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Commentary

Neuroimaging of Cerebral Infarction Associated with Coronary Revascularization

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Coronary artery bypass surgery is the most frequently performed procedure that utilizes cardiopulmonary bypass. Most of the data pertaining to neurologic complications of open heart surgery have been derived from studies of this operation, but they are applicable generally to other forms of cardiac surgery not involving transplantation. Many technical improvements, including membrane rather than bubble oxygenators and in-line filtration, have decreased the risk of introducing macroemboli (>25 μm) into the circulation, although they cannot protect against microemboli of air, fat, and particulate matter [1]. Since the advent of transcranial Doppler imaging as a simple and noninvasive technique for real-time monitoring of intracranial blood flow velocity, two recent studies have described the use of this technique for the intraoperative detection of air and particulate matter microemboli that migrate to the brain during coronary bypass surgery [2, 3]. Pathologic studies in patients and experimental animals following cardiopulmonary bypass surgery have revealed small capillary and arteriolar dilatations (SCADs) ranging from 10 to 40 μm in diameter, most commonly in the cortex and deep nuclei. The speculation that SCADs are sites of previous air or fat emboli needs to be confirmed and correlated with clinical neurologic manifestations [4].

Several retrospective studies on the complications of coronary artery bypass surgery have suggested that the rate of focal cerebral infarction in these patients is from 0.3–2% [5–9]. Prospective studies relying on clinical examination rather than chart review have suggested a higher rate of 4.7–5.2%, of which 2% of patients were severely affected [10, 11]. In the prospective study by Breuer et al. [11] of 421 patients undergoing coronary artery bypass surgery at the Cleveland Clinic, the authors were unable to correlate a large number of preoperative, intraoperative, and postoperative variables with the risk of stroke. These variables included age, prior transient ischemic attack or stroke, carotid bruit, prior myocardial infarction or dysrhythmias, duration of pump time or aortic cross-clamp time, intraoperative hypertension, type of oxygenator, or reoperation within 48 hr. Although the small percentage of strokes may be a factor in limiting the demonstration of statistical significance of some of these variables, only a history of prior stroke approached statistical significance with an increased risk of stroke during coronary artery bypass surgery. However, potential mechanisms of stroke could be identified in 73% of the patients: these included intraoperative and perioperative cardiac arrhythmias, internal carotid artery atherosclerosis, air embolism, carotid artery trauma during internal jugular vein cannulation, aortic atherosclerosis at the site of aortic cross-clamping, and prolonged intraoperative hypotension [12].

Extracranial carotid artery atherosclerosis is often suggested as an important cause of stroke during coronary artery bypass surgery, which presupposes that severe carotid occlusive disease combined with intraoperative hypotension results in cerebral ischemia. However, most perioperative strokes occur in the absence of significant carotid occlusive disease or have a delayed onset in the postoperative period [13]. Intraoperative transcranial Doppler monitoring of ipsilateral middle cerebral artery blood flow during coronary artery

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bypass surgery has failed to demonstrate significant velocity changes in patients with high-grade internal carotid artery stenosis [14]. In a retrospective study that identified 155 stenotic (≥50%) or occluded carotid arteries in 144 patients who had coronary artery bypass surgery, ipsilateral cerebral infarction occurred in 1.1% of patients with 50–90% stenosis, 6.2% of patients with >90% stenosis, and 2% of patients with carotid occlusion [15]. In a recent study, Hertzer et al. [16] randomly assigned a group of 23 patients with symptomatic or bilateral >70% carotid stenosis to staged or simultaneous coronary artery bypass surgery and carotid endarterectomy. Among this small group of patients, who had undergone urgent coronary artery bypass grafting alone because of unstable coronary disease, only two (8.7%) developed a perioperative stroke. Although these small studies have too few patients to permit any statistical analysis, they suggest there is a subgroup of patients with carotid disease who are at a somewhat increased risk for stroke during coronary artery bypass surgery but that our ability to identify them preoperatively remains elusive.

In this issue of the *AJNR*, Hise et al. [17] describe neuroradiologic studies of patients suffering major neurologic events after coronary artery bypass surgery. Their data support the proposal that cerebral embolism is the main mechanism of cerebral infarction in the perioperative and postoperative periods. Cerebral hypoperfusion, as implied by a watershed pattern of cerebral infarction, was much less common and related to a complete ipsilateral internal carotid artery occlusion in at least one patient studied with angiography. Their data also support the observation that intracranial hemorrhage is a distinctly uncommon type of stroke after coronary artery bypass grafting.

Postoperative alteration in consciousness ranging from encephalopathy to prolonged coma is often multifactorial and may be related to drug effects, hypoxia, hemodynamic instability, metabolic derangements, sepsis, and intensive care psychosis. The frequency of clinically detectable diffuse encephalopathy ranges from 3–12% [8, 11, 18]. However, careful neuropsychological testing has demonstrated subtle but significant cognitive deficits occurring in up to 30% of patients [19, 20]. A prospective analysis of multiple preoperative, intraoperative, and postoperative variables [11] demonstrated a correlation with the use of intraaortic balloon pump counterpulsation and pressor agents in the postoperative period, which are both markers of severe hypotension. This suggests that in some individuals undergoing coronary artery bypass surgery, cerebral hypoperfusion may be a more important cause of encephalopathy than are focal neurologic deficits. Multifocal microemboli have also been implicated as a mechanism for diffuse encephalopathy, as suggested by the SCAD descriptions, although one recent study using intraoperative transesophageal echocardiography demonstrated that microbubbles, often detected during surgery, were not predictive of postoperative neurologic complications [21]. Further neuroradiologic investigation is desperately needed in this area, perhaps employing MR imaging as a high-resolution method for detecting microemboli.

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