Cardiopulmonary Bypass During Pregnancy: A Case Report

Robin G. Sutton and James P. Dearing
Medical University of South Carolina
Charleston, South Carolina

Keywords: case report; pregnancy; technique, cardiopulmonary bypass

Abstract

(J. Extra-Corpor. Technol 20(2):67–71, 39 references) **Reported cases of the pregnant patient during cardiopulmonary bypass are rare but not unique. This case report of the pregnant patient during cardiopulmonary bypass is unique for two reasons. First, the patient was also a Jehovah's Witness and refused blood products. Secondly, this case report provides a literature review and specific cardiopulmonary bypass considerations pertinent to the perfusionist.**

Recent literature reports that heart surgery is relatively safe during pregnancy. It is important to monitor fetal heart rate during the procedure not only to determine fetal distress but also to further knowledge in the area by documenting effects. The perfusion circuit must be prepared for the increased blood volume of the patient and still maintain minimal priming volumes.

Uterine blood flow is not autoregulated so it is important to maintain adequate cardiac output or blood flow and perfusion pressure during the operation. A cardiac index of 3 liters per minute per meter squared and mean blood pressure of 60 mmHg during our case gave us no signs of fetal distress. The pregnant patient should be maintained at normothermia and bypass times should be kept to a minimum.

Introduction

Cases involving cardiopulmonary bypass during pregnancy are still relatively few. In 1983, a survey of members of the Society of Thoracic Surgeons by Dr. Ronald Becker[1] showed 20% had had some experience with cardiac operations in pregnant women. 11% of the surveys returned included the use of cardiopulmonary bypass.

Our recent experience at the Medical University of South Carolina with a 21-year-old woman, with congenital aortic stenosis, who was in her second trimester of pregnancy led us to this investigation of the literature.

Normal Pregnancy

Even in normal pregnancy there is an increased stress on the heart. Starting from the 8th week of gestation, the blood volume begins to expand. By the 36th week, blood volume is increased to 35 percent above the non-pregnant level. The plasma volume is increased by 40 percent whereas the red blood cell volume has only increased by 20 percent.[2] By the end of the first trimester, the cardiac output has increased by 30–40 percent. This changes insignificantly later in the pregnancy.[3,4,5] Oxygen consumption during pregnancy increases by 15 to 18 percent.[6] These changes are attributed to the low resistance shunt created by the placenta, water and sodium retention, renin and prolactin secretion, and changes in plasma hormone levels.[7]

History

Cardiac surgery performed during pregnancy was reported as early as 1953. Brock,[8] Cooley and Chapman,[9] Logan and Turner,[10] and Mason[11] reported one maternal death and one premature delivery in a total of 11 closed mitral commissurotomies.

The first use of extracorporeal circulation in pregnancy was in 1959.[12] A pulmonary commissurotomy and an atrial septal defect repair were performed at 6 weeks gestation. Although the patient survived, she had a spontaneous abortion 3 months later.

In 1969, Ralph S. Zitnik et al.[13] conducted a random survey of 20 cardiovascular surgeons. Of the patients they reported, the average maternal age was 31 years and the average gestation at the time of surgery was 12 weeks. The maternal mortality was 5 percent and the

Direct communications to: Robin G. Sutton, 1173 Island View Drive, Mt. Pleasant, SC 29464.

fetal mortality was 33 percent. Zitnik states that only one fetal death occurred during the immediate post-operative period, but that cardiac surgery could not be ruled out as contributor to the other fetal deaths.

Statistics for survival improved in a survey conducted in 1983 by Becker[1] which showed a 1 percent maternal mortality and 16 percent fetal death. In 1986, a survey by Bernal et al.[14] found a fetal mortality of 9.5 percent compared to 1.6 percent fetal mortality in pregnant women without signs of cardiac disease.

