Clinical Reports

Pulmonary hypertension and cardiomyopathy: anaesthetic management for Caesarean section

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Pulmonary hypertension in pregnant women is uncommon but is associated with a high mortality. We present the case of a 14-yr-old parturient with pulmonary hypertension and cardiomyopathy who required a Caesarean section. Management goals included: (1) maintaining right ventricular function, (2) avoiding the haemodynamic effects of general endotracheal anaesthesia, and (3) minimizing narcotic-related neonatal respiratory depression. While most authors agree on invasive pulmonary and systemic monitoring, opinions differ as to the optimal method of providing anaesthesia for these patients. The successful use of lumbar epidural anaesthesia with lidocaine and fentanyl is described. When the local anaesthetic was administered slowly and in increments, epidural anaesthesia was safe for both mother and fetus.

L'hypertension pulmonaire chez les femmes enceintes n'est pas fréquente, mais elle est associée à un taux élevé de mortalité. On présente le cas d'une parturiente de 14 ans atteinte d'hypertension pulmonaire et d'une cardiomyopathie qui a requis une césarienne. Le plan d'action incluait: 1) le maintien de la fonction ventriculaire droite, 2) le contrôle des effets hémodynamiques de l'anesthésie générale endotrachéale et 3) le contrôle de la dépression respiratoire néonatale reliée aux narcotiques. Alors que la majorité des auteurs s'accordent sur la surveillance systémique et pulmonaire invasive, les opinions divergent sur la méthode optimale de fournir l'anesthesie à ces patientes. L'utilisation avec succès de l'anesthésie épidurale lombaire avec de la lidocaïne et du fentanyl est décrite. Quand l'anesthésique local fut administré lentement et avec des doses de rajouts, l'anesthésie épidurale fut sécuritaire tant pour la mère que pour le foetus.

Key words
ANAESTHESIA: obstetric;
ANAESTHETIC TECHNIQUES: epidural;
HEART: cardiomyopathy, patent foramen ovale;
LUNG: pulmonary hypertension.

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Primary pulmonary hypertension is a rare condition with a mortality of up to 79% within five years from the time of diagnosis.[1] In women with pulmonary hypertension (PH) the physiological changes of pregnancy may be tolerated poorly.[2] This report details the anaesthetic management of a patient with the combination of primary PH and cardiomyopathy who underwent Caesarean section.

Case report

A 14-yr-old gravida 1 para 0 North American Indian girl at 25 wk gestational age presented to an obstetrician for antenatal and peripartum care. She was known to have pulmonary hypertension, cardiomyopathy and a patent foramen ovale (PFO) for at least the past five years. In 1985, 1986 and 1987 she underwent cardiac catheterization (Table I). Endocardial biopsies of the right ventricle (RV) and the left ventricle (LV) were performed in 1987. Biopsy results were consistent with a congestive cardiomyopathy, although the cardiologist considered that pulmonary hypertension was the most important of her problems. At the time of presentation, pregnancy had been well tolerated for 25 weeks. Neither the patient nor her obstetrician thought termination was indicated. At 34 wk gestation she was admitted to hospital for failure to gain weight. She reported occasional dyspnoea when climbing one flight of stairs but was otherwise asymptomatic.

Initial physical examination showed a frail, acyanotic girl without evidence of clubbing. Heart rate (HR) was 80 beats per minute (bpm) and regular, blood pressure

TABLE I Previous cardiac catheterization data

Date	*RAP*	*PAP*	*Mean PAP*	*PCWP*	*LV O_2 Sat*	*LA O_2 Sat*
Apr 11/85	3	71/39	46		94%	
Jan 23/86		47/13	20	4		96%
Feb 26/87		45/15	24	9	96%	

RAP = right atrial pressure (mmHg), PAP = pulmonary artery pressure (mmHg), mean PAP = mean pulmonary artery pressure (mmHg), PCWP = pulmonary capillary wedge pressure (mmHg), LV O_2 Sat = oxygen saturation in the left ventricle, LA O_2 Sat = oxygen saturation in the left atrium.

