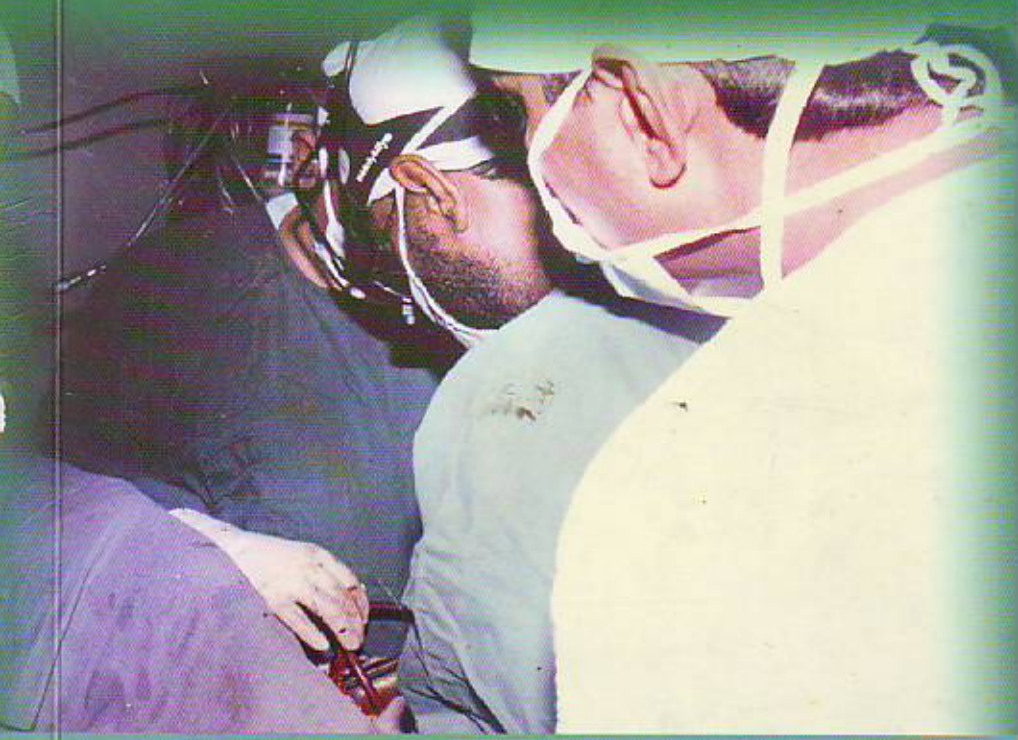
# METHODS & TECHNIQUES

## *IN CLINICAL*

# PERFUSION



*PROF.DR. N.A. KAMRUL AHSAN*

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# PERFUSION

(Hand book of methods & techniques in clinical Cardio-Pulmonary Bypass and tissue perfusion)

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# METHODS & TECHNIQUES IN CLINICAL PERFUSION

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# Preface

Open heart surgery along with cardiopulmonary bypass (CPB) has continued to develop in technique and equipments.The essence of bypass, perfusion however has extended in newer fields & techniques during the last era.

The education & training of perfusionist is evolving too.Now mostly perfusionist are trained in formal institutional programe in our country.Untill now there is lacking of organised educational programs with on-hand training.All these have brought about raising of the educational background of newly trained coming generation perfusionist. This is mandatory too for the role & acceptance of perfusionist in the surgical team.

Unfortunately the availability of texts for learners are not also in pace with the art of the subject.Students have to run to a variety of texts,hardly available here,to obtain necessary information in the specific field.

With this in mind we tried to accumulate most necessary practical informations in this short text with a view to help the learners and the perfusionist on pump.

We have avoided clinical, surgical,anatomical & normal physiological discussions intentionaly to keep the volume short.

I wish to thank our members of the Anaesthesiology & Surgical faculty of National Institute of Cardiovascular Disesases,Dhaka for whole hearted co-operation.

Particularly I thank Dr.Tajkera B and Dr. Bilkis B,Perfusionists of our team to take the trouble to go through the script & give necessary suggestions. I thank Dr. F.Maruf, Assistant Professor & Dr. Asif A, Cardiac Surgeon of my team for their warm support & help in writing and publishing this book .

10 October 2009
The Dhaka

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# 1

## Introduction

## METHODS & TECHNIQUES IN PERFUSION

Cardiopulmonary Bypass (CPB) for whole body perfusion is conceptually simple & relatively easy. Blood which normally returns to the right atrium is drained in device in which oxygen is supplied to this blood and carbon dioxide is removed. Then the newly oxygenated blood is pumped from the device into the aorta.

### Unnatural events in CPB:

The newly circulating blood

1. Does circulate through the non-endothelial lined channels.
2. Contains gaseous & particulate emboli.
3. Experiences non-physiologic shear stresses.

Besides during the procedure body experiences **an unaccustomed absence of any appreciable pulmonary blood flow and a minimum pulsatile** aortic pressure. These untoward circumstances are in addition to the *major surgical trauma.*

In spite of all these events the patient survives after operation & CPB.

### Cardiopulmonary Bypass

History of CPB is difficult and almost impossible to determine, who first gave the idea of artificial circulation?

At the last part of the 19th century Frey & Gruber worked

with oxygenator in 1885. After that a score of reports are available from different laboratories.

Revolution appeared with the development of heparin and blood biocompitable plastic material along with modern anesthetic and surgical technique. Undoubtedly at Massachusetts general hospital in late 1939. John Gibbon did a major contribution to the development of CPB to a successful one in clinical setup. During subsequent 20 years of his work took little note of medical world.

But Gibbon continued his work and in 1953 performed successful operation with CPB for Atrial septal defect in a young women.

Then unfortunately his subsequent 4 patients died & became discouraged.

Mean while few other began to work. Dennis & Richard at University of Minnesota attempted clinical application by CPB in 1951in a patient thought to have an ASD but the patient died and at autopsy found to be a case of AVSD (atrio-ventricular septal defect). In Stockholm Bjork & Senning also worked during late 1940s & early 1950s.

After Dennis's unsuccessful attempt Lellehi at Minnesota used controlled cases circulation in laboratory with living subject as oxygenator & in 1954 they used father/ mother as a oxygenator for a series of operation. But the technique was abandoned .

In early 1950s at Mayo Clinic John Kirklin started Laboratory work led to first successful repair of Ventricular Septal defect an March 22, 1955. & subsequently world first publication of a device of intracardiac operation appeared.Today the method is being used in every country of the world.

## Body Perfusion:

CPB is consequently simple and equipments are available. Most of the patient's blood (Systemic) that returns to right atrium is diverted to a device for exchange of $O_2$ and $CO_2$.

The newly oxygenated blood is pumped to the aorta.

The complications appear from:

(1) Non-endothelial surface of the circuit through which the blood circulates
(2) Continuous particulate/gaseous mater
(3) Stimulated physiologic stress response.

Remarkable fact is that patients survive and recover well. Prevalence of unfavouorable effects are not well defined.

When all systemic blood is returned to oxygenator is called total Cardiopulmonary bypass. On the other hand some goes to the lung and back to left atruim known as partial CPB.

# 2

# Coronary Circulation & Myocardial Preservation

## DEVELOPMENT

- The heart is developed in splanchnic mesoderm
- The primitive heart tube is separated from myoepicardial mentle by 'cardiac jelly'.
- Coronary arteries are developed from epicardial mentle

## FUNCTION OF THE HEART

- Transformation of metabolic energy($o_2$,substrate) to mechanical energy (Circulatory pressure,Flow)
- Chemo-Mechanical energy transducers are Sarcomares (Functional units)

## FLOW

- Normal coronary flow:0.7-0.9 ml/g/min
- O2 extraction 75% at rest,100% at stress
- With adequet perfusion pr. flow is auto- regulated by arteriolar resistance (influenced by metabolic demand)

## FOCUS OF ACTION

- Myosin cross-bridge

$Ca^{+}$>Myosin>Hydrolyse>ATP>ADP +Phosphate Splitting ATP release CHEMICAL ENERGY, brings >conformational changes in MYOSIN BRIDGE > Contraction of Myosin Mechanical Changes (Cross-Bridge dynamics)

- Dysfunction of Cross-bridge dynamics is similar( in characteristics -metabolic,energetic & functional) in all Cardiac Diseases (Valvular /Ischemic).

- Core dysfunction > SR
- With critical /significant(75%)stenosis coronary pressure distal to lession falls & blood flow shifted away from endocardium (due to high intramural pressure).

**CAPACITY OF CORONARY BED**

- Capacity defined as flow at which coronary pressure is equalled systemic pressure
- Disease free primary coronary trunk (LAD,Cx,RCA) capacity is 100 ml/min
- Flow depends on arterial pressure, cavity pressure.& transmural pressure.
- Ischemic necrosis begins in sub- endocardium
- This ischemic injury progresses exponentially with time
- Explains the importance of time in Cardiac Surgery

**[SCORING OF PERFUSION ]**

- Result of treatment remains imprecise unless quantification of obstruction,distribution & severity of perfusion is determined
- Simpler Scoring methods acts as accurate guide line to surgeons
- No substitute to see personally by surgeons decicing for /against operation

*Segmental Coronary Arteries( CAs):*

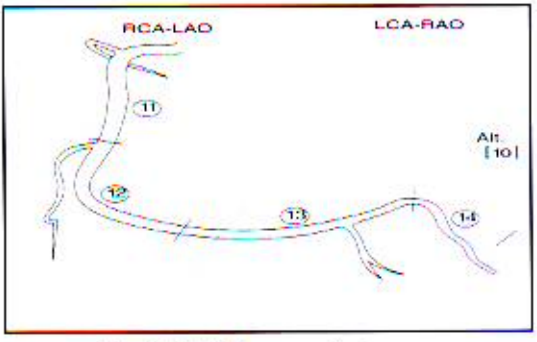



*Fig 1: Right Coronary Artery*

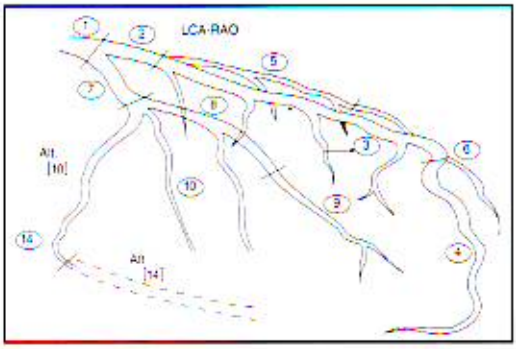



*Fig 2: Left Coronary Artery*

- 50% diameter (75% cross section) less-Important
- 67% diameter(90% cross sec.) less- severe

**Patient Specific Prediction & Comparison of outcome in CABG :**

- Indicaton of surgery based on time-related probability of good out-come after operation & Comparison of this outcome with other alternative treatment or no treatment
- Prediction is based on:

1) Time related probability of freedom unfavourable outcome(death)
2) Time related predicted comparative benifit of CABG Vs other options of treatments.

- In IHD this comparisom is complex for multivariable factors & treatment Options in IHD
- At unrealistic minimum number of factors to be considered :

1. Number of systems with important stenosis
2. Left ventricular function
3. Severity of reversible ischemia
4. Presence of acute Infarction

[For a realistic prediction 'Soft ware' is necessary]

**MORPHOLOGICALSCORING**
(Extent & Severity)

Grade 1 - <50%
2 - 50-69%
3 - 70-95%
4 - 95-99%
5 - 100%

**Diffuse lession** - If >70% (i.e. 3-5 Grs) involving:

3 of 5 segments of LAD
3 of 5 „ Cx
2 of 4 „ Cx (non dominant)
2 of 4 „ RCA

INDICATION OF REVASCULARISATION DEPENDS ON ADEQUATE DISTAL VESSELS (size & run-off).

**MYOCARDIAL PRESERVATION:**

- MYOCARDIAL STUNNING: is Perfusion-contratility mismatch (normal flow but contraction low or diminished )
- Damage from a period of ischemia resulting both systolic & diastolic dysfunction of variable period without necrosis (hours to days)
- Some investigastors found 6 hrs normothermic ischemia compatible to myocardial cell Survival
- Ischemia leads to necrosis (MI)
- Necrosis takes 20 mins
- STUNNING OCCURS WHEN AFTER AN ACUTELY DIMINISHED FLOW, REPERFUSION STARTS.
- Inspite of normal flow >diminished contraction (Perfusion-contratility mismatch)

Causes :Hypothesis

- Diminished consumption (to protect necrosis)

**(some denied this hypothesis as stunned cell shows high consumption)**

- May be abnormal energy utilization (other than high energy $Po_4$) – Unlikely
- Current hypothesis -

Free radicle (neutrophil etc) - Experimentaly Super oxide dismutase introduced before ischemia can prevent stunning

- $Ca^{++}$ influx during reperfusion also responsible -"stone heart"
- HYBERNATION : IS PERFUSION – CONTRACTION MATCH ( both flow & contraction are low )

**It is Chronic potentially reversible state of dysfunction**

# 3

## Myocardial Management

**Objectives:** To limit ischemic injury by some combination of:

-Hypothermia
-Electro-mechanical arrest
-Wash out
-O2 / substrate enhancement
-Oncotic / Buffer manipulation

**No single method unequivocally best.**

**FACTORS TO CHOICE:**

- Technique that influences duration of Cross clamp.
- Surgeons conviction that injury prevention possible despite complexity of procedure
- Institutional enviornment
- Costs

**METHODS:**

1) Continous Normokalemic perfusion

**Normothermic - Not ideal**

- Flow distribution is abnormal (small heart)
- Collaterals are impeded
- Transmural infarction occures

**Mild to Moderate Hypothermia (25-30°C ) - Good result**

- 2) Fibrillating Heart perfusion:
  - Normothermia(37°c) - Fibrillation by current
  - Hypothermia(Moderate) - Fibrillation

sponteneous / current

[sub endocardium injury in hypertrophy]

- 3) Moderate Hypothermia 25-30°c ( Intermittant ) Surgeon works on clamp for 15 mins than release - Does not provide adequet exposure
- 4) Profound Hypothermia -22°c for 45- 60 min cross clamp- May be used in infant surgery
- 5) Drug mediated protection – blockers, $Ca^+$ channal blockers,hypothermia & Intermitant ischemia
- 6) Cold cardioplegia (multidose)-

**Asanguinous** - low $K^+$ for maintainance & substrate added (commercially available)

**Sanguinous** - Hyperkalemic cold - works well

**Bucukberg formulation** – (Blood- crystalloid mix + free $Ca^+$ & Glucose + Buffer) – as good as blood cardioplegia but the latter is more simple.

- 7) Blood Cardioplegia (Cold) - less costly, only Blood & $K^+$(22 mmol/L)

**MODE:**

ANTEGRADE:

- 150 ml//min/$m^2$ - for 3 min ( average adult 750 ml)

  [If root pr. < 30 mmHg Rate to increase- Not total dose]
- Re Infusion –after 25 min for 1min( K+ reduced to 10 mmol/L)

[If serum K+ 7-8 mEq /L > Bolus of 400mg/Kg Glucose(50%)+ 0.2U/Kg of Sol.Insulin .]

**RETROGRADE :**

Through Coronary Sinus at Pressure < 50mmHg

***RESULT:***

With cold cardioplegia 'safe' duration is not unlimited, Probably 100 min is safe

- Continuous (cold) perfusion (Ante / Retro) - Alternative to single dose / Multidose intermitant

❖ Continous( Warm) perfusion (Ante / Retro) -

Provides good protection. But some time surgically inconvenient

- 8) Cold cardioplegia with controlled Aortic root perfusion & warm cardioplegia induction:

(Minimize reperfusion injury & stunning with better performance. Not widely accepted )

Circuit: 1. Mini Heat Exchanger
2. Two pumps

**Technique:**

Warm,hyperkalemic modified blood infusion upto 70 mmHg >Total dose 500ml then > normothermic, normokalemic unmodified blood > continued till sinus rythm returns(usually 20min) > cross clamp released after deareation

**ANCILLARY PROTECTION**

- o ANAESTHESIA to CPB start - Patient is at high risk of damage & should pay attention
- Control of Hypertention
- Care of Oxygen demand
- Manipulation of catecholamines
- Anxiety

# 4

## Variables to be Controlled

Out put from oxygenator to patient is achieved by generation of a large pressure gradient by the pump & the roller pump generates non-pulsatile flow simple and reliable.when they are occlusive( in practice they are set to near occlusive). when they are occlusive truma to the formed elements of blood is increased. When too non-occlusive they are unable to maintain same rate of flow against wide range of resistance offered by the vascular system & arterial cannula. Silicon rubber tubing head is better than Tycon( polyvinyl) in respect of elasticity that decreases in hypothermia.

**Rollar tubing are kept occlusive** *(Defined by a fall of inch /minute of column of water kept vertically in the tube)*

When fully primed the pressure in the system is 300 mm Hg.

***Volume output of pump is more certain when output resistance is high than negative pressure generated in input side(>200mmHg)***

### Centrifugal pump:

Flow generated by the controlled Vortex pump varies with the changes with the resistance to flow into and out of the pump. *When pressure in the output line is more than 500 mm Hg.both out/inflow becomes zero.* Again when in flow pressure in the inflow line is <-500 mm Hg. again out/inflow becomes zero. **So rotation per minute in case of roller pump can not estimate flow in centrifugal pump.** A flow meter must be placed on arterial & venous line.

If arterial line is completely occluded flow ceases but pressure in arterial line will not exceed >500 mm Hg.. So not ruptured. Blood trauma and air transfer is similar with pump system.

## Venous input:

Venous inflow is due to pressure gradient from patients to machine. Pressure needed to drain into machine is less than to push from the machine to patient.(due to both to patient's venous & arterial system & for canulae).

**Sufficient negative pressure for venous input can be generated :**

- Maintaining controlled vacuum in reservoir
- Using Siphon system with gravity
- Using pump to create negative pressure within venous line

## Factors:

(Venous drainage)

**a) Vacuum assisted:**

- Creating controlled vacuum in this reservoir(By patient & machine level)
- Hard shell reservoir attached to wall suction (-40 to -60 cm $H_2O$) can develop vacuum assisted negative pressure.These are available.
- Allowes short tubing, smaller reservoir, low priming volume
- Valuable for infants; & children.
- Help increasing haematocrit & to reduce use of blood & blood products intraoperatively.

