The Royal Melbourne Hospital

Cardiopulmonary Bypass during Pregnancy

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Objective:

To understand the Cardiopulmonary Bypass (CPB) Strategy for the pregnant patient.

- Perfusion techniques unique to the pregnant patient.
- Physiological and Anticoagulation requirements.
- Fetal/Placental pharmacological consideration.

Background:

- First used during pregnancy in 1959 by Dobourg, Pulmonary Valvotomy and Atrial Septal Defect at 6 weeks of gestation, Mother survived, Fetus spontaneously aborted 3 months later.
- Heart disease is the leading cause of death in pregnancy, approximately 30% of all maternal deaths.
- Valvular disorders are the most common cardiac problem associated with pregnancy.
- Maternal mortality - 3%
- Fetal mortality - 25-50%

Pregnancy:

The pregnant patient requires special consideration by the perfusionist. The perfusionist not only manages the mother while on bypass, but also manages the unborn fetus. Pregnancy lasts about 38 weeks from conception to birth, although it may be 40 weeks from the first day of the last menstrual period. The placenta is formed from the parts of the woman and the embryo. The placenta is responsible for providing nutrients to the fetus and for removing waste products. The blood of the mother does not cross the placental barrier to mix with the that of the fetus, However. The mother experiences numerous physiologic changes during pregnancy. These changes often unmask pre-existing cardiac problems such as congenital defects.

Placenta: The Interface between Mother and Fetus

- Major source of Human chorionic gonadotropin (HCG).
- Secretes steroid hormones such as progesterone, lactogens and estrogens.
- Functions as a large arterio-venous fistula, which reduces the vascular resistance of the mother.
- Acts similarly to a true membrane oxygenator as the fetal and maternal blood do not mix, but gas exchange occurs.

Timing of Surgery:

When cardiac disease must be treated with surgical intervention, it should be undertaken prior to maximal maternal hemodynamic load and before cardiac decompensation occurs. Multiple authors have suggested surgical intervention as early in the second trimester as possible following the conclusion of first trimester organogenesis. Staging of such surgical intervention following these gestational events lessens the probability of spontaneous disruption of pregnancy in the first trimester as well as induction of premature labour with later intervention.

1ST TRIMESTER

- Organogenesis occurs
- If CPB is utilized during this period there is a risk of teratogenesis (formation of birth defects).
- Risk of teratogenesis from CPB is associated with hypoxia and hypoperfusion.

2ND TRIMESTER

- Ideal timing to perform surgery utilizing CPB
- Organogenesis has completed, eliminating risk of teratogenesis
- Less risk of premature labour vs. 3rd trimester

- Hypervolemia and anaemia less in mother vs. 3rd trimester
- Because of smaller fetal size there is less uterine blood flow and hemodynamic demands of fetus are less.

3RD TRIMESTER

- Increased risk of premature labour
- Physiological changes in mother are most profound
- During pregnancy uterine blood flow represents 10-15% of Cardiac output (CO) vs. 1% in nine pregnant patients.

Physiology & Hemodynamic:

Successful execution Of CPB during pregnancy requires acute awareness of the normal physiological and hemodynamic changes associated with pregnancy.

Blood Volume (BV)

- Beginning from 8th week of gestation, maternal BV expands in a linear progression to 30-50% above pre-pregnant levels by the 36th week.
- Erythrocyte volume is increased by 20-30% above normal
- Plasma volume (PV) increased by at least 50%
- Drop in Haematocrit (HCT) of about 7% (RBC volume doesn't increase as much as total volume)

Cardiac Output (CO)

- CO progressively increases from the beginning and plateaus around the 20th week.
- Peak CO of 35-45% above non pregnant resting output at about 20th week
- Such increases are common and result from increase in heart rate (10-12 beats/min) and stroke volume.
- Towards term, the enlarged uterus and fetal movement may disrupt venous return by compressing inferior vena cava resulting in reduction of CO.
- Caval compression is most pronounced in the supine position and least notable in the left lateral decubitus position.

Oxygen Consumption (VO2)

- Changes in CO are potentiated further by an increased oxygen demand with VO2 gradually increasing 15-18% by the end of 12th week.
- Values of arterial pCO2 decrease by approximately 10mmHg below normal with a coincidental decrease in bicarbonate, contributing to the maintenance of a normal pH.

Vascular Resistance (VR)

- Dilation of uterine blood vessels early in pregnancy results in decreased VR
- Decreased resistance is however, is frequently well compensated by an increased CO with mild decrease or no change in blood pressure (BP).
- Both preload and ventricular end diastolic volume (VEDV) are increased, Ventricular end diastolic pressure (VEDP) and Central venous pressure (CVP) remains unchanged attesting to enhanced cardiac compliance and a decreased VR.

