merse in halothane (*left*, fig. 1) underwent dissolution to the extent depicted in 5 min. The cartridges immersed in isoflurance and enflurane appeared undamaged to a cursory inspection after 48 h, but cracking was apparent on close inspection.

Communication with Ohmeda revealed that most plastics are disrupted to some extent by immersion in liquid agents, but that they have good resistance to disruption on exposure to the vapors of the three commonly used agents. Ohmeda does not recommend use that would involve immersion.§

Because of this incident we recommend that one of two precautions be taken during closed-system anesthesia using the injection technique: 1) The TVX® flow cartridge should be placed upstream of the injection port, so that anesthetic agent does not pool on it. A possible arrangement is to place the flow cartridge between the Y-piece and the expiratory tubing. As long as the flow detector of the volumeter is placed appropriately for the direction of the expiratory flow, the flow cartridge can be inverted and still function. 2) Alternatively, the flow cartridge could be omitted and another technique of measuring ventilatory volume chosen.

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Anesthetic Management of Cesarean Section in a Patient with Idiopathic Hypertrophic Subaortic Stenosis

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Idiopathic hypertrophic subaortic stenosis (IHSS) is a disease characterized by obstruction to left ventricular outflow secondary to hypertrophy of septal myocardium just beneath the aortic valve. The stiff, hypertrophied muscle mass leads to diastolic dysfunction as well as systolic obstruction; this is manifested as congestive heart failure (with exertional dyspnea and easy fatigability), dysrhythmias, and sudden death. Inheritance is autosomal dominant, with initial presentation of symptoms in childhood or young adulthood.

Obstruction to left ventricular outflow by the hypertrophic myocardium may be affected by several of the physiologic alterations occurring in pregnancy and during labor. The hypervolemia associated with pregnancy can improve circulatory dynamics. However, any decrease in preload, as would occur in blood loss, supine positioning (with aortocaval compression by the uterus), and the Valsalva maneuver, could increase left ventricular outlet obstruction by making the left ventricular cavity smaller. The consequent decrease in cardiac output could compromise uterine blood flow. During labor and delivery, catecholamine release secondary to pain, fear, and anxiety can increase the inotropic state of the myocardium, thereby increasing subvalvular stenosis.

Reports,[1,2] of cesarean deliveries in these patients have failed to define their anesthetic management; therefore, we are reporting the results of such a case.

Report of a Case

A 26-yr-old primagravida, with known twin pregnancy at 32 weeks gestation, was admitted with possible toxemia of pregnancy and premature cervical dilation and effacement.

Ten years prior to admission, IHSS was diagnosed by cardiac catheterization and ultrasound. (These were performed at another hospital and the results were unavailable to us.) She was receiving propranolol 40 mg po qid, and, because of ventricular tachycardia noted on ambulatory electrocardiographic monitoring approximately 1 yr earlier, was also being treated with procainamide 750 mg po qid. Past surgical history was remarkable only for strabismus surgery and tonsillectomy in childhood which were uncomplicated. Family history revealed two brothers with IHSS who died suddenly in young adulthood.

The patient was asymptomatic on admission. Physical examination revealed an arterial blood pressire of 112/60 mmHg. The lungs were clear. Cardiac examination revealed normal first and second heart sounds. There were no third or fourth heart sounds. A grade 3/6 systolic ejection murmur was audible at the left lower sternal border and a grade 3/6 holosystolic murmur was heart at the apex. The ankles

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had moderate pitting edema. Cervical examination revealed 1.0 cm dilation with 50% effacement. There was 1+ (out of 4+) proteinuria on urinalysis; other laboratory values were within normal limits. Ultrasound examination confirmed twins with one fetus in breech presentation. Two days later, the patient began having uterine contractions, and her cervix dilated to 3 cm with 90% effacement. Her premature labor was managed with iv magnesium sulfate, which was preferred over beta-adrenergic agonists because of its negative inotropic effects. Several hours later, after a decrease in arterial blood pressure to 80–90 mmHg systolic, magnesium sulfate was discontinued and a radial artery catheter was inserted. The patient was scheduled for emergent cesarean section because of failure to respond to tocolysis and breech presentation of one fetus.