**b) Siphon Drainage (Gravities)**

- Common method of drainage .
- Appropriate level of table is a problem.
- A narrow range of alteration is available
- Easily interrupted by large boluses of air in venous line
- Large pump volume is needed.
- ***Simple reliable, effective method***

**c) Pumps:**

- Controlled vortex pump are used rather than roller pump
- Large pressure gradient can be developed (that can obstruct canulae tips) needed to be controlled by use of small cannulae
- Suitable for percutaneous cannulation (Long 18-20 F)in adult
- Facililitates MICS (Mininally Invasive Cardiac Surgery)

## Exchange of Gas :

- Oxygenator is important componant of the system
- Also an important surface for blood damage (the contrast, only a small proportion of blood elements,formed/unformed come in contact with tubing)
- Gas exchange occurs at the interface of the system (vertical screens//cylinders) in bubble oxygenators.
- Accross tiny pores in membrane oxygenator of hollow fibres micropores polypropylene
- Damage to blood elements are less in membrane systems
- Silicon rubber membrane provide no blood damage as there is interface & can allow CPB for more than 24 hours with reasonable safety
- The devices (hollow fibres/membrne) do not depend on gas flow for $CO_2$ regulation. Gas flow allows allows precise regulation of $PO_2$ & $PCO_2$

## Arterial $O_2$:

- $PaO_2$ at 250 mm Hg. is easily accomplisbed
- Higher $PaO_2$ is toxic to patient & forms bubble.
- $PaO_2$ has less than 85 mm Hg. is not acceptable may result cell damage if <65 mm Hg
- $PaO_2$ is related to temperature depended to oxygen consumption ($VO_2$) blood flow rate (Q), Performance of Oxygenator, flow rate and composition of gas.
- ***Reducing body temperature reduces $VO_2$ increases $PVO_2$ resulting increased $PaO_2$***
- During rewarming increasing $VO_2$ and metabolic debt accumulated gives low mixed venous $O_2$ level demand maximal $O_2$ transfer capacity of the Oxygenator

## Arterial $CO_2$ Pr:

- PaCO2 is controlled by varying the ratio between gas flow rate in Oxygenator (VO2 ventilator /m) & blood flow rate through Oxygenator (Q)
- Inline PCo2 & PH meters, fascilate control of PaCO2 & PH

Clinical perfusions either are performed **at normothermia(37°C)** or at hypothermia of various level: **mild(36°-32°C), moderate(25°-30°C) or deep(<25°C).**

**it is necessary to control line $PaCO_2$ & indirectly $P^H$**

**The "alpha- stat"** strategy is based on -

- **Using the $P^H$ measured at 37°C & uncorrected for temperature of patient blood**

- **Maintaining this level at 7.4** (i.e ventilation of the oxygenetor s maintened at level of body temperature of 37oC irrespective of level of hypothermia)

***This strategy results in optimum function of number of important enzyme systems***

## Perfusate:

A balanced electrolyte solution with a near-normal $P^H$ with ion containg that of plasma are ued.

Some centers use glucose & lactate.

Colloidal solutions like Dextran40,Dextran70 or Hydroxyethyl starch can be used to priming volume to attenuate fluid loss from intravascular space

## Hb-Concentration :

Some centers do not control haematocrit in CPB both in adults & youngs.

In human at 37°C normal haematocrit of 40%-50% is optimal for oxygen transport. Provides sufficient $O_2$ to maintain mitochondrial $PO_2$ levels of 0.05-1.0 mmHg & average intracllular $PO_2$ of 5 mmHg.This results in $PvO_2$ 0f 40 in mixed venous blood ($SvO_2$ of around 75%).

Higher $O_2$ conentration increases blood viscosity is increased & thereby decreases blood flow [$O_2$ transport varies directly with Hct & inversly with viscosity of blood which is again is determined by Hct]

Hypothermia increases viscosity & at low temperature lower Hct is needed than at normal

- A lower than normal Hct is desirable during hypothermia as the perfusate has lower viscosity with low shear stress provides better perfusion (Hct 20% is optimal in moderate & deep hypothermia)
- During rewarming higher Hct is desirable for increased $O_2$ demand. May be done by **Ultrafiltration or adding packed cells**

- Ammount of Blood/PCV needed to achieve desired Hb level can be calculated before CPB is started
- Banked blood stored for <24H is used
- More older is acceptable in adults only (Acidosis can be neurtalised heparin ,calcium & buffer)
- Usually 3ml $Cacl_2$(10%) is added for each unit of banked blood.Add no more until ionized calcium is measured **(Normal- 1.2 mmol/L, Total Calcium - 2.5mmol/L)**
- High level of ionized calcium may be deleterious to the patients

## Hypothermia & Perfusion

It is necessary to consider strategy for control of line $PaCo_2$ and indirectly pH..

The "alpha-stat" is based on (1) Using the pH measured at 37°C and uncorrected for temperature of the patients blood (2) maintained level at 7.4. Ventilation of Oxygenator is maintained at the level appropriate for temperature of 37°C irrespective of hypothermia

Hyperventilation during hypothermia decreases $PaCo_2$ and raises pH when the values for these are corrected the alpha stat strategy results in optimal function of the enzyme systems of body. $P^H$ stat strategy demands for same Value of pH & $PaCo_2$ corrected to the temperature of blood.

State of respiratory acidosis & hypercarbia exists. Cerebral flow increases. But may expose potential cerebral emboli.

At cellular enzyme level alpha stat strategy is good. But is still under investigation.

### Heparin in CPB

- A heterogeneous group of biological products called glycosaminoglycan
- A purified form derives from porcine intestinal mucosa but form derived from bovine lung is used.

- Studies suggest that lung heparin is preferable because of its reliable protein neutralization response.
- Heparin binds to and activates anticoagulant III and presents anticoagulant activity.
- Heparin is added IV/IM to the patient before initiation of CPB(300-400U/Kg)

Heparin concentration during CPB 3·5-4·0 U/ml .This measures corelates with activated clotting time (ACT)

In heparin resistance 2-3 times usual dose of heparin can produce satisfactory anticoagulation activity (ACT of 480s)

- Activation of clotting mechanism in CPB is not completely neutralized inspite of satisfactory ACT
- Increasing dose of heparin does not prevent subclinical coagulation during CPB
- Maintaining ACT at 300-350s rather 450s results no more subclinical coagulation than does the traditional method using less heparin and associated with less bleeding after operation.
- **Aprotinin prolongs both clotting time and ACT**. Its use has changed the concept the use of heparin.Recommandation is to use initial usual dose of heparin & than additional dose to maintain ACT>700s,when aprotinin is used along activating agent other than Kaolin.
- **If Kaolin is used with aprotinin as a activating agent maintaining usual** ACT at 480s is recommanded.

## Diluent:

A balanced electrolyte solution used to prime the pump oxygenator system wholly or in part for erythrocyte free addition during CPB. In some centres glucose and lactate containing solutions are used.

## Haemoglobin:

In some centres no effort is made to control hemoglobin concentration and pump routinely filled with salt solution.

In human at 37°C the hematocrit of 40% – 50% is optimal for oxygen transport assuring normal red blood cell hemoglobin. This provides sufficient $O_2$ delivery to normal mitochondrial $PO_2$ of 0.50 to 1.0 mmHg and average intra cellular $PO_2$ of 7 mmHg (reflected normal oxygen level of P $O_2$ 40 in mixed venous blood.

- When hematocrit is high the $O_2$ content is also high and the increased viscosity decreases the flow.
- The rate of $O_2$ transport varies directly with haematocrit and inversely with viscosity.
- Hypothermia increases the viscosity of blood, therefore at a lower hematocrit is more effective than at 37°C.
- Lower hematocrit provides better perfusion of microcirculation.
- **20% hematrocrit (Hct) is optimum for moderate and deep hypothermia.**
- Higher Hct (Blood cell addition) is necessary at normothermia.
- Desired Hct concentration can be determined before start of CPB and thus blood priming or adding blood to priming volume can be used.
- Banked blood less than 48 hours old in used. Older blood are accepted for adults.
- Banked blood is rendered calcium free by anticoagulant solution (CPD) which is acidotic & may need addition of heparin,calcium & buffer before CPB.Some do not use calcium until rewarming to 28°C.Then the ionized calcium level is determined & calcium added accordingly **(Normal - 1.2mmol/L.Total calcium 2.5mmol/L).**
- Reasonable practice would be to add initially 3ml of calcium chiloride (10%) rather that 5 ml for each blanked blood.

**Albumin Concentration:**

- Concentration is affected by haemodilution.

- With low albumin in hemodilution extracellular volume increases rapidly.
- There is difference of opinion to add albumin to the prime in adult.
- Colloids (Dextran 40, 70) can also be added to the priming volume to attenuate the loss of fluid from intravascular space.
- None have proved of definite benefit.

**Other additions:**

- Osmotic diuretic may be advisable ((Manitol 0.5g/Kg.)
- Diuretic during CPB is generally useful (1-2 mg/kg bolus) at the start of rewarming.
- Short acting adrenergic -receptor blocking agent (Phentolamine) is capable to antagonize vasoconstriction by catecholamine. Beneficient from even cooling/rewarming (0.2mg /kg bolus) at the start of cooling & at rewarming after circulatory arrest.
- Long acting -blocker (phenoxybenzamin) can be used in infants/children at (1 mg/kg) for 15 minutes before start of CPB and at rewarming after circulatory arrest.
- Continuous infusion of nitroprusside during cooling and again during rewarming is preferred. It reduces arterial blood pressure maintaining cerebral flow during moderately hypothermic CPB.
- Corticosteroids improves tissue perfusion and lessen the extra-cellular water that accompanies CPB. But routine use is arguable Methylpredrisolone single dose (30mg/kg) or dexamethasone (1mg/kg) at the onset of CPB, not repeated may be advantageous (attenuate complement activation).
- Epsilon – Aminocaproric acid and Tranexamic acid are two anti-fibrinolytic agent that can be administered before, during and after CPB to reduce bleeding.

***(Epsilon: 150mg/kg at incision and then 30mg/kg for 4 hours upon initiation of CPB.***

***Imperically - 10g before skin incission, 10g during operation & 19g during early post operative period***

***Tranexemia: 1g at incision, 500mg at pump priming and 400 mg/H during procedure).***

**Changes during CPB:**

- During CPB there is steady depletion of patient machine blood volume.
- This is due to blood loss in operative field,increase in interstitial volume and urine out put
- Usual practice of perfusionist to add balanced electrolyte solution to maintain volume at safe level (in adult upto 2000 ml). As a result severe hemodilution occurs and persists in post bypass period unless attention is paid (avoiding more addition,Ultrafiltration at the end).
- In Neonate and infant ultra filtration at the end of CPB is recommended (before removal of cannulae).
- In children and adult ultra filtration may be performed during later part of CPB if Hct is below 0.25.
- Alternatively ultra filtration of the volume remains in the pump, after CPB discontinued and then concentrated blood is infused through arterial cannula.

**Total Systemic flow:**

During total CPB systemic flow is controlled by perfusionist.

- In practice for infant and children ($\leq$ 4 years),at body temperature of $\geq 28^0$C - Flow of 2.5L/ $m^2$/min .
- For older patients(>4years) - 2.2 L/$m^2$/min.
- For adults a flow of - 1.8 to 2.0 L/$m^2$/min may be chosen.
- In moderate hypothermia CPB flow is reduced to 1.7 L/$m^2$/min for prolonged period.
- At 18~ 20°C in infants/neonates/adults flow of 1 L/$m^2$/min for prolonged period is adequate. (Flow of 0.5 L/$m^2$/min is adequate to protect brain at this temperature).

- Less than optimal flow rate, results less perfusion of total capillary bed resulting lactic academia and metabolic acidosis.
- Mixed venous $O_2$ saturation ($S\tilde{v}O_2$) and mixed venous O2 pressure ($P\tilde{v}O_2$) are used widely as indices of adequate perfusion flow rate. [$S\tilde{v}O_2$) inversely related to oxygen consumption
  ( $vO_2$) ].

**Arterial Pressure Waveform :**

*During CPB pulse pressure is narrow and nonpulsatile. Pulsatile flow can be achieved by:*

- Partial CPB
- Using IABP during bypass.
- Pulsatile arterial pumps.

**Disadvantages by nonpulsatile flow:**

- Vascular resistance increases.
- RBCs aggregate
- Renal function is impaired with release of renin
- Cellular Hypoxia

**But Benefit of pulsatile flow is not well established.Evidences are insufficient to draw conclusion that pulsatile flow importantly reduces the effects of relatively short period of CPB in great majority of patients**

**Systemic Venous Pressure:**

- Cross sectional area and length of the venous cannulae as well as the size of venous tubing determines venous pressure during CPB.
- Largest cannulae are compatible commonly.
- Venous pressure should be kept to zero and positively not more than 10 mmHg to minimize extra cellular fluid.

**Pulmonary venous pressure:**

- Ideally should be at zero and not more than 10mmHg.
- Higher pressure causes pulmonary oedema
- Monitoring of LA pressure may be done.
- If pressure increases, suctioning pulmonary trunk reduces LA/LV pressure.

**Temperature:**

- Can be controlled by perfusionist with heat exchanger.
- Hypothermia adds flexibility of CPB when moderate hypothermia is added.
- This always lower pump flow rates & less trauma.
- Better myocardial or organ protection are also achieved than normothermic CPB.
- Provides a lengthy arrest time.
- **Mild hypothermia ($32^0$ - $36^0$ C) advisable in all cases.**
- **Moderate hypothermia ($28^0$-$31^0$C) is used in many patient.**
- **$14^0$- $20^0$C is chosen for circulatory arrest.**
- Temperature greater than $42^0$C damages blood.
- **Blood temperature should be kept below $39.5^0$C during rewarming and water temperature should not be more than $42^0$ C.**
- Heat exchanger should be at up stream (proximal) to the oxygenator to avoid gas formation during warming.
- Maintenance of temperature gradient of exchanger and blood should not be more than 10-$12^0$C to prevent bubble formation (*Solubility of gas in blood is decreased when warmed*).
- Some surgeons use $4^0$c perfusate temperature once CPB is initiated.

**Unfavourable Responses:**

Evident during the early days of open cardiac surgery. These were:-

(1) Diffuse bleeding (2) small patients became edematous during the procedure (3) occasionally severe hyperthermia (4) Pulmonary dysfunction (5) Dysfunction of the heart as anticipated. Yet many patients were free of these developments and most survived.

**Non specific Inflammatory Response to Pump-Oxygenator:**

Diversion of blood through non-endothelialized tubing's is recognized as 'non self'. Specific (immune) and non specific (inflammatory) responses are activated.

- Immune response is evident during first few days after CPB but not generally strong.
- Non specific Inflammatory response rapidly appears and called whole body inflammatory response or systemic inflammatory response syndrome (SIRS), that can be stimulated by other processes similar to CPB .

**Humoral :**

Initiated by the contact of plasma with tubing

- Starts with activation of plasma protein
- Inspite of heparinization coagulation cascade, complement, Kallikrine, fibrinolytic and other cascades respond immediately to foreign contact.
- Factor XII (Hageman factor) is initially activated.
- Platelets are independently activated at the same time.
- Found in the blood during and after bypass.
- Disappear probably to some-extent by metabolisms, taken up by specific cell receptors, dissipated into extra cellular fluid and excreted in urine.
- Products of activation have powerful physiological effect directly and by activation of others, complement cascade.

- Contact activation of factor XII also activates Kallikrein-bradykinin cascade with a feed-back loop to form plasmin – whose basic function is to digest fibrin, thrombin, and thus fibrinolytic cascade is activated.

**Cellular Response:**

Blood cell and endothelial cells participate in non-specific inflammatory response. Mast cells (Basophilic) participate well. Lymphocytes (B-cell, T-cells) participate little but NK cells are well participating like monocytes once activated.

**Leukocytes (neutrophilic)** : Play a major role in CPB. They change shape become adhesive & secretes cytotoxic substances including free radicals *(in tissue but in blood during CPB)*. Neutrophils may be activated by other mediators and agents (Kallikrein, Cytokines).

**Platelets :** Strongly affected. Activation occurs within one minute. Probably due to direct surface contact, shear stress and exposure to one agonists. Many surface receptors are exposed (Fibrinogen glycoprotein receptor, GPIIb-IIIa complex) and bounding of fibrinogen are in essence of adhesion.

**Endothelial Cells** : Have complex activities and affected when connected with CPB. Mechanism lies with abnormal pressure and shear stress, ischemia and chemicals. Endothelial cells and other cells express arachidonic acid which are important mediators of inflammation.

**Metabolic Response:**

*Elevated catecholamine levels in blood is a measure of severity of stress reaction during CPB.* The metabolic response to any stress is also necessary for recovery.

**Oxygen Consumption:**

Total Body $O_2$ Consumption **($vO_2$) :** During CPB at normothermia (***37 $^0C$***) should be that of intact human under anaesthesia $0_2$ consumption. Temperature of patient is also related to total body $O_2$ comsumption.

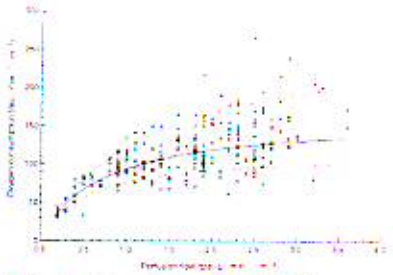


*Fig 3. Relationship of total body O2 consumption to perfusion flow rate at normotherrmia*

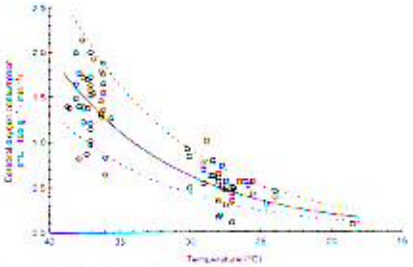


*Fig 4. Relationship of body temperature & cerebral O2consumption at full flow*

- Pulsatility of arterial input does not affect $vo_2$ (when flow>1.41/m/$m_2$ or with PH.
- Factors that affect $vo_2$ are pericapillary oedema & catecholamine release.

