SEVERAL authors have focused on change in blood pressure as the key hemodynamic variable during neuraxial anesthesia for cesarean delivery in patients with severe preeclampsia. The optimal anesthetic technique for cesarean delivery in women with severe preeclampsia remains controversial. A more relevant and interesting problem is how we can improve monitoring of other hemodynamic parameters in preeclamptic parturients. The article by Dyer et al. in this number of ANESTHESIOLOGY introduces two interesting issues.

First, the authors focus on cardiac output as the variable of greater interest for assessment of the circulation. Though often edematous, untreated preeclamptic patients typically have a small plasma volume, reduced diuresis, hypertension, and vasoconstriction compared with healthy parturients. Dyer et al. find considerable heterogeneity in cardiac output among the patients included in their study, ranging from less than 4 l/min to more than 10 l/min, and a correspondingly large variability in systemic vascular resistance. This could be due to different preoperative treatments and to different responses to treatment. Using pulmonary artery catheters, Visser and Wallenburg found smaller hemodynamic variability in 87 untreated preeclamptic patients compared with treated patients. Dyer et al. argue that keeping blood pressure at baseline is not an optimal strategy and that maintaining cardiac output is a better approach. They report no measures of uterine circulation in the current study to verify that cardiac output is more important than blood pressure for maintaining uterine blood flow, but other studies have demonstrated that increasing cardiac output is beneficial for uterine blood flow. Valensise et al. showed an inverse correlation between cardiac output and uterine resistance index in healthy pregnant women. They recently published a study showing increased cardiac output and improved uterine blood flow after plasma volume expansion and a nitric oxide donor in hypertensive pregnant women. These authors argue that previous studies have focused on blood pressure without sufficiently considering the effects on cardiac output and systemic vascular resistance.

Second, Dyer et al. use a new minimally invasive technique (the LiDCOplus; LiDCO Ltd., Cambridge, United Kingdom) for hemodynamic monitoring. The LiDCOplus is a cardiovascular monitor, providing continuous measurement of cardiac output and derived variables. This is achieved by two proprietary algorithms: a continuous arterial waveform analysis system (PulseCO) coupled to a single-point lithium indicator dilution calibration system (LiDCO). The technique requires only peripheral arterial and venous cannulation. Although the device has been validated in other patient groups, few data have been published using this device in pregnant women. The impact of delivery on the cardiac output measurements provided by this technology may be substantial. Using this device in pregnant women for the past 3 yr, we have experienced that recalibration is necessary after delivery. The recommendation by the manufacturer to recalibrate every 8 h is, in our opinion, not adequate in pregnant women during delivery.

Compared with previously available invasive techniques (i.e., the pulmonary artery catheter), the threshold for using new minimally invasive techniques should be low. Previously, invasive hemodynamic monitoring in severe preeclamptic patients has been recommended only in those patients with complications, such as pulmonary edema and renal failure. With more available invasive techniques, one might reduce the incidence of serious complications by using the hemodynamic information to guide the optimal treatment.

In healthy pregnant women, we found that 5 U oxytocin increased cardiac output and decreased systemic vascular resistance by around 60% and caused a decrease in systolic blood pressure by 33% compared with baseline. Preeclamptic patients may respond differently to oxytocin compared with healthy parturients with smaller compensatory increase in cardiac output as a response to the massive vasodilatation caused by oxytocin. Dyer et al. found no significant increase in stroke volume in their patients who received 2.5 U oxytocin. There was apparently less increase in cardiac output after oxytocin in patients with low cardiac output compared with those with high values. Controversies about oxytocin use often arise from traditional preferences in...
different hospitals. In parturients with cardiac disease with invasive monitoring of cardiac output, we give oxytocin as repeated boluses of only 0.1 U until the uterus contracts, rarely exceeding a total of 0.5–1.0 U. Even with these small doses, one observes minor short-lasting hemodynamic changes. Carvalho et al.\textsuperscript{14} found that the ED\textsubscript{95} of oxytocin for adequate effect on uterus contraction is 0.35 U. These findings should be of consequence for clinical practice.

New methods for continuous invasive monitoring of cardiac output may herald a new era in which we focus on blood flow instead of blood pressure as the key hemodynamic variable in obstetric anesthesia. This will provide more information and ultimately a better understanding of the hemodynamic changes in women with severe preeclampsia. This may improve our management of severe preeclamptic patients with respect to choice of fluids, diuretics, and vasoactive substances and allow tailored treatment in this heterogenous patient group. Improved hemodynamic monitoring in patients with severe preeclampsia may help to reduce the risk of peripartum pulmonary edema, renal failure, and cerebral complications.

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\textbf{References}