Heart Disease During Pregnancy

The incidence of heart disease during pregnancy is 1–2 percent.[15] Most women with cardiac disease do not require surgery during pregnancy. They can be managed medically until the pregnancy is concluded.[16]

Heart disease is the number one cause of maternal death; it accounts for 10% of all maternal deaths.[17] The majority of cases are due to mitral stenosis caused from rheumatic fever incurred earlier in life, but some are congenital defects which only become apparent during the increased cardiovascular stress of pregnancy. A patient with mitral stenosis may be compensating well before pregnancy; but with increases in cardiac output brought on by pregnancy, the mitral valve gradient will increase. The resulting increase in left atrial, pulmonary venous and pulmonary capillary wedge pressure can cause atrial fibrillation or pulmonary congestion. Mitral regurgitation and aortic insufficiency are better tolerated because of the fall in systemic vascular resistance in pregnancy.[7]

Mild to moderate aortic stenosis is also well tolerated during pregnancy, but in severe aortic stenosis where the cardiac output cannot compensate for any reduction in preload, possible complications include cardiac and cerebral ischemia and a decrease in blood flow to the uterus.[7] Reported maternal mortality in these cases has been as high as 17 percent and fetal mortality as high as 32 percent.[18]

Women with congenital heart defects have a 10 times greater chance of having a child with congenital heart disease. This may be hereditary or due to poor fetal development secondary to the hemodynamic status of the mother.[16]

The most common congenital defects discovered during pregnancy are atrial septal defects and patent ductus arteriosus.[19] Both are well tolerated during pregnancy. Ventricular septal defects are not commonly seen during pregnancy because the loud murmur causes it to be noticed and repaired early in life. If not repaired before pregnancy occurs, ventricular septal defects are well tolerated if the shunt is small. Large shunts can cause pulmonary edema during pregnancy.

The bicuspid aortic valve is the most common form of congenital valvular aortic stenosis.[7] As in the acquired disease, unless the stenosis is severe, it is well tolerated during pregnancy.

Coarctation of the aorta, although usually found in males, is especially dangerous for the pregnant woman. When not repaired before pregnancy, it can be a great risk to the patient during pregnancy. Mortality during pregnancy due to such factors as aortic rupture, cerebral vascular accident due to rupture of a berry aneurysm in the Circle of Willis and congestive heart failure is 3.5 percent.[20]

Uncorrected right to left shunts such as in Tetralogy of Fallot are generally not well tolerated during pregnancy.[7] The decrease in systemic vascular resistance causes an increase in the right to left shunt.

Pregnancy in patients with Eisenmenger's Complex, which is an elevated pulmonary vascular resistance in a patient with a previous left to right shunt, is poorly tolerated with a maternal mortality of 30 to 70 percent.[7]

Severe pulmonary artery hypertension has a maternal mortality of 50 percent.[7] Even when the mother survives, fetal mortality is 40 percent.[21] Surgery is only attempted when the mother's life is threatened and the risk of surgery is outweighed by such complications as pulmonary edema, congestive heart failure, or bacterial endocarditis.

Effects of Cardiopulmonary Bypass on the Maternal Fetal Unit

The surgeon must determine the risk to the patient and fetus with or without surgical treatment. If it can be determined that surgery will be required at some point during the pregnancy, it is best that it be performed early in the second trimester.[1,4,16,17,22] The rationale is to avoid the potential for teratogenesis or fetal deformity in the first trimester, when the internal organs of the fetus are being formed, as well as the risk of additional hemodynamic stress and premature labor later in pregnancy.[22]

Although there has been no correlation reported between fetal death or deformity and the length of gestation at the time of surgery, a survey by Jose Bernal et al.[14] in 1986 found of 45 cases, in the 3 infants born with congenital malformations, the mother had been placed on cardiopulmonary bypass during the first trimester of pregnancy. On the other hand, 6 patients reported in Becker's study[1] underwent cardiopulmonary bypass in the first trimester without knowledge of their pregnancy and thus no special precautions were taken. All 6 pregnancies in that study resulted in normal deliveries.

Cardiac outputs can be decreased up to 20 percent in the pregnant patient when they are in the supine position.[23,24] This is especially seen later in pregnancy when the venous return can be decreased due to compression of the inferior vena cava by the uterus. Positioning of the patient in the left lateral position will relieve this

compression. Since this may not be feasible during cardiac surgery, a right lateral pelvic tilt may suffice.[1] Positioning of the pregnant patient during cardiopulmonary bypass may be important where cardiac output is relatively controlled and obstruction of the inferior vena cava either by cannulation or patient position can have harmful effects on the placenta.[22,25] Experiments by Howard and Goodson[26] demonstrate the extreme case where inferior vena cava obstruction in dogs at term produced abruptio placenta, which is a tearing of the placenta from the uterus.

