Can J Anesth/J Can Anesth (2012) 59:1119–1124
DOI 10.1007/s12630-012-9793-6

CASE REPORTS/CASE SERIES

Cardiac output monitoring during Cesarean delivery in a patient with palliated tetralogy of Fallot

Monitorage du débit cardiaque au cours d'un accouchement par césarienne chez une patiente ayant une tétralogie de Fallot opérée

Jefferson Clivatti, MD · Rebecca L. Smith, MD · Mathew Sermer, MD · Candice Silversides, MD · Jose C. A. Carvalho, MD, PhD

Received: 4 July 2012 / Accepted: 14 September 2012 / Published online: 12 October 2012

Abstract

Purpose *Tetralogy of Fallot (TOF) is one of the most common causes of cyanotic congenital heart disease. The anesthetic management of parturients with uncorrected TOF is challenging and controversial, especially for Cesarean delivery (CD). We describe the use of noninvasive cardiac output (CO) monitoring to assist the management of CD for a woman with palliated TOF under general anesthesia.*

Clinical features *A 34-yr-old woman presented for elective CD at 38 weeks gestation. Having been born with TOF, she underwent a modified Blalock-Taussig shunt at six years of age, followed nine years later by creation of an aortopulmonary connection. The patient's functional status was New York Heart Association class I despite evident central cyanosis. A CD was performed under general anesthesia. Fentanyl, etomidate, and succinylcholine were utilized for induction, and intrathecal morphine was administered for postoperative pain control. The baseline CO (7.2 L·min^{-1}), blood pressure (156/74 mmHg), heart rate (74 beats·min^{-1}), and total peripheral resistance (1,059 dynes·sec^{-1}·cm^{-5}) remained stable throughout the procedure. Maintenance anesthesia consisted of rocuronium, sevoflurane, and an oxygen/nitrous oxide mixture. Upon delivery, an infusion of oxytocin combined with ergometrine was administered. Hemodynamic parameters remained stable and no vasopressor was required.*

Conclusion *Balanced general anesthesia and careful titration of uterotonic agents provided stable hemodynamic conditions during CD in a patient with a palliated TOF, as assessed by a continuous noninvasive CO monitor. Noninvasive CO monitoring may improve our understanding of the hemodynamic implications of various anesthetic techniques for CD in cardiac patients.*

Résumé

Objectif *La tétralogie de Fallot (TF) est l'une des causes les plus fréquentes de cardiopathie congénitale cyanosante. La prise en charge anesthésique des parturientes ayant une TF non corrigée est controversée et relève du défi, en particulier en cas d'accouchement par césarienne. Nous décrivons l'utilisation d'un monitorage non invasif du débit cardiaque (DC) pour aider la prise en charge de la césarienne chez une femme ayant une TF opérée sous anesthésie générale.*

Caractéristiques cliniques *Une femme âgée de 34 ans se présente pour une césarienne programmée à 38 semaines de grossesse. Elle est née avec une TF et a subi une intervention modifiée de Blalock-Taussig à l'âge de six ans, puis neuf ans plus tard la création d'une connexion aorto-pulmonaire. Le statut fonctionnel de la patiente*

Author contributions *Jefferson Clivatti, Rebecca L. Smith, Mathew Sermer, Candice Silversides,* and *Jose C. A. Carvalho* cared for the patient. *Jefferson Clivatti, Rebecca L. Smith,* and *Jose C. A. Carvalho* reviewed the literature. *Jefferson Clivatti* and *Rebecca L. Smith* wrote the manuscript. *Mathew Sermer, Candice Silversides,* and *Jose C. A. Carvalho* revised the manuscript, and *Jose C. A. Carvalho* submitted the final version of the manuscript.

