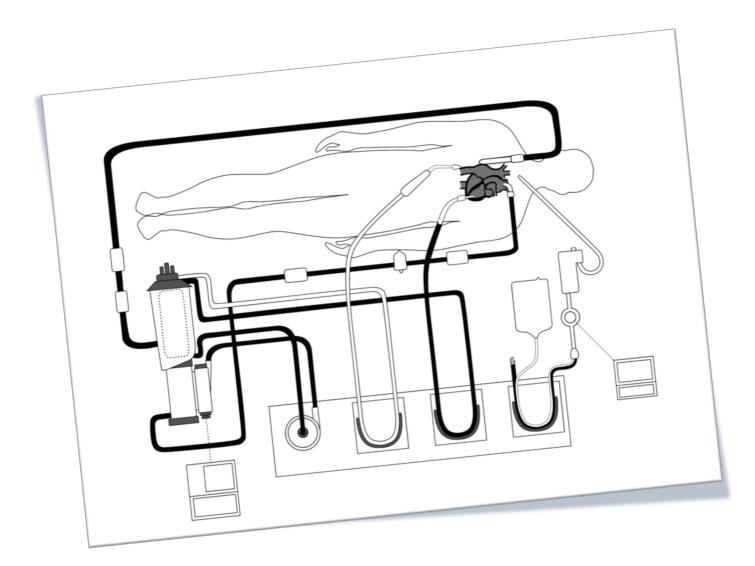


CARDIOPULMONARY BYPASS HANDOUT

Shuo Liu, MD; Ruby Feng, BA; Yanick Baribeau, BS; Robina Matyal, MD;

Ruma Bose, MD; Feroze Mahmood, MD



CARDIOPULMONARY BYPASS - SIMPLIFIED

TABLE OF CONTENTS

Section I: Anatomy And The Physiology Of Heart

Section II: Historic Development Of Cardiopulmonary Bypass

Section III: CPB Hardware

Section IV: Oxygenator And Pump

Section V: Hemodilution And Priming Solution

Section VI: Temperature And Acid Base Management

Section VII: Myocardial Protection

Section VIII: Conduction Of Cardiopulmonary Bypass

Section IX: Troubleshooting During CPB And Management

Section X: Organ Protecting During Cardiopulmonary Bypass

Section XI: Deep Hypothermic Circulatory Arrest

Section XII: Non-Cardiac Uses Of Cardiopulmonary Bypass

Section XIII: Heparin-Protamine Axis

Section XIV: Perioperative Cell Salvage

Section XV: Off Pump CABG-Role Of The Perfusionist

Section XVI: Intra-Aortic Balloon Pump

Section XVII: Ventricular Assist Devices

Section XVIII: ECMO

Section XIX: Minimally Invasive Surgeries

Section XX: Checklist

SECTION I: ANATOMY AND PHYSIOLOGY OF THE HEART

Introduction

The heart is a muscular organ that functions as the body's circulatory pump. It takes in deoxygenated blood through the veins and delivers it to the lungs for oxygenation before pumping it into the systemic arteries. The heart is located in the thoracic cavity medial to the lungs and posterior to the sternum.

On its superior end, the base of the heart is attached to the aorta, pulmonary arteries and veins, and the vena cava. The inferior tip of the heart, known as the apex, rests just superior to the diaphragm. The base of the heart is located along the body's midline with the apex pointing toward the left side.

Because the heart points to the left, about 2/3 of the heart's mass is found on the left side of the body and the other 1/3 is on the right.

Pericardium

The heart sits within the pericardial cavity. The walls and lining of the pericardial cavity are a special membrane known as the pericardium. Pericardium is a type of serous membrane that produces serous fluid to lubricate the heart and prevent friction between the ever beating heart and its surrounding organs. Besides lubrication, the pericardium serves to hold the heart in position and maintain a hollow space for the heart to expand into when it is full. The pericardium has 2 layers: a visceral layer that covers the outside of the heart and a parietal layer that forms a sac around the outside of the pericardial cavity.

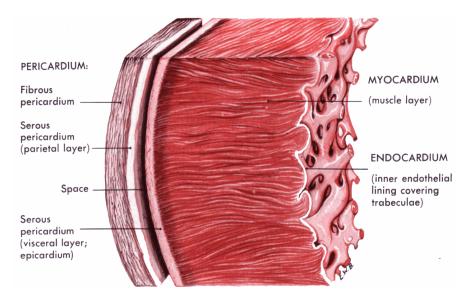
Structure of the Heart Wall

The heart wall is made of 3 layers: epicardium, myocardium and endocardium

- 1. <u>Epicardium</u> The epicardium is the outermost layer of the heart wall and is just another name for the visceral layer of the pericardium. Thus, the epicardium is a thin layer of serous membrane that helps to lubricate and protect the outside of the heart. Below the epicardium is the second, thicker layer of the heart wall, the myocardium.
- <u>Myocardium</u> The myocardium is the muscular middle layer of the heart wall that contains the cardiac muscle tissue. Myocardium makes up the majority of the thickness and mass of the heart wall and is the part of the heart responsible for pumping blood. Below the myocardium is the thin endocardium layer.
- 3. <u>Endocardium</u> Endocardium is the simple squamous endothelium layer that lines the inside of the heart. The endocardium is very smooth and is responsible for keeping blood from sticking to the inside of the heart and forming potentially

deadly blood clots.

The thickness of the heart wall varies in different parts of the heart. The atria of the heart have a very thin myocardium because they do not need to pump blood very far – only to the nearby ventricles. The ventricles, on the other hand, have a very thick myocardium to pump blood to the lungs or throughout the entire body. The right side of the heart has less myocardium in its walls than the left side because the left side has to pump blood through the entire body while the right side only has to pump to the lungs.

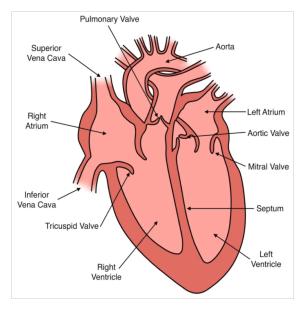


Section of heart showing layers of heart wall

Chambers of the Heart

The heart contains 4 chambers: the right atrium, left atrium, right ventricle, and left ventricle. The atria are smaller than the ventricles and have thinner, less muscular walls than the ventricles. The atria act as receiving chambers for blood, so they are connected to the veins that carry blood to the heart. The ventricles are the larger, stronger pumping chambers that send blood out of the heart and are connected to the arteries that carry blood away from the heart.

The chambers on the right side of the heart are smaller and have less myocardium in their heart wall when compared to the left side of the heart. This difference in size between the



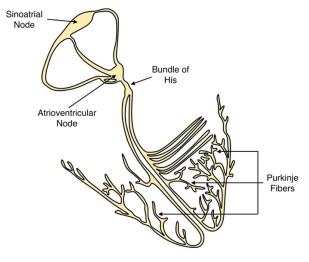
Anatomy of the Human Heart

sides of the heart is related to their functions and the size of the 2 circulatory loops. The right side of the heart maintains pulmonary circulation to the nearby lungs while the left side of the heart pumps blood all the way to the extremities of the body in the systemic circulatory loop.

Valves of the Heart

The heart functions by pumping blood both to the lungs and to the systems of the body. To prevent blood from flowing backwards or "regurgitating" back into the heart, a system of one-way valves is present in the heart. The heart valves can be broken down into two types: atrioventricular and semilunar valve.

- 1. <u>Atrioventricular valves</u> The atrioventricular (AV) valves are located in the middle of the heart between the atria and ventricles and only allow blood to flow from the atria into the ventricles. The Av valve on the right side of the heart is called the tricuspid valve because it is made of three cusps (flaps) that separate to allow blood to pass through and connect to block regurgitation of blood. The Av valve on the left side of the heart is called the mitral valve or the bicuspid valve because it has two cusps. The Av valves are attached on the ventricular side to tough strings called chordae tendineae. The chordae tendineae pull on the Av valves to keep them from folding backwards and allowing blood to regurgitate past them. During the contraction of the ventricles, the Av valves look like domed parachutes with the chordae tendineae acting as the ropes holding the parachutes taut.
- 2. <u>Semilunar valves</u> The semilunar valves, so named for the crescent moon shape of their cusps, are located between the ventricles and the arteries that carry blood away from the heart. The semilunar valve on the right side of the heart is the pulmonary valve, so named because it prevents the backflow of blood from the pulmonary trunk into the right ventricle. The semilunar valve on the left side of the heart is the aortic valve, named for the fact that it prevents the aorta from



Conduction system of heart

regurgitating blood back into the left ventricle. The semilunar valves are smaller than the AV valves and do not have chordae tendineae to hold them in place. Instead, the cusps of the semilunar valves are cup shaped to catch regurgitating blood and use the blood's pressure to snap shut.

Conduction System of the Heart

The heart is able to both set its own rhythm and to conduct the signals necessary to maintain and coordinate this rhythm throughout its structures. About 1% of the cardiac muscle cells in the heart are responsible for forming the conduction system that sets the pace for the rest of the cardiac muscle cells. The conduction system starts with the pacemaker of the heart-a small bundle of cells known as the sinoatrial (SA) node. The SA node is located in the wall of the right atrium inferior to the superior vena cava. The SA node is responsible for setting the pace of the heart as a whole and directly signals the atria to contract. The signal from the SA node is picked up by another mass of conductive tissue known as the atrioventricular (AV) node.

The AV node is located in the right atrium in the inferior portion of the interatrial septum. The AV node picks up the signal sent by the SA node and transmits it through the atrioventricular (AV) bundle. The Av bundle is a strand of conductive tissue that runs through the interatrial septum and into the interventricular septum. The Av bundle splits into left and right branches in the interventricular septum and continues running through the septum until they reach the apex of the heart. Branching off from the left and right bundle branches are many Purkinje fibers that carry the signal to the walls of the ventricles, stimulating the cardiac muscle cells to contract in a coordinated manner to efficiently pump blood out of the heart.

Coronary Systole and Diastole

At any given time the chambers of the heart may found in one of two states:

- 1. <u>Systole</u> During systole, cardiac muscle tissue is contracting to push blood out of the chamber.
- <u>Diastole</u> During diastole, the cardiac muscle cells relax to allow the chamber to fill with blood. Blood pressure increases in the major arteries during ventricular systole and decreases during ventricular diastole. This leads to the 2 numbers associated with blood pressure--systolic blood pressure is the higher number and diastolic blood pressure is the lower number. For example, a blood pressure of 120/80 describes the systolic pressure (120) and the diastolic pressure (80).

The Cardiac Cycle

The cardiac cycle includes all of the events that take place during one heartbeat. There are 3 phases to the cardiac cycle: atrial systole, ventricular systole, and relaxation.

 <u>Atrial systole</u> – During the atrial systole phase of the cardiac cycle, the atria contract and push blood into the ventricles. To facilitate this filling, the AV valves stay open and the semilunar valves stay closed to keep arterial blood from re-entering the heart. The atria are much smaller than the ventricles, so they only fill about 25% of the ventricles during this phase. The ventricles remain in diastole during this phase.

- <u>Ventricular systole</u> During ventricular systole, the ventricles contract to push blood into the aorta and pulmonary trunk. The pressure of the ventricles forces the semilunar valves to open and the AV valves close. This arrangement of valves allows for blood flow from the ventricles into the arteries. The cardiac muscles of the atria repolarize and enter the state of diastole during this phase.
- 3. <u>Relaxation phase</u> During the relaxation phase, all 4 chambers of the heart are in diastole as blood pours into the heart from the veins. The ventricles fill to about 75% capacity during this phase and will be completely filled only after the atria enter systole. The cardiac muscle cells of the ventricles repolarize during this phase to prepare for the next round of depolarization and contraction. During this phase, the Av valves open to allow blood to flow freely into the ventricles while the semilunar valves close to prevent the regurgitation of blood from the great arteries into the ventricles.

Blood Flow through the Heart

Deoxygenated blood returning from the body first enters the heart from the superior and inferior vena cava. The blood enters the right atrium and is pumped through the tricuspid valve into the right ventricle. From the right ventricle, the blood is pumped through the pulmonary semilunar valve into the pulmonary trunk.

The pulmonary trunk carries blood to the lungs where it releases carbon dioxide and absorbs oxygen. The blood in the lungs returns to the heart through the pulmonary veins. From the pulmonary veins, blood enters the heart again in the left atrium.

The left atrium contracts to pump blood through the bicuspid (mitral) valve into the left ventricle. The left ventricle pumps blood through the aortic semilunar valve into the aorta. From the aorta, blood enters into systemic circulation throughout the body tissues until it returns to the heart via the vena cava and the cycle repeats.

The Electrocardiogram

The electrocardiogram (ECG) is a non-invasive device that measures and monitors the electrical activity of the heart through the skin. The EKG produces a distinctive waveform in response to the electrical changes taking place within the heart.

The first part of the wave, called the P wave, is a small increase in voltage of about 0.1mV that corresponds to the depolarization of the atrial during atrial systole. The next part of the ECG wave is the QRS complex, which features a small drop in voltage (Q), a large voltage peak(R), and another small drop in voltage (S). The QRS complex corresponds to the depolarization of the ventricles during ventricular systole. The atria also repolarize during the QRS complex, but have almost no effect on the ECG because they are so much smaller than the ventricles.

The final part of the ECG wave is the T wave, a small peak that follows the QRS complex. The T wave represents the ventricular repolarization during the relaxation phase of the cardiac cycle. Variations in the waveform and distance between the waves of the ECG can be used clinically to diagnose the effects of heart attacks, congenital heart problems, and electrolyte imbalances.

Heart Sounds

The sounds of a normal heartbeat are known as "lubb" and "dupp" and are caused by blood pushing on the valves of the heart. The "lubb" sound comes first in the heartbeat and is the longer of the two heart sounds. The "lubb" sound is produced by the closing of the Av valves at the beginning of ventricular systole. The shorter, sharper "dupp" sound is similarly caused by the closing of the semilunar valves at the end of ventricular systole. During a normal heartbeat, these sounds repeat in a regular pattern of lubb-dupp-pause. Any additional sounds such as liquid rushing or gurgling indicate a structure problem in the heart. The most likely causes of these extraneous sounds are defects in the atrial or ventricular septum or leakage in the valves

Cardiac Output

Cardiac output (Co) is the volume of blood being pumped by the heart in one minute. The equation used to find cardiac output is:

Co= Stroke Volume x Heart Rate

Stroke volume is the amount of blood pumped into the aorta during each ventricular systole, usually measured in milliliters. Heart rate is the number of heartbeats per minute. The average heart can push around 5 to 5.5 liters per minute at rest.

SECTION II: HISTORICAL DEVELOPMENT OF CARDIOPULMONARY BYPASS

The world's first heart hospital was developed in the University of Minnesota with an 80-bed facility in 1951.

In 1952, John Lewis successfully closed a secundum ASD using inflow stasis and moderate hypothermia.

By 1955, Lewis reported 33 ASD closed at 12% mortality rates.

Gross performed ASD closure using blind atrial well technique. Mortality rate was 30%.

Cardiac surgery has widely been regarded as one of the most important medical advances of the twentieth century. The concept of a CPB machine arose from the *technique of "cross circulation"* in which the arterial and venous circulations of mother and child were connected by tubing in series. The mother's heart and lungs maintained the circulatory and respiratory functions of both, whilst surgeons operated on the child's heart (*Dr Walton Lillehei*, Minnesota, 1953). Modern CPB machines have evolved to incorporate monitoring and safety features in their design.

John Gibbon (Philadelphia, 1953) is credited with developing the *first mechanical CPB system*, which he used when repairing an atrial secundum defect (ASD). On May 6, 1953, Gibbon performed first ASD closure on cardiopulmonary bypass using film oxygenator in 18-year-old patient named *Cecelia Bavolek*.

Initially, the technology was complex and unreliable and was therefore slow to develop. The equipment used in a typical extracorporeal circuit has advanced rapidly since this time and although circuits vary considerably among surgeons and hospitals, the basic

Electrical Activity	Graphic Depiction	Associated Pattern
Atrial Depolarization		P Wave
Delay at AV Node	-01-	PR Segment
Ventricular Depolarization		QRS Complex
Ventricular Repolarization	-10-	T Wave
No electrical activity	-1-0	Isoelectric Line

ECG wave patterns

concepts are essentially common to all CPB circuits.

Cooley coined the term cardiopulmonary bypass

Azygous Flow Principle

Earlier, it was thought that minimum flow for CPB should be 100-165 ml/kg/min in animals and human. However, during some canine experiments, it was discovered that if azygous vein is not clamped and the cavae are temporarily occluded, the resultant very small cardiac output (8-14 ml/ kg/min) was sufficient to sustain vital organ functions for 30 minutes. This brought the concept of controlled cross circulation in 1954. Controlled refers to use of *sigmamotor pump* to precisely control balance of the volume of blood flowing into and out of the donor and the patient.

Lillehei performed 45 surgeries by controlled cross circulation. The thought of taking healthy donors to operating room was criticized and even considered immoral by some. However, there was no donor mortality and no long lasting donor sequelae.

Cross circulation was successful because the donor automatically corrected all the various hematologic and metabolic derangements. This changed the concept of *sick human heart theory.*

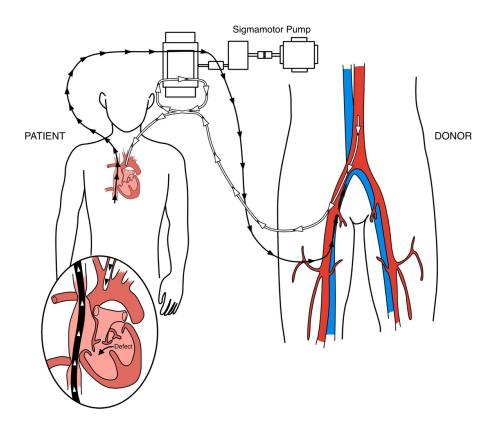


Fig-Controlled cross circulation using sigma motor pump

Evolution Of Oxygenators

- 1. Mayo clinic Gibbon oxygenator
- 2. De Wall and Lillehei Bubble oxygenator
- 3. Kay cross Rotating disc film oxygenator
- 4. Kolff and Berk membrane oxygenator

Hemodilution

It was realized that pump oxygenators could be primed with non-blood solutions. This improves quality of perfusion by reduction in viscosity and safety by reducing foreign blood.

Progress In Pump Design

- Sigmamotor pump
- Roller pump
- Centrifugal pump

History Of Cardiopulmonary Bypass

<u>Charles-Édouards Brown-Séquard</u> pointed out that the success of perfusion of blood depends on the use of oxygenated blood. He observed that rigor mortis of guillotined criminals disappear temporarily when they are perfused with their own blood.

<u>Bubbling method</u> – Waldemar Von Schroder introduced this technique of oxygenation of blood. When bubbles of gas is passed through blood, the bubbles are surrounded by a thin layer of blood that in turn absorb oxygen, give off carbon dioxide, and then burst and leave the blood free of gas. However foaming of blood and gas embolism were main difficulties.

<u>Filming method</u> – described by Max von Frey and Max Gruber. A thin film of blood is exposed to oxygen that provides a gas exchange.

Patterson and starling – oxygenated the blood with animal's own lung.

<u>Charles Lindbergh</u> – was appointed to work in Carrel's institute. Gave details of heart pump. However, he neglected an important issue of oxygenating the blood.

Very shortly, John Gibbon walked onto stage.

John Gibbon was born in Philadelphia. He wanted to become poet and writer.

He completed his medical education in Jefferson Medical College of Philadelphia. He was the sixth generation doctor in his family. After completing medical education, he worked as a fellow in Churchill's lab in Boston. He married his technical assistant, Mary Hopkinson.

On October 3, 1930, at Massachusetts General Hospital in Boston, a female patient, following uncomplicated cholecystectomy, suddenly developed discomfort in her chest and was diagnosed to have pulmonary embolism. John Gibbon was given the task of monitoring the vitals of the patient.

The complication ended fatally. But this incident gave birth to an idea in the development of heart lung machine - a machine that would remove deoxygenated blood from patient, put oxygen into that blood, allow carbon dioxide to escape from it, and then inject oxygenated blood to patients arteries.

John started working on his idea. In 1939, he reported in the American Association of Thoracic Surgery an indefinite survival of cats in good condition is achieved after a period of total CPB. His work was discontinued in World War II. In 1945, Thomas Watson, chairman of IBM helped Gibbon in his work. A larger machine with a rotating drum was made. The experiments yielded better results. It was observed that, turbulence of flow could increase oxygenation as much as eight times. Using the screen for the oxygenator could produce the desired turbulence. A device suitable for human use was then constructed. It took 23 years to bring the idea into reality.

On May 6, 1953, 18-year-old Cecelia Bavolek became John's patient. She was diagnosed as a case of atrial septal defect. Gibbon decided to operate her. Her heart was exposed through bilateral fourth intercostals incision. Digital exploration of the atrium revealed a defect the size of a silver dollar. Left subclavian artery was cannulated, as SVC and IVC total bypass was instituted. The defect was closed directly with continuous suture. The atrial incision was closed. The extracorporeal circuit had been in place for 45 minutes. Patient made an uneventful recovery.

Important Contributions Of Gibbon In CPB

- 1. Rinsing of the circuit before CPB
- 2. Use of colloid for priming solutions
- 3. Small priming volume to reduce Hemodilution
- 4. Importance of measuring O2 saturation of venous blood to assess tissue perfusion
- 5. Systemic pressure to be maintained at least 50-65 mm Hg
- 6. Perfusate and blood trapped in the CPB should be salvaged and returned to patient
- 7. His equipment included safety devices for shutting off the pump automatically in case the blood in the reservoir reached too low a level or line pressure became too high.
- 8. He experimented with pulsatile flow, incorporated a device for arterial filtration, and used plastic tubing
- 9. He demonstrated successful surgical procedures in heparinized subjects
- 10. He described the phenomenon of heparin rebound, noted that protamine could

produce hypotension and advocated use of myocardial temperature probes.