**Cerebral $O_2$ Consumption:** It is an important consideration during & particularly at hypothermic CPB at low flow. Because reduced $vo_2$ at a given temperature causes uneven cerebral perfusion.

- Total ($vo_2$) consumption changes with CPB due to variation in flow.
- Cerebral $O_2$ consumption does not appear to change with the variation of blood flow during CPB. (Fig. 4)

**Mixed venous Oxygen level:**

- Related with controlled variables of flow rate, haemoglobin concentration of perfusate & $PO_2$.

- When most of microcirculation is perfused venous $O_2$ reflect the main value for tissue oxygen level.
- **If mixed venous level in CPB is normal ($P\bar{v}O_2$ 30 to 40 mmHg; $SO_2$-60% to 70%) and $vo_2$ is normal than tissue $O_2$ levels are relatively normal.**
- The whole body perfusion is meeting the metabolic demand.

**Metabolic Acidosis:**

Usually tends to develop due to uneven distribution of flow or underperfusion with release of lactic acid and usually not severe enough.

**Hemolysis:**

Well recognised during CPB. Hemoglobin binds with heptoglobins or free. When heptoglobn binding sites are saturated are removed from circulation by reticulo endothelial system and kidney. ***When plasma free Hb-level exceeds 40mg/dl cast may be formed in renal tubles.*** Renal shut down is unlikely unless more than 100 mg/dl.

- RBC was often decrease during 1st hours of CPB. Complement activation may play role in red cell destruction.

**Systemic Vascular Resistance & Arterial Blood Pressure**

- Normothermic & moderate hypothermia decreases resistance at the onset abruptly.
- Gradually increases throughout CPB & may be higher than normal.
- There is considerable variation from patient to patient.
- *In patients with Ischemic heart disease higher resistance tends to develop.*
- Mechanism suggested WBC in Ischemic arrested heart when returns to machine and then to circulation develops vasodilatation for its substances.
- **In general it is unnecessary to manipulate SVR Pharmaceutically during CPB. However cerebral**

**blood flow is lower when arterial pressure is bellow 40 mmHg (in normo or moderate hypothermia).**

- **Rational pharmaceutical approach is then to maintain effective arterial pressure.**
- **When mean arterial pressure is too high (> 100 mmHg) it is important to make it less.**

**Cerebral Blood flow:**

- Flow proportionately similar in all age group with very little effect of age and CPB.
- In normothermia & moderate hypothermia flow is also not importantly altered with variation of BP.
- This is similar in awake patient under different BP (60-150mmHg).
- In deep hypothermia near BP should be above 25 mmHg.
- $PaCO_2$ increases (hypercrabia) increases flow and vice versa.
- Increase in cerebral flow under CPB causes 'Luxury perfusion' and micro-embolism.

**Venous tone:**

Increases in CPB for some hours afterwards may be due to catecholamine.

**Catecholamine Response:**

- Epinephrine → from adrenal medulla.
- Norepinephrine → sympathetic nervous system discharge. It is not clear that CPB response is more than normal ,in any form stress.
- With start of CPB epinephrine rises and decline after bypass. But persistent elevation one hour after occurs in patients with post operative hypertension.
- *Neonatal, infants and young children also shows marked increase concentration during CPB.*

The increase response, particularly of non-epinephrine is partly due to **during CPB blood does not pass through the lungs, where non-epinephrine is inactivated.**

**Adrenal Hormones & Pituitary Armurs:**

Markedly elevated (cortisol & vessopressin) for more than 24 hours with start of CPB. Deleterious or beneficial effects of these hormones are not demonstrated.

**Body Fluid:**

- After CPB body fluid increases at the interstitial compartment. Plasma volume tends to decrease. *The magnitude of increase is directly proportion to CPB duration and greater when there is haemodilution.*
- Exchangeable $Na^+$ increases after CPB and $K^+$decrease.
- Intracellular Potassium is also decreased.
- These changes are due to acute changes in membrane permeability.
- Other factors (hormonal) may also play role.

**Damaging Agents:**

- ***Foreign surfaces*** ® Pump oxygenerator filters, exchanger, bubbles system.
- ***Shear Stresses*** → Pumps, suction system, change in flow, cavitation around the cannula.

Both leukocytes, RBC are damaged.

**Safe Duration of CPB:**

- Partial CPB is better tolerated may be in days when a true membrane oxygenerator is used.
- Safe duration of total CPB is shorter in hours.
- **Duration is clearly a risk factor for morbidity & mortality after cardiac surgery and similar type of oxygenerator.**
- **Small and older patients are also risk factors for CPB & this is well documented.**
- **Relationship of safety & duration of CPB depends on number of factors.**
- ***Safe duration is surely measured in hours not minutes or days and is surely closer in general to 3 hours than 1 hour.***

**Clinical Use:**

CPB should be a flexible clinical tool due to its recognized risks limitation and damaging effects.

- Importance of hypothermia in CPB adds safe period longer with low perfusion flow(0.5L/min/$m^2$ ) or circulatory arrest.
- **Normothermia CPB provider higher cardiac output with low SVR at early post-operative period.**
- Size of arterial and venous cannulae is determined primarily by perfusion flow rate and type of venous return.
- Flow rate is not an absolute quantity but it includes a range of value.
- Two venous cannulae may be used as a routine in congenital heart disease.
- A single two-stage having additional roles may be used for coronary grafting operations for aortic valve mitral valve and ascending aorta and for combination of these procedures. Also for some congenital heart disease.
- Some times single cannulae are used without cross clamping for single operations like replacement of valve conduit.
- *Methods of cannulation, use of left atria, left ventricular vent and other aspect of methodology should be flexible within certain limit.*
- Combined experience of team should adopt surgical situation for patient safety.

**Heparinization / Protamin:**

- Dose is to be individualized for each patient.
- Base line ACT is obtained after sternotomy.
- Heparin (300 to 400 unit /Kg *or 3-4 mg/Kg*) is given and ACT is obtained after 3-5 minutes.
- Additional dose is given if ACT is less than 400 second to perform cannulation.

**Pump-Oxygenerator:**

- Heparin **(3cc/ml)** is added to the priming volume of the oxygenerator.
- After CPB started ACT is determined every 30 minutes and additional dose is added to maintain > 400 Sec.ACT during hypothermia (<$30^0$C) should be > 480s.

**Protamin:**

After removal of all cannulae protamin sulfate is given. The biological agent from sperm of fish, a low molecular weight protein forms heparin – protamine complex.

- This complex may be partially metabolized.
- May also react with fibrinolysin and thus freeing heparin and causes 'heparin rebound'.
- Widely used dose of protamine among many calculation, 1-1.5mg of protamine for each 100 units (each milligram) of heparin.
- **1 mg of protamine nutrilizes about 85 units of heparin. Extra protamine is given to prevent heparin rebound due to heparine released from tissue store or from heparin protamine -complex or to compensate the short half life**

# 5

## Commencing Bypass

On command of the Surgeon the perfusionist starts CPB and gradually increases flow to about 2.2 L/ $m^2$ /min and then cooling is started.

- In neonates and infants with deep hypothermia prime temperature to be maintained at $18^0$-$22^0$C.
- If cold prime volume is used heart slows down and functions impaired.
- Therefore pump must reach full flow to maintain systemic perfusion with a care not to distend the heart.
- If cardiac contraction is maintained at the start of CPB it is good to continue ventilation till full flow is achieved.
- For rewarming water temperature is raised at $42^0$C and arterial blood temperature should not exceed $39^0$C.
- It is advantageous to maintain nasopharyngeal temperature at $37^0$C for 10 minutes before CPB is discontinued.

**Pump - Oxygenator:**

Changing pump-oxygenator apparatus have some important general points.

***Componants are:***

**Venous Reservoir:**

- Used for gravity drainage. Hard-shell reservoir with vacuum assisted drainage is also used.
- This is incorporated in the housing of oxygenator.

**Open Circuit :** Traditionally used and venous drainage is free flow by gravity and is open to atmosphere.

**Closed Circuit** : Not in contact with atmosphere. Blood are collected in a collapsed bladder bag. Needs a separate open hard shell reservoir to handle excess volume than bladder. This system has advantage of reduced air blood interface and thus reduce the surface tension at the interface to minimize damage.

***In spite of all the open system is most favorable to use.***

**Oxygenator:**

Most important part. It is also the most damaging component of the CPB system.

- **Microporous & true membranous oxygenators** are better than bubble oxygenators.

**Heat Exchanger:**

- An effective exchanger is important.
- This may be free standing or integral part (less efficient).
- Integrated exchanger reduces priming volume.

**Arterial Pump:**

- **Usually roller pump.**
- To be adjusted to make slightly non occlusive and frequently calibrated.
- A centrifugal pump is also used.

**Pumps:**

**Roller Pump** – Developed by Debakey in 1934.These 'Milk' a constrained piece of tubing and capable to generate both a positive & negative pressure to pump blood through circuit.

**Hemolysis:**

In the early years hemolysis was a concern of roller pumps. **Now it is the air-blood interface in the suction at high velocity is considered cause.**

**Centrifugal Pumps:**

Centrifugal is an alternative for many years. **(But simplicity of roller pump is well accepted is most paediatric cases).**

High speed rotator compels blood at out let that depends on after load (resistance).

*High shear stress at the vortex may be a cause of hemolysis & satgnation in this system.* Limitation of operation is mainly with Cardiotomy suction or vent source of venous drainage. Disposable pump head is another limitation of cost.Centrifugal pumps works on the principle of high speed rotating radial wheel with vanes impelling blood to the out let of pump head. *The flow have disadvantage of dependance on after load resistance* (this is used as a safety feature of accidental line occluson).This afterload dependance causes a constant low flactuation.

- Also increases priming volume
- Newer generations have volume of 50 ml
- Since the head of these pumps are not occlusive /partially occlusive,if rotation per minute falls below a certain level retrograde flow from patient to reservoir is possible silently.
- Air embolism is also posible from these heads.

Additional problem lies with the electromagnetic flow meter used in these pumps that must be adjusted with every use & further the accuracy of flow probe may be interfared by electromagnetic interfarence.

**Filters:**

- The need is somewhat controversial.
- Currently low porosity filters are found beneficial.

**Arterial filter:**

**These are only in widespread use in paediatric cases.** Prime objection in use in children is the relative large volume needed to prime the filters.

**Designs:** Filters must remove air & particulate matter. Currently screen arterial line filters are of pore size 40 μn, *made of polyester woven into two dimensional screen.* They are

universally used. Pores are liable to block after use,In some filters hydrophobic membrane are incorporated at the top to expel air into atmosphere( *Pall Biomedical AV3SV , AV6SV* ).

They remove air by causing sudden decrease in flow velocity as the blood enters into the filters.This sudden decrease in velocity causes air bubble to rise to the top of the filter where they are purged from the system. Latest generation filters direct air tangentially to the top of filters.

Pre-Bypass filter:Circulation of clear priming solution through the circuit before blood is added ,is a good prctice Additonally a temporary filter(0.5-5µm)in the circuit can remove particulate matter created during manufacturing of ciruit.The filter is discarded before the blood is added.

**Crystalloid filters:** These are membrane (0.2µn size) filters & **bacteriostatic** *(Pall BioMedical CPSO2)*

**White Cell filters:**

These filters are used to make blood free of ***white cells (activated),*** both autogenous or transferred by adding a filter in the circuit. . In many centers Blood Bank service do this filtration before addition to bypass circuit. Autologous WBC as well with homologous are filtered continuously through out the bypass period

*Disadvantage this with the addition of priming volume.*

Release of sequestrated '***activated WBC***' (lung,sleen etc) after weaning has raised the doubt in its effectiveness.

**Microembolic:**

- More *documented in bubble oxygenator* during hypothermic cooling when the solubility of both $O_2$ & $CO_2$ are increased.
- Modern hollow fibre membrane oxygenator generates few emboli.
- *Filters are more useful in membrane oxygenator when vacuum assisted venous drainage is employed.*

**Paediatric, Filters: (Arterial)**

*Designed to keep priming low as well as high flow rate.*

- **Pall Biomedical**: Low prime volume (35ml) with maximum flow rate of 3 lit/minute. Adapted with 1/4 wide tubing.
- **Terumo Capiox**: Prime volume of 40ml & flow rate 2.5 lit/min. Accept 1/4 inch tubing.
- **Dideco 736**: Prime volume of 40 ml & flow rating of 2 lit/minute. It has got internal bypass feature.

**Ultrafiltration device:**

- Used in the **line between cardiotomy reservoir and arterial line**.
- During CPB if there is excess volume, the device is activated and serum water is removed.

**Haemo Concentration & Ultra-filtration:**

- Arterial line filters is to remove particulate matter greater than 40µm.
- **Ultrafiltration** is to make blood concentrated by removing water, dissolved ions and small molecules.
- **Bio-active smallest molecules other than albumen (65 kDa) like heparin, inflammatory mediators (IL-6, C3a, C5a), smaller than albumin can be removed are currently used**.

**Conventional Ultrafiltration:**

Ultrafiltration applied during CPB to achieve *hemoconcentration is called conventional*

Ultrafilter is placed with its inlet to the arterial line and out to venous reservoir.

- Transmembrane pressure difference can change filtration rate. Positive pressure on the arterial side can increase transmembrane pressure. Perfusionist can manipulate by applying negetive pressure on the effluent side.

- Blood flow rate- managed by Perfusionist.
- Membrane thickness, depth of pores, numbers of pores and size of pores determine rate of filtration.
- ***Low hematocrit increases the rate and thus hemoconcentration.***

**Modified filtration; Widely applied in paediatric cardiac surgery**

- Allows hemoconcentration of both circulating blood and perfusate in reservior after bypass.
- **Blood is drawn retrograde from arterial cannula, and reservoir to oxygenator and heat exchanger by a roller pump connected to the ultrafilter.**
- Negative pressure can be applied to the ultrafilter and increase the rate of flow.
- Filtered blood from the reservoir is returned to the patient through the venous line.
- Widely applied by many centers although there is debate in its use (dilution).
- ***If hematocrit is maintained more than 30% during bypass this ultrafiltration is less beneficial.***
- *Disadvantages lies with the complexity ,air trapping in the arterial line.*
- *Reduced prime volume with modern circuits with use of conventional ultrafiltration a hematocrit of 30% is well achieved before bypass is off.*

**Cardiotomy Suction:**

- Contain two suction ports.
- ***Low porosity filters*** are to be used to remove particulate matters.
- Reduction of length of cardiotomy suction is not too important but still reduction in size & length lessen the shear stress and floor air interface, that minimizes the damage to the blood.

**Arterial line pressure:**

- Must be continuously monitored.
- If *exceeds 250-300 mmHg* disruption of line and cavitation at the cannula site increases.

**Bypass Circuit In Neonats & Infants:**

Importantly the development in recent era is the circuit for neonates & infants in reducing priming volume (**as low 45-60 ml**) along with the calibre and ***length of the tubing***. Currently the **'centrifugal pump'** is placed closed to patients head and thus reducing the tubing length, although these are not in widespread use.

Table 1 : **Optimum Venous Cannula Size** (Fr)

| DIP Metal Tip Right Angle | | | RIM Venous | |
|---|---|---|---|---|
| Weight | SVC | IVC | VC | IVC |
| <3.5 | 12 | 12 | 16 | 16 |
| 3.5-6 | 12 | 14 | 16 | 18 |
| 6-8 | 12 | 16 | 18 | 18/20 |
| 8-12 | 14 | 16 | 18 | 20 |
| 12-16 | 14 | 18 | 20 | 20 |
| 16-22 | 16 | 18 | 20 | 22 |
| 22-28 | 16 | 20 | 22/24 | 22/24 |
| 28-32 | 18 | 20 | 24/26 | 24/26 |
| 32-40 | 18 | 20 | 26 | 26/28 |
| >40 | 20-22 | 22-28 | 26/28 | 26/28 |

SVC=Superior Vena Cava IVC = Inferior Vena Cava

**Heparin bonded Circuit :**

*All the tubing, shell & filters are heparin coated cannulas,.* Reduces the activation of complement and other markers. **Clinical efficiency is demonstrated in adult only.**

**Pulsatile / Non Pulsatile Perfusion:**

Advantage of Pulsatile Perfusion has been difficult to demonstrate. Roller head can be put to discontinuation fashion to generate pulse & easy to transmit through bubble

oxygenator. In membrane oxygenator and current tubings as well in paediatric circuit it is difficult to travel the the pulsatile flow through. *Alternatively balloon, diaphragm have been used like ventricular assist device but not in general use yet.*

**Hematocrit in CPB :**

- $0_2$ transport peak at hematocrit of 30%.
- Lower hematocrit is desired when there is greater degree of hypothermia.
- *In early phase of cooling the Brain is still warm and metabolically acute so with less Hct injury may occur.*
- ***General practice is higher hematocrit for higher temperature bypass.***
- A hematocrit level at low at 12% may be safe during CPB.
- There is important interaction of hematocrit, PH, temperature and flow rate.
- Low Hct with lower $0_2$ carrying capacity can be compensated for some extent by increased flow rate, reduced temperature or non acidotic $P^H$.
- ***But it is important to consider temperature maximum at a target hematocrit rather than minimum temperature.***
- In children 30-35% Hct should be the target for majority.
- **25% Hct should be the minimaly acceptable hematocrit for any CPB.**
- More studies are being conducted in this topic to find the extremes.

**Vasoactive agent addition**

**Vasodilators:**

Nitric Oxide doners (***Nitroglycerin, Nitroprusside*** ) are using as additive in CPB to improve the uniformity of both cooling and warming, particularly in deep hypothermia.