J. Clivatti, MD · R. L. Smith, MD · J. C. A. Carvalho, MD, PhD
Department of Anesthesia and Pain Management, Mount Sinai Hospital, University of Toronto, 600 University Avenue-Room 781, Toronto, ON M5G 1X5, Canada

M. Sermer, MD · C. Silversides, MD · J. C. A. Carvalho, MD, PhD (✉)
Department of Obstetrics and Gynecology, Mount Sinai Hospital, University of Toronto, Toronto, ON, Canada
e-mail: jose.carvalho@uhn.on.ca

C. Silversides, MD
Department of Cardiology, Toronto General Hospital, University of Toronto, Toronto, ON, Canada

correspond à la classe I de la New York Heart Association en dépit d'une cyanose centrale évidente. La césarienne a été réalisée sous anesthésie générale. L'induction a été réalisée avec du fentanyl, de l'étomidate et de la succinylcholine; de la morphine a été administrée par voie intrathécale pour contrôler la douleur postopératoire. Les variables initiales étaient au départ : DC (7,2 L·min^{-1}); tension artérielle (156/74 mmHg); fréquence cardiaque (74 battements·min^{-1}); et résistance périphérique totale (1 059 dynes·sec^{-1}·cm^{-5}); elles sont restées stables tout au long de l'intervention. L'entretien de l'anesthésie a été assuré avec du rocuronium, du sevoflurane et un mélange oxygène/protoxyde d'azote. Au moment de la naissance, une perfusion associant ocytocine et ergométrine a été administrée. Les paramètres hémodynamiques sont restés stables et aucun vasopresseur n'a été nécessaire.

Conclusion *Une anesthésie générale balancée et un ajustement prudent des agents utérotoniques a procuré des conditions hémodynamiques stables au cours de l'accouchement par césarienne chez une patiente ayant une TF opérée, tel qu'évalué par le monitorage continu non invasif du DC. Le monitorage non invasif du DC pourrait améliorer notre compréhension des implications hémodynamiques des différentes techniques anesthésiques pour césarienne chez les patientes avec pathologie cardiaque.*

Tetralogy of Fallot (TOF) is one of the most common causes of cyanotic congenital heart disease. The majority of these patients survive to adulthood with a reasonable quality of life.[1] The literature contains many cases of successful pregnancy outcomes in patients with corrected TOF.[2,3] As there is little resultant hemodynamic compromise, management is typically similar to that of the healthy parturient. Pregnancy in women with uncorrected TOF, however, has a maternal mortality rate (MMR) as high as 12%.[4] The anesthetic management of such parturients remains challenging and controversial. An in-depth understanding of the underlying anatomical configuration and its impact on cardiorespiratory physiology is paramount. Although much is known about the pathophysiology of congenital heart disease in pregnancy, little is known about the hemodynamic consequences of various anesthetic techniques. The recent development and wide availability of noninvasive cardiac output (CO) monitors may further contribute to the understanding of the impact of anesthetic techniques and other interventions. Such knowledge may facilitate the tailoring of each patient's anesthetic management to maintain physiological equilibrium. We describe the use of noninvasive CO monitoring to assist the management of Cesarean delivery (CD) for a woman with palliated TOF under general anesthesia. Written consent for publication of this case report has been obtained.

Case description

A G2P0A1 34-yr-old woman (71 kg, 164 cm) presented for elective CD at $38^{3/7}$ weeks gestation after declining vaginal delivery following extensive discussions with different members of the team, including a patient care conference. Having been born with TOF, she underwent a modified Blalock-Taussig shunt (BTS) at six years of age. A second procedure at 14 yr of age was intended to be fully corrective but was not considered technically feasible. Instead, an aortopulmonary connection was created. Further surgery offered in adulthood was declined. The patient's first pregnancy resulted in an uncomplicated first trimester therapeutic abortion under general anesthesia.

