
Effect of prophylactic ondansetron and/or continuous infusion of phenylephrine on spinal anesthesia-induced hypotension

Sir,

We read with interest the article by Ortiz-Gómez *et al.* reporting the effect of prophylactic ondansetron and/or continuous infusion of phenylephrine on spinal anesthesia-induced hypotension; we acknowledge their work.^[1] The investigators reported, in a double-blind, randomized, placebo-controlled trial, a reduction of 50% in the incidence of maternal hypotension during elective caesarean delivery. We appreciate that the authors mentioned the usually forgotten reverse Bainbridge reflex with the well cited one study by Bezold-Jarisch in an attempt to probably explain the prophylactic effect of ondansetron on the hypotension associated with spinal anesthesia.^[2] At the same time we must mention that ondansetron, expected to inhibit hypotension and bradycardia via inhibiting the Bezold-Jarisch reflex, triggered many cases of severe bradycardia and even cardiac arrest.^[3]

We do not fully share with the authors the opinion when they stated that the main contributors to hypotension with sympathetic nerve blockade are the decreases in cardiac output and systemic vascular resistance. Studies have proven many years before the publication of the article in question that the typical hemodynamic effects of spinal anesthesia in healthy pregnant women are a decrease in the systemic vascular resistance and a compensatory increase in cardiac

output, which might increase again after fetal extraction by cancellation of uterine aortocaval compression syndrome.^[4] These scientific facts are made possible by the utilization of minimally or noninvasive monitoring of cardiac output whose increase is the result of a partial rise in stroke volume and tachycardia.^[4,5] Thus, phenylephrine is the first-line vasopressor from physiological point of view, regardless of the effect of both phenylephrine and ephedrine on the fetal acid-base status.^[4,5] However, when hypotension is associated with bradycardia, ephedrine becomes the recommended option.^[5]

We do have also a great concern about the results reporting significant differences between the groups in heart rate. After a thorough glance to these findings, we noticed that atropine was not administered at all in the control and ondansetron groups but was given in 25.4% and 19.1% in phenylephrine and ondansetron plus phenylephrine groups, respectively. For us this is a proof that bradycardia seen in the latter two groups was a reactionary response to blood hypertension caused by continuous phenylephrine infusion and mediated by arterial baroreceptors.^[2] Another direct implication is that the phenylephrine dosage used in the present trial was causing hypertension rather than preventing hypotension as was the aim of the investigators.

Last but not least, pregnant women are now classified ASA 2 instead of ASA 1 according to the recent ASA classification update.^[6] In fact, physiological changes related to pregnancy influence perioperative management and outcome.

We wonder how the trial was carried out to give solutions for spinal anesthesia-induced hypotension, and at the same time, we remark in the demographic and anesthetic data that dural puncture to skin incision and skin incision to fetal extraction are both lasting for 10 min in average. This means that delivery took in the study several minutes which is known to have possible negative impacts on maternal and fetal prognosis even under spinal anesthesia.

Finally, we really appreciate the authors' initiative to accomplish this work which will certainly help to clarify and animate the debate about spinal anesthesia-induced hypotension as was suggested by meta-analyses.^[7]

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Conflicts of interest

There are no conflicts of interest.

**SALAH MHAMDI, MOHAMED KAHOUL,
ALAEDDINE HAFSA¹, ALI MAJDOUB²**

Department of Anesthesia and Intensive Care,
Teaching Hospital of Sahloul, University of Medicine,

¹Department of Anesthesia and Intensive Care,
Teaching Hospital of Farhat Hached,

University of Medicine Ibn Al Jazzar, Sousse,

²Department of Anesthesia and Intensive Care,
Teaching Hospital of Taher Sfar Mahdia,
University of Medicine Fattouma Bourguiba,
Monastir, Tunisia


Address for correspondence:

Dr. Salah Mhamdi,
Department of Anesthesia and Intensive Care,
Teaching Hospital of Sahloul, Sousse, Tunisia.
E-mail: dr_mhamdi@yahoo.fr

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