International Journal of Obstetric Anesthesia (2014) **23**, 66–85

CASE REPORTS

ELSEVIER
www.obstetanesthesia.com

Peripartum cardiomyopathy: postpartum decompensation and use of non-invasive cardiac output monitoring

G. Lorello,[a] J. Cubillos,[a] M. McDonald,[b] M. Balki[a]
[a] *Department of Anesthesia and Pain Management,* [b] *Department of Cardiology, Mount Sinai Hospital, Toronto, ON, Canada*

ABSTRACT
The utility of a non-invasive cardiac output monitor (NICOM™) in guiding the peripartum management and identification of postpartum complications in a patient with severe peripartum cardiomyopathy is reported. A 31-year-old nulliparous woman at 35 weeks of gestation presented with a three-week history of worsening dyspnea and progressive functional deterioration. A transthoracic echocardiogram showed severe left ventricular systolic dysfunction with an ejection fraction <20%. Cardiac status was monitored using NICOM™ during labor and delivery. The baseline values were: cardiac output 5.3 L/min, total peripheral resistance 1549 dynes.sec/cm^5, stroke volume 42.1 mL and stroke volume variation 18%. She received early epidural analgesia during labor, titrated slowly with a loading dose of 0.0625% bupivacaine 10 mL and fentanyl 25 μg, followed by patient-controlled epidural analgesia (0.0625% bupivacaine with fentanyl 2 μg/mL, infusion at 10 mL/h, bolus dose 5 mL and lockout interval 10 min). After epidural drug administration, total peripheral resistance decreased, cardiac output increased, and satisfactory analgesia was obtained. She had an uneventful vaginal delivery with a forceps-assisted second stage after prophylactic administration of furosemide 20 mg. NICOM™ was discontinued after delivery. Fifteen hours post-delivery, the patient developed cardiogenic shock, which resolved after aggressive therapy with inotropes and furosemide. NICOM™ can be used to guide treatment during labor and delivery in patients with critical peripartum cardiomyopathy. We suggest that use of NICOM™ be extended into the postpartum period to detect signs of cardiac decompensation in such patients.

Keywords: Peripartum cardiomyopathy; Cardiogenic shock; NICOM™

Introduction

Peripartum cardiomyopathy (PPCM) is characterized by left ventricular systolic dysfunction (left ventricular ejection fraction (LVEF) <45%) typically presenting within the last month of pregnancy and up to five months postpartum, in the absence of previously known heart disease.[1] The incidence of PPCM in the USA is 1 in 2289–4000 live births.[2] Its etiology is unknown, although genetic and environmental factors may play a role.[3] The pathophysiology includes deteriorating systolic function with a reduced LVEF and an increased risk of congestive heart failure, thromboembolism, arrhythmias, and sudden cardiac death.[4] While nearly 50% of patients recover cardiac function within 3–6 months, PPCM may recur in subsequent pregnancies. Overall mortality exceeds 10%.[3]

The purpose of this case report is to demonstrate the use of a non-invasive cardiac output monitor (NICOM™, Cheetah Medical Inc, Portland, OR, USA) as a supplement to clinical examination, and to discuss the management and postpartum complications encountered in a case of PPCM.

Case report

A 31-year-old nulliparous woman at 35 weeks of gestation (92 kg, 157 cm, body mass index 37.4 kg/m^2) presented with a three-week history of worsening dyspnea, orthopnea, paroxysmal nocturnal dyspnea and progressive functional deterioration consistent with New York Heart Association (NYHA) class IV symptoms. She had no significant past medical history. Pregnancy had been complicated in the third trimester by

Accepted October 2013
Correspondence to: Dr. Mrinalini Balki, Department of Anesthesia and Pain Management, Mount Sinai Hospital, 600 University Ave, Toronto, ON, Canada M5G 1X5.
E-mail address: mrinalini.balki@uhn.ca

well-controlled insulin-dependent gestational diabetes mellitus.

Evaluation of dyspnea included a ventilation/perfusion scan, reported as low risk for pulmonary embolism, and duplex compression ultrasonography of the legs which revealed no evidence of deep vein thrombosis. Subsequently, transthoracic echocardiography (TTE) showed severe global left ventricular systolic dysfunction, with a LVEF of <20%, a left ventricular end diastolic diameter of 6.4 cm (normal <5.7 cm) and a dilated left atrium. Liver enzymes were mildly elevated, and an abdominal ultrasound revealed a diffuse fatty liver with no morphological stigmata of chronic liver disease. She was transferred to the coronary care unit (CCU) where she was normotensive but in frank pulmonary edema. Intravenous furosemide and digoxin and a nitroglycerin patch were given with good initial improvement and hemodynamic stability. The next morning she went into spontaneous labor and was transferred to the delivery unit.