**Vaso constrictors :**

- These drugs are risky to use in pediatric patient.
- In adults with hemodilution and consequent hypotension may cause uneven distribution of flow.

- ***Phenylephrine*** is used in adults to maintain perfusion pressure and to counteract hypotension
- **In children without vascular disease, needs deep hypothermia or arrest. Vasoconstrictor may cause spasm and should be avoided in paediatrc patients and certainly not be added in pump prime.**

**$P^H$ & $CO_2$ in CPB :**

- **$CO_2$** is potent systemic vasodilator & conversly alkalosis & low $CO_2$ is vesoconstrictor . It is similar in cerebral circulation.
- On the other hand **Hypercarbia** resulting respiratory acidosis again causes vaso constriction.
- **$CO_2$** and **$P^H$** have opposite effects on ***systemic and pulmonary circulation***. Thus in case of AortoPulmonary collaterals and shunts (System/Pulmonary), any change in $CO_2/P^H$ can cause marked shift in distribution of flow between system and pulmonary circulation.

**Acid Base Balance :**

- Optimum Enzyme function depends on stable intracellular $P^H$ close to neutrality of water ($P^H$) when hydrogen and hydroxyl ions are equal.
- In human body this corresponds to **intracellular $P^H$ 7.1**.
- **To maintain 7.1 extra cellular $P^H$ should be between 7.36 & 7.44**
- *This is achieved by buffer systems.*

**Buffers**

- Hemoglobin, Proteins – are most important buffers. Amino acid histidine is principal protein buffer found in plasma protein and importantly in hemoglobin. This is reduced in hemodulation in CPB.
- Phosphate – is less important buffer system.
- **Bicarbonate** – Principal buffering system in plasma. **Red Blood Cell** plays part in bicarbonate buffering providing **carbonic anhydrous** (CA).

- CA facilitates conversion of carbonic aid to $CO_2$ and $H_2O$ and this prevents accumulation of carbonic acid washing out $CO_2$ by exhalation.
- Body can tune finely the $CO_2$ level through respiration and bicarbonate through renal urine. So bicarbonate buffer is an important buffer system.

**Physiology at CPB :**

- Various manipulation by the perfusionist overrides the body homeostasis mechanism and causes important impact.
- Flow rate – If falls causes inadequate tissue oxygenation.
- A flow index ***of 2.4L/Min/M²*** is considered full flow in normothermic bypass.
- This is between **½ to** of normal cardiac output – ***important to remember***.
- Monitoring of various blood saturation is used to monitor adequate flow.
- **There is strong correlation between Lactic acidosis and venous saturation.**
- *Acidosis should be considered in similar way in ICU as it indicates the late sign of hypoperfusion suggesting imminent tissue damage or has already occured.*

**Dilution :**

Non-bicarbonate buffer

- Normal non-bicarbonate buffer (Plasma& RBC-containing imidazole group of protein) **strength is 28 slykes***(RBC 20,Plasma 8) . Adult with normal plasm protein & 40% Hb contains 30mmol imidazole per liter.*
- Buffering capacity is a measure of titration of specific amount of acid or alkali added to a closed system, causing a change of 1 unit of $P^H$ and the unit is called ***slyke.***
- If crystalline is added to prime there is much reduction in buffer & falls to 33% (28 to 20 slykes).

- If Hct is reduced to 24 – 28% on CPB there will be 20% reduction in non-bicarbonate buffering.
- **Hemodilution thus significantly increases the chance of developing acidosis.**

**Hypothermia :**

- $H_20$ molecules dissociated with temperature change and alters Hydrogen ion concentration.
- As temperature increases more Hydrogen ions forms and $P^H$ falls.
- With cooling number of hydrogen ions decrease and $P^H$ increases.

**Strategy management** : Acid-Base Balance

**Al pha stat :**

- Cold-blooded animals' (Ectotherms) follow this srategy with fall of temperature. These animals maintain a constant ratio of hydroxyl to hydrogen ion across intra/extra cellular compartment in wide range of temperature with parallel change in pH.
- **But they maintain a constant ratio** (*also called alpha*) **of dissociated to non dissociated imidazole group protein buffers of blood**
- These animals behave oposite to' hibernating naimals'(Hetrotherms).Their strategy is to maintain their blood pH constant at 7.4 for all temperature. **Ths technique of pH management is called pH stat**

**$P^H$ stat Strategy :**

- ***Strategy to maintain blood $P^H$ at 7.4 for all temperature*** like hibernating animals ($P^H$ stat strategy).
- Interestingly not all organs in animal (hibernation) do not remain constant as blood.
- Heart and Liver shift to alkaline direction.
- The vital organ thus maintain pH independently in hibernators (Heterotherms).

$P^H$ Stat / Alpha Stat Strategy

- Widely used pH stat strategy.
- Addition of $CO_2$ is done for shifting of $0_2$ dissociation curve to right to make more $0_2$ available.
- Also more $CO_2$ cause vasodilatation and improve cerebral circulation.
- Disadvantage lies in increased microemboli.
- Alpha stat preserves cerebral auto regulation until lower limit of near arterial pressure 30 mmHg.
- **Still there is debate which strategy is good. More trials are suggested.**

$P^H$ Stat Strategy:

- Superiority to alpha stat strategy is well documented.
- Needs addition of $CO_2$ to oxygenator as mixture of different concentration (97/3, 96/4, 95/5, 94/6).

**For CBP in Children:**

- ***Evidence suggests that original approach of $P^H$ start strategy in hypothermic CPB in children is still preferable.***
- In circulatory arrest pH strategy also gives better cognitive result than alpha strategy (Retrospective Study).
- **$P^H$ strategy is also preferred for circulatory arrest in adults.**

**Oxygenation in CPB:**

- **Hyper oxygenation with modern oxygenator aggravates ischemia – reperfusion injury with generation of $0_2$ derived free radicals particularly in heart.**
- Causes higher lipid peroxidation, increased nitric oxide formation and worse cardiac contractility.
- *Other studies suggested that normoxic CPB results greater cerebral injury than hyperoxic CPB.*
- **Although there is controversy normoxic bypass is probably as safe as hyperoxic bypass in continuous bypass with membrane oxygenerator and arterial filter.**

- Normoxic CPB is not appropriate for any age in circulation arrest.
- *Normoxic strategy needs (in clinical practice) mixture of air and oxygen.*

**Neonatal / Paediatric Oxygenators:**

- Essential component of bypass circuit.
- Responsible for gas exchange ($O_2$ / $CO_2$), volatile anaesthetic gases.
- Incorporates heat exchanger for cooling and rewarming of the patient.
- ***For last 2 decades developed models have markedly reduced morbidity of CPB.***

Designs of Oxygenators:

**Disc & Screen Oxygenator:**

- Earliest oxygenator made of mesh screen or solid discs that were covered with thin layer of blood on which interface gas exchange occurs.
- Needs large prime volume.
- **First used in 1953 by Gibbon.**

**Bubble Oxygenator:**

- Lillehi with Dewall was first introduced in clinical practice. Oxygen is bubbled through blood and are liable to produce massive emboli.
- **Became obsolete in late 1980s.**

**Membrane Oxygenerator:**

- 2 types are available.**True membrane** & **Microporous membrane**
- *True membrane resembles lung separating gas & blood in the alveolus.*
- **Microporous membrane can not separate blood completely .**
- Surface area of membrane oxygenator (10-15%) is smaller than lung.

- Oxygenator creating turbulance during exchange increases the exposure of blood to gas at the cost of potential blood injury.

**True Membrane Oxygenerator:**

- Silicon membrane is used.These are stable, heat and chemical resistant (**'thermoset plastic'**) as well anti thrombosis.
- It propels blood and prevent adhesion and highly permeable to gases.
- Prevents foaming and denaturation of proteins.
- Gas transfer occurs with pressure difference by molecular diffusion because of concentration gradient.
- **Only one true membrane series is commercially available (Medtronic 600, 800, 1500).**
- ***Model of choice for prolonged perfusion.***

**Microporous Oxyenerator:**

- Intermediate of membrane and bubble oxygenator.
- Membrane composed of **microporous polypropylene** (**'thermoplastic'**).
- Does not allow diffusion significantly through membrane itself.
- Multiple microporous openings (3-5 μm) in diameter allows a transient direct interface between gas and blood is created.
- *As bypass is started proteins are deposited in the pores and direct contact is lost.*
- Further surface tension of blood against small pores prevents significant blood movement through pores during bypass.
- *After several hours significant amount of serum leaks into the gas compartment causing less performance of exchange.*
- Hollow fibre oxygenerators are not ideal for long perfusion.

- Gas embolism may occur if negative pressure develops in blood side (when filter is not used).
- **Negative pressure may develop when collecting blood sample from the oxygenator clamping inlet and outlet tubes of oxygenerator.**

**Basic Design of (*microporous polyproylene membrane* ) Oxygenators**: 2 types:

Hollow fiber:

- Two types of hollow membrane oxygenerator –*'Blood inside fibres' & 'Blood outside fibres'*.
- Gas pases within the fibres surrounded by blood is known' Blood outside fibre'
- Blood is within the fibre surrounded by the gas.
- Blood inside type is liable to thrombosis in the fibers & blood has to face a higher resistance through the fibre (*Terumo Capiox 300 series*).
- Blood out side(principle) series (Sorin / Dideco Lilliput 1&2, Polystan Micro and Terumo Baby Rx) have less resistance to blood flow.Blood may flow perpendicular /Parallel to the fibre.When parallel blood flows in direction opposite to the gas
- Constrauctions of outlet / inlet manifold is also important to reduce turbulance

**Folded membrane:**

- Microporous is flat sheet folded to create plates that separates blood.
- These membranes has been superseded by hollow fiber oxygenators .

*Development:*

- Few innovative modification have been done since 1955.
- Integrated oxygenator – Heat exchanger pump – filter system (**vortex pump**) **and membrane oxygenator** is major advance.

- Now addition **is vacuum controlled reservoir** from venous cannulae.

**Heat exchanger : Principle & Design**

- These should not increase prime volume in paediatric setting
- Due to extreme differences of temperature used in neonates and infants, air embolism due to change in gas solubility is a problem in these setting than adults.
- Early heat exchanger (**Heater / cooler system**) works on the principle of convection *(prefusate passed actively through tubes / coils surrounded by water ensure the temperature of which is controlled in crude fashion).*
- These are inefficient and works quite gradually.
- The early non integrated heat exchanger needs large prime and difficult in sterilization & cleaning.
- **These are usually placed at the arterial side of the line** *(to avoid pressure drop accross the unit).*
- **Liable to cause gas formation while warming.**
- Development of integral heat exchanger inside the disposable oxygenerator changed the difficulties of old ones.
- **It is possible with these integrated exchanger to pass water through the tubes rather than vice versa and thus the pressure drop that occurs** (when blood passed through isolated exchangers) **is much less.**
- The exchange is placed in the venous side and no chance of gas embolism.
- **Patients intravascular gas formation still occurs if cold blood is rapidly warmed where the patient is still warm during cooling rather than rewarming phase of CPB.**
- Additional prime volume can be avoided in integrated system.
- Stainless steel used in integrated exchanging causes poor heat exchange and more cost (disadvantages).

- *Opposite directon of blood & water flow , addition of steel coils can minimize these problems.*
- Recent heat exchanger changed from convoluted tube to that sheet design similar to gas exchange sheet.
- **These have improved heat exchange efficiently significantly reducing prime volume.**
- Blood passes around the elements not through the system.
- **Difference of 10°C water to venous blood temperature minimizes gas embolism while warming. Blood temperature should not exceed 40°C.**
- **During cooling water temperature should be as was as 4°C.**
- In large children while rapidly cooling chance of micro embolism is small.
- In Infant and neonate rapid cooling increases the risk of micro embolism when cold perfusate enters in warm body (Higher difference).
- *Clinical studies argue against rapid cooling.*

**Heater / Cooler Units:**

- Various model are available.
- Modern units have separate cooling and rewarming chamber.
- It avoids cooling / warming of large volume of reservoir.

**Blood Conservation:**

- Pump – oxygenerator should incorporate ultrafiltration device for concentration the blood left in the machine after CPB, including plasma proteins (*in contrast to cell saver*).
- This concentrated blood can be administered to the patient.
- *Blood sucked from the surgical field can be processed through cell saver system.*
- Cell saver systems separate, wash and concentrate the RBC which is then transferred to the patient. **The plasma components are lost.** Heparin is added.

- Upto 12 hours after operation shed blood from the mediastational and plearal tubes can be collected in the reservoir and returned to the patient.
- **This shed blood has been defibrinated in the patient before collection and contains no clothing factors.**
- Prophylactic antifibrinolytic drugs decreases frequency of reoperation for bleeding.
- **Mostly platelet dysfunction and activation of fibrinolytic cascade are the causes of bleeding.**

**CPB in Neonates, Infants & Children:**

- Preparation for patient is same as adult.
- Surface cooling is employed in addition to core cooling with a cooling blanket and ice around head so that temperature reduces to 30-32°C when the CPB is initiated.

**Aortic Cannula:**

- During hypothermic circulatory arrest the purstring around the aortic cannula can allow air to enter when the pump is stopped (due gravity suction develop in the arterial system and thus air embolism with the aorta at zero pressure.
- **A clamp should be always applied by the perfusionist before discontinuation of CPB.**
- ***Alternatively a clamp distal to cannulation can also protect air embolism, when aortic cannulation can be used for cardioplegic.***

**Cooling :**

- Started with high flow (***2.2 – 2.5 L/mm/mm²***).
- Temperature can be reduced to 18°C in 10 minutes on neonates & small infants. But should not be hurried and **take 20-30 minutes**.
- Temperature of 16°-18° is reasonable for circulatory arrest.
- *Management of gas exchange and arterial $P^H$ is important.*

- Distention of heart is to be avoided.
- A single injection of Cardioplegia is usually sufficient when circulatory arrest does not discards 30 minute.
- Intermittent perfusion during circulatory arrest if there is time constrain is useful.

**Choosing the best Oxygenator:**

*Sould consider -*

- Performance specifications including priming volume heat exchange and gas exchange efficiency.
- Reliability.
- Predictability.
- Easy set-up and use of infusion lines/port.
- Cost.

The final choice lies with joint decision of perfusion, surgical start considering the need of patient.

**Hemodilution:**

- As the experimental (**1937**) and clinical introduction (**1950**) the prime volume was blood.
- Soon the problem of **homologous banked blood syndrome** became apparent.
- In 1960 concept of hemodilutation was presented and largely introduced by Kirklin in clinical practice using 5% glucose, 0.2% Nacl; albumin and 2 liters of blood to make a volume of 3 liters.
- Until late the limitation of dilution was not investigated.

**Physiology:**

- **Dilution of normal hematocrit of 40% to 20% decreases total oxygen content by 50%.**
- The effect of hemodilution in extreme blood is no longer continuous homogenous source of oxygen at tissue levels.
- There is a *critical cell separation distance* ,if exceed the continuous delivery hampers.

- Under normal conditions red cell spacing does not affect oxygenation.

**Viscosity & shear stress:**

- **Viscosity is shear stress divided by shear rate (dynes/cm$^2$).** Shear rate is velocity gradient and the shear stress is the tangential force applied.
- Viscosity of blood is related with shear rate inversely when shear increases (viscosity decreases and decreased shear rate increases viscosity and rouleaux (aggregation) formation.
- In micro circulation when and where flow rate is low and shear rate reduced, viscosity increases.
- In formal microcirculation shear rate is relatively high at pre & post capillary segment and lowest shear rate at post capillary venules and vein.
- **Resistance ratio due to flow rate and shear results increase the capillary pressure.**
- **This is of important role in accumulation of fluid during CPB.**

**Hemodilution & viscosity:**

- When viscosity decreases with dilution the flow characteristic of blood becomes more Newtonian (*i.e. constant linear in viscosity with increase shear rate*).
- **Normally blood is non Newtonian (i.e. changes its viscosity with change of shear rate).**

**Hemodilution and Protein:**

- Dilution decreases colloid oncotic pressure.
- **Colloid is better than crystalloid for hemodilution to prevent tissue ocdema.**
- ***This is more important in neonates than adults.***

**Coagulation factors and hemoglobin:**

- All are reduced with hemodilution.
- Increase bleeding risk and infection.

**Blood for Priming volume:**

- Depends upon determination of hematocrit desired.
- *Formula for calculation* :

  **Prime RBC vol. = ( On bypass HCT ) x ( Pt BV + Prime BV ) – ( Pt. RBC vol.)**
- **Blood should be as fresh as possible**. Ideally for infants or small patients should be less than **72 H** for priming.
- Fresh blood should be reserved for after bypass.
- **Stored blood reduces availability to deliver oxygen as well various electrolyte & metabolites increases within 24-72 H.**
- RBC in banked blood is alive and metabolizes using glucose in CPD solution and develop acidosis that needs correction in prime volume.Nees correction of pH adding $NaHCO_3$ 8.4%.
- Anticoagulant used in banking is mostly citrate. Excess of citrate patients calcium after bypass,which can be corrected by heparinizing the unit & adding calcium to achieve normal calcium before bypass is started
- Some accept this low ionized calcium during hypothermia & do not correct until rewarming
- To avoid citrate some centre heparinize blood for neonatal& infant surgery.

**Crystalloid / Colloid:**

- Controversy exists which one is more safe. No study clearly resolves.
- **Earliest use of clear (5% dextrose) prime resulted higher mortality and complication.**
- There is consensus that crystalloid for children should not include **dextrose/ lactate**.
- Hyperglycemia not only draw water out also cause neurological injury. (Particularly in circulatory arrest)
- **Plasmalyte a crystalloid is being currently used by many centers (particularly for children)**.

