform RMO
- EUC, FBC, COAGS post op
- Monitor haemodynamic status

**~ Treatment**

- Control hypertension, MAP < 70mmHg
- Protamine
- PEEP
- Sedation
- Correct coagulopathies
- Transfusion of blood products / colloid
- Ensure clots are continuously removed from chest drains
- Decision to take patient back to OT will be influenced by the rate and amount of bleeding, and surgeons assessment



**Chest re-opened  
due to bleeding**

**Arterial bleed**

**Cardiac Tamponade:****~ Definition**

Occurs when blood accumulates in the pericardium or anterior mediastinum causing mechanical compression of the heart due to the increase in fluid in the pericardial space. This limits the normal functioning of the heart and prevents ventricular filling.

**~ Causes**

- Bleeding from chest drains, then become blocked due to clotting of drainage
- Bleeding of the myocardium post removal of pacing wires
- Thoracic aortic aneurysm

- 
- End stage lung cancer
  - Pericarditis due to bacterial or viral infections
  - Trauma to heart

**~ Clinical Findings**

- There may be sudden cessation of chest drainage after previously bleeding as the drains become blocked with clots
- Decrease MAP
- Increase CVP, PAP, PAWP
- Tachycardia
- Decrease cardiac output
- Widened mediastinum on CXR
- Neck vein distension
- Pulsus paradoxus (fall of 100mmHg in BP during inspiration)
- Cold and clammy
- Cardiac arrest
- Muffled heart sounds

**~ Nursing Responsibilities**

- 15min observations
- Early recognition of clinical findings

**~ Treatment**

- Airway, 100% oxygen
- Inotropes / colloids to maintain BP
- Return to OT
- Emergency chest re opening

Cardiac Tamponade showing enlargement of the cardiac silhouette with characteristic “water bottle” appearance



## **Emergency Sternotomy**

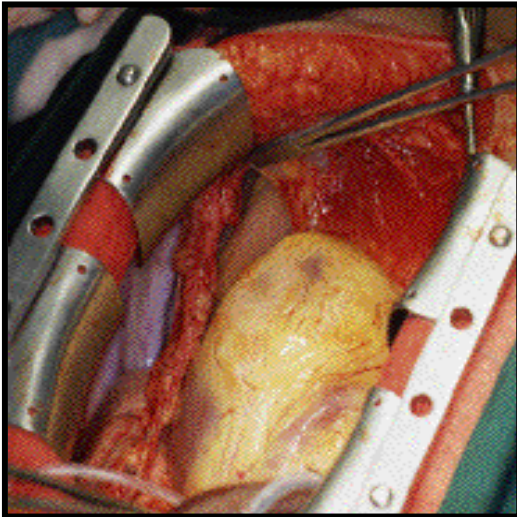
Massive sudden haemorrhage and impending or cardiac arrest may require the chest to be opened in ICU.

The purpose of this is to find the bleeding site, release the pressure in the chest from tamponade, and to improve cardiac function.

Refer to ICU guideline: Emergency Resternotomy in ICU 2015

Once the decision is made to open the chest the procedure is:

- Calling 666 and stating CTOT(Cardiothoracic operating theatre) switch then calls all staff who are on call for cardiothoracic
- Organise staff roles i.e. airway, preparation of trolley, runners
- Any one involved should wear gown, mask, gloves and protective eye wear
- Obtain arrest trolley
- Obtain chest re-opening trolley and spare trolley
- Open drapes and gowns first
- Open betadine and swabs to prepare patients skin
- Scalpel, retractor and sternal wire cutter
- Instrument tray, surgical sponges, sterile yankeur sucker and tubing
- OT count sheet is to be filled in and raytex sponges and gauze and instruments need to be accounted for.
- Internal defibrillator opened as needed



## **Hypertension:**

If left untreated it can cause rupture or leakage of suture lines and increase bleeding.

### **~ Causes**

- History of hypertension
- Increased levels of catecholamine's or rennin
- Pain
- Hypothermia

### **~ Treatment**

- Treat the underlying cause
- Sedation if blood pressure transiently high
- Sodium nitroprusside (SNP) if systolic is > 150 and /or MAP >90.
- Anti-hypertensive's

**Pericardial Effusion:**

May represent haemorrhage or an increased volume of pericardial fluid due to an inflammatory process from opening the pericardial sac.

**~ Clinical Findings**

- Dyspnoea
- Fatigue
- Pulsus paradoxus
- Unexplained hypertension
- Large effusions can cause cardiac tamponade if not treated

**~ Treatment**

- Insertion of ICC
- Physio
- Oxygen

**Coronary Artery Spasm:****~ Definition**

- Transient narrowing, either total or partial, of a large coronary artery.
- The narrowing should be sufficient to cause ischaemia
- All grafts can be partially or completely blocked off or collapse upon themselves, especially distal anatomises.
- All coronary arteries, LIMA's and radials are more prone to spasming due to
  - ~ Their unique anatomical make up,
  - ~ Irritability from handling during surgery
  - ~ Irritability from the sutures.

**~ Clinical Findings**

- Pain, nausea, diaphoresis
- Hypotensive
- tachycardic
- ECG changes – ST elevation or depression, peaked T waves, transient Q waves
- heart blocks of varying degrees
- Ventricular Tachycardia, Ventricular Fibrillation

**~ Treatment**

Aimed at preventing constriction and promoting coronary vasodilatation.

- Angiogram in cardiac cath lab
- Nitroglycerin (GTN) usually runs prophylactically, 50mgs / 500ml 5% glucose at 10mls /hr.
- Calcium Channel Blockers.
  - ~Patients post CABG that has radial artery grafts and bilateral internal mammary artery grafts (Dr Dignan) have a verapamil infusion. (See Pharmacology guideline below)

**Arrhythmias:**

Arrhythmias compromise cardiac output, which therefore decreases coronary artery perfusion and increases myocardial oxygen demand.