Peripheral Edema

- Significant elevation in sodium levels contributes to increase in BV.
- Elevated sodium levels also contribute to increase in extracellular fluid
- Compounded by caval compression of the gravid uterus results in peripheral edema.

Uterine Blood Flow

- The prepregnant uterine blood flow is normally around 50ml/min and accelerates to term values of 500 to 1000ml/min.
- Accounts for 20% of CO and can be affected by maternal positioning as well as pharmacological intervention.

Hypercoagulable State

- Increases in clotting factors, particularly Factors VII, VIII, and XIII, contribute to a hypercoagulable state during pregnancy.
- Slight increase in platelet and a decrease in plasminogen activator levels results in a decrease in fibrinolysis, further contributing to the hypercoagulable state.
- Beneficial for the control of haemorrhaging in the normal postpartum course
- Not beneficial when extracorporeal intervention becomes necessary.

Perfusion Considerations:

Fetal Heart Rate Monitoring

Monitoring the fetal heart rate is essential during CPB in the pregnant patient, it provides the surgical team immediate information related to the physiological status of the fetus. Low blood flow rates during CPB result in diminished blood delivery to the fetus and may be associated with fetal distress. Fetal bradycardia may develop as a direct result. The normal fetal HR is between 120 and 160 bpm. All available reports have shown a direct relationship between fetal bradycardia and blood flow during CPB regardless of maternal acid-base status. During periods of increased distress, the fetus will compensate by autoregulating available blood flow to the brain, heart and adrenal glands and decrease VO2 by 50%. Insufficient fetal blood flow and hypoxia may result from insufficient uterine blood flow, insufficient umbilical blood flow, or a decrease in maternal arterial oxygen content, after ten minutes of oxygen deprivation fetal survival is unlikely, with lesser periods of hypoxemia associated with increased incidence of cerebral palsy or profound neurological sequelae. Fetal bradycardia may be favourably influenced by adjustment of CPB flow rates, further measures include modification of maternal position to eliminate umbilical cord compression, correction of maternal haemorrhaging quickly with fresh whole blood and minimising or eliminating use of vasopressors, increase maternal oxygen saturations during fetal hypoxia, correction of maternal acid-base deficits with sodium bicarbonate, and close monitoring and maintenance of maternal glucose levels with glucose infusions to replenish fetal glycogen stores lost during fetal hypoxia.

Priming Volumes

Since the pregnant patient presents with greatly expanded BV, a reduced pump prime is advised. Consideration should also be given to the selected pump circuit to accommodate anticipated volume, During CPB, the perfusionist should anticipate a decrease in predicted haemoglobin levels due to the disproportionate increase in plasma volume. The haematocrits for CPB in the pregnant patient should be maintained between 20-25%.

Perfusion Flow Rates

The pregnant patient's resting CO is increased by as much as 43% with co-existing decreased VR, therefore, a higher flow rate will be required to achieve adequate tissue perfusion and mean pressure, Pump flow rates should be calculated utilizing a cardiac index (CI) of 2.6-3.0 L/min/sqm with adjusted up or down in response to the fetal heart rate. A second method to

estimate pump flow is to use a CI of 2.4L/min/sqm and use pre-pregnant measurements to determine body surface area (BSA) and add 30-50% to the calculated flow. Regardless of the methodology selected to determine flow, a direct correlation should be noted between blood flow and fetal HR. Fetal arterial oxygen saturation varies between 52-65%. Despite of these low saturations, O2 supply is adequate when adequate blood flow is maintained.

Temperature Management

Multiple authors have reported fetal compromise through cardiac arrhythmias or fibrillation in association with exposure to moderate and deep hypothermia and they have concluded that fetal rewarming results in decreased fetal HR and induces uterine contractions and may precipitate premature labour. During CPB, maternal core temperature should start near normothermic levels and be allowed to drift slowly to 34*C. Maternal blood temperature should not be permitted to fall below 30*C.

Scavenging of Cardioplegic Solution

Regardless of the cardioplegic mixtures selected to accomplish maternal electro-mechanical cardiac standstill, clinicians must be conscious that potassium ions easily traverse the placental barrier and may induce depression of the fetal heart activity or cardiac arrest. Therefore, administration of high dose potassium cardioplegia may necessitate bi-caval venous cannulation followed by opening of the right atrium and scavenging of the cardioplegia from the coronary sinus. Retrograde cardioplegia can be used with venting of the aorta to a separate clamped out cardiotomy or waste bag. An ultrafiltrator or the cell washer may be used to avoid fetal hyperkalaemia.

Anticoagulation Management

Due to the elevated BV in pregnancy and a disproportionate PV increase, pregnant patients frequently present with an increased antithrombin III titre and require substantially higher than usual heparin doses. In addition, since heparin does not cross the placental barrier due its large molecular weight (MW =20,000), the fetus is not in jeopardy of intra-abdominal or intracranial bleeding seen with the heparinization of neonates. However, maternal heparinization is reported to carry increased risks of uterine haemorrhage and placental disruption. Therefore, strict heparin monitoring is required to avoid both subtherapic and excessive anticoagulation.

