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Low-dose *versus* a Higher-dose Bupivacaine Spinal Anesthesia for Cesarean Delivery

To the Editor:—We read with interest the article by Langesæter *et al.*[1] that investigated the hemodynamic effects of a low-dose *versus* a higher-dose bupivacaine spinal anesthesia for cesarean delivery.

While the LiDCOplus (LiDCO Ltd., Cambridge, United Kingdom) monitor for continuous hemodynamic measurements seems promising because of its minimal invasiveness, the use of low-dose bupivacaine for spinal anesthesia during cesarean delivery poses several practical questions.

First, we would like to remark that among the various methods studied while incurring less frequent hypotension during cesarean delivery with spinal anesthesia, the only technique to date that has been shown to be effective is the combination of high-dose phenylephrine and crystalloid cohydration.[2]

Our primary concern regarding the study by Langesæter *et al.*[1] is the high incidence (7.5%) of incomplete spinal block encountered with the low-dose local anesthetic. Also, we wonder why the upper target sensory level was T8 and not T4-5, and what the actually recorded upper sensory level of the block with both doses of spinal anesthesia was.

In addition, from a practical and safety point of view, it seems illogical to administer prophylactic phenylephrine with a systolic blood pressure of 140 mmHg.

The concern that the hemodynamic stability might come on the account of the quality of anesthesia is further emphasized by Ben David *et al.*,[3] who found that with low-dose bupivacaine plus fentanyl, 8 out of 16 patients noted transient pain or pressure with stretching of the incision and/or with uterine fundal pressure at delivery.

We believe that a low-dose spinal anesthesia for cesarean delivery should only be employed with the combined spinal-epidural approach where epidural supplementation is feasible (as it was done in the present study). However, such an epidural supplementation may lead to hemodynamic instability by itself. If spinal anesthesia has to be supplemented with epidural local anesthetics, then a rapid-onset local anesthetics such as lidocaine is the preferable option.

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In Reply:—We thank Ngan Kee and Khaw for their response to our study[1] concerning invasive hemodynamic monitoring during spinal anesthesia for C-section. Some of the apparent disagreements seem to depend on the misinterpretation that we recommend a clinical practice identical with the strict study algorithm used in an experimental study.

We do not recommend an arterial line as routine in healthy parturients undergoing C-section. This was a study conducted to investigate the maternal hemodynamic changes induced by different variants of spinal anesthesia with the use of invasive monitoring. By using continuous invasive monitoring, we showed the immediate and prominent hemodynamic changes associated with spinal anesthesia in this patient group. This study documented that cardiac output increases—not decreases—after spinal anesthesia, which is contrary to previous assumptions published regarding the effects of spinal anesthesia.[2] This is the first study describing continuous invasive monitoring in healthy pregnant women during spinal anesthesia for C-section. Spinal anesthesia has theoretically been assumed to decrease cardiac output based on the reflex bradycardia induced by reduced preload on the cardiovascular receptors in the vena cava, right atrium, and left ventricle. The vasodilatory effect of sympathectomy has been thought to be moderate. Our study demonstrated a large decrease in systemic vascular resistance, and also that the changes were immediate within less than 3 min. To counteract these prominent and immediate changes, vasopressor can be given prophylactically, and the logic choice of vasopressor is an α-agonist, which counteracts the vasodilatory effect. Ephedrine, which mainly acts as a β-agonist, would further increase cardiac output. Ngan Kee and Khaw misinterpret us when they say we suggest to delay or withhold administration of phenylephrine to permit a decrease in blood pressure below baseline. On the contrary; we recommend starting phenylephrine prophylactically simultaneously with the spinal anesthesia, as we did in our study. In clinical practice, we also suggest giving a bolus of phenylephrine at start, in addition to the infusion, to further reduce the rapid hemodynamic changes observed in our study. Thus, the recommendation by Ngan Kee *et al.*[3–5] of prophylactic phenylephrine infusion during spinal anesthesia for C-section is supported by our article. When we designed our study, we did not know that we would demonstrate such immediate hemodynamic changes in less than 3 min. If we were to conduct this study today, we would have included a start bolus of phenylephrine, based on these findings.

In our study, a bolus of phenylephrine 30 μg IV was given as rescue medication when systolic blood pressure was below 90 mmHg. Ngan Kee and Khaw compare our invasive beat-to-beat blood pressure recordings with their results obtained by noninvasive intermittent blood pressure measurement.[3] We do not think such direct comparison is justified. In their study, 96% of patients had one or more intermittent measurements below their target of 80% of baseline systolic blood pressure, with accompanying nausea and vomiting in 40%. Obviously, their low-height patients receiving a large dose of spinal bupivacaine had considerably more prominent hemodynamic instability, as compared with our study patients. In our group receiving low-dose bupivacaine and low-dose phenylephrine, the mean maximum decrease in systolic blood pressure was 17%, measured with a beat-to-beat technology. With an intermittent noninvasive technique, these changes might not have been detected.