110/70 mmHg and weight 53 kg. The lung fields were clear to auscultation. Cardiovascular examination revealed a slight RV heave, an accentuated P2, and a grade I–II/VI pulmonary systolic murmur. No signs of right ventricular failure were detected. Laboratory tests revealed a microcytic anaemia with a haemoglobin concentration of 97 $g \cdot L^{-1}$. Urinalysis and culture demonstrated a urinary tract infection due to E. Coli. The ECG showed normal sinus rhythm with right axis deviation, right atrial hypertrophy and RV hypertrophy and the echocardiogram showed an LV of normal size and systolic function. The RV was dilated, with septal shift in both systole and diastole. The pulmonary artery was also dilated and peak pulmonary systolic pressures were estimated at 50–60 mmHg. Mild tricuspid regurgitation was noted. No shunting across the PFO was demonstrated. A chest radiograph was not obtained.

Obstetrical management consisted of bedrest, limited physical activity, and nocturnal oxygen. Consultations were obtained with cardiology, anaesthesia, perinatology, and the intensive care unit (ICU) physician. At 36 wk gestation an amniocentesis was performed; the L/S ratio suggested fetal lung maturity. A conference was held to allow all the consultants to voice their opinions and concerns. After this, the patient's obstetrician decided to perform an elective Caesarean section in two days. However, the afternoon after amniocentesis the patient went into labour spontaneously. The obstetrician elected to proceed with Caesarean section that evening.

Two hours before the Caesarean section a 14-gauge peripheral catheter was inserted in a left arm vein and an epidural catheter was placed at the $L_{2,3}$ interspace. The patient was sedated with morphine 6 mg and scopolamine 0.3 mg *im* before transfer to the operating room. Oxygen 3 $L \cdot min^{-1}$ by nasal prongs was begun at the time of premedication. Once on the operating table, the patient was placed supine with 15 degrees left uterine displacement. The patient's mother was present throughout the procedure. Intravenous fentanyl was given incrementally to 100 μg for further sedation. Under local anaesthesia, a radial arterial catheter and an oximetric pulmonary artery catheter (PAC) were inserted. Table II shows the initial haemodynamic information and results of arterial blood gas analysis. Care was taken to avoid the injection of air in the intravenous lines because of the possibility of paradoxical embolus. Ampicillin and gentamicin were administered according to standard regimens as prophylaxis against bacterial endocarditis. A test dose of 3 ml lidocaine 1.5% with epinephrine 1/200,000 was given through the epidural catheter. This was followed by lidocaine 2% in 3 ml increments over 30 min for a total volume of 23 ml lidocaine (8.1 $mg \cdot kg^{-1}$). The patient also received epidural fentanyl 100 μg. Forty minutes after the test dose, epidural blockade to T_5 bilaterally was detected. An increase in cardiac output (CO) and a decrease in systemic vascular resistance (SVR) were observed. The systemic and pulmonary artery pressures remained stable and the mixed venous oxygen saturation remained at about 80%. An attempt was made to keep *iv*

TABLE II Intraoperative arterial blood gas analysis and haemodynamic variables

Initial ABG in the OR:
pH 7.39 PCO_2 33 PO_2 205 HCO_3^- 20 BE −4

Variable	*Pre-induction*	*T_5 block*	*Postpartum*
HR	56	66	66
BP	127/67	151/67	157/70
MAP	87	88	95
PAP	71/30	86/40	86/38
Mean PAP	46	59	57
CVP	1	3	4
CI	3.38	4.78	5.00
SVRI	2035	1420	1024
LVSWI	71.4	86.7	97.8
RVSWI	37.7	58.2	58.7

Pre-induction = before epidural block, T_5 Block = after attainment of a T_5 epidural block, postpartum = after delivery of the infant, HR = heart rate ($beats \cdot min^{-1}$), BP = blood pressure (mmHg), MAP = mean arterial pressure (mmHg), PAP = pulmonary artery pressure (mmHg), Mean PAP = mean pulmonary artery pressure (mmHg), CVP = central venous pressure (mmHg), CI = cardiac index ($L \cdot min^{-1} \cdot m^{-2}$), SVRI = systemic vascular resistance index ($DE \cdot cm^{-5} \cdot m^{-2}$), LVSWI = left ventricular stroke work index ($g \cdot m^{-1}$), RVSWI = right ventricular stroke work index ($g \cdot m^{-1}$).

