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CASE REPORTS/CASE SERIES

Noninvasive cardiac output monitoring during general anesthesia for Cesarean delivery in a patient with severe aortic stenosis

Monitorage non effractif du débit cardiaque pendant une anesthésie générale pour un accouchement par césarienne chez une patiente atteinte de sténose aortique grave

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Abstract

Purpose *The anesthetic management of women with severe aortic stenosis (AS) undergoing Cesarean delivery (CD) remains controversial. We used a relatively new bioreactance technology to highlight the continuous hemodynamic changes related to anesthesia, delivery, and recovery in a parturient with severe AS.*

Clinical features *A 29-yr-old woman, New York Heart Association Class II, with a congenital bicuspid aortic valve and AS presented for CD at 36.5 weeks of gestation. The estimated aortic valve area on echocardiogram was 0.75 cm^2, and the maximal transvalvular gradient was 64 mmHg. Cesarean delivery was performed under general anesthesia with an epidural catheter placed prior to induction for postoperative analgesia. Noninvasive cardiac output (CO) monitoring based on bioreactance was used throughout the procedure. Cardiac output increased from 7-12 $L{\cdot}min^{-1}$ following delivery primarily due to an increase in stroke volume. Both stroke volume variation and total peripheral resistance decreased, while the patient's heart rate did not change. Increased stroke volume, likely associated with decreased afterload and increased preload, contributed to an increase in CO from 7-12 $L{\cdot}min^{-1}$.*

Conclusion *Continuous CO data obtained from bioreactance-based monitoring suggests that pregnant women with severe AS may experience an increase in CO under certain circumstances. This result is in keeping with data obtained from non-pregnant individuals and is an interesting finding that warrants further study. Noninvasive CO monitoring may improve our understanding of the peripartum changes in women with heart disease.*

Résumé

Objectif *La prise en charge anesthésique des femmes atteintes de sténose aortique (SA) grave et subissant un accouchement par césarienne demeure controversée. Nous avons utilisé une technologie de bioréactance relativement nouvelle afin de mettre en évidence les changements hémodynamiques continus liés à l'anesthésie, à l'accouchement et à la récupération chez une patiente atteinte de SA grave.*

Éléments cliniques *Une femme de 29 ans, de statut II selon la classification de la New York Heart Association, avec une valve aortique bicuspide congénitale et une SA, s'est présentée pour un accouchement par césarienne à 36,5 semaines de grossesse. La surface estimée de la valve aortique à l'échocardiogramme était de 0,75 cm^2, et le gradient transvalvulaire maximal de 64 mmHg. Un accouchement par césarienne a été réalisé sous anesthésie générale avec un cathéter péridural installé avant l'induction pour fournir l'analgésie postopératoire. Un monitorage non effractif du débit cardiaque (DC) s'appuyant sur la bioréactance a été utilisé tout au long de l'intervention. Le débit cardiaque a augmenté de 7-12 $L{\cdot}min^{-1}$ après l'accouchement, principalement en raison d'une augmentation du volume d'éjection. La variation du volume d'éjection et la résistance périphérique totale ont toutes deux diminué, alors que la fréquence cardiaque de la patiente n'a pas changé. Un volume*

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d'éjection accru, probablement associé à une post-charge réduite et une pré-charge accrue, a contribué à une augmentation du DC de 7-12 L·min^{-1}.

Conclusion *Des données continues de DC obtenues grâce à un monitorage fondé sur la bioréactance indique que les femmes enceintes atteintes de SA grave pourraient avoir un DC accru dans certaines circonstances. Ce résultat correspond aux données obtenues de personnes non enceintes; en outre, il est intéressant et justifie des études plus approfondies. Le monitorage non effractif du DC pourrait contribuer à notre compréhension des modifications survenant au cours de la période périnatale chez les femmes atteintes de maladies cardiaques.*

The anesthetic management of patients with severe aortic stenosis (AS) undergoing Cesarean delivery (CD) remains controversial. The stenotic aortic valve limits blood leaving the heart and may lead to a relatively fixed cardiac output (CO).[1–7] General anesthesia is advocated for its cardiostable properties.[4] The safety of neuraxial anesthesia continues to be questioned, but recent case reports[3,7] and series[2,8] have documented its successful use in this high-risk patient group.

