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Stroke Associated with Coronary Artery Bypass Surgery

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Medical records and neuroimaging studies of 30 patients with major neurologic events after coronary artery surgery were reviewed. Two thousand and twenty-nine coronary artery bypass graft operations were performed in our institution between October 15, 1985, and December 27, 1989. Of these, there were 30 documented neurologic events suggesting acute ischemic injury during the intraoperative or the postoperative period. Clinical manifestations included hemiparesis, monoparesis, aphasia, bilateral cortical dysfunction, cortical and brainstem dysfunction, and left homonymous hemianopsia. There were five deaths directly attributable to neurologic injury. Twenty-two patients had a CT scan of the head, of which 15 showed evidence of acute infarction, two suggested watershed lesions from cerebral hypoperfusion, and the remainder showed findings consistent with multiple cerebral emboli or primary intracranial occlusion. Five carotid arteriograms and one digital subtraction arteriogram of the carotids were obtained. Angiographic findings revealed two common carotid artery occlusions, one callosal marginal artery occlusion, and two cases of bilateral high-grade internal carotid stenoses.

Our findings support the contention that in patients who suffer cerebral infarction associated with coronary artery bypass grafting, the main mechanism of injury is cerebral embolization rather than cerebral hypoperfusion.

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In the vascular and neurologic literature, there has long been interest concerning neurologic events after cardiac surgery. Shaw et al. [1] performed a prospective study of neuropsychological symptoms in patients after coronary artery surgery that showed neuropsychological symptoms and/or moderate or severe intellectual dysfunction in a significant number of patients (31% and 24%, respectively). Sotaniemi et al. [2] also evaluated neuropsychological, neurologic, cardiologic, and electroencephalographic findings in patients who had cardiac valve replacement and showed significant long-term neuropsychological consequences in a substantial percentage of these patients. Most recently, Kittner et al. [3] studied historical features relevant to the diagnosis of cardiac embolic strokes and pointed out the need for study of the radiologic features of cerebral vascular events with a possible cardiac source. Major neurologic events are a well-known complication in a small percentage of patients who have undergone coronary artery bypass graft procedures. The purpose of this study was to evaluate the radiologic findings in the CNS in this patient population and attempt to characterize the possible causes of major neurologic events in these patients.

Materials and Methods

The charts, postoperative CT scans, and postoperative cerebral angiograms were reviewed in all patients with clinically documented neurologic events occurring in the postoperative period following coronary artery bypass grafting. Nineteen of the 30 documented neurologic

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events occurred within 24 hr of the surgical procedure. Seven occurred within 24–48 hr of surgery. The remaining four occurred between 48 hr and 11 days following surgery. The time interval between clinical symptoms or signs and the CT scan of the head in 20 of the 22 patients who had CT scans ranged from 2 hr to 96 hr, with a mean under 40 hr. The time interval between clinical signs and symptoms and CT scan in one of the 22 patients is not known, and in one patient was under 20 days.

Results

Of the 30 patients with neurologic deficits, 22 had head CT scans. The clinical presentations included hemiplegia or hemiparesis (19), monoparesis (one), aphasia (three), homonymous hemianopsia (one), "bioccipital stroke" (one), and diffuse cortical dysfunction or cortical and brainstem dysfunction (10). Two patients had head scans consistent with watershed infarction. In the first patient, the lesion was along the junction between the left anterior cerebral artery and the left middle cerebral artery distribution (Fig. 1). In the second patient, the lesion was located at the junction of the left middle cerebral artery and left posterior cerebral artery territories. An arteriogram obtained in this patient showed complete occlusion at the origin of the left internal carotid artery with ulceration. Of the remaining positive head scans, eight revealed infarctions within a single vascular territory while five had studies demonstrating infarction in more than one vascular territory (Fig. 2). Six of the patients in this group had angiographic studies, the results of which are summarized in Table 1. There were five deaths directly attributable to neurologic injury. Two of the patients had postmortem examinations. One of the two had a prior CT scan. The mortality rate associated with CNS infarction following bypass surgery was 16%.

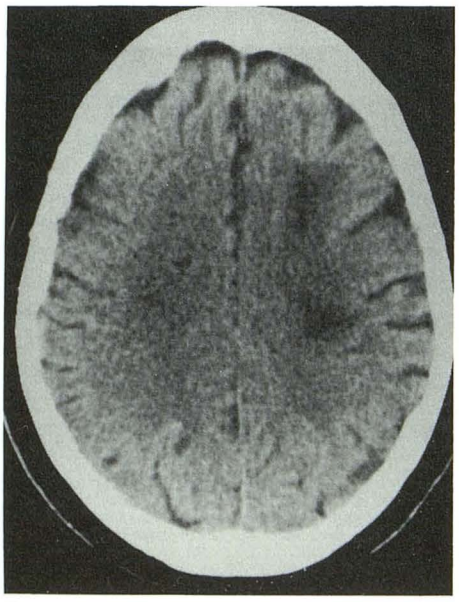
Discussion

Coronary artery bypass grafting is the most frequently performed cardiac surgery in the United States, with approximately 230,000 procedures performed in 1987 [4].