- In adult practice colloid is reserved for compromised myocardium to avoid myocardial oedema

**Colloids:**

- ***Albumin*** - This is natural,constitute half of the total plasma protein & give 80% of osmotic pressure.Uniform molecular size(molecular mass 69 kDa)
- Commercially Obtained by plasmapheresis/ Fractionation and available at 4.5% concentration (*Iso-osmotic*) and 10% / 20% (*hypertonic*).
- ***Gelatins*** – (Haemacel) is widely used in Europe.It is breakdown product of collagen.It contain calcium & when given with citrated blood can cause clotting. Commonly like other synthetic colloids the gelatins have small risk of anaphylactic reaction.
- ***Dextrans*** – Like hydoxyethyl starches(also used as synthetic colloid) dextrans are modified polysacchorides – Commonly use are Dextrose 40( molecular mass 40 KDa), Dextan70 ( molecular weight 170 KDa).Like hydroxyethyls molecular weight of dextrans determine its retention in circulation.
- **Dextrans are effective plasma expander with some side effects (allergy, coagulation problems).**
- **Fresh frozen plasma (FFP) and while blood** – Whole blood or RBC are primed with FFP gives additional load of colloidal protein,contains considerable quantity of albumin and coagulation factors.These will be less diluted than using other colloids.

**Ultrafiltration:**

- **Technique to maintain desired Hct during CPB.In case of modfied ultrafltration hemoconcentration is done after CPB**
- ***Widely applied in Paediatric cases.***
- Ultra-filter is palaced parallel to patient circuit and both circuit is on run same time.

- Negative pressure is applied to the filter 7 fluid is drawn through the micropores of the membrane.Filtration of molecules depends upon the pore size of the membrane.
- *Also used in circulatory arrest or pre-bypass period to concentrate the prime to desired hematocrit..*
- **Conventional / modified both are useful. The later is with relative complexity.**
- **Current favourable technique involves Hct (30-35%) during CPB with aggressive application of conventional ultra-filtration.**
- At late phase of rewarming Hct can be increased to greater than 35%.
- *Yet considerable experimental work is demanded to define optimal hematocrit for an individual patient.*

# 6

## Assembling & Techniques

**[ Assembling of P.O.U* ]** - *differs with models*

1. Patient side terminals of Input line Drain line and Suction line should be clean.

   Reservoir and Artificial lung is installed to main body of P.O.U. Hand over vent circuit to the operation field in clean state without opening and receiving later from the same field.

2. Connect the input line with artificial lung. Connection part is located at the bottom of artificial lung and covered with red cap. Do not hang it on the roller yet. (Explained later). Hang the air sucker on the hook. Cut the end of heating line by scissors and connect with Reservoir. (This is the circuit in which trial circulation for blood heating is done and air is sucked at the same time.)

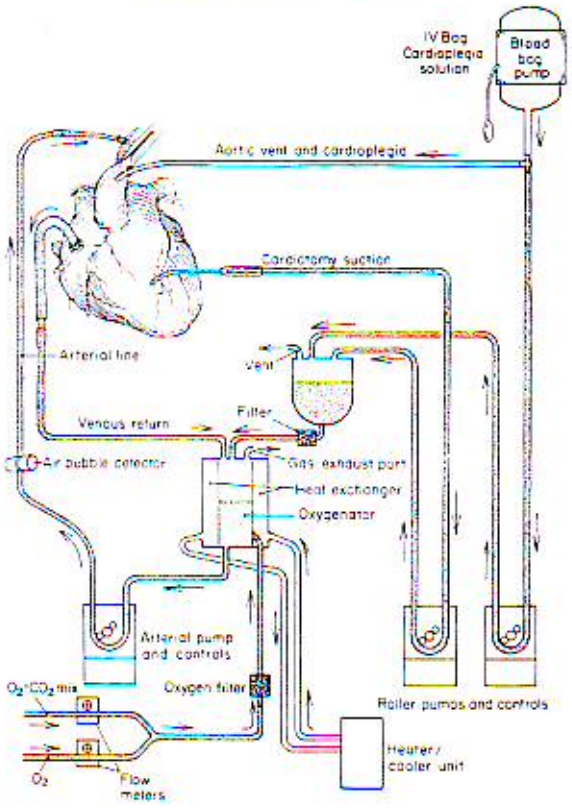
3. Connect drain line with artificial lung. It is easy to find, as it is thicker one with blue cap. Connect the line which connects the reservoir and artificial lung with the blue cap which is located next to the bigger one. (smaller diameter, shorter line than drain line).

4. After suction circuits are hung on roller, connect them with reservoir. Suction circuits are marked with yellow and brown color. Clear distinction is necessary, as suction of either of them has to be enhanced or weakened according to the request by the operator. (Rotation adjusting dials are also differentiated by colors.) .Cut the tip of it by scissors and connect it with the blood transfusion set. The same for the other suction circuit.

* *P.O.U- Pump Oxygenator Unit*

5. After receiving vent circuit from the operation field, hung it on the roller and connect it with the reservoir.
6. Connect the gas suction circuit (filter is attached) with the artificial lung. The circuit has a green cap or written as $O_2$.
7. Insert the thermometer for the input temperature.
8. Connect the expansion tube having three-way cock with artery and vain side respectively in order to gather blood. (In case vain side is omitted, fix the cap firmly and confirm no leakage of the solution.)
9. Connect the heat exchanger with artificial lung by two water pipes. The water flow is IN – OUT, OUT – IN. Confirm indication. Fill two places with ice and water, and hot water respectively and keep the switch on.

*Fig.5 Diagram of Pump-Circuit*




**[ Priming ]**

1. Before desired priming from let O2 and CO2 flow. Without gas flow, the solution makes reverse flow. (When it gets dirty, the procedures have to be done once again from the beginning.).
2. Clamp the forceps at the position shown in the drawing. The path in which the solution pooled in the artificial lung leaks is
   (1) input line
   (2) drain line and
   (3) gas line mentioned the above.

3. Start priming from.Turn the suction dial properly. As there is no loss of suction pressure due to the forceps at the patient end, the solution is sucked quickly from the prime mouth. Forceps at the bottom of the reservoir is to make assurance double sure.

4. Confirm following items after all priming solution enter into the artificial lung:
   (1) Is gas flowing ? (If there is bubbling at the top of the artificial lung, O.K.)
   (2) If forceps on input and drain lines are firmly clamped and there is no leak.
   (3) If three way cocks on blood gathering line are firmly stoppered.
   If the caps are firmly fitted in case vein side is not used.

5. If it O.K., open forceps and introduce prime solution to the input line by taking advantage of head until it pass over the roller. In order to remove the air, hang the line on the roller after tapping it with the forceps. (The air bubbles are hard to be removed at the connection parts of lower side of the artificial lung and the roller.)

6. If the blood pass over the roller, turn the input dial a little bit, and introduce slowly the solution into the air suction part .(Clamp the line which goes to the patient, with forceps) And remove the bubbles completely. After filling up this part, send the solution to the reservoir.
   In this way, the closed circuit from the artificial lung to this reservoir is completed, and make heating possible before bypass starts.

7. Avoid long time heating, as it destroy the blood components. Try to remove the air bubbles during heating. If it is heated up to 36°–70°C, stop and wait.

Filling Preparation Table

| BSA | Optimal Perfusion Index | Size of Circuit (ml) | Priming Volume (ml) | Priming Blood (ml) | Priming Fluid (ml) | Sodium Bicarbonate | Manitol | Antibiotics | $O_2$ Flow | Size of Cannula | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | Aortic | Venous |
| 0.2~ | | S (3/8") | 1000 | 800 | 200 | 20 | | | | 10F | 12~14F |
| 0.4~ | 2.8 | | 1000 | 800 | 200 | 30 | 5ml/ | 1.0 G | | 12F | 16~20F |
| 0.5~ | $L/m^2$ | 30 ml/ RPM | 1200 | 800 | 400 | 30 | Kg | | 1.5 | 14F | 20~24F |
| 0.7~ | | L | 2000 | 1600 | 400 | 30 | | | | 14F | 22~26F |
| 1.0~ | 2.5 | (5/8") | 2000 | 1200 | 800 | 50 | 200 | | | 16F | 26~30F |
| 1.2~ | $L/m^2$ | 80ml/ | 2200 | 1200 | 1000 | 60 | ml | 2.0G | 1.5 | 18F | 28~32F |
| 1.5~ | | RPM | 2400 | 1200 | 1200 | 70 | | | | 20F | 30~34F |

- In case of the infant under 10kg, give FFP 3 packs (200ml) instead of Lactate 200ml
- Heparin 1 ml (10mg) per ACD blood 200 ml
- $CaCl_2$ 2ml per ACD blood 200 ml
- Initial Heparin 3 mg/kg (0.3 ml/kg)
- Additional Heparin 1 mg/kg (0.1 ml/kg)/hour
- In case of non-blood filling, don't add Heparin and $CaCl_2$. The blood is substituted by Lactate.

Total Perfusion Flow =Perfusion Index X BSA

[Normal Flow =2.2-2.5L/$m^2$]
LowFlow =2.5l/$m^2$/min(40ml/Kg) >31$^0$C
HighFlow=3.5L/$m^2$/min(55-70ml/Kg)>28$^0$C

**[ The Way How to Hand over Circuit ]**

1. Hand over input line drain line and suction line to the operating field. If they are stained by mistake, this procedure has to be done over again.

2. Procedures are as follows:

   1. Remove the tape which fixes the yellow cover.
   2. Hold the circuit with left hand and pull the cover about 10 cm
   3. Hold the circuit with left hand and pull the cover about 50 cm with right hand.
   4. Ask the nurse or the assistant operator whose hands are clean to hold the clean part of the circuit.
   5. Have the circuit pulled while the cover is held. Do not pull the cover from this side, as the end of clean portion might happen to drop and contact something.

**[ Just before Starting of P.O.U ]**

1. Connect the input line and the drain line in the operating field. When forceps Fig (e) is clamped and forceps Fig (f) is opened, the solution is sent from the input line to the artificial lung through the drain line. The air in the input line is carefully removed in the operating field and the solution is sent to the drain line. Here remove the air in the drain line by clamping the two drain lines alternately with the forceps. (The air in the drain line has no direct danger but causes improper drainage.)

2. Before cannulation of input and drain in the operating field, Heparin is added. As the prime solution temperature drops again by this time, heat it again. When blood temperature is raised enough ($36 - 37^0C$), the input and drain lines are separated in the operating field. In this moment, open the forceps on the drain line and return the blood to the drain line so that it will not be lost at the time of separation, and clamp it again with forceps.

3. Confirmation of CVP monitor and the rectal temperature should be done by the anesthesiologist, in order to know if they are functioning properly.

4. Declamp the forceps on the suction circuit, and confirm the prime inlet (d) is closed. (If it is opened, suction get poor.)

5. Start suction in the suction circuit and the vent circuit.

6. Cannulation of the input tube is done in the operating field. Confirm the forceps on the input line side (f) is clamped. (If the forceps is not clamped while pump is operated, too much blood is supplied.) After cannulation, connect the input tube with the drain cannula tube, but if there is too much bleeding in the operating field, slow down input according to the instruction by the operator or the anesthesiologist.

7. Cannulate the drain tube. Supply of the blood is necessary if there is too much bleeding.

8. Remove the forceps (f) on the input line and wait in ready to start at any time. At this time, only forceps (b) (e) on the heating line and (g) on the drain line are clamped. Confirm that the artificial lung is the highest position.

[ **Start of Bypass** ]

1. Start partial bypass by the instruction of the operator.

   Start input; Rotate the pump slowly and deliberately looking at the arterial pressure and CVP, and increase input up to 1/5 – 1/4 of total flow (about several seconds) (The blood pressure and CVP goes up slightly.)

2. Start drainage. (Start the bypass also at this time.)

   Open forceps slowly and slightly. Keep watching on the solution level of the artificial lung. When the level comes up due to drainage, increase input at the same time.

   Confirming the solution level of the artificial lung goes up because of slow and slight opening of the forceps, continue to increase input by that amount and open

the forceps fully, and at the same time, increase the pump rotation to the extent of total flow.

3. Blood pressure and CVP drop with the start of drainage, but the level of the artificial lung is more important and it is the best indicator of drain condition.

4. If there is no rise of the solution level due to bad drainage, stop the further input. (Otherwise, total amount of blood will be sent at once, resulting the air supply to the patient.) If there is no level up, stop input and decrease rotation of the pump to the extent where there is no more level down.

   Caution to arterial pressure is necessary at this time. CVP does not drop. (In case of good drainage, CVP drops remarkably.) Inform the operator of bad drainage in order to adjust the position of the drain tube. There are many cases of wrong position, but even if the position is adjusted, bad drain remains sometimes, especially in the case of a thin tube for the infant.

5. Remove forceps on drain line. If drain was done well and the total flow of input was completed, let the anesthesiologist and the operator know this, and lower the position of the artificial lung and keep the adequate level.

6. CVP is almost 0 or under 0. There is sudden initial drop of blood pressure but it goes up soon. Initial drop can be countered by slightly increasing input or by increasing 0 flow. But dosing of – stimulant (Phenylephrine , Methosamine ] has little effect to the increase of the blood pressure.

**[Start of Cooling Arrest]**

1. Turn the switch of the heat exchanger to "Cool" position, and start cooling. (Hot water for heating is kept at the constant temperature not constant. Take Care.) As the temperature of ice water is around 10°C, avoid rapid cooling. (It causes cardiac arrest.) Turn the switch off 5 –

10 seconds after the switch is on, and observe the input temperature. It starts to drop with a little time lag. Make cooling again, after confirming the temperature goes up again after it drops once. Repeat this process with care, and obtain the desired low temperature.

2. Rectal temperature takes time to drop (In case of rectum thermometer disorder, the esophageal temperature is used. Esophageal temperature react quickly as it is near to the heart, aorta and venacava.) The temperature difference (Blood – Rectum) within 5°C is desirable.

3. When the blood temperature drops into 28° – 30°C, clamp the aorta to make anoxic arrest. Rectum temperature has not yet dropped enough 30°C is desirable, it does not matter even if it is higher, as the heart is cooled by the ice water in the operating field.

   Start to measure the arrest time by stop watch on the instruction of the operator, and ventilation is stopped by the anesthesiologist.

4. Cardioplegia is injected and the cardiac arrest takes place. Cardioplegia comes out to RA through coronary and is introduced to the artificial lung by the drain tube, and the level goes up.

5. Continue further cooling after cardiac arrest, and obtain desired low body temperature, As rectum temperature, care is necessary so that there will be no too much cooling of the blood.

6. As cooling proceeds, the blood which dropped once, goes up again.

7. After 5 min. from starting the bypass, gather the blood and subject it to inspection. Check the blood gas, electrolyte, and the blood sugar.

**As soon as the heart stopped beating, LV expands, therefore, over-distension must be avoided. In many cases, LV vent comes in before arrest.**

[ **Maintenance of Bypass** ]

1. Perfusion flow is as follows, but adjust the flow within tolerance depending on the circumstances.

| Input Tube | BSA | Flow | Minimum | Maximum |
|---|---|---|---|---|
| S | 0.2 - 0.4 $m^2$ | 2.8 l min/$m^2$ | 100 ml/kg/min | 1.20 ml/kg/min |
| M | 0.5 - 0.7 | 2.6 | 100 ml/kg/min | 1.20 ml/kg/min |
| L | 1.0 - 1.5 | 2.4 | 60 | 80 |

There are three sizes (S, M, L) in the input tube. As respective input amounts per rotation of pump is fixed, rotational frequency of pump can be known from the desired amount of perfusion.

| Input Tube | S | 28 ml/RPM |
|---|---|---|
| | M | 48 |
| | L | 74 |

e.g. *When perfusion is done to the patient whose weight is 60 kg at 60 ml/kg/min, total flow is 60 x 60 = 3600 ml/min.*
*In case of the input tube L,*
*3600 , 74 = 49 rpm -- 50 rpm*
*(49 - 50 can not be differentiated by the scale of meter.)*

2. Blood is oxidized by gas flow which was sent to the artificial lung, and $CO_2$ is removed. As a reference, the ratio of $O_2$ to infusion amount is 1:1, $CO_2$ is 1/100 of $O_2$.

   If infusion amount is 4 L/min, $O_2$ flow is 4 L/min and $CO_2$ is 40 ml/min.

   $PCO_2$ should be over 35 TORR

   If $PCO_2$ drops, raise $CO_2$ flow or lower $O_2$ flow (= total flow) in order to raise $PCO_2$.

3. Keep the following parameters in normal values:

| | | | |
|---|---|---|---|
| **Average Arterial Pressure** | | : | **50 - 80 TORR** |
| **CVP** | | : | **near 0** |
| **Blood Gas** | **PH** | : | **7.35 - 7.45** |
| | **$PO_2$** | **Over 150 TORR** | |

| | |
|---|---|
| $PCO_2$ | 35 – 45 TORR |
| $PVO_2$ | 45 – 45 TORR |
| **Electrolyte** | **$K^+$ over 4.5 mEq/1** |
| **Hematocrit** | **20 – 30% (Hb 7.0 – 10.0 g/dl)** |
| **ACT** | **400 – 450 sec** |
| **Amount of Urine** | **Over 1 ml/kg/hr** |

High $PVO_2$ is caused by A-V shunt, and it is high under the low body temperature. Low $PVO_2$ means the rise of $O_2$ extraction, and means the bad tissue-infusion. Check the state of infusion again and increase the flow.

4. **Electrolyte**

Sometimes K indicates the low value due to the blood dilution, increase of the urine volume or the shift to the cells. It is corrected by KCl (1 mEq/ml). If the urine volume is large, and the decrease is remarkable, more KCl should be used. Since Cardioplegia solution is added every 30 min., K is absorbed into the artificial lung, and K concentration increases. (It is sometimes sucked by Wall Suction and thrown away outside. Take care.)

**Adult**

**Cardioplegia:** Young solution
2 ml/kg (100 ml)

G I K solution
10ml/kg (500-100ml)

**Components of GIK solution**

| | |
|---|---|
| 5% C | 500 ml |
| KCl | 10 mEq |
| Meilon | 5ml |
| Insulin | 0.25 ml (5 u) |
| Albumin | 50 ml (Adult only) |

If K concentration is high, increase the urine volume. (Mannitol, Lasix etc.)

