Non-invasive cardiac output monitoring for cesarean delivery under epidural anesthesia in a patient with Marfan syndrome and cardiomyopathy

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ABSTRACT
Maternal cardiac output and stroke volume increase significantly at the time of cesarean delivery. Parturients with baseline myocardial dysfunction are at increased risk of cardiovascular decompensation in the peripartum period and close hemodynamic monitoring is warranted. We report our use of intraoperative non-invasive cardiac output monitoring during cesarean delivery under epidural anesthesia in a 24-year-old woman with dilated cardiomyopathy secondary to Marfan syndrome, aortic arch, aortic valve and mitral valve replacements and a left ventricular ejection fraction of 37%. Three distinct hemodynamic trends were noted. After achieving adequate surgical anesthesia with 2% lidocaine 20 mL, cardiac output and stroke volume rose for approximately 20 min from baseline values of 6.3 L/min and 69 mL, respectively, to 9 L/min and 107 mL. Values subsequently trended down and remained depressed for nearly 20 min following delivery. The lack of immediate post-delivery increases in both cardiac output and stroke volume were attributed to acute blood loss, intravascular volume depletion from fluid restriction, and slow infusion of oxytocin. By the end of surgery, cardiac output and stroke volume ultimately increased by 66% and 84% of baseline values, respectively. Systemic blood pressure, heart rate and cardiac output did not appear to correlate despite the use of phenylephrine to manage hypotension. The patient remained hemodynamically stable with no evidence of acute volume overload.

Keywords: Non-invasive cardiac output monitoring; Marfan syndrome; Cardiomyopathy; Phenylephrine; Oxytocin

Introduction

Maternal cardiovascular disease complicates 0.2–4% of all pregnancies in western industrialized countries.[1] Non-invasive cardiac output (CO) monitoring has been recommended for obstetric patients with high-risk cardiovascular conditions due to the large fluctuations in maternal CO and stroke volume (SV) that occur at delivery.[2–7] No large-scale outcome data for this population currently exist.[8] We describe our use of the non-invasive CO monitoring device, NICOM™ (Cheetah Medical Inc, Newton Center, MA, USA), as an adjunctive hemodynamic monitor for a woman with Marfan syndrome and a left ventricular ejection fraction (EF) of 37% undergoing cesarean delivery (CD) under epidural anesthesia. Our report contributes to the limited body of literature detailing the intraoperative hemodynamic trends of obstetric patients with baseline ventricular dysfunction undergoing CD.

Accepted November 2015
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Case report

A 24-year-old, 98 kg G3P2 woman with Marfan syndrome, aortic arch, aortic valve (AV), and mitral valve replacements presented to her cardiologist in the 33rd week of pregnancy with shortness of breath and palpitations. A transthoracic echocardiogram demonstrated a dilated, diffusely hypokinetic left ventricle with a decline in EF from 64% before pregnancy to 37%. A 0.7 cm non-obstructing AV thrombus was identified on a subsequent transesophageal echocardiogram despite the patient having been maintained on warfarin for her prosthetic valves, with a therapeutic international normalized ratio (INR) of >2.5 in the third trimester. The patient was admitted to the cardiac intensive care unit (ICU) for intravenous heparin therapy and was started on carvedilol 12.5 mg twice daily for persistent sinus tachycardia.

On hospital day 13, urgent delivery was planned due to a worsening fetal biophysical profile. Following a multidisciplinary meeting between the obstetric, cardiology, and obstetric anesthesiology services, it was felt that the risk of AV thromboembolism during labor without anticoagulation outweighed those of surgical delivery and she was scheduled for CD. We elected to use intraoperative non-invasive CO monitoring as an

adjunct to standard heart rate (HR) and invasive arterial blood pressure (BP) monitoring given her low EF and the potential for acute heart failure from fluid shifts at the time of delivery.

In the operating room following discontinuation of heparin for 6 h and normalization of her coagulation profile, a radial arterial line, two 18-gauge peripheral intravenous catheters and an L4–5 epidural catheter were placed. After positioning the patient supine with left uterine displacement, four CO monitor electrodes were placed on the chest wall with data collection at 1-min intervals. The epidural was dosed incrementally with 2% lidocaine with 1 in 200 000 epinephrine to a total volume of 20 mL. Epidural fentanyl 100 μg was also given. Intravenous fluid administration was limited to approximately 30 mL/h following a 700 mL crystalloid bolus during epidural placement to minimize the risk of volume overload and pulmonary edema given her low EF. Supplemental oxygen at 3 L/min by nasal cannula was provided. A phenylephrine infusion was started at 20 μg/min during epidural dosing to minimize hypotension from sympathetic blockade and titrated to maintain a mean arterial pressure (MAP) of 70–90 mmHg. Additional phenylephrine boluses (80–120 μg) were given intermittently throughout the case to treat hypotension. Baseline data obtained were: CO 6.3 L/min, SV 69 mL, HR 92 beats/min and MAP 79 mmHg.

