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Reply:

Thank you for the opportunity to respond to Dr. Mannion's letter in which he describes that "epidural spread after posterior lumbar plexus block (PLPB) depends on the approach used," and suggests that the technique used by the author, and described by Capdevila and colleagues,[1] is preferable to the approach used in our patient[2] in minimizing epidural spread and thus adverse hemodynamic consequences, particularly in patients with severe aortic stenosis.

Epidural spread with bilateral symmetrical[3] or unilateral[1] anesthesia after PLPB has been reported, but as acknowledged by Dr. Mannion the extent to which epidural spread occurs or factors that contribute to epidural spread after PLPB are still not defined. In our experience, and that of others, this is variable and multifactorial and may be related to the position of the needle prior to the injection, unintended injection close to the intervertebral foramen via a medially directed needle, accidental epidural injection via a misplaced catheter,[4] large volume of local anesthetic used, and the presence of spinal deformity (e.g., scoliosis),[4] etc. Our patient did not have any obvious spinal deformity and we chose the L3 approach with no medial direction of the needle, as described by Parkinson et al.,[3] because L3 has the longest lumbar transverse process and allows the injection to be made furthest away from the intervertebral foramina, thus minimizing epidural spread. Parkinson et al. report bilateral symmetrical anesthesia in 16% of patients using the above approach with an initial injection volume of 0.5 $mL \cdot kg^{-1}$.[3] Interestingly, although the point of needle insertion, as described by Capdevila et al.,[1] is more medial than in our patient, unilateral epidural anesthesia was seen in only 6.5% of patients,[1] suggesting that factors other than the approach may be responsible. Also of note is the lower initial volume and dose (0.4 $mL \cdot kg^{-1}$ of 0.2% ropivacaine) of local anesthetic used.[1] We used only 15 mL (0.3 $mL \cdot kg^{-1}$) of ropivacaine 0.5% for the PLPB and there was no clinical evidence of epidural anesthesia in our patient,[2] the drop in blood pressure in our patient notwithstanding. Moreover, the propofol infusion used for sedation during the procedure may also have been partly responsible for the drop in blood pressure. Therefore, before we can recommend a certain approach or technique of PLPB as being superior to another in causing less epidural spread or adverse hemodynamic consequences, further research is required.

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Nociception is the root cause of postoperative nausea and vomiting: hypothesis

To the Editor:

Postoperative nausea and vomiting (PONV) has long plagued clinicians as a problem in search of a solution. It has caused discomfort and dissatisfaction to patients across the globe. Nevertheless, little light has been shed on its etiology during the operative period. In light of this unclear etiologic picture, clinical focus often is wrongly placed on pharmacologic antiemetics for prophylaxis.

This author's hypothesis regarding the cause of PONV is as follows: "iatrogenic nociception is the root cause of intraoperative genesis of PONV and a sense of malaise. Nociception confers a predisposition to PONV in the face of a depth of anesthesia not commensurate with suppression of nociperception by brain." Nociception herein is defined as a deleterious effect on the brain secondary to injury or a noxious agent or stimulus anywhere in the body. Nociperception is