The use of fetal heart rate monitoring during cardiopulmonary bypass was first reported by Koh et al.[25] in 1975. This is usually accomplished by ultrasound. But phonocardiography or electrocardiography can also be employed. Normal fetal heart rate is 120 to 160 beats per minute but heart rates as low as 100 are not of significant concern.[19] Fetal hypoxia is a major cause of fetal bradycardia.[19] All cases reported in the literature had fetal bradycardia at the initiation of cardiopulmonary bypass.[25,27,28,29,30] This was attributed to one or more of the following: loss of maternal pulsatile blood flow, decreases in cardiac output from prebypass, decreases in maternal blood pressure, hypothermia, and particulate or bubble emboli interfering with uterine blood flow. No reported cases employed the use of pulsatile blood flow during cardiopulmonary bypass. In some cases, the fetal bradycardia was alleviated by increases in blood flow or maternal blood pressure. Werch and Lambert[28] reported a direct correlation between maternal blood flow and fetal heart rate when blood flows were decreased by 50 percent. Brinkman and Woods[31] studied uterine blood flow in the nonpregnant ewe and found it to be 2 percent of the cardiac output, during pregnancy this increased to 20 to 30 percent of the cardiac output, demonstrating the increased demands of the placenta and fetus. Since uterine blood flow is not autoregulated, it would seem advantageous to the fetus to employ blood flows higher than routine during cardiopulmonary bypass.

A transient fetal tachycardia is often reported postbypass. This may be a result of an oxygen debt incurred by the fetus.[25]

If the pregnant patient is cooled, the same cold blood which may cause her to fibrillate can also cause the fetus to fibrillate. This complication has not been reported probably because the cases reported utilized normothermia or mild hypothermia. Normothermia should be employed unless cross clamp time is expected to be long.

Drug Administration During Pregnancy

When drugs are given to the pregnant woman, concern is given to their effects on uteroplacental blood flow as well as the teratogenic properties. Reduction in uteroplacental blood flow is the most significant contributor to fetal and neonatal morbidity and mortality. Drugs should be given only if the potential benefit justifies any potential risk to the mother or fetus.

Epinephrine, a sympathomimetic drug, appears to be the vasopressor of choice during pregnancy, since at low doses it has a primary beta stimulatory effect.[31,32] Because the uterine vasculature is primarily under alpha control, alpha adrenergic agents such as norepinephrine and phenylephrine cause decreases in uteroplacental blood flow and should not be used during pregnancy.[31]

Hydralzine is the antihypertensive drug of choice during pregnancy. In low doses it will decrease the blood pressure while increasing renal and uterine blood flow, though higher doses have been shown to decrease uterine blood flow.[31] Antihypertensive drugs must be administered carefully during pregnancy because of the dependency of uteroplacental blood flow on maternal blood pressure.

Sodium nitroprusside carries the risk of cyanide toxicity to both fetus and mother.[34] By decreasing the peripheral vascular resistance it can cause blood flow to be shunted away from the uterus. Studies show that its use in an acute hypertensive situation during pregnancy causes no deleterious fetal effects.[35,36]

Oral anticoagulants such as coumadin are known to cross the placental barrier. The teratogenic effects and the risk of fetal hemorrhage with oral anticoagulant use are well documented.[37]

Heparin because of its large molecular weight does not cross the placental barrier.[16] Heparin still carries the risk of uterine hemorrhage.[38,39] No deleterious effects have been documented with the use of relatively large doses of heparin given during cardiopulmonary bypass. During pregnancy concentrations of coagulation factors such as fibrinogen, prothrombin, factor VIII, and factor X are increased.[19](p. 388) There is no evidence that this increase in coagulation factors is responsible for the hypercoagulability seen in pregnancy.[19](p. 337) The concentration of antithrombin III is also increased during pregnancy. It could be considered that with increases in clotting factors, antithrombin III and plasma volume that potentially there could be a change (increase or decrease) in a pregnant patient's response to heparin. Neither is documented.