**Brady arrhythmias:**

Include Sinus Brady, heart blocks, idioventricular, and junctional.

Valve patients are more at risk due to the proximity of the cardiac conduction system to the mitral and aortic valves

**~ Causes**

- Occurs post op due to depression of the conduction system cells by the use of cardioplegia or injury to nodes and conduction pathway during surgery.
- Sutures and oedema
- Valve patients at risk due to the proximity of the conduction system to mitral and aortic valves

**Heart blocks**

**1<sup>st</sup> degree heart block**

- All P waves are conducted

**2<sup>nd</sup> degree Heart Block**

~ Type 1 or Mobitz I or Wenckebach

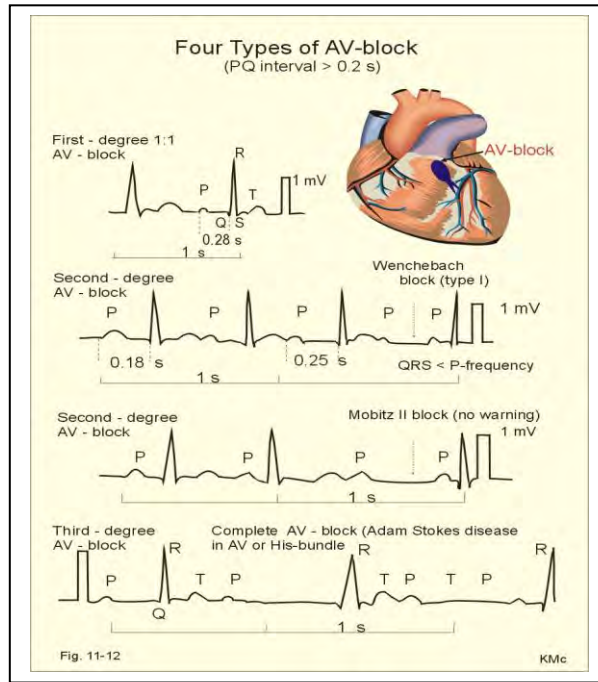
- Progressive delay of conduction of the AV node until conduction is completely blocked
- PR interval is longer with each beat until QRS is dropped

~ Type 2, Mobitz II

- 2 – 4 p waves before each QRS
- Potential to progress to 3<sup>rd</sup> degree hear block
- Ventricular rate less than atrial rate

**3<sup>rd</sup> degree Heart Block**

- No P waves are conducted



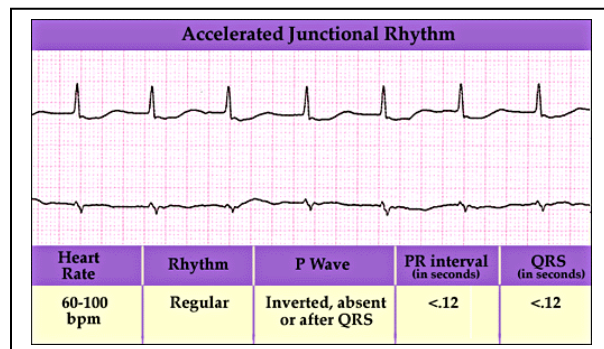
Textbook Medical Physiology 2000

**Treatment**

- Epicardial pacing wires may need to be used 24 –48hrs post op (Refer to pacing learning package)

**Junctional rhythm**

- AV node is the fastest pacemaker & so starts the impulse
- P often absent. "buried" in the QRS complex
- P waves may be upside down or after the QRS



Frank G Yanowitz MD 1997

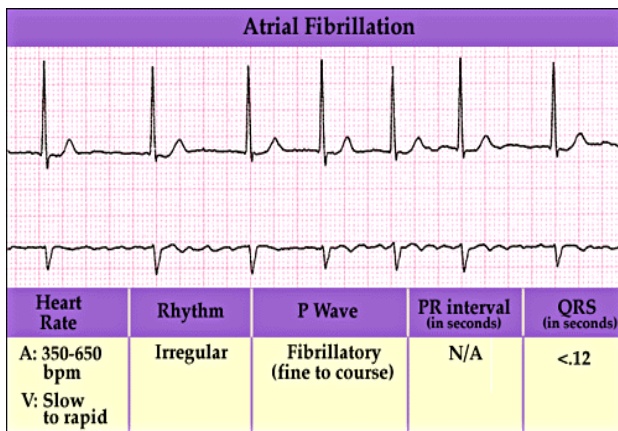
**Atrial Fibrillation**

**~ Causes**

- Very common especially in mitral valve patients due to long-standing atrial dilatation and stretching.
- Chronic AF
- Low MgSO<sub>4</sub>
- Caused by multiple re-entry circuits in atria
- Atrial trace shows fibrillation waves rather than P waves and always irregular
- Loss of atrial kick and ventricular filling

**~ Treatment**

- Amiodarone
- Check electrolyte
- Treat underlying cause



Marquette KH 1996

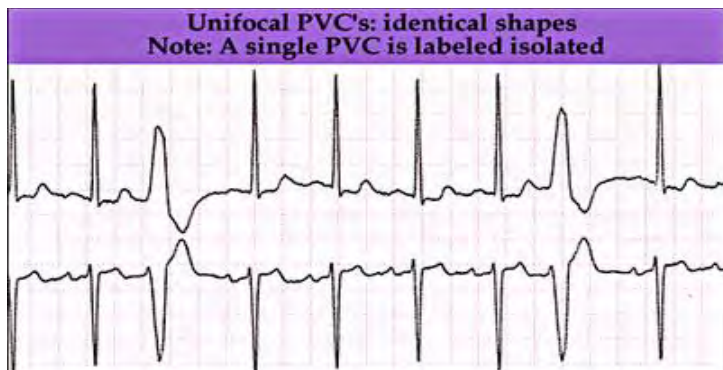
Premature Ventricular Contractions (PVC's)

