Commentary: Optimizing cerebral oxygenation in cardiac surgery: Neurocognitive and perioperative outcomes

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Central Message

Larger, more adequately powered, interventional randomized controlled trials are needed to more fully examine the influence of cerebral oximetry guidance on the optimisation of both cerebral and noncerebral outcomes after cardiac surgery.

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Uysal and colleagues¹ add a small, although no less important, piece of information to the seemingly ever-expanding puzzle surrounding perioperative neurocognitive disorders (PNDs) and the potential factors that may influence them. Their randomized controlled study has a number of strengths, but it also shares some deficiencies that have frequently been seen in similar investigations of cerebral oximetry to optimize cerebral outcome after cardiac surgery. Although the study appears to include a nominal sample size ($n = 245$), the actual number of patients who had both cerebral oximetry monitoring and baseline neurocognitive testing performed was relatively small ($n = 59$), somewhat reducing the confidence one might have in their conclusions. Indeed, discerning the factors that influence the often subtle changes in neurocognitive function that occur in this setting are challenging in small studies. That said, one of this study's notable strengths is that it interrogated for PND at both 3 and 6 month time points after cardiac surgery. These relatively longer-term postoperative PND end points are consistent with the latest nomenclature guidelines that have been promulgated in an attempt to bring better consistency and reliability to studies of PND.^{2,3} Indeed, although recent meta-analyses have suggested a potential relationship between intraoperative management guided by cerebral oximetry and a reduction in cognitive dysfunction,⁴ the frequent variability in defining these cognitive changes (both in terms of cognitive domains that are being measured and the time points at which the assessments are made) brings considerable heterogeneity to the subject.

Despite the cognitive outcome measurements that Uysal and colleagues¹ report (ie, group mean memory change score) being better at 6 months in the group using cerebral oximetry-guided interventions, the overall presence, duration, and severity of cerebral desaturation

were not associated with the cognitive change scores. On the surface, this may seem counterintuitive. However, the very nature of using cerebral oximetry thresholds suggests that interventions to treat cerebral desaturation are often instituted before the actual threshold is reached; thus, the benefit of the intervention might be seen without a prolonged period of desaturation actually having occurred. Cerebral oximetry also can have a significant impact on other noncerebral organs, and interventions that might not fully reverse the cerebral desaturation could improve other noncerebral outcomes that might affect cognition. Thus, although it is convenient to think that preventing cerebral hypoperfusion/ischemia itself can be of benefit in reducing neurocognitive disorders, the various factors influencing perioperative cognitive changes are complex enough that this is but one aspect that could be influential.

Most importantly, this study provides another glaring example of what is lacking more broadly in the field of cerebral oximetry research. That is, a large adequately powered ($n > 1000$) interventional randomized controlled trial examining the influence of cerebral oximetry guidance to optimize both cerebral and noncerebral postoperative outcomes is desperately needed. Before we have such a large study, which is disappointingly still lacking despite more than 2 decades of clinically available cerebral oximeters,

we will not be able to more fully understand the impact of this potentially important monitoring technology on patients undergoing cardiac surgery.

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