In Reply:

We thank Dr. Gambling et al. for their interesting and pertinent comments. Parturients presenting for spinal anesthesia for elective cesarean delivery are not only fluid replete but also have the expanded blood volume of pregnancy. In a recent investigation, we demonstrated that the initial 20% decrease in mean arterial pressure after induction of spinal anesthesia was associated with a partial compensatory increase in maternal cardiac output mediated by an increase in stroke volume and heart rate, provided adequate lateral tilt was used and a rapid crystalloid colloid was administered.1 In this scenario, phenylephrine seems to be the ideal initial vasopressor, in that the correction of systemic vascular resistance reduces cardiac output and increases blood pressure to levels approaching baseline values. In addition, blood pressure is restored more rapidly after phenylephrine than ephedrine, and this could prevent maternal symptoms, in particular nausea and vomiting. Heart rate was found to be a good surrogate marker of maternal cardiac output. Doses of phenylephrine causing a marked increase in blood pressure and sinus bradycardia significantly depress maternal cardiac output below baseline values and should be avoided.

We agree that, in certain circumstances, the rapid onset β-adrenergic effects of ephedrine have an important role to play. In particular, in the small proportion of patients in whom the response to spinal anesthesia is bradycardia and hypotension (which indicates a decreased cardiac output), anticholinergics or ephedrine would seem to be a much better choice, once the uterus has been adequately displaced. In low-resource environments in which preoperative maternal assessment is sometimes less than ideal, the beta effects of ephedrine may be important in undiagnosed cases of ventricular dysfunction in which the hemodynamic response to spinal hypotension is inadequate.2

We agree that in most cases of spinal hypotension in which the fetus is healthy, the minor degree of fetal acidosis induced by ephedrine is probably clinically insignificant. Indeed, a minor degree of stimulation of metabolic activity may be beneficial.3 However, it is probable that if the fetus is compromised and there are further complications such as maternal hypotension and a long uterine incision to delivery time, large doses of ephedrine are to be avoided because an increase in fetal metabolic rate could adversely affect the oxygen supply to demand ratio. It should also be noted that the median doses of vasopressor used predelivery in the recent article to which Dr. Gambling alludes, namely 61 mg of ephedrine and 1,300 µg of phenylephrine, are considerably higher than those used by most clinicians in clinical practice.4

Therefore, in summary, we agree with Dr. Gambling that phenylephrine given in doses adequate to restore the baseline heart rate is the vasopressor of choice in most cases and that ephedrine has an important role to play when indicated by the maternal hemodynamic response to spinal anesthesia.

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