The frequency of stroke among patients undergoing coronary revascularization has been reported to be between 0.9% and 5.9%. Although the exact causes of stroke following coronary artery bypass graft are largely unknown, there has been extensive discussion of this complication in the surgical and neurologic literature [5–10]. Intraoperative hypotension, microemboli consequent to cardiopulmonary bypass apparatus, macroemboli from diseased heart valve, left ventricular thrombus, or an atheromatous aorta during cross clamping or cannulation have been implicated as causes of cerebral complications following bypass surgery [7]. Emboli forming at the proximal anastomosis of the vein graft have also been implicated as a cause of infarction following bypass. Inadvertent puncture of a carotid artery while attempting internal jugular vein cannulation, cardiac arrhythmias, and postoperative hypotension are other possible causes [6].

There is little information on this subject in the radiology literature and we found only a single reference in which CT scanning was used to search for the cause of bilateral visual field defects in a patient who had undergone a coronary artery bypass graft and excision of a myocardial aneurysm [11].

Of our patients with positive head scans, 13 showed evidence of injury suggesting an embolic source or, less likely, a primary intracranial occlusion. Five of the head scans were quite striking, with multiple acute infarctions in different vascular territories. Primary intracranial occlusion as a cause of infarction was considered less likely than embolism. All patients with single vascular territory occlusions presented in the immediate postoperative period and the occlusion pre-



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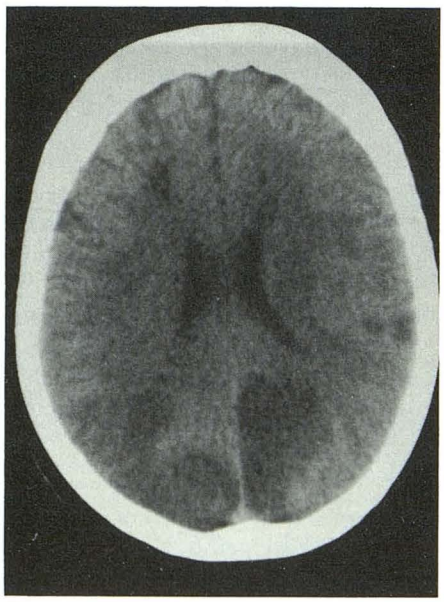

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Fig. 1.—CT scan of watershed infarction at boundary of left middle cerebral artery and left anterior cerebral artery.

Fig. 2.—CT scan of multiple infarctions in several internal carotid artery branch distributions, including right posterior cerebral artery, left posterior cerebral artery, right middle cerebral artery, and left middle cerebral artery, consistent with an embolic origin.

TABLE 1: Findings in 30 Patients with Cerebral Events After Coronary Artery Surgery

