CORRESPONDENCE

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Spinal Anesthesia in Preeclamptic Patients—"Supportive" Evidence

To the Editor:—I congratulate Hood and Curry[1] for attempting to provide outcome-based evidence "supporting" the routine use of spinal anesthesia in severely preeclamptic patients. I agree, the evidence is supportive, but not "convincing." Hood and Curry[1] and Santos[2] appropriately discuss the weaknesses and applicability of the data from a retrospective study. I would like to discuss three points.

First, there were no guidelines as to induction of epidural anesthesia, specifically (1) volume of local anesthetic injected; (2) time course for dosing; and (3) physiologic end point (*e.g.*, sensory dermatome). Is injecting up to a "10 ml bolus" of epidural local anesthetic, especially 3% 2-chloroprocaine, analogous to inducing spinal anesthesia? If yes, then why should there be a difference in outcome between spinal and epidural anesthesia?

Second, Hood and Curry[1] theorize greater levels of pain-induced circulating catecholamines in laboring as compared with nonlaboring patients. With induction of spinal anesthesia, secretion of circulating catecholamines is suddenly attenuated, leading to maternal hypotension greater than what is seen in nonlaboring patients.[3] If this is so, then why are baseline maternal blood pressures similar between laboring and nonlaboring patients?[1,3] Does blood pressure decrease less after induction of regional anesthesia in laboring patients than in nonlaboring patients?[4,5]

Third, why were only 65% of severely preeclamptic patients receiving magnesium sulfate therapy? Magnesium sulfate therapy causes (1) vasodilation of uterine and systemic arterial vasculature[6,7]; (2) worsens hypotension seen with hemorrhage[8] (*e.g.*, at delivery) and induction of epidural anesthesia[9]; and (3) attenuates the effects of catecholamines, including angiotensin II, on systemic and uterine vasculature.[10] Could magnesium sulfate influence the degree of maternal hypotension and volume of hydration and dose of ephedrine infused in response to the more rapid onset of sympathectomy accompanying spinal anesthesia? What was the magnitude of the decrease in blood pressure in the patients who received magnesium sulfate *versus* those who did not receive this drug? Are the results of the study by Hood and Curry[1] applicable to my preeclamptic patients who are uniformly (*versus* 65% of the time) treated with magnesium sulfate?

It is commonly accepted that regional anesthesia is "safer" than general anesthesia in a severely preeclamptic patient.[2] In the authors' superb care, spinal anesthesia probably is as "safe" for the mother as epidural anesthesia. However, the other product of our endeavors is neonatal outcome; to that end, I am encouraged by their neonatal outcome data. As the authors imply, there are still practitioners who believe that even aggressively treated episodes of maternal hypotension may be detrimental to a relatively "ischemic" (especially in a preterm), fetoplacental unit. I must admit that I should be counted among this dwindling number! I still await a prospective analysis to "convince" me that spinal anesthesia and epidural anesthesia are equal and interchangeable techniques in severely preeclamptic patients.

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