**~ Causes**

- Surgical trauma and manipulation
- Electrolyte imbalance
- Changes in pH and pO<sub>2</sub>
- Pulmonary artery catheter positioning
- Chest tube placement

**~ Treatment**

- Treat underlying cause

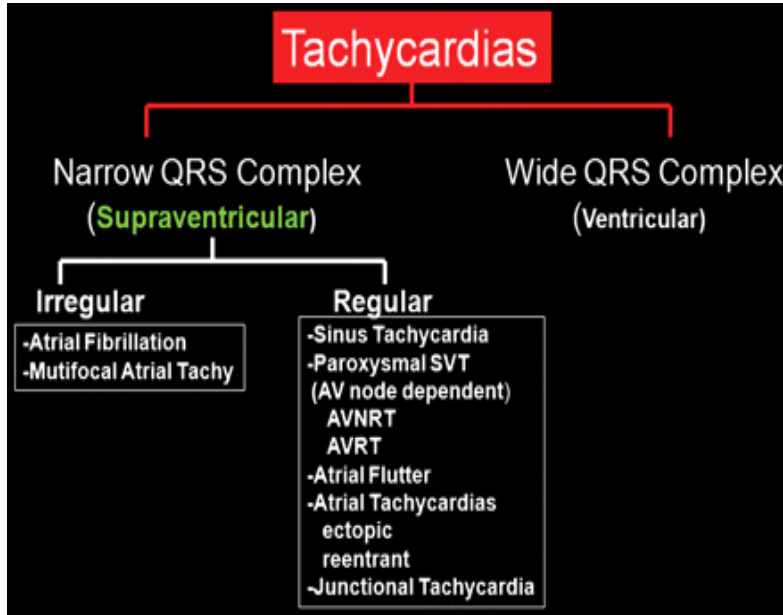


**Tachycardia's****~ Causes**

- Hypovolemia
- Catecholamine release during surgery
- Pain, anxiety, fever

**~ Treatment**

- Treat underlying cause
- Beta blockers

**Classification of Tachycardias**

Radiological Society of North America 2010

**Supraventricular tachycardia:**

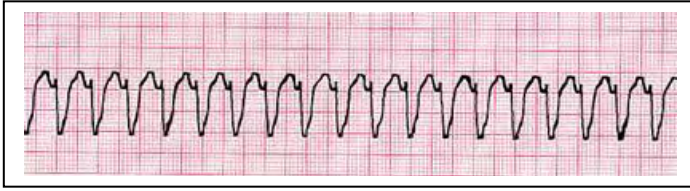
Emergency care cardiac guidelines  
Nursing CEU 2010

**~ Causes**

- Tachycardia arising from atria or AV junction
- We use it to describe fast narrow-complex tachycardias
- Usually caused by a re-entry circuit returning to the atria

**~ Treatment**

- Treat underlying cause
- Adenosine(See ICU medication protocols)
- Amiodarone
- Cardio version if haemodynamically unstable
- Check electrolytes

**Ventricular Tachycardia**

Emergency Care Cardiac Guidelines  
Nursing CEU 2010

**~ Causes**

- Due to a reentry circuit or to increased automaticity
- Associated with MI, cardiomyopathy, valvular heart disease & long QT syndrome
- Usually regular, rate greater than 100
- Wide complexes (greater than 3 small squares), best seen on V1 & V2

**~ Treatment**

- Defibrillate if unconscious
- Cardioversion if conscious & haemodynamically unstable
- Amiodarone
- Lignocaine ( See ICU Pharmacology Protocol)
- Correct electrolytes
- 4 H's (hypoxaemia, hypovolaemia, hypo/hyperthermia, hypo/hyperkalaemia)
- 4T's ( tamponade,tension pneumothorax,toxins,thrombosis)

**Renal**

Renal blood flow is decreased during bypass, blood cells are damaged, and free haemoglobin is released from red blood cell destruction. Cellular debris and free haemoglobin can damage renal tubules. This risk accelerates with increasing time on bypass and pre-existing renal dysfunction.

**~ Causes**

- Hypotension
- Vasoconstrictors
- Micro emboli
- Multiple transfusions
- Low cardiac output
- Age
- Long bypass time
- Nephrotoxic drugs
- Hypothermia

**~ Manifestations**

- Oliguria and or polyuria
- Rising urea and creatinine

**~Treatment**

- Adequate systemic blood pressure
- Diuretics
- Renal replacement therapy when indicated



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**Respiratory Failure:**

A common side effect of bypass is atelectasis and pleural effusions that can cause respiratory failure. The left lower lobes are the most common site for atelectasis due to reduced lung volume during surgery.

**~Manifestations**

- Decreased breath sounds
- Basal collapse on CXR
- Poor ABG

**~Treatment**

- Aggressive chest physio
- Adequate analgesia according to pain scale
- Early mobilisation
- CPAP
- Chest drain for effusions when indicated

**Hypothermia:**

Hypothermia is induced when the patient goes on bypass for myocardial preservation and to decrease the metabolic requirements of the patient. Moderate hypothermia (28deg to 32 deg) decreases normal oxygen requirements by approx 50% therefore providing major organs some protection against ischaemia.