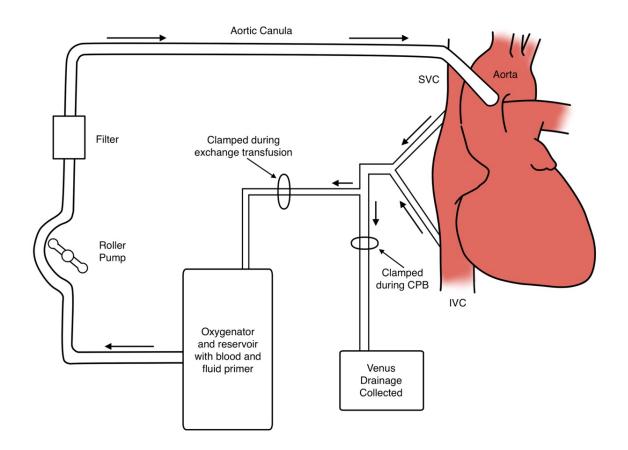
- 11. He considered heparin/ protamine titration test after CPB, once it became available
- 12. He knew that amount of hemolysis was related to length of CPB
- 13. He was aware the necessity of an assistant who could give an undivided attention to CPB, the forerunner of perfusionist today.

SECTION III: CPB HARDWARE

Cardiopulmonary Bypass

Basic components of CPB

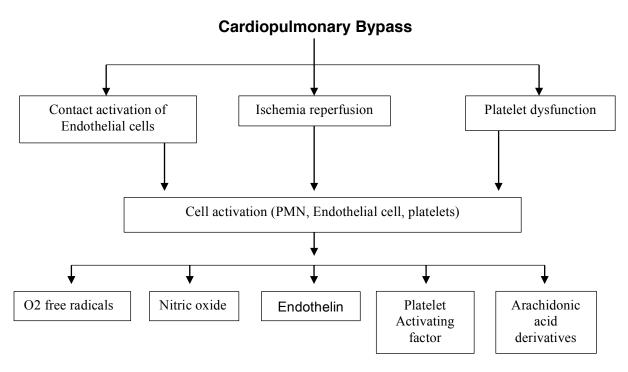
- Venous cannula
- Cardiotomy suction pump
- Venous reservoir that collects blood by gravity
- Oxygenator
- Heat exchanger
- Perfusion pump
- A blood filter in arterial line
- Arterial cannula
- Cardioplegia system



Pathophysiology Of CPB

It relates to

- 1. Blood contact with the biomaterial surface
- 2. Cardiotomy suction
- 3. Effect of non-pulsatile flow
- 1. Initiates intrinsic coagulation pathway through activation of factor XII
- 2. Kallikrein activates fibrinolytic system
- 3. Bradykinin is a key mediator of increased capillary permeability and. development of tissue edema
- 4. Activates complement system \rightarrow recruitment of leukocytes
- 5. Release of cytokines → myocardial dysfunction



Pathophysiology of Cardiopulmonary Bypass

Platelet dysfunction in CPB is due to

- 1. Hypothermia
- 2. Hemodilution
- 3. Cardiotomy suction
- 4. Platelet aggregate formation
- 5. Formation of platelet leukocyte complexes
- 6. Platelet adhesion to artificial surfaces leads to platelet consumption during CPB

Various emboli produced during CPB are

- 1. Fibrin
- 2. Platelet aggregates
- 3. Neutrophilic RBC debris
- 4. Gaseous
- 5. Spallated materials

Strategies used to decrease systemic inflammatory response to CPB

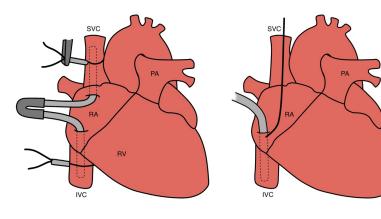
- 1. Optimal cardiac output clears inflammatory mediators
- 2. Aprotinin-serine protease inhibitor with anti-inflammatory properties.
- 3. Modified Ultrafiltration decreases tissue edema and circulating inflammatory properties
- 4. Leukocyte depletion filters
- 5. Pulsatile perfusion
- 6. Coating of membranes and tubings with heparin

Cannulation Techniques

Venous Cannulation

Venous cannulation for CPB allows deoxygenated blood to be drained from the patient into the extracorporeal circuit.

The type of venous cannulation used is dependent upon the operation being undertaken. For cardiac surgery that does not involve opening the chambers of the heart, for example, coronary artery bypass grafts (CABG), a two-stage venous cannula is often used. An alternative method of venous cannulation for CPB is bicaval cannulation-this uses two single-stage cannulae that sit in the inferior and superior vena cavae, respectively. The two single-stage cannulae are connected using a Y-connector to the venous line of the CPB circuit. Bicaval cannulation is generally used for procedures that require the cardiac chambers to be opened.



A. Bicaval cannulation

The femoral veins may also be used as a cannulation site for more complex surgery. In this instance, a long cannula, which is in essence an elongated single-stage cannula, may be passed up the femoral vein into the vena cava in order to achieve venous drainage.

Types and size of cannula

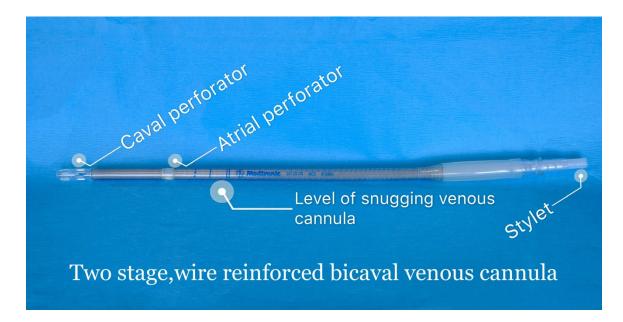
Venous cannulas are either single stage or double stage. Double stage cannula has a wider portion with holes in the section designed to sit in the RA and a narrower tip designed to rest in the IVC. Cannulas are made up of plastic and are wire reinforced to prevent kinking. They may be straight or right-angled. Tips of cannula may be **plastic or metal**, which provides best internal diameter (ID) to outer diameter (OD) ratio.

The size of cannula is selected such that one third of total flow drain through SVC cannula and two third of total flow drain through IVC.



Single stage venous cannula (A. Curved metal tip wire reinforced single stage venous cannula B. straight plastic tip wire reinforced single stage venous cannula)

Cannula Size (French) – DLP metal tip right-angled venous			
Weight	SVC	IVC	
<3.5	12	12	
3.5-6	12	14	
6-8	12	16	
8-12	14	16	
12-16	14	18	
16-22	16	18	
22-28	16	20	
28-32	18	20	
32-40	18	22	
>40	20-22	22-28	



Method of venous cannulation

- 1. Bicaval single canulas are inserted in SVC and IVC
- 2. Cavoatrial with two-stage canula.
 - It is used routinely in
 - Aortic valve procedures
 - Coronary bypass procedures
 - AVR+CABG procedure
 - Bentall procedure

The disadvantage of two stage venous cannula is it rewarms the heart \rightarrow less

myocardial protection.

Venous drainage is usually accompanied by *gravity siphonage*. Venous reservoir must be below the level of patient. Lines must be full of priming fluid and air lock should be prevented.

The determinants of venous drainage are

- Height of the patient above venous reservoir
- Patients' blood volume
- Resistance of tubing
- Cannula dimensions

Cause of low venous return

- Reduced venous pressure
- Inadequate height of patient above venous reservoir
- Malposition of venous cannula
- Obstruction or excess resistance of lines and cannula
- Kink/ air lock

Augmented Venous Return

It is achieved by three methods

- Placing a roller pump in venous line
- Centrifugal pump in the venous line (Kinetic assisted venous drainage)
- Vacuum assisted venous drainage

Adequate venous drainage is achieved with speeds of 1000-2000 RPM of kinetic pump or application of 20 mmHg vacuum to venous.

Potential Problems Of Augmented Venous Return

- Hemolysis
- Collapse of right atrium resulting in impaired venous drainage and chattering of venous line
- Micro air aspiration

Peripheral Cannulation

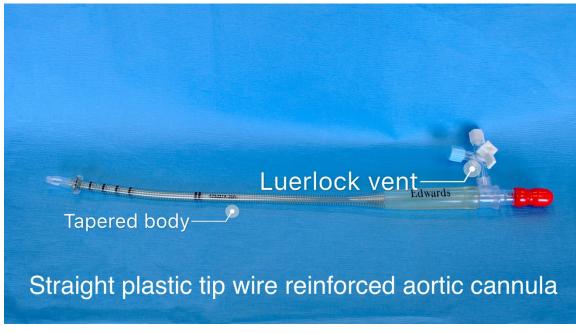
At times, venous cannulation is done peripherally, usually through femoral or iliac veins. Less commonly used sites are innominate vein and axillary vein.

Indications Of Peripheral Cannulation

- 1. Emergency closed cardiopulmonary assist
- 2. Support of ill patients before induction of anesthesia
- 3. Before sternotomy for reoperations
- 4. Minimal access surgery
- 5. Certain aortic and thoracic surgery

Arterial Cannulation

The arterial cannula is used to connect the "arterial limb" of the CPB circuit to the patient. It delivers oxygenated blood from the heart-lung machine directly into the patient's arterial system. The required size is determined by the size of the vessel that is being cannulated & blood flow required. The ascending aorta is the most common site of arterial cannulation. Arterial cannulas are sized by internal diameter. It has a metal or plastic tip.



Aortic cannula

Advantages of metal tipped cannula

- 1. It provides best internal diameter (ID) to outer diameter (OD) ratio
- 2. Easy to cannulate.

Disadvantages of metal tipped cannula

- 1. Jet effect \rightarrow Hemolysis
- 2. No side holes to reduce sandblast effect
- 3. Traumatic \rightarrow may damage posterior intima

Performance index – Pressure gradient versus OD at any given flow.

Tip may be straight of right angled. In small sized cannulas, the right-angled configuration may aggravate Hemolysis.

Thin-walled wire-wound cannulas like the BioMedicus (Medtronic) provide the best flow characteristics and are very resistant to kinking. This style of cannula is very effective for very small aortas e.g. interrupted aortic arch, and for minimally invasive procedures.

The arterial cannula is placed in the ascending aorta just proximal to the innominate artery. The outflow should be directed into the arch, not into the innominate artery. Cannula size is determined by the anticipated flow rate for the patient based on body surface area, so as to minimize line pressure and sheer force.

Weight (kg)	Cannula Size (French)
<5	10
5-10	12
10-14	14
14-28	16
28-50	18
>50	20

Complications Of Aortic Cannulation

- 1. It is preferable to select a cannula that will provide adequate flow with <100 mmHg pressure gradient. Pressure gradients >100 mm Hg may cause excessive <u>Hemolysis and protein denaturation</u>.
- 2. Jetting effect produced by small cannulas may damage interior aortic wall, dislodge atheroemboli (sandblasting) and cause arterial dissections. Proper position of the cannula tip is essential while inserting the cannula. If a right-angled cannula tip is used, it is initially directed towards the heart and rotated 180 degrees to confirm intraluminal placement. Bleeding through the cannula is then allowed to eliminate air or atheromatous debris and further confirm intraluminal placement.

Aortic dissection should be suspected when

- Increased CPB systemic line pressure
- Sudden decrease in both venous return or arterial pressure
- Decreased organ perfusion (oliguria, dilated pupil, myocardial ischemia, EEG changes)
- Blue discoloration of aortic root
- Bleeding from cannulation site

<u>Management</u>

• Prompt cessation of CPB

- Re-cannulation distal to dissection (usually from femoral but occasionally into distal aortic arch)
- Induction of deep hypothermia and a period of circulatory arrest while aorta is opened and the extent of injury analyzed and repaired by direct closure
- Use of patch or replacement of the ascending aorta with a tube graft
- 3. Atherosclerosis frequently involves ascending aorta and pose problems during cannulation, application of clamps and vascular grafting. Sandblasting effect of jet is thought to be a major cause of *perioperative stroke, aortic dissection and postoperative renal dysfunction.* Traditionally, surgeons rely on palpation to detect these changes and select site of cannulation. *Epiaortic ultrasonic scanning* is complementary. If patient has extensive atherosclerosis of ascending aorta, then alternative routes for cannulation should be considered, mainly femoral, axillary, subclavian or innominate artery cannulation.
- 4. During arterial cannulation, mean pressure should be optimum (70-80 mmHg in adults). If too high, there is greater chance of tears, dissection and blood loss. If too low, aorta tends to collapse and there is greater risk of damage to back wall of aorta.

Femoral Cannulation

Indications

- 1. Aneurysm of ascending aorta or diffuse atherosclerosis of ascending aorta
- 2. In unstable patients before induction
- 3. Reoperations
- 4. When Antegrade dissection complicates aortic cannulation
- 5. Minimally invasive cardiac surgery

Main complications of femoral cannulation are retrograde arterial dissection and limb ischemia. Placing a Y connector in the arterial line can minimize the risk of distal ischemia. A small cannula is inserted into distal femoral artery to maintain perfusion of the leg.

Axillary Cannulation

Advantages over femoral A cannulation are it is less likely to be involved by atherosclerosis and it has a good collateral blood flow, thereby decreasing risk of ischemic complications.

Tubing And Connectors

Desirable characteristics of tubing

It should be

- 1. Transparent
- 2. Inert
- 3. Biocompatible
- 4. Smooth inner surface
- 5. Low spallation rate
- 6. Flexible and kink resistant
- 7. Re expands after compression
- 8. Resists collapse, cracking and rupture
- 9. Tolerance to heat sterilization and blood compatibility
- 10. Medical grade PVC seems to meet these standards.

Keeping the tube as short as possible reduces prime volume, pressure gradients and blood trauma.

To minimize blood trauma, avoid velocities above 100 cm/s and avoid Raynaud's number above 1000.

Connections must be tight enough to prevent leakage of blood when exposed to positive pressures and aspiration of air on the venous side.

Patient Weight (kg)	Arterial Line (inches)	Venous Line (inches)
<8	1/4	1/4
8-17	1/4	3/8
17-50	3/8	3/8
>50	3/8	1/2

Size of tubing

Biomaterial Tubing

Binding heparin or other surface modifying agents into inner surface of the tubing may improve biocompatibility.

Examples

- 1. Biomembrane mimicry Tubing are coated with a derivative of phosphorylcholine (Memys, Sorin)
- 2. Heparin coated circuits
 - Heparin bound to tubing is slowly released into circulation (Duroflo II, Baxter)
 - Heparin is permanently bound covalently to biomaterial surface (Carmeda,

medtronic and Trillium, Medtronic)

- Hybrid surface-combination of heparin releasing and heparin immobilized (Bioline, Jostra)
- 3. Surface modified additives -Terumo Corporation has developed CPB circuits coated with poly 2-methoxy-ethylacrylate which has hydrophobic properties and little tendency to react with blood products.

Results

In vitro studies have shown potent antithrombotic behavior. Clinically, there is no evidence of decreased thrombogenecity. However, inflammation related to complement activation is decreased.

Heat Exchangers

Heat exchangers are necessary to control body temperature during cardiopulmonary bypass. They are included as integral part of disposable oxygenators. They are usually located proximal to gas exchange section of circuit to minimize risk of releasing micro-bubbles.

<u>Hardware</u>

Primary form of heat transfer is *forced convection*. The heat transfer surface is made up of stainless steel, aluminum or polypropylene. To maximize efficiency of heat transfer blood and water pathways flow in counter current direction. Water circulates within heat exchanger between 1-2C and 42C. Blood should not be heated above 42C to avoid injury to blood proteins.

The temperature difference between circulating water and the blood determines heat transfer. There is risk of gas embolism if temperature difference between heat exchanger and patient's blood is >10C.

Cardiotomy Reservoir And Field Suction

<u>Components</u>

- 1. Defoaming chamber -pore size of Cardiotomy filter is 40 u.
- 2. Storage chamber
- 3. Macro or micro filter

Blood is returned to venous reservoir (with membrane oxygenators) or into the bubble oxygenator, either continuously or intermittently.

Cardiotomy suction is found to be a major source of hemoylsis, particulate microparticles, inflammatory mediators and platelet injury.

Methods to minimize adverse effects of Cardiotomy suction

- 1. Doing off pump cases
- 2. Minimize aspiration of air
- 3. Filtration of Cardiotomy suction blood
- 4. Not employing Cardiotomy suction
 - Discard shed blood
 - Use cell salvage and washing system
- 5. Hemostasis to minimize shed blood

Cardiac Venting

The normal sources of blood returning to left ventricle during CPB include bronchial and the basian veins. Abnormal sources include LSVC, PDA or BT shunt, VSD, anomalous systemic venous drainage into left heart and aortic regurgitation.

Venting of left heart is indicated whenever the left ventricle is unable to handle the amount of blood that is returning to it.

Purpose

- 1. Aid visualization and provide bloodless field
- 2. Avoid chamber distension
- 3. Reduce myocardial rewarming
- 4. Prevent cardiac ejection of air

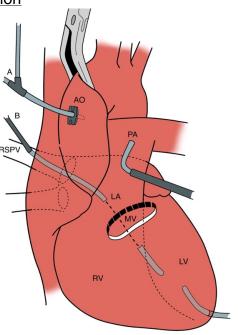
Potential sites of cardiac venting

- 1. Pulmonary artery
- 2. Right superior pulmonary vein
- 3. Left atria
- 4. Left ventricle
- 5. Aortic root cardioplegia cannula

Monitoring of LV distension

- Inspection and palpation of left heart
- PA catheter
- Left atrial monitoring line
- TEE

Major complication of cardiac venting is air embolism. To avoid this complication, heart is allowed to fill before vent insertion and vents are preferably removed while insertion site is covered with fluid.



Sites of Cardiac venting

Arterial Filters

These filters remove air and particulate matter while allowing passage of cellular elements of blood. In modern practice, screen arterial line filters made of nylon or polyester are almost universally used generally with a pore size of 40 um. Surface area varies between 650-800 cm².

Limitations

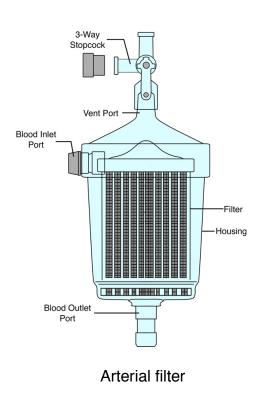
- Relatively large volume (200 ml) is required to prime the filters
- Problems related to cellular components of blood -Hemolysis, platelet loss and complement activation
- Lack of proven benefit
- A paradoxical risk of increased particulate embolism.

Specific arterial filters

Pall Biomedical, Terumo Capiox, Dideco 736

Leukocyte depletion filters

These contain non-woven polyester fibers that have been surface modified to remove leukocytes. Neutrophil are important contributor to ischemia reperfusion injury. Neutrophils cause tissue injury by release of enzymes, reactive oxygen species and other toxic substances.



Neutrophil filtration in CPB circuit has been proved beneficial to provide myocardial protection.

LD filters may be placed in many different locations in the ECC, including arterial line, venous line and cardioplegia line. The disadvantage of LD filters is that it adds further prime volume to the circuit.

Minimal Extracorporeal Circuits

They all feature decreased foreign surface area, priming volume and blood air contact.

They are basically a closed venoarterial loop incorporating a single centrifugal pump, a membrane oxygenator and some type of surface modification but exclude venous reservoir and cardiotomy suction and often heat exchanger and arterial filter.

Priming volume is 500ml.

Used successfully in CABG, aortic and mitral valve surgeries.

Some RCTs have found less inflammation, less Hemodilution, and improved neurological and renal function while others have failed to detect clinical benefits.

Examples

- Performer Medtronic
- Synergy Sorin Biomedical

Limitations

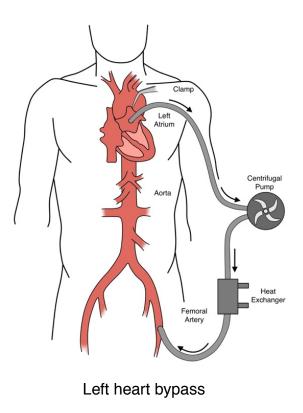
- The greatest concern is about systemic air embolization because of lack of venous reservoir and absence of arterial filter.
- Lack of venous reservoir to handle excess venous return and to permit immediate volume infusion when needed.
- Lack of heat exchanger
- Need to use a separate cell processing system
- Increased cost

Left Heart Bypass

This method relies on patient's lungs to provide gas exchange. Blood is removed from left heart with large venous cannula placed directly into left atrium or through left pulmonary veins. This carries risk of air embolism if one is not careful during insertion and removal of this cannula.

Arterial return may be into descending aorta or femoral artery. Flows range from 1-3 L/min.

Extra-corporeal circuit (ECC) consists of tubing and centrifugal pump. It does not include a reservoir, heat exchanger or bubble trap. This minimizes need for heparinization. If no reservoir is included in the ECC, the anesthesiologist must be prepared to administer intravascular volume expanders. This support system is not truly CPB as there is no oxygenation of blood, but simply



pumping of oxygenated blood from left atrium to distal arterial vessels.

Indications

Patients with descending thoracic or large thoracoabdominal aortic aneurysms.

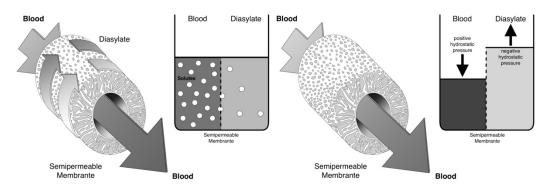
Hemofilter/Ultrafiltration Devices

Dialysis

It refers to a process in which blood is separated from crystalloid solution or dialysate by a semipermeable membrane

Ultrafiltration

It refers to movement of water across a membrane due to transmembrane pressure gradient. No dialysate on the opposite side of membrane is required.

