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Crit Care Clin 24 (2008) 83–98

CRITICAL
CARE
CLINICS

Mechanisms of Cerebral Injury from Cardiac Surgery

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Cerebral injury is a frequent complication of cardiac surgery, and it has been associated with high mortality, morbidity, hospital costs; an increased likelihood of admission to a secondary care facility after hospital discharge; and impaired quality of life [1–4]. There are various manifestations of perioperative cerebral injury, including ischemic (or, less commonly, hemorrhagic) stroke that occurs in 1.5% to 5.2% of patients, encephalopathy affecting 8.4% to 32% of patients, and neurocognitive dysfunction affecting 20% to 30% of patients 1 month after surgery [1–4]. The range in reported incidences between studies is likely caused by different patient populations (eg, patient age and risk status, types of procedures), diagnostic definitions, and the intensity of clinical surveillance. Contemporary studies using sensitive brain MRI with diffusion-weighted imaging report that as many as 45% of patients who have undergone cardiac surgery have new ischemic brain lesions that are often clinically undetected [1,5].

The prevailing hypothesis, although not definitively proven, is that all forms of injury associated with cardiac surgery (ie, stroke, encephalopathy, and neurocognitive dysfunction) have a similar etiology and that the manifestations depend on the extent and location of brain injury (eg, motor cortex versus areas subserving cognition). Many earlier studies that described long-term neurocognitive changes after cardiac surgery have failed to include a nonsurgical control group [1,6]. In a longitudinal study of

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0749-0704/08/$ - see front matter
doi:10.1016/j.ccc.2007.09.004 *criticalcare.theclinics.com*

patients who had coronary artery disease undergoing either percutaneous coronary interventions or coronary artery bypass grafting (CABG), there were no differences in cognitive measures 36 months after either procedure [7]. These data imply that the effects of cardiac surgery on cognition may be short-lived (ie, approximately 3 months) and that progression of inherent cerebral vascular disease is a more important determinant of long-term cognitive decrements. These results further underscore the low sensitivity and specificity of psychometric testing for detecting cerebral injury in elderly populations with a high prevalence of pre-existing cognitive impairment [1].

This article examines postulated mechanisms for cerebral injury from cardiac surgery. Most emphasis has been placed in the past on the intraoperative interval as being the period of highest cerebral vulnerability. Many clinical cerebral events, however, occur in the postoperative period. the authors have reported, in fact, that more than 20% of clinical strokes occur after recovery from surgery and anesthesia [2,8]. Thus, patients must be considered vulnerable to cerebral injury any time during the perioperative period.

Cerebral embolism

Cerebral injury from cardiac surgery is primarily ischemic, secondary to embolism and/or cerebral hypoperfusion [1]. Primary intracerebral hemorrhage is found on brain imaging in less than 1% to 2% of patients, depending on the type of surgery [9]. Neuronal injury likely is exacerbated by inflammatory processes resulting from cardiopulmonary bypass (CPB) and ischemia/reperfusion [1,10]. Between 30% and 50% of perioperative strokes detected with brain imaging are caused by cerebral macroembolism, likely arising from the ascending aorta [8,11–14]. Encephalopathy and neurocognitive dysfunction are believed to result primarily from cerebral microembolism [1,15–21]. Microemboli are either gaseous or particulate in composition. Gaseous emboli can arise form an open left-sided cardiac chamber or from air entrained into the CPB circuit [1]. The view that microemboli are the proximate cause of neurocognitive dysfunction is based in part on indirect data from retinal angiographic evaluations, autopsy studies, and animal experiments where cognitive end-points were not assessed [1,22,23]. A more direct link is suggested by studies showing that the number of transcranial Doppler detected arterial embolic signals during CPB is related to cognitive impairment after CABG surgery [11]. It is increasingly apparent, however, that postoperative cognitive dysfunction is not explained solely by this mechanism and is likely multifactorial [24–27]. For example, in studies of open-chamber cardiac valve surgery, cognitive decline was not correlated with the number of Doppler cerebral microembolic signals, and reducing cerebral microembolism (eg, with off-pump surgery) does not significantly improve cognitive outcomes [27,28].