Throughout the current pregnancy, the patient's functional status was New York Heart Association class I. Physical examination revealed central cyanosis, clubbing, and a grade 3/6 continuous murmur best heard at the apex. Baseline oxygen saturation was approximately 85% with hematocrit of 56%. An electrocardiogram revealed normal sinus rhythm, prominent P waves, and right ventricular hypertrophy. An echocardiogram showed normal left ventricular size and function, a mildly enlarged right ventricle with normal function, a large ventricular septal defect (VSD) with bidirectional flow, an absent pulmonary valve, a functional BTS, and turbulent flow above the aortic valve consistent with the aortopulmonary shunt. There was no pulmonary hypertension. Medication consisted of levothyroxine for subclinical hypothyroidism.

A CD was performed under general anesthesia. The patient had fasted for ten hours. Aspiration prophylaxis consisted of ranitidine, metoclopramide, and sodium citrate. Pre-induction management included insertion of a bubble trap (MED-RX®, Benlan Inc, Oakville, ON, Canada) in the intravenous line, insertion of an arterial line, placement of a noninvasive CO monitor (NICOM™ Cheetah Medical Inc, Portland, OR, USA), and administration of intrathecal morphine 100 μg for postoperative pain control. Anesthesia was induced in modified rapid sequence with fentanyl 250 μg, etomidate 20 mg, and succinylcholine 120 mg. Cricoid pressure was applied and the patient's trachea was safely intubated. Baseline pulse oximetry was 78-83% on room air. Following preoxygenation, the baseline pulse oximetry increased to 87% and was maintained above 90% during ventilation. Noninvasive CO monitoring data were collected at one-minute intervals beginning 30 min prior to induction until six hours post delivery. Preceding any fluid administration,

baseline hemodynamic data were obtained with the patient positioned supine with a wedge under her right hip: CO 7.2 L·min^{-1}; stroke volume (SV) 97.4 mL; heart rate (HR) 74 beats·min^{-1}; blood pressure (BP) 156/74 mmHg; and total peripheral resistance (TPR) 1,059 dynes·sec^{-1}·cm^{-5}. Maintenance anesthesia consisted of rocuronium 30 mg for muscle relaxation and sevoflurane 0.9-1.1% in an oxygen/nitrous oxide mixture (50:50 mixture). We used volume-controlled ventilation with a tidal volume of 8 mL·kg^{-1}, a respiratory rate of 8-12 cycles·min^{-1}, and a positive end-expiratory pressure of 5 cmH_2O. Following induction, the CO, BP, and TPR remained unchanged; HR increased to 89 beats·min^{-1}; SV reduced to 86.6 mL, and the stroke volume variation (SVV) was 11%. Following delivery, an infusion of oxytocin 20 IU with ergotamine 0.25 mg in 1 L of Ringer's lactate was initiated at 120 mL·hr^{-1}. Two boluses of oxytocin 0.5 IU facilitated uterine contractions, one at the delivery of the neonate's anterior shoulder, the other within the next five minutes. Following delivery, CO, SV, and TPR remained stable; BP reduced to 128/76 mmHg, and HR returned to baseline. No vasopressor was required. Intraoperative analgesia consisted of morphine 9 mg *iv*, ketorolac 30 mg *iv*, and acetaminophen 1,300 mg rectally. Muscle relaxation was reversed with neostigmine 2.5 mg and glycopyrrolate 0.4 mg. Throughout the procedure, 800 mL of Ringer's lactate was administered, including the initial preload of 5 mL·kg^{-1}. The estimated blood loss was 400 mL. A healthy neonate was delivered weighing 2,450 g, with Apgar scores of 9 and 9 at one and five minutes, respectively. After uneventful tracheal extubation, the patient was kept on the labour and delivery unit for 24 h, and discharged home on the third postoperative day. The patient's hemodynamic profile during the procedure is depicted in Figs 1 and 2.