TABLE III Haemodynamic data in the intensive care unit postoperatively

Variable/Time	*0020*	*0326*	*0600*
HR	59	57	65
BP	110/74	138/75	137/72
MAP	91	98	93
PAP	74/33	82/26	90/35
Mean PAP	54	50	56
CVP	4	0	1
PCWP	10	7	8
CI	3.93	3.49	3.21

Time = actual time (patient arrived in the ICU at 2330 hr), HR = heart rate (beats $\cdot$ min^{-1}), BP = blood pressure (mmHg), MAP = mean arterial pressure (mmHg), PAP = pulmonary artery pressure (mmHg), Mean PAP = mean pulmonary artery pressure (mmHg), CVP = central venous pressure (mmHg), PCWP = pulmonary capillary wedge pressure (mmHg), CI = cardiac index (L $\cdot$ min^{-1} $\cdot$ m^{-2}).

fluid administration to a minimum. Intraoperative analgesia was excellent and surgery proceeded uneventfully. A 3020 g female infant was born with Apgar scores of nine and nine at one and five minutes. The cord pH was 7.30. The baby did not require naloxone or assistance with ventilation. After delivery an infusion of oxytocin 20 u $\cdot$ L^{-1} in lactated Ringer's solution was started at a rate of 200 ml $\cdot$ hr^{-1}. Epidural morphine 3 mg was given for postoperative analgesia. Total blood loss was estimated at 300 ml and the total volume of *iv* fluid administered was 1100 ml. The patient was transferred to the ICU after surgery for monitoring. Her course was uneventful (Table III) and she went to the postpartum ward the next day. Six days later she was discharged home feeling weak but otherwise well. At four months postpartum she tires easily and continues to feel weak but has not required hospitalization.

Discussion

Obstetrical management of patients with primary PH frequently involves termination of the pregnancy. This has been reported as late as 20 wk gestation.[3] Due to our patient's presentation at 25 wk, stability of the pulmonary hypertension, and adequate fetal growth, the obstetrician estimated that the pregnancy could be continued with no additional increase in risk. The method of delivery of these women is controversial but most previous reports describe vaginal delivery[4–9] (Table IV). We believe that the choice between vaginal delivery and Caesarean section[10] should be made primarily for obstetrical reasons. However, there are several advantages to a planned caesarean delivery. Coordination between the obstetrician, the anaesthetist and the ICU is facilitated; appropriate monitoring can be instituted on an elective basis; and a bed in the ICU can be assured.

When vaginal delivery is chosen, careful consideration of induction[4] versus spontaneous delivery[5–9] is required. In particular, the physiological effects of oxytocin and prostaglandins must be reviewed. The vasodilator properties of oxytocin can lead to hypotension with a compensatory tachycardia. Patients with PH and limited cardiac reserve may not tolerate these haemodynamic changes. Oxytocin can also lead to water retention (vasopressin-like effect). By avoiding the use of hypotonic intravenous solutions and high doses of oxytocin, water retention can be minimized. Prostaglandins, most commonly E2 (PGE2) and F2α (PGF2α) are often used to "ripen the cervix".[11] Both PGE2 and PGF2α can cause nausea, vomiting, diarrhoea, restlessness, tachypnoea, tachycardia and pyrexia. Haemodynamic effects of PGE2 include decreased SVR and mean arterial pressure (MAP), increased CO and HR, and no change in PVR. Prostaglandin F2α can cause increased MAP, HR, CO, and PVR. Bronchoconstriction can occur with PGF2α. For these reasons prostaglandins are best avoided in patients with PH. With care, oxytocin can be used safely for induction and/or augmentation of labour.

Mangano[12] has outlined the important principles of the management of patients with primary PH for delivery. The first is to define the severity of the pulmonary hypertension. Thus, invasive monitoring of both systemic and pulmonary pressures is advocated. After placement of a pulmonary artery catheter, Roessler and Lambert[10] suggested testing the response of the pulmonary circulation to fluid challenge and vasoactive drugs. We believe, however, that the risks to both mother and fetus posed by trials of fluids and vasoactive medications outweigh the potential benefits. The other principles suggested by Mangano and supported by Joyce and Palacios[13] are: (1) to avoid increases in PAP or PVR, (2) to avoid marked increases or decreases in RV preload, (3) to maintain LV afterload and (4) to maintain RV contractility.

Premedication with morphine 0.1 mg $\cdot$ kg^{-1} and scopolamine 6 μg $\cdot$ kg^{-1} *im* was given one hour before transfer to the operating room. We postulated that avoiding maternal anxiety and the associated stress response was more important than possible neonatal depression. It was anticipated that neonatal respiratory depression, if present, could be managed with pulmonary ventilation and naloxone. Furthermore, the doses of morphine and scopolamine used were unlikely to cause hypercapnia and acidosis, factors known to aggravate PH. In addition, the patient received supplemental oxygen at the time of premedication to avoid hypoxaemia, which is another potent pulmonary vasoconstrictor.