The mechanism by which pregnant patients with severe AS tolerate vasodilatory events in the peripartum period is not well understood. However, studies of non-pregnant patients with severe AS and heart failure suggest that careful vasodilation may increase CO,[1] but it is unclear whether this information is applicable to pregnant women.

The availability of new minimally invasive and noninvasive technologies for CO monitoring may provide valuable information on how pregnant patients, especially those with AS, respond to labour, delivery, and various anesthetic techniques. We used a noninvasive CO monitoring device to highlight the continuous hemodynamic and cardiodynamic variations related to anesthesia, delivery, and recovery in a parturient with severe AS. The patient gave written consent for publication of this case report.

Case description

A 29-yr-old woman (160 cm, 85 kg) in her second pregnancy with one living child (i.e., G2P1) presented for Cesarean delivery at $36^{4/7}$ weeks of gestation for premature rupture of membranes (PROM) and transverse lie. The woman had a congenital bicuspid aortic valve, a palpable Grade V/VI heart murmur (loudest in the aortic area), and AS. Three years previously, she had tolerated her first pregnancy well. Subsequently, her aortic valve area (AVA) was calculated at 1.0 cm^2. At 15 weeks of gestation in this pregnancy, she complained of shortness of breath after climbing seven steps, and she reduced her activity to minimal levels. Her functional status improved to New York Heart Association Class II. At 29 weeks of gestation, she was placed on bed rest for PROM.

Electrocardiogram revealed normal sinus rhythm and echocardiograms were performed each trimester from ten weeks of gestation onwards with calculation of the AVA by the continuity equation. The aortic valve area was 0.77 cm^2, 0.82 cm^2, and 0.75 cm^2 for the first, second, and third trimesters, respectively. The dimensionless index was 0.28, and the maximal aortic transvalvular gradient was 64 mmHg measured three weeks prior to delivery. The left ventricular function was good, and the left ventricular mass index was 85.9 g·m^{-2}. There was trivial mitral regurgitation; the ascending aorta was dilated at 4.3 cm, and there was no aortic regurgitation.

Cesarean delivery was performed under general anesthesia, and an epidural was sited preprocedure for management of postoperative pain. Electrical stimulation of the epidural catheter was used to confirm its position. An arterial line was inserted prior to induction of anesthesia, and a noninvasive CO monitor (NICOM^{TM} Cheetah Medical Inc, Portland, OR, USA) was applied. The patient was premedicated with midazolam 1 mg, ranitidine 50 mg *iv*, and sodium citrate 0.3 M. Anesthesia was induced with fentanyl 250 μg (0.3 μg·kg^{-2}) and etomidate 16 mg (0.2 mg·kg^{-1}). Succinylcholine 120 mg (1.5 mg·kg^{-1}) was used for muscle relaxation, and the trachea was safely intubated. Rocuronium 20 mg (0.25 mg·kg^{-1}) was administered for maintenance of muscle relaxation, and the patient was maintained on sevoflurane in an oxygen/air mix at a minimum alveolar concentration of 0.6-0.7. An etomidate infusion of 4 μg·kg^{-1}·min^{-1} was run concurrently to minimize cardiac instability, and epidural morphine 2.5 mg was administered close to delivery. An oxytocin infusion of 20 IU·L^{-1} Ringer's lactate (500 mU bolus followed by 40 mU·min^{-1}) was given following delivery. Morphine 5 mg *iv* was then given followed by epidural administration of 0.125% bupivacaine 10 mL and fentanyl 100 μg in two divided doses 15 min apart. The total amount of Ringer's lactate administered was 1,400 mL. Six hundred and fifty micrograms of phenylephrine were administered for maintenance of blood pressure. The patient's trachea was extubated safely, and she was transferred to the coronary care unit for ongoing continuous monitoring of the electrocardiogram and arterial line recordings. After 24 hr, she was transferred to the ward and then discharged home on the fifth postoperative day.