Clinically, she was not in respiratory distress. Examination revealed a blood pressure (BP) of 133/88 mmHg, heart rate (HR) of 126 beats/min, respiratory rate (RR) of 18 breaths/min, and oxygen saturation of 95% on supplemental oxygen 15 L/min via a face mask. Arterial blood gases were unremarkable: pH 7.41, $PaCO_2$ 32 mmHg, PaO_2 111 mmHg, HCO_3 20 mEq/L, base excess −4.6 mmol/L, SaO_2 98%. Airway examination revealed very edematous soft tissues, unrestricted atlanto-axial extension in a short neck, thyromental distance of two finger breadths, and poor mandibular subluxation; her Mallampati score was IV. She was given another intravenous bolus of furosemide 40 mg.

A multidisciplinary plan for early titrated epidural analgesia with an assisted second stage vaginal delivery was made. A repeat TTE was essentially unchanged and ruled out an intra-cardiac thrombus (Appendix A).

An 18-gauge intravenous cannula and a 20-gauge intra-arterial cannula were inserted. A peripherally-inserted central cannula was placed under ultrasound guidance since conventional cannulation was difficult due to edematous tissues. In addition to standard monitoring, NICOM™ was used. Baseline values were: BP 133/88 mmHg (mean arterial pressure 103 mmHg), HR 126 beats/min in sinus rhythm, cardiac output (CO) 5.3 L/min, total peripheral resistance (TPR) 1549 dynes.sec/cm^5, stroke volume (SV) 42.1 mL and stroke volume variation (SVV) 18% (SVV <10% is most likely not responsive to fluids; >12% is most likely responsive to fluids). Although she became increasingly short of breath, her hemodynamic status was unchanged from baseline. A lumbar wedge was used to promote left uterine displacement and limit aortocaval compression. NICOM™ data were recorded at 1-min intervals until the end of her stay on the delivery unit (Figs. 1 and 2).

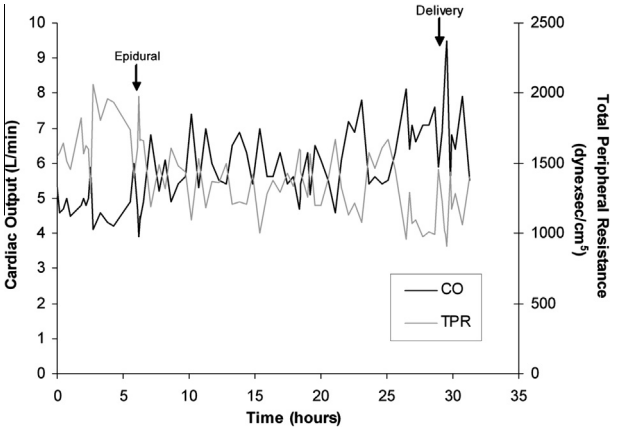

Fig. 1 Cardiac output (CO) and total peripheral resistance (TPR) according to NICOM™ measurements as a function of time during labor. Epidural insertion and delivery are demonstrated by the arrows.

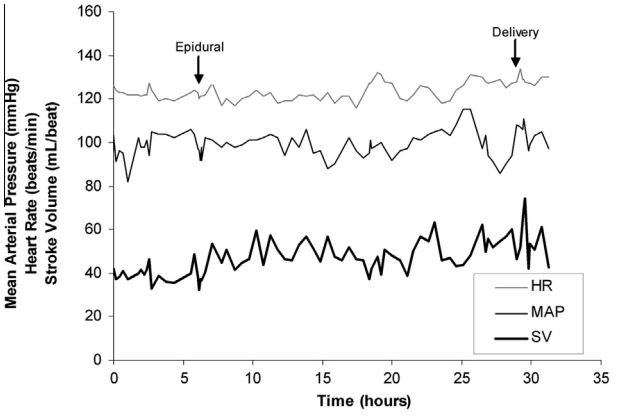

Fig. 2 Mean arterial pressure (MAP), heart rate (HR), and stroke volume (SV) according to NICOM™ measurements as a function of time during labor. Epidural insertion and delivery are demonstrated by the arrows.

The fetus was monitored with continuous cardiotocometry.