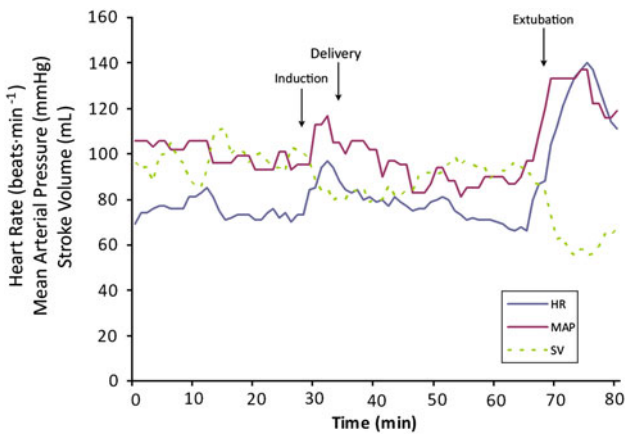

Fig. 1 Heart rate (HR), mean arterial pressure (MAP), and stroke volume (SV) measured by NICOM during Cesarean delivery

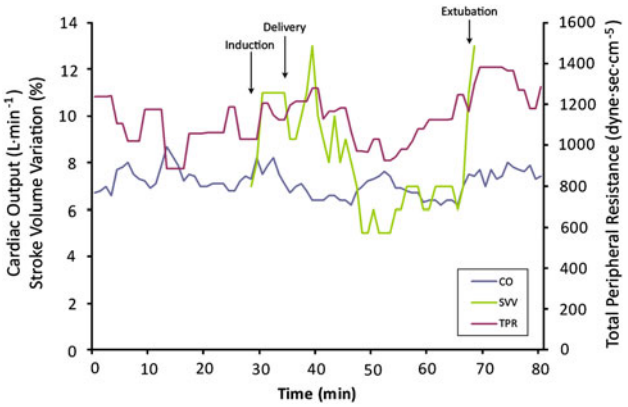

Fig. 2 Cardiac output (CO), total peripheral resistance (TPR), and stroke volume variation (SVV) measured by NICOM during Cesarean delivery

Discussion

The eighth report of the Confidential Enquiries into Maternal Deaths in the United Kingdom found cardiac disease to be the most common cause of maternal mortality in 2006-2008. The MMR for cardiac disease was 2.31 per 100,000 maternities, six percent of which was attributed to Grown-up Congenital Heart Disease (GUCHD).[5] Approximately 85% of the 0.8% of infants born worldwide with congenital heart disease survive to reproductive age.[2]

Tetralogy of Fallot accounts for 7-10% of all congenital cardiac defects.[6] It consists of a VSD, an overriding aorta, and right ventricular hypertrophy resulting from right ventricular outflow tract obstruction (RVOTO) that favours a right-to-left shunt. Intracardiac pressures tend to equalize across the VSD, and cyanosis occurs due to shunting at a ventricular level and a reduction of blood volume in the pulmonary vasculature from the RVOTO.[7] The relative resistances of the pulmonary and systemic circulations determine the degree of shunt. Both systolic and diastolic ventricular dysfunction occurs due to increased ejection work. Gradual deterioration of ventricular function is attributable to the RVOTO. Right ventricular failure, arrhythmias, progressive tricuspid valve regurgitation, and sudden cardiac death may all result.[3] In palliation, the modified BTS utilizes a polytetrafluoroethylene graft interposed between the subclavian artery and the pulmonary artery. This increases the volume of blood entering the pulmonary vasculature; however, it rarely causes overperfusion.[8] This procedure will improve the systemic oxygenation but does not fully reverse cyanosis as there is still a shunt at the ventricular level. Over time, there is strain on the low pressure pulmonary circulation, which eventually leads to pulmonary hypertension and right ventricular failure.[9]

In patients with palliated TOF, the burden of pregnancy is significant. Cyanotic lesions, such as palliated TOF, without the presence of pulmonary hypertension are considered to be of moderate risk with a mortality rate in pregnancy of 1-5%.[1] The MMR in the presence of pulmonary hypertension is higher. Poor prognostic indicators in parturients with TOF include a hematocrit $> 60\%$, oxygen saturations of $< 85\%$, and a history of syncope. Morbidity tends to be highest amongst those with a poor ventricular functional class, pulmonary hypertension, severe RVOTO, ventricular dysfunction, and pre-pregnancy anticoagulation.[4] Our patient could be classified as moderate risk as she did not have pulmonary hypertension, her hematocrit was below 60%, and she had no history of syncope.

Cyanotic congenital heart disease in itself is not an indication for CD unless there is significant aortic pathology.[10] Vaginal delivery inherently carries fewer risks for both mother and fetus.[2] Epidural analgesia is recommended for labour, and loss of resistance to saline is utilized to avoid accidental air embolism.[11] The effective sympathectomy minimizes hemodynamic fluctuations related to catecholamine surges; however, careful titration of epidural analgesia is required to avoid significant decreases in preload and afterload. A passive second stage and instrumental delivery is preferred in most women.[10]

If a CD is indicated, the anesthetic technique of choice remains controversial. The primary consideration is to maintain the shunt fraction at, or below, its baseline level. This requires maintenance of the systemic vascular resistance (SVR), the intravascular volume, and venous return.[12] Patients should not be fasted excessively as dehydration will further predispose them to thromboembolic events.[13] Decreases in BP and SVR or threatening cyanotic crises should be managed by pure vasoconstrictors, such as phenylephrine. Reflex bradycardia is less of a concern for the parturient hemodynamics, but may compromise the uteroplacental perfusion and fetal circulation.[13] Arrhythmias should be managed promptly, as should blood loss; fluid shifts and tachycardia in response to volume depletion will strain an already compromised cardiovascular system. Hemorrhage occurs more commonly due to the angiogenesis associated with chronic hypoxemia as well as a reduction in both the absolute number and function of platelets.[1] The hematocrit should be kept close to preoperative levels, and invasive BP monitoring is performed in the contralateral arm to the BTS.[11] Central venous line insertion may be problematic due to previous surgical reconstruction or anatomic abnormalities.[9] An air bubble trap is advisable to prevent paradoxical air embolism, and uterotonic agents should be administered slowly to avoid hemodynamic instability.

In our case, the patient insisted on a CD despite extensive discussions with a multi-professional team and no clear medical or obstetrical contraindication to vaginal delivery. We considered that general anesthesia would be more suitable for this case based on the analysis of risks and benefits. The literature does not favour one procedure over the other, especially in palliated cases. In our view, there is a lack of understanding of the anesthetic effects on patients with this condition, and noninvasive CO monitoring of cases performed under both general as well as regional anesthesia may help anesthesiologists make better decisions in the future.

Neuraxial anesthesia may be used in these patients. The patient benefits most from neuraxial anesthesia as she is then able to witness the birth of her child. Spinal anesthesia is typically considered to be contraindicated due to the sudden and significant reduction in SVR.[13] From the anesthesiologist's viewpoint, a carefully titrated epidural will confer a degree of hemodynamic stability; however, the peripheral vasodilatation reduces preload and SVR, raising the shunt fraction that bypasses the pulmonary tree.[9] This deleterious effect of neuraxial anesthesia can be reversed with the use of peripheral adrenergic vasopressors.[1] Although infrequent, unplanned or urgent conversion from neuraxial to general anesthesia may be fraught with difficulty. Anxiety, pain, and distress will have significant impact on the cardiorespiratory dynamics.

General anesthesia may improve oxygenation not only by allowing manipulation of the shunt fraction through ventilation strategies, but also by reducing the work of breathing.[13] Mechanical ventilation may prevent factors that increase the pulmonary vascular resistance (PVR), such as hypoxia, hypercarbia and acidosis. Concerns regarding general anesthesia include the hemodynamic response to laryngoscopy, the possibility of failed intubation and aspiration, and drug-induced myocardial depression.[12] Noninvasive CO monitoring may assist in minimizing these risks. Intermittent positive pressure ventilation may reduce venous return while increasing PVR and dead space due to a greater shunt fraction. The oxygen saturation probe is perhaps the most important monitor in these patients as it reflects the effective pulmonary blood flow.[9] The induction agent is of ultimate importance and should be chosen judiciously. A faster arm-brain circulation time is to be expected, and drugs may display exaggerated cardiovascular effects.[10]