5. **Hematocrit / Hemoglobin**

It should not be 20% or less, and Hb should not be 7.0 g/dl or less. ACD blood (fresh), ACD blood (stored) and the concentrated red blood cell should be supplied. Especially in case of the priming without blood, dilution is remarkable. Take care. And also be careful not to delay the timing of transfusion of the blood. Consultation with the surgeon and the anesthesiologist is necessary.

6. **ACT**

3 mg/kg of Heparin is dosed by RA or CVP, just before starting the bypass. ACT value necessary for the bypass is 400 sec or more. If it goes down to 300 sec, coagulation in the circuit might occur. Therefore, it should not be leas than 400 sec.

At the time of priming, Heparin is added by 10 mg (1 ml) per 1 pack (200ml) of blood. Half life of Heparin is 90 min.

**[Note] Normal ACT value is 107 sec (81 – 133 sec).**

*Due to the lack of coagulation factor, decrease of the number of thrombocyte does not affect ACT value. It is prolonged considerably under the low body temperature.*

Usually, 1 mg/kg of Heparin is added every 1 hr after the first dosing so that ACT value can be kept 400 sec or more. If finishing of the bypass is soon, and if still ACT value is fairly prolonged, the amount of Heparin to be added should be controlled.

7. **Amount of Urine**

Even if the amount of urine between the beginning of operation and the starting of the bypass is small, sometimes pretty large amount of urine is discharged after starting the bypass. In this case, pay attention to the decrease of $K^+$, and the level of Oxygenator

In case of priming without blood, rapid decrease of level can be seen. When the amount is small,

(1) Recheck the renal function of the patient.

(2) Pay attention to the increase of K and save the additional dosing of K.

(3) Check the flow. (sufficient?)

(4) Check the average arterial pressure (too low?) (If the blood pressure is too low, the urine is not discharged even if the flow is proper.) Then, diuretics (Mannitol, Lasix, etc.) are dosed. Especially in case of hemoglobin urine, diuretics are necessary in order to maintain proper amount of urine.

8. Other

Time Keeper:

Measure the arrest time , tell it to the surgeon every 30 min., and let him know the time of Cardioplegia injection.

**Chart Recording**:

Blood pressure, CVP (SVC, IVC), blood temperature, rectum temperature, amount of urine, used drugs, added blood and etc. are recorded on a chart.

**[ Check-points in Bypass ]**

**1. Arterial Pressure**

Pay attention not only to the digital indication, but also to the wave forms on the monitor. It might be "dull" due to blood drawing from the arterial line or due to bad positioning of cammula. Checking by an anesthesiologist is necessary.

**2. CVP**

CVP should be checked in the same way. Three-way cock might be off due to dosing of drugs, drawing of the blood & etc.

**3. Blood Drawing, Heparin Adding**

Check the values every 30 min. after starting the bypass, in order to prepare for the abnormal values. Adding of Heparin should be always kept in mind.

**4. Blood**

It should be kept in mind what kind of, and how many bottles of blood remain.

**5. Amount of Urine**

If suddenly the flow of Urine became very slow, there may be obstructions in the route from the bladder → Foley, Catheter .

If there are some bubbles, "Air Lock" occurs, and the urine can not be discharged.

**6. Line, Piping, etc.**

A lot of cords for various equipments, or input (drain) tubes, are used in the state of "Spaghetti Syndrome". Be careful to avoid disconnection, especially not to step onto the blood input tubes.

**7. Suction**

Useless suction may cause the blood distraction or hemolysis. Stop it when the operator does not use it, and when it is used, do it as quickly as possible.

**8. Vent Circuit**

Sufficient revolution (Speed) should be given to the vent circuit. Take care to avoid over-distention in the left chamber.

**[Preparation for Weaning]**

1. After intracrdiac operation is finished, recovery of temperature is started at the sign from the operator. The switch of heat exchanger is turned to "Hot". Take care never to raise the temperature of the water in the chamber up to 42°C or more. Unlike cooling, the switch is always set to "Hot". Blood temperature increases rapidly, but the rectum temperature does not increase so soon.
2. After declamping of the aorta, the blood starts to flow again in to the coronary artery, and the self-pulsation

starts. But sometimes self pulsation does not start if the blood temperature did not yet recovered to about 32°C.

3. After declamping of the aorta and loosening of taping of SVC & IVC the blood flows into the heart, and the level of Oxygenator and the average arterial pressure decreases.
4. Check the arrest time.
5. Between aorta-declamping and bypass-weaning, various treatments are done in the operating field. In the meantime, recovery of temperature should be done sufficiently.

(Blood temperature → 37°C, Rectum temperature → 34°C).

In the operating field, LV vent circuit is removed, and LA catheter is inserted. Meanwhile, the position of Oxygenator is raised so that the air does not flow from LA to LV. → Aorta, or if necessary, the venous drainage tube is clamped with forceps by 1/3 – 1/2, in order to put the blood back into the patient, and to fill the blood into the heart. After LA catheter is inserted, P.O.U. is put down to the former position, (the forceps is removed), and the blood is put back into Oxygenator.

6. If LAP is shown, O point is confirmed by the anesthesiologist. During operation,

sometimes the operating table might be moved up and down, or rotated. Wave forms

of LAP should be also confirmed (Digital indication shows the mean value.) In this

time, LAP is around O.

7. Blood in the vent circuit is put back into A.L. (Artificial Lung)

* Thus, considerable amount of blood can be saved, especially in case of priming without blood.

8. After the hemostatic treatment is finished in the operating field, and after the rectum temperature goes up to 34°C, bypass weaning is started. But beforehand, following points should be checked.

**(a) Rectal temperature --- 34°C**

**(b) Blood gas, Electrolyte, Hct**

**$K^+$ ----- 4.0 mEq/l or more, Hct -------- 20% or more**

**(Hb ----- 7.0 mg/ml or more)**

**(c) $Ca^{++}$ is within the normal range ?**

**(d) ACT value**

**(e) Wave forms of artery, and of LA, are all right ?**

**Aren't they "dull"? O point is all right ?**

**(f) CVP route is all right ?**

*** This route is sometimes used as the route for Catecholamine or for vasodilators, by the anesthesiologist, and it might be switched off. (Confirmation by the anesthesiologist is necessary).**

**(g) Level of A.L. is sufficient ?**

**If it is not sufficient, the blood or Lactate Ringer solution should be provided so that weaning can be done with ease.**

**[ Weaning ]**

1. Fundamental rules of weaning are as follows:

**(1)** Weaning should not be done in haste. All operations should be done gradually.

**(2)** Always pay attention to the level of A.L. in order not to put the air in.

**(3)** LAP value rises later. Be careful not to raise LAP value too high, because over-distention is very dangerous. (CVP rises far later.)

2. At first, raise A.L. up to the highest position. The level goes down, and LA 6 CVP go up. Sometimes wave forms due to cardiac beats are shown in the arterial wave forms.

3. Drainage tube is clamped with forceps by 1/4, to raise LAP. If the level keeps falling, the input of blood should be decreased. Then clamp the tube more with another forceps in order to decrease the drainage. While LAP rises, the operator should check the "distention" of the heart, in order to avoid over-distention. In accordance with the rise of LAP, the arterial wave forms become bigger due to the cardiac beats. Do pre-loading when the cardiac minute volume is the maximum. The best LAP value is determined, maintaining the systolic blood pressure at 80 – 100 $^{TORR}$. This LAP is said to be 10 – 15 $^{TORR}$, but actually it varies more or less depending on the transducer position, or on the small change of 'O' point.

4. Amount of drainage is decreased, by clamping little by little (1/4 → 2/4 → 3/4), in order to get the proper LAP value. Amount of input is also decreased gradually so that the level of Oxygenator does not fall too low.

5. After the drainage is decreased, and pre-loading is done, if still the systolic blood pressure is not enough and LAP value keeps rising because of bad function of the heart, the input should not be decreased too much. Increase the drainage first, and lower LAP value, avoiding over-distention. Then the anesthesiologist starts to dose Catecholamine & vasodilators.

6. If the heart beats are good, and also if LAP and the blood pressure are proper, the whole drain tube is clamped with forceps when the amount of drainage is finished. (Bypass finishes).

7. Stop the input of blood, and check. Depending on hemorrhage in the operating field, expansion of the peripheral (blood vessels) or the volume of urine,

shortage of circulating blood may occur, and LAP may fall gradually. Therefore, input of blood should be repeated little by little, paying attention to LAP value.

8. Drain tubes are taken off one by one from the right atrium, but input tubes are left till the end to prepare for unexpected hemorrhage. (Small amount of hemorrhage occurs when drain tubes are taken off.)

9. If the state of circulation is stable ,input tubes are taken off.

10. When hemostatic operations are finished to some extent, the anesthesiologist doses Protamine, reverses Heparine, and starts the blood transfusion. Bypass is not yet finished, because the blood might be input again on the instruction of the anesthesiologist in case of unexpected hemorrhage, or blood pressure fall, etc. (Input to the right atrium)

11. If the state of circulation is stable, the input line to the right atrium is taken off.

12. Thus, the bypass is finished. But if the state of circulation turned bad, the bypass might be started again. Therefore, clearing should not be done immediately. While waiting, calculate the water balance, the blood balance, dilution, etc.

[**Counter measures in Case of Emergency** ]

**1.** If the pump stopped suddenly,

(1) Turn the handle by hand. (Handle is attached on the right side of A.L)

(2) Check the power source inlet. In case of power failure, wait until it is recovered.

**2.** If the air is put in,

(1) Stop the input pump.

(2) Clamp the drain line. (If too late, it might cause the death of the patient.)

(3) Turn off the switch of input pump, and reverse its revolution by hand.

(4) Remove the air through air sucker in the operating field.

(5) Start to input the blood.

3. If the blood over flowed Oxygenator due to over-drainage,

(1) Lower the position of the reservoir to reserve the blood in it.

(2) Be careful especially in case of Giant LA.

4. If the drainage volume decreased suddenly,

(1) In case CVP is high.

(a) The operator might step on drain tubes.

(b) Drain tubes might be folded under the sheet in the operating field.

In both cases, the operator should be informed again correctly.

(2) In case CVP is low,

(a) The blood might be drained from the wall suction.

(b) Too much hemorrhage from the incised part (in case of Femoral incision)

In both cases, do the blood transfusion.

5. If the pump is driven with the input tube clamped.

**Do not declamp immediately, because the rubber tube is expanded.**

(1) At first, turn off the switch of pump.

(2) Open the clamp gradually.

(3) Start the input again.

[ **Clearing** ]

1. Straight parts of the suction circuit are cut off and handed to a nurse. (-to be recycled as suction tubes).

2. Follow the instructions or know-how of your superiors.

(2) Throw the blood into the bucket

(3) Be careful not to throw away the forceps.

3.Thus, **clearing** is finished.

# PAEDIATRIC PERFUSION

# 7

## Paediatric Perfusion

Paediatric patients need separate consideration in planning vigilance during perfusion. All equipment are to be made available during the procedure.

These patients differ:

- *In size*
- *The heart itself is proportionately larger than the body*
- *Circulating volume is reduced*
- *Vascular system is more reactive*
- *Differs in doses of drugs*
- *Different formulae are used in preoperative calculation*

**Perfusion Techniques also differ in paediatric patients.**

Such as :

- Low flow state/Circulatory arrest
- Pulsatile flow/Constant flow
- Apha stat/ $P^H$ stat

Until Certain technique gain acceptance perfusionist should be strict to the general concept and deal the situation in the ways that are accepted by most perfusionists.

**Calculation of Blood flow:**

Paediatric patients have higher metabolic rate than the adults.

*Flow is determined by multiplying the body surface area by the cardiac index at full flow.*Mostly cardiac index of 2.8-3.2L/$m^2$/min is used to calculate the flow.

Flow rate of 60-80ml/kg/min for larger children & 80-125 ml/kg/min is necessary for the infants.

Low rate can also be calculated with the help BSA chart with the followig table:

## Flow calculation using BSA :

| | |
|---|---|
| New born~2 yrs | 3.0-3.2 xBSA |
| 2~4 yrs | 2.8 x BSA |
| 4~6 yrs | 2.6 xBSA |
| 6~10 yrs | 2.5 xBSA |
| 10~ above | 2.4 xBSA |

## Circuit Size

*(Appropriate size is necessary to avoid dilution/low flow)*

| Weight | Arterial | Venous |
|---|---|---|
| <4.5 kg | ¼ | ¼ |
| 4.5~9.0 kg | ¼ | 3/8 |
| 9~40 Kg | 3/8 | 3/8 |
| >40 kg | 3/8 | ½ |

## TUBING VOLUME

| Tubing Inside Diameter | ml / ft |
|---|---|
| 1/4 in | 9.65 |
| 3/8 in | 71.71 |
| 1/2 in | 38.61 |
| 5/8 in | 48.00 |

**Commercially available Oxygenators wth flow :**

OXYGENATOR

**AVECOR**

| | RATED FLOW (LPM) | Prime Vol (ml) | $O_2$TRANSFER ( ml/min@LPM ) |
|---|---|---|---|
| ULTROX III | 4.0 | 395 | 230@4.0 |
| 0400-2A | 0.35 | 60 | |
| 0800-2A | 1.2 | 100 | |
| 1500-2A | 1.8 | 175 | |

| | | | |
|---|---|---|---|
| **COBE** | | | |
| VPCML PLUS | 1.3 | 70 | 64@1.3* |
| | 2.7 | 140 | 135@ 2.7* |
| | 4.0 | 210 | 215@4.0* |
| **DIDECO -SORIN** | | | |
| MASTERFL INFANT | 2.0 | 135 | 80@2.0 |
| MASTERFLOP ED | 3.5 | 160 | 125@3.5 |
| **MEDTRONIC** | | | |
| **MINIMAX** | 1.5 | 140 | 83.2@1.5 |
| **MINIMAX PLUS** | 2.3 | 149 | 94@2.3 |
| **SARNS** | | | |
| SMO/INF | 2.5 | 170 | 132@2.5 |
| **TERUMO** | | | |
| **CAPIOX CX308** | 0.8 | 80 | 30@.08 |
| **CAPIOX CX320** | 2.0 | 200 | 80@2.0 |

( • *At barometric pressure of 620 mm Hg.* )

**Arterial line Filters are listed in this table with appropriate flows and priming volumes.**

## FILTERS

| Name | Maximum Flow | Prime |
|---|---|---|
| Pall ¼ in | <2 LPM | 50 ml |
| Baxter AF 540D | <3 LPM | 115 ml |
| Sorin Micro P | <3 LPM | 100 ml |

## Parameters for Cannulae

### ARTERIAL CANNULAE

**SARNS:**

| Name | Size | Flows |
|---|---|---|
| *High Flow Aortic Arch* | 3.8 | <1.6 LPM |
| *High Flow Aortic Arch* | 5.2 | <3.5 LPM |
| *High Flow Aortic Arch* | 6.5 | <5.2 LPM |

**SORIN BIOMEDICAL:**

| *Size* | *Flows* | *Size* | *Flows* | *Size* | *Flows* |
|---|---|---|---|---|---|
| **3.0 mm** | <1.2 LPM | **8F** | <0.6 LPM | **18F** | <4.0 LPM |
| **3.8 mm** | <1.8 LPM | **10 F** | <0.9 LPM | **20F** | <6.5 LPM |
| **4.5 mm** | <2.9 LPM | **12F** | <1.5 LPM | | |
| **5.2 mm** | <4.1 LPM | **14F** | <2.5 LPM | | |
| **6.58 mm** | <6.3 LPM | **16F** | <3.0 LPM | | |

**BARD:**

| | | |
|---|---|---|
| FEMORAL ARTERY | **16F** | <2.4 LPM |
| FEMORAL ARTERY | **18F** | <3.5 LPM |
| FEMORAL ARTERY | **20F** | <4.3 LPM |
| FEMORAL ARTERY | **22F** | <6.2 LPM |

**Priming volume:**

Priming volume of oxygenator,filter,tubings & reservoir are calculated to find out total priming volume.Dilution effect then can be calculated.

**Priming componants:**

- Infants have higher Haemoglobin than adult.But it is lower than adult by three months.
- Paediatric patients contain more blood /kg wt than adults

**Blood volume by weight**

| Weight | Volume |
|---|---|
| New born(15~30 min) | 76 ml/kg |
| New born(24 H) | 83 „ |
| 05~10 kg | 85 „ |
| 11~20 kg | 80 „ |
| 21~45 kg | 75 „ |
| 45kg > | 70 „ |

*[Example:*

- *If wt is 5 kg then volume is 5x 85 =425 ml*
- *If Hct is 55% then RBC volume of Pt. is 425 x 0.55 =234 ml*
- *To Calculate the vol of RBC to make Hct 25% at bypass 425 +800(priming vol) x 0.25 = 306*
- *Pt has 234 ml RBC but needs 306 ml.*
- *So 306-234=72 ml of RBC are required*
- *If Hct of ppacked RBC is 70% then 72÷0.70=103ml of packed RBC is needed to have Hct of 25%*
- *Then 103 ml of priming volume to be taken off to prevent dilution by adding 103ml packed cells*
- *Now priming vol stands 697 ml in addition to 103 ml packed RBC]*

- Fibrinogen,a coagulaton factor decreases with haemodlution.Usually **100mg/dl** is minimun leve to be maintained.Fresh frozen plasma may be added *( after calculating total quantity needed)*
- Albumin may be added to prime to maintain colloidal osmotic pressure about **25 mmHg**

The contains **of Prime :**

**Crystalloid**
**25% albumin**
**FFP**
**RBCs**

**Use of Whole blood :**

- Use of heparinized whole bood is available that eliminates use of fibrinogen.
- Commonly used in neonatal bypass as bilirubin is lower than stored blood & citrate from CPD solution is not present
- Blood is with poor $O_2$ carrying capacity & low $p^H$

The increased metabolc requirements dictates a heigher perfuson rate in small patients. **60~80 ml/kg/min** appears to be adequet for paediatric patients.
**80~100 ml/kg/min** is preffered in small paediatrics & infants.

