Cerebral Air Embolism

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Objectives:

At the conclusion of this lecture, the participants should be able to:

1. Recognize the potential sources for arterial air embolism in cardiac surgical patients.
2. Explain the pathophysiology of arterial and cerebral air embolism.
3. Discuss the therapeutic options available for the treatment of cerebral air embolism.

Neurologic and neurocognitive deficits following cardiac surgery utilizing cardio-pulmonary bypass (CPB) remain as common and severe complications with impact to patients’ quality of life. The etiology of these adverse outcomes is most likely multifactorial, including mainly the effects of cerebral emboli and generalized cerebral hypoperfusion next to inflammatory reactions. Cerebral emboli during CPB may present as particulate emboli released from atheromatous plaques during manipulation of the thoracic aorta or as gaseous emboli mainly entrained during open chamber procedures or generated within the CPB-circuit. High-intensity transient signals (HITS) are the ultrasonic signatures of air or solid emboli in the brain circulation as detected by transcranial Doppler (TCD). In patients undergoing open-heart surgery, most embolic signals are likely associated with air microbubbles but an unknown fraction may be represented by solid microemboli.

Cerebral embolism in combination with hypoperfusion exacerbated by ischemia/reperfusion injury are likely to be the primary underlying causes for cerebral injury following cardiac surgery. In addition, inflammation (both cerebral and systemic), cerebral edema, blood brain barrier dysfunction, hyperthermia, and a genetic susceptibility to injury or genetically defined inability to repair following injury have all been implicated. Embolization of particulate and gaseous material into the cerebral microvasculature resulting in focal areas of cerebral ischemia has been well studied, but evidence showing a direct association between cerebral microembolic load and post-operative cognitive dysfunction is conflicting. Gerriets et al.
report that cerebral micro-embolization may contribute to cognitive decline after CABG, which in their hands was measurable 3 months post-operatively. The same authors report that reducing the amount of gaseous emboli using a dynamic bubble trap may mitigate neuropsychological side effects.\(^{16}\) In another recent clinical study that included low-risk patients undergoing CABG surgery, there was no demonstrable correlation between the counts of HITS and POCD.\(^{17}\) However, several methodological issues may account for the variability among studies. These include, but are not limited to small sample sizes, the technique and location where transcranial Doppler was placed, differences in intensity thresholds used to detect HITS, the quality of TCD recordings, and the effects of several confounders such as the duration of CPB and core body temperature.

Ultimately, it may be that quality rather than quantity of cerebral emboli plays a more important role in the pathogenesis of cerebral injury. As discussed, during CPB, both gaseous and solid emboli (ie, fat, clots, platelet aggregates, atherosclerotic plaque material) may be delivered to the brain. While particulate emboli are more likely associated with adverse neurological events such as stroke and perhaps cognitive deficits, they are more frequently released during certain surgical maneuvers. Surgical technical maneuvers designed to reduce emboli production may improve neurobehavioral outcome. This was demonstrated in a study by Hammon et al., who showed that modifications of surgical technique (e.g. single cross-clamp technique, increased venting of the left ventricle, and application of transesophageal and epiaortic ultrasound scanning to locate and avoid trauma to aortic atherosclerotic plaques) was beneficial.\(^{18}\)

Shed blood suctioned from the operative field via standard cardiotomy suction has been shown to contain high levels of lipid microparticles and other cellular debris.\(^{19}\) There is also evidence that lipid microparticles may embolize into the brain as they can be found in cerebral vasculature after cardiac surgery.\(^{20}\) The use of blood salvage devices to process the blood prior to returning it to the venous reservoir may decrease the amount of particulate/lipid-laden material, which most likely originates from the sternotomy incision. Two recent prospective randomized double-blind trials investigating the effect of blood processing via a cell saver system as opposed to cardiotomy suction on cognitive outcome after cardiac surgery addressed this issue. The first trial included 264 patients undergoing CABG and/or aortic valve surgery and failed to show any clinical evidence of a beneficial effect on postoperative cognition.\(^{21}\) In the second trial that also compared the use of cell saver with cardiotomy suction (defined as control), Djaiani et al. report that processing shed blood resulted in a significant reduction in
postoperative neurocognitive dysfunction after CABG surgery in the elderly. There were many differences in the design of these two trials that may have contributed to conflicting results, most notably that Rubens et al. used standardized definitions of cognitive dysfunction, while Djaiani et al did not, and two different cell saver systems with uniquely diverse processing capabilities were used. Reduction of both platelet and coagulation factors through its intrinsic washing processes is a undesirable side effect of the processing of cardiotomy blood and both trials demonstrated an increase in bleeding and transfusion, a risk that may outweigh any potential benefit of this technique.

Gaseous emboli can be created, or enlarged if already present, by factors related to the process of CPB such as augmented venous return. The intrinsic ability of the circuit to allow air entrained from the venous return cannula to pass through the oxygenator varies considerably between manufacturers but remains a significant source of air in the circuit. As significant quantities of air can be entrained into the heart from incompletely de-aired cardiac chambers, flooding the field with CO₂ has been proposed as a potentially preventative strategy. However, studies have demonstrated no benefit of CO₂ insufflation on neurocognitive outcomes.

References:


