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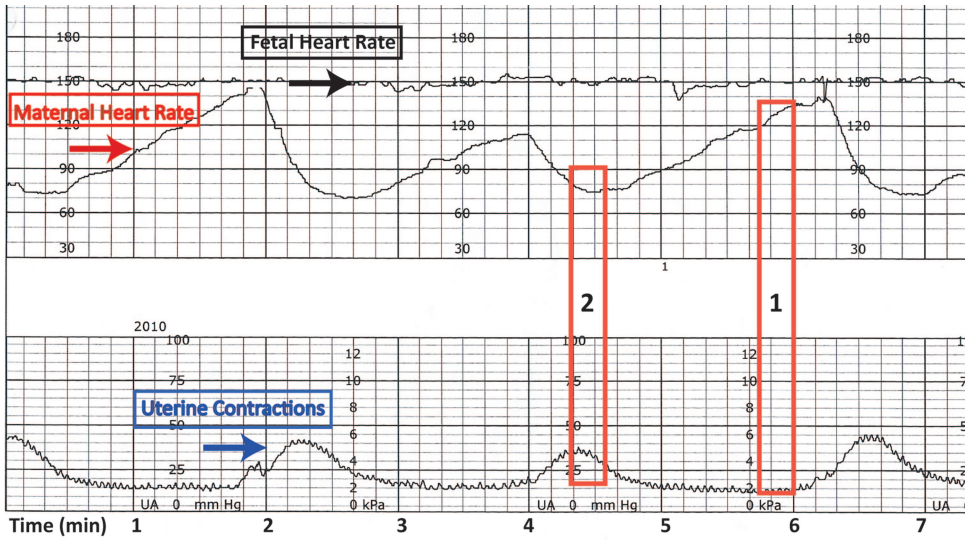
Hemodynamic Effects of Aortocaval Compression and Uterine Contractions in a Parturient with Left Ventricular Outflow Tract Obstruction

Emily J. Baird, M.D., Ph.D.,* Valerie A. Arkoosh, M.D., M.P.H.

* Department of Clinical Anesthesiology and Critical Care, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania
emily.baird@uphs.upenn.edu



A 21-YR-OLD parturient underwent induction of labor at 37 weeks gestation for cardiac decompensation in the setting of a bicuspid aortic valve and subaortic membrane. Cardiac evaluation revealed a left ventricular outflow tract gradient of 80 mmHg. Epidural analgesia was initiated with ropivacaine (0.1%) and fentanyl (0.0002%) (8 ml/h). As labor progressed, a phenylephrine infusion was required to maintain systolic systemic blood pressure more than 100 mmHg. Cyclic variations of maternal heart rate (red arrow) correlating with uterine contractions (blue arrow) subsequently developed. Fetal heart rate (black arrow) was unaffected. Evaluation of the parturient revealed she was normotensive with the phenylephrine infusion and resting comfortably in the supine position.

The sinusoidal maternal heart rate pattern suggests the tachycardia was secondary to intermittent hemodynamic changes. Given the patient's position, aortocaval compression undoubtedly was a contributing factor. At term gestation, there is nearly complete obstruction of the inferior vena cava in the supine position and venous return occurs incompletely *via* collateral veins.[1] Because venous return is impaired, stroke volume decreases, and heart rate increases to maintain cardiac output (box 1). In a laboring patient, ~400 ml of blood is displaced into the central circulation with each contraction.[2] This transient fluid influx leads to an increase in preload, with a concomitant increase in stroke volume and a reflexive decrease in heart rate (box 2).

Parturients with stenotic aortic lesions are extremely sensitive to changes in intravascular volume. Whereas hypovolemia leads to decreases in cardiac output, increases in preload are associated with pulmonary edema and atrial arrhythmias.[3] Management requires meticulous attention to positioning, intravascular volume, and perfusion pressure. The cyclic heart rate pattern resolved after a 400-ml crystalloid bolus and repositioning into left uterine displacement. The remainder of labor progressed uneventfully with the vaginal delivery of a healthy fetus *via* a planned vacuum-assisted delivery in second stage.

References

1. Kerr MG: The mechanical effects of the gravid uterus in late pregnancy. J Obstet Gynaecol Br Commonw 1965; 72:513–29
2. Lee W, Rokey R, Miller J, Cotton DB: Maternal hemodynamic effects of uterine contractions by M-mode and pulsed-Doppler echocardiography. Am J Obstet Gynecol 1989; 161:974–7
3. Hameed A, Karaalp IS, Tummala PP, Wani OR, Canetti M, Akhter MW, Goodwin I, Zapadinsky N, Elkayam U: The effect of valvular heart disease on maternal and fetal outcome of pregnancy. J Am Coll Cardiol 2001; 37:893–9

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