**~ Manifestations**

- Cardiac depression, reduced cardiac output
- Bradycardia
- Acidosis
- Increased peripheral vasodilatation
- Shivering which increases metabolic rate, heart rate, blood pressure and peripheral vasoconstriction

**~Treatment**

- Slowly warm patient with warm blanket
- Sedatives or pethidine to control shivering
- Vasodilators if SVR is high
- Volume replacement as rewarming occurs and vascular space increases

**Fluid and Electrolyte Disturbances:**

Following bypass the total body fluid volume increases as a result of

- Haemodilution which decreases the colloid osmotic pressure
- Increased vasopressin levels
- Non pulsatile renal perfusion
- Activation of the renin-angiotensin-aldosterone mechanism

Large amounts of fluid shifts from the intravascular to the interstitial space during and up to 6 hours post bypass.

The most common electrolyte disturbance is potassium, magnesium and phosphate due to serum dilution, fluid shifts, altered cellular transport mechanisms and diuresis

**~Manifestations**

- Low CVP
- Intravascular hypovolaemia
- Low PAWP
- Low cardiac output
- Hypotension
- Reduction in renal blood flow
- hypothermia

**~Treatment**

- Replace fluids
- Check electrolytes and replace accordingly

**Neurological Deficits:**

These can include

- Transient ischemic attacks
- Cerebral vascular accidents
- Seizures
- Impairment of concentration, disorientation, periods of agitation and confusion

**~ Causes**

- Inadequate perfusion and emboli from bypass
- Prolonged bypass time
- Emboli can be due to left ventricular thrombus, or mobilisation of atherosclerotic plaque

**Psychological Disturbances:**

These can include

- Mild depression
- Disorientation
- Agitation
- Hallucinations
- Aggressive behaviour
- Psychosis

**~ Causes**

- Patients with untreated psychological disturbances
- Extreme pre op anxiety
- Sleep deprivation from long stay in ICU
- Elderly
- Alcohol abuse
- 

**~Treatment**

- Generally these complications are transient and are treated symptomatically by supporting the patient.

**Wound Complications:**

These can include

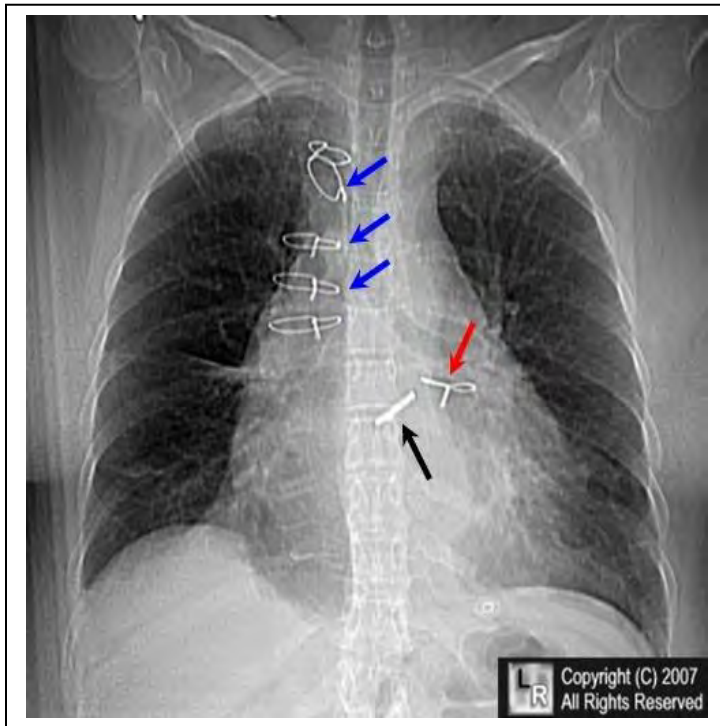
- Sternal wound complications
- Sternal dehiscence
- Superficial or deep wound infection
- Infection of graft/ leg incisions

**~ Manifestations**

- Copious purulent discharge
- Extensive cellulitis surrounding sternal incision
- Localised tenderness
- Fever
- Sternal instability
- Malaise

**~Treatment**

- Antibiotics
- If dehiscence occurs operative surgery to do sternal debridement may be necessary. This may involve plastic surgery using the Pectoralis major muscle

**Sternal dehiscence.**

Blue arrows: point to one group of sternal wires that are displaced to the right of the midline

Red arrow points to a lower wire that has travelled with the left half of the dehiscenced sternum

Black arrow points to a prosthetic aortic valve.

Normally, the sternal wires should align in the midline.

Wandering Wires: Frequency of Sternal Wire Abnormality in Patients with Sternal Dehiscence  
American Journal Of Roentgenology

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## COMPLICATIONS ASSOCIATED WITH CARDIOPULMONARY BYPASS

CPB harms patients, even while it enables life-preserving operations. In a time-dependent fashion, CPB activates plasma proteins and blood and endothelial cells. These complex reactions activate the complement, clotting, and fibrinolytic cascades and cause a bleeding tendency, micro emboli and fluid retention (Salenger et al 2003)

### A. Volume and Pressure

There are factors that lead to extra vascularisation of fluid into the interstitial compartment during both bypass and the early post-operative phase. The length of bypass is proportional to these effects.

- Haemodilution which lowers intravascular colloid osmotic pressure
- Damage to platelets and other elements secondary to contact with the pump surfaces which release vasoactive substances such as bradykinin that increases capillary wall permeability.
- Labile MAP due to third space losses and relative hypovolaemia. Requiring large amounts of filling to maintain pressures.
- Relative hypovolaemia due to dilatation as a result of rewarming.

### Treatment

- Volume management should be guided by MAP, peripheral perfusion and urine output
- Filling with colloids and/or blood products as required.

