Response to Letter Regarding Article, “Intraoperative Magnesium Administration Does Not Improve Neurocognitive Function After Cardiac Surgery”

We thank Dr Derakhshan for his interest in our recently published study on magnesium and postoperative cognitive decline. The questions he raises on handedness and hemispheric dominance have already been elegantly addressed in a previous response to a similar query. In essence, handedness may be unreliable as a representation of hemispheric specialization.

Dr Derakhshan also refers to the work of Messerotti Benvenuti et al in asserting that flow to the right hemisphere is not relevant to postsurgical outcome. We think that such an assertion may be quite premature. Although the demonstration by Messerotti Benvenuti et al that preoperatively reduced cerebral blood flow velocity in the left (but not the right) middle cerebral artery was associated with postoperative cognitive decline is certainly intriguing, it should be considered preliminary for several reasons. This study involved only 31 patients evaluated with a limited cognitive battery; did not account for baseline cognition or intraoperative cerebral blood flow velocities; and assessed cognition only at hospital discharge. In a follow-up study of the same patients assessed at 3 months after surgery, the association between cerebral blood flow velocity and postoperative cognitive decline was no longer significant (P=0.08).

To their credit, Messerotti Benvenuti et al urged caution in interpreting their findings because “it seemed unlikely that neuropsychological tests activate the left hemisphere in isolation” and supported the need for a validation study with broader assessment (eg, visuospatial tasks).

The most puzzling comment in the letter from Dr Derakhshan is his critique of our expectation that magnesium treatment would reduce the incidence of cognitive deficits to 25%. It would seem that he is simultaneously arguing that the cognitive deficit rate can never exceed 50% and that it cannot be lowered. If cortical hypoperfusion is in fact a significant predictor of postoperative cognitive decline, magnesium-induced vascular smooth muscle relaxation would improve cerebral blood flow and potentially lower the cognitive deficit rate. As we describe in our study, magnesium has also been reported to exert neuroprotective properties via other mechanisms independent of blood flow, including preservation of cellular energy metabolism, noncompetitive inhibition of the N-methyl-D-aspartate receptor, attenuation of presynaptic excitatory amino acid release, potentiation of presynaptic adenosine, and blockade of voltage-gated calcium channels. For all of these reasons, it is entirely logical to postulate that intraoperative magnesium administration would reduce postoperative neurocognitive dysfunction.

Disclosures

None.

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