Case No.	Age (years)	Sex	Time to Clinical Signs or Symptoms	Time from Signs or Symptoms to CT Scan	Clinical Presentation	Cerebral CT Scan Findings	Cerebral Angiography or Postmortem Findings
1	53	M	<24 hr	<48 hr	Rt hemiplegia	Lt middle cerebral artery infarction	
2	66	F	<24 hr	<48 hr	Rt hemiparesis	Lt middle cerebral artery infarction, L lacunar infarction	Cerebral angiogram with 75% stenosis of the L internal carotid artery and 90% stenosis of the R internal carotid artery 10 days before coronary artery surgery
3	69	M	52.5 hr[a]	<96 hr	Lt homonomous hemianopsia	Rt posterior cerebral artery infarction	
4	72	M	22 hr	–	"Bioccipital stroke"		
5	70	M	24–48 hr	Unknown	Coma; death	Ventriculomegaly and Lt cerebellar infarction	Lt cerebellar hemorrhagic infarction and tonsillar herniation at autopsy
6	61	M	<12 hr	<12 hr	Coma; death	Infarctions in the Lt posterior cerebral artery, Lt middle cerebral artery, and Rt middle cerebral artery distributions	
7	61	F	<12 hr	60 hr	Coma	Infarctions in the Lt posterior cerebral artery, Lt middle cerebral artery, Rt posterior cerebral artery, Rt middle cerebral artery distributions	
8	70	M	Immediate postoperative period	–	Coma; death		
9	79	M	Immediate postoperative period	75 hr	Rt hemiplegia	Normal	
10	76	M	48 hr	26 hr	Rt hemiplegia; confusion	Atrophy	
11	69	M	24–48 hr	15 hr	Aphasia; Rt hemiparesis	Lt middle cerebral artery–Lt posterior cerebral watershed infarction	Cerebral angiography with Lt common carotid occlusion and ulceration
12	70	F	<24 hr	–	Death		Rt parietal and pontine infarctions at autopsy
13	66	M	11 days[b]	2 hr	Rt hemiparesis	Normal	Cerebral angiogram normal 2 days after coronary artery surgery
14	81	M	<48 hr	–	Lt hemiplegia		
15	65	M	<48 hr	<48 hr	Lt hemiparesis	New Rt posterior cerebral artery and Rt middle cerebral artery infarctions, old Lt middle cerebral artery and L posterior cerebral artery infarctions	Angiogram several years prior demonstrated minor bilateral arteriosclerotic disease
16	60	F	<24 hr	36 hr	Lt arm and Rt leg weakness; coma	Bilateral middle cerebral artery and Lt cerebellar infarctions	
17	89	M	24 hr	12 hr	Coma	Large Rt posterior cerebral artery infarction	
18	51	F	<24 hr	7 hr	Rt hemiplegia	Large Lt middle cerebral artery infarction	
19	66	F	<24 hr	<20 days	Rt hemiplegia	Lt anterior cerebral artery–Lt middle cerebral artery watershed infarction	
20	65	M	<24 hr	12 hr	Rt hand weakness	Subdural fluid collections	
21	69	M	<48 hr	<36 hr	Rt hemiparesis, coma; death	Small Lt anterior cerebral artery infarction	Postoperative angiogram demonstrated Lt callosal marginal artery occlusion
22	68	M	48–96 hr	6 hr	Aphasia	Normal	Digital subtraction angiogram demonstrated a Rt common carotid artery occlusion

TABLE 1: Continued.

Case No.	Age (years)	Sex	Time to Clinical Signs or Symptoms	Time from Signs or Symptoms to CT Scan	Clinical Presentation	Cerebral CT Scan Findings	Cerebral Angiography or Postmortem Findings
23	73	M	Immediate post-operative period	–	Lt hemiparesis		
24	69	M	<24 hr	<88 hr	Rt hemiplegia	Lt middle cerebral artery infarction	
25	70	M	<24 hr	56 hr	Lt hemiparesis	Atrophy	
26	76	M	5 days	<24 hr	Aphasia/Rt hemiparesis	Old Lt lacunar infarction	Postoperative angiogram demonstrated 50% stenosis in the Lt internal carotid artery, >50% stenosis in the Rt internal carotid artery, high-grade stenosis in the supraclinoid portion of the Lt internal carotid artery
27	72	F	Immediate post-operative period	–	Lt hemiparesis		
28	72	F	Immediate post-operative period	–	Lt hemiparesis		
29	68	M	<48 hr	–	Lt hemiparesis		
30	71	M	<24 hr	<84 hr	Coma	Lt posterior cerebral artery, Lt middle cerebral artery, and Rt middle cerebral artery infarctions	

Note.—Rt = right, Lt = left.
[a] This was the first time the patient used his glasses.
[b] This neurologic event may be unrelated to the coronary artery surgery.

sumably would have occurred during a time when the patient was anticoagulated. One of the two patients with a watershed pattern infarction on the CT scan had a carotid angiogram showing complete carotid occlusion. The patient with the watershed pattern infarction at the junction of the left anterior and middle cerebral artery territories had a CT scan showing a subcortical infarction in this distribution. Given the unilaterality of the finding and the positive correlation with the acute neurologic deficit, it is unlikely that this lesion could represent preexisting microangiopathic leukoencephalopathy. There were five patients with either a previous CT or MR examination of the brain available for review. This group's postoperative scan findings included two patients with single distribution infarctions, two patients with multiple distribution infarctions, and one patient with a watershed distribution infarction. None of these lesions was present on previous CT or MR examinations. Of the three remaining patients with positive head scans and postoperative cerebral arteriograms, one showed direct evidence of emboli with an abrupt cutoff of the left callosal marginal artery and clinical findings of diffuse cortical dysfunction suggesting multiple other emboli below the limits of angiographic resolution. Another patient had carotid angiograms in the postoperative period that showed stenoses at both common carotid artery bifurcations with 75% stenosis on the left and 90% stenosis on the right. The infarction in this patient occurred in the distribution of the left carotid artery. A single patient who did not have a CT scan of the head underwent an autopsy. The autopsy findings showed pontine and right parietal infarctions consistent with an embolic origin.

Although hypotension and watershed infarction play a significant role in neurologic injury during and following coronary artery bypass surgery, our findings suggest that the majority of patients with severe neurologic dysfunction have suffered embolic infarctions.

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The reader's attention is directed to the commentaries on this article, which appear on the following pages.