Noninvasive CO monitoring data were collected at one-minute intervals beginning 15 min prior to induction of anesthesia and continuing until the end of the procedure. The following data were recorded at baseline prior to

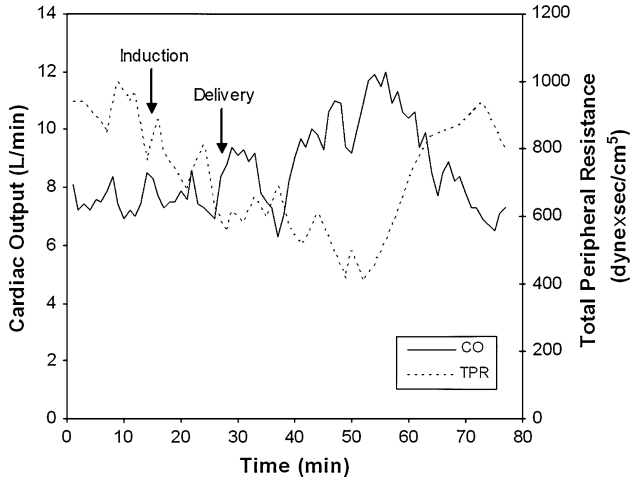

Fig. 1 Cardiac output (CO) and total peripheral resistance (TPR) measured by NICOM during Cesarean delivery

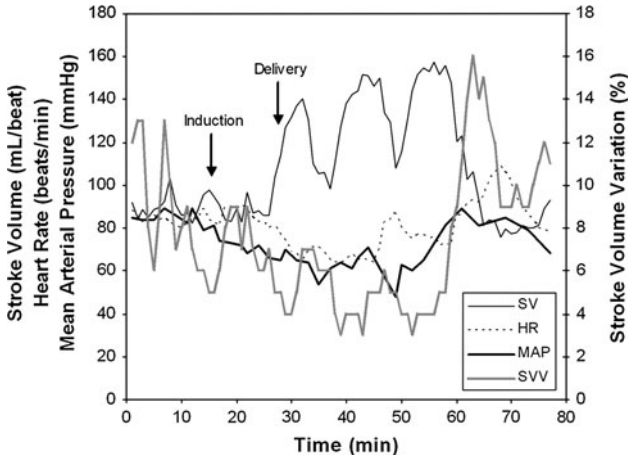

Fig. 2 Stroke volume (SV), heart rate (HR), mean arterial pressure (MAP), and stroke volume variation (SVV) measured by NICOM during Cesarean delivery

induction of anesthesia: CO, 7.47 (0.41) L·min^{-1}; stroke volume (SV), 88.25 (5.1) mL; heart rate (HR), 83.9 (2.16) beats·min^{-1}; blood pressure, 101 (3.4) /77 (2.16) mmHg; stroke volume variation (SVV), 10.5% (2.5%); and total peripheral resistance (TPR), 899 (34.8) dynes·sec·cm^{-5}. Following induction of anesthesia, the following data were recorded: SV, HR, and CO remained unchanged; total peripheral resistance decreased to 764.1 (64) dynes·sec^{-1}·cm^{-5}; and SVV decreased to 7.25% (1.63%). Following delivery, CO increased from 7.47 (0.41) L·min^{-1} to a maximum of 12 L·min^{-1}; stroke volume increased to a maximum of 151 mL; SVV decreased to 4%; TPR decreased to a nadir of 411 dynes·sec^{1}·cm^{-5}; heart rate at maximal CO was 76 beats·min^{-1}; and blood pressure was 85/55 mmHg. Cardiac output subsequently declined to baseline levels toward the end of surgery. The 60% increase in CO was attributable primarily to the increase in stroke volume and was not related to changes in HR. Figs. 1 and 2 detail these changes.

