Management of anesthesia for open heart surgery during pregnancy

F. George Estafanous, M.D.
Department of Cardiothoracic Anesthesia

S. Buckley, M.D.
Department of General Anesthesia

Rheumatic heart disease, particularly mitral stenosis, is the principal type of heart disease during pregnancy,[1,2] and the leading, indirect cause of maternal mortality in the United States.[3]

During pregnancy blood volume and cardiac output are increased 30% to 50% and oxygen consumption is increased 20%. These changes make patients with mitral stenosis more vulnerable to acute heart failure and emergency heart surgery is often indicated.

Mitral commissurotomy was first performed on the pregnant patient in 1952,[1] and in 1958 Leyse et al[4] reported the first use of cardiopulmonary bypass during pregnancy. In 1967, Harthorne et al[3] reported the first insertion of a Starr-Edwards prosthesis during pregnancy. They reviewed a series of 394 patients who had heart surgery during pregnancy; maternal mortality was 1.8% and fetal mortality was 9%. The maternal mortality of pregnant patients from the same age group who were managed medically during their pregnancies was 4.2% to 18.7% and fetal mortality as high as 50%.[3] Currently, it is believed that surgical mortality and morbidity following open mitral commissurotomy are not increased during pregnancy.[5]

Surgical experience in heart surgery during pregnancy was frequently reviewed; however, the anesthesia literature lacked such information.

Case report

A 31-year-old white woman in the 22nd week of gestation was admitted to the Cleveland Clinic for acute hemoptysis. She had had a heart murmur since age 3 and in the past 3 years dyspnea on mild exertion and occasional tachycardia developed. She denied having any ankle edema or paroxysmal nocturnal dyspnea. The night prior to admission she suddenly coughed up red blood and was treated at a local hospital with morphine and furosemide. She had two additional episodes of hemoptysis.

On admission blood pressure was 100/70 mm Hg; pulse rate, 75 beats per minute and regular. Chest examination revealed scattered rales at both lung bases, and diastolic thrill at the apex. An opening snap was heard followed by a systolic accentuation. Uterine enlargement was compatible with the gestational age, and the remainder of the physical examination was normal. Severe, decompensating mitral stenosis was diagnosed with the aid of echocardiography and immediate open mitral commissurotomy was recommended.

As premedication the patient received 15 mg Pantopon and 15 mg promazine. On arrival in the induction room, the electrocardiogram and arterial mean pressure were continuously monitored. After preoxygenation with 100% oxygen, anesthesia was induced with 200 mg thiopental sodium and 7 mg pancuronium bromide. The patient was intubated and mechanically ventilated. Anesthesia was maintained with nitrous oxide and oxygen in a 50:50 ratio to which was added 0.3% to 0.5% methoxyflurane. Before bypass the patient received the usual dose of heparin. During bypass the flow of the pump was adjusted to maintain mean blood pressure of 70 to 80 mm Hg, and no vasopressors were used. Upon termination of bypass which lasted 29 minutes the blood volume was adjusted by correlating the blood pressure, central venous pressure, and pulse rate. The residual effect of heparin was reversed by protamine sulfate (15 mg protamine for each 1000 USP units). During the entire procedure 750 ml fresh Rh negative blood and 1650 ml lactated Ringer solution were used. Arterial blood gases were analyzed every half hour, and the parameters of ventilation were adjusted to maintain a PaO_2 of 150 mm Hg and a $PaCO_2$ of 30 to 35 mm Hg. At the end of surgery the fetal heart rate was 125 beats per minute and regular.

Postoperatively the patient was ventilated mechanically for 24 hours. She was sedated with Pantopon 5 to 10 mg intravenously every 3 hours, and arterial blood gases were kept at the previously mentioned levels. Fetal heart sounds were monitored every half hour; there was no significant change in the rate and there was no vaginal bleeding or abnormal uterine contractions. On the first postoperative day the patient was extubated and received 5 liters oxygen per minute by nasal catheter. Her recovery was uneventful and she was discharged from the hospital 10 days after surgery. Subsequently, the patient delivered a full-term child without any signs of deformity or congenital abnormality.

Discussion

When anesthesia is administered during pregnancy every effort must be made to ensure continuation of pregnancy, the safety of the mother, and no injury to the fetus. Proper preparation of the patient as time permits and selection of appropriate anesthetic agents and techniques are mandatory to prevent miscarriage or affect fetal organogenesis.

Acute, uncontrollable heart failure, mainly in the second or third trimester is usually the indication for emergency heart surgery.[6] Neither the mother nor the fetus are expected to be in an ideal condition.

