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$S\dot{v}_{O_2}$ Monitoring during Spinal Anesthesia and Cesarean Section in a Parturient with Severe Cyanotic Congenital Heart Disease

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ADVANCES in palliative and corrective cardiovascular surgical procedures have lead to increasing numbers of patients with congenital heart disease in the obstetric population.[1] We describe a parturient with palliated complex congenital heart disease. Our peripartum treatment of this challenging patient included mixed venous oximetry and continuous spinal anesthesia.

Case Report

The patient is a 20-yr-old woman with cyanotic congenital heart disease who presented for initial obstetric care at 21 weeks' gestation. She was born with tricuspid atresia and a severely hypoplastic right ventricle. (fig. 1) She also had a discrete stenosis at the junction of the right pulmonary and main pulmonary arteries. Outflow from the right atrium was through an atrial septal defect into the left atrium. At 4 days of age she underwent placement of a Waterston shunt from the aorta to the pulmonary artery. As a result of the shunt, pulmonary hypertension developed in the right lung. The left lung remained grossly underperfused, with its only blood supply coming from the shunt in a retrograde fashion through the stenotic right pulmonary artery. There was very little, if any, flow *via* a tract from the left ventricle to the pulmonary artery. At presentation, her exercise tolerance was poor with significant shortness of breath after one flight of stairs. She had no history of syncope, orthopnea, or arrhythmias, and she had never been administered any cardiac medications, although her medical care had been sporadic.

At examination, she was noted to be very thin, 50 kg and 155 cm, with a Mallampati class I airway. Her heart sounds were regular with a continuous systolic murmur and a prominent parasternal lift. She had significant digital clubbing and peripheral cyanosis. Her baseline oxygen saturation ranged from 80 to 83%, with precipitous drops to the low 70s by pulse oximetry with minimal activity, such as walking to her bathroom. Her hematocrit was 53%. A room air arterial blood gas showed a *p*H of 7.42, a pressure of carbon dioxide (P_{CO_2}) of 30 mmHg, and a pressure of oxygen (P_{O_2}) of 50 mmHg. An echocardiogram showed asymmetric left ventricular hypertrophy with evidence of volume overload. The left ventricle was dilated with a mildly diminished ejection fraction. No abnormalities of the mitral or aortic valves were seen. A 1993 cardiac catheterization revealed a patent shunt and pulmonary artery pressures averaging two thirds of systemic pressures. The shunt fraction ($Qp/Qs = SA_{O_2} - Sv_{O_2}/Spv_{O_2} - SpA_{O_2}$) at that time was calculated as 2.5:1.

Fetal ultrasonography revealed severe intrauterine growth restriction with the fetus at the <1 percentile for both weight and head circumference. The patient was admitted and placed on bed rest and oxygen.

At 27 weeks of gestation a decision was made to perform a cesarean

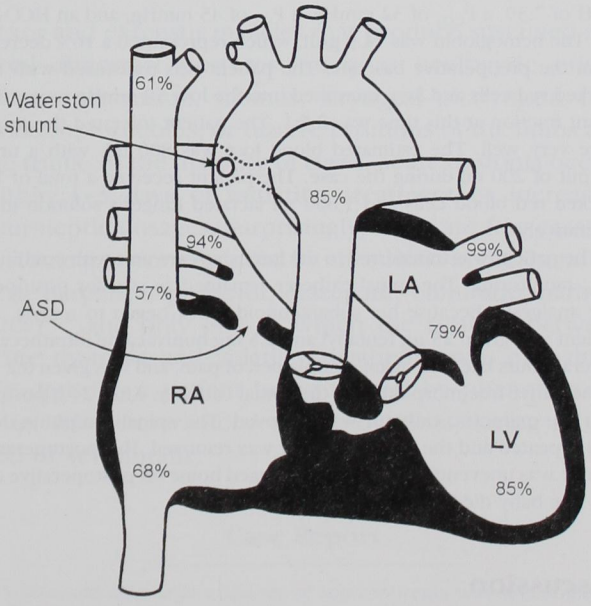

Fig. 1. Schematic drawing of the patient's heart included to help visualize her cardiac anatomy. The numbers represent oxygen saturations measured during cardiac catheterization.