Discussion

We used a noninvasive CO monitor based on bioreactance technology to monitor CO and other hemodynamic variables in a patient with severe AS undergoing Cesarean delivery under general anesthesia. We observed an increase in CO following delivery. The increase in CO was attributable primarily to an increase in stroke volume as the HR did not change.

Stroke volume variation, which reflects respiratory changes in aortic blood flow, is an accurate measure of fluid responsiveness.[9] The magnitude of these changes is highly dependent on fluid status. The lower the SVV, the patient is less likely to respond to fluid, suggesting that the patient is more fluid replete. Stroke volume variation in this patient decreased from 10.5% (2.5%) at baseline to 4% at the time of maximal CO. Delivery of the fetus with release of aortocaval compression, uterine autotransfusion, and intravenous fluid administration contributed to this change.[10] Soon after delivery, oxytocin, a potent vasodilator,[11] was administered as a bolus and then infusion. To optimize analgesia and reduce the risk of postoperative tachycardia, morphine *iv* was administered followed by initiation of epidural analgesia. These events contributed to the prolonged reduction in TPR observed. The combination of reduced TPR and reduced SVV in the setting of normal HR likely facilitated the increase in stroke volume and CO.

Aortic stenosis is defined as severe when the valve area is <1 cm^2, the mean gradient is >40 mmHg, and the jet velocity is >4 m·sec^{-1}.[12] In pregnancy, the continuity equation for the calculation of AVA is least affected by changes in stroke volume and may best estimate the severity of the stenotic lesion.[8] The transvalvular gradient and jet velocity that are measured during pregnancy may be higher than in the post-pregnancy state.[13,14] Therefore, one could argue that this was not a true case of severe AS. However, the reduction in transvalvular gradient following pregnancy is not associated with a reduction in the degree of stenosis.[13]

In AS, an increase in left ventricular wall thickness and mass, i.e., ventricular hypertrophy, to maintain normal wall stress and unimpaired contractions may bring adverse consequences, such as a potential for cardiac ischemia and diastolic dysfunction.[15] Our patient had an elevated left ventricular mass index, but she did not meet established criteria for ventricular hypertrophy.[16] In the study by Tzemos *et al.*, 40% of pregnant patients with AS had

hypertrophy[13]; however, the degree of hypertrophy was not predictive of adverse outcomes. This result contrasts with findings in older patients with AS where ventricular hypertrophy was associated with adverse outcomes.[15,17]

Severe AS is thought to lead to a relatively fixed CO with an inability to increase stroke volume. Cardiac output is thus determined primarily by HR.[2–6] It is thought that vasodilator therapy is relatively contraindicated as it may rapidly decrease systemic vascular resistance, resulting in decreased blood pressure, decreased coronary blood flow, and a compensatory tachycardia which can be hazardous. However, there is evidence that careful vasodilation may increase CO in AS. Khot *et al*.[1] administered careful vasodilator therapy (sodium nitroprusside) to 25 patients with severe AS in cardiac failure. Cardiac output was increased successfully in all study patients, and stroke volume was increased from 34 (10) mL to 58 (23) mL. The authors referred to the physics principle that resistance in series is additive. Total resistance incorporates both valvular and peripheral resistance. A decrease in peripheral resistance will reduce total resistance. As CO is inversely proportional to resistance, a reduction in total resistance will facilitate an increase in CO once there is adequate ventricular filling.