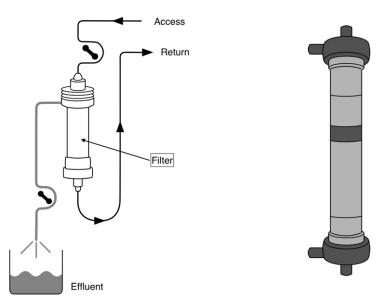


Comparison of Hemofiltration & Dialysis

It contains semipermeable membrane that permits passage of water and electrolytes out of blood. The semipermeable membrane is manufactured in hollow fiber configuration. The hollow fibers are between 180-200um in diameter and the pores of microporous membrane are between 5-10nm. The rate of fluid removal depends on:

- Membrane permeability
- Blood flow
- Transmembrane pressure
- Hematocrit

Ultrafiltration removes excess fluid. Studies have shown that patients who are ultrafiltered demonstrate increased hematocrit, decreased lung waters and decreased tissue edema. It also improves patient's hemodynamics, cardiac contractility and oxygenation.



Mechanism of hemofiltration by positive hydrostatic pressure

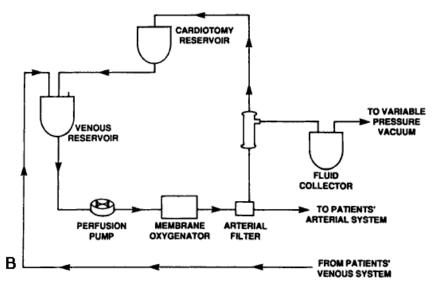
Compared to cell washer

- They conserve platelets and coagulation factors → improve perioperative hemostasis
- They conserve albumin \rightarrow reduce lung water and post operative ventilator support
- Cheaper

Molecules up to a molecular mass of 20,000 Da are removed. At least some heparin is removed and therefore, adequacy of heparinization should be checked.

Conventional Ultrafiltration (CUF)

It refers to the practice of withdrawing blood from the patient through venous reservoir during rewarming period when the patient is on CPB, and passing it through a hemoconcentrator and pumping it back into the patient through venous reservoir.



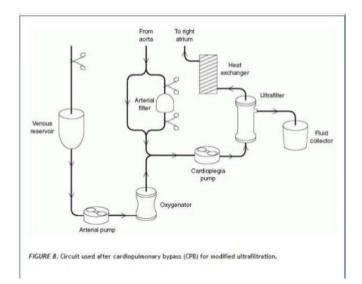
Conventional ultrafiltration

Zero balanced Ultrafiltration

The volume of plasma water removed is replaced by an equal amount of balanced electrolyte solution. The purpose is to remove inflammatory mediators.

Modified Ultrafiltration (MUF)

Introduced by Elliot. It refers to the practice of withdrawing blood from the patient through aortic cannula, after weaning from CPB, and passing it through a hemoconcentrator and pumping it back into the patient through venous cannula.



Modified Ultrafiltration

In most centers, arteriovenous configuration is used. Venovenous MUF carries less risk of air cavitation and hemodynamic instability but it does not deliver oxygenated blood into pulmonary vasculature.

Advantages

Naik et al noted decreased blood loss, fewer blood transfusions, and increase in arterial blood pressure. MUF seems to be most effective in pediatric patients.

Disadvantages

Patient needs to remain cannulated for 10-20 minutes after CPB termination and to maintain integrity of ECC, protamine should not be administered during MUF. Air aspiration is the major concern. Because of the risk of air entering into arterial circuit, Antegrade flow should not be permitted once MUF is begun.

	Conventional Ultrafiltration	Modified Ultrafiltration
Inlet	Venous reservoir	Arterial end
Outlet	Venous reservoir	Venous end
Timing	During rewarming, on CPB	Off CPB
CPB time	Does not increase CPB time	Increase CPB time
Risk of air aspiration	Less	Most
Circuit design	Technically simple	Technically complex requiring prior planning

SECTION IV: OXYGENATOR AND PUMP

Pump

The pump is a machine that imparts motion to fluids. During cardiopulmonary bypass the heart is functionally out of circuit and a blood compatible pump must supply sufficient flow at an adequate pressure.

The ideal pump for CPB should have the following characteristics

- It must be able to pump blood at a flow rate of 7 L/min against a pressure of 500 mm Hg
- The pumping motion should not damage the cellular or acellular components of blood.
- All parts in contact with the bloodstream should have a smooth continuous surface with no dead space to cause stagnation or turbulence, should be disposable, and should not contaminate the permanent parts of the pump.
- Calibration of pump flow should be exact and reproducible so that blood flow can be accurately monitored.
- In the event of a power failure, the pump should be manually operable.

Types of pumps

There are two principle types of pump.

- 1. Displacement pump
 - Roller pump
 - Non occlusive roller pump
- 2. Rotary pumps
 - Axial (Archimedes Screw)
 - Diagonal
 - Radial (centrifugal)

Roller Pump

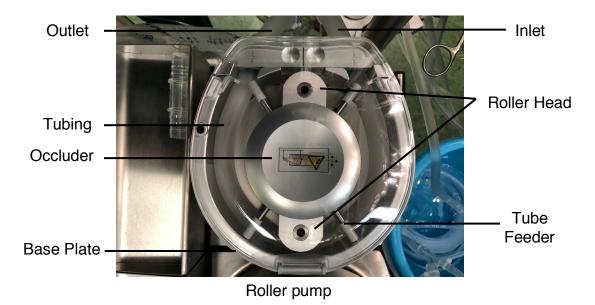
Developed by DeBakey in 1934

Principle

Two rollers, placed opposite to each other, roll the blood through piece of tubing.

Hardware

- The roller pump consists of two rollers, 180° apart, which rotate through a raceway that is an arc of approximately 2000
- Tubing made up of PVC or silicon is placed between the rollers and raceway so that the rollers barely occlude the tubing at 180 mm Hg back pressure
- As one roller begins to compress the tubing, the opposite roller releases so that



blood within the tubing is continuously propelled in one direction.

Roller pump occlusion procedure

The pump occlusion is compression of the tubing as the roller presses tubing against the race

STEP-1: Load the unprimed tubing in the roller pump raceway.

- STEP-2: Introduce the appropriate amount of priming solution in to the oxygenator
- STEP-3: While holding a segment of tubing on the positive side of pump vertically, advance a column of crystalloid priming solution approximately 30cm above the level of pump.
- STEP-4: Occlusion of each roller pump must be adjusted in such way that it will allow a drop of 1 inch/minute of priming solution of the tubing.

Roller pumps are capable of generating *more powerful pulsatile flow* than centrifugal pumps, and therefore they are used as arterial pumps.

Flow rate depends on two variables – Size of tubing and number of rotations of pump head. A key issue for controlling the output is the occlusion setting of the rollers; Under-occlusion results in inadequate flow while over-occlusion results in Hemolysis.

Tubing Diameter (inches)	Stroke Volume (ml)
3/16	7
1/4	13
3/8	27
1/2	54

Complications

- Malocclusion
- Loss of power or pump failure
- Fracture of pump tubing
- Spallation repeated compression of rollers on the tubing will generate micro particle, a process is called spallation
- May pump large amount of air

Centrifugal Pump

Principle

Fluid moves by creating a pressure gradient between the inlet and outlet of the pump.

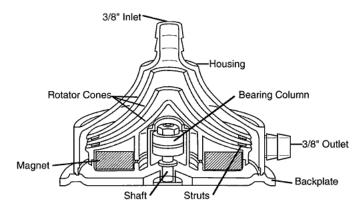


Diagram showing components of centrifugal pump

It consists of a nest of smooth plastic cones located inside a plastic housing. The disposable housing is coupled magnetically with electric motor. When rotated at 2,000-3000 rpm these pumps generate a pressure difference that causes movement of fluid. Rate of blood flow depends on pressure gradient and resistance at the outlet of pump.

Unlike roller pump they are totally **non-occlusive and afterload dependent** i.e. an increase in afterload decreases forward flow. This results in constant fluctuations in blood flow. Therefore, a **flowmeter** must be incorporated in the arterial line to quantify pump flow. Further, when the pump is not running, blood will flow backwards leading to exsanguinations of the patient or aspiration of air. Therefore, **when the centrifugal pump is not running, arterial line must be clamped.**

It is preferred in pediatric patients and in ECMO circuit.

Examples of centrifugal pumps

- Maquet Rotaflo
- Terumo Capiox

- Medtronic biomedicus
- Sorin revolution

Advantages

- Reliability
- Ease of use over a wide range of flows
- Absence of spallation
- Low risk of massive air embolism. Pump stops if > 50 ml of air is introduced into the circuit.
- Less blood trauma, less activation of coagulation and produce fewer micro-emboli.

<u>Disadvantage</u>

- Susceptible for air locks
- Power outage can be a disaster
- Centrifugal pumps increase total priming volume
- Centrifugal pump cannot be operated in cardiotomy suction or vent positions.
- Disposal pump head adds to cost.

Characteristics of centrifugal pulse:

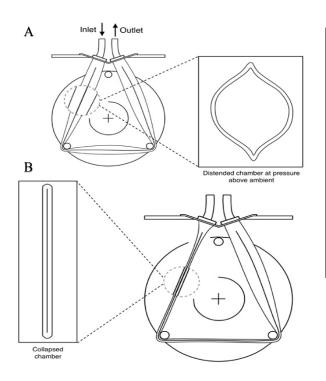
- Controllable stroke volume and pulse rate
- Capable to produce wide range of output which are linerarly proportional to pulse rate& independent of afterload.
- Minimal transfer of kinetic energy to blood.
- Calibration easy and reproducible
- Automatically controlled and operated for routine use, with battery backup.

Sr	Feature	Roller Pump	Centrifugal Pump
1	Principle	Two rollers, placed opposite to each other, roll the blood through piece of tubing	Fluid moves by creating a pressure gradient between the inlet and outlet of the pump.
2	Rate of blood flow	Depends on Size of tubing and number of rotations of pump head	Depends on pressure gradient and resistance at the outlet of pump
3	Pulsatile flow	++	+
4	Air embolism	++	+
5	Blood Trauma	++	+
6	Need of flow meter	Absent	Present
7	Occlusion	Occlusive	Non occlusive
8	Spallation	Present	Absent
9	Cost	Less	More
10	Cardiotomy suction vent	Can be used	Cannot be used
11	Afterload	Not dependent	Dependent

Non-Occlusive Roller Pump

MC3 pump is a passive filling, peristaltic pump that combines many advantages of both centrifugal pump and the roller pump. This pump is non-occlusive and used in combination with flow meter.

The rollers are mounted on rotor. Rotation of rotor imparts a peristaltic motion to the blood within pumping chamber. When the inlet of the chamber is supplied with blood at a pressure above ambient, blood is moved towards outlet. When the inlet pressure is below ambient pressure, pump chambers become flat and the pump stops. This prevents pumping of air.



Schematic drawing of non-occlusive roller pump

A: Operating principle of the triple roller pump and pumping chamber. Polyurethane pumping chamber is stretched over the rollers, and roller rotation is counterclockwise.

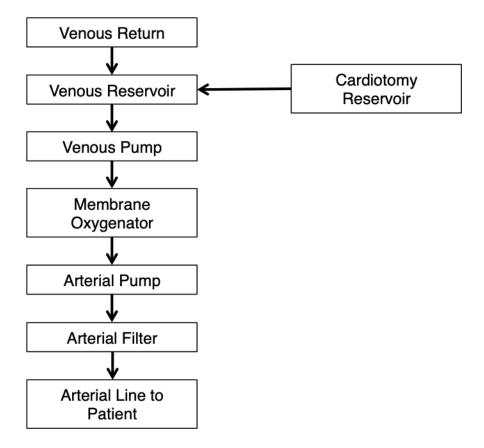
B: Pumping chamber inlet collapsed when blood is not supplied at a pressure above ambient.

Pulsatile Flow On Cardiopulmonary Bypass

Several investigators have shown that use of pulsatile flow may have physiologic benefits on vital organ function during and after bypass. Others have found that production of nitric oxide is increased during pulsatile flow.

Pulsatile CPB Can Be Achieved By

- Use of IABP during bypass
- Partial CPB.
- Diagonal pump (Deltastream, Medos)
- Placement of second pump beyond membrane oxygenator



Membrane oxygenator with pulsatile flow circuit

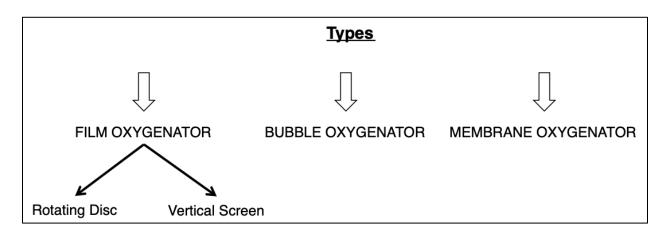
Oxygenator

The fundamental purpose of oxygenator is to arterialize venous blood by removing excess CO2 and increase PO2.

The use of oxygenators for long-term support > 6 hrs is described as ECMO. Short term devices<6hrs is described as traditional CPB oxygenators.

There are three types of oxygenators

- 1. Film
- 2. Bubble
- 3. Membrane



Film Oxygenator

<u>Rotating Disc Oxygenator</u> – The rotating disc oxygenator is a horizontal Pyrex glass cylinder with stainless steel end plates. A central axel is rotated on bearing set. It contains stainless steel discs with spaces between them, flat and convoluted. There is direct contact of blood and gas in it.

<u>Vertical Screen Oxygenator</u> – Cabinets containing a series of upright screen in an oxygen atmosphere. In this type of oxygenator, venous blood is introduced by way of slits at the top of the cabinet. Oxygenated blood collects at the bottom of the cabinet. Since the flow of the blood through the oxygenator varied consistently, a recirculation pump was necessary.

Bubble Oxygenator

Principle

The blood enters a venous inlet and crosses a heat exchanger. Oxygen is then bubbled through this venous blood and gas exchange occurs. The oxygenated blood then flows through a defoamer and into arterial reservoir. The arterial blood is then returned to the patient.

Gas bubbles are dispersed in to the venous blood in the bubble column and act as vehicle for transfer of both oxygen and carbon dioxide. Oxygen diffuses from bubble in to the blood film surrounding the bubble. Carbon dioxide diffuses from blood film into the bubble. Bubble size is important for adequate gas transfer. Smaller bubble increases oxygenating capacity but not available for carbon dioxide removal whereas larger bubble increases the carbon dioxide removal but compromises the oxygen transfer. The ideal bubble size for gas transfer is 3 mm to 7 mm. As bubble/blood mixture moves out of the bubble column and through the defoaming area, the bubble will burst releasing their contents, which are then vented from the device. This gas environment provides secondary oxygen and carbon dioxide transfer. If secondary oxygenation decreased total

oxygen transfer may be significantly diminished. Higher the operating levels, smaller the area operated outside of the bubble column for secondary oxygenation.

Membrane Oxygenator

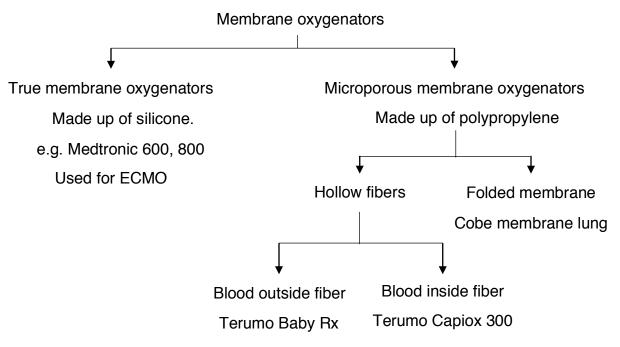
The membrane oxygenator physically separates the blood from gas with the gas permeable membrane material. It consists of three parts

- Oxygenator
- Venous reservoir
- Heat exchanger

Membrane material

- 1. Silicon rubber used for long durational cases (ECMO)
- 2. Polypropylene Flat and hollow fiber

Types of membrane



Currently employed materials are *silicon rubber and polypropylene*. The silicon rubber is homogenous, non-porous material where as the polypropylene is heterogeneous, micro-porous, hydrophobic membrane.

The micro porous membranes are developed by stretching the membrane material and forming rents in the substance of membrane that act as pores for gas transfer. Pores are 0.03 um to 0.07um in effective diameter and cover half of the membrane surface. The pore size is in the sub-micron range and is hydrophobic. It does not act as diafilter. However, gas can be forced in to the blood path if the gas path exceeds that of the blood path.

Hollow Fibers

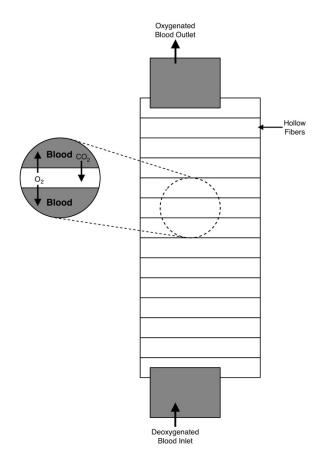
- 1. Blood inside fiber If some of the fibers rupture, blood will spill in to the gas phase and decreases the total surface available for oxygen transfer. This may not allow visualization of air bubbles in blood path during priming or operation.
- 2. Blood outside fiber if some of the fiber ruptures blood will spill only in to fiber affected and will not change the total surface area available for oxygen transfer. Allow visualization of air bubbles in the blood path during priming or operation.

Body Surface Area	Medtronic	Medos	Maquet	Sorin
Infant (0.2-0.34)	Pixie	Hilite1000	Quadrox1000	Liliput
Children (0.34-0.8)	Minimax	Hilite2800	Quadrox3000	EOS
Adult (0.8-2.2)	Affinity	Hilite7000	Quadrox7000	Apex

Commonly used oxygenators include

Characteristics of ideal oxygenator

- 1. Oxygenation of venous blood: It can provide oxygenation over a range of venous inlet wide conditions. There must be safe and efficient means where by venous blood is brought in to a proximity to a source for oxygen transfer.
- 2. Carbon dioxide removal: Must be safe and efficient elimination of carbon dioxide in sufficient quantity to avoid hypercarbia or hypocarbia.
- 3. Minimun trauma to blood: The gas transfer process must be associated with minimum damage to blood cells, platelets and This protein. can be accomplished by careful design. Trauma induced by the direct exposure of gas to blood must be considered.



Illustrated diagram of oxygenator

4. Small priming volume: Oxygenator must be able to perform adequate gas exchange in low or small priming volume of extracorporeal circuit. Hemodilution can effect on oxygen delivery and also on protein concentration

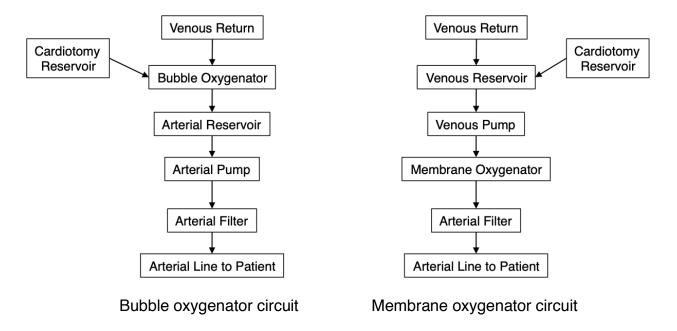
- 5. <u>*Safety:*</u> Device must be easily assembled, prime and operated. It must be constructed to minimize the possibility of air embolism during normal procedure.
- 6. <u>Ease to use</u>: Material used should be transparent to give clear visualization. Ports must be labeled to avoid confusion. It should be easily changeable in case of emergency.

All currently used oxygenators fulfill the requirement of ideal oxygenator.

Differences/Comparison Of Bubble Oxygenator And Membrane Oxygenator

Film Oxygenator	Bubble Oxygenator	Membrane Oxygenator
Reusable	Disposable	Disposable
Difficult to use	Easier to assemble	Easiest to assemble
Direct contact of blood and gas	Direct contact of blood and gas	Blood and gas are separated by porous membrane
Maximum damage to blood cells and protein	Lesser damage blood cells and protein	Minimum damage to blood cells and protein
Higher chances of micro air emboli	Lesser chances of micro air emboli	Minimal chances of micro air emboli
Highest prime volume	Higher prime volume	Lesser prime volume
Maximum hemolysis	Lesser hemolysis	Minimum hemolysis
Cost is cheapest than the other oxygenators	Cheaper than the membrane oxygenator	Most expensive
Oxygenated blood runs through the pump head	Oxygenated blood runs through the pump head	Deoxygenated blood run through the pump
Gas and blood are in opposite direction	Gas and blood are from the same point in the same direction	Gas and blood are in opposite direction
No independent control of PO2 and PCO2	No independent control of PO2 and PCO2	Independent control on PO2 and PCO2
The sterilization used is autoclave	Sterilization is ethylene oxide(ETO)	Sterilization is ethylene oxide(ETO)

Circuit Diagrams



Venous Reservoir

The venous reservoir is of two types: closed collapsible bag & open hard shell.

<u>Closed Collapsible Bag</u> – The blood volume in the bag is not exposed to room air surrounding the device. Excess volume coming in to the bag is stored either in the cardiotomy or in patient's veins. Air collected in the bag must be removed through the purging parts situated at the top of the bag. Bag reservoir does not have direct defoaming capability. It provides an element of safety, as it collapses and does not allow massive air being delivered to membrane.