Etomidate was chosen as the induction agent in this case as it has been shown to be associated with great hemodynamic stability in critical patients. Thiopental is currently not available in Canada, and propofol, currently used for induction in CDs, is well known for its cardiovascular depressant effects. Ketamine may have been a suitable induction agent in this case, but it is known to induce hypertension and overall cardiovascular stimulation.

Although the Canadian formulary does not include etomidate, this drug is available at our hospital through a special access program approved by Health Canada. Fentanyl was used to prevent adverse response to laryngoscopy and intubation. The patient's mean TPR and CO remained unchanged, with only a minor decrease in the SV, and no vasopressor was needed. Desflurane was avoided, as increasing concentrations may abruptly raise the PVR.[9] Instead, sevoflurane was used as the maintenance agent. During the procedure, the SVV remained consistently low, indicating euvolemia. Upon delivery of the fetus we infused a combination of oxytocin and ergotamine with the goal of optimizing myometrial contraction with smaller doses of oxytocin, and counteracting the oxytocin-induced vasodilatation with the ergotamine-induced vasoconstriction.

The CO monitoring should be considered an integral part of the clinical management of GUCHD cases as well as any other case involving a complex or severe cardiac condition. In some cases, the hemodynamic data will be used to guide clinical management, while in others, it will be used to affirm clinical management decisions, as in the current report.

We used the NICOM, a noninvasive CO monitor based on bioreactance technology. The NICOM system's mode of operation has been discussed at length by other authors.[14] In brief, the system measures the bioreactance or the phase shift in voltage across the thorax. Unlike bioimpedance, bioreactance-based CO measurements do not use the static impedance and do not depend on the distance between the electrodes for the calculations of SV, two factors that reduce the reliability of the result. The CO, as measured by bioreactance, has been shown to be highly correlated with that measured by thermodilution and pulse contour analysis. It is operator independent, simple to use and has an improved signal-to-noise ratio when compared with bioimpedance. Its accuracy has been tested against that of thermodilution in both animals and humans,[15] and it has been used in pregnant patients.[16-18] The NICOM has been found to have acceptable accuracy, and it correlates well with more established methods of CO monitoring.[14] Little has been studied regarding the use of NICOM in patients with grossly abnormal cardiovascular anatomy; however, the underlying physics of bioreactance technology is based on thoracic pulsatility, which takes into account the entire volume of blood in the thoracic cage irrespective of which side it occurs in the cardiac anatomy. While the actual values generated cannot be guaranteed to be a true and exact representative of the CO and SV, they do provide a baseline, and these baseline values allow anesthetic management to proceed in such a manner as to return these values to their baseline. Triggers for the treatment of identified changes should be based on the entire clinical picture and not on the generated numbers alone, and they should incorporate the patient's clinical status at the time. The presence of intracardiac shunts makes it extremely difficult to assess and validate CO readings because there is no accurate standard for comparison. Thermodilution, echocardiography, and Fick's law are all unreliable methods in these cases. NICOM's signal originates from the aortic outflow tract, and thus it is postulated that intracardiac shunts do not impact the recordings; however, this assumption is yet to be confirmed.

There is no generic recipe for success in cases such as this. Each patient with GUCHD requires an individualized plan of care, and understanding each patient's particular physiology is the first step towards a successful outcome. Cardiac output monitoring may be helpful in this setting to assess the response to anesthesia and surgical stress. Early multidisciplinary collaboration at a centre of excellence is recommended. The anesthetic technique should be adapted to minimize the hemodynamic stress of labour and delivery on the underlying pathophysiology. All staff should be aware that the early postpartum period remains a high-risk period. Due to the advances in medical and surgical care and the associated increase in patient life expectancy, we should expect to face the challenge that this particular group of parturients poses on an increasingly frequent basis.