An epidural catheter was placed at the L3-4 interspace using ultrasound guidance. Correct placement was tested using the epidural electrical stimulation test, which consists of stimulating the epidural catheter using an electric current at 1–10 mA to elicit a motor response.[5] To limit changes in TPR, a 10 mL loading dose of 0.0625% bupivacaine with fentanyl 25 μg was titrated over 15 min, followed by patient-controlled epidural analgesia using 0.0625% bupivacaine and fentanyl 2 μg/mL, with an infusion rate of 10 mL/h, bolus dose of 5 mL, a lockout interval of 10 min and a 4-hourly limit of 80 mL.

After epidural drug administration, TPR decreased (13.9%), CO increased (13.8%), SV increased (18%),

and HR and SVV were unchanged compared to baseline on admission to the labor floor. Labor was augmented with an oxytocin infusion (15 U in 0.9% saline 250 mL, rate incrementally increased from 2 to 14 mU/min according to local protocol, total dose 4361 mU over 7 h 45 min). NICOM™ values fluctuated throughout labor. Boluses of intravenous furosemide 20–40 mg (total 160 mg) were given based on clinical judgment. Intravenous fluids were given at variable rates (average 75 mL/h) and were decreased or stopped if worsening pulmonary edema was detected clinically. While in the delivery unit, total fluid input was 2592 mL and total urine output was 3575 mL.

Labor was uneventful and lasted 25 h. At full cervical dilation, a prophylactic dose of intravenous furosemide 20 mg was given and she was transferred to the operating room for a forceps-assisted vaginal delivery. A male infant weighing 1940 g, was delivered with Apgar scores of 6, 8, and 9 at 1-, 5-, and 10 min, respectively. Oxytocin infusion (20 U/L in 0.9% saline) was administered at the rate of 40 mU/min for 8 h (total volume 960 mL). TPR decreased (32.9%) and SV increased (54.1%), which led to a significant increase in CO (58.5%). Her HR, BP and SVV remained unchanged. These changes lasted for approximately 25 min, after which hemodynamic parameters returned to those observed before delivery. A second-degree vaginal laceration was repaired. Epidural morphine 2.5 mg was given for post-delivery pain relief.[6]

Before transfer to CCU for on-going monitoring, the epidural catheter and NICOM™ were removed. At our facility, NICOM™ is used on the delivery unit for patients with cardiac conditions and is not available for use in other hospital areas.

Fifteen hours later, while in CCU, her condition deteriorated with decreased urine output, decreased capillary refill, mottled skin and worsening pulmonary edema. A pulmonary artery (PA) catheter was inserted for invasive hemodynamic assessment (Figs. 3 and 4).

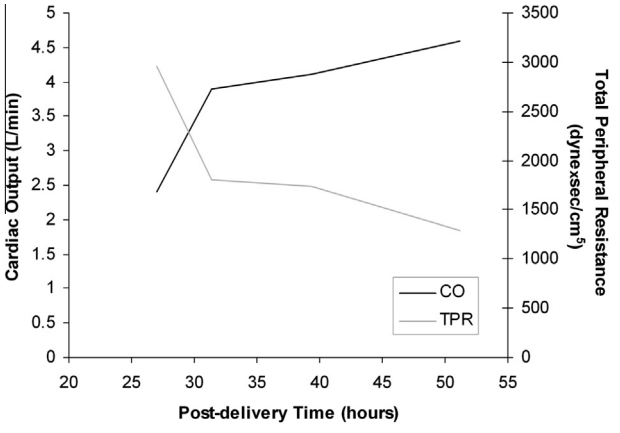

Fig. 3 Cardiac output (CO) and total peripheral resistance (TPR) according to pulmonary artery catheter measurements as a function of time after delivery.

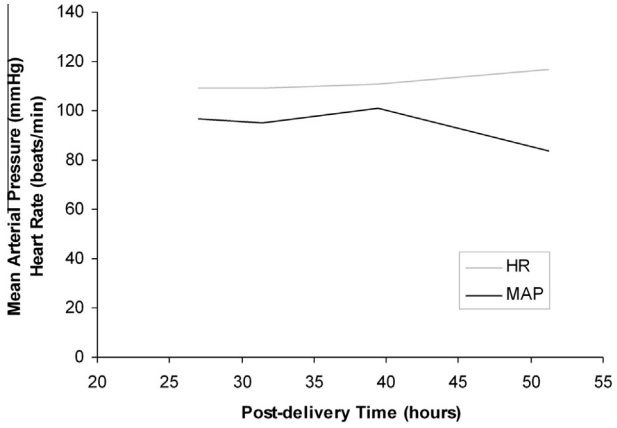

Fig. 4 Mean arterial pressure (MAP) and heart rate (HR) according to pulmonary artery catheter measurements as a function of time after delivery.