### B. Renal Function

- Can either be polyuric or oliguric during the early post-operative phase.
- Polyuria is secondary to excretion of large volumes secondary to haemodilution or may be due to mild hyperglycaemia (osmotic diuresis) secondary to CPB.
- Oliguria is seen to be generally due to hypothermia and high vasopressin levels during CPB. Hypothermia increases SVR and decreases CO. While vasopressin induces renal vasoconstriction thus decreasing blood supply to the renal bed leading to a reduction in urine output.
- Non-pulsatile flow of CPB can initiate renin, angiotensin system to cause oliguria.

### Treatment

- Fluid replacement in accordance with PAWP, BP, HR observations
- Re-warming patient

### C. Hypertension

- Increased catecholamine and renin secretion in combination with baroreceptor response to non-pulsatile blood flow leads to an increase in sympathetic tone.
- Vasoconstriction secondary to hypothermia.

**Treatment**

- Anti-hypertensive's, sodium nitroprusside, sedation

**D. Direct Cardiac Effects**

- Hypothermia leads to cardiac depression which can reduce the cardiac output and may lead to bradycardias.
- Acidosis leads to decrease in cardiac contractility and increase in peripheral vasodilatation.

**Treatment**

- Re-warm patient
- Fluid replacement in accordance with PAWP,BP and Cardiac output studies

**E. Haemolysis**

- Mechanical contact with the bypass circuit causes damage to red blood cells releasing Hb into the serum.
- Can present as decreased haematocrit or as haemoglobinuria.

**F. Bleeding**

- May be surgical in nature.
- Coagulopathy post CPB is seen due to inadequate reversal of heparin, mechanical destruction and consumption of clotting factors. This is secondary to the initiation of the clotting cascade and platelet dysfunction or clumping

**Treatment**

- Should be corrected by infusion of packed cells and platelets.
- Excessive bleeding may require chest re-opening.

**G. Ventilation**

- Lungs are collapsed during CPB leading to insufficient alveolar distension to activate surfactant causing possible alveolar collapse and secretion retention. Atelectasis is very common.
- Non perfusion of lungs during CPB causes breakdown in capillary walls which can lead to micro thrombi formation. This in turn may lead to pulmonary shunting and pulmonary oedema.

**Treatment**

- Aggressive physiotherapy
- Sit out of bed and ambulate Day 1 if stable
- May require CPAP post extubation

**H. Hyperglycaemia**

- Increased glyconeogenesis and decreased insulin secretion secondary to sympathetic response.
- Hypothermia reduces pancreatic cell release of insulin.
- Circulating insulin may adhere to bypass tubing.
- Mild hyperglycaemia is experienced.

**Treatment**

- Second hourly BSL
- Insulin infusion if BSL greater than 10mmol

**I. Electrolyte Disturbances.**

- In particular Na<sup>+</sup>, K<sup>+</sup> and Mg<sup>+</sup> due to serum dilution, fluid shifts, altered cellular transport mechanisms and diuresis.

**Treatment**

- All should be monitored, in particular K<sup>+</sup>.
- K<sup>+</sup> kept above 4.0mmol and MgSO<sub>4</sub> above 1.0mmol

**J. Cerebral Dysfunction**

- Due to inadequate perfusion and emboli.
- Emboli can be due to left ventricular thrombus, mobilisation of atherosclerotic plaque, platelets, platelet clump, and fat or air embolus.

<b>SUMMARY OF COMPLICATIONS CARDIOPULMONARY BYPASS</b>	
<b><u>CAUSE</u></b>	<b><u>EFFECT</u></b>
Haemodilution	Fluid Retention, interstitial fluid accumulation
Platelet destruction and aggregation	Thrombocytopenia
Neutrophil activation, decrease in surfactant	Pulmonary dysfunction
Embolus formation	Stroke, neurological dysfunction
Cannulation, bubble oxygenator	Air Emboli
Release of catecholamine's due to hypothermia	Hypertension
Diuresis	Hypokalaemia, hypovolaemia
Hypothermia	Myocardial depression, Oliguria,
Glucagon release	Hyperglycaemia
Systemic Heparinisation	Bleeding
Leukocyte release	Capillary leakage
Renin-angiotension activation, vasopressin release	Oliguria
Damage to red cells	Haemolysis, haemoglobinuria

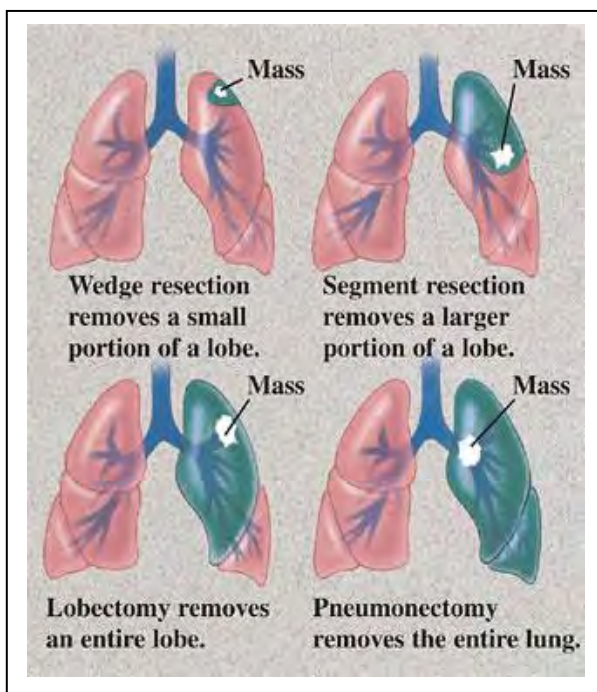
## THORACIC OR LUNG SURGERY

Lung surgery or thoracotomy is done for various reasons. It involves opening the chest wall to repair or remove part of or all of the lung tissue.