<u>Open Hard-Shell</u> – Blood in the reservoir exposed to air. The advantages of this type of reservoir is-it gives better visibility of levels, larger volume storage capacity, a easier passive elimination of air entering from the venous and or cardiotomy lines. It acts more like a bubble oxygenator. If it gets accidentally empty during cardiopulmonary bypass can potentially deliver air in to the circuit.

Advantages of Open Reservoir	Advantages of Closed Reservoir
Easy to measure volume	
Handle venous air more effectively	Reduce risk of massive air embolism
Larger capacity	because they collapse when emptied and do not permit air to enter the
Easy to prime	systemic pump
Permit application of suction for vacuum assisted venous return	

Functions

- 1. It serves as a buffer for fluctuation and imbalance between venous return and arterial flow.
- 2. It serves as a gross bubble trap for air that enters venous line. The pore size in venous reservoir filter is $150-200\mu$.
- 3. It acts as a site where blood, fluid or drugs may be added, into which cardiotomy reservoir empties, and a ready source of blood transfusion to the patient.
- 4. It provides time for the perfusionist to act if venous drainage is sharply reduced to avoid pumping the CPB system dry and risking massive air embolism.

With membrane oxygenators, the reservoir is the first component of ECC, directly receiving venous drainage and cardiotomy drainage.

When bubble oxygenator is used, the reservoir is placed beyond oxygenating and defoaming chambers.

SECTION V: HEMODILUTION AND PRIMING SOLUTION

Introduction

The cardiopulmonary bypass (CPB) circuit must be primed with a fluid solution, so that adequate flow rates can be rapidly achieved on initiation of CPB without risk of air embolism. Priming fluid fills both venous and arterial limbs of the circuit and maintains an adequate reserve volume in the venous reservoir to ensure that air is not entrained into the arterial side of the circuit during initiation of CPB.

The optimum composition of the CPB priming solution is still a matter for debate. Currently used primes provide a fluid that when mixed with blood is capable of maintaining oxygen delivery, carbon dioxide and physiological homeostasis.

Historical perspective

Blood was used to prime CPB circuit in an attempt to preserve a high Hematocrit; early in the evolution of cardiopulmonary bypass this was thought to be an important determinant for successful outcome.

It later became clear however, that use of crystalloids in the prime improve outcome due to hemodilution.

Zuhdi et al developed the process of hemodilution in 1961. DeWall confirmed the benefits of hypothermic hemodilution.

Benefits of hemodilution

- Decreases blood viscosity → Improved regional blood flow
- Improved oxygen delivery to tissues
- Decreased exposure to homologous blood products
- Improved blood flow at lower perfusion pressure especially during hypothermic perfusion

Hematocrit on CPB

- It is appropriate to target hematocrit< 30% if the temperature is reduced to 30°C, and lower hematocrit up to 25 % are preferred if temperature is reduced below 25°C.
- Hematocrit< 20% may be associated with abnormal distribution of blood flow to organs.
- Hematocrit >34% in CABG patients is associated with greater risk of Q wave infarct, worsened LVEF and increased mortality.

Priming

1. BLOOD PRIMING

2. NON-BLOOD PRIMING:

A] CRYSTALLOID PRIMES

- Dextrose
- Balanced Crystalloid Fluid
- Mannitol
- **B] COLLOD PRIMES**
 - Albumin
 - Gelatins
 - Dextrans
 - Hydroxymethyl Starch
- 1. Blood priming: Reducing the degree of hemodilution
 - **Indications**
 - Pediatric
 - Low hematocrit

Blood is a non Newtonian fluid. Its viscosity depends on flow rates.

Poiseuille equation

 $Flow = \frac{\alpha \, Perfusion \, Pressure}{Vascular \, Resistance \times Viscosity}$

$$Q = \frac{\pi (P1dP2)R^4}{8uL}$$

Q= Flow P1dP2= pressure drop along a tube of radius R and length L u=viscosity

3. Non-blood priming

A] Crystalloid Prime

DEXTROSE

Dextrose 5% is slightly hypotonic and acidotic and becomes more so as dextrose is metabolized in vivo.

Function

- 1. Reduces the mechanical damage to erythrocytes and improves intra operative and postoperative diuresis.
- 2. Crystalloid prime containing dextrose has also been found to lead to decrease preoperative fluid requirement and reduced postoperative fluid retention.

<u>Disadvantages</u>

- 1. Dextrose is metabolized & the dilutional effect on plasma bicarbonate may cause systemic metabolic acidosis.
- 2. As serum glucose and insulin concentrations are elevated due to the effect of cardiopulmonary bypass, adding dextrose to the prime may further increase the level of blood glucose. This is especially a concern for diabetic patients.

The glucose containing priming solution may increase the risk of CPB related neurological complications although there was a lack of significant clinical evidence.

BALANCED CRYSTALLOID FLUID

Balanced crystalloids are the fluids formulated to have a neutral PH and concentration of electrolyte ions similar to that of human plasma. Ringer's lactate and Hartmann's solution are typical examples of a balanced crystalloid and contain lactate as a source of bicarbonate.

However, a large volume of fluid containing lactate should be used with caution in diabetic patients, as lactate may be converted in to glucose in vivo through the gluconeogenic pathway.

A further example of a balanced crystalloid is plasmolyte solution which contains acetate and gluconate for bicarbonate production. It also contains magnesium which is an important intra cellular caution involved in cellular process of energy transfer and in cellular process of energy transfer and in myocardial ATP metabolism.

MANNITOL

Mannitol is a hypotonic, low molecular weight crystalloid widely used in clinical practice to stimulate dieresis.

A particular advantage of mannitol is its protective effect on renal function. As a volume expander, mannitol draws fluid initially across the capillary into the plasma. Then it rapidly diffuses volume of the whole extracellular phase by withdrawing water from the body cells.

B] Colloid Prime

ALBUMIN

Molecular weight 69000 daltons. It accounts for 75% to 80% of the plasma oncotic pressure, responsible for the maintenance of body plasma volume. Addition of 200ml of 25% albumin in the bypass circuit had no beneficial effect on preoperative fluid balance, cardiopulmonary and renal function.

Albumin prime reduces post-operative bleeding whereas hydroxyethyl starch prime did not. Albumin can induce anaphylactic or anaphylactoid reactions and may also carry the risk of transmission of viral disease. For these reasons, and also because albumin is rather expensive, a number of synthetic colloidfluids are chosen as priming fluids.

DEXTRANS

Molecular weight 40,000daltons to 70,000 daltons. The dextrans molecule is a polysaccharide produced from sucrose by the bacterium leuconostoc mesenteroides. Dextran 40 has a colloid osmostic pressure twice as high as that of plasma, and so has a strong effect in mobilizing water from the extracellular into the intravascular space. Dextran 40 prepared in 10% solution is a more effective volume expander than dextran 70. As it is contains almost twice as much as colloid per litter.

However the action of dextran 40 is much less sustained, as the small molecules allow it to be rapidly eliminated by the kidneys. As a priming fluid for cardiopulmonary bypass, dextran reduces blood viscosity and prevents the adhesion of leukocytes in the microcirculation.

It is recommended that the total dose of dextran infusion does not exceed 1. 4g/kg/day as dextran may impair *hemostasis*. This dose should be further limited in patients undergoing cardiopulmonary bypass because heparin is used in these patients. Anaphylactoid reactions to dextrans may occur, but the incidence of reaction is much lower than that caused by gelatin.

GELATINS

Obtained from bovine collagen. Molecular weight 30,000 to 35,000 daltons.

Types

- Urea Linked Gelatin
- Succinyl linked Gelatin

<u>Disadvantage</u> – Gelatin is relatively high incidence of anaphylactoid reactions compared with other artificial colloid.

HYDROXYETHYL STARCH

Hydroxyethyl starch is synthetic colloid that consists of hydroxyethylated polymers of glucose derived from amylopectin. Compared with albumin as a colloid priming fluid, hydroxyethyl starch appeared to achieve the similar clinical effects of volume expansion in cardiac surgical patients with low incidence of anaphylactoid reactions containing balanced electrolytes sodium, chloride, calcium, magnesium and potassium as well as glucose and lactate.

Factors Affecting Hematocrit During CPB:

- 1. Patient's weight
- 2. Preoperative anemia or polycythemia
- 3. Preoperative hypovolumia or hypervolumia
- 4. Cardiopulmonary bypass circuit volume
- 5. Cardiopulmonary bypass prime volume
- 6. Pre cardiopulmonary bypass blood
- 7. Pre cardiopulmonary bypass volume administration
- 8. Crystalloid cardioplegia volume
- 9. Added crystalloid or colloid prime to maintain flow
- 10. Urine output.

Additional Components In Priming Fluid

Component	Function
1. Heparin	Heparinization
2. Mannitol	 Prevent edema maintains osmotic pressure oxygen free radical scavenger
3. Calcium	Prevent Chelation
4. Corticosteroids	Prevent inflammatory reactions due to CPB.
5. Bicarbonate	Act as buffer when unbalanced prime solutions are used

Assessing Adequacy of perfusion with Hemodilution

- Global cerebral function (Electroencephalogram EEG)
- Blood flow velocity (Transcranial Doppler)
- Renal function, urine output.
- ABG monitor oxygenator function, and measurement of mixed venous oxygen tension.

SECTION VI: TEMPERATURE AND ACID BASE MANAGEMENT IN CPB

Hypothermia exerts its protective effects by multiple mechanisms

- 1. It reduces metabolic rate and O₂ consumption
- 2. It reduces excitatory neurotransmitter release
- 3. Slows down enzymatic reactions
- 4. Decreases inflammatory response to cardiopulmonary bypass

Hypothermia Level	Patient Temperature (°C)	Safe Circulatory Arrest Time (min)
Mild	37-32	5-10
Moderate	32-28	10-15
Deep	28-18	15-60
Profound	<18	60-90

In cardiac surgery, hypothermia allows lower pump flow, better myocardial protection, less blood trauma and better organ protection compared to normothermic perfusion.

Disadvantages of hypothermia

- 1. Prolongation of cardiopulmonary bypass -due to periods of cooling and rewarming
- 2. Infection -?
- 3. Prolonged post operative recovery

Acid base management strategy

During hypothermia, solubility of CO2 increases. Also with hypothermia, pH increases, due to influence of buffers, such as imidazole moiety of histidine. Thus, *hypothermia leads to alkalosis and hypocarbia*, which triggers decrease in cerebral blood flow.

Alpha stat management

It refers to acid base management strategy in which Net charge of proteins remains constant as temperature changes. Typically, exogenous CO2 is not added to the system when following alpha stat strategy. Alpha stat management results in lower cerebral blood flow as compare to pH stat management. In adults, emboli appear to play a prominent role in adverse neurologic outcomes. Reduced CBF associated with alpha stat management limits dispersion of cerebral microemboli.

PH stat management

It refers to acid base management strategy in which PH remains constant as

temperature changes. This is achieved by adding 5% CO_2 to the system to maintain pH of 7.4. It results in excessive blood flow that may be detrimental. In children, hypoperfusion appear to play a prominent role in adverse neurologic outcomes. Therefore, pH stat strategy may be beneficial.

Protection of the brain may be best achieved by a mixed acid base strategy

- pH stat during initial cooling phase
- alpha stat strategy during reperfusion, rewarming and termination of CPB

Alpha Stat Management	pH State Management
Seen in poikilothermic animals	Seen in hibernating animals
Net charge of proteins remains constant as temperature changes	pH remains constant as temperature changes
Exogenous CO_2 is not added to the system	Exogenous CO2 is added to the system
Decrease in cerebral blood flow	Increase in cerebral blood flow
Preferable in adult patients	Preferable in pediatric patients

SECTION VII: MYOCARDIAL PROTECTION

The pathophysiology and underlying molecular biology of myocardial injury are complex.

Ischemia-reperfusion injury

Definition

It refers to functional, metabolic and structural alterations caused by restoring blood flow after a period of temporary ischemia

Pathophysiology

It is due to

- 1. Intracellular calcium accumulation
- O₂ free radical release-The generation of reactive oxygen species is believed to be mediated by xanthine oxidase, activation of neutrophils or dysfunction of mitochondrial electron transport chain. The major reactive oxygen species are superoxide (O₂), Hydrogen peroxide (H₂O₂) and hydroxyl ions(OH⁻)
- 3. No reflow phenomenon-after a period of ischemia, despite restoring blood flow to preischemic levels, some areas remain underperfused. There are two components of no reflow
 - Physiologic sustained vasoconstriction following release of endothelin
 - Mechanical capillary plugging by adherent neutrophils
- 4. Cellular edema

Myocardial injury

It is classified as

Reversible injury

Transient depression in cardiac performance, myocardial edema and resolves without long-term sequelae. It manifest as *stunning or hibernation*.

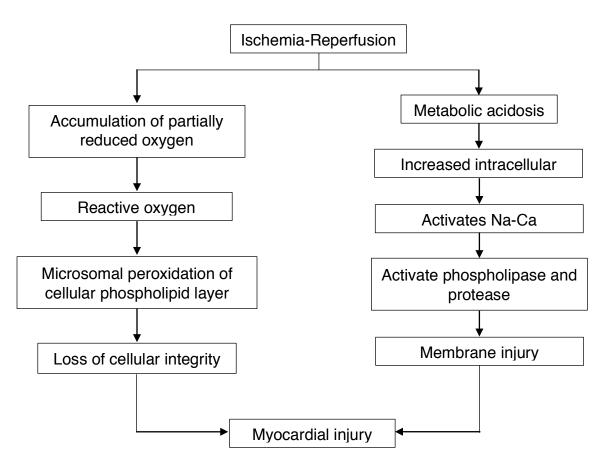
Stunning refers to mechanical dysfunction that persists after reperfusion in absence of myocellular damage. It is characterized by perfusion/ contraction mismatch. It occurs due to decrease in myofilament Ca⁺ responsiveness.

Hibernation is a chronic, potentially reversible state of segmental contractile dysfunction. Hibernation is a perfusion/ contraction match. It occurs due to decrease in Ca⁺ transits at cellular level.

Irreversible injury

Occurs by two morphologically distant pathways, necrosis and apoptosis. It results in ECG charges, increased cardiac markers and lasting abnormalities in ventricular function. Ultrastructural changes include-

- Cell swelling
- Contraction band necrosis
- Calcium loading of mitochondria
- Loss of glycogen granules



Mechanism of ischemia reperfusion injury

Stunning	Hibernation
Mechanical dysfunction that persists after reperfusion in absence of myocellular damage	Chronic, potentially reversible state of segmental contractile dysfunction.
Perfusion/contraction mismatch	Perfusion/contraction match
Decrease in myofilament Ca+ responsiveness	Decrease in Ca+ transit at cellular level.
Improves with resolution of myocardial edema and cardiac uploading	Improve with revascularization

Characteristics of dysfunctional but viable myocardium

Characteristics	Technique
Contractile reserve	Dobutamine Echocardiography
Intact cell membrane	Thallium 201 SPECT
Intact mitochondria	Tc-99m SPECT
Glucose metabolism	FDG imaging

Myocardial protection

Myocardial protection strategies

- 1. Cardioplegic arrest
- 2. Intermittent fibrillation
- 3. Off pump techniques
- 4. Deep hypothermic circulatory arrest

Cardioplegia

History

- In 1955, Melrose described cardioplegia using potassium citrate to induce cardiac arrest
- Hearse et al in St. Thomas hospital (London) developed St Thomas solution which provided reliable cardiac arrest and reasonable. myocardial protection.
- In 1979, Buckberg identified blood as optimal cardioplegic vehicles.
- Buckberg documented efficacy and safety of retrograde and combined Antegrade-retrograde infusion in valvar and coronary surgery
- Del Nido solution was used in 1990 at Boston children hospital, primarily for pediatric

Principle goals of cardioplegia are

- 1. Electromechanical arrest of myocardium
- 2. Avoidance of substrate depletion
- 3. Sustained cooling of myocardium and flushing out unwanted toxins
- 4. Limit myocardial edema and provide buffering capacity
- 5. Limit ischemia and reperfusion injury to myocardium

Delivery method

Goal is to provide adequate and uniform distribution of cardioplegia solution to the myocardium. Different delivery methods are-Antegrade, retrograde and combined

Antegrade cardioplegia

Can be delivered through

- Aortic root
- Coronary ostia
- Bypass conduits after distal anastomosis is completed



Fig-Antegrade cardioplegia needle

Advantages

- 1. It mimics the natural mechanisms of coronary flow through myocardial microcirculation
- 2. Simple method

Limitations

- 1. Antegrade cardioplegia delivery may be inadequate in situations in which coronary arterial circulation is severely diseased
- 2. Presence of aortic insufficiency limits its feasibility
- 3. Densely calcified ostia can result in dislodgement or dissection of tissue around the ostia
- 4. Lifting or retracting the heart for CABG or mitral valve procedures will distort

aortic valve to the point that it becomes incompetent. Therefore, in CABG, heart is repositioned and in mitral valve surgery, the retraction is released and then antegrade cardioplegia is given. This delays conduct of surgery.

Delivery through bypass grafts

<u>Advantages</u>

- 1. Cardioplegia is delivered to an area presumably underserved by antegrade coronary flow
- 2. Cardioplegia can be given without disruption of surgery
- 3. It gives an indication regarding runoff and patency of bypass grafts

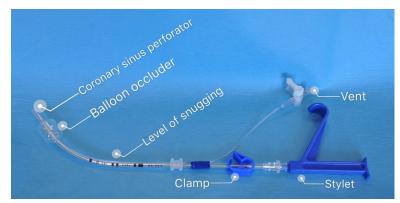
Retrograde cardioplegia

First used by Lillehei

It improves subendocardial perfusion

<u>Advantages</u>

- 1. It overcomes limitations of antegrade cardioplegia
- 2. It provides distribution of cardioplegia independent of coronary artery disease(CAD)
- 3. Cardioplegia can be delivered without disruption of surgery
- 4. Retrograde cardioplegia can be given for deairing of coronary arterial vasculature and aortic root.



Self retaining, retrograde cardioplegic cannula

Limitations

- 1. Retrograde catheters are difficult to place. TEE can be used to guide retrograde catheter placement
- Although retrograde delivery of cardioplegia is excellent to the left ventricle, the septum and RV are often underperfused. This problem can be solved by delivery of cardioplegia through right-sided vein grafts or through right coronary ostia.

- 3. Retrograde cardioplegia pressure should be maintained at 20-35 mm Hg. Pressures >50 mm Hg produce myocardial edema, hemorrhage and myocardial injury.
- 4. In presence of left SVC draining into coronary sinus, there is difficulty in maintaining arrested and cold myocardium.

Absolute contraindicates to retrograde cardioplegia

- 1. Left SVC
- 2. Unroofed coronary sinus

Method of putting retrograde cardioplegia

- Pursestring on RA-1 cm away from the AV groove and at the junction of upper 2/3rd and lower 1/3rd of RAA-IVC junction
- Keep volume in the heart
- Stab and hold the cannula with one hand keeping other hand over the coronary sinus
- Put cannula through RA anterior to two stage venous cannula & direct it posteriorly. Once the cannula enters CS opening, direct it towards left shoulder.
- Withdraw the style and see for pulsatile black blood (SpO2 at CS 25 %) coming out
- Connect it to pressure line to see the pressure tracings (4-5mmHg)

Combined Antegrade-retrograde cardioplegia technique

<u>Benefits</u>

- Prompt arrest
- Ensure distribution
- Limit cardioplegia volume
- Uninterrupted procedures
- Avoid ostial cannulation
- Flush coronary debris/air

Cardioplegia may be given as intermittent or continuous.

	Intermittent	Continuous
Advantages	Improved exposure Lower cardioplegia volume	Normal perfusion Increased post op LV function
Disadvantages	Increased inter dose myocardial aciosis	Operative field may not be dry complex

Cardioplegia composition

- Crystalloid cardioplegia
- Blood cardioplegia consist of 4 parts of blood to one part of crystalloid solution

Benefits of blood cardioplegia

- 1. Ability to carry oxygen
- 2. Excellent buffering capacity
- 3. Blood scavenge free radicals
- 4. Limit Hemodilution
- 5. Maintain oncotic pressure

Concerns associated with blood cardioplegia

- 1. Shift of oxyhemoglobin dissociation curve to left, thereby reducing release of O2 at tissue level
- 2. Potential of sludging and red cell rouleux formation

CABG patch trial

It was conducted in high-risk patients (Ejection fraction<35%) undergoing CABG to determine the contribution of cardioplegia composition and delivery route on morbidity and mortality of the patients.

Mortality was lower, myocardial infarction was less frequent and shock was less common in blood cardioplegia group

Use of inotrope use, IABP use and right ventricular dysfunction was less in patients receiving combined cardioplegia

Additives in cardioplegia

- 1. Nikorandil
- 2. L-Arginine
- 3. Insulin
- 4. Adenosine
- 5. Kreb's cycle intermediates -L Glutamate, Aspartate
- 6. Free radical scavangers-Allopurinol, Desferroxamine
- 7. Calcium antagonists-verapamil, diltiazem

Nikorandil

- It selectively open K ATP channel. Therefore cardiac arrest is achieved with less toxic doses of potassium containing cardioplegia
- Nikorandil may protect against perioperative coronary spasm
- Nikorandil acts as preconditioning agent.

<u>L-Arginine</u> – Minimize endothelial dysfunction by protecting coronary microcirculation by producing nitric oxide (NO)

<u>Insulin</u> – Evaluated as an adjunct to cardioplegia in an attempt to improve myocardial protection.

Studies show that, insulin enhanced cardioplegia did not provide superior myocardial protection.

<u>Adenosine</u> – Adenosine induces hyperpolarized cardiac arrest by antagonizing calcium channels and has been shown to inhibit both SA and AV nodes. Adenosine is also used during reperfusion period, which acts as substrate for ATP. It is a potent coronary dilator. It also retards no reflow effects through its antiplatelet and antineutrophil activity. Half-life of adenosine is 10 seconds. It is cleared through cellular uptake, primarily by erythrocytes and vascular endothelial cells.