Pulmonary congestion, edema, and both maternal and fetal hypoxia are expected. However, the use of digitalis, diuretics, or aminophylline have no harmful effects on the fetus, and these drugs can be used preoperatively to control heart failure.

Preoxygenation and the rapid induction of anesthetic agents avoid hypoxia during intubation. Arterial blood gases are analyzed frequently during surgery and postoperatively to treat any possible hypoxia or pulmonary changes which can be deleterious for both the mother and fetus. We strongly recommend mechanical ventilation for 18 to 24 hours following open heart surgery to avoid postoperative hypoxia.[7] However, in this particular situation an added advantage of mechanical ventilation is that it will allow general sedation of the mother and help prevent any undesirable uterine contractions at this stage.

Both hypotension and the use of vasopressors have deleterious effects on the placental circulation and can lead to premature separation of the placenta. We prefer the muscle relaxant pancuronium bromide because it is free from hypotensive effects. Blood volume should be properly adjusted following cardiopulmonary bypass to prevent either pulmonary congestion or hypotension.

Most anesthetic and analgesic agents used cross the placenta to the fetus. The effect and fate of the drugs in the fetus differ from that in the mother because (a) fetal enzyme systems are still immature, (b) fetal serum proteins and pH differ from that in the mother,[8] and (c) the placenta is a site for biotransformation of certain drugs.[9] Only fully investigated anesthetic and analgesic agents free from any effects on organogenesis must be used in these patients. Unlike anesthesia for delivery, there is no concern about the effect of anesthetic agents on the fetal respiration, since the fetus is still receiving oxygen from the mother, and oxygenation in the mother is well maintained by mechanical ventilation.

Currently, nonpulsatile flow is used during cardiopulmonary bypass. The perfusing pressure during bypass was maintained around 70 mm Hg. Such perfusion pressure is usually adequate for the adult and should be adequate for the fetus, since the fetus is accustomed to a relatively hypoxic state in the uterus[10] and arterial oxygenation during bypass is higher than that in the mother (200 to 250 mm Hg). Therefore, the oxygen available to the fetus should be adequate. However, it is important to use flows from cardiopulmonary bypass higher than usual to compensate for the increased output needed to maintain the placental circulation. Higher flows are also indicated to maintain the arterial pressure without resorting to the use of vasopressors. With the use of the new blood filters during bypass, the risks of emboli reaching the placental circulation are almost eliminated. Although we maintain optimal conditions for the survival of the fetus during surgery and bypass, it may be advisable to monitor the fetal heart rate during surgery. Koh et al[11] recently described a simple ultrasound monitoring system which provides both audible and visual display of the fetal heart rate during surgery.

Summary

Anesthesia can be administered and surgery safely performed for heart surgery during pregnancy, provided that the factors which may

lead to miscarriage, such as hypoxia, hypotension, pH changes, vasopressors, and uterine excitability are avoided, and the possibility of fetal malformation is avoided by using only drugs free from any effect on organogenesis.

References

1. Ueland K: Cardiac surgery and pregnancy. Am J Obstet Gynecol **92:** 148–162, 1965.
2. Kozam RL, Conklin EF, Giannelli S, et al: Open-heart surgery for mitral stenosis during pregnancy. J Thorac Cardiovasc Surg **53:** 587–591, 1967.
3. Harthorne JW, Buckley MJ, Grover JW, et al: Valve replacement during pregnancy. Ann Intern Med **67:** 1032–1034, 1967.
4. Leyse R, Ofstun M, Dillard DH, et al: Congenital aortic stenosis in pregnancy, corrected by extracorporeal circulation; offering a viable male infant at term but with anomalies eventuating in his death at four months of age; report of a case. JAMA **176:** 1009–1012, 1961.
5. Collins HA, Daniel RA Jr, Scott HW Jr: Cardiac surgery during pregnancy. Ann Thorac Surg **5:** 300–309, 1968.
6. Lee WH, Pate JW: Surgical aspects of heart disease in pregnancy. GP **28:** 78–83, 1963.
7. Estafanous FG: Respiratory care following open heart surgery. Surg Clin North Am **55:** 1229–1241, 1975.
8. Editorial: Placental transmission of anaesthetic agents. Br J Anaesth **41:** 799, 1969.
9. Juchau MR, Pedersen MG, Fantel AG, et al: Drug metabolism by placenta. Clin Pharmacol Ther **14:** 673–679, 1973.
10. Mannix EP Jr, Mahajan DR: Open-heart surgery during pregnancy; a report of three cases. J Thorac Cardiovasc Surg **53:** 592–601, 1967.
11. Koh KS, Friesen RM, Livingstone RA, et al: Fetal monitoring during maternal cardiac surgery with cardiopulmonary bypass. Can Med Assoc J **112:** 1102–1104, 1975.