

In Response:

We thank Dr. Mazze for his interest in our article (1). According to Miss Konno, assistant manager, Tokyo Sales Department, Dräger Japan (Tokyo, Japan), Dräger (Luebeck, Germany) changed the old CO₂ absorbent, "Drägersorb 800" to a new absorbent "Drägersorb 800 plus" in 1999 all over the world. As indicated in our article (1) and Dräger Medical's material safety data sheet, Drägersorb 800 contains some NaOH (1%–2%) and KOH (2%–3%), whereas Drägersorb 800 plus contains some NaOH (1%–2%) and only trace amounts of KOH (0.003%). We prepared these old and new CO₂ absorbents in our study and measured Compound A concentrations under low-flow sevoflurane anesthesia using four absorbents, including Drägersorb 800 and Drägersorb 800 plus (1).

We were also surprised by the results of Stabernack et al. (2), because it has long been thought that KOH has a greater involvement than NaOH in the production of Compound A (3). Considering the results obtained by Stabernack et al. (2), the difference in the production of Compound A among the absorbents cannot be explained by base composition. Factors other than base composition might be involved. In any event, all studies were in agreement in that Amsorb (Armstrong Medical Ltd., Londonderry, UK) did not cause the degradation of sevoflurane (1,2,4,5).

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Note: Drs. Eger and Yamakage chose not to respond.

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Absence of Reflex Tachycardia After Spinal Anesthesia in the Elderly

To the Editor:

We have read with interest the recent dose-response study of IV atropine after spinal anesthesia in elderly patients (1). The authors hypothesized that depressed reflex tachycardia might contribute to the pathogenesis of hypotension and demonstrated that a small dose of IV atropine effectively reduced the need of ephedrine to treat predefined, spinal anesthesia-induced hypotension in patients >60 yr of age. Their finding was clinically useful; however, their explanation for the depressed reflex tachycardia raises some questions.

First, the authors stated that the absence of reflex tachycardia might result from the blockade of cardioaccelerator sympathetic fibers with a reference (2), although higher level of sensory analgesia had not been determined in this article. In fact, we frequently observe in elderly patients after subarachnoid blockade that no clinically significant increase in heart rate (HR) occurs even with the higher level of analgesia below T10 and with clinically significant hypotension, which would otherwise produce HR increases in younger patients. Second, the authors also speculated "reverse" Bainbridge reflex as another etiology. However, physiological importance or even the existence of Bainbridge reflex is questionable in humans (3–5). Rather, depressed arterial baroreflex control of HR (6,7) and age-dependent reduction of β_1 -receptor-mediated responsiveness (8) are more likely explanations for the attenuated HR response to spinal anesthesia-induced hypotension in the elderly.

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In Response:

Thank you for the constructive comments from Drs. Tanaka and Nishikawa. We agree that our study raised more questions than the study results could answer (1). Hypotension and lack of reflex tachycardia during spinal anesthesia are very common and we believe that the cause for this phenomenon is likely to be multifactorial. Although our study demonstrated that a lack of reflex tachycardia after spinal anesthesia could be a contributing factor for the hypotension, it also showed that it could not be the only factor in causing the hypotension. The exact mechanism and cause for the relative bradycardia during spinal anesthesia remains unknown and speculative. Blunting of the baroreceptor reflex is a well-known phenomenon in the elderly patients. Although we agree that it may be a contributing factor, we do not believe it is the most important factor. We observed similar phenomenon in younger patients undergoing spinal anesthesia. In fact, Ure et al. (2) demonstrated the same phenomenon in a group of young obstetric patients undergoing spinal anesthesia. In that study, the use of IV glycopyrrolate increased the heart rate and also reduced the severity of hypotension.

In addition, the level of sympathetic denervation in neuraxial anesthesia is even higher than the level of sensory anesthesia (3). Hence, blockade of cardioaccelerator fibers may still be one of the factors contributing to hypotension in our patients.

In summary, while blunting of baroreceptor reflex may be a contributing factor in causing bradycardia during spinal anesthesia in the elderly, it cannot be the only factor. In fact, this phenomenon is likely to be caused by more than one factor.

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End-Tidal Carbon Dioxide Tracing Configuration Depends on Sampling Size

To the Editor:

Placing a filter in the anesthesia circuit has become popular in our institution and in others as an effective way to preserve the heat and