Temperature of cardioplegia

By definition, warm cardioplegia refers to cardioplegia given at temperature of 34-35°C, while cold cardioplegia refers to cardioplegia given at 4°C.

There is disagreement over benefits of cardioplegia temperature in regards to myocardial protection.

Ascione et al – there is less myocardial injury and less ischemic stress in patients receiving cold cardioplegia

Franke et al – Warm cardioplegia was effective and potentially provided superior myocardial protection compared with cold cardioplegia

Warm induction is especially useful in energy depleted hearts like-

- Cardiogenic shock
- LVF
- LM disease with unstable angina

In such patients, warm cardioplegia is given for 5 minutes to replete energy stores. This is followed by delivery of cold cardioplegia.

St Thomas Cardioplegia

First described by Mark Brianbridge and Hearse

St Thomas I in 1975 and St Thomas II in 1981.

Composition (mmol/L)	St Thomas I	St Thomas II
К	20	16
Na	142	117
Mg	16	16
Са	1.7	1.2
HCO3	30-40	25
Procaine	1	1
рН	7.4	5.5-7
Osmolarity	320	294

Del Nido cardioplegia

Developed at Boston children hospital

It was originally developed for use in pediatric patients; its use for adult patients is expanding

It has a unique four parts crystalloid to one part whole blood formulation that is generally used in single dose formulation. Blood cardioplegia to preserve myocardial metabolism and function result in less repurfusion injury.

It contains base solution plasma Lyte A, which has electrolyte composition similar to extracellular fluid

Na	140 mEq/L
К	5 mEq/L
Mg	3 mEq/L
CI	98 mEq/L
Acetate	27 mEq/L
Gluconate	23 mEq/L
Са	Trace
рН	7.4

In 1 L plasma Lyte solution, following components are added

Mannitol 20%	16.3 ml	Scavenge free radical and reduce cell swelling
Mg 50%	4ml	Calcium channel blocker
Soda bicarb 8.4%	13ml	Buffering solution
KCL(2mEq/ml)	13ml	Deloparizing arrest
Lidocaine	13ml	Block sodium channel

Bretschneider's HTK solution (Custodiol)

HTK-Histidine, tryptophan, Ketoglutarate

<u>Uses</u>

- 1. Cardioplegia
- 2. Preservation of organs for transplantation

Properties of custodial

- 1. Low sodium concentration for safe organ inactivation
- 2. Histidine provides extended buffer
- 3. Tryptophan supports membrane integrity
- 4. Alpha ketoglutarate serves as a substrate for energy production

Component	Concentration (mmol/L)
Na	15
К	9
Mg	4
Histidine	18
Histidine hydrochloride monohydrate	18
Tryptophan	2
Mannitol	30
Са	Traces
Potassium hydrogen 2 ketoglutarate	1

Cardioplegia delivery

Dose of cardioplegia- 20 ml/ kg

Pressure at which cardioplegia is delivered

Antegrade	120 mm Hg-1st cardioplegia 75 mm Hg-in subsequent cardioplegia
Retrograde	25 mm Hg

Cardioplegia	Electromechanical arrest time	
St Thomas	20 minutes	
Custodiol	60 minutes	
Del Nido	120 minutes	

Phases of Myocardial Arrest

Phase	Strategy
Pre-arrest	Optimize hemodynamics
	Optimize anesthetic induction
	Early institution of partial CPB
Arrest	Warm substrate enhanced resuscitation
	Hyperkalemic arrest with maximum cardioplegic distribution
Maintenance	Hypothermic intermittent oxygenated cardioplegia with maximum distribution
Reperfusion	warm substrate enhanced, hypocalcemic cardioplegia
	Leukocyte depletion and free radical scavenge
Post-arrest	Modified Ultrafiltration
	Optimize hemodynamics
	Unload damaged myocardium – IABP, ECMO, LVAD

Integrated myocardial protection

It refers to a comprehensive cardioplegic strategy taking maximal advantage of each aforementioned cardioplegic method. It includes

- Warm/cold blood cardioplegia
- Antegrade/retrograde delivery
- Intermittent/ continuous perfusion
- Blood/crystalloid cardioplegia

<u>Microplegia</u> – cardioplegia technique that consists of mixing blood from CPB circuit with small quantities of concentrated additives, potassium being the most frequently used.

Infusion pump are used to deliver precise doses of additives. This minimizes quantity of crystalloid in the plegia.

Cardioplegia strategy – cardiogenic shock

- 1. Warm blood cardioplegic induction
- 2. Substrate enrichment (glutamate, aspartate)
- 3. Grafts are placed into contractile muscle first. Vessels supplying regions with smaller functioning mass are grafted next. The last grafts are placed into infracted region.
- 4. Distribution of cold cardioplegia through all grafts into newly revascularized segments is done after each anastomosis is completed
- 5. Warm blood cardioplegic reperfusion

Pediatric myocardial protection

Neonatal heart differs from adult heart in that

- 1. Immature myocardium is not able to increase stroke volume the same degree as adult myocardium per increase in preload. It is because-
 - Immature myocytes are smaller in size
 - There is increase in ratio of type I to type III collagen in neonatal hearts
 - Myofibrils may have incomplete sarcomeres
 - Mitochondria are present in fewer numbers
 - Only 30% of myocardial mass in the neonate comprises contractile tissue, compared with 60% in the mature myocardium
- 2. Immature myocardium has less well-developed sarcoplasmic reticulum and T tubule system. This is likely to account for the far greater dependency upon extracellular calcium demonstrated by immature myocardium. Calcium is therefore an effective short-term inotropic agent following Cardiotomy in the newborn and infant
- **3.** Neonatal myocyte metabolism is based on carbohydrate metabolism, in sharp contrast to mature myocardium that relies primarily upon free fatty acid metabolism. The immature myocardium has greater myocardial stores of glycogen and therefore, tolerates ischemia far better than mature myocardium.
- 4. Cardiac output in neonates in primarily heart rate dependent.

Ischemic Preconditioning (IPC)

First described by *Murry*.

<u>Definition</u> – It is a phenomenon in which an initial sublethal ischemic injury gives protection against subsequent ischemic insults.

Mechanism

Mechanisms have not been completely understood. However, the protective effects are related to Heat shock proteins, adenosine receptor, protein kinase and alterations in K ATP channel.

The effects of IPC are bimodal, consisting of

- Early phase -transitory with infarct limitation being lost if ischemia continues beyond 30-120 minutes
- Delayed late phase-occur 24-96 hours after induction of IPC.

Clinical application

The application of IPC remains to be determined. Studies have shown no benefit of IPC compared to old blood cardioplegia; because CPB per se induces preconditioning In OPCABG, IPC is induced by temporarily occluding coronary arteries before anastomosis. The results are conflicting. Some studies show significant lower cardiac troponin levels compared with control. Other studies show no significant difference between IPC and control groups.

Remote ischemic preconditioning

Defined as transient ischemia and reperfusion in a tissue remote from the heart, such as extremities, has been demonstrated to protect the heart after surgically induced myocardial ischemia by protecting mitochondrial integrity. The mechanism is thought to be systemic responses that interact with known IPC effectors. The cardioprotection afforded is limited and much reduced compared with IPC.

Post conditioning

It consists of repeated episodes of ischemia and reperfusion, which decreases effects of reperfusion injury. The mechanism appears to be similar to those of IPC.

SECTION VIII: CONDUCT OF CARDIOPULMONARY BYPASS (CPB)

When essentially all systemic venous blood returns to pump oxygenator instead of the heart, the situation is termed total cardiopulmonary bypass.

When some systemic venous blood returns to right heart and is pumped into the lungs, then passed back into left atrium and is pumped back by the left ventricle to the aorta, the situation is termed partial cardiopulmonary bypass.

Safe conduct of CPB includes

- 1. Selection of appropriate equipment
- 2. Assembly and priming of system
- 3. Completion of checklist
- 4. Initiation and reversal of heparin
- 5. Monitoring basic physiologic function and CPB circuit during bypass
- 6. Resumption of normal cardiopulmonary function once the patient is off bypass.

Surgeon, anesthesiologist, perfusionist and nurse should work together as a team for successful outcome. Before each operation, surgeon must develop a plan for conducting the operation, incision, method of cannulation, temperature desired and possible need for low flow or circulatory arrest.

- 1. <u>Selection of equipment</u> for most adult cardiac surgical procedures, the circuit is standardized. But for pediatric cases and for infrequently performed procedures, the circuit is often modified.
- 2. Assembly
 - The exact sequence of circuit assembly varies among perfusionist Components include oxygenator, Cardiotomy reservoir, cardioplegia delivery set and arterial reservoir. The components are connected with precut sterile tubing and mounted on CPB console.
 - Water source to heat exchanger and cardioplegia delivery system is then turned on and tested to verify adequate flow and absence of water leak.

Tubing Diameter for Different Patients Weights		
Weight	Arterial line(in.)	Venous line(in.)
<8	1/4	1/4
8-17	1/4	3/8
17-50	3/8	3/8
>50	3/8	1/2

3. Priming

Balanced electrolyte solution is then added to circuit. Tubing length is kept

minimum to decrease priming volume. The degree of Hemodilution may be calculated before bypass is initiated, and if the expected priming volume would cause an unacceptable anemia, occasionally packed red cells may be added to the circuit.

Pt blood volume × *wt*×*Hct*

Hemodilutional hematocrit = $\frac{1}{Pt blood volume \times wt + priming volume (ml)}$

- 4. Setting occlusion It is defined as a fall of 1 inch per minute of a column of water in tubing held vertically 30-40 inch above the roller pump Clinically, when the pump oxygenator is fully primed, the system is pressurized to 300 mmHg (measured by an onboard manometer) by applying a tubing clamp. The roller heads are adjusted to allow a slight pressure drop over 1 minute.
- 5. Pre CPB checklist should always be done and never omitted.
- 6. Heparinization protocol
 - Give 300 IU/Kg heparin into right atrium. Add 4 IU/ml in pump prime to CPB circuit.
 - Draw ACT after 3min. ACT must be >2.5 times baseline value before initiating CPB.
 - ACT is monitored every 20 minutes during normothermic CPB, every 30 minutes during
 - hypothermic CPB and more frequently if the patient shows heparin resistance.
 - Maintain ACT between 400-600 seconds during CPB
- 7. Heparin substitute Bivalirudin protocol
 - 50 mg in pump prime
 - 1mg/kg bolus 20 minutes prior to CPB •
 - 2.5mg/kg/h continuous infusion following bolus dose
 - Maintain ACT between 400-600 seconds during CPB
 - Check ACT every 10 minutes on bypass
 - Discontinue bivalirudin following termination of CPB
 - Do NOT use blood cardioplegia. Use crystalloid cardioplegia
- 8. After heparinization, surgeon taps arterial line to exclude any air bubbles. Recirculation is then stopped and arterial line is then clamped at the pump and table. The surgeon divides arterial line.
- 9. Aortic purestrings \rightarrow Heparinization \rightarrow cannulation
- 10. The perfusionist manually palpates pulsation on arterial flow line pressure monitor to rule out malposition of cannula. Venous cannulation is done. Upon instruction from the surgeon, CPB begins.
- 11. Initiation of CPB
 - a. Check ACT
 - b. Remove the arterial clamp and then slowly remove venous clamp
 - c. Gradually increase the pump flow

- d. Initial gas : Flow ratio of 1 : 1 with FiO2 of 80%
- e. Initiate water flow through heat exchanger.

Sudden release of venous clamp may cause chattering of line-due to vacuum effect, intima gets sucked into side holes of cannula resulting in sudden decrease in flow.

12. Systemic flow is most often indexed to patient BSA. Generally accepted indices are 2.2 to 2.4 L/min/m² or 50-65 ml/ kg when normothermic or when cooling. Higher indices are used in pediatric patients and rewarming.

Calculating blood flow rates in children			
Age(years)	Blood flow rate	Body weight(kg)	Blood flow rate(ml/min/kg)
0-2	BSA <i>×</i> 2.6	0-5	150
2-4	BSA <i>×</i> 2.5	6-10	125
4-6	BSA×2.4	11-15	100
6-9	BSA <i>×</i> 2.3	16-25	90
>9	BSA×2.2	>25	70

13. After commencement of cardiopulmonary bypass, heart should empty, as indicated by collapse of pulmonary artery, indicating good systemic venous drainage.

After going on CPB, if heart is still full then search for

- SVC/ IVC cannula too much in
- Kink in venous line
- Associated LSVC
- Associated PDA
- 14. The aorta is clamped and antegrade cardioplegia is delivered for 2 minutes at a rate of 200 ml/min. During cardioplegia delivery, distension of heart is avoided. If it occurs, ventricles should be promptly decompressed.

Indicators of cardioplegia delivery are

- Aortic root distension
- Heart stops immediately
- Generalized cooling of myocardium
- Coronary veins become bright red and distended
- Blood returning from opposite coronary ostia if direct coronary injection is given

If myocardial activity continues even after delivery of cardioplegia then possibilities are

- Partially applied clamp
- Aortic regurgitation
- Incomplete venous drainage

• K+ is not added

Intermittent delivery of oxygenated cardioplegic solution at an interval of 15-20 minutes ensures complete electromechanical arrest, allows for oxygen delivery, removes metabolic waste, buffers accumulated acids, delivers metabolic substrates and maintains the desired temperature of heart.

15. <u>Use of vents</u> – All vents should be tested before use by briefly immersing the tip of vent in a basin of saline to verify its suctioning effect. Sometimes, a small gauge needle is inserted in the vent line to relieve pressure.

Monitoring during bypass

Clinical variables

- Total systemic blood flow
- CVP, PA and LA pressures
- Urine output, temperature of perfusate and patient
- Depth of anesthesia

Laboratory variables

- Hct, ABG, CO2
- ACT
- Mixed venous saturation is used as an indicator of adequate tissue perfusion.
- ECG
- Neuromonitoring NIRS, Transcranial Doppler

Circuit variables

- Venous reservoir volume should be equal to 25% of systemic blood flow to allow for 15-second reaction time.
- Induction and reversal of hypothermia should be guided by maintenance of 8-10°C gradient between arterial blood and patient temperature to avoid emboli formation
- Systemic line pressure

Desired values on pump

ACT	>480 seconds	
	>350 seconds for biocompatible circuits	
Systemic Flow rates	2-2.5 L/min/m ² at 37°C	
	1.7-2.0 L/min/m ² at 30°C	
Systemic blood pressure	50-70mmHg	
Arterial blood gases	PO2>250 Torr, PCO2 40-50 Torr with pH7.4	
SVO2	>70%	
Hematocrit	>25%	
Blood glucose	100-180mg/dl	

ABG is performed every 10 minutes

If PaO2 decreases	Increase FiO2
If PaO2 increases	Decreased FiO2
If PCO2 decreases	Decrease gas flow rate
If PCO2 increases	Increase gas flow rate

Termination of CPB

- 1. <u>Rewarming</u> Temperature required for complete rewarming is 37 °C nasopharyngeal or 35°C rectal temperatures.
- 2. Controlled reperfusion using warm modified blood cardioplegia (Hot shot). The benefits of buffered, hypocalcemic, hyperosmolar, and substrate-enhanced reperfusion of ischemic myocardium are demonstrated.
- 3. Correction of metabolic abnormalities
 - Metabolic acidosis
 - Potassium-academia and administration of cardioplegic solution results in hyperkalemia.
 - Serum potassium levels fall after CPB due to rewarming and increased glucose utilization, dieresis and increased β_2 receptor stimulation from the stress of CPB.
 - Hypocalcaemia results from Hemodilution and transfusion of citrate containing blood products. If ionized hypocalcaemia is present (<0.8 mg/ dl) after complete rewarming, it should be corrected with calcium chloride to improve myocardial contractility and SVR. Otherwise calcium should not be administered to avoid risk of reperfusion injury.
 - Magnesium should be administered during CPB to provide vasodilatation of coronaries and to prevent arrhythmias
 - Hyperglycemia should be avoided as it increases risk of infection, causes osmotic dieresis and increase susceptibility of brain to hypoxic damage
- 4. <u>Anesthetic management</u> Narcotics and muscle relaxants are empirically administered. Endotracheal suctioning is performed
- 5. Deairing of heart
 - Patient is placed in 30° head low position
 - Caval tourniquets are loosened and heart is filled by reducing venous return to minimize air entrapment.
 - The lungs are intermittently ventilated to express air from pulmonary veins
 - Heart is gently massaged while the vent in LV continues to drain. Ventilation is resumed
 - Once heart resumes its contractions, Antegrade cardioplegia cannula is placed on suction.
 - Once the contractions are satisfactory, arterial pressure is reduced to 50 mm Hg and aortic clamp is removed, while suction in cardioplegia cannula is maintained.

- 6. Transesophageal echocardiogram
 - TEE provides an excellent assessment of residual air in the heart
 - Left ventricular short axis view provides a useful indicator of LV size and filling
 - Regional wall motion abnormality provides important indicator of myocardial ischemia and inadequate flow through a specific CABG graft
 - Evaluation of valvular function after valve repair
 - Evaluation of repair of complex congenital heart disease
- 7. Heart rate and rhythm
 - **a.** Ventricular fibrillation is a likely reperfusion arrhythmia due to calcium overload of ischemic myocardium. Lidocaine is often given before the cross clamp is released to prevent ventricular fibrillation. The heart should be immediately defibrillated as soon as possible. Electrolytes should be treated if abnormal. Recurrent ventricular fibrillation should be treated with amiodarone and repeat defibrillation.
 - **b.** Sinus bradycardia is treated with atrial pacing.
 - c. Sequential AV pacing is indicated for AV conduction block.
 - **d.** In patients with low ejection fraction and preoperative conduction system abnormalities, temporary biventricular pacing may improve postoperative hemodynamics and prevent LV dyssynchrony.
- 8. <u>Prophylactic inotropic support</u> Inotropic drugs are started at low dose (Dopamine at 5 ug/kg/min) to allow smooth separation from bypass without causing cardiac distension, hypotension and reinstitution of bypass.

Preexisting low EF, dilated left ventricle, elevated left ventricular end diastolic volume, longer duration of aortic cross clamp time and age are positive predictors of need for post operative intropic supports.

9. Termination of CPB

Ensure

- Contractility
- Color of myocardium
- Normal sinus rhythm
- Hemodynamics
- Temperature
- Arterial blood gases

- 1. Adjust rate and rhythm-pace if needed
- 2. Partially occlude venous line -fill the heart
- 3. Decrease arterial flow from the pump (partial bypass); ejection begins
- 4. Measure arterial blood pressure

P	1	¥
5.	Completely occlude venous.	5. Maintain partial bypass
6.	Stop arterial pump after ventricle seems appropriately	6. Carefully adjust ventricular volume
	full.	7. Begin appropriate vasoactive drugs
		 Reduce flow – readjust volume and drugs
		9. Stop arterial pump
	Ļ	Ļ

Estimate or measure preload and stroke volume (contractibility)

Steps of termination of bypass

- 1. Switch off gas flow
- 2. Switch off water circulation inside heat exchanger
- 3. Slowly reduce arterial pump flow and occlude the venous backflow
- 4. Clamp venous line
- 5. Clamp arterial line
- 10. Give test dose of protamine and continue protamine full dose keeping a watch on contractility of heart, pulmonary artery and hemodynamics. When protamine is 50% over, remove arterial cannula.

SECTION IX: TROUBLESHOOTING DURING CPB AND MANAGEMENT

Philip Kay and Christopher Munsch (2004) in" Techniques in Extracorporeal Circulation" state: "*Cardiopulmonary bypass is a dynamic artificial environment conferring a shock state on the body with its own potential for severe morbidity and mortality.*" Vigilance is thus paramount to the conduct of cardiopulmonary bypass. Human error is a far greater Cause of accidents than mechanical mishap. In this chapter, few important mishaps of cardiopulmonary bypass are described. Timely management of these mishaps can prevent further disasters.

1. Inadequate Blood Flow

Common causes of inadequate blood flow include-

- Incorrect orientation of cannula tip
- Small size of cannula
- Decreased volume-dehydration, hemorrhage
- Aortic dissection during cannula placement
- Undetected blood leaks either from the CPB circuit

Small quantity of venous line air rarely causes decrease in CPB blood flow. However, if the entire venous line becomes filled with air, the gravity effect is lost (referred to as air lock). In such scenario, CPB is temporarily stopped & venous line is clamped to allow surgeon to refill the venous with fluid. The air is then moved down the venous line into the venous reservoir to reestablish effective drainage.

2. Low Level In The Venous Reservoir

Common causes include

- Inappropriate size of venous cannula
- Systemic vasodilation
- Malposition, clamping or kinking of venous cannula
- The basic cause is to be searched for and treated accordingly

3. Inadequate Gas Exchange

Mechanical problems related to inadequate gas exchange by the oxygenator include **oxygenator failure and gas delivery system failures**. Oxygenator failure is diagnosed by observing dark-colored blood existing the oxygenator that cannot be corrected by increasing the concentration of oxygen in the ventilating gas. Causes may include

- Loss of gas supply to the CPB oxygen/air blender
- Failure of the blender
- Leaks or obstructions in the gas delivery system

Inadequate anticoagulation leading to clotting in the oxygenator can cause

inadequate gas transfer. The use of propofol, an anesthetic agent, administered directly into the membrane oxygenator can potentially affect gas transfer, presumably by blocking pores or otherwise interfering with the membrane surface in the blood-contacting compartment of a microporous membrane. Therefore, propofol should be administered peripherally to permit de-emulsification before it reaches the membrane oxygenator.

