Anaesthetic management of a parturient with pulmonary stenosis and aortic incompetence for Caesarean section

N. Campbell¹, O. P. Rosaeg¹* and K. L. Chan²

¹Department of Anesthesiology, The Ottawa Hospital—Civic Campus and ²The Adult Congenital Heart Clinic, University of Ottawa, Ottawa, Canada

*Corresponding author: Department of Anesthesiology, The Ottawa Hospital—Civic Campus, University of Ottawa, 1053 Carling Avenue, Ottawa, Ontario K1Y 4E9, Canada. E-mail: norse@cyberus.ca

Anaesthetic management of Caesarean section in a parturient with severe pulmonary stenosis and aortic regurgitation is described. The valvular sequelae resulted from previous unsuccessful surgical correction (Ross procedure) of congenital aortic stenosis. This case demonstrates the importance of multi-disciplinary assessment and careful anaesthetic planning, to avoid deterioration in perioperative cardiac performance in parturients with complex valvular disease.

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Congenital heart disease is now more common than rheumatic heart disease in pregnant women in the western world.¹ Many children with significant congenital valve disease undergo heart surgery, resulting in a new subpopulation of parturients who have had previous cardiac procedures. Previous surgery may produce some residual valvular defect or new, complicated valvular dysfunction.² The anaesthetic management of these parturients must be based on individual assessment of cardiac function and reserve, to anticipate the impact of anaesthetic techniques for vaginal delivery or Caesarean section on cardiac performance.

Case report

A 20-yr-old primigravid parturient was referred to the obstetric anaesthesia service at 29 weeks gestation for assessment before elective Caesarean delivery. She had undergone cardiac surgery (Ross procedure: aortic valve autograft and pulmonary homograft) for congenital aortic stenosis, 5 yr earlier. Unfortunately, she developed severe pulmonary stenosis and aortic incompetence after this procedure. She experienced one episode of right ventricular failure, with associated dyspnoea, New York Heart Association (NYHA) class III, palpitations and dizziness, at 24 weeks gestation. Balloon angioplasty of the main pulmonary artery at that time resulted in some temporary improvement in the gradient between right ventricular and pulmonary artery pressures. The baseline pressure measurements were 53/0 mm Hg in the right ventricle and 15/6 mm Hg in the pulmonary artery before balloon angioplasty. The pressure in the right ventricle was 35/7 mm Hg and 16/11 mm Hg in the pulmonary artery after the angioplasty procedure.

However, at 28 weeks gestation, she again experienced shortness of breath on exertion, NYHA class II. There was no chest pain, dizziness or palpitations. On examination, she was comfortable and not cyanosed. She had a heart rate of 80 beats min⁻¹ and her arterial pressure was 118/62 mm Hg. Jugular venous pressure was not elevated. There was a palpable right ventricular heave with a systolic murmur (grade 4/6) at the right sternal edge, and an early diastolic murmur (grade 2/4) heard at the left sternal edge. There was no evidence of pulmonary oedema, hepatomegaly, or peripheral oedema.

An echocardiogram at that time demonstrated severe aortic regurgitation, pulmonary stenosis (mean gradient 40 mm Hg), a right ventricular systolic pressure of 95 mm Hg, and increased left ventricular size with normal systolic function. Haematological and biochemical investigations were within normal limits. The electrocardiogram (ECG) showed sinus rhythm of 82 min⁻¹ with a normal axis, borderline left ventricular hypertrophy and non-specific intra-ventricular conduction delay. During the admission, she experienced several episodes of atypical chest pain. She received subcutaneous, unfractionated heparin 7 500 IU
twice daily for deep venous thrombosis prophylaxis, furosemide 20 mg and atenolol 25 mg p.o. once daily, and i.m. dexamethasone. She remained stable in hospital, and serial ultrasound examinations confirmed that fetal growth was normal. It was determined that she should undergo elective Caesarean section at 32 weeks gestation. The activated partial thromboplastin time (aPTT) was normal before epidural catheter insertion. In the operating room, non-invasive arterial pressure monitoring, 5-lead ECG with ST-segment analysis, and pulse oximetry were applied. Her oxygen saturation, breathing room air, was 96%. I.v. access was secured. Her arterial pressure was 104/58 mm Hg and heart rate was 110 beats min⁻¹ before insertion of invasive arterial pressure monitoring and an epidural catheter. The patient was anxious and required fentanyl 25 µg and midazolam 2 mg i.v. to facilitate the invasive procedures. She received an i.v. infusion of Ringer’s lactate solution 300 ml. A 20G polyurethane epidural catheter was inserted at L3–L4 using a 16G Tuohy needle and loss of resistance to saline technique. The patient was placed in the supine position with left uterine displacement and given supplemental oxygen.

