Cardiopulmonary Bypass In Pregnancy: An Experience Of Three Different Clinical Scenarios

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Citation

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Abstract

Since foetal mortality is extremely high, open heart surgeries during pregnancy should be tried with extreme caution. Some deviation from routine cardiopulmonary bypass protocols like higher perfusion flow, pressure and arterial oxygen tension, use of Tocolytic agents might result in improved foetal survival though maternal survival seems to depend on cardiac disease per se than on cardiopulmonary bypass. Foetal bradycardia is the earliest indication of foetal distress and must be aggressively managed.

KEY MESSAGE

Cardiopulmonary bypass surgery during pregnancy is a high risk procedure. Some deviation from routine cardiopulmonary bypass protocols like higher perfusion flow and pressure and arterial oxygen tension, use of Tocolytic agents, aggressive management of foetal bradycardia might result in improved foetal survival.

INTRODUCTION

Cardio pulmonary bypass surgeries during pregnancy are very high risk surgeries which should be undertaken with extreme caution. Foetal mortality remains elevated as a consequence of CPB factors that stimulate uterine activity and contractions and reduce placental perfusion. Adjusting the perfusion flow, perfusion pressure and FiO_2 can contribute to improve placental blood flow and allow for better exchange with foetal blood. Tocolytic agents may improve placental perfusion[1].

The goal of this article is to high light the point that some deviation from routine cardiopulmonary bypass protocols might result in improved foetal survival though maternal survival seems to depend on cardiac disease per se than on cardiopulmonary bypass.

CASE HISTORY

Three cardiopulmonary bypass (CPB) surgeries during pregnancy occurred in our institute in 2006. Ages of patients at the time of surgery were 24, 28 and 30 years, gestational ages were 24, 25 and 32 weeks respectively. Indications of surgery were severe mitral stenosis with atrial clot, post BMV severe acute MR and heart failure secondary to severe mitral stenosis not amenable to medical treatment respectively. Two patients belonged to New York Heart Association Class III and one to class IV preoperatively, so were operated on.

While the patients were on 100% oxygen in supine position with wedge under right hip, monitors attached, wide bore intravenous access was secured. Under local anaesthesia arterial line and central venous line established, invasive blood pressure monitoring and CVP monitoring started. Preoperative vitals were systolic B.P. 90-100 mmHg, CVP 12-15 cm H_2O, heart rate 100-130 bpm, sPo_2 90-92. Foetal heart rate was monitored by obstetrician using Doppler monitoring.

General anaesthesia with endotracheal intubation achieved using intravenous inj.midazolam 0.1 mg/kg and inj.fentanyl 10 mcg/kg for induction, inj.pancuronium 0.1 mg/kg as muscle relaxant. NICO monitor was attached to ETT. Maintenance of anaesthesia was done with continuous intravenous infusion of midazolam, fentanyl and atracurium. We maintained ACT between 480 and 600 seconds by using 300U/Kg Heparin. We used mixed priming with blood and ringer's lactate, maintaining hematocrit at 30-35. Pump flow was between 3.6-4.9 l/min/m^2 sufficient to maintain a mean arterial pressure above 70 mmHg (70-90 mmHg). Inj.terbutaline and inj.magnesium sulphate were used to cease the uterine contractions .Surgeries were performed at mild hypothermia (34-36°C).. Duration of CPB were 55, 70

and 48 minutes with duration aortic cross clamp 35, 48 and 31 minutes respectively. Postoperatively all three patients were shifted ICU, and electively ventilated for 3, 4.5 and 4 hrs respectively.

Later, two patients delivered live neonates by elective caesarean section, while one is still carrying with normal pregnancy which is confirmed by ultrasonography. Birth weights of neonates were 2700 and 2850 gms. There were no congenital abnormalities. Patient data is shown below in table1.

Figure 1

Table 1: The characteristics of the patients and foetal outcome after open heart surgery.

Patient No.	Week of Pregnancy	Heart Operation	T (°C)	CPB time (clamp time)	Prime Quality	Pump Flow, L/min	MAP	Foetal Outcome	Maternal Outcome
1	28	MVR	34-36	55 (35)	Mixed	3.6-4.1	80-90	Good	Good
2	32	MVR	34-36	70 (48)	Mixed	4.3-4.9	80-90	Good	Good
3	24	MVR	34-36	48 (31)	Mixed	3.8-4.6	80-90	FHS Good	Good

DISCUSSION

Operative cardiac interventions have been performed on pregnant women with varying degrees of success since the late 1950s. But even after more than 50 years of scientific progress CPB during pregnancy is associated with a maternal mortality rate of 3% to 15% and a foetal mortality of 20% to 33%[2].

Because of the increase in cardiac output, red cell mass, and oxygen consumption during pregnancy, the standard approach to CPB (i.e., hemodiluted, nonpulsatile, and low flow) might be detrimental. Potential adverse effects of CPB include changes in coagulation, alterations in the function of cellular and protein components of the blood, release of vasoactive substances from leukocytes, complement activation, particulate and air embolism, nonpulsatile flow, hypothermia and hypotension[3,4,8].

Anaesthesia can affect pregnancy adversely. Mechanical hyperventilation results in a decrease in uterine blood flow of 25% during hypocarbia. These adverse effect on uterine blood flow was also attributed to the mechanical effects of positive-pressure ventilation - that is, a decrease in venous return and cardiac output [3,5,6]. Though most anaesthetic agents, intravenous and inhalational, and paralyzing drugs are devoid of teratogenic effects, doubt exists[7]. It is important to understand that most of the factors which affect the foetus during CPB do not act directly on the foetus. Their influence is exerted on the placenta and the uterus. Only a few factors such as hyperkalemia may directly affect the foetal myocardium. Hypothermia produces acid-base changes, dysrrythmias and uterine contractions[8,9,10]. Uterine contractions occur frequently during cardiopulmonary bypass, assumed to result from dilution of progesterone and other gestational hormones, considered to be the most important predictor of foetal death[10].

The most common foetal reaction is bradycardia, which very often occurs after a few minutes of CPB. Most of times the bradycardia is an indication of foetal distress and should be criteriously identified and managed. The transition from pulsatile to linear flow of the arterial pump, the lower mean arterial pressure, and hemodilution, at the beginning of CPB occasionally associated with uterine contractions or increased tonus, can all contribute to reduce the placental blood flow [10]. Both, increasing perfusion flow and increasing arterial pO_2 favour oxygen exchange at the placental interface.

Cardiotocography, Ultrasonography and Doppler Monitoring are few widely used techniques to monitor foetal rate. We used Doppler monitor to measure foetal heart rate.

Pump flow was between 3.6-4.9 l/min/m^2, sufficient to maintain a mean arterial pressure above 70 mmHg, with FiO_2 0.6 achieving arterial pO2 200-300 mmHg, although a few authors have suggested higher levels [1, 7]. However, the best demonstration of an adequate pump flow and arterial pressure is the foetal response to CPB. Foetal bradycardia is an indication to elevate perfusion flow and pressure in order to increase placental blood flow. We conclude all these seemingly little modifications can make significant change in the eventual out come.

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