Some of the types of thoracic surgeries are

- ~ Lobectomy
- ~ Wedge resection
- ~ Segmentectomy
- ~ Pneumonectomy
- ~ Decortication
- ~ Pleurodesis

Below is a diagram explaining some of the various types of surgeries



Midwestern Cardiac Surgery 2009

### Pleurodesis

- ~ Is a procedure that is performed that causes the membrane (pleural) around the lung to stick together
- ~ It prevents build up of fluid in the spaces between the membranes
- ~ Irritants such as Bleomycin, Tetracycline or talc powder are instilled in pleural space. This causes inflammation which makes the membranes stick together

### Decortication

- ~ Is removal of the pleural layer due to the lung being covered by thick inelastic pleura which restricts lung expansion

**Post op care**

Post op thoracic patients may require to return to the ICU some may not. Usually dependant on pre-existing conditions and what procedure is performed. Mostly lobectomy and pneumonectomy patients will require High dependency care.

**Lobectomy**

~ Is the removal of an entire lobe of the lung

~ Goal of care is to maintain and optimise respiratory function

**Physiotherapy**

- High semi-fowlers
- Sit out of bed and ambulate Day 1
- Deep breathing and coughing
- Shoulder and leg exercises
- Triflow 2<sup>nd</sup> hourly

**Observations**

- Full physical assessment
- Heart rate, BP, Respiratory rate, O<sub>2</sub> saturation, air entry
- Oxygen therapy to maintain saturation above 95%

**Chest Drains**

- Hourly observations for bubbling, oscillation, drainage
- Suction at 20cmH<sub>2</sub>O
- Clamps at bedside
- Removed when ordered by surgical team

**Pathology/ x-ray**

- UEC, FBC, Coagulation (APTT, PT, INR), ABG on return, then as indicated
- CXR on return and daily

**Analgesia**

- Pain and sedation scores
- PCA observations 1hrly- 2<sup>nd</sup> hrly
- Epidural obs

**Wound Care**

- Dressing intact until day1 then daily PRN
- Observe for ooze, redness, inflammation

**Nutrition**

- IV fluids and clear fluids Day 0 then diet as tolerated.

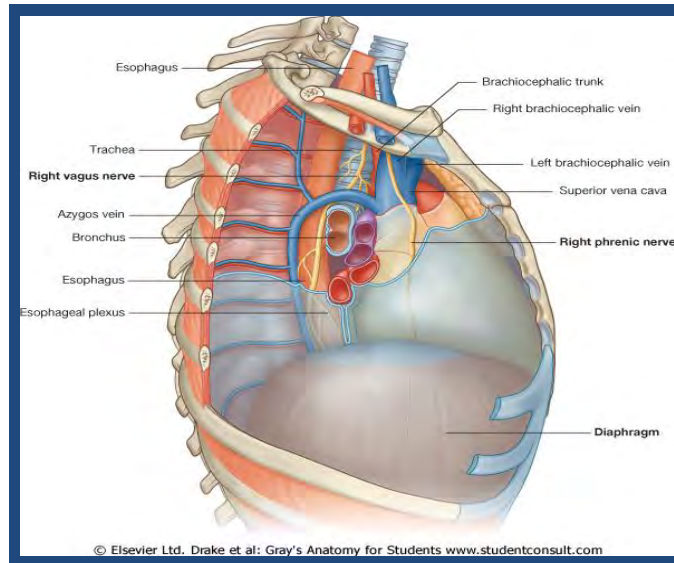


## Pneumectomy

~ is the removal of an entire lung and may involve removal of surrounding glands and lymph nodes

~ Most often used to treat

- patients with lung cancer
- Asbestos related lung disease
- Traumatic or acute injury to lungs
- Complications during CABG and pulmonary artery rupture



### Thoracic cavity

#### **Procedure**

- Performed via a thoracotomy, which is an incision that extends from below the shoulder blade along the curvature of the ribs at the front of the chest. Part of or a rib may be removed for a clearer view of the lung and ease in removing the diseased organ
- Surgeon then
  - ~ deflates the lung
  - ~ ties off the major blood vessels
  - ~ Clamps and cuts main bronchus to prevent fluid from entering the air passage
  - ~ removes the lung
  - ~ staples or sutures the end of the bronchus
  - ~ makes sure that air is not escaping from the bronchus
  - ~ inserts a chest tube between the layers of the pleura and the surgical cavity
  - ~ closes the chest



**Thoracotomy incision** (Encyclopedia of Surgery 2010)

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**Post op care****~ Airway**

- Attend full respiratory assessment- rate, depth, frequency, rise and fall of chest
- Oxygen mask according to assessment and ABG
- If requires NIV consult with ICU and CT teams

**~ Circulation**

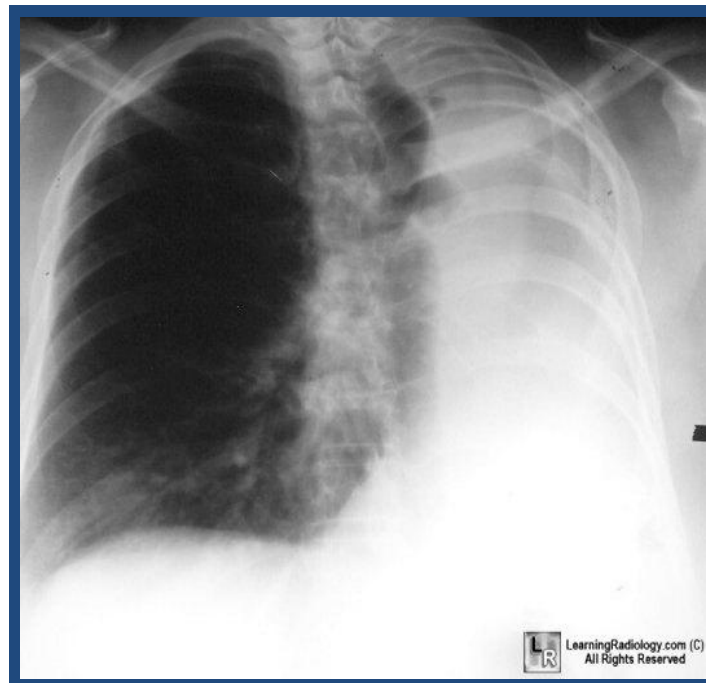
- Continuous haemodynamic monitoring
- 2<sup>nd</sup> hourly recording of Heart rate, BP, Temp, O<sub>2</sub> saturation
- Full physical assessment
- Post op 12 lead ECG, then daily or as indicated

