Clinical Reports

David R. Gambling MB BS DRCOG FRCPC,
Mary Louise Flanagan MD FRCPC,
Victor F. Huckell MD B SC FRCPC FACC,
S. Brian Lucas MD FRCPC FACA, James H. K. Kim MD FRCPC

Anaesthetic management and non-invasive monitoring for Caesarean section in a patient with cardiomyopathy

This paper describes the anaesthetic management of a 29-year-old woman for an elective repeat Caesarean section. A diagnosis of peripartum cardiomyopathy (PPCM) had been made after her first delivery by Caesarean section three years earlier. Although the patient was currently asymptomatic, recent echocardiography demonstrated persistent left ventricular dilatation. The choice of haemodynamic monitors and the clinical significance of PPCM in this situation are discussed.

Peripartum cardiomyopathy (PPCM) is defined as the development of heart failure in the last trimester of pregnancy or up to the sixth postpartum month. There is absence of demonstrable heart disease prior to the last trimester and no aetiology can be found.[1,2]

The major concerns in a patient with PPCM during Caesarean section are to optimise fluid administration and to avoid factors which may cause myocardial depression. Appropriate haemodynamic monitors should be employed and chosen according to the clinical condition of each patient. Our criteria for selecting a non-invasive monitoring technique are described in the following Case Report.

Key words

From the Departments of Anaesthesia and Cardiology, University of British Columbia and Grace Hospital, Vancouver, British Columbia.

Address correspondence to: Dr. D. R. Gambling, Department of Anaesthesia, Grace Hospital, 4490 Oak Street, Vancouver, British Columbia V6H 3V5.

Case report

A 29-year-old G2 P1 presented for an elective repeat Caesarean section at term. Past obstetric history revealed a first pregnancy three years earlier, complicated by pre-eclampsia which necessitated an urgent delivery by Caesarean section, under epidural anaesthesia. After delivery of a healthy male infant, general anaesthesia was induced, as the patient had a grand mal seizure, for undetermined reasons. Twenty-four hours after delivery, after uneventful recovery from the anaesthetic, the patient developed pulmonary oedema which responded to medical therapy. Chest x-ray at that time revealed global cardiac enlargement and an echocardiogram showed a dilated, hypocontractile left ventricle with a thin, hypokinetic interventricular septum. No cause for the sudden onset of heart failure could be determined and the patient was discharged from hospital on the seventh postoperative day, under the care of a cardiologist.

In the interim, the patient remained asymptomatic but serial echocardiograms taken during this period demonstrated persistent left ventricular dilatation and mild impairment of myocardial contractility. Her ECG was normal apart from frequent asymptomatic uniform ventricular premature depolarisations. Physical examination was unremarkable and she was normotensive.

The repeat Caesarean section was performed under epidural anaesthesia using incremental boluses of carbonated lidocaine (two per cent) with 1:400,000 epinephrine to a total of 15 ml·min⁻¹ via an epidural catheter inserted at the L_{2-3} interspace. She was given 6 L·min⁻¹ of oxygen by mask and positioned with a left lateral tilt of the uterus.

Prior to this, a Cordis® introducer had been placed via the right internal jugular vein. The initial CVP was 4 cm H_2O. Routine monitors included ECG (modified V_5 configuration) and an automatic blood pressure device. In our institution non-invasive haemodynamic data are obtained from an impedance cardiograph, the NCCOM3

TABLE I   Haemodynamic parameters from the non-invasive cardiac output monitor during administration of epidural anaesthesia

	Mean BP (mmHg)	Systemic vascular resistance (dyn·sec·cm⁻⁵)	Cardiac output (L·min⁻¹)	Cardiac Index (L·min⁻¹·m⁻²)	CVP cmH₂O
Before epidural (supine with wedge)	77	730	8.4	5.0	+4
T₅ sensory block	81	770	8.4	5.0	+4
12 minutes after delivery (Ephedrine 5 mg IV given)	57	560	8.3	4.9	+2
13 minutes after delivery (Ephedrine 5 mg IV given)	50.3	550	7.4	4.4	+2
18 minutes after delivery	61.7	600	8.2	4.9	+4
In postanaesthesia room 37 min post partum	66.7	725	7.3	4.35	+6
In postanaesthesia room 60 min post partum	90	1050	6.9	4.15	+8

(BoMed)®, which provides us with continuous values for stroke volume, cardiac output and other parameters from which we can derive systemic vascular resistance, cardiac contractility and an index of thoracic extravascular fluid.[3-5]

Surgery and anaesthesia were uneventful, although the mean BP fell approximately ten minutes after delivery due to a reduction in SVR, with no significant fall in cardiac output. This responded to intravenous ephedrine in two 5 mg boluses and an increase in the rate of infusion of intravenous saline to achieve a CVP of 8 cm H_2O (Table I). The patient made a good recovery postoperatively and a five-month follow-up showed her to be clinically well with normal LV dimensions on echocardiogram.