Monitoring of uterine contractions is advantageous during cardiac surgery. Contractions may be caused by rewarming or may be a side effect of medications given to the patient.

Case Study

The patient was a 21-year-old white female, 16 weeks pregnant, who presented with persistent fatigue, palpitations, dizziness and chest pain. A heart murmur had been discovered at birth and she was told 3 years prior

to this admission, during her first pregnancy, that she had mild aortic stenosis. Few cardiac symptoms were noted until this second pregnancy.

She underwent cardiac catheterization which revealed an 80 to 100 mmHg aortic gradient, consistent with severe aortic stenosis.

Because of her symptoms, it was felt that she should undergo aortic valvotomy or possibly aortic valve replacement. An additional complication was that the patient was a Jehovah's Witness and consequently did not wish blood products to be utilized unless absolutely necessary.

Seven days after cardiac catheterization, the patient was taken to the operating room for repair or replacement of her aortic valve.

There were several perfusion protocol changes made for this case. Because of the patient's increase in blood volume due to her pregnancy and valve disease, our normal blood volume calculation of 65 ml of blood per kilogram body weight was changed to 100 ml per kilogram.

To keep hemodilution to a minimum, we utilized a Shiley M2000 Membrane oxygenator[a] set up without a reservoir bag and a Travenol 5M1471 filtered cardiotomy reservoir.[b] Minimal level in the cardiotomy reservoir prebypass allowed for a priming volume of 1800 ml as well as an adequate reserve for the patient's increased blood volume. The system was primed with 1300 ml lactated Ringers[b] and 500 ml 6 percent hetastarch.[c]

Ultrafiltration using a Cordis Dow C-DAK 135 dialyzer[d] was set up parallel to the bypass circuit to aid in handling the large blood volume as well as for hemoconcentration during and after cardiopulmonary bypass.

Fetal heart rate was monitored by doppler and a baseline fetal heart rate of 140 beats per minute was obtained.

Heparin was given at 300 units at per kilogram of body weight and five minutes later and hand tilted activated clotting time was 510 seconds.

The ascending aorta was cannulated and a dual stage venous cannula was inserted though the right atrium. The patient was placed on cardiopulmonary bypass at a blood flow of 3 liters per minute per meter squared of body surface area. Her mean perfusion pressure without drug intervention stayed at 55 to 65 mmHg. Prebypass her mean blood pressure was 80 mmHg. Fetal heart rate fell to 120 beats per minute, which is still within normal limits. The patient's esophageal temperature was maintained at 35 degrees centigrade. Venous oxygen saturations, monitored with a Bentley Oxysat Meter[e] stayed at 70 to 75 percent throughout cardiopulmonary bypass. The aorta was cross clamped and 700 ml of crystalloid potassium cardioplegia was infused through the aortic root. An aortotomy was performed. The aortic valve was found to be bicuspid and an aortic valvotomy was performed. The aortotomy was closed and air evacuated from the heart. Aortic cross clamp time was 18 minutes and the heart was defibrillated with one countershock.

During this time the patient was warmed to 36.5 degrees esophageal temperature. The hematocrit fell to 15 percent during bypass after the administration of cardioplegia and since the bypass time was only 22 minutes, it did not allow time to ultrafilter a significant amount of plasma water during bypass.

The patient came off bypass without difficulty and within a short period of time the fetal heart rate returned to baseline. The patient's aortic valve gradient was measured at 30 mmHg. This was considered to be satisfactory since her cardiac output at the time of measurement was 9 to 12 liters per minute. It was felt that this would allow her to complete this pregnancy and when her hemodynamic status returned to normal post partum, the gradient would decrease.

The mother and fetus tolerated the operation well. After surgery, the patient was admitted to surgical intensive care. She was extubated and transferred to the post surgical floor on the first post-operative day.

She was discharged on the sixth post-operative day with a hematocrit of 20 percent. No blood products were utilized during her hospital stay.

Four and a half months later, she delivered a healthy baby at the Charleston Naval Hospital.

Discussion

A woman with asymptomatic heart disease may become symptomatic with the increased cardiovascular stress of pregnancy. If the patient warrants heart surgery at this time, the perfusionist must understand the unique considerations of this patient in order to provide safe cardiopulmonary bypass for both mother and fetus. Patient blood volume, drug administration, cardiac index, patient temperature, and fetal monitoring are all areas which should be of concern to the perfusionist.