Her TPR had increased significantly to 191%, and CO decreased to 45%, of baseline on admission. Intravenous dobutamine 5 μg/kg/min was started. TTE was unchanged. Mechanical circulatory support with a short-term biventricular assist device was considered but her condition improved rapidly with inotropic support and intermittent intravenous furosemide. Within 24 h, oral vasodilator therapy with ramipril 5 mg twice daily was started, as were spironolactone 25 mg daily and digoxin 0.125 mg daily. Due to the risk of left ventricular thrombus, therapeutic anticoagulation with enoxaparin was started. She was discharged from the hospital 17 days after delivery.

A six-month follow-up TTE showed an LVEF of 20–29% with severe global hypokinesis, a mildly dilated left atrium, a hypokinetic right ventricle, a right ventricular systolic pressure of 38 mmHg, and no intra-cardiac thrombus. At eight months postpartum, she was asymptomatic while taking oral candesartan, metoprolol, spironolactone, digoxin, and furosemide. Despite good functional capacity (NYHA class I), persistent left ventricular systolic dysfunction and the potential for malignant ventricular arrhythmias may in due course indicate the need for insertion of a prophylactic defibrillator.[1]

Discussion

We employed NICOM™ in conjunction with clinical examination to monitor cardiodynamic parameters and guide treatment in a patient with severe PPCM undergoing labor and delivery.[7] NICOM™ has been previously validated in the obstetric population for continuous bedside hemodynamic monitoring.[8] For CO monitoring, it compares favorably to thermodilution techniques and is superior to bioimpedence.[9–11]

NICOM™ provides continuous non-invasive hemodynamic monitoring using bioreactance technology.

Bioreactance is based on an analysis of phase shifts that occur when an alternating current is applied to the thorax; pulsatile blood flow through the large thoracic arteries causes the amplitude of the applied thoracic voltage to change, leading to a phase shift between the applied current and the measured voltage. The phase shifts are strongly correlated with stroke volume and aortic blood volume changes, and are independent of artifacts caused by respiration phase, thoracic fluid, chest wall thickness, and other electrical equipment. In contrast, bioimpedance-based systems rely only on changes in signal amplitude and are subject to these artifacts.

In our patient, following epidural drug administration, TPR decreased and CO increased (Fig. 1), presumably due to an increase in SV since HR remained similar throughout labor (Fig. 2). SV changes are likely to reflect the balance between preload and afterload in the presence of depressed myocardial contractility. Titration of epidural drug doses permitted stability in MAP and HR, and avoided precipitous decreases in afterload that may occur after sudden sympathectomy. SVV, a measure of respiratory change in blood flow, assesses fluid responsiveness and its magnitude is highly dependent on fluid status.[12] It remained unchanged during labor, suggesting constant intravascular volume, and an appropriate balance between diuresis produced by furosemide and repletion via intravenous fluid administration. The postpartum changes in SVV are likely to have been caused by uterine autotransfusion and release of aortocaval compression, which may also have contributed to a spike in CO at delivery. Of note, the validity of SVV has not been established in spontaneously breathing patients, pregnancy or severe left ventricular dysfunction. Assessment of the intravascular volume status was challenging in our patient in view of the opposing effects of furosemide, promoting diuresis, and oxytocin infusion, promoting fluid retention and altering hemodynamics. Epidural morphine analgesia may have also contributed to the prolonged reduction in TPR by suppressing sympathetic nervous system stimulation secondary to pain.[6]

The postpartum course of patients with PPCM undergoing vaginal delivery is not well documented in the literature but is more frequently reported after cesarean delivery with spinal[13–15] or general anesthesia.[14,16] Approximately 15 h postpartum, the patient developed pulmonary edema and cardiogenic shock requiring inotropic support. We hypothesize that decompensation was secondary to interstitial fluid mobilization, sympathetic stimulation, possibly from pain, and loss of both the low resistance placental circulation and the vasodilatory effects of pregnancy. Treatment of pulmonary edema with furosemide, dobutamine and nitroglycerine had a gratifyingly dramatic response. Mechanical circulatory support was considered but, given the rapid response to dobutamine, was not required.