Discussion
Four excellent reviews of PPCM have been published in the last three years.[1,2,6,7] The incidence of PPCM is between 1:1300 to 1:4000 live births in the U.S.A., with an overall mortality rate of 25–50 per cent.[2] Morbidity and mortality rates can be even higher in subsequent pregnancies if the LV dimensions have not normalised between six to 12 months post partum.[8,9] Those patients with a return to normal LV size have only a 25 per cent chance of developing pulmonary oedema in subsequent pregnancies, with a negligible mortality rate.[8] Those patients with persistent cardiomegaly at the onset of their next pregnancy show a 60 per cent mortality and 80 per cent morbidity rate.[1] Even though most of these figures come from studies looking at patients from low socioeconomic groups with inadequate antenatal care, the numbers are comparable with recently published data of middle class women who had excellent antenatal care and cardiological evaluation.[7]

In light of these facts it was felt that our patient would be at risk throughout the delivery period. Most pregnant women show a volume-loading phenomenon of pregnancy seen as right ventricular dilatation on echocardiogram, but significant left ventricular dilatation, especially

in the late post partum period, is considered to be abnormal.

A diagnosis of PPCM can only be made after excluding the other causes of acute heart failure. The differential diagnosis includes non-cardiac causes such as toxaemia (pre-eclampsia),[10] beta₂-adrenergic tocolysis,[11] pulmonary embolism, amniotic fluid embolism[12] and aspiration pneumonitis. Cardiac causes include decompensated valvular and subvalvular disease, myocardial ischaemia and infarction, fluid overload and cardiomyopathy.

A decision to use regional anaesthesia for surgery was made in order to avoid potentially cardiodepressant general anaesthetic agents and perhaps allow an earlier diagnosis of cardiovascular compromise in the awake patient.

The choice of a non-invasive technique for following cardiac output was made because of concern for subjecting an otherwise healthy mother and her fetus to the potential risks of invasive monitoring with a Swan Ganz catheter (SGC).[13-15]

Adequate management of the haemodynamic alterations that may occur during surgery requires vigilant attention to patient response and appropriate monitoring aids. In this case, the patient with only mild impairment of LV function can be satisfactorily monitored non-invasively and the trends in cardiac output provide us with an excellent overall picture of cardiac function. We do not feel it is necessary to "fine tune" or maximise cardiac output by manipulating pulmonary capillary wedge pressure (PCWP) if our baseline cardiac output is satisfactory and the patient falls within the categories listed in Table II.

Elective use of invasive monitoring is justified in a symptomatic patient with an elevated JVP, third and fourth heart sounds, orthopnoea, PND or shortness of breath at rest, with clinical evidence of a low cardiac output or echocardiographic evidence of significant myocardial depression (poor contractility, LV wall motion abnormalities) (Table III).

TABLE II Indications for non-invasive monitoring in patients with PPCM

Asymptomatic
Physical examination normal
Mild impairment only of LV contractility by echocardiography
Relative contraindication to pulmonary artery catheter
– Coagulopathy
– Uncontrolled ventricular dysrythmias
– Lack of experienced personnel and equipment
– Pulmonary/tricuspid valve stenosis or prosthesis
– Transvenous temporary pacemaker *in situ*

Non-invasive monitoring can be achieved using a Doppler technique but this is not without its technical difficulties.[16] The impedance technique produces reliable trends in various haemodynamic parameters and has allowed confident, successful management of severe pre-eclampsia in our institution. This approach is far more acceptable to our patients and when they are asymptomatic, well controlled epidural anaesthesia and non-invasive monitoring provides optimal management with a minimal risk/benefit ratio.

Toxaemia of pregnancy can complicate 15–30 per cent of pregnancies associated with PPCM which is an incidence four times higher than in the normal pregnant population.[17] It is not known whether this relationship is coincidental or cause-and-effect, but it is likely to worsen the prognosis of PPCM due to an increased afterload further straining an already weakened myocardium. The presence of toxaemia in a patient with PPCM will thus favour an invasive approach, using a SGC. Even so, a study by Benedetti has shown that PCWP correlates well with CVP when the latter is less than $6 \, cm \, H_2O$.[10] This patient had an uneventful course, but had decompensation

TABLE III Indications for invasive monitoring in patient with PPCM

History
Dyspnoea At Rest Or On Minimal Exertion
PND/Orthopnoea
Severe Chest Pain

Examination
Elevated JVP (CVP > $10 \, cm \, H_2O$)
Gallop Rhythm
Toxaemia of Pregnancy (Moderate to Severe)

CXR
Radiological Evidence of Pulmonary Extravascular Fluid

ECG
Evidence of Myocardial Ischaemia

Echocardiography
Severe Impairment of Myocardial Contractility
LV Wall Motion Abnormality

occurred we had access for Swan Ganz catheter insertion via the Cordis introducer, enabling us to follow PCWP valves.

Pulmonary oedema in the peripartum period should never be treated lightly. All pregnant patients who have unexplained congestive heart failure should receive appropriate therapy and, when stable, undergo diagnostic echocardiography and complete assessment by a cardiologist. Some of the causes are treatable and some patients will show mural thrombi requiring long-term anticoagulation.[18]

A recent report describing two cases of pulmonary oedema associated with Caesarean section reminds us that the risk to our patients with PPCM is real.[19] It must be stressed that invasive monitoring is not always required and each case must be assessed individually and appropriate monitors selected accordingly.