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Table 1. Hemodynamic and Laboratory Data

Event	Sa_{O_2} (mmHg)	Sv_{O_2} (mmHg)	BP (mmHg)	Q_p/Q_s	$pH/p_{CO_2}/p_{O_2}/HCO_3$	Intervention
Initial values	90	57	142/68		7.46/29/52/20	
Spinal catheter placement	90	46	134/67			O_2 off, iv fentanyl
T4 sensory level	86	56	135/68	2.3:1	7.38/36/47/21	
5 min after delivery	84	38	138/57	2.6:1	7.39/32/45/21	1 unit PRBC
30 min after delivery	83	52	149/57	1.8:1		

section for poor fetal well being. Fetal ultrasonography revealed no interim growth of the fetus, and umbilical artery Doppler studies showed reverse diastolic flow. There had been no change in her physical status since her admission 5 weeks previously. She was brought to the operating room and placed in left uterine displacement. Standard monitors were placed. Her Sp_{O_2} on 6 l/nasal cannula was 90%. A left radial 20-g arterial line and a right internal jugular introducer were placed, and an oximetric catheter was inserted with its tip in the superior vena cava. Baseline Sv_{O_2} was 57 mmHg (table 1). A continuous spinal catheter (Braun Perifix® 20-g open-tip catheter; B. Braun Medical Inc., Bethlehem, PA) was then placed at the L3–4 interspace using a 17-g Touhy needle with the patient in a sitting position. During placement of the spinal catheter the Sv_{O_2} was noted to gradually decrease from 57 to 49 mmHg. Discontinuation of supplemental oxygen resulted in Sp_{O_2} decreases to the mid 80% range. The patient was given 25 μg fentanyl intravenously. A total of 15 mg hyperbaric bupivacaine plus 25 μg fentanyl were administered incrementally over 10 min. A T4 sensory block with good surgical anesthesia was obtained. At this point the Sv_{O_2} had increased to 56 mmHg. The shunt fraction (Aosat − Sv_{O_2}/P_Vsat − $P_A$sat) was 2.3:1 The fetal heart rate pattern remained stable throughout this period.

In the minutes after delivery of a 365-g infant, the patient's Sv_{O_2} decreased to 38 mmHg, with an arterial oxygen saturation (Sa_{O_2}) of 87% and a shunt fraction of 2.6:1. Room air arterial blood gas showed a *p*H of 7.39, a P_{CO_2} of 32 mmHg, a P_{O_2} of 45 mmHg, and an HCO_3 of 19. The hemoglobin was 14.9 g/dl, which represented a 16% decrease from the preoperative baseline. The patient was transfused with 1 U packed red cells and Sv_{O_2} increased into the low 50 mmHg range. The shunt fraction at this time was 2.5:1. The patient tolerated the procedure very well. The estimated blood loss was 800 ml, with a urine output of 200 ml during the case. The patient received a total of 1 U packed red blood cells and 1,800 ml lactated Ringer's solution intraoperatively.

The patient was transferred to the intensive care unit with continued Sv_{O_2} monitoring. The spinal catheter remained *in situ* for postoperative analgesia. Because her subarachnoid block began to recede, the patient was given 25 μg fentanyl and 2.5 mg bupivacaine intrathecally. Several hours later she again complained of pain, and was given 0.2 mg preservative-free morphine *via* the spinal catheter. After 24 h postpartum the oximetric catheter was removed. The spinal morphine dose was repeated and the spinal catheter was removed. Her postoperative course was uneventful. She was discharged home on postoperative day 12. The baby died on day of life 8.

