Central Nervous System Complications of Open Heart Surgery

ANTHONY J. FURLAN, M.D., AND ANTHONY C. BREUER, M.D., F.A.C.P.

Stroke and encephalopathy remain major causes of morbidity and occasional mortality complicating open heart surgery. Open heart surgery poses many hemodynamic, embolic, and metabolic threats to the brain, yet the cause of neurological damage often remains uncertain in the individual patient. Failure to recognize the multitude of potential causes for severe or subtle brain dysfunction after open heart surgery can lead to overemphasis of a few highly visible factors such as extracranial vascular disease.

Cardiac Catheterization

Central nervous system complications are rare during heart catheterization. In the course of over 30,000 catheterizations done at our hospital during a five-year period, 35 patients sustained a focal deficit (carotid 15, vertebral-basilar 20), and two patients experienced diffuse encephalopathy. In 19 patients the deficit resolved, usually within 48 hours. In 18 patients the deficit persisted, and two patients died as a result of cerebral injury sustained during catheterization. The etiology was embolism or systemic hypotension in six patients; in two patients a vertebral artery was inadvertently injected with contrast. No clear etiology was identified in the remaining patients, although many had a past history of stroke, documented cerebrovascular disease, or potential cardiac embolic risk factors, such as ventricular hypokinesia, mural thrombus, or valvular disease.

Brain Infarction

In the setting of open heart surgery, "stroke" generally refers to focal brain infarction in a specific vascular territory. This risk has decreased since the 1960's, partly as a result of improved monitoring and surgical techniques, along with the introduction of membrane oxygenators and in-line filtration which decrease the release of microaggregates into the circulation. Since the 1970's there has been little change in the stroke rate, which ranges between 2 and 5% in various studies.

In a prospective study, we found a 5% rate of focal brain or ocular infarction among 421 patients undergoing coronary artery bypass graft surgery. Only 2% suffered major neurological disability. In our institu-

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TABLE 1  Central Nervous System Complications in Patients Undergoing Various Types of Open Heart Surgery

<table>
<thead>
<tr>
<th>Valve Type</th>
<th>Stroke</th>
<th>Encephalopathy</th>
<th>Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left heart valves (n = 421)</td>
<td>5</td>
<td>12</td>
<td>2</td>
</tr>
<tr>
<td>Ventricular aneurysm (n = 19)</td>
<td>8</td>
<td>10</td>
<td>9</td>
</tr>
<tr>
<td>Aortic aneurysm (n = 99)</td>
<td>0</td>
<td>5</td>
<td>0</td>
</tr>
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</table>

*CABG = coronary artery bypass graft.

Diffuse Encephalopathy

In our prospective series 11.6% of patients had clinically detectable prolonged encephalopathy after open heart surgery. More subtle changes in personality noticeable to the family and detectable only through psychological testing may be a more common problem than previously recognized. Postsurgical encephalopathy is often multifactorial and related to post-anesthetic effect, drugs, intensive care psychosis, metabolic derangements, hemodynamic instability, and a host of other factors acting alone or in combination. A decrease in blood pressure was the only factor we could statistically correlate with the risk of developing encephalopathy. This finding suggests that a decrease in cerebral blood flow above the frank infarction threshold can result in altered mentation in some patients.

Carotid Stenosis and Stroke

Since major stroke occurs in less than 2% of all patients undergoing open heart surgery, from a statistical standpoint it would be extremely difficult to demonstrate that one variable significantly alters that risk. Nonetheless, extracranial carotid artery disease is often suggested as an important cause of stroke during open heart surgery. This theory proposes that infarction may occur ipsilateral to a severely stenosed internal carotid artery (ICA) if there is a drop in blood pressure during surgery. The logic is deceptively persuasive, but the evidence suggests that extracranial ICA stenosis is a relatively insignificant factor in causing stroke.

The frequency of hemodynamically significant ICA stenosis in this population is uncertain. Asymptomatic neck bruits are poor indicators of the severity of underlying ICA stenosis, and they are useless for predicting stroke risk during elective surgery. There have been a few studies using noninvasive tests to detect carotid stenosis in patients undergoing open heart surgery, none with angiographic confirmation. The data are conflicting, the results partly depending on which noninvasive test was employed. Tests such as oculoplethysmography and Doppler may give information regarding the proximal hemodynamic significance of an ICA stenosis, but they do not provide a measure of cerebral blood flow or autoregulatory capacity. With Doppler techniques hemodynamically significant asymptomatic ICA stenosis has been found in 6 to 12% of surgery patients. Using oculoplethysmography, Kartchner and McRae found a rate of 31% in a group undergoing nonspecified cardiovascular procedures. The reasons for this marked discrepancy are unclear, although it appears that oculoplethysmography was considered “positive” with lesser degrees of stenosis. A “positive” Doppler study does not correlate with stroke risk during open heart surgery. Kartchner and McRae found a much higher stroke risk in patients with “positive” oculoplethysmography, but they do not comment on the vascular distribution of the strokes nor on other potential causes of stroke in their series.