The literature search on this case report was accomplished using the Index Medicus and a Medline search with the keywords cardiac surgery during pregnancy.

References

1. Becker, R.M.: Intracardiac Surgery in Pregnant Women. *Ann. Thorac. Surg.* 36(4): 453–458, 1983.
2. Lund, C.J., Donovan, J.C.: Blood Volume during Pregnancy. *Am. J. Obstet. Gynecol.* 98:393–403, 1967.
3. Lees, M.M., Taylor, S.H., Scott, D.B., Kerr, M.G.: A Study of Cardiac Output at Rest Throughout Pregnancy. *J. Obstet. Gynaecol. Br. Commonw.* 74:319–328, 1967.

a Shiley, Inc., Irvine, CA 92714
b Travenol Laboratories, Deerfield, IL 60015
c American Critical Care, McGaw Park, IL 60085
d C. C. Medical, Miami Lakes, FL 33014
e America Bentley, Inc., Irvine, CA 92714

4. Metcalfe, J., Ueland, K.: Maternal Cardiovascular Adjustments to Pregnancy. *Prog. Cardiovasc. Dis.* 16: 363–374, 1974.
5. Rovinsky, J.J., Jaffin, H.: Cardiovascular Hemodynamics in Pregnancy. *Am. J. Obstet. Gynecol.* 95: 787–794, 1966.
6. Ueland, K., Hansen, J.M.: Maternal Cardiovascular Dynamics III Labor and Delivery under Local and Caudal Analgesia. *Am. J. Obstet. Gynecol.* 103: 8–18, 1969.
7. Lang, R.M., Borrow, K.M.: Pregnancy and Heart Disease. *Clinics in Perinatal.* 12(3): 551–569, 1985.
8. Brock, R.C.: Valvotomy in Pregnancy. *Proc. Roy. Soc. Med.* 45: 538–540, 1952.
9. Cooley, D.A., Chapman, D.W.: Mitral Commissurotomy During Pregnancy. *J.A.M.A.* 150: 1113–1114, 1952.
10. Logan, A., Turner, R.: Mitral Valvotomy in Pregnancy. *Lancet* 1(26): 1286, 1952.
11. Mason, J.: (In discussion of Stabler, F.E. and Szekely, P.J.) Cardiac Disease in Pregnancy. *J. Obst. Gynaec, Brit. Emp.* 59: 569, 1952.
12. Dubourg, G., Broustet, H., Bricaud, H.: Correction of a Triad of Fallot with Extracorporeal Circulation in a Pregnant Woman. *Arch. Mal. Coeur. Vaiss.* 52: 1389–1391, 1959.
13. Zitnik, R.S., Brandenburg, R.O., Sheldon, R., Wallace, R.B.: Pregnancy and Open-Heart Surgery. *Circulation* 39 (Suppl-1): 257–262, 1969.
14. Bernal, J.M., Miralles, P.J.: Open-heart Surgery and Pregnancy. *Obstet. Gynecol. Surv.* 41 (1): 1–6, 1986.
15. Szekely, P., Snaith, L.: *Heart Disease and Pregnancy.* London Edinburgh: Churchill-Livingstone, 1974, pp. 86–90.
16. Ueland, K.: Cardiac Surgery and Pregnancy. *Am. J. Obst. Gynec.* 92(1): 148–162, 1965.
17. Hibbard, L.T.: Maternal Mortality due to Cardiac Disease. *Clin. Obstet Gynecol.* 18(3): 27–36, 1975.
18. Artal, R., Pineda, J.: Aortic Stenosis in Pregnancy. *J. Reprod. Med.* 20: 229–232, 1970.
19. Metcalfe, J., McAnulty, J.H., Ueland, K.: *Burwell and Metcalfe's Heart Disease and Pregnancy: Physiology and Management.* Boston/Toronto: Little, Brown and Co., 1986, pp. 223–224.
20. Deal, K., Wolley, C.: Coarctation of the Aorta and Pregnancy. *Ann. Intern. Med.* 78: 706–710, 1973.
21. McGaffrey, R.N., Dunn, L.J.: Primary Pulmonary Hypertension in Pregnancy. *Obstet. Gynecol. Surv.* 19: 567–591, 1964.