There are relatively few data on CO in pregnant patients with AS, particularly in the post-delivery period. Careful review of previous studies where pulmonary artery catheters were used for intermittent CO monitoring suggests a rise in CO in patients with severe AS following both vaginal and Cesarean delivery.[8] Brian *et al*.[7] detailed the hemodynamic changes in a pregnant woman with severe AS (transvalvular gradient 90 mmHg) undergoing Cesarean delivery with lumbar epidural anesthesia using a pulmonary artery catheter. They noted a decrease of 44% in systemic vascular resistance following oxytocin administration, with a subsequent net 21% increase in cardiac index, while HR remained unchanged. There are relatively few data to suggest that CO or SV is fixed in parturients with AS, and case reports suggest that CO may increase at delivery secondary to an increase in SV.

Due to the rapidity of the changes occurring following delivery, continuous CO monitoring devices may build on the information available from intermittent measurement devices. Avoidance of complications associated with insertion of invasive devices makes noninvasive devices attractive. Noninvasive CO monitoring is based on bioreactance technology. The mode of operation and the basic science around bioreactance have previously been reported.[18] Noninvasive CO monitoring involves the application of four sensors on the thorax. Changes in aortic blood flow drive phase shifts of propagating waves which are detected by the sensors as the frequency changes. These frequency changes correlate well with instantaneous changes in blood volume and blood flow in the aorta. Bioreactance was validated in various non-pregnant populations which manifested hemodynamic instability,[19–21] and it has been shown to produce hemodynamic profiles in the pregnant population consistent with other more invasive forms of monitoring.[22] Noninvasive CO monitoring provides continuous HR data, performs noninvasive blood pressure measurements at one-minute intervals, and records one-minute running averages of CO, stroke volume, SVV, and TPR. Noninvasive CO monitoring appears to report directional changes in CO well,[19,20] but the absolute values in pregnancy are not well validated. Therefore, in our view, the trends seen in this report may be of greater importance than the absolute CO values.

Mortality from AS in pregnancy was previously quoted as high as 17%.[6] More recent studies in the series from Silversides *et al.* suggest a better outcome with morbidity in 3/49 pregnancies and with no mortality. [23] Tzemos *et al.* reported early morbidity in 7/70 pregnancies and no mortality in their extended follow-up.[13] Regional anesthesia was previously avoided in patients with more severe forms of AS based on the assumption of fixed CO. However, there is newer evidence of good outcomes in patients with severe AS receiving neuraxial analgesia for both vaginal and Cesarean delivery. In the recent series by Ioscovich *et al*.,[2] two patients with severe AS had epidural analgesia for vaginal delivery, and one patient with severe AS had epidural anesthesia for Cesarean delivery. Two patients with AS, both of whom had an AVA of 0.5 cm^2, had Cesarean delivery under general anesthesia. General anesthesia appeared to be reserved for the most severe cases of AS, and there was no morbidity or mortality reported. The effects of oxytocin administration on parturients with AS are not discussed in the majority of reports and reviews other than in cases where the associated vasodilation has caused an ill effect.[7]

In summary, this case report suggests that pregnant women with AS may increase CO to compensate for vasodilation induced by anesthesia and oxytocin in the peripartum period. Furthermore, noninvasive continuous CO monitoring may contribute to our understanding of the peripartum hemodynamic changes in patients with AS and other cardiac lesions.

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Conflict of interest None declared.

References

1. *Khot UN*, *Novaro GM*, *Popovic ZB*, *et al*. Nitroprusside in critically ill patients with left ventricular dysfunction and aortic stenosis. N Engl J Med 2003; 348: 1756-63.

