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Commentary

A New Role for Radiologists in the Development of Cardiac Surgery

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Question: What iatrogenic situation will permanently injure the brain of more than 150,000 Americans this year *and* has never been the subject of an article in the radiologic literature?

Answer: Neurologic or neuropsychological complications of cardiac surgery. Hise et al. [1] deserve credit for bringing this matter to our attention.

The introduction of cardiopulmonary bypass (CPB) pump perfusion, the result of two decades of design and experimentation by Dr. John H. Gibbon at Massachusetts General Hospital and later Jefferson Medical College, has made cardiac surgery feasible. Its use has become widespread: 400,000 operations are performed yearly in the United States using this technology [2]. Putting this figure into perspective, the rate is nearly 30 times that of intracranial aneurysm surgery. One measure of a successful operation is that all of the noncardiac organs function at least as well after surgery as before. There is mounting evidence that one, the brain, does not. In a retrospective review of complications among cardiac surgery patients [3], the frequency of altered mental state was reported to be 3.4%, with a 1% reported rate of stroke. Hise et al. [1] have described the CT findings in a similar group of patients; that is, the 1% who will be found to have "stroke" retrospectively.

More recently, prospective testing has revealed a 24–34% rate of occurrence of permanent neurologic or neuropsychological deterioration after cardiac surgery [4–7]. One study [8] showed increased levels of a CSF enzymatic marker of brain injury (adenylate kinase) after heart surgery. (The disturbing issue of brain dysfunction in a smaller but significant number of older patients undergoing major surgery apart from the heart [6] must be investigated further.)

Early CPB devices were reused, so that in spite of meticulous cleaning, many problems were caused by inadvertent injection of old blood products. There were and continue to be problems related to trauma to blood elements and foaming in the oxygenator. Filters have been developed for the system, but because they may cause trauma to blood elements if the filter size is below 40 μm, their value has been questioned [5]. Oily silicone antifoam agents were introduced into the system to remove air bubbles, but these agents were shown to embolize to the brain [9–11] and they are no longer used [12, 13]. Fat emboli have been documented after CPB [14, 15]. Air bubbles are known to embolize to the brain, but with the use of membrane oxygenators they are no longer considered to be a problem [13].

Focal neurologic deficits can occur in association with a large air embolism as a result of restarting cardiac pulsation without completely evacuating air in the cardiac chambers. Focal neurologic deficits can also occur in association with local hypoperfusion and particulate *macro*emboli (atherosclerotic plaque disruption at the site of aortic cannulation, calcium or vegetation fragments from valve manipulation, other exogenous debris from field-aspirated fat globules or blood clots). *Diffuse* neurologic and neuropsychological deficits are presumably due to *global* hypoperfusion or myriad *micro*emboli. Microembolic agents could be platelet aggregates, chylomicrons, tiny air bubbles, glove powder, tube fragments, silicone, antifoam materials, or phospholipid remnants of dam-

This article is a commentary on the preceding article by Hise et al.

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aged red blood cell membranes. Another source of emboli could be the fat globules that accumulate on the surface of field-aspirated blood.

In spite of recent improvements in CPB, there is evidence that emboli still occur. Blauth et al. [16] reported abrupt termination of blood flow in retinal vessels as seen by fundus fluorescein angiography during CPB, presumably resulting from small emboli. Using transcranial Doppler sonography, Padayachee et al. [17] noticed disturbances believed to be arterial microemboli with both membrane and bubble oxygenators during CPB. Moody et al. [18] demonstrated multitudinous small emboli in brain and other tissues in autopsy material from humans and research dogs following CPB. In that study histochemical staining of thick sections permitted visualization of long segments of the microvasculature on a single slide. In another study [19], investigators established that cerebral blood flow dramatically diminishes during CPB and does not return to normal levels at the end of the procedure. Some global phenomena—such as autoregulation, decreased metabolic requirement, or reaction to cooling—are thought to be the cause. Such a response is also compatible with multiple semipermanent microembolic events. In all four studies [16–19] the origin or composition of the microemboli was not determined conclusively. There may be several different varieties of these offending agents.

Recently, two groups of investigators have reported prospective MR imaging in cardiac surgery patients. First, Schmidt et al. [20] described findings in 10 patients before and after coronary artery bypass grafting (CABG). No change was found in the MR studies, but the three patients with postoperative neurologic complications had infarcts or basal ganglia lacunae prior to surgery. Therefore, it is possible that a subgroup exists, identifiable by MR imaging, that can be expected to have a poor neurologic outcome with heart surgery. Second, DeLaPaz et al. (paper presented at the annual meeting of the ASNR, Washington, DC, June 1991) reported findings in 16 patients who underwent cardiac valvular surgery. One half of the patients had new findings on MR studies after the operation, and two of these had appropriate neurologic deficits. Valvular surgery is believed to be more dangerous than CABG. My own judgment is that the conclusions of imaging studies purporting to establish the cause (hypoperfusion vs embolization, or embolism of "hard" products such as calcium vs "soft" products such as air or blood clots) of brain injury in this situation are questionable at best.

During cardiac surgery pulsatile perfusion is replaced most often by constant-pressure perfusion. It is possible that the pulsatile nature of flow has an as yet unknown salutary effect. Certain deep vascular beds of the brains of elderly patients may be irrigated only during the successive peaks of pulsatile perfusion [21].

Cardiac surgery has become safer, and further improvements can be anticipated. In the future, improved design of materials in the CPB apparatus may incite less inflammatory response and may be less traumatic to blood cells. Anesthesia protocols are also under investigation [2]. *N*-methyl-D-aspartate antagonists, calcium channel blockers, perfluorocarbons, blood glucose management, and other manipulations that might protect brain tissue during CPB are under consideration for clinical trials.

Sensitive new instruments for imaging alterations in brain composition (MR) and metabolism (MR spectroscopy, PET, SPECT) will be instrumental in the next phase of the development of cardiac surgery. I believe there will be a role for radiologists in the development of modern cardiac surgery in the area of patient selection and outcome studies.

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