Conflicts of interest None declared.

References

1. *Hepburn L*, *Kelleher AA*. Grown-up congenital heart disease. Anaesth Intensive Care Med 2009; 10: 451-6.
2. *Uebing A*, *Steer PJ*, *Yentis SM*, *Gatzoulis MA*. Pregnancy and congenital heart disease. BMJ 2006; 332: 401-6.
3. *Veldtman GR*, *Connolly HM*, *Grogan M*, *Ammash NM*, *Warnes CA*. Outcomes of pregnancy in women with tetralogy of Fallot. J Am Coll Cardiol 2004; 44: 174-80.
4. *Kaur H*, *Suri V*, *Aggarwal N*, *Chopra S*, *Vijayvergiya R*, *Talwar KK*. Pregnancy in patients with tetralogy of Fallot. World Journal for Pediatric and Congenital Heart Surgery 2010; 1: 170-4.
5. *McClure JH*, *Cooper GM*, *Clutton-Brock TH*. Centre for Maternal and Child E. Saving mothers' lives: reviewing maternal deaths to make motherhood safer: 2006-8: a review. Br J Anaesth 2011; 107: 127-32.
6. *Starr JP*. Tetralogy of Fallot: yesterday and today. World J Surg 2010; 34: 658-68.
7. *Luxenberg D*, *Torchen L*. Tetralogy of Fallot. In: Abdulla R, editor. Heart Disease in Children: A Pediatrician's Guide. 1st ed. New York: Springer; 2011. p. 167-76.
8. *Yuan SM*, *Shinfeld A*, *Raanani E*. The Blalock-Taussig shunt. J Card Surg 2009; 24: 101-8.
9. *Chassot PG*, *Bettex DA*. Anesthesia and adult congenital heart disease. J Cardiothorac Vasc Anesth 2006; 20: 414-37.
10. *Fernandes SM*, *Arendt KW*, *Landzberg MJ*, *Economy KE*, *Khairy P*. Pregnant women with congenital heart disease: cardiac, anesthetic and obstetrical implications. Expert Rev Cardiovasc Ther 2010; 8: 439-48.
11. *Arendt KW*, *Fernandes SM*, *Khairy P*, *et al*. A case series of the anesthetic management of parturients with surgically repaired tetralogy of Fallot. Anesth Analg 2011; 113: 307-17.

12. *Maitra G*, *Sengupta S*, *Rudra A*, *Debnath S*. Pregnancy and non-valvular heart disease—anesthetic considerations. Ann Card Anaesth 2010; 13: 102-9.
13. *Weiss BM*, *Atanassoff PG*. Cyanotic congenital heart disease and pregnancy: natural selection, pulmonary hypertension, and anesthesia. J Clin Anesth 1993; 5: 332-41.
14. *Marik PE*. Noninvasive cardiac output monitors: a state-of the-art review. J Cardiothorac Vasc Anesth 2012. doi:10.1053/j.jvca.2012.03.022.
15. *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.
16. *Ohashi Y*, *Ibrahim H*, *Furtado L*, *Kingdom J*, *Carvalho JC*. Non-invasive hemodynamic assessment of non-pregnant, healthy pregnant and preeclamptic women using bioreactance [corrected]. Rev Bras Anestesiol 2010; 60: 603-13.
17. *Doherty A*, *Ohashi Y*, *Downey K*, *Carvalho JC*. Non-invasive monitoring based on bioreactance reveals significant hemodynamic instability during elective cesarean delivery under spinal anesthesia. Rev Bras Anestesiol 2011; 61: 320-5.
18. *Fanning N*, *Balki M*, *Sermer M*, *Colman J*, *Carvalho JC*. Non-invasive cardiac output monitoring during general anesthesia for cesarean delivery in a patient with severe aortic stenosis. Can J Anesth 2011; 58: 837-41.