Editorial

Stroke and Encephalopathy After Cardiac Surgery
The Search for the Holy Grail

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See related article, pages 562–571.

The incidence of clinically obvious strokes after coronary artery bypass graft operations (CABG) is reported to be between 0.8% and 5.2%. It is estimated that between 5000 and 35 000 new strokes develop as a result of this procedure, which possibly makes coronary artery bypass surgery the single largest cause of iatrogenic stroke in the United States.1 The typically poor postoperative course of patients who develop stroke after cardiac surgery underlines the need for timely recognition, prevention/modification of factors that predispose to stroke. In the present study, McKhann et al2 are touching on the mechanisms, risk factors and outcomes of postoperative stroke after cardiac surgery. They also suggest possible algorithms for management of postoperative stroke and ways to prevent the occurrence of stroke after cardiac surgery. Previous authors have identified several preoperative, intraoperative, and postoperative risk factors of stroke after cardiac surgery, such as episodes of hypotension during or after the operation (requiring inotropic support or placement of intra-aortic balloon pump), atrial fibrillation, carotid artery disease, history of cerebrovascular accident, manipulation of aorta and others. Preventive strategies such as routine screening of patients for carotid artery disease with carotid duplex are useful. The application of diffuse-weighted MRI to diagnose silent brain infarcts will be more cost effective if applied to the patients identified as high-risk for postoperative stroke, such as elderly patients, patients with low ejection fraction, atrial fibrillation, diabetes, hypertension, and carotid artery disease. This subset of patients will benefit the most by intraoperative maneuvers, such as use of epiaortic scanning, the “no touch” technique to avoid manipulation of the aorta, higher perfusion pressures or use of arterial line filters.

Previous studies have suggested that coronary bypass surgery without cardiopulmonary bypass is associated with a lower risk of stroke.3,4 To date, there is no randomized trial evidence to suggest a lower incidence of stroke after off-pump surgery. The timing of occurrence of stroke is different between the 2 approaches suggesting different pathophysiological mechanisms. Embolic phenomena have been previously implicated in the pathophysiology of stroke after On-pump CABG, whereas myocardial stunning and hypoperfusion may be possible mechanisms associated with delayed onset of stroke after Off-pump CABG.5 The timely administration of platelet inhibitors and/or perioperative anticoagulation, as well as prevention of hypotensive episodes may be indicated in Off-pump CABG as preventive measures against delayed onset of stroke.

Moreover, the occurrence of clinically obvious stroke, the outcome measure of most studies, likely represents only the tip of the iceberg. Postoperative cognitive impairment that may not necessarily fall under the rubric of “stroke,” possibly represent multiple territory cerebral microinfarcts occurring as a result of embolic phenomena. These patients may be classified in other categories, such as delirium, depression, or dementia. Thus, the complication rate of clinically obvious stroke reported in the previous articles most likely represents an underestimate. Unfortunately, diagnosing these patients has traditionally been difficult in the past. Newer modalities such as diffusion-weighted MRI may show promise in that regard.

The increasing recognition of the role played by aortic atheroma and the innovative steps taken to minimize this risk, including epiaortic scanning and the potential for intra-aortic filtration, are all currently undergoing large-scale, prospective evaluations. Previous authors have demonstrated that use of epiaortic scanning and of a “Y” graft, which uses the radial artery joined to the pedicled left internal mammary artery in a Y graft fashion was associated with a significantly decreased incidence of cerebral embolization secondary to aortic instrumentation. Aortic manipulation during CABG is a contributing mechanism for postoperative stroke. The incidence of postoperative stroke increases with increased levels of aortic manipulation. We previously demonstrated that patients who had a full and a tangential aortic clamp applied were 1.8 times more likely to have a stroke versus those without any aortic manipulation (P<0.01) and 1.7 times more likely to develop a postoperative stroke than those with only a tangential aortic clamp applied.6 A modification of the surgical strategy, such as the “no-touch technique” described by Mills and Everson, might also be important in these patients.

In addition, pharmaceutical agents such as gangliosides, glutamate receptor antagonists, and antioxidants may potentially minimize neuronal damage and decrease the occurrence of stroke. It has also been suggested that prostacyclin infusion during cardiopulmonary bypass may lower the incidence of encephalopathy and stroke during coronary artery bypass by preventing adhesion of platelets to the extracorporeal tubing and subsequent microembolization. Further studies are

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needed to prospectively investigate the potential benefits of pharmaceutical agents in reducing the incidence of stroke after coronary artery bypass. Coronary artery bypass without cardiopulmonary bypass needs further investigation as an approach for decreasing the incidence of stroke and should potentially be considered in patients with carotid artery disease, or other high-risk characteristics for stroke. However, the benefits of this technique have not been evaluated in a prospective randomized setting. All these interventions may eventually prevent or decrease the effects of these catastrophic complications after cardiac surgery. The elusive “holy grail” may at last be within grasp.

References

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