In the presence of severe left ventricular dysfunction, repeated TTE was performed in order to rule out an intra-cardiac thrombus and assess cardiac status. Pregnancy, being a pro-thrombotic state with depressed cardiac contractility and increased blood stasis, predisposes women to intra-cardiac thrombi,[13–15] especially if LVEF is <35%. Prognosis in PPCM worsens if the LVEF falls below 20% or if the LV diameter exceeds 6 cm.[17] Anticoagulation is indicated.[2]

Anesthetic considerations of sympathetic stimulation for labor and cesarean delivery are similar. In labor, an early, titrated epidural is desirable to prevent sympathetic nervous system stimulation secondary to pain and to prevent a precipitous drop in TPR. Balancing preload is critical as excessive volume can lead to pulmonary edema whereas acute intravascular depletion can result in decreased CO. For cesarean delivery, a denser block is needed, which can result in a significant decrease in TPR, and can be catastrophic in the presence of poor ventricular function. Careful titration of epidural anesthesia is essential to improve hemodynamic control. The epidural catheter also allows drug administration for postoperative pain management.

Uterotonic agents may cause considerable cardiovascular effects. A rapid intravenous bolus of oxytocin in healthy patients can result in a decrease in BP and TPR, and an increase in CO produced by an increase in HR and SV,[18] while doses larger than 45 mU/min may produce an antidiuretic effect.[19] These effects are poorly tolerated in patients with left ventricular dysfunction and can be minimized by administration of oxytocin in low doses and at a slower infusion rate titrated to effect.[20] Ergometrine, a potent vasoconstrictor that can induce coronary artery spasm,[21] and prostaglandin F2α, which may cause bronchospasm, pulmonary edema, ventilation–perfusion mismatch and hypoxemia,[22–24] should be used with extreme caution in hemodynamically compromised patients.

NICOM™ was stopped after delivery. Continuing its use postpartum may have allowed early detection of signs of heart failure but was not possible for logistic reasons. Bedside TTE, whose use has been described in pregnant women, was performed.[25,26] The PA catheter was inserted in CCU due to the presence of cardiogenic shock and assessment of the need for mechanical circulatory support. Studies have shown good correlation between PA catheter continuous cardiac output monitoring and the CO observed by NICOM™, with correlation coefficients between $r = 0.62$ and 0.78 and minimal bias as defined by Bland-Altman plots.[7,11,27]

This case describes the use of NICOM™ as an adjunct to clinical examination in a woman with severe PPCM during labor; although her condition deteriorated postpartum but responded with pharmacological

management. A literature review suggests that this is the first case to highlight the hemodynamic course using NICOM™ of a patient with critical PPCM during labor and second stage assisted vaginal delivery. Prolonged postpartum NICOM™ monitoring may be warranted to detect early signs of decompensation, and lead to prompt intervention and optimization of hemodynamic support.

Disclosure

The authors have no relevant conflicts of interest and no funding was provided.

Acknowledgements

The authors would like to thank Kristi Downey for her help with the graphical representation.