Over the course of 26 min from the start of epidural dosing until skin incision, CO and SV increased to 8.5 L/min (41%) and 103 mL (54%) respectively (Fig. 1). For the next 31 min, CO and SV declined back toward baseline despite a transient 6% increase in the first minute following delivery of a healthy 3050 g infant. An oxytocin infusion (20 U/1000 mL) was started immediately following delivery of the placenta and infused over the remainder of the case. There was no evidence of uterine atony or excessive hemorrhage reported by the obstetrician following delivery and no additional uterotonic drugs were indicated. After reaching nadir values of 6.7 L/min and 83 mL 18 min post-delivery, CO and SV then began to rise as the hysterotomy and deep tissue layers were closed. Final values obtained at skin closure were: CO 10.5 L/min, SV 126 mL, HR 83 beats/min, MAP 60 mmHg. Total crystalloid administration was 1700 mL and estimated blood loss was 800 mL.

The patient remained hemodynamically stable and was transported back to the ICU. Her immediate postoperative hemoglobin concentration was 10.8 g/dL and systemic anticoagulation with heparin and warfarin was initiated several hours later. On postoperative day 5, her hemoglobin concentration decreased to 5.6 g/dL and a large hemoperitoneum was noted on imaging. She received two units of packed erythrocytes but remained hemodynamically stable. She was discharged home on day 7 on enalapril, carvedilol and warfarin.

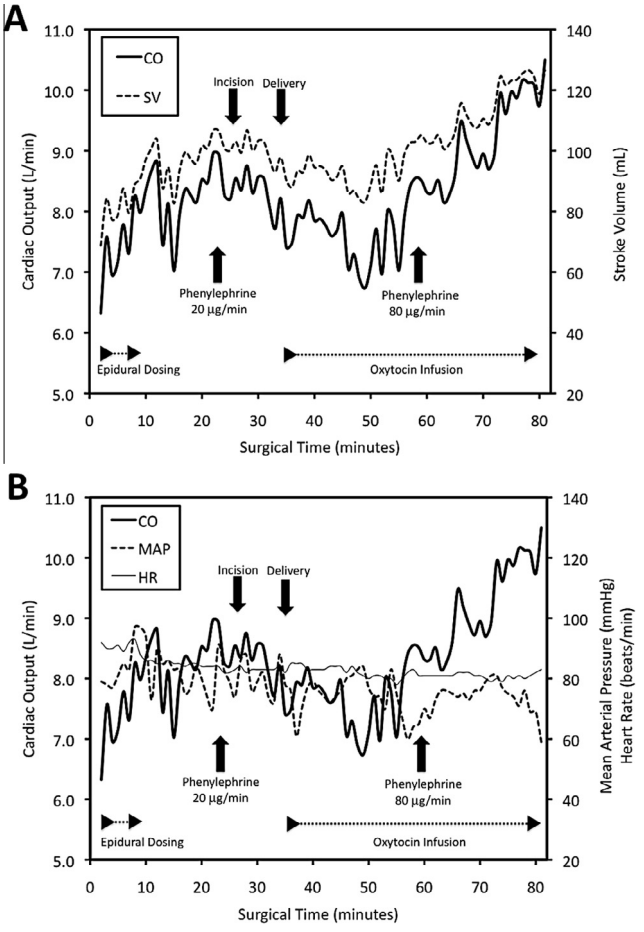

Fig. 1 Intraoperative hemodynamic trends during cesarean delivery under epidural anesthesia with key events noted (black arrows). (*A*) Cardiac output (CO) and stroke volume (SV) trends over time. (*B*) CO, mean arterial pressure (MAP), and heart rate (HR) trends over time.

An echocardiogram three months later demonstrated an EF of 41% with no evidence of AV thrombus.

Discussion

Using non-invasive CO monitoring, we were able to identify three distinct hemodynamic trends in this patient with cardiomyopathy undergoing CD (Fig. 1). Cardiac output first increased by 41% during initiation of epidural anesthesia (minutes 1–8) and was attributed to both intravascular volume loading and lowered systemic vascular resistance (SVR) from sympathetic blockade. This suggested that myocardial contractile function was preserved despite her cardiomyopathy and that she may have been relatively intravascularly volume depleted from preoperative fluid restriction in the ICU.

Cardiac output and SV subsequently declined just before skin incision and remained depressed for nearly 20 min following delivery despite the modest 6% increase