Physical examination just prior to surgery revealed an 88-kg woman with rapid, shallow respirations, an arterial blood pressure of 80/45 mmHg, and a heart rate of 60 beats/min. Left basilar rales were heard on auscultation of the chest as were the aforementioned murmurs. Moderate pitting edema at the ankles was noted. Fetal heart monitoring at this time showed no evidence of fetal compromise.

Prior to the induction of general anesthesia, sodium citrate (30 ml) was administered po, and a pulmonary artery catheter was inserted *via* the right internal jugular vein. The preinduction pulmonary artery diastolic pressure was 40 mmHg; the heart rate was 64 beats/min and the systemic arterial blood pressure had risen to 140/95 with iv hydration and cessation of magnesium therapy. A urinary catheter was inserted. Following denitrogenation of the lungs, a rapid-sequence induction of anesthesia was performed using 250 mg of thiopental and 140 mg of succinylcholine iv. Cricoid pressure was maintained until the trachea was intubated. Laryngoscopy and tracheal intubation were uneventful, with minimal hemodynamic alteration. Enflurance (0.5–1.25%) with 99% O_2 (to ensure maximal oxygenation of both mother and fetus) was administered prior to delivery of the infants. After delivery, 50% nirous oxide was added and supplemented with two iv boluses of 50 μg each of fentanyl, while the enflurane concentration was maintained between 0.5–1.25%. Propranolol (2 mg) and procainamide (100 mg) were administered iv to continue the maintenance regimen. Ten units of oxytocin in 500 ml of lactated Ringer's solution was administered iv over 1 h following the delivery of the infants. Gentamicin 80 mg and ampicillin 2 g were given iv for endocarditis prophylaxis.[1-7] A total of 2,300 ml of crystalloid was administered iv. Blood loss was estimated at approximately 800 ml.

On emergence from anesthesia, the trachea was extubated and the patient was transferred to the intensive care unit. On arrival there, arterial blood pressure was 100/70 mmHg, and her heart rate was 60 beats/min. The pulmonary artery pressure was 60/40 mmHg with a pulmonary capillary occluded (wedge) pressure of 27 mmHg. The cardiac output was 3.5 l/min. The patient was awake and comfortable except for mild abdominal pain and mild dyspnea. A 12-lead electrocardiogram revealed a sinus bradycardia at 55 beats/min with a right bundle branch block (prior ECGs were unavailable for comparison). Chest roentgenogram revealed pulmonary vascular congestion, increased interstitial fluid (Kerley B lines), and moderate cardiomegaly. The patient was breathing 100% O_2 *via* a face mask and gradually "weaned" to breathing room air over the next day.

On the first postoperative day, the patient was comfortable after a diuresis of 1.5 l following the administration of 10 mg of furosemide iv. Her pulmonary artery diastolic pressure and pulmonary capillary wedge pressure (PCWP) decreased to 28 mmHg and 21 mmHg, respectively. Cardiac output was essentially unchanged. Subsequently, the pulmonary artery catheter was removed. The patient was discharged home 4 days later, asymptomatic, and was receiving the same doses of propranolol and procainamide as she had prior to pregnancy.

The first-born twin, a girl, weighing 1,540 g, with Apgar scores of 7 and 8, did not require resuscitation. At 2 days of age she developed two episodes of apnea and bradycardia and was treated with iv theophylline. She was discharged 1 week later. The second child, a 1,460 g boy, with Apgar scores of 8 and 8, also did not require resuscitation at delivery but subsequently required ventilatory support for respiratory distress syndrome (RDS). He was discharged 2 weeks later.