**Haemodilution may also be calculated as follows :**

**Estimated Blood Voume = 80 ml/kg x wt (kg)**
**Estimated RBC vol = Hct x EBV**
**Total Circulating vol (TCV) = EBV + Prime vol + IV fluid vol**
**Estimated Haemodilution HCT = Estimated RBC Volume / TCV**

**ACT OF BYPASS:**

- Done mostly in traditional manner.Remove the occluding clamps slowly to avoid shift particularly in paediatric patients.Flow are gradually adjusted to calculated full flow.At first the patient may show hypotension due to dilution. **Mostly it is thought that BP >50 mmHg is best.**
- **If BP does not rise to normal level after few minutes,it may be necessary to add agents (neosynephrine).To start in low dose(5~19 mcg) to see the response.Dose can be adjusted accordingly.**
- **If hypertension flow may have to be decreased while medications to take action.Drugs commonly used for hypertenson are Halothane, Nipride, Fentanyl ,Na-Pantothal.** Keeping flow some-what low is of no harm with BP elevated.
- *Arterial $pO_2$ should not be high in infants to avoid retinal damage.*Safe $pO_2$ is not estabished but danger of high level to be recognized.
- Cardioplegia to be administered at low pressure to avoid damage to small heart.
- Circulatory arrest is used routinely in small patients althogh the long term effect is yet unknown
- Low urine output is common is paediatric patients but some urine output is desirsable.
- If mannitol in the prime is not sufficient diuretics may be added
- Coming off bypass is the traditional with care taken not to overfill & distend the small heart.

# 8

## IABP

**Counter pulsation : IABP [INTRA AORTIC BALLON PUMP ]**

**Physiology & mechanism :**

IABP only augments cardiac function by reducing afterload & increasing diastolic pressure.This is contrary to VADs that can completely replace pumping function of failing heart

*Basic Strategy to use devices:*

Mostly IABP is firstly used.can be put in ICU.
If IABP *+Pharmacological support* fails adequet tissue perfusion LVAD is indicated

**INDICATIONS : IABP**

1. Post cardiotomy cardiogenic shock ( inability to wean from CPB)
2. Cardiogenic shock after
   *AMI,unresponsive to medical therapy*
   *Primary myocardial dysfunction*
   *VSD*
   *MR( papillary rupture)*
3. Unstable Angina
   *Pre MI & Post MI*
   *Failed angioplasty - travelling to operation*
4. Ventricular tachyarrhythmias caused by ischemia
5. Bridge to transplantation
6. High-risk cardiac patients undergoing general surgery
7. Adjunct to mechanical ventricular assistance

**CONTRAINDICATION:**

1. Aortic Regurgitation
2. Dissection
3. Thoracic aneurysm

4. Peripheral Vascular Disease(severe)
5. Blood dyscarasias
6. Irreversible Brain Injury
7. End stage Ventricular failure

**IABP** : Inflation & deflation of ballon in synchrony with cardiac cycle can optimize $O_2$ consumption. 3 parameters can be adjusted accordibg to changing requirement of patients.

**Ballon size :4.5 -12.0 F** (Typica adult requires 8.5 -9.0F cath, 40cc ballon) Synchronization achieved by various trigger modes:

- ***ECG*** - Most frequently used
  HR >150/m decreases IABP efficiency
  **Usefull in arrest**
  **Usefull in AF**
- ***Pressure*- Inconsistant ECG trigger**
  **Usuing electrocautery**
  **Requires systolic BP >50 mmHg**
- ***Pacer*** **- For A-V / Ventricular pacing**
  **Requires 100% pacing**
- ***Internal*** **– When NO CARDIAC OUTPUT**
  **Set rate**
  **When BP<50mmHg**
  **(Augmentation should be <1/2 )**

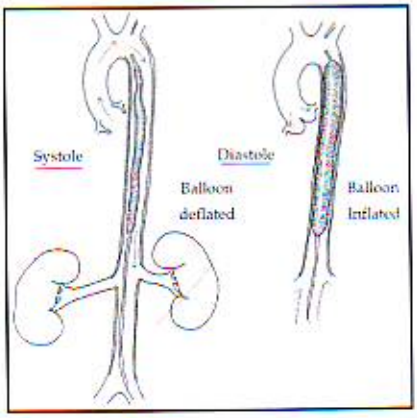



*Fig: Correct positioning IABP*

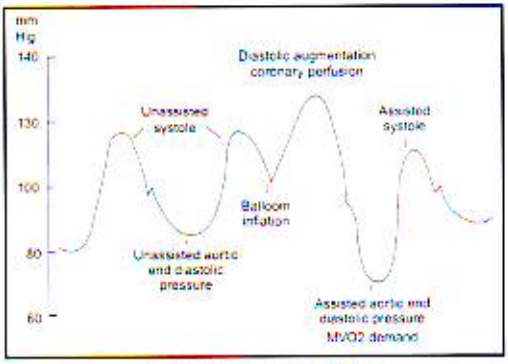



*Fig: Arterial waveform*

Inflation must occur just after AV closure & deflation as AV opens for proper augmentation.Optimum timing can be determined by wave form.The same wave form & Pt. status reveal TIMING ERRORS.

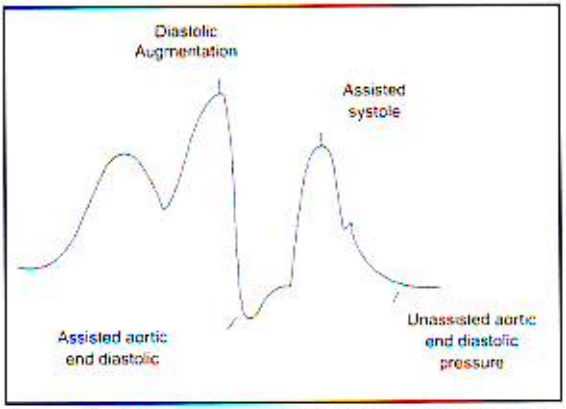



*Fig. Early Deflation (Premature deflation in diastole)*

Characters:

1.Sharp drop after diastolic augmentation
2.Suboptimal augmentation
3.Assisted EDP equal / greater

Effects:

1.Suboptimal CA perfusion
2.Retrograde CA perfusion
3.Angina.
4.Suboptimal afterload

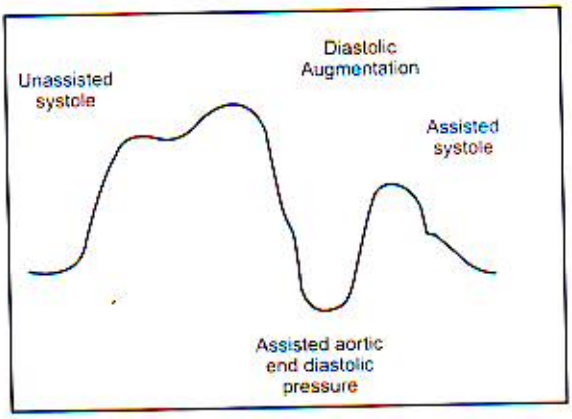



*Fig. Early Inflation*

Character:
1.Inflation before notch
2.May encroach systole

Effects:
1.Premature closure AV
2.LVEDP/ PCWP ?
3.Increased MVO2 demand

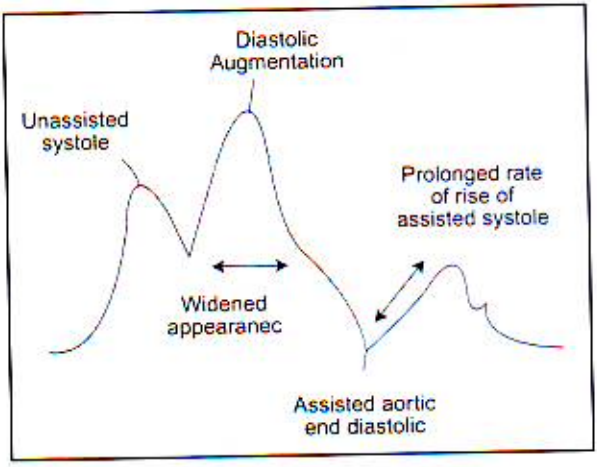



*Fig. LATE DEFLATION*

Characters:
1.Assisted EDP may be equal
2.Diastal augmentation widen

Effects:
1.Afterload ? essentially absent
2.Impade LV ejection
3.MVO2 ?

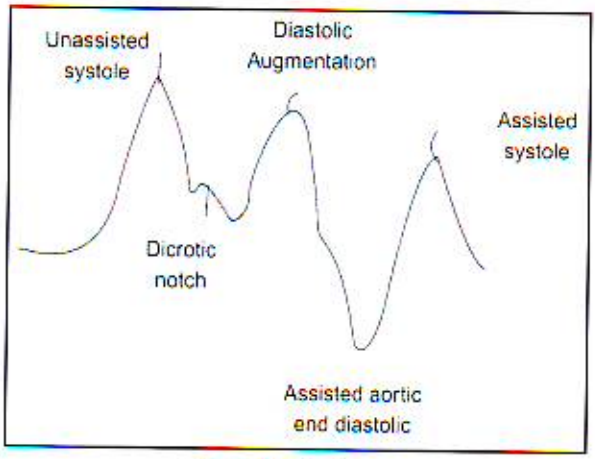



*Fig. Late Inflation*

Character:

1.Inflation after notch

2.Sub optimal augmentation

3Abscence of sharp V

Effects:

1.Sub optimal coronary perfusion.

INFLATION FREQUENCY SET UP :

By **FREQUENCY: HR ( 1:1 , 2:1 )**

**APPROPRIATE ADJUSTMENT WITH INFLATION / DEFLATION TIMING.**

**WAVEFORM & PATIENTS STATUS REVEALS CORRECT TIMING**

**WEANING FROM IABP :**

***PRINCIPLE*** : To withdraw support incrementally & to asses haemodynamics at each step

- Pt must be stable with minimum inotrops
- CI >2 L/min/m2
- Systolic Pressure >90mmHg
- LA , RA pressure <20 mmHg
- HR <100/min
- Urine >0.5 ml/kg/H

Decrease inflation frequency 1:1 to 2:1 to 3:1 at 1-2 hr interval

Decrease amount of augmentation to minimum of 50% for prevention of thrombosis

**When minimum support tolerated for hours**

**withdraw taking care to purge clot from proximal & distal femoral artery**

Direct pressure for some minutes(20),apply sand bag for 6hrs, confined to bed for 12 hrs

**IABP : COMPLICATION OF**

1) Limb Ischemia
   Most common (5-19%)
   Related to cardiac output, catheter diameter,intimal injury,thrombosis.
2) Perforation –Common in shock,PVD
   Sup.Femoral Artery – thrombosis,leg ischemia
   Abdominal vessel - Retroperitoneal haemorrhage
3) Incorrect position
   Visceral ischemia,Aortic insufficiency
4) Aortic dissection ( <5%)-Usually retrograde often seals with own
5) Wound complication (1-3%)
6) Catheter failure (Gas escape)

**VADs** : When function of heart remain inefficient inspite of ballon, inotrops use of VAD may be considered

- ***VADs takes over the function of heart but ballon only optimize cardiac function***

# 9

## Ventricular Assist Devices ( VADs)

**VENTRICULAR ASSIST DEVICES (VADs) - TEMPORARY**
**Uses:**
**1)After Surgery**
**2)Bridge to transplant**
**3)Bridge to recovery**
**May be used with IABP**

FDA approves 5 devices :

| | | |
|---|---|---|
| 1.ABIO MED BVS 5000 | Pneumatic | Temporary |
| 2.Thoratec VAD(Rt/Lt) | ,, | ,, |
| 3.Thoratec Heart Mate (IP1000LVAD) | ,, | Parmanant |
| 4.Novacor | Electrical | ,, |
| 5. Thoratec Heart Mate (TCI LVAD) | ,, | ,, |
| All are pulsatile flow | | |

**VADs - Parmanant**
**Paracorporeal : Thoratec VAD ( PNEUMATIC)**

- **LV / RV or both**
- **Untill recovery LV**
- **Bridge to transplant.**
- **TO PLACE OVER ABDOMINAL WAL**

**VADs -Parmanant :**
**Implantable:**

- **Pneumatic Thoratec Heart mate (IP1000 LVAD)**
  - **long pipe line**
  - **out side dreiver console**
- **Electrical - Thoratec Heart mate (TCI SVE LVAD)**
  - **Novocor**

**Use: Normal Life style**
**Long wait**

A small percentage of patient undergoing Cardiac Surgery can not be winned from bypass despite of all efforts.Even they are refractory to IABP.Besides these patients with Low out put syndrome & dysarrythmias need VADs.

**Points of consideration :**

1. Age – Elderly with poor health are not good candidate for long support
2. Costly & time consuming
3. A perfusionist must be present for care
4. "Days" may be required before the patient is wined /transplanted

**Materials:**

Mostly Centrifugal pump is used for VADs. The pump works on the principle to propel blood in smooth nontraumatic manner for long support.

**Priming**: By using 3 liters of solution ensurng all air is expelled from the curcuit simultinously filling the tube & ragutating the pump by the perfusionist.

**Cannulation :** Is done by introducing through the LV apex,LA or retrograde accross aortic valve. **A venous single stage cannula is used.** The infusion cannula is directly placed into aorta. Allowing LV is bypassed .

There are other methods of priming also.

# 10

## Bi -Ventricular Assist Device (BIVADs)

This requires 2 pumps & circuits. In addition to LVAD,a right ventricular assist device (RVAD) is used.For RVAD inlet cannula is introduced in Right Atrium & the outlet cannula into Pulmonary Artery using aortic cannula. In RVF central Venous pressure is high & pulmonary wedge pressure is low.Truly RVAD is used with LVAD in most cases where VAD is required

**Action**

When the patient is taken-off from the bypass VADs are started slowly to 2.2 L/min/$m^2$. Venous drainage is to be watched.If difficulty is suspected the flow must be decreasd to avoid air entry.Fluid volume may be added to meet urgent drainage fall.

LA presssure measurement is necessary to monitor LVAD( 5~15 mmHg).Here also the risk of air embolism is present.

When biventrcular devices are used the LVAD is started first. Correct tunning makes the flow appropriate with LA Pr with in 5~15 mmHg.Through out the period ACTs are maintained 150~200 s.

**Ventilation**

- Pt. needs ventilation & $Po_2$ should be maintained >75 mmHg.
- When IABP is used, shoul be usually maintained.
- SVR should be in normal range (88~1200).
- Colloidal osmotic pressure to be maintained normally (25mmHg).This is important to asses the effect of huge dilution some time happens with VADs

**Weaning:**

To be attempted after the heart starts ejecting after a rest of 24 hours. Cardiac out put are assesed.Arterial pressure tracing dictates the ejection.If not satisfactory the return to pump is necessary for next trial after some time.

# 11

## ECMO : Extra-Corporeal Membrane Oxygenator

Principle is simillar to CPB is to remove $CO_2$ & to deliver $O_2$ by using oxygenation

Respiratory failure due to

- LV failure
- Lung trauma
- ARDS (increased capillary permeability)
- Infection

Can be managed by ECMO in adults.Although this is a time consuming & expensive attempt with poor result still some time gives excellent result.

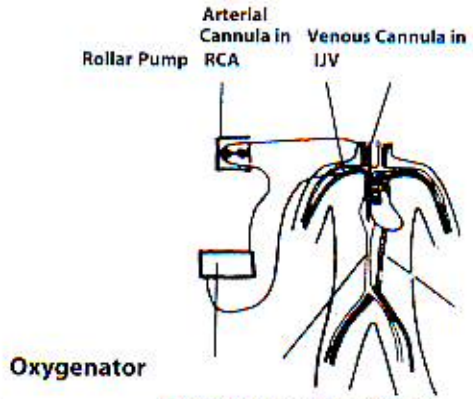
**Cannulation:**

2 way of Cannulation:

- Veno-arterial
- Veno-Veno

**Veno-arterial ( Partial CPB)ECMO:**

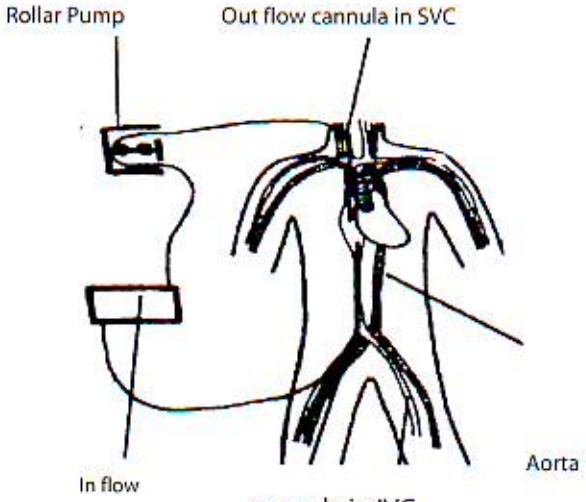
Cannulation of the RA through Right Internal jugular vein is done to drain blood to oxygenator.Oxygenated blood is delivered through the right common carotid artery.




VENO -ARTERIAL ECMO

## Veno-venous ECMO:

Venous blood is drained from the right Internal jugular vein(IJV).After oxygenation blood is returned to Right femoral vein




**NB. The heart must be ejecting to maintain circulation of the oxygenated blood as no cardiac out put support is provided in this way. Otherwise the blood will simply come back & forth to patient and the oxygenator**

**Tubing :**

Usually 3/8 inch is used for all except smaller patients.

**Hemoconcentration & Heparinization:**

May be done by adding another rollar pump in the circuit. ACT at the range of 180~220 s is used. Hematocrit. osmotic Pressure & fluid balance are very important to watch to avoid further damage to lung.

**Weaning:**

When the lung improves the flow rate will be decreased to 25% over several hours. If it is tolerate then the weaning will be attempted allowing the patients lung to supply oxygen to the tissue.

