Neurocognitive decline in cardiac surgery—Distraction rather than destruction?

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Abstract

Background: A neurocognitive decline is an undesirable event that can be observed in patients after cardiac surgery. It has been related to the use of cardiopulmonary bypass (CPB). Minor embolic or hyperinflammatory mechanisms are thought to be responsible. In this issue of the Journal of Cardiac Surgery, the neurocognitive decline was observed in 22 of 30 patients after cardiac surgery with CPB. Repeatable neuropsychological status tests were used and scores 4 days after surgery were 5%–15% lower than before. Mechanistic investigations with glycemic control and transcriptomic and cytokine analyses failed to provide an explanation but the frequency of this observation is worrisome.

Discussion: However, available evidence suggests that neurocognitive dysfunction disappears within a few months, and later on no difference to controls that did not undergo surgery can be detected. In addition, similar degrees of neurocognitive dysfunction can be observed after noncardiac surgery and even after percutaneous coronary intervention (PCI). A most recent comparison of memory decline after CABG and PCI also suggests no difference between the two invasive treatment options for coronary artery disease. All these findings argue against a primarily CPB-associated mechanism. Interestingly, test subjects from a consumer investigation showed a 10% decline in their working memory just by placing their cell phone on the table, suggesting that being distracted may also affect neurocognitive function. Given the reversibility of surgery- and intervention-associated neurocognitive dysfunction, we question destructive, embolic, or inflammatory-associated mechanisms. Distractive aspects of intervention-associated stress may also play a role.

Conclusion: In any case, neurocognitive decline after cardiac surgery does not appear to be surgery-specific.
COMMENTARY

Cardiac surgery became a reproducible treatment option for many patients around 50 years ago. For the following three decades, there were no or few treatment alternatives for many patients and cardiovascular practice was characterized by the documentation of risks and activities to improve outcomes.

Cardiopulmonary bypass (CPB) is a key component in most surgical procedures, as it provides the basis for the safe conduct of all the different techniques described to address a cardiac condition today. However, CPB has also been associated with undesirable adverse events by causing, for instance, coagulation disorders, inflammatory response syndrome’s or neurological events in varying degrees. Even mild changes in neurocognitive function are often attributed to the use of CPB, suggesting that minor particulate or minor air embolism affects cerebral function. For decades such conditions (also known as “air head” or “pump head”) were considered a given risk and “part of the package.” With the advent of alternative treatment strategies with an ever-growing portfolio of interventional techniques not requiring CPB, such complications may receive new value and new attention.

In this issue of the Journal, Scrimgeour et al. assessed neurocognitive decline in patients undergoing cardiac surgery using CPB. They performed Repeatable Battery Assessment of Neuropsychological Status tests pre- and 4 days postoperatively. The test assesses changes in short and long-term memory, language, attention, global cognition, and visuospatial function. They found that 22 of 30 patients examined showed significant declines in neurocognitive function. Scores four days after surgery were often 5%–15% lower than before surgery. The authors excluded a possible relation to glycemic control but were unable to provide more revealing insights despite sophisticated transcriptomic and cytokine analyses. Irrespective of possible molecular mechanisms explaining this finding, the fact that three-quarters of patients appear to experience neurocognitive decline after cardiac surgery is highly disturbing and requires deeper investigation.

The good news upfront: despite being a frequent clinical condition, the neurocognitive function is usually restored after 3 months, and at 1 year, cognitive function is indistinguishable from matched controls. In addition, postoperative neurocognitive dysfunction is common in all kinds of surgery and its incidence has been described to be 5%–15% and in certain high-risk groups (such as hip fracture patients), it may reach over 60%. Thus, the association to the use of CPB may not be as clear as we thought.

The neurocognitive decline has also been reported in interventional patient populations. In patients with NSTEMI, for instance, undergoing percutaneous coronary interventions (PCIs), its incidence has been reported with 28.8%. A recent cohort study from the American Health and Retirement Study on 1680 patients above 65 years of age assessed the change in the rate of memory decline after CABG or PCI. The authors found no difference between the two treatment options in the rate of memory decline. They did not compare the results to a cohort that did not undergo invasive coronary artery diseases treatment, but the lack of differences in this long-term outcome between CABG and PCI questions again a specific technical aspect of surgery as the sole cause of neurocognitive dysfunction.

The current literature suggests that neurocognitive outcomes may be due to a hyperinflammatory state, which may be the common denominator for all these observations. Scrimgeour et al. also based their hypothesis on such an inflammatory scenario and therefore investigated diabetic patients and glycemic control, where a proinflammatory condition is supplemented by the use of CPB. Their findings as well as the other observations of neurocognitive dysfunction in the absence of CPB question CPB as a key contributor to the incidence of neurocognitive dysfunction and argue against a strictly embolic nature of this complication.

While the present study is too small to draw definitive conclusions, the findings may be used to question the role of cytokines, gene expression, and glycemic control as causes of perioperative cognitive dysfunction. Future studies need to address the relation of inflammatory states to neurocognitive decline and brain function as a whole. Importantly, they need to do this addressing the question of whether the decline is reversible and whether reversibility or lack of it is linked to cellular damage with or without recovery potential.

This line of thinking may support an alternative assessment of this phenomenon. Embolic and/ or inflammatory mechanisms may trigger loss of cerebral function by the destruction of cerebral tissue or cells. But, what if surgical stress affects the allocation of cognitive capacities, so that neurocognitive decline 4 days after cardiac surgery would not be the result of the destructive embolic or inflammatory but rather a distractive mechanism. It is interesting to review a recent publication from the consumer/marketing area. Ward et al. subjected consumers to mental ability tests in the presence or absence of their cell phones. It may be striking to note that the test subjects’ working memory or ability to solve riddles experienced a decline of around 10% of their cell phone was on their desk rather than in the room next door.

It may be accidental that the amount of functional decline due to this distraction was similar to the neurocognitive decline measured in the above study 4 days after cardiac surgery using CPB, where a distractive mechanism is entertained. Although the many different tests and characteristics used and assessed may not be directly comparable, such a consideration may be able to explain why postoperatively noted neurocognitive decline is temporary, reversible, not affected by diabetes mellitus, and not different between surgical and other interventional procedures. We, therefore, would like to ask, whether neurocognitive decline after cardiac surgery may be more due to mental distraction than cellular destruction. “May be”, but in the absence of convincing evidence, our thoughts may also be a distraction from possible destruction. In any case, the phenomenon is there, but it is not necessarily surgery-specific.

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CONFLICT OF INTERESTS
The authors declare that there are no conflict of interests.
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