**~ Analgesia**

- All patients to have PCA
- Some may have extra-pleural catheters with a ropivacaine infusion @ 10mls/hr. Continues for 24-48hrs until documented to cease by cardiothoracic team

**~ CXR/Pathology**

- CXR post op then daily to assess line and chest tube placements and structural changes
- Post op UEC, COAGS, FBC, Mgs<sub>04</sub>, Po<sub>4</sub>, LFTs, CKMB, then daily PRN as indicated



**Left Pneumonectomy: the left thoracic cavity will gradually fill up with fluid**

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**~ Fluids/Nutrition**

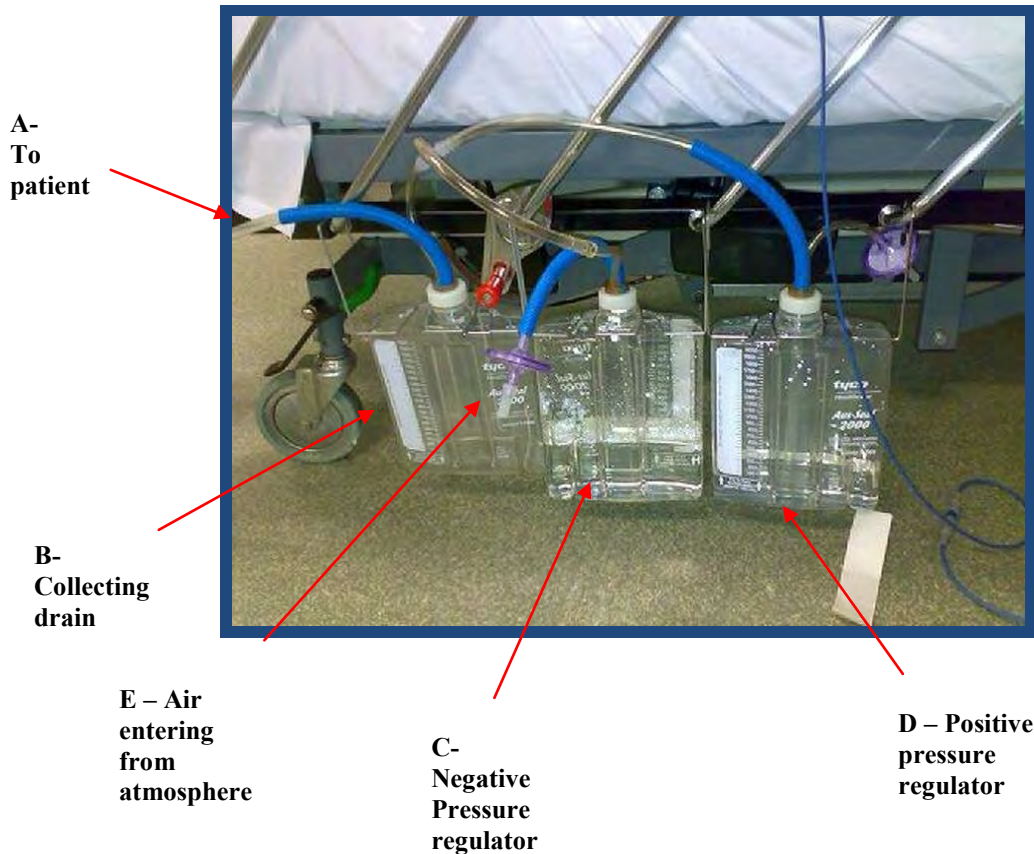
- Day 0
  - ~ NBM, IV fluids as ordered being careful not to over hydrate
- Day 1
  - ~ Left pneumonectomy – ice to suck
  - ~ Right pneumonectomy – NBM
    - NGT on free drainage, till day2
  - ~ Right pneumonectomy is likely to have respiratory compromise if they develop gastric distention.
- Day 2
  - ~ Clear fluids for left and ice to suck for right pneumonectomy
- Day 3
  - ~ Light diet for left and clear fluids for right pneumonectomy
  - ~1500ml fluid restriction
  - ~ Daily weight

**~ Chest drain management**

- Pneumonectomy patients have a balanced chest drain system which prevents mediastinal shift
- The balanced chest drain system **MUST NOT BE CONNECTED TO SUCTION**
- During early post op period , extrusion of air from the pleural space may result in mediastinal displacement toward the side of the pneumonectomy
- Retention of air in addition to fluid accumulation may result in deviation of mediastinum toward the remaining lung
- This can compromise lung function and impair venous return if there is a shift toward the remaining lung
- Deviation toward the operative side can cause dysrhythmia, hypotension, cardiac herniation and pulmonary oedema

**~ Chest drain management**

- Drain B
  - ~ collects drainage from chest
- Drain C
  - ~ If pressure in chest becomes more than 15cmH<sub>2</sub>O negative, air will move into the pleural cavity.  
This is why E needs a bacterial filter
- Drain D
  - ~ If pressure in chest becomes more than 1cmH<sub>2</sub>O positive the air will come out of chest
- E
  - ~ Bacterial filter