A test dose of lidocaine 2%, 3 ml with epinephrine 1:200 000 was administered to exclude i.v. or intrathecal placement of the epidural catheter. Epidural anaesthesia was initiated with lidocaine 2%, 12 ml with epinephrine 1:200 000, given in 4 ml aliquots at 5 min intervals, with epidural fentanyl 100 µg. A total of 15 ml of the anaesthetic solution resulted in a loss of sensation to pinprick to T10. A further 5 ml of solution was given, resulting in an extension of the block to T4. At this time, the patient experienced chest discomfort and dyspnoea. The arterial pressure decreased abruptly to 78/40 mm Hg, with a junctional rhythm of 80 min⁻¹. A bolus of ephedrine 5 mg i.v. was given immediately, followed by pentastarch 10%, 250 ml. Her arterial pressure increased to 140/76 mm Hg after administration of ephedrine, the chest pain resolved and normal sinus rhythm returned. There were no ST-segment changes associated with this episode. Surgery proceeded uneventfully, and a healthy infant was delivered with an Apgar score of 9, at 1 and 5 min. An i.v. infusion of oxytocin 3.0 IU h⁻¹ was started. Her arterial pressure increased to 160/62 mm Hg with a heart rate of 120 beats min⁻¹ after delivery of the infant. Metoprolol 2 mg i.v. was given to decrease the heart rate and reduce the arterial pressure. Furosemide 10 mg i.v. was given after delivery of the placenta, to promote diuresis and reduce the increase in preload induced by auto-transfusion from uterine contraction and relief of inferior vena cava obstruction. The heart rate decreased to 100 beats min⁻¹ and the arterial pressure to 130/72 mm Hg after administration of i.v. metoprolol. The remaining intra-operative course was uneventful. Estimated blood loss was 500 ml. Preservative-free morphine 3 mg was given via the epidural catheter for postoperative analgesia and ondansetron 4 mg i.v. was given for antiemetic and antipruritic prophylaxis.

She remained in the post-anaesthesia care unit for observation overnight. Creatine kinase was elevated after surgery, but troponin concentrations were normal and there were no new ECG changes. She was discharged home, in good condition, on the fourth day after operation.

The patient returned to hospital for cardiac surgery 2 months after the Caesarean section. She underwent a Bentall procedure, where the stenotic pulmonary homograft was replaced by a new one, in addition to insertion of a mechanical aortic valve. The entire old pulmonary homograft was found to be contracted and very stenotic (1.0 cm²), and the aortic valve incompetence was largely because of aortic root dilation. There were no postoperative complications, and the patient was discharged home on coumadin and acetylsalicylic acid on the fifth day after operation.

**Discussion**

Recent advances in paediatric surgical treatment of congenital heart disease have resulted in ~90% of these patients surviving to adult life. The anaesthetic management of these women at delivery requires an understanding of the original valvular defect, the corrective cardiac surgery procedure, and the sequelae to that procedure. The impact of the normal cardiovascular changes of pregnancy and the anaesthetic technique can then be anticipated.

The Ross procedure uses the patient’s own pulmonary valve to replace the stenotic aortic valve, and a cadaveric valve to replace the pulmonary valve. This allows the patient to avoid anticoagulation after surgery. This operative procedure is suitable for women of child-bearing age as the pulmonary autograft has the potential to grow. The autograft also has greater longevity than a bioprosthesis (e.g. porcine) valve, which deteriorates more rapidly than an autograft.

Pulmonary artery stenosis increases the intraventricular pressure and work of the right ventricle. Severe pulmonary stenosis can reduce left ventricular output because of reduced forward flow. It is important to maintain preload to optimize myocardial contractility; however, excessive preload can precipitate right heart failure and atrial arrhythmias. During pregnancy, preload may be reduced by aortocaval compression, and at delivery by the vasodilatation associated with neuraxial sympathetic block.