Discussion

There are numerous reports but few actual studies of the maternal implications of cyanotic heart disease. One report noted an increased incidence in the frequency of congestive heart failure and deterioration in functional status.[2] A retrospective study by Presbitero *et al.*[3] identified maternal risk factors in 44 patients with cyanotic heart disease, without Eisenmenger's physiology. Type of maternal disease, ability index, and prepregnancy hemoglobin and Sa_{O_2} were identified by univariate analysis as factors that discriminate between successful and unsuccessful outcomes. Prepregnancy hemoglobin and oxygen saturation were the most predictive of poor maternal and fetal outcomes. Because of the great variability in anatomy and functional capacity in these patients, further classification in terms of pulmonary blood flow and physiology is useful. The presence of pulmonary hypertension alone in the parturient carries a mortality risk of 30–50%, depending on its nature and severity.[4] The fetal risks of maternal cyanotic heart disease are well characterized. These include spontaneous abortion, preterm delivery, and severe growth restriction related to the low level of maternal oxygen saturation and high blood viscosity. A prepregnancy hemoglobin of greater than 20 and an SaO_2 of less than 85% are the worst prognostic indicators of poor fetal outcome.[3] Our patient's prepregnancy values included a hemoglobin of 19 g/dl and an Sa_{O_2} of 83%.

After discussions with her cardiologists, and after reviewing the echocardiogram and cardiac catheterization films, the consensus was that myocardial failure was likely in this patient. Inotropic support during the peripartum period was anticipated. Our anesthetic management was planned with the goal of maximizing systemic oxygen delivery and monitoring for myocardial dysfunction in the event of expected cardiovascular and fluid changes. A regional anesthetic was chosen. The continuous spinal provided a reliable, dense, titratable block with peripheral vasodilation. The monitoring options were limited because of the patient's anatomy. We thought that simply following the mean arterial pressure and Pa_{O_2} values in this patient would not provide sufficient information about her cardiac status and that central venous pressure measurements alone would not

provide much additional information. Traditional thermodilution cardiac output measurements are inaccurate in the setting of an intracardiac shunt, and because of the patient's anatomy, placement of such a catheter was not an option.

An oximetric central venous catheter was therefore chosen to give at least indirect information about systemic oxygen delivery and myocardial function. Following shunt fractions and potentially manipulating the balance between pulmonary and systemic blood flow was another concern. Increasing the Pa_{CO_2} and lowering the Pa_{O_2} before incision aimed to divert blood away from the pulmonary bed by causing pulmonary vasoconstriction. The rising Sv_{O_2} suggested an increase in systemic oxygen delivery. The estimated shunt fractions remained fairly constant throughout the procedure and no interventions were made on this basis alone.

In our opinion, the use of central venous Sv_{O_2} monitoring substantially facilitated the care of our patient. We also believe that the continuous availability of Sv_{O_2} would have proven extremely helpful in the event of any untoward cardiovascular events. We suggest that anesthesiologists consider such monitoring carefully when confronted with patients such as ours.

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Acute Dystonia during Sevoflurane Induction

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DRUG-INDUCED extrapyramidal syndromes occur most frequently with use of neuroleptic agents, but can also be induced by many drugs acting on the central nervous system.[1] Among these syndromes, dystonic reactions are characterized by involuntary contractions in opposing flexor and extensor muscles that produce sustained and fixed abnormal postures, such as oculogyric crises, tongue protrusion, trismus, laryngeal–pharyngeal constriction, torticollis, or bizarre positions of the limbs and the trunk. In the majority of cases, acute dystonia occurs within 1–3 days of initiating treatment or increasing neuroleptic dosages. Surprisingly, there are few cases of dystonic reactions induced by anesthesia in the literature (*e.g.,* narcotics, propofol, diazepam, enflurane, barbiturates)[2–5] and only one in which the relation between acute dystonia and volatile anesthetics was probable.[6] We describe a patient being treated by phenothiazine drug, who experienced severe dystonia during inhalation of sevoflurane.

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Key words: Antipsychotic medication; dopaminergic pathways; halogenated drug; torticollis.

Case Report

A 19-yr-old man with a history of schizophrenia was scheduled for removal of multiple impacted teeth during general anesthesia. He had been treated by cyamemazine, a phenothiazine antipsychotic drug, in a daily dose of 75 mg for 2 yr, along with 180 mg dihydroergotamine