The presence of a PFO necessitated concern for both inter-atrial shunting and paradoxical embolization. Central venous pressure (CVP) was monitored and kept at 0–5

TABLE IV Previous reports of outcome in patients with pulmonary hypertension and pregnancy

Author	*PH*	*Mat age*	*Gest age*	*Mode del*	*Anaesth*	*Maternal outcome*
Nelson[2]	Primary	20	32	Vaginal	Epidural	Alive
Slomka[5]	Primary	32	38	Vaginal	Epidural	Alive
Power[6]	Primary	27	38	Vaginal	Epidural	Died
Roessler[10]	Primary	21	30	C/S	Epidural	Died
Roberts[9]	Primary	26	34	C/S	G.A.	Alive
Robinson[4]	Secondary	28	35	Vaginal	Epidural	Died
Sorensen[7]	Secondary	23	37	Vaginal	Epidural	Died
Abboud[8]	Secondary	34	39	Vaginal	Intrathecal	Died
Roberts[9]	Secondary	31	36	C/S	Epidural	Died

PH = primary or secondary pulmonary hypertension, Mat age = maternal age at delivery (yr), Gest age = gestational age at delivery (wk), Mode del = mode of delivery, C/S = Caesarean section, Anaesth = type of anaesthesia used, Outcome = maternal outcome (Alive = alive at 2 months postpartum, Died = death within 6 weeks postpartum).

mmHg. Care was taken to ensure that no air entered the *iv* lines and the patient was positioned 5–10° head-up during surgery.[14,15]

We chose epidural anaesthesia because we considered it to be a safe alternative to general anaesthesia. The stress of laryngoscopy and tracheal intubation, and the potential problems of positive pressure ventilation were avoided. Epidural anaesthesia was induced very gradually. Epinephrine was used only in the test dose as it can cause a more profound sympathetic block and a resultant decrease in SVR.[16] Furthermore, we were concerned about the α_1 effects of epinephrine on the pulmonary circulation.[16] Fentanyl was added to the epidural lidocaine to improve the quality of the block obtained.[17,18] The slow onset of epidural blockade to the T_5 level over 40 min may have allowed a compensatory increase in cardiac index (CI) and maintenance of MAP (Table II). Right ventricular preload (CVP) was maintained to keep RV filling optimal. Although SVRI decreased, MAP, and therefore myocardial perfusion pressure, was maintained. The PAC did not wedge easily. Repeated attempts to obtain a wedge pressure measurement were avoided because of the increased risk of pulmonary artery rupture in patients with PH.[19] Thus the PVR could not be calculated. After delivery of the infant, an intravenous solution containing oxytocin 20 u · L^{-1} was administered slowly in an attempt to minimize the vasodilating effects of oxytocin.

The choice of epidural anaesthesia for patients with PH is controversial. Mangano[12] advised general anaesthesia with an inhalational induction. Joyce and Palacios[13] advocated general anaesthesia with ketamine induction and avoidance of nitrous oxide. Had we chosen or been forced to administer general anaesthesia, we would have induced anaesthesia with alfentanil. Batson *et al.* have described the use of alfentanil in a patient with mitral stenosis and PH.[20] A high-dose narcotic anaesthetic may provide maximum haemodynamic stability and minimize response to stimuli such as tracheal intubation. We planned to induce general anaesthesia if: (1) we were unable to place the epidural catheter, (2) the patient would not tolerate insertion of the PAC, (3) satisfactory epidural blockade could not be obtained or (4) the patient became uncooperative. While we agree with the basic principles outlined by Mangano mentioned above, we also believe that epidural anaesthesia has a role and can be used safely in selected patients. Our patient had survived five years with the diagnosis of PH (and the PH had decreased over the years) and a co-existent PFO.[21,22] These factors probably gave her a better prognosis than many other patients with PH. The risks and benefits of epidural versus general anaesthesia must be assessed on an individual basis.

In summary, this report demonstrates that Caesarean section can be performed under epidural anaesthesia in a patient with pulmonary hypertension, cardiomyopathy and a patent foramen ovale. Standard text books[12,13] advocate general anaesthesia, but most recent case reports describe the use of epidural anaesthesia for these patients. After thorough assessment, and consideration of each patient's pathology, epidural anaesthesia may be safely performed in selected patients.

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