Regurgitation of blood flow through an incompetent aortic valve reduces cardiac output and coronary artery blood flow. Reducing afterload improves forward flow, and maintaining a high-normal heart rate reduces the time for regurgitant flow. The reduction in afterload and increase in heart rate associated with pregnancy may improve symptoms in patients with aortic regurgitation. In patients with severe aortic incompetence, coronary arterial flow occurs predominantly during systole; diastolic coronary blood flow is markedly reduced.

Maternal cardiac morbidity and mortality (and fetal outcome) during pregnancy correlate strongly with maternal
functional status. Although this primigravid woman was acyanotic and NYHA class II, she had significant pulmonary artery stenosis with pulmonary hypertension, in addition to severe aortic regurgitation. Elective Caesarean delivery was planned at 32 weeks gestation to maximize fetal lung maturation and to avoid deterioration in maternal cardiac status. The cardiovascular changes associated with labour and vaginal delivery, including a further increase in cardiac output and oxygen consumption and Valsalva manoeuvres, were thought to be too hazardous to attempt induction of labour.

As a compromise, the haemodynamic objectives for this patient were to maintain a normal heart rate. Ideally a low-normal heart rate is preferable for patients with pulmonary stenosis, whereas patients with aortic regurgitation require a high-normal heart rate. Also, to maintain cardiac output we would aim to maintain preload, but tolerate a moderate reduction in afterload which would favour forward flow through the incompetent aortic valve. We chose to slowly induce epidural anaesthesia with incremental injections of small aliquots of lidocaine and fentanyl to provide effective anaesthesia, while minimizing haemodynamic changes during surgery. Regional anaesthesia also avoided the need for general anaesthesia with its well-known risks of aspiration, difficult tracheal intubation, and a hypertensive response to intubation. Invasive arterial pressure monitoring facilitated early recognition and treatment of deleterious intraoperative changes in arterial pressure. The brief hypotensive episode with junctional rhythm was likely attributable to a combination of reduced preload, and epidural local anaesthetic block of the sympathetic cardioaccelerator nerves to the heart. Ephedrine, which stimulates cardiac beta receptors to a greater degree than peripheral alpha receptors, was the preferred sympathomimetic drug in this situation; administration of a pure alpha agonist (e.g. phenylephrine) would have increased afterload considerably, and therefore reduced forward flow across the regurgitant aortic valve. We elected not to attempt to insert a central venous catheter because we anticipated that this procedure would be too stressful for this anxious patient, and likely result in a further increase in heart rate. Also, central venous catheterization is associated with several well-known complications. Although right-sided pressures would not have reflected left-sided filling pressures in this patient, some information regarding the intraoperative trend in central venous pressure might have been obtained. Insertion of a central venous catheter via an antecubital vein might have been a feasible alternative, and would probably have been acceptable to the patient.

There are few data in the literature to guide the anaesthetic management of parturients with severe pulmonary or aortic valve disease. Epidural anaesthesia, using incremental injections of bupivacaine 0.5%, has been successfully used for Caesarean delivery in a woman with Watson’s syndrome who had pulmonary stenosis. Analgesia for labour and delivery has been provided with an intrathecal sufentanil infusion, and a 15 mg bolus of lidocaine 1% for vacuum extraction, in another parturient with isolated severe pulmonary stenosis. A precipitous and marked decrease in afterload can, however, result in profound hypotension and myocardial ischaemia in patients with severe aortic insufficiency. Cardiovascular collapse and death has been described in a woman with aortic regurgitation and pre-eclampsia who received a single bolus of 18 ml of epidural bupivacaine 0.5% (after a 2 ml test-dose of the same solution) for Caesarean delivery. Patients with significant multivalvular heart disease require careful pre-operative, multi-disciplinary assessment and anaesthetic planning before delivery, to optimize cardiac function during the peripartum period and make informed decisions regarding mode of delivery and anaesthetic technique. Preconceptual consultation with a cardiologist specializing in congenital heart disease is also advisable for women with complex cardiac valvular disease.

References

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