Discussion

Most patients with IHSS develop exacerbation of their disease during pregnancy, and especially during labor and delivery,[7] although other reports[1,2] have shown pregancy and delivery to be well tolerated in these patients. Normally, in the first and second trimester and peaking at about the 20th week, there is an approximately 40% increase in plasma volume and a 20% increase in red blood cell volume. This, along with a slight increase in heart rate (10–15 beats/min) and a decrease in peripheral vascular resistance, leads to a 40–50% increase in cardiac output. With twin gestation, these changes occur earlier and are of greater magnitude. While the hypervolemia helps minimize obstruction to ventricular outflow, the decrease in peripheral vascular resistance, especially with supine positioning, decreases venous return, which can aggravate symptoms. Furthermore, while pulmonary artery pressure decreases during normal pregnancy, the increased plasma volume with increased hydrostatic pressure (plus decreased colloid osmotic pressure due to dilutional hypoalbuminemia) can cause pulmonary edema earlier in patients with preexisting heart failure. At term with increased catecholamines during labor, the contractile state of the myocardium increases, causing further narrowing of the subaortic outflow tract.

Beta-adrenergic blocking drugs, because of their negative inotropic effect, have been the mainstay of medical treatment. However, chronic use of the drugs throughout pregnancy may increase uterine tone, leading to a small placenta and a low birth weight infant.[8-11] Beta-adrenergic blockade is also potentially harmful to the fetus if hypoxia occurs during labor, because it will attenuate the fetal sympathetic response.[12] Fetal and neonatal hypoglycemia are potential problems,[12] although the clinical significance of the latter abnormalities has been disputed.[11] Some authors feel that beta-adrenergic drugs should be used quite cautiously,[12] especially in asymptomatic patients during pregnancy. Use during labor and delivery (either vaginal or abdominal) is more clearly beneficial[1,2,10] because of increased adrenergic output at this time. Until recently, elective abdominal delivery was recommended for these patients because of the potential hazards of labor and vaginal delivery. Cesarean delivery is now reserved for obstetric indications only.[1,2]

Optimal anesthetic management of cesarean delivery in IHSS has not been determined, except that regional (spinal or epidural) anesthesia is relatively contraindicated because of resultant vasodilation associated with sympathetic blockade of the lower extremities.[2,13] Regardless

of the anesthetic technique selected, left uterine displacement should be employed routinely, cross-matched blood should be readily available and, if required, vasopressors devoid of inotropic effect (*i.e.*, phenylephrine or methoxamine) may be used following delivery. Prior to delivery, alpha-adrenergic agonists should be avoided as they may decrease uterine blood flow; intravascular volume expansion should be used instead. Oxytocin, due to its vasodilating properties, may be detrimental in such patients; therefore, ergonovine is the preferred oxytocic[1,2] as it has vasoconstrictor action (although our patient demonstrated no adverse effects to a slow oxytocin infusion). Caution must be exercised with concomitant use of ergonovine and vasopressors as they may potentiate each other's effect. While propranolol may be beneficial to the mother, the neonate should be closely monitored for several days for bradycardia, impaired respiration, and hypoglycemia. Calcium channel blockers (*i.e.*, verapamil) have recently come into use for IHSS[14,15] Maintenance of euvolemia or slight hypervolemia is essential, and monitoring of central venous pressure or pulmonary artery pressures is helpful.

The choice of inhaled anesthetic is not clear. Volatile anesthetics that are myocardial depressants may be beneficial. Halothane[16] improves hemodynamics in IHSS. Because of relatively greater decrease in systemic vascular resistance (SVR) and increase in heart rate with enflurane and isoflurane, respectively, these agents have not been recommended by some authors.[13] However, we chose to use enflurane in our patient because of possible aggravation of her ventricular ectopy with halothane. Sudden death, presumably due to dysrhythmias, occurs in up to 30% of patients with IHSS.[3]

In summary, anesthetic management of the parturient with IHSS undergoing abdominal delivery is complex. Adequate preload and afterload must be maintained, which relatively contraindicates regional anesthesia. Myocardial contractility must be depressed, for which the volatile anesthetics are useful, but the ideal anesthetic for this is not available as the increased heart rate from isoflurane, decreased SVR from enflurane and isoflurane, and potential for ventricular ectopy with halothane all pose potentially serious problems. Ready availability of antidysrhythmics, vasopressors, cross-matched blood, beta-adrenergic blockers, and possibly calcium channel blockers is essential, and ergonovine is preferable to oxytocin for uterine contraction.

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