The patient who is either too lightly anesthetized or is hypothermic with inadequate muscle relaxation may have higher oxygen consumption that can lead to decreased mixed venous hemoglobin saturation and decreased arterial PO2 if left uncorrected.

4. Electrical Problems

Minor and major electrical problems are relatively common during CPB but only rarely cause adverse patient outcome. The entire CPB console or individual components can fail to operate as a result of wall power or electrical cord failures. Some newer CPB consoles have built in battery power to provide continuous function in the event of electrical failure. All CPB pumps should have provision for manual hand cracking readily available in the event backup electrical sources fail.

5. Air embolism:

Air embolism (venous or arterial) may occur not only from CPB (pump air) but during a variety of surgical procedures (surgical air) or at the head of the table from actions or inaction by anesthetic (anesthetic air).

Surgical (operative) air

In 1914, the danger of air embolism during cardiac surgery was reported by Carrel who wrote, "The opening of the ventricles or of the pulmonary artery and the aorta is always followed by entrance of air into the heart. "Support for his statement was the observation of fibrillation and death in animals after coronary artery air embolism.

CPB (pump) air

Air embolism originating from the CPB circuit may enter the patient's vascular system either from the arterial line or by other mechanisms. Arterial line air embolism due to emptying of the CPB reservoir is been a common cause. However, inattention to the reservoir level can still cause air to be transmitted to the CPB systemic flow line. High pump flows in conjunction with vertical CPB reservoir outlets may cause vortexing of air into the systemic flow line. Newer model reservoirs with angled or horizontal outlets are less prone to this condition. The arterial roller pump head tubing may rupture causing arterial air embolism

Accidental disconnects, punctures, cuts, or openings, such as stopcocks left open to atmosphere, in the arterial line can cause air embolism depending on flow conditions. Generally, such disruptions will cause blood loss from the circuit, but under low CPB systemic flow conditions, air may enter the arterial line.

Anesthetic air

Inappropriate ventilation of the patient during insertion of CPB cannulas or a left atrial monitoring line can cause air embolism. Expanding the lungs fully to displace pulmonary venous air is an important adjunct to surgical de-airing maneuvers; if not performed properly, air may be retained and later embolize to the arterial circulation.

Management of air embolism

If coronary arterial embolism results in myocardial dysfunction, the circulatory needs of the patient can be met by the CPB circuit. Systemic heparinization, a requisite for CPB, has been shown to be of benefit in reducing interactions of bubbles with blood but may be deleterious if brain infarction has occurred. Hemodilution to levels commonly used during CPB will reduce blood viscosity and improve tissue perfusion when air embolism occurs.

When the chest is open, the surgeon is able to aspirate air directly from the heart chambers or vessels. Venting, induction of hypothermia, retrograde coronary sinus or cerebral perfusion, or direct cardiac massage are all available if air embolism occurs during the open operation. If right coronary artery air embolism is suspected (isolated acute ST segment elevation in the inferior ECG leads is highly suggestive), the surgeon can transiently raise the pressure in the proximal aorta by gently pinching the aorta distal to the arterial cannula with the left hand while using the index finger of the other hand to access proximal aortic pressure. The transiently increased aortic pressure will push intracoronary air bubbles through the coronary circulation, which often can be seen if significant right coronary air embolism has occurred.

In cases of massive air embolism, packing the patient's head in ice will decrease cerebral metabolism and may be beneficial. Ventilating the patient with 100% oxygen favors bubble resolution and can limit cerebral ischemia.

If air has filled the arterial line, the arterial line can be disconnected from the arterial cannula. After clamping the venous cannula(e) and disconnecting the venous line, the arterial line and venous line are joined so perfusate can quickly recirculate back to the CPB reservoir to remove the air. Once cleared of air, the lines are reconnected to the appropriate cannulas and CPB may be resumed, Drug therapy for arterial air embolism is aimed first at raising the arterial blood

pressure to force bubbles through tissue vasculature to the venous side of the circulation. Corticosteroids, diuretics, antiplatelet agents, anticonvulsants, and barbiturates have been advocated to decrease cerebral manifestations of ischemic injury due to air embolism.

6. Infection To The Patient

The CPB circuit or its components may become contaminated by inattention to detail during setup or operation. The obvious risk of bacterial contamination via the patient's bloodstream necessitates observing sterile technique at all stages of use of CPB. Infection after cardiac surgery is an uncommon but serious complication leading to prolonged duration of mechanical ventilation and intensive care unit stay. One third of these patients may die of causes related to infection.

7. Drug Errors

The common drug error includes overdosage of either a vasodilator or vasoconstrictor. Although these types of errors may be short-lived and of no clinical consequence.

Drug errors related to anticoagulation can be fatal. Protamine administration during CPB can effectively render the oxygenator, reservoir, and arterial line filter unusable due to development of gross clot.

8. Incompatible Blood Transfusion

Another serious complication is transfusion of ABO-incompatible bank blood. The manifestation is an acute hemolytic reaction that can damage the kidneys. Prevention lies in adherence to strict double-checking of blood units against the patient's information. It can be life threatening.

9. Heparin Resistance

<u>Definition</u> – If ACT< 300 seconds after administration of up to 600 IU/kg heparin, it is hepatin resistance.

<u>Causes</u> – Heparin resistance may be seen in

- Antithrombin II deficiency -congenital/ acquired
- Prolonged administration of heparin
- Septicemia
- Thrombocytosis (platelet counts >7.00, 000/HL)
- Hypereosinophilic syndrome
- NTG treatment

<u>Treatment</u> – Administer 2 units FFP or 1000-2000 units Antithrombin III concentrate.

10. Hematologic problems

Cold agglutinins

Cold agglutinins are serum antibodies that become active at decreased blood temperature and produce agglutination or hemolysis of red blood cells. These antibodies are classically directed against antigens on the red blood cells but can also be nonspecific. The most clinically relevant characteristic of cold agglutinins is thermal amplitude, the temperature below which the antibodies become activated. As temperature drops below this threshold, antibody activity increases exponentially. In general, this activity reverses as rewarming occurs.

Rewarming activates the complement system to induce hemolysis in patients with cold agglutinin. For hemolysis to occur, the cold agglutinins and complement activities must overlap. That is, the temperature must be low enough for the cold agglutinins to activate but warm enough for a complement fixation to occur.

Safety Devices

Gas bubble detector

The gas bubble detector is uses a light source and detector to detect bubble in the arterial line distal to the arterial pump head. The device is wired in to the motor controller and will stop one ml bubble within one foot of the device.

Level sensor

The level sensor fall into one of the several systems such as

- Capacitance system
- Weight system
- Light system

Capacitance system

It uses a strip of metal tape, which is placed on the side of the oxygenator at whatever level the perfusionist wants the alarm to sound. It is only the audible alarm and does nothing to stop the pump.

Light system

It is the device which attaches to the oxygenator holder and is moved up against the side of the oxygenator at whatever levels the perfusionist wishes the device to alarm. As long as blood is in front of the light source, then the right is reflected back on the sensor. When the sensor does not see a reflected right, the device alarms. The device can be set in the automatic mode to automatically stop the arterial pump if it should alarm. The disadvantage of this device is that if clot should form in front of the light source, then the sensor will always see reflected light.

Weight system

The weighting device actually weights the holder, oxygenator and contents of the oxygenator. As the weight in the oxygenator increases due to increasing volume the arterial pump head runs faster in order to maintain a set arterial reservoir level. The tragic effect in this device is that the device cannot differentiate blood in the arterial reservoir and someone leaning on the weight arm.

Arterial line filter

The arterial filter is also function as bubble trap. Many perfusionists use the vent line to measure the pressure in the arterial line filter to monitor loading of the filter. The use of the filter in this configuration gives the perfusionist a safety margin of only an average of 3.2 seconds.

Left heart vent valve

It is the connector with a duckbill-type rubber valve inside it. It has an extremely small orifice which increases hemolysis. Manufacturers of the device claim that air cannot be pumped into the patient should the vent be reversed through the pump head using this valve.

"The best safety device available today is the perfusionist. One who is well-trained experienced and qualified to handle routine as well as emergency situations."

SECTION X: ORGAN DAMAGE DURING CARDIOPULMONARY BYPASS

Organ damage during cardiac surgery has been primarily attributed to the use of cardiopulmonary bypass (CPB).

Triggers Of Organ Damage

The key mechanisms in causing organ damage associated with CPB are

- 1. Activation of systemic inflammatory response syndrome (SIRS) following CPB
- 2. Hemodilution and reduced blood viscosity
- 3. Ischemia/reperfusion injury to heart, lungs and organs supplied by the splanchnic
- 4. Circulation
- 5. Laminar rather than pulsatile flow during CP

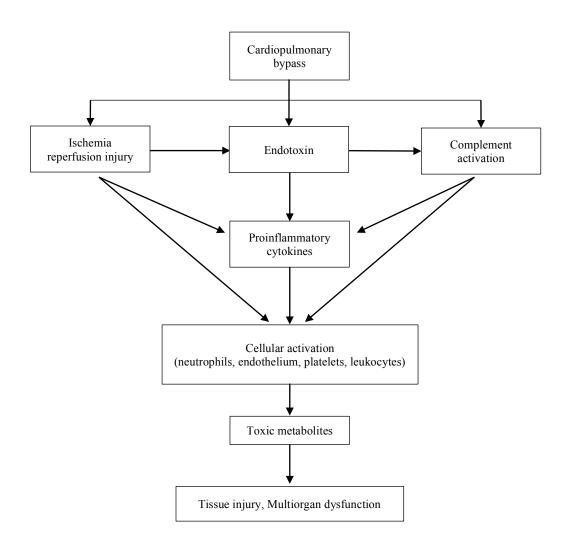


Fig-Mechanism of SIRS following CPB

Systemic inflammatory response syndrome (SIRS) following CPB occurs due to

- Activation of complement
- Activation of fibrinolytic and kallikrein cascades
- Synthesis of cytokines
- Oxygen radical production
- Activation of neutrophils with degranulation and release of protease enzymes.

Ischemia-reperfusion injury

It refers to cellular injury that occurs on resumption of normal perfusion to an organ after a period of relative or complete ischemia. During the ischemic period, intracellular calcium accumulates due to the failure of ATP-dependent cellular pumps. On reperfusion, intracellular calcium levels further increase secondary to oxidative dysfunction of sarcolemma membranes. This cellular and mitochondrial calcium overload ultimately

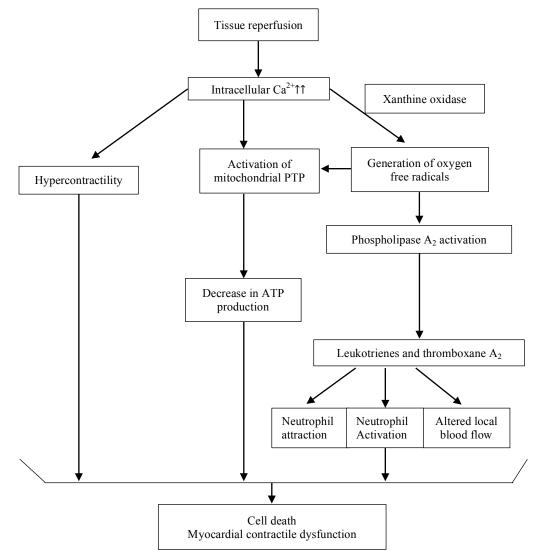


Fig-Ischemia reperfusion injury

induces cardiomyocyte death.

Organ damage following CPB

- 1. Post-operative bleeding
- 2. Thromboembolism
- 3. Neurocognitive dysfunction -Stroke, Delirium, Focal neurological deficit
- 4. Kidney- Acute renal shutdown requiring dialysis
- 5. Pulmonary-Atelectasis, Adult Respiratory Distress Syndrome (ARDS)
- 6. Gastrointestinal-Cholecystitis, Pancreatitis, GI bleed
- 7. Myocardial dysfunction
- 8. Multi-organ failure

Therapeutic strategies

A number of strategies have been employed to ameliorate the extent of the SIRS seen during CPB.

- 1. <u>Pharmacological</u> using steroids prior to the onset of CPB, antioxidants and proteolytic enzyme inhibitors. None of these interventions have had clinically meaningful impact.
- <u>Heparin-bonded circuitry</u> can be used with the intention of reducing the degree of complement activation, but has proven to be less effective in attenuating coagulation or fibrinolysis
- 3. <u>Hemofiltration/ultrafiltration</u> has shown to reduce the circulating levels of pro-inflammatory mediators
- 4. <u>Leukocyte-depleting filters</u> incorporated in the CPB circuit, to reduce the number of circulating activated white cells. Their value is presently unclear, but leukocyte depletion may have a protective effect in reducing the severity of lung and myocardial injury observed post-CPB. The most consistent benefit is found in high-risk patients with pre-existing lung disease, ventricular dysfunction or those receiving long CPB times.

SECTION XI: DEEP HYPOTHERMIC CIRCULATORY ARREST (DHCA)

Introduction

Cardiac arrest with cardiopulmonary bypass (CPB) to maintain perfusion to other organs. However, in certain situations, the nature of the surgical procedure or the pathology of the underlying condition necessitates complete cessation of blood flow. For example, safe removal of large tumors encroaching on vascular structures requires provision of a bloodless field to enable dissection, or operation on the aorta itself may preclude application of a cross-clamp because of the pathological anatomy. Preservation of organ function during the period of total circulatory arrest can be aided by reducing the core temperature of the body. The technique of core cooling combined with cessation of blood flow is termed "deep hypothermic circulatory arrest" (DHCA).

Definition

It refers to complete cessation of cardiopulmonary bypass (CPB) flow at a core body temperature of less than18°C.

Advantages of DHCA

- 1. Clarity of surgical field free of blood and ancillary equipment
- 2. Shorter exposure to CPB
- 3. Reduced edema

Indications of DHCA

- 1. Cardiac
 - Aortic surgery
 - Pulmonary thromboendarterectomy
 - Complex neonatal surgery
- 2. Neurologic
 - Cerebral aneurysms
 - Arteriovenous malformations
- 3. Other Renal cell carcinoma with caval invasion

Effects of hypothermia

Advantages

- 1. It reduces metabolic rate and O2 consumption
- 2. It reduces excitatory neurotransmitter release
- 3. It slows down enzymatic reactions
- 4. It decreases inflammatory response to CPB

Disadvantages

1. Arrhythmias

- 2. Increased plasma viscosity
- 3. Vasoconstriction impairing microcirculation
- 4. Impaired coagulation
- 5. Metabolic acidosis
- 6. Hyperglycemia
- 7. Effects on pharmacodynamics and pharmacokinetics

Surgical technique

Cardiopulmonary bypass

Aortic cannulation. Single venous cannulation into right atrial appendage. Patient on CPB. Cooling is started. During cooling, temperature gradient is kept 10°C to avoid crash cooling, which is linked with neurologic dysfunction. Cooling duration is minimum of 20 minutes and the head is packed with ice to reduce ambient temperature. A nasopharyngeal temperature of 16-18°C and EEG silence is a reasonable criterion to establish circulatory arrest. A single infusion of cardioplegic solution will usually suffice if circulatory arrest does not exceed 30 minutes.

Circulatory arrest

Once the circulatory arrest is achieved, right atrial cannula is removed from the heart. If arch reconstruction is a part of the operation, the aortic cannula is removed as well. The heart and great arteries are opened where appropriate and repair is performed. When the intracardiac repair is completed, cardiotomy is closed.

Rewarming

The aortic and right atrial cannula are reinserted. When CPB is resumed after a period of DHCA, hypothermic perfusion should be maintained for 10 - 20 minutes before rewarming commences. This reduces risk of raised intracranial pressure.

During rewarming, temperature gradient should not exceed 10°C to avoid air emboli. Rewarming should be more gradual, over a period of 30 minutes, to prevent rebound hypothermia after separation of CPB.

Cerebral protection

Cerebral damage may occur due to

- Inadequate cerebral perfusion
- Cerebral edema
- Micro emboli

In many centers, the duration of safe DHCA is extended by use of selective Antegrade cerebral perfusion (SACP) or retrograde cerebral perfusion(RCP).

Techniques of cerebral perfusion

Antegrade – with a side graft attached to right subclavian artery Retrograde – with a cannula in superior vena cava

	Selective Antegrade Cerebral Perfusion	Retrograde Cerebral Perfusion
Advantages	Prolongs safe length of DHCA more than RCP	Helps maintain cerebral cooling
Disadvantages	Risk of embolism	Cerebral edema Raised ICP

Continuous low flow selective cerebral perfusion is associated with improved neurological outcomes. Flow is administered at the rate of 10-15 ml/kg/min.

<u>Pharmacologic neuroprotection</u> – Drugs used for this purpose include thiopental, propofol, steroids and mannitol.

Neurologic monitoring during DHCA

- Transcranial doppler sonography
- Near infrared spectroscopy
- EEG

Near Infrared Spectroscope(NIRS)

It is a non-invasive technique that provides continuous real time assessment of microcirculatory oxygenation at patient's bedside. The optical range of infrared light is 700-900 nm. Within this range, oxygenated and deoxygenated Hb has different absorption spectra.

NIRS can provide real time information about tissue oxygenation and tissue perfusion

It is used in operating room or ICU

Uses

- 1. Regional cerebral perfusion in aortic arch surgery
- 2. Lumber NIRS- during surgical repair of coarctation of aorta

Acid base management

pH stat strategy leads to increased cerebral oxygen delivery and is thought to give more uniform cooling of the brain at the expense of increasing number of microemboli to brain.

It is recommended that pH stat strategy is used during cooling before DHCA. During

rewarming, the use of alpha stat strategy is beneficial as it prevents increased cerebral blood flow and the risk of cerebral edema.

Glycemic control

Hyperglycemia during periods of hypothermia worsens the impact of ischemia through increased glycolysis and intracellular acidosis. Virtually, all patients undergoing DHCA develop impairment of glucose metabolism and will require control of glucose with insulin.

Summary of Boston circulatory arrest trial-

- 1. Most patients tolerate 30 minutes of DHCA without significant neurologic dysfunction
- 2. DHCA>40 minutes is associated with increased risk of neurological injury
- 3. Uninterrupted DHCA >60 minutes is universally perceived detrimental
- 4. Continuous perfusion techniques or intermittent perfusion should be employed if lengthy repairs are anticipated

With today's techniques of circulatory arrest i.e hematocrit 30 %+ pH stat strategy, hyperoxygenation, intermittent reperfusion at 20-30 minute intervals and avoidance of a total duration>1 hour, it is highly unlikely that any differences could be detected in neurodevelopmental outcome relative to continuous cardiopulmonary bypass.

SECTION XII: NON-CARDIAC USES OF CARDIOPULMONARY BYPASS

The majority of cardiac operations performed use CPB, but CPB can be an essential adjunct in certain non-cardiac procedures. This chapter discusses various indications to non-cardiac uses of cardiopulmonary bypass.

Non-cardiac surgical applications of cardiopulmonary bypass (CPB)

- 1. Profound hypothermia with circulatory arrest-for repair of vascular anomalies such as intracranial aneurysms. Studies show that a core temperature of 15°C provides up to 60 minutes of safe ischemic time during circulatory arrest.
- 2. Urology-Renal cell carcinoma extending into IVC
- 3. Thoracic procedures-CPB is required for carinal tumour resection, tracheal procedures, resection of large anterior mediastinal masses and massive pulmonary embolism
- 4. Isolated left heart bypass- Tandem heart pVAD (percutaneous VAD) is used for left ventricular bypass. Femoral vein is cannulated and a 21 F catheter is threaded into right atrium and across the interatrial septum into left atrium(LA). Oxygenated blood is drained from LA through femoral venous cannula and directed to a centrifugal pump. The blood is then pumped into femoral arterial cannula in a continuous, non-pulsatile manner. The system has no reservoir, no oxygenator. The device can deliver flows up to 2-5L/min.

<u>Uses</u>

- Cardiogenic shock following myocardial infarction
- LVAD following cardiac surgery
- Percutaneous coronary angioplasty of patients with left main coronary lesions who are not surgical candidates and the risk of cardiac collapse is high
- 5. Venovenous bypass-It differs from CPB in that-
 - Oxygenator is not used
 - Arterial system is not perfused. Therefore, pressures within the system are lower.

Indications

- Respiratory failure
- Liver transplantation, during anhepatic phase

The cannula is placed into femoral vein \rightarrow centrifugal pump \rightarrow axillary vein. Flow rate is 1-2 L/min

<u>Drawbacks</u>

- Flows must be maintained to prevent clot development in the circuit as well as to provide adequate right heart filling
- System is closed, so no volume can be added directly once the circuit is primed
- 6. Accidental hypothermia patient can be more rapidly rewarmed. There is decreased risk Ventricular Fibrillation and if Ventricular Fibrillation occurs, circulation and oxygenation can be supported until heart is defibrillated.
- 7. Overdose of heroin or barbiturates can also be successfully resuscitated with

bypass. One of the advantages of bypass is the ability to include a filter to dialyse barbiturates.

8. Orthopedic – bypass techniques are used to administer high concentration of chemotherapeutic agents to discrete regions of body, mostly neoplasms of lower extremity

SECTION XIII: HEPARIN PROTAMINE AXIS

Heparin

Introduction

Discovered by Mc Lean in 1916 from the liver extract (hence the name) It is glycosaminoglycan derived from porcine intestinal mucosa (previously from beef lung)

Advantages of mucosal heparin

- Lower mean molecular weight-more effectively inhibits factor Xa
- Lower cost
- More cross-linked polysaccharide structures

Disadvantage of mucosal heparin

 Protamine partially neutralizes effect → greater postoperative bleeding Unfractionated heparin (UFH) consists of spectrum of molecular weights between 1000-30000 Da with a mean of 15,000 Da

1 unit of heparin is the amount of heparin that is required to maintain fluidity of 1 ml of citrated sheep blood for 1 hr, at 37° C

Mechanism of action

Heparin binds to antithrombin III and brings a conformational change in it leading to its activation. Antithrombin III inhibits coagulation factor thrombin (IIa), Xa, IXa, XIa and XIIa. Anticoagulation thus depends on presence of adequate amounts of circulating antithrombin.