Cannulation

Unless critical emergency situations predominate, cannulation of the femoral artery and vein should be avoided due to the possibility the fetal and uterine position may obstruct the vena cava and cause reduced venous return. Additionally, femoral cannulation with non-physiologic retrograde arterial blood flow may result in hypoperfusion of uterine blood vessels, even in the presence of increased pump flows.

Positioning

To optimise venous return to the caval cannula, the patient should be placed in mild, left lateral position (right flank slightly raised - 15*) for surgery or transport. The supine position should always be avoided due to aortocaval compression. Weight of uterus on the vena cava can decrease venous return by as much as 20%, also weight of uterus of abdominal aorta can cause supine hypotensive syndrome.

Monitoring:

Mother

- Non-invasive Blood Pressure
- Invasive Arterial Blood Pressure
- End Tidal CO2
- Oximetry (Pulse and Cerebral)
- EKG

- Foley Temperature
- Oesophageal Temperature
- Pulmonary artery Catheter (swan ganz) SVO2
- TEE
- EEG or Bispectral Index (BIS)

Fetus

- Tocometer: uterine activity
- Fetal HR

Selection of Medications:

The pregnant patient has certain special responses to cardiovascular drugs. the five concerns are:

1. The effect on the uteroplacental blood flow.
2. The effect on the uterine muscle tone and labour.
3. The direct and indirect effects.
4. Undesirable maternal and fetal side effects.
5. Dilutional effects due to higher PV.

Cardiovascular Drugs for the Pregnant Patient

Diuretics

Furosemide

- Used for edema and hypertension.
- Crosses the placenta and may decrease placental perfusion.
- May prevent normal plasma volume expansion.

Thiazide

- May increase risk of congenital defects as a result of placental transfer.
- Induces electrolyte imbalance - hypoglycaemia, hyponatremia, hypokalaemia and thrombocytopenia.
- Secondary fetal bradycardia

Inotropic Agents

Digoxin

- crosses the placenta without causing fetal harm

- considered drug of choice for persistent fetal tachycardia

Dopamine

- no known adverse effects
- increases uterine blood flow and may stimulate uterine contractions.

Norepinephrine

- may cause constriction of uterine blood vessels and reduce blood flow.
- may stimulate uterine contraction.
- primarily alpha agonist.

Phenylephrine

- same as norepinephrine

Epinephrine

- crosses the placenta and may cause fetal tachycardia
- human teratogenic has not been shown.
- at low doses it has beta effect.
- vasopressor of choice.

Vasodilators

Hydralazine

- crosses the placenta
- in low doses will decrease BP in mother while increasing renal and uterine blood flow.
- no association seen with congenital defects.
- drug of choice for hypertensive crisis

Sodium Nitropruside

- contraindicated in pregnant patients because of the risk of cyanide toxicity to both mother and fetus.
- may shift blood flow away from the uterus.
- crosses the placenta

Nitroglycerin

- use of nitro-glycerine sublingually for angina without fetal harm has been shown.

Antiarrhythmic Agents

Quinidine

- not related to congenital defects

- crosses the placenta and related to fetal thrombocytopenia.
- used with digoxin to treat fetal supraventricular tachycardia.

Atropine

- rapidly crosses the placenta
- no evidence found for an association with fetal malformations.
- no significant changes were noted in fetal heart rate.
- no effect on uterine activity.

Beta-Blocking Agents

Propranolol

- readily crosses the placenta
- intrauterine growth retardation may be related to drug.
- fetal bradycardia and fetal toxicity found to suggest a relationship to malformations.
- lower umbilical and uterine blood flow.

Atenolol

- crosses the placenta with no fetal malformation noted.
- a decrease in fetal heart rate may be observed.

Calcium- Channel Blockers

Verapamil

- placenta passage was demonstrated with no relationship to congenital defects
- may reduce uterine blood flow with fetal hypoxia a potential risk.

Nifedipine

- no adverse effects in fetus observe

Antibiotics

Cefazolin

- cephalosporin antibiotic.
- readily crosses the placenta
- adverse effects have not been observed
- antibiotic of choice

Summary:

- Postpone surgery if possible

- Position patient with left uterine displacement (Right hip raised)
- Monitor uterine contractions
- Monitor fetal heart rate
- Prime circuit with bank blood if HCT <22-25%
- CPB flow 2.5-3.0 L/min.sqm.
- Maintain MAP>65mmHg.
- Stay normothermia or mild hypothermia(>32*C)
- Be prepared to administer progesterone and beta agonist.
- Scavenge cardioplegia (retrograde is best).
- Have obstetrician and neonatologist on stand-by in case a caesarean section is needed (gestational age >28wk).

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