- **Nursing Issues**

- ~ **Never connect the system to suction**

- ~ **Do not cover air vents on Drain C and D.** They are to remain open to air at all times

- ~ Check all connections to make sure they are connected properly and air tight

- ~ Check water levels in C and D ( if different to below DO NOT change volume. Set by CT surgeon according to each patient)

- C –should be at 10 – 15 cm (negative pressure regulator bottle)

- D –should be at 1cm (water seal bottle)

- ~ **Do not clamp chest drain** as the balanced system maintains the mediastinum in the normal position while still allowing the drainage of excess post op blood or fluid

- ~ Hourly chest drain observations, oscillation, bubbling

- ~ Drain C should be the only chamber that has bubbling, if in any other chambers notify ICU team

- ~ If drainage is more than 150ml/hr notify the ICU and CT teams

(Pneumonectomy Care: Chest drain Considerations Protocol Liverpool Hospital 2005)

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**~ Physiotherapy**

- Position in high semi fowlers position to re expand lungs and for adequate gas exchange
- Deep breathing and coughing 2<sup>nd</sup> hourly
- Shoulder and leg exercises
- Sit out of bed Day 1 and ambulate as soon as possible

**~ Wound Care**

- Dressing intact till day 2 unless oozing
- Observe wound for ooze, redness, discharge

(Nursing care for patients after pneumonectomy surgery: Liverpool hospital policy 2010)

**~ Complications post Pneumonectomy**

- Mediastinal shift
- Pulmonary oedema/ effusions
- Pulmonary hypertension
- Surgical emphysema
- Pneumothorax
- Arrhythmias
- Post pneumonectomy syndrome

~ Rare complication after a pneumonectomy

~ It consists of excessive mediastinal shift resulting in compression and stretching of the tracheobronchial tree and oesophagus (European Journal of Cardiothoracic Surgery)



CT chest showing significant mediastinal shift to right with tracheal deviation after pneumonectomy

## CARDIAC PHARMACOLOGY

The following cardiac intravenous drugs are discussed in this section:

Adrenaline	Verapamil
Noradrenaline	Protamine
Dobutamine	GTN
Milronone	Levosimendan
Sodium nitroprusside	Amiodarone
Adenosine	

The following oral cardiac drugs will also be discussed

Calcium Channel blockers
Beta Blockers
ACE inhibitors

### ORAL CARDIAC PHARMACOLOGY

This section concentrates on the following classifications:

- Calcium Channel Blockers
- Beta-Blockers
- Ace Inhibitors.

#### Calcium Channel Blockers

~ are used to treat

Angina  
Hypertension  
Arrhythmias.

#### Action

- Calcium channel blockers bind to L-type calcium channels located on the vascular smooth muscle, cardiac myocytes, and cardiac nodal tissue (sinoatrial and atrioventricular nodes).
- These channels are responsible for regulating the influx of calcium into muscle cells, which in turn stimulates smooth muscle contraction and cardiac myocyte contraction.
- In cardiac nodal tissue, L-type calcium channels play an important role in pacemaker currents and in phase 0 of the action potentials.
- Therefore, by blocking calcium entry into the cell, Calcium Channel Blockers cause vascular smooth muscle relaxation (vasodilation), decreased myocardial force generation (negative inotropy), decreased heart rate (negative chronotropy), and decreased conduction velocity within the heart

(negative dromotropy), particularly at the atrioventricular node)

(Cardiovascular Pharmacology Concepts 2008)

**Examples of calcium channel blockers**

- Amlodipine (Norvasc)
- Diltiazem (Cardizem)
- Felodipine (Plendil)
- Nifedipine (Adalat)
- Verapamil (Isoptin)

**Side Effects**

- ~ Flushing
- ~ Headache
- ~ Nausea
- ~ Dizziness
- ~ Mild peripheral oedema

For more specific information, please refer to the MIMs manual

**BETA-BLOCKERS**

~ Are used to treat

- Hypertension
- Angina
- Myocardial infarction
- Arrhythmias
- Heart failure

**Action**

- Effectively reduces the myocardial stimulant, vasodilator, bronchodilator, and metabolic actions of catecholamines.
- They may be classified as nonselective or selective.
- Non-selective Beta-blockers blocks both beta<sub>1</sub> and beta<sub>2</sub> receptor sites.
- All available beta receptor blockers are effective competitive antagonists at beta receptors, but differences do occur in the properties of the individual drugs.
- Selective beta-blockers block only the Beta<sub>1</sub> receptor sites.
- Reversible competitive blocking action at beta-adrenergic receptor sites, resulting in decreased heart rate and force of contraction. Slows atrioventricular conduction, decreases plasma renin, and lowers BP.

**Examples of Beta-blockers**

- Metoprolol (Betaloc)
- Propranolol (Inderal)
- Atenolol (Tenormin)

**Side Effects**

- Drowsiness
- Light headedness
- Lethargy
- Nausea
- Bradycardia
- Bronchoconstriction
- Hypoglycaemia
- Reduced exercise capacity

**ACE INHIBITORS**

~ Ace Inhibitors are used to treat

Hypertension

Heart failure

**Action**

- Inhibits the angiotensin converting enzyme that hydrolyzes inactive angiotensin 1 to active angiotensin 2 in the plasma and lungs.
- Peripheral vascular resistance is lowered; salt and water retention is reduced.
- Increase in cardiac output
- Renal blood flow is increased.

**Examples of Ace Inhibitors**

Ramipril ( Altace)

Captopril (Capoten)

Lisinopril (Zestril)

**Side Effects**

- Tachycardia
- chest pain
- palpitations
- hypotension
- Nausea
- gastric irritation,
- diarrhoea, vomiting
- Persistent Cough
- Headache
- Renal impairment



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