2. *Ioscovich AM*, *Goldszmidt E*, *Fadeev AV*, *Grisaru-Granovsky S*, *Halpern SH*. Peripartum anesthetic management of patients with aortic valve stenosis: a retrospective study and literature review. Int J Obstet Anesth 2009; 18: 379-86.
3. *Suntharalingam G*, *Dob D*, *Yentis SM*. Obstetric epidural analgesia in aortic stenosis: a low-dose technique for labour and instrumental delivery. Int J Obstet Anesth 2001; 10: 129-34.
4. *Whitfield A*, *Holdcroft A*. Anaesthesia for caesarean section in patients with aortic stenosis: the case for general anaesthesia. Anaesthesia 1998; 53: 109-12.
5. *Brighouse D*. Anaesthesia for caesarean section in patients with aortic stenosis: the case for regional anaesthesia. Anaesthesia 1998; 53: 107-9.
6. *Arias F*, *Pineda J*. Aortic stenosis and pregnancy. J Reprod Med 1978; 20: 229-32.
7. *Brian JE Jr*, *Seifen AB*, *Clark RB*, *Robertson DM*, *Quirk JG*. Aortic stenosis, cesarean delivery, and epidural anesthesia. J Clin Anesth 1993; 5: 154-7.
8. *Easterling TR*, *Chadwick HS*, *Otto CM*, *Benedetti TJ*. Aortic stenosis in pregnancy. Obstet Gynecol 1988; 72: 113-8.
9. *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.
10. *Silversides CK*, *Colman JM*. Physiological changes in pregnancy. *In*: Oakley C, Warnes CA (Eds). Heart Disease in Pregnancy, 2nd ed. Malden, MA, USA: Blackwell Publishing; 2007.
11. *Langesaeter E*, *Rosseland LA*, *Stubhaug A*. Haemodynamic effects of repeated doses of oxytocin during caesarean delivery in healthy parturients. Br J Anaesth 2009; 103: 260-2.
12. *Warnes CA*, *Williams RG*, *Bashore TM*, *et al*. ACC/AHA 2008 guidelines for the management of adults with congenital heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing committee to develop guidelines for the management of adults with congenital heart disease). Circulation 2008; 118: e714-833.
13. *Tzemos N*, *Silversides CK*, *Colman JM*, *et al*. Late cardiac outcomes after pregnancy in women with congenital aortic stenosis. Am Heart J 2009; 157: 474-80.
14. *Lesniak-Sobelga A*, *Tracz W*, *KostKiewicz M*, *Podolec P*, *Pasowicz M*. Clinical and echocardiographic assessment of pregnant women with valvular heart diseases—maternal and fetal outcome. Int J Cardiol 2004; 94: 15-23.
15. *Kupari M*, *Turto H*, *Lommi J*. Left ventricular hypertrophy in aortic valve stenosis: preventive or promotive of systolic dysfunction and heart failure? Eur Heart J 2005; 26: 1790-6.
16. *Levy D*, *Savage DD*, *Garrison RJ*, *Anderson KM*, *Kannel WB*, *Castelli WP*. Echocardiographic criteria for left ventricular hypertrophy: the Framingham Heart Study. Am J Cardiol 1987; 59: 956-60.
17. *Pellikka PA*, *Sarano ME*, *Nishimura RA*, *et al*. Outcome of 622 adults with asymptomatic, hemodynamically significant aortic stenosis during prolonged follow-up. Circulation 2005; 111: 3290-5.
18. *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.
19. *Marque S*, *Cariou A*, *Chiche JD*, *Squara P*. Comparison between Flotrac-Vigileo and Bioreactance, a totally noninvasive method for cardiac output monitoring. Crit Care 2009; 13: R73.
20. *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.
21. *Benomar B*, *Ouattara A*, *Estagnasie P*, *Brusset A*, *Squara P*. Fluid responsiveness predicted by noninvasive bioreactance-based passive leg raise test. Intensive Care Med 2010; 36: 1875-81.
22. *Ohashi Y*, *Ibrahim H*, *Furtado L*, *Kingdom J*, *Carvalho JC*. Noninvasive hemodynamic assessment of non-pregnant, healthy pregnant and preeclamptic women using bio-reactance. Rev Bras Anestesiol 2010; 60: 603-13, 335-40.
23. *Silversides CK*, *Colman JM*, *Sermer M*, *Farine D*, *Siu SC*. Early and intermediate-term outcomes of pregnancy with congenital aortic stenosis. Am J Cardiol 2003; 91: 1386-9.