It is of interest that five months after her second delivery the long standing ventricular dilatation had returned to normal. We are not aware of any report of a successful delivery significantly reducing LV size as rapidly as that seen in this patient.

Conclusion

PPCM is a diagnosis made by exclusion but should never be regarded as insignificant. Follow-up by a cardiologist with serial echocardiography is essential. Anaesthetic management and appropriate haemodynamic monitoring is aimed at optimising fluid therapy, avoiding cardiodepressant drugs and providing a minimal risk/benefit ratio to both mother and fetus with a well controlled epidural anaesthetic.

Choice of monitors will depend on the severity of the myocardial disease and degree of symptomatology. Persistent LV dilatation at the onset of subsequent pregnancies carries a higher morbidity and mortality incidence. If the patient is asymptomatic, non-invasive haemodynamic monitoring can provide a safe, reliable means of following the cardiopulmonary status of the patient.

References

1 *Veille JC.* Peripartum cardiomyopathies: a review. Am J Obstet Gynecol 1984; 148: 805–18.

2 *Homans DC.* Peripartum cardiomyopathy: current concepts. N Engl J Med 1985; 312: 1432–7.

3 *Kubicek WE et al.* Development and evaluation of an impedance cardiac output system. Aerospace Med 1966; 37: 1208.

4 *Milsom I, Forssman L, Biber B, Dottori O, Sivertsson R.* Measurement of cardiac stroke volume during Caesarean section: a comparison between impedance cardiography and the dye dilution technique. Acta Anaesthesiol Scand 1983; 27: 421–6.

5 *Secher NJ, Amsbo P, Andersen LH, Thomsen A.*
Measurement of cardiac stroke volume in various body
positions in pregnancy and during Cesarean section. A
comparison between thermodilution and impedance
cardiography. Scan J Clin Lab Inves 1979; 39: 569–76.

6 *Julian DG, Szekely P.* Peripartum cardiomyopathy. Prog
Cardiovasc Dis 1985; 27: 223–40.

7 *O'Connel JB, Costanzo-Nordin MR, Subramanian R et al.*
Peripartum cardiomyopathy: clinical, hemodynamic,
histologic and prognostic characteristics. J Am Col Cardiol
1986; 8: 52–6.

8 *Demakis JA, Rahimatooloa SH, Sutton GC et al.* Natural
Course of peripartum cardiomyopathy. Circulation 1971;
44: 1053–61.

9 *Seftel H, Susser M.* Maternity and myocardial failure in
African women. Br Heart J 1961; 23: 43.

10 *Benedetti TJ, Kates R, Williams V.* Hemodynamic
observations in severe pre-eclampsia complicated by
pulmonary edema. Am J Obstet Gynecol 1985; 152:
330–4.

11 *Hawker F.* Pulmonary oedema associated with B_2
sympathomimetic treatment of premature labour. Anaesth
Intensive Care 1984; 12: 143–51.

12 *Mainprize TC, Maltby JR.* Amniotic fluid embolism: a
report of four probable cases. Can Anaesth Soc J 1986;
33: 382–7.

13 *Elliot CG, Zimmerman GA, Clemma TP.* Complications
of PA catheterisation in care of critically ill Patients.
Chest 1979; 76: 647–52.

14 *Barash PA.* Catheter induced PA perforation. J Thor
Cardiovasc Surg 1981; 82: 5–12.

15 *Nadequ S, Noble W.* Misinterpretation of pressure
measurements from the pulmonary artery catheter. Can
Anaesth Soc J 1986; 33: 352–63.

16 *Mark JB, Steinbrook RA, Gugino LD.* Continuous non-
invasive monitoring of cardiac output with esophageal
Doppler ultrasound during cardiac surgery. Anesth Analg
1986; 65: 1013–20.

17 *Silverman RI, Ribner HS.* Peripartal cardiomyopathy. *In*:
Cardiac Problems in Pregnancy. New York, Alan R
Riss Inc Publ, 1982.

18 *Hodgman MT, Pessin MS, Homans DC et al.* Cerebral
embolism as the manifestation of peripartum
cardiomyopathy. Neurology 1982; 32: 668.

19 *Malinow AM, Butterworth JF, Johnson MD et al.*
Peripartum cardiomyopathy presenting at Caesarean
delivery. Anesthesiology 1985; 63: 545–47.

Résumé

*Cette étude décrit la conduite anesthésique chez une femme de
29 ans subissant une deuxième césarienne élective. Trois ans
auparavant on avait diagnostiqué une cardiomyopathie péripar-
tum (CMPP) à la suite de son premier accouchement par
césarienne. Bien que la patiente fut présentement asymptomati-
que, une échocardiographie récente démontrait une dilatation
ventriculaire gauche persistante. On discute du choix de
moniteurs hémodynamiques et de l'importance clinique de la
CMPP dans cette situation.*