Half-life

Heparin is cleared by two mechanisms

- Rapid-binding to endothelial cell receptors and macrophages
- Slow- renal

This makes anticoagulant response of heparin non linear at therapeutic doses, with both duration and intensity of the effect rising disproportionately with increasing doses. Thus biological half-life increases from 30 minutes with IV bolus of 25 IU/kg to 60 minutes with IV bolus of 100 IU/Kg to 150 minutes with IV bolus of 400 IU/Kg.

Heparinization protocol on CPB

Give 300 IU/Kg heparin into right atrium. Add 4 IU/ml in pump prime to CPB circuit. Draw ACT after 3min. ACT must be >2.5 times baseline value before initiating CPB ACT is monitored every 20 minutes during normothermic CPB, every 30 minutes during hypothermic CPB and more frequently if the patient shows heparin resistance.

Maintain ACT between 400-600 seconds during CPB

Cardiovascular Procedure	Heparin Dosage
Vascular	100IU/Kg
OPCABG	200IU/Kg
ONCABG and acyanotic heart disease	300IU/Kg
Cyanotic heart disease	400IU/Kg

<u>Heparin protocol in ACS</u> – Bolus dose of 70 IU/ Kg followed by infusion of 12 IU/ kg to obtain aPTT levels of 60-80 seconds

<u>Heparin protocol in PTCA</u> – Bolus dose of 100IU/Kg to maintain ACT of 250-300seconds.

Heparin protocol in DVT – Bolus dose of 80IU/Kg followed by 18 IU/kg/min.

ACT monitoring

ACT is an automated test and it involves addition of blood to a test tube containing blood activator (celite ot kaolin)

- 1. Hemochron ACT device- when a clot forms, resistance to movement of a small magnet in the tube is detected and the timer stops.
- 2. Hemotec ACT device-uses kaolin as an activator, uses a plastic plunger for continuous mixing. The absence of plunger fall after a clot forms is detected photooptically. It is preferred when aprotinin is used.

Procedure	Target ACT (seconds)
Ventricular assist divices	180
ECMO	200
Minimum safe ACT for cardiopulmonary bypass	300
Before initiating cardiopulmonary bypass	480

Side effects of heparin

- It decreases systemic vascular resistance by decreasing amount of ionized calcium
- Disseminated intravascular coagulation
- Osteopenia
- Heparin induced thrombocytopenia
- Heparin resistance

HITT(Heparin induced Thrombocytopenia and Thrombosis)

It is characterized by 50% reduction in platelet count.

Incidence is 1-3%. It is higher with bovine heparin.

Two types

- 1. Type I HITT- mild decrease in platelet count due to proaggregatory effects of heparin. on platelets.
- Type II HITT- more severe form, often occurs >5 days of heparin administration and is immune mediated. It is an IgG mediated, heparin-PF4 complex on the platelet surface is the target antigen. Ongoing platelet activation results in increased thrombin production and a systemic hypercoagulable state. ELISA test is used to confirm the diagnosis.

<u>Treatment</u>

- Patients with HITT 1 can receive heparin safely
- Patients with history of HITT II with undetectable antibodies and who have not received heparin for >90 days can receive heparin for CPB
- Patients with active HITT II should be anticoagulated with alternative anticoagulant for CPB. Bivalirudin is the alternative agent when HITT is a concern.

Heparin resistance

Definition

If ACT<300 seconds after administration of up to 600 IU/kg heparin, it is heparin resistance.

<u>Causes</u>

Heparin resistance may be seen in

- Anithrombin III deficiency-congenital/acquired
- Prolonged administration of heparin
- Septicemia

- Thrombocytosis (platelet counts >7,00, 000/ul)
- Hypereosinophilic syndrome
- NTG treatment

Acquired AT III deficiency is seen in

- Decreased synthesis- liver cirrhosis
- Drugs-Estrogen, heparin, L Asparginase
- Increased excretion -nephritic syndrome, inflammatory bowel disorder
- Increased consumption-DIC, surgery
- Diltional-cardiopulmonary bypass

<u>Treatment</u>

Administer 2 units FFP or 1000-2000 units Antithrombin III concentrate.

Heparin reversal

UFH is reversed with protamine sulfate -1-1.3 mg of protamine for each 100 IU of heparin.

Heparin rebound

Post CPB, after neutralization of heparin with protamine, the heparin that is bound to plasma proteins does not get neutralized. This is released after few hours leading to free heparin in the blood. It should be suspected if signs of bleeding appear after a period of dryness in the operating field.

Alternative to UFH in cardiopulmonary bypass

These should be considered in situations where heparin should be avoided e.g. heparin allergy, protamine allergy, HITT.

These include

- 1. Direct thrombin inhibitors (Hirudin, bivalirudin and argatroban)-hold promise as the best alternative. Hirudin is monitored using ecrine clotting time. Bivalirudin has been demonstrated as safe as heparin for on pump surgery. It has short half time.
- 2. LMWH is not suitable, as there is >90% immune cross reactivity with UFH and significant bleeding.
- 3. Heparinoid(Danaparoid)-may be useful, but no neutralizing agent
- 4. Ancrod-derived from snake venom
- 5. Fondaparinaux-synthetic pentasaccharide, selectively binds antithrombin and causes rapid and predictable inhibition of factor Xa.

Protamine

Originally, it was derived from salmon sperm but now it is produced primarily through recombinant biotechnology.

Chargoff and Olson in 1937 discovered that heparin can be neutralized by peptide protamine.

It is strongly alkaline because its amino acid composition is 67% arginine. Protamine can immediately reverse anticoagulation effects of heparin by a non specific acid base interaction.

Other clinical uses

- 1. Protamine zinc insulin
- 2. NPH insulin

Assessment of reversal of anticoagulation

- 1. Fixed protamine dose regimen- 1-1.3 mg of protamine for each 100 IU of heparin
- 2. Heparin activated clotting time dose response curve -It involves determining three ACT values and plotting them on a graph Vs heparin dose. The calculated amount of heparin is then neutralized by giving 1.3 mg of protamine per 100 IU of heparin at the conclusion of CPB.
- 3. Heparin levels
- 4. Protamine titration

Method	Advantage	Disadvantage
Fixed dose	Simple	Inadequate or excessive protamine
ACT Heparin dose response curve	Rapid, easy to use in OR More accurate protamine administration	Relies on ACT
Heparin levels	Less protamine given	Requires peripheral lab Time consuming Assumes point on static curve
Protamine titration	Less protamine given No rebound effect seen with small protamine doses	Dependence on blood volume estimate Several steps for potential error Assumes point on static curve

Other drugs used to neutralise heparin

- Platelet factor 4- Recombinant PF4 Is cloned in E. Coli. It neutralizes heparin as effectively as protamine in vitro. Advantages
 - PF 4 has no effect on WBC count, platelet count or complement levels, unlike protamine.
 - No hypotension or pulmonary vasoconstriction, unlike protamine

- Can be administered rapidly within 2 minutes
- Disadvantage emergency return to CPB makes heparinisation difficult
- 2. Protamine variants (Designer protamine)
 - +18 BE(Standard protamine is +21 BE)
 - Low molecular weight protamine molecule with RGD side chain (Arginine, Glycine and aspartate
- 3. Heparinase neutralises heparin by enzymatic cleavage of alpha glycosidic linkage at antithrombin III binding site. Emergency return to CPB makes heparinisation difficult
- 4. <u>Heparin removal devices</u> The principle of plasmapheresis is applied in the heparin removal device system to expose heparinised plasma from patient to poly L lysine. The negatively charged heparin molecules bind irreversibly with positively charged poly L lysine and are removed from plasma. The time taken to reverse heparin activity is 30 minutes which is currently unacceptable for routine clinical practice.

Protamine reaction

Classification (Horrow)

Туре І	Hypotension from rapid administration due to histamine release
Type IIa	Anaphylactic reactions (Ig E)
Type IIb	Immediate anaphylactoid reactions (IgG)
Type IIc	Delayed anaphylacroid reactions (IgG)
Type III	Pulmonary vasoconstriction (IgG/complement mediated)

True anaphylactic reaction are seen in

- 1. Patients with prior protamine exposure
- 2. Patients with fish allergy
- 3. Vasectomized patients
- 4. Patients receiving NPH or protamine zinc insulin

Type III reaction-Complement mediated pulmonary vasoconstriction

<u>Mechanism</u>

Uptake of protamine-heparin complex in pulmonary macrophages Non immunologic activation of assic complement pathway Release of Thromboxane Pulmonary vasoconstriction

- Incidence is1-2%
- It can be decreased by giving drug slowly and giving the drug on the left side of circulation.
- Diagnosis is clinical. Screening antibodies IgG and IgE are assessed with Radioimmune assay.

Prevention and therapy

- 1. Slow administration of protamine. In practice, a rate of 15 mg/min is well tolerated.
- 2. Stop infusion at the first sign of any adverse reaction.
- 3. Treat anaphylaxis with fluids and adrenalin
- 4. Supportive strategies
- 5. Reinstitution of bypass may be considered
- 6. Nitric oxide inhalation

The episode usually abates after few minutes. Protamine may be given at the rate 5mg/min following resolution of episodes and after resumption of hemodynamic stability.

SECTION XIV: PERIOPERATIVE CELL SALVAGE

Cell salvage and washing system

Allogenic RBC transfusion is associated with well-known adverse effects. These include febrile, anaphylactic and hemolytic transfusion reactions, transfusion related acute lung injury and infection risk.

Use of autologous blood is attractive as being less harmful than donated, allogeneic blood. Methods for autologous blood transfusion include

- 1. Predeposition
- 2. Perioperative normovolemic Hemodilution
- 3. Perioperative cell salvage

Cell salvage system

Principle

Blood from the surgical field is collected, filtered, washed and transfused back to patient. This technique can be used during intraoperative or early postoperative period

Types of cell salvage system

Continuous auto transfusion system (CATS)-Fresenius Terumo Discontinuous auto transfusion system (DATS)-Medtronic sequestra 1000

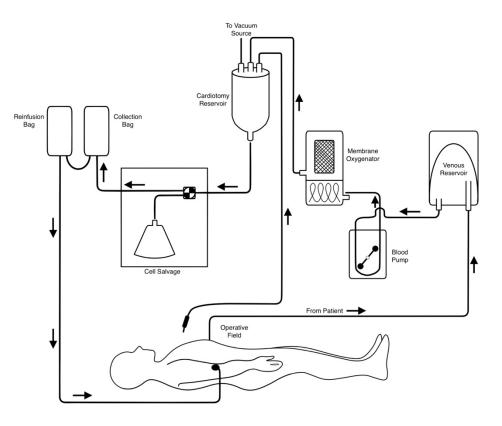
Procedure

 <u>Suction</u> – The blood is sucked away from the operative site via a double lumen tube, which mixes blood immediately with anticoagulant into a custom made reservoir.

The anticoagulant used is heparin or citrate Heparin saline is usually prepared with 30,000 units of heparin in 1 L of normal saline and mixed with blood at a rate of 40-60 drops/min.

- <u>Filtration</u> The contents of reservoir are filtered to remove large clots and debris. Most collection reservoirs have filters in the 40-150 um range.
- <u>Separation</u> The remaining volume is drawn into a centrifuge to be processed. Sterile isotonic saline is pumped into centrifuge bowl. Centrifuge process separates more dense RBC from less dense plasma, where it spills into waste bag.
- <u>Disposal</u> Waste products, including white blood cells, platelets, plasma, anticoagulant, fat, clotting factors and free Hb are collected in a bag and are disposed as a clinical waste.
- 5. <u>Cell salvage</u> packed RBC are collected in a separate bag kept at room

temperature. The collected red cells are reinfused immediately or for up to 4 hr after processing it kept at room temperature.



Cell salvage system

Advantages

- 1. Reduce demand for allogenic blood
 - Reduce risk of transmission of infection
 - No risk of blood incompatibility
 - Option in case of rare blood group
 - Acceptable for Jeehovah's witness
- 2. Removal of activated clotting factors and inflammatory cytokines
- 3. Cells reinfused at room temperature.

Disadvantages

- 1. Cost of the initial set up, disposables and staff training
- 2. Delay in processing and blood availability
- 3. Loss of plasma proteins, coagulation factors and platelets
- 4. Need for operator time and attention
- 5. Risk of bacterial contamination

SECTION XV: OFF PUMP CABG-ROLE OF PERFUSIONIST

In this technique, the entire operation is performed without using heart lung machine.

Off pump coronary artery bypass surgery is an alternative to on pump CABG. The comparison of long term results of both the techniques are still debatable and largely dependent on surgeon preferences.

The main benefits of off pump CABG are

- It avoids pump related complications
- Full heparinization is avoided
- Hemodilution is avoided
- Less blood transfusion is required
- Oxidative stress and markers of inflammation (IL-8, TNF and E-selectin) are significantly reduced

Myocardial protection for off pump CABG

During Off pump CABG, myocardial protection should be maintained. This is achieved by various means

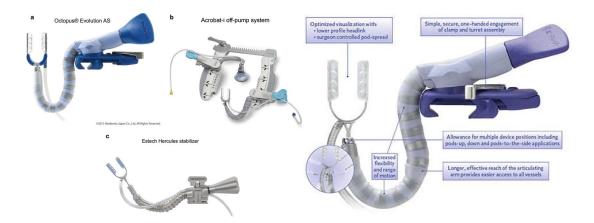
- 1. Maintenance of adequate systemic blood pressure
- Thoughtful selection of order of distal anastomosis As a general rule, collateralized vessels are grafted first. The collateralizing vessel can then be safely grafted.
- 3. Careful use of traction sutures, apical heart positioners and coronary stabilizers
- 4. Intracoronal shunts
- 5. IABP counterpulsation

Self-retaining coronary stabilizers

It consists of two pronged forks attached to retractor by means of an articulating arm. These stabilizers are connected to suction tubings (-100 to -300 mmHg). The epicardium is sucked into these ports, allowing the stabilizer to grip the heart.

They have added advantage of providing traction and countertraction of the fat surrounding the coronary artery, making them well suited for coronaries deep within the fat.

Currently available stabilizers are **Octopus evolution (Medtronics) and ACROBAT** (Maquet).



Three kinds of stabilizer and Octopus evolution stabilizer

Limitations of suction stabilizer

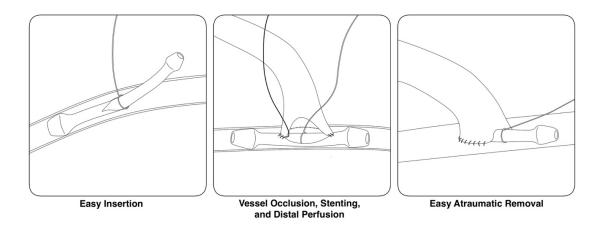
- Myocardial damage to epicardium and myocardium (suction injury)
- Produce arrhythmogenic areas
- Damage to venules

Apical suction heart positioner

Starfish (Medtronics) and Xpose (Maquet) It displaces the heart by holding the apex with a suction cap and fixing the position with an articulating arm.

Intracoronary shunts (clearview)

Range in size from 1.0 to 3.0 mm. It provides significant flow to distal vessels and a reasonable bloodless field. The major advantages of the shunt are myocardial protection and hemostatic effects. The shunt allows distal perfusion while maintaining Hemostasis of the anastomotic site. The major drawback of shunt is endothelial injury during insertion.



If patient is not maintaining hemodynamics during surgery then IABP needs to be inserted.

During surgery, if heart is arrested suddenly and not responding to cardiac massage and inotropes, then patent needs to be put on emergency cardiopulmonary bypass. Perfusionist should be well prepared with these two dreaded complications before taking patient in operation room.

Checklist for off pump CABG

- Confirm IABP assembly and Balloon
- Confirm CPB assembly.
- Confirm availability of assembly for off pump CABG-shunts, octopus, mist blower
- Discuss every case preoperatively with surgeon. Complications are more likely to happen in patients with low ejection fraction (<30 %)or patients with Left main disease.

SECTION XVI: INTRA-AORTIC BALLOON COUNTERPULSATION

Concept was put by Moulopolous and clinical application was by Kantrowitz

It is used to support hemodynamics in cardiogenic shock and to relieve medically refractory ischemia in patients with severe coronary disease.

Indications

- 1. Cardiogenic shock
 - Bridge to revascularization IABP placement is an ACC/AHA class I recommendation in patients with STEMI presenting with cardiogenic shock, not responding to medical therapy. IABP in this setting is a stabilization measure for prompt revascularization.
 - Bridge to tertiary center -Thrombolytic therapy alone is less successful than early revascularization. However, addition of IABP to thrombolysis can improve outcomes in patients with cardiogenic shock.
- 2. Acute MI without cardiogenic shock- benefits are uncertain
- 3. High-risk percutaneous revascularization includes left main (LM) disease, Ejection fraction (EF) <40% and heart failure. IABP placement in these setting is associated with less cardiac events.
- 4. Mechanical complications of MI such as mitral regurgitation and ventricular septal defect
- 5. Refractory unstable angina IABP placement is a class I indication in STEMI patients and class IIa indication in unstable angina/NSTEMI patients with continued or recurrent symptoms, not responding to medical therapy
- 6. Weaning from CPB-for patients with severe LV dysfunction difficult to wean from bypass after open-heart surgery, IABP support improves hemodynamics and facilitates weaning from CPB.
- 7. Refractory ventricular arrhythmia post MI class IIa indication
- 8. Decompensated aortic stenosis can be managed with temporary IABP support to improve stroke volume and reduce transvalvular gradient before AVR.
- Support during non-cardiac surgery Patients with severe coronary disease, recent MI, and severe LV dysfunction are at high risk for cardiac complications when they undergo non-cardiac surgery. IABP support is used to stabilize these patients and avoid complications.

Contraindications to IABP placement

- 1. Aortic dissection may worsen aortic dissection
- 2. Abdominal or thoracic aneurysm-can precipitate aortic dissection, dislodgement of atheroemboli, or aortic rupture
- 3. Severe peripheral vascular disease limb ischemia and threatened limb viability
- 4. Coagulopathy or contraindications to heparin
- 5. Moderate to severe aortic insufficiency by inflating during diastole, IABP may worsen aortic insufficiency.

Shuttle gas used for IABP - either of the following is used

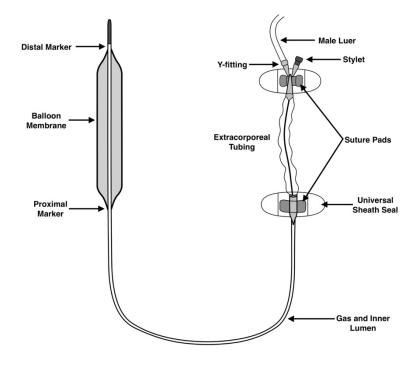
- 1. Helium-preferred as it has low viscosity, enabling rapid inflation and deflation.
- 2. CO2-it is more viscous but has better safety margin.

Hemodynamics of balloon pump

Placement of IABP results in decrease in afterload and augmented coronary perfusion.

During systole, the balloon deflates rapidly. This creates a negative pressure in THE aorta, which improves forward flow from LV and increase in cardiac output of approximately 20%.

During diastole, the balloon inflates. It displaces blood to proximal aorta and increases coronary perfusion.



Components of intra-aortic balloon

Insertion of IABP

Size of balloon is based on patient's height.

Balloon size	Patients height
50	6'
40	5'4"-6'
34	5-5'4"
25	<5

The leg with strongest pulses and best ankle brachial pressure index should be chosen for access. The two commonly available balloon catheter sizes are 8 F and 9.5 F. The balloon is advanced until proximal tip is positioned 1 cm below left subclavian artery and 2 cm above carina. Insertion is done preferably under fluoroscopic guidance. If fluoroscopy is not available, the distance from angle of Louis to the umbilicus and then to common femoral artery insertion site is measured to determine approximate distance.

Occasionally, balloon pump can be inserted surgically by directly exposing the common femoral artery or by suturing a 6-12 mm prosthetic graft end to side to femoral artery to provide a conduit for catheter. With this method, distal limb ischemia is reduced. However, the graft must be removed surgically and femoral artery should be repaired during removal of IABP.

IABP should always be put below inguinal ligament, as manual hemostasis is extremely difficult above the ligament.

Other sites of insertion of IABP are

- 1. Aortic pursestring
- 2. Axillary -to make patient ambulatory (as a bridge to transplant)
- 3. Iliac
- 4. Abdominal aorta
- 5. Pulmonary artery (for pulmonary artery counterpulsation)

Monitoring of IABP

- 1. CXR is immediately obtained after IABP placement to verify position.
- 2. *IV heparin* to maintain aPTT of 50-70 seconds.
- 3. Daily Hb and platelet counts to monitor Hemolysis and thrombocytopenia

Care of the patient with IABP

- 1. The patient should be kept supine in bed
- 2. Patient should be evaluated for possible limb ischemia
- 3. Prophylactic antibiotics are not indicated
- 4. Blood samples should not be obtained from central lumen of IABP.

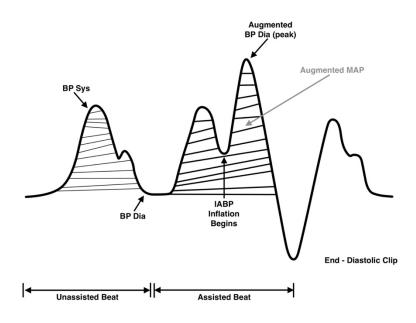
Balloon pump triggering and timing

Balloon pump inflation can be triggered by

1. Surface ECG - triggered after the R wave. If the IABP fails to trigger properly

from surface ECG, check surface electrode placement or increase the ORS gain on the console monitor.