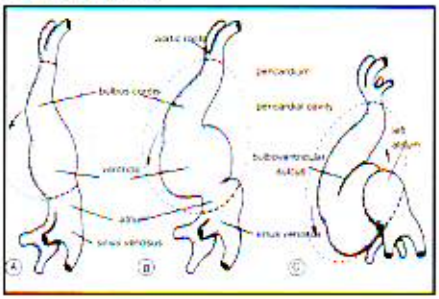
# COMMON
# CONGENITAL HEART DISEASES

# 12

## Congenital Heart Diseases

### DEVELOPMENT

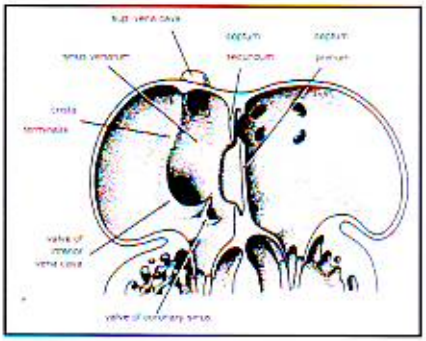
- Developing heart tube in splanchnic mesodermal pericardial cavity attached to the dorsal mesocardium
- Forms the bulboventricular portion
- Atria,Sinus venosus remains outside in septum transversum
- Next events => Rapid growth of bulboventricular portion than cavity



- Elongated tube forced to bend in cavity
- Cephalic part bends ventrally, caudally& to right
- A-V junction comes to left & dorsally
- Atria still paired & connected to ventricle through A-V Canal
- THE LOOP FORMS-Rt.ventricularOut flow tract(conus cordis,trancus)& Left ventricle
- Traco-conus shifts medially & atrium buldges both sides to Rt. & Lt atria(with oblique conus)

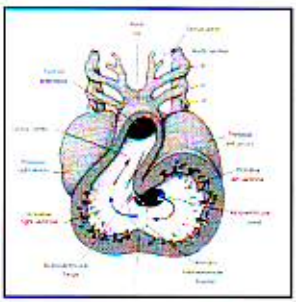
### Sinus venosus

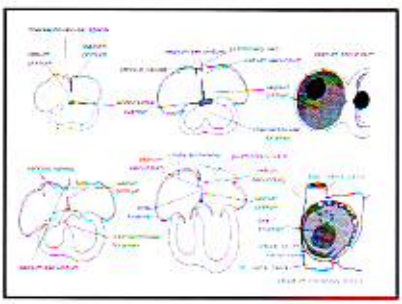
- PAIRED HORNS Left disappears to coronary sinus
- Right horn incorporated to atrium through right venous valve that gradually incorporated & forms valves of Inferior vena cava,CS



SEPTATION

- Trancus depresses the roof of atrium forms a crest that forms the septum primum grows to cushions => forms ostium primum
- Primary septum unites with cushion to form septum primum,meanwhile perforation appears => Ostium secundum
- Interseptovalvar space develops septum secundum => grows & overlaps osteum secundum



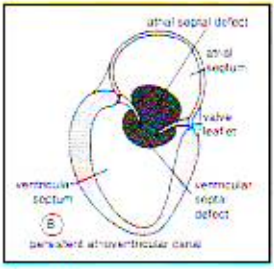


***ENDOCARDIAL CUSHION***

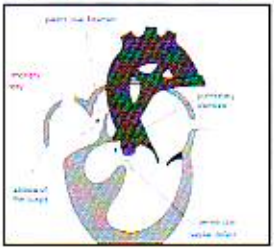
- Appears supro inferior boarder of AV canal
- At this stage conus tube separated from ventricle by a flange- bulboventricular flange
- At times this comes to mid point of supr. cushion
- With further growth the flange divides canal into rt.& lt. AV orifices

## ABNORMAL AV CANAL

- ***PERSISTANT AV CANAL : Primitive cannal persists***



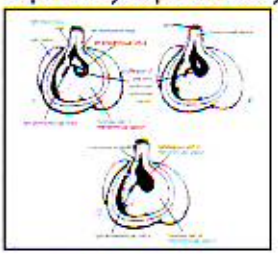

- ***TRICUSPID ATRESIA***



- **EBSTEIN'S ANOMALLY** - Apical displacement of septal cusp of TV

## Septation Ventricle

- Dilatation of primary ventricle,diverticulation with trabeculation => forms muscular septum inferiorly
- superiorly & posteriorly by apposition of flange



## SEPTATION OF CONUS

- Ridges appear inside truncus > Rt.sup & Lt inferior >divides in two channel with formation of aorticopulmonary septum.It twists while growing distally.

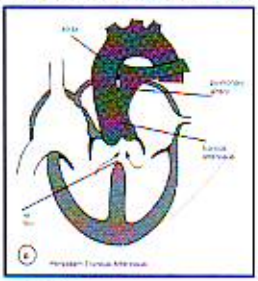
- Similarly two conus ridges divides the conus =>Proximal end of Rt.meets superior border of Rt.AV orifice => completes Rt.ventricle
- Lt conus swelling extend along right side of muscular septum.Conus septum thus forms divide conus antero-lateral & post ero lateral part
- Post-lateral part continuous with Lt.definite ventricle
- This reduces size of inter ventricular foramen
- Further closure done by growth of membrane from cushion to meet abutting edges of conus septum

**Abnormal IVS**

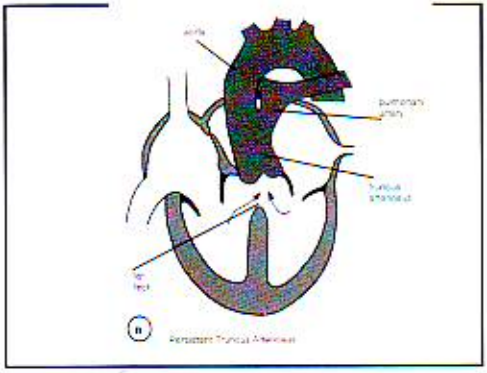
- SUPRA CRYSTAL VENTRICULAT SEPTAL DEFECT (VSD)

- **INFRA CRYSTAL VSD**
- **MASCULAR VSD**
- **SINGLE VENTRICLE**

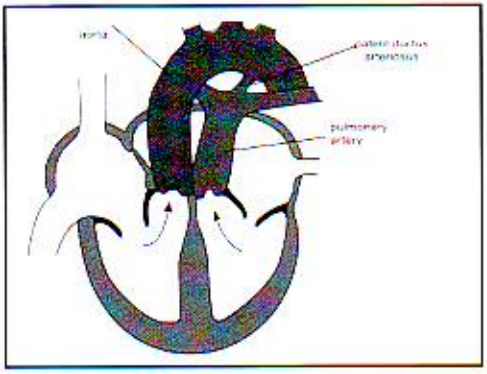
**ABNORMAL TRANCO-CONUS**



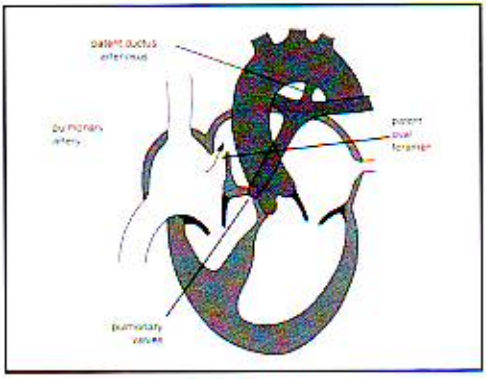
**TOF/DORV**



TGA



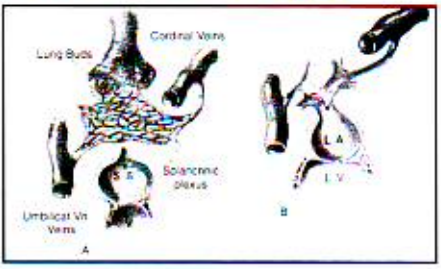
PTA



PS/PA

## PULMONARY VEINS (PVs)

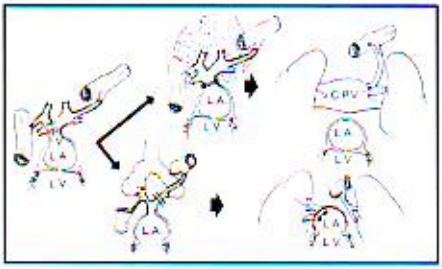
- Splanchnic plexus drains lung bud – shares connection with cardinal,umbilical vein
- Common pulmonary Vein invaginated from LA> joins the splanchnic plexus
- Pul. Vein drains to LA > primitive connections disappear
- With differential growth Pulmonary Veins are incorporated in LA & CPV disappear



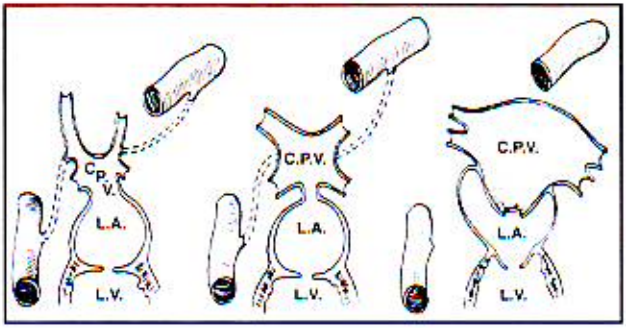

## ABNORMAL PV DEVELOPMENT

### TAPVC

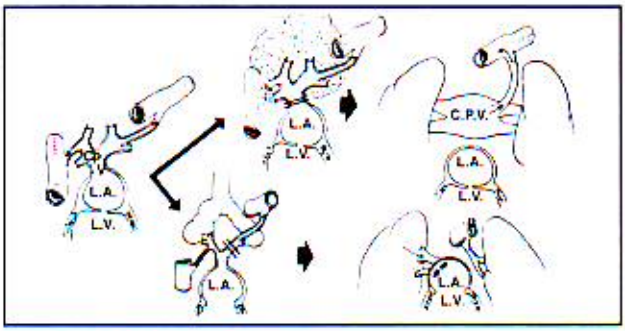

### PAPVD



**CPV >LA**




**Cor triatriatum > CPV**




**Cerebral & Spinal Protection in Aortic Surgery /CPB**

# 13

## Injury Protection

### Cerebral Damage

- Duration of circulatory arrest is a clear determinants of brain damage
- Cerebral oxygen consumption in hypothermic CPB at low flow reduced below normal-due to decreased density
- At 37°C it is same as before CPB
- Safe duration is affected by many known& unknown factors
- *Damage is rarely difuse in adult & manifested by intellectual / motor deficit.*
- *In neonats & children by seizures,choreoathetoid movements*

### Risk factors:

- Duration
- Temperature
- Rate of cooling & rewarming
- Flow & distribution
- BP
- Electrical activities
- Management of reperfusion

**Evident that circulatory arrest of 60 minutes more at 15°~18°C associated with irreversible damage although tolerated by some**

### Brain Injury: Aortic Surgery

- Temporary dysfunction- Agitation,Disorientation,Psycosis,Chorea,seizure
- Stroke

**Cerebral Protection**

Hypothrmic Circulatory arrest

- Simple & widely used
- Provides bloodless field
- Scrupulous apllication of technique is important to minimize complications(20%)
- For frequent cognitive dysfunction safe duration is close to 30min
- Adjunct(Retrograde cerebral perfusion) may increse safe duration.

***Retograde Brain Perfusion :***

- Meaningful metabolic benefit is debated.
- Plays Primary role as adjunct
- Bicaval cannulation is required
- Cooling at 18 °C
- Needs 3rd pump head.
- All retrograde cardioplagia & no clamping of Aorta
- Bypass line (3/8") from arterial to SVC
- Venous line to pump is clamped .Arterial line to Femoral Artery is clamped
- CVP should not be >30

**Antegrade perfusion:**

- Low flow hypothermic perfusion reserves pH & energy
- Direct cannulation of brachiocephalic arteries
- Used also retrogrde after arch replacement through a separate 10mm tube
- Flow 800~1200 ml/min at 20°C
- May not be safe more>80 min

## *Spinal cord Protection*

**Spinal Artery anatomy :**

- Variability of origin of Arteria Redicularis magna - $T_9$ ~ $T_{12}$ (75%)
- Clamping distal aorta is more safe

**Collaterals :**

- Disease where good collaterals are developed incidence is zero (e.g. in Coarctation) compare to degenerative diseases
- Incresed Intraspinal & Decreased aortic presure with clamp => reduces collateral flow

**Paraplegia/Paraparesis with/without bladder** is immediate or late result of unprotected cord during surgery for–

Distal arch

*Descending aorta*

Thoracoabdominal aorta

***Risk Factors :***

- Duration of clamping
  - No Probability when <15 min
- Temperature
  - Moderate to profound have lower incidence of injury than normothermia
  - 20°C allows clamping >100 min
  - Irigation of cold(4 °C )saline in epidural space for 24~28 °C CSF is adjunctive
- Level of clamp
  - Beyond renal arteries($L_2$) for 60 min incidence is less(0.1%)
  - At diaphragm for 60 min(10%)
  - Distal to subclavia for 60min incidence 80%
  - With increase in distance of 2 clamps number of intercostals exclude from spinal collaterals

**Protection of Cord :**

- Hypothermia & Low room temperature
- Cooling/Heating blanket (30~32 °C ) with additional cooling of pleural space for 15 min
- It is inadviseable to operate on descending & thoracoabdominal aorta with surface cooling
- Perfusion of Distal Aorta
- To maintain 60~70mmHg for spinal flow maintenance ( LA-FA bypass , Aorto-aortic shunt, Partial CPB )
- Grafting of Intercostal & Lumber arteries
- Most of the posterior wall to graft to the tube
- Favourably arteries below $T_6/T_7$

**THORACO ABDOMINAL REPLACEMENT**

- Combination Femoro-Femoral CPB, hypothermic arrest, grafting of intercostals,additional arterial cannula graft - **widely accepted**

**The End**

# 14

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***Product Manufacturer***

**********

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BARD CARDIOPULMONARY DIVISION
Baxter Healthcare corporation
**BENTLEY DIVISION**

**COBE Cardiovascular, Inc.**

**DATASCOPE CORPORATION**
**BIO – MEDICAL**

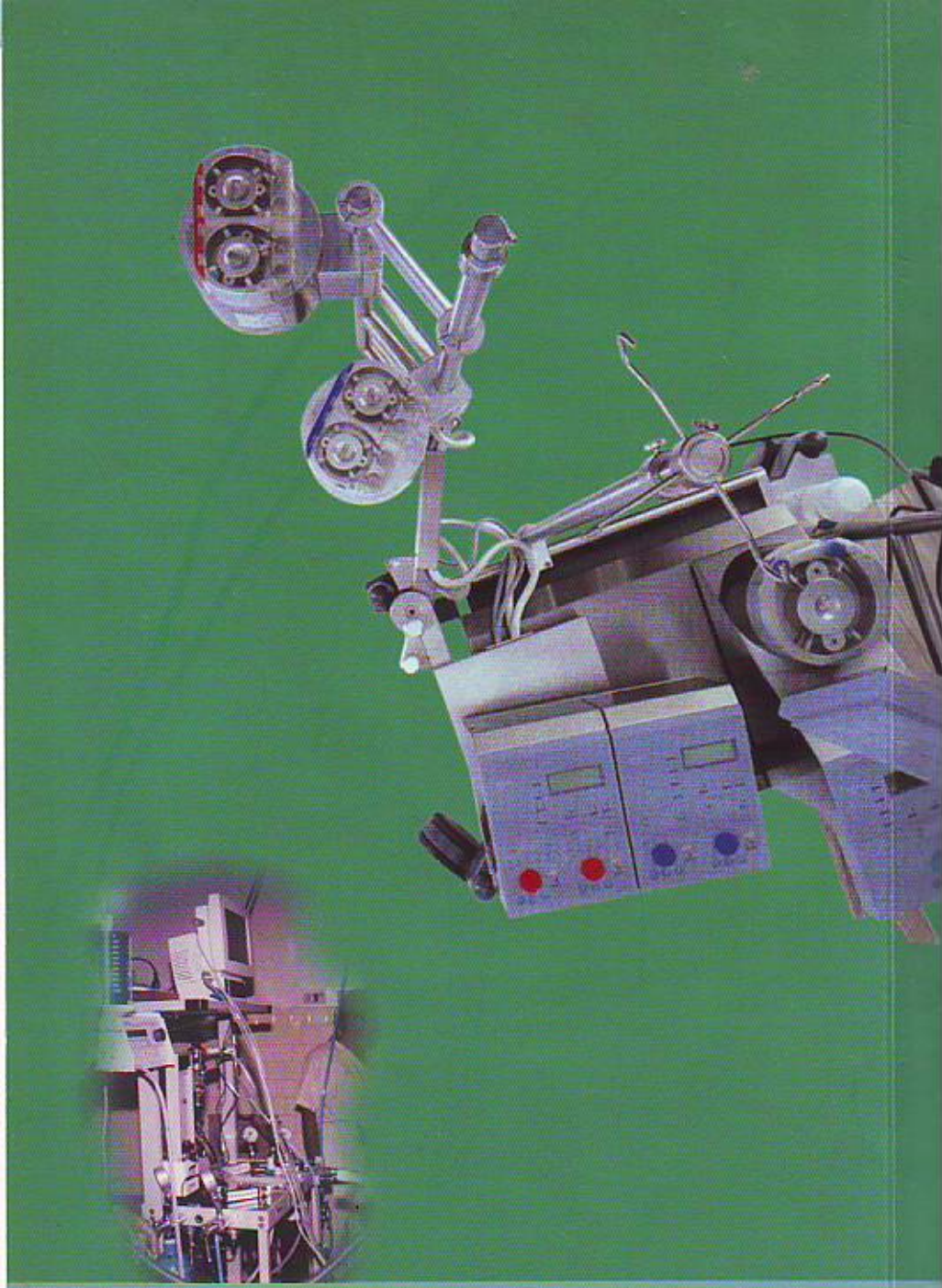
***Medtronic***
Cardiac Surgery

**MILES**
Pharmaceutical Division

**SORIN**

**BIOMEDICAL**

**3M Health Care**
**Cardiovascular Systems**



*To Encourage Understanding Medical Science*