22. Meffert, W.G., Stansel, H.G.: Open Heart Surgery During Pregnancy. *Am. J. Obstet. Gynecol.* 102(8): 1116–1120, 1968.
23. Ueland, K., Novy, M.J., Peterson, E.N., Metcalfe, J.: Maternal Cardiovascular Dynamics 1V: The Influence of Gestational Age On the Maternal Cardiovascular Response to Posture and Exercise. *Am. J. Obstet. Gynecol.* 104: 856–864, 1969.
24. Quilligam, E.J., Tyler, C.: Postural Effects on the Cardivascular Status in Pregnancy: A Comparison of the Lateral and Supine Postures. *Am. J. Obstet. Gynecol.* 130: 194–198, 1978.
25. Koh, K.S., Friesen, R.M., Livingstone, R.A., Peddle, L.J.: Fetal Monitoring During Maternal Cardiac Surgery with Cardiopulmonary Bypass. *Can. Med. Assoc. J.* 112: 1102–1104, 1975.
26. Howard, B.K., Goodson, J.H.: Experimental Placental Abruption. *Obstet. Gynecol.* 2: 442–446, 1953.
27. Levy, D.L., Warriner, R.A., Burgess, G.E.: Fetal Response to Cardiopulmonary Bypass. *Obstet. Gynecol.* 56(1): 112–115, 1980.
28. Werch, A., Lambert, H.M.: Fetal Monitoring and Maternal Open Heart Surgery. *South. Med. J.* 70:1024, 1977.
29. Katz, J., Hook, R., Barash, P.: Fetal Heart Rate Monitoring in Pregnant Patients Undergoing Surgery. *Am. J. Obstet. Gynecol.* 125: 267–269, 1976.
30. Trimakos, A.P., Maxwell, K.D., Berkay, S., Gardner, T.J., Achuff, S.C., Fetal Monitoring During Cardiopulmonary Bypass for Removal of a Left Atrial Myxoma During Pregnancy. *Johns Hopkins Med. J.* 144: 156–160, 1979.
31. Brinkman, C.R. III, Woods, J.R.: Effects of Cardiovascular Drugs During Pregnancy. *Cardiovasc. Med.* 1: 231–251, 1976.
32. Pedersen, H., Mieczyslaw, F.: Anesthetic Risk in the Pregnant Surgical Patient. *Anesthesiology.* 51: 439–451, 1979.
33. Eilen, B., Kaiser, I.H., Becker, R.M., Cohen, M.N.: Aortic Valve Replacement in the Third Trimester of Pregnancy: Case Report and Review of the Literature. *Obstet. Gynecol.* 57: 119–121, 1981.
34. Rigg, D., McDonogh, A.: Use of Sodium Nitroprusside for Deliberate Hypotension during Pregnancy. *Br. J. Anaesth.* 53: 985–987, 1981.
35. Ellis, S.C., Wheeler, A.S., James, F.M. III, Rose, J.C., Meis, P.J., Shihabi, Z., Greiss, F.C. Jr., Urban, R.B.: Fetal and Maternal Effect of Sodium Nitroprusside Use to Counteract Hypertension in Gravid Ewes. *Am. J. Obstet. Gynecol.* 143: 766–770, 1982.
36. Stempel, J.E., O'Grady, J.P., Morton, M.J., Johnson, K.A.: Use of Sodium Nitroprusside in Complications of Gestational Hypertension. *Obstet. Gynecol.* 60: 533–538, 1982.
37. Stevenson, R.E., Burton, D.M., Ferlauto, G.J., Taylor, H.A.: Hazards of Oral Anticoagulants During Pregnancy. *J.A.M.A.* 243: 1549–1551, 1980.
38. Hall, J.G., Paul, R.M., Wilson, K.M.: Maternal and Fetal Sequelae of Anticoagulation During Pregnancy. *Am. J. Med.* 68: 122–140, 1980.
39. Mahairas, G.H., Weingold, A.B.: Fetal Hazard With Anticoagulant Therapy. *Am. J. Obstet. Gynecol.* 85: 234–237, 1963.