References

1. Howlett JG, McKelvie RS, Costigan J, et al. The 2010 Canadian Cardiovascular Society guidelines for the diagnosis and management of heart failure update: heart failure in ethnic minority populations, heart failure and pregnancy, disease management, and quality improvement/assurance programs. *Can J Cardiol* 2010;**26**:185–202.
2. Pearson GD, Veille JC, Rahimtoola S, et al. Peripartum cardiomyopathy: national heart, lung, and blood institute and office of rare diseases (National Institutes of Health) workshop recommendations and review. *JAMA* 2000;**283**:1183–8.
3. Bhattacharyya A, Basra SS, Sen P, Kar B. Peripartum cardiomyopathy: a review. *Tex Heart Inst J* 2012;**39**:8–16.
4. Sliwa K, Fett J, Elkayam U. Peripartum cardiomyopathy. *Lancet* 2006;**368**:687–93.
5. Tsui BC, Gupta S, Finucane B. Determination of epidural catheter placement using nerve stimulation in obstetric patients. *Reg Anesth Pain Med* 1999;**24**:17–23.
6. Macarthur A, Imarengiaye C, Tureanu L, Downey K. A randomized, double-blind, placebo-controlled trial of epidural morphine analgesia after vaginal delivery. *Anesth Analg* 2010;**110**:159–64.
7. Keren H, Burkhoff D, Squara P. Evaluation of a noninvasive continuous cardiac output monitoring system based on thoracic bioreactance. *Am J Physiol Heart Circ Physiol* 2007;**293**:H583–9.
8. Ohashi Y, Ibrahim H, Furtado L, Kingdom J, Carvalho JC. Non-invasive hemodynamic assessment of non-pregnant, healthy pregnant and preeclamptic women using bioreactance. *Rev Bras Anestesiol* 2010;**60**:603–13,335–40.
9. Armstrong S, Fernando R, Columb M. Minimally- and non-invasive assessment of maternal cardiac output: go with the flow! *Int J Obstet Anesth* 2011;**20**:330–40.
10. Critchley LA. Pulse contour analysis: is it able to reliably detect changes in cardiac output in the haemodynamically unstable patient? *Crit Care* 2011;**15**:106.
11. Squara P, Denjean D, Estagnasie P, Brusset A, Dib JC, Dubois C. Noninvasive cardiac output monitoring (NICOM): a clinical validation. *Intensive Care Med* 2007;**33**:1191–4.
12. Marik PE, Cavallazzi R, Vasu T, Hirani A. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. *Crit Care Med* 2009;**37**:2642–7.
13. Langesaeter E, Dyer RA. Maternal haemodynamic changes during spinal anaesthesia for caesarean section. *Curr Opin Anaesthesiol* 2011;**24**:242–8.
14. James CF, Banner T, Caton D. Cardiac output in women undergoing cesarean section with epidural or general anesthesia. *Am J Obstet Gynecol* 1989;**160**:1178–84.
15. Langesaeter E, Rosseland LA, Stubhaug A. Continuous invasive blood pressure and cardiac output monitoring during cesarean delivery: a randomized, double-blind comparison of low-dose versus high-dose spinal anesthesia with intravenous phenylephrine or placebo infusion. *Anesthesiology* 2008;**109**:856–63.
16. Fanning N, Balki M, Sermer M, Colman J, Carvalho JC. Noninvasive cardiac output monitoring during general anesthesia for cesarean delivery in a patient with severe aortic stenosis. *Can J Anaesth* 2011;**58**:837–41.
17. Witlin AG, Mabie WC, Sibai BM. Peripartum cardiomyopathy: a longitudinal echocardiographic study. *Am J Obstet Gynecol* 1997;**177**:1129–32.
18. Secher NJ, Arnsbo P, Wallin L. Haemodynamic effects of oxytocin (syntocinon) and methyl ergometrine (methergin) on the systemic and pulmonary circulations of pregnant anaesthetized women. *Acta Obstet Gynecol Scand* 1978;**57**:97–103.
19. Abdul-Karim R, Assali NS. Renal function in human pregnancy. Effects of oxytocin on renal hemodynamics and water and electrolyte excretion. *J Lab Clin Med* 1961;**57**:522–32.
20. Thomas JS, Koh SH, Cooper GM. Haemodynamic effects of oxytocin given as i.v. bolus or infusion on women undergoing caesarean section. *Br J Anaesth* 2007;**98**:116–9.
21. de Groot AN, van Dongen PW, Vree TB, Hekster YA, van Roosmalen J. Ergot alkaloids. Current status and review of clinical pharmacology and therapeutic use compared with other oxytocics in obstetrics and gynaecology. *Drugs* 1998;**56**:523–35.
22. Harber CR, Levy DM, Chidambaram S, Macpherson MB. Life-threatening bronchospasm after intramuscular carboprost for postpartum haemorrhage. *BJOG* 2007;**114**:366–8.
23. Andersen LH, Secher NJ. Pattern of total and regional lung function in subjects with bronchoconstriction induced by 15-me PGF2 alpha. *Thorax* 1976;**31**:685–92.
24. Baduni N, Sanwal MK, Jain A. Acute pulmonary edema after intramyometrial prostodin. *J Anaesthesiol Clin Pharmacol* 2011;**27**:275–7.
25. Belfort MA, Rokey R, Saade GR, Moise Jr KJ. Rapid echocardiographic assessment of left and right heart hemodynamics in critically ill obstetric patients. *Am J Obstet Gynecol* 1994;**171**:884–92.
26. Dennis AT. Transthoracic echocardiography in obstetric anaesthesia and obstetric critical illness. *Int J Obstet Anesth* 2011;**20**:160–8.
27. Squara P, Rotcajg D, Denjean D, Estagnasie P, Brusset A. Comparison of monitoring performance of Bioreactance vs. pulse contour during lung recruitment maneuvers. *Crit Care* 2009;**13**:R125.

Appendix. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.ijoa.2013.10.008.

0959-289X/$ - see front matter
http://dx.doi.org/10.1016/j.ijoa.2013.10.008