- 2. Arterial pressure waveform triggered when the aortic valve closes (dicrotic notch)
- 3. A paced rhythm in patients who are 100%paced.
- 4. Internal asynchronous mode in arrested patient



IABP waveform

Ideal balloon pump timing occurs when the balloon inflates on the downslope of systolic pressure. Waveform before the dicrotic notch and deflates before the onset of next systolic waveform.

Early inflation	Inflation of IABP before aortic valve closure
Late inflation	Inflation of IABP well after aortic valve closure
Early deflation	Deflation of IABP before isovolumetric LV contraction
Late deflation	Deflation of IABP after the onset of systole

In tachyarrhythmia, when the heart rate approaches 150 BPM, augmentation become difficult, adjusting balloon inflation to 1: 2 can sometimes improve augmentation with tachyarrhythmia.

Troubleshooting – check for

- 1. Loose connections in the gas line or arterial pressure tubing
- 2. Blood in the tubing
- 3. Poor augmentation
 - Adjust timing
 - Change trigger mechanism

• Evaluate catheter position to check balloon kinking

Complications of IABP

- 1. Limb ischemia If ischemia develops in the accessed leg, balloon catheter and sheath should be removed and hemostasis obtained at the access site. Surgical intervention in the form of thrombectomy, surgical bypass grafting or rarely amputation may be required.
- 2. Bleeding controlled with prolonged manual pressure at the access site. Sometimes, direct arterial pressure may be required.
- 3. Infection
- 4. Balloon rupture should be considered if blood is detected in the gas driveline lumen or if balloon augmentation ceases.
- 5. Balloon entrapment occurs when balloon rupture causes a clot to form within the balloon, preventing deflation during removal. When resistance is encountered during balloon catheter removal, balloon entrapment should be considered. Forceful removal of partially deflated catheter may cause vascular injury. Management involves surgical extraction.
- 6. Other complications
 - Acute renal failure
 - Mesenteric ischemia
 - Paraplegia
 - Aortic dissection and aortic perforation
 - Hemolysis and thrombocytopenia

Removing of IABP catheter

- 1. Weaning
 - The usual practice is to change IABP inflation to 1: 2 for a few hours and then to 1: 3 with close hemodynamic monitoring.
 - Inotropic drugs are used to maintain adequate cardiac output
 - If weaning is tolerated hemodynamically, balloon can be removed
- 2. <u>Withdrawal of balloon catheter and sheath</u>
 - Discontinue heparin 4 hrs before removal of catheter
 - Percutaneously placed catheters can be removed manually, but surgically placed catheters should be removed by direct arterial repair.
 - Balloon is put on standby mode and gas drive line is disconnected
 - Balloon is deflated. Catheter is pulled back until resistance is met, indicating that catheter in the sheath. The sheath and catheter are then withdrawn together as a unit
- 3. <u>Hemostasis</u>
 - After balloon is removed, puncture site is allowed to backbleed for 1-2 seconds while pressure is held distal to puncture site to evacuate proximal thrombi.
 - Then manual pressure is applied proximal to puncture site and backbleeding is repeated to evacuate distal thrombi.
 - Manual pressure is then applied for 30-45 minutes over the puncture site until

adequate hemostasis is achieved.

• A compressive dressing is applied.

IABP in children

Use of IABP in children is limited because

- Lack of balloon catheters small enough for children <5 years of age
- The compliant aorta in young children may prohibit effective diastolic augmentation
- Synchronizing balloon action to higher heart rate is difficult
- Children have additional RV/ pulmonary artery disease

SECTION XVII: VENTRICULAR ASSIST DEVICES (VAD)

History

Dr. Michael DeBakey implanted the first mechanical assist device in 1963. Dr. Denton Cooley reported the first successful bridge to transplant using mechanical support in 1978.

Introduction

Despite advances in management of heart failure, few options exist in current era. Orthoptic heart transplantation is the gold standard of therapy for end stage heart failure. With a growing population of end stage heart failure and a static donor pool, additional treatment modality must be considered. To date, ventricular assist device is the most promising alternative treatment for end stage heart failure patients.

VAD can be used to provide temporary ventricular assistance after cardiac surgery. VAD can also be used for prolonged circulatory support with the intent of bridge to transplant, bridge to recovery and as a destination therapy.

Principle

Implantable VAD function by removing oxygenated blood from apex of left ventricle, passing it through a mechanical pump and returning pressurized blood to ascending aorta.

Indications

- 1. Cardiogenic shock after acute myocardial infarction
- 2. Post cardiotomy cardiogenic shock
- 3. Myocarditis
- 4. Refractory ventricular arrhythmias
- 5. Chronic heart failure as a bridge to transplant
- 6. Chronic heart failure destination therapy.

Classification of LVAD

Devices for short-term mechanical support (< 30 days)

- 1. Intra-aortic balloon pump (IABP)
- 2. Extraacorporeal membrane oxygenation (ECMO) Biomedicus biopump (Medtronic)
- 3. Levitronics centrimag
- 4. Abiomed BV 5000 and ABS 5000
- 5. Abiomed Impella
- 6. TandemHeart (CardiacAssist) percutaneous LVAD

Devices for long-term mechanical support

First generation-pulsatile pump

- 1. Thoratec Heartmate XVE
- 2. Thoratec paracorporeal
- 3. Thoratec intracorporeal

Salient features

- Axial blood pumps
- Produce cyclic, pulsatile flow
- It has inflow and outflow prosthetic valves
- Principle pulsatile chamber that fills passively and is compressed by pusher plate

Secondary generation - centrifugal pump

- 1. Thoratec Heartmate II
- 2. Jarvik 2000
- 3. Micromed DeBakey
- 4. Incor Berlin heart

Salient features

- Centrifugal blood pump
- Produce continuous, non pulsatile blood flow
- It has no valves
- Principle continuous flow driven by spinning rotor around central shaft

Advantages of second generation devices over first generation devices include

- 1. Less extensive surgical dissection for implantation
- 2. Absence of valves that are primary sites of wear
- 3. Higher efficiency with less energy requirement
- 4. Smaller percutaneous lead

Third generation – magnetic levitation technology

- 1. Ventracor Ventrassist
- 2. Terumo Duraheart
- 3. Worldheart LevaCor
- 4. Thoratec HeartMate III

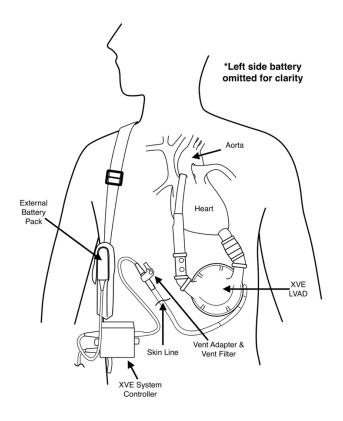
Salient features

- Centrifugal blood pump
- Produce continuous, non pulsatile blood flow
- It has no valves
- Principle continuous flow driven by electromagnetic suspended spinning rotor.

REMATCH trial – was performed in 2001, patients with irreversible heart failure who were not eligible for transplantation were randomized to either maximal medical therapy or HeartMate XVE implantation. LVAD implantation doubled the 1-year survival rate of these patients form 25% to 52%.

HeartMate XVE – currently, it is the only device approved for a destination therapy. It consists of a positive displacement pump made of titanium with a polyurethane diaphragm and a pusher plate accelerator that converts electrical energy to mechanical energy. Cannulation involves LV apical cannula and an outgrow graft to ascending aorta. The most unique feature of HeartMate XVE device is a blood-priming surface, which consists of titanium microspheres that promotes formation of pseudointima that is resistant to thrombogenesis. The device requires only aspirin for effective anticoagulation.

Unfortunately, long-term durability of the device is poor. The device malfunction occurs within 18-24 months due to bearing wear.



HeartMate XVE

LVAD implantation

Whenever possible, LVAD implantation should be performed under elective circumstances. Appropriate patient selection and proper timing of implantation are more important for optimal outcome.

- The key factors that should be assessed preoperatively include
- Right ventricular function
- Status of valves and presence of ASD or VSD-patients with severe tricuspid regurgitation, severe aortic regurgitation, atrial septal defect or ventricular septal defect should have their lesions corrected at the time of VAD implantation.
- Nutritional status
- Renal and hepatic function
- Gastrointestinal bleeding, pulmonary function, coagulation status
- Infection, neurologic function
- Psychosocial support to the patient

Implantation of long term LVAD should not be considered in patients with

- Irreversible major end organ dysfunction
- Sever hemodynamic instability
- Unknown neurologic status
- Irreversible respiratory failure requiring mechanical ventilation
- Sepsis
- Right heart failure

In patients who present with cardiogenic shock and end organ dysfunction, temporary mechanical support with *TandemHeart percutaneous VAD* may be beneficial. The device can be inserted under fluoroscopic guidance in cathlab. Patients who show improvement in end organ failure are then considered for subsequent implantable LVAD therapy.

The goal of LVAD is mechanical unloading of failing left ventricle and an increase in aortic perfusion.

Postoperative management

The LVAD placement requires post-operative blood pressure monitoring, anticoagulation and management of right ventricular function.

Mean arterial pressure is maintained at 70-80 mmHg, Patient is started on aspirin and warfarin post operatively to maintain INR of 1.5-2.5.

Right ventricular function is managed by

• Intraoperative myocardial protection

- Postoperative-milrinone, inhaled prostaglandin, nitric oxide
- Frequent post operative echocardiography adjusting pump speed to avoid deflection of interventricular septum either into or away from right ventricle
- Right ventricular support should be considered if cardiac index is < 2 L/min/m² and central venous pressure is >20 mm Hg.

Potential complications

- 1. Perioperative hemorrhage
- 2. Infection
- 3. Right sided heart failure
- 4. Acute renal failure
- 5. Gastrointestinal bleeding
- 6. Ventricular arrhythmias

A successful LVAD program is reliant on a team of cardiac surgeon, heart failure cardiologist, electrophysiology cardiologist, nurse, VAD coordinators, nutritionist, pharmacist, social worker, and transplant coordinator.

Pediatric VAD

To address the challenge of providing mechanical support in small patients, Berlin Heart EXCOR is developed to include multiple pump sizes. The pump contains polyurethane trileaflet inflow and outflow valves. It is available in sizes of 10 ml, 25 ml and 50 ml.

SECTION XVIII: EXTRACORPOREAL MEMBRANE OXYGENATION (ECMO)

ECMO remains the most commonly used form of mechanical circulatory support in children. It has a limited role in order patients.

Advantages of ECMO

- 1. It provides biventricular support.
- 2. ECMO provides respiratory support in conditions in which advanced pulmonary disease is also present.
- 3. Peripheral cannulation and quick priming makes ECMO a potential resuscitation tool for patients in cardiac arrest.
- 4. ECMO can provide a period of stabilization for patients in cardiogenic shock prior to implantation of more permanent circulatory assist devices or transplantation
- 5. It is less costly than other forms of mechanical support.

Indications of ECMO in pediatric patients

- 1. <u>Respiratory failure</u>
 - Congenital diaphragmatic hernia
 - Meconium aspiration
 - Sepsis
- 2. Cardiac indications
 - Acute myocarditis
 - Bridging to heart transplant, following transplant
 - Treatment of intractable ventricular arrhythmias
 - Cardiac resuscitation in the event of cardiac arrest rapid deployment ECMO
 - Post cardiotomy failure to wean from CPB
 - Complex congenital obstructed TAPVC, ALCAPA, TGA
- 3. Combined cardiopulmonary
 - pulmonary hypertension following cardiac surgery
 - infants with unrepaired cyanotic heart disease who acquire common viral respiratory illness

Contraindications to ECMO

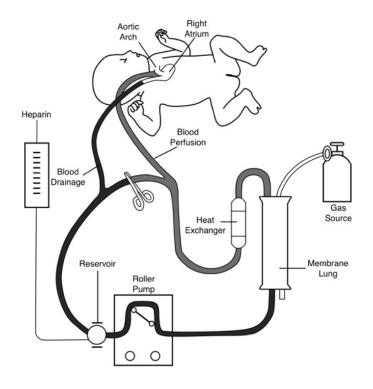
- 1. Incurable malignancy
- 2. Advanced multisystem organ failure
- 3. Extreme prematurity
- 4. Severe CNS damage

Types

ECMO is used to provide either combined cardiopulmonary or isolated pulmonary support.

Venoarterial ECMO provides total cardiopulmonary support by removing deoxygenated blood from venous circulation, exchanging oxygen and carbon dioxide and then pumping it back into patients arterial circulation. Studies show that, temporary total cardiopulmonary support provides an environment that promotes myocardial and lung recovery.

Venovenous ECMO provides pulmonary support by withdrawing deoxygenated blood from venous system, exchanging oxygen and carbon dioxide and then returning the blood to the venous system.

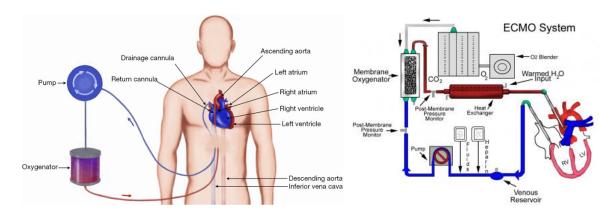


Components of basic ECMO circuit

Equipment

The typical ECMO circuit consists of three components

- 1. Pump roller/centrifugal
- 2. Oxygenator membrane/hollow fiber
- 3. Heat exchanger



Components of basic ECMO circuit

Additional components include tubing and monitoring devices

Monitoring devices

- 1. Bladder reservoir to provide access for administration of drugs, blood products and fluid and to remove air from circuit
- 2. Pressure transducer detect circuit thrombosis or kinking of tubing.
- 3. Bubble detector to decrease air embolism
- 4. Bridge a short segment of tubing that allow direct communication between arterial and venous limbs of circuit during temporary period of ECMO removal from patient, as in circuit thrombosis.

Cannulation

Transthoracic cannulation of right atrial appendage and ascending aorta

Cannulation of right internal jugular vein and common carotid artery is the preferred peripheral cannulation site in infants and young children

Groin cannulation of femoral vessels provide adequate venous drainage and perfusion for adults

Management of ECMO

ECMO flow rates are generally kept in the range of 80-150 cc/kg/min. Adequate flow rates can be judged from urine output, peripheral temperature, arterial lactate and mixed venous oxygen saturation.

Patients are routinely kept on low dose dopamine and vasodilators.

Patient is kept on moderate levels of ventilator support

ACT levels kept at 180-200 seconds with heparin infusion. Argatroban is beneficial in patients who have heparin sensitivity. Heparin coated circuits significantly decrease exogenous heparin requirements and activation of coagulation system. Use of aminocaproic acid significantly reduces bleeding.

Early institution of enteral nutrition maintains gut mucosal integrity and reduces sepsis

Weaning

As native cardiac and pulmonary functions improve, ECMO support is decreased. Weaning is performed under echo guidance to assess ventricular filling and function. Flow rates are gradually turned down for several hours. Concurrently, ventilator support and inotropic dosage are increased to appropriate levels. Decannulation is performed bedside.

Cardiac catheterization is considered when patient fails to wean from ECMO following surgery, especially if myocardial function appears to have recovered.

Complications related to ECMO

- 1. Bleeding
- 2. Oxygenator failure
- 3. Circuit thrombosis
- 4. Neurologic-cerebral thrombosis/ hemorrhage

Results of ECMO

A registry of patients that have received ECMO is maintained by the Extracorporeal life support organization (ELSO). The last publication of ELSO registry data reported outcomes on nearly 51,000 patients with 75% survival for neonatal respiratory failure, 56% survival for pediatric respiratory failure, and 55% survival for adult respiratory failure.

SECTION XIX: MINIMALLY INVASIVE CARDIAC SURGERY

Preoperative evaluation

Standard preoperative workup used for conventional surgery

Additionally, peripheral vascular Doppler and TEE should be performed to evaluate degree of atheromatous disease in aortic arch, thoracic aorta and peripheral vessels.

Percutaneous myocardial protection system (Heartport)

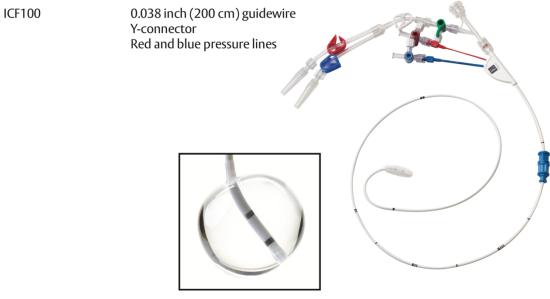
It is an endovascular bypass system consisting of

- 1. Y-shaped femoral artery cannula
- 2. Femoral venous cannula for drainage of right atrium
- 3. Endopulmonary vent catheter
- 4. Endoaortic occlusion device (Endoclamp)

The endoclamp is a 120 cm long, flexible balloon tip, triple lumen catheter.

- A. The first lumen is used for endoclamp balloon inflation
- B. The second lumen enables monitoring of aortic root pressure proximal to balloon
- C. Third lumen facilitates contrast lumen injection to secure position of catheter, cardioplegia delivery and ascending aorta venting.

39.3 inch (100 cm) overall length



Close up of tip

Endoclamp

Transesophageal echocardiography

Every case of minimally invasive surgery should undergo routine intraoperative TEE to evaluate

- 1. Intraluminal aortic atherosclerotic pathology
- 2. Coronary sinus cardioplegia catheter placement
- 3. Femoral venous cannula positioning
- 4. Endoaortic catheter positioning
- 5. Residual intra-cardiac air
- 6. The surgical result and post operative cardiac function

Cardiopulmonary bypass (CPB) considerations

- Reduction in surface area-priming volume
- Use of biocompatible surface coating
- Augmentation of venous drainage vacuum assisted or kinetic assisted

Arterial cannulation

The options for cannulation site include ascending aorta, femoral artery and axillary artery

Commonly used arterial cannulas are

- Port access femoral artery cannula (Edwards)
- Remote access perfusion arterial cannula (Estech)
- Biomedicus (Medtronic)

Venous cannulation

Right femoral vein is commonly cannulated and placement is guided via TEE. The femoral venous cannula can be advanced through the right atrium and into superior vena cava for bicaval single cannula drainage or can be placed into right atrium for bicaval dual cannula drainage.

Cardioplegia considerations

Endoclamp catheter	Facilitate endovascular aortic cross clamping, Antegrade cardioplegia delivery and aortic root pressure monitoring
Endoplege sinus catheter	Retrograde cardioplegia via coronary sinus
Endovent pulmonary artery catheter	Pulmonary artery venting

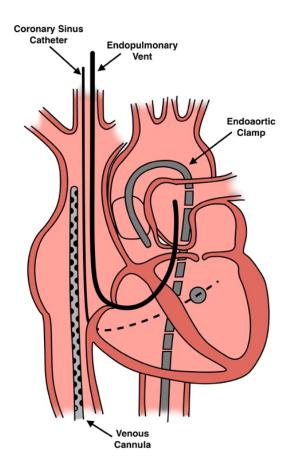
Procedure

Double lumen endotracheal tube is used to enhance visualization. Use of TEE probe is

essential.

Patient is positioned with right chest elevated to 30°. Groin dissection is performed to isolate femoral artery and vein. After full dose heparinization, cannulation is done with femoral arterial inflow cannula (19 or 21Fr *Biomedicus cannula, Medtronic*) and kinetic venous drainage through a femoral (22 or 23/25Fr) and right internal jugular vein (17 Fr).

Aortic occlusion is done by direct external clamping or endoaortic balloon occlusion (*Heartport*). Cardioplegia can be administered directly into ascending aorta or retrogradely via a catheter placed through right atrium into coronary sinus under TEE guidance.



CPB is initiated and patient is cooled to 30°C

The intracardiac procedure is conducted in a standard technique using specially designed, long shafted instruments. The procedure is performed by direct vision of operative field. Use of 30° High definition endoscope improves visual acuity. For aortic valve surgery, a small right anterior second or third intercostal incision is made to expose aortic root. For mitral valve surgery, incision is made in right inframammary groove, and fourth intercostal space is entered.

After completion of procedure, de-airing is performed under guidance of TEE. CO2 is flooded into thoracic cavity to eliminate danger of gas bubbles and then endoclamp is deflated. Rewarming is done and patient is weaned off bypass. Femoral cannulas are removed and femoral vessels are repaired. Thoracotomy incision is closed in layers.

Advantages over conventional open surgery

- 1. Less postoperative pain
- 2. Postoperative blood loss is significantly less
- 3. Earlier hospital discharge
- 4. Earlier extubation
- 5. Lower incidence of postoperative SVT
- 6. The advantage of avoiding sternotomy incision is specifically meaningful in geriatric patients, in terms of fewer pulmonary complications and infections

CPB CHECKLIST

- 1. Check all power cord, cables gas lines and temperature probe.
- 2. Connect heater cooler line
- 3. Note oxygenator, pump set and CP serial number
- 4. CO2 flush circuit
- 5. Check sucker occlusion
- 6. Check patients history
- 7. Prepare drugs, Signature_____
- 8. Prepare CP solution and check for occlusion
- 9. Check blood
- 10. Connect gas line to oxygenator
- 11. Prime and debubble arterial line, filter and circuit
- 12. Check and add drugs
- 13. Set various clamps
- 14. Activate bubble detector and air emboli detector
- 15. Set and enable arterial pump alarms
- 16. Set and enable cardioplegia pump alarms
- 17. Prime temperature <u>°C</u>
- 18. Set arterial pump occlusion
- 19. Attach venous saturation monitor
- 20. Take pre bypass gas sample

Signature_____

Date: / /