Despite the lack of convincing data, it is common practice in some hospitals to perform a “prophylactic” carotid endarterectomy in asymptomatic patients discovered to have a hemodynamically significant ICA stenosis prior to open heart surgery. Whether there is a group of patients who might benefit from such surgery

TABLE 2  Myocardial Revascularization. Possible Etiology of Focal CNS Deficits

<table>
<thead>
<tr>
<th>Cause</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac arrhythmia intra/peroperatively</td>
<td>6</td>
</tr>
<tr>
<td>Internal carotid artery atherosclerosis</td>
<td>5</td>
</tr>
<tr>
<td>Air embolism from left ventricle</td>
<td>2</td>
</tr>
<tr>
<td>Carotid artery trauma during internal jugular vein cannulation</td>
<td>1</td>
</tr>
<tr>
<td>Aortic atherosclerosis at site of clamping</td>
<td>1</td>
</tr>
<tr>
<td>Prolonged intraoperative blood pressure decrease</td>
<td>1</td>
</tr>
</tbody>
</table>

*Cause identified in 16 of 22 patients (73%).

FIGURE 1. Cholesterol embolus within a brain arteriole in a patient who never awakened after open heart surgery. There were numerous brain and systemic emboli; a very brittle aorta had been cross-clamped during surgery.
FIGURE 2. CT showing bihemispheric watershed infarctions in a patient who failed to awaken after open heart surgery. There was significant intraoperative hypotension. The extracranial vessels were patent at postmortem.

requires further study. Presently there is no evidence that staged or combined endarterectomy improves stroke risk during open heart surgery.11 12

Predicting, Detecting, and Preventing Brain Damage

Our experience and that of others indicates that there are few preoperative, intraoperative, or postoperative factors which can reliably predict the risk of stroke or encephalopathy during open heart surgery. Obviously, marked drops in blood pressure should be avoided, but many patients sustain relatively prolonged periods of hypotension without neurological deficit. Potential embolic situations such as a brittle aorta, ventricular thrombus, air in the heart or bypass lines, and dysrhythmias should be watched for and dealt with appropriately.

Since hypotension is a definite possibility during any open heart procedure, it would be of interest to develop a reliable regional cerebral blood flow (rCBF) "stress test" for assessing autoregulatory capacity preoperatively. There are several invasive and noninvasive techniques for measuring rCBF, including xenon inhalation and injection, positron emission tomography, and single photon emission computed tomography. Autoregulation can be tested by using a tilt table or inhaling 5% CO₂. None of these techniques has been widely applied before or during cardiopulmonary bypass. The available studies indicate that cerebral blood flow drops during cardiopulmonary bypass.13 The analysis of cerebral blood flow during surgery is complicated by the effects of general anesthesia and hypothermia, which might raise the ischemic threshold, and by other factors such as nonpulsatile flow14 and the release of vasoactive substances which have an uncertain effect on brain blood flow.

Ordinarily, cerebral blood flow is constant between a mean arterial pressure of 60 and 160 mm Hg. Autoregulation is achieved mainly through changes in the diameter of the arteriolar resistance vessels. The extracranial vasculature serves primarily as a pressure head reservoir. While high grade internal carotid artery stenosis may contribute to impaired cerebral blood flow, the status of the arteriolar resistance vessels is more critical in determining the effects of systemic hypotension on the brain. In patients with arteriolar disease, for example, elderly hypertensive patients, the autoregulation curve shifts to the right so that cerebral blood flow may begin to fall even with a mean arterial pressure exceeding 100 mm Hg. Although most patients on cardiopulmonary bypass do well with a mean pressure greater than 50 mm Hg, it would seem prudent to maintain a mean pressure of 60 mm Hg or greater in patients with possible defective cerebral blood flow autoregulation. Some cases of prolonged encephalopathy or unexplained coma may be due to relative hypotension in patients with defective autoregulation.15

Other causes of brain dysfunction after open heart surgery must be considered. Both Halothane and Ethrane may produce alterations in neuronal microtubules16 and thereby alter axonal transport. Ethrane may produce occult seizures17 unwitnessed during general anesthesia, resulting in a prolonged postictal encephalopathy in some patients. Complement activation during bypass,18 complement-induced granulocyte aggregation,19 or altered thromboxane synthesis with effects on platelet function during extracorporeal circulation20 may also play a role in producing some central nervous system dysfunction. However, since all patients undergo anesthesia and all have their blood exposed to the surfaces of the disposable plastic extracorporeal tubing, yet only a few develop CNS dysfunction, other factors must be involved.

The electroencephalogram (EEG)21 may be a useful means of detecting the time of onset of focal or global brain ischemia intraoperatively and might provide clues to injury mechanisms. However, it is impractical to monitor all surgical patients with EEG. Other techniques for detecting brain damage, such as analysis of BB-CPK isoenzyme in the cerebrospinal fluid, are being investigated.22 The development of a preoperative rCBF stress test might allow identification of a subgroup of high risk patients who could then be selectively monitored with EEG. The rCBF analysis
might also allow a more rational approach to prophylactic endarterectomy in the patient with asymptomatic carotid disease.

The setting of open heart surgery provides an ideal opportunity for testing new treatment protocols for modifying focal or global brain ischemia. Various hemodynamic and pharmacological treatment strategies could be quickly instituted either at the time of brain ischemia or, preferably, before onset of ischemia in high risk situations. Thus, a better understanding of brain ischemia during heart surgery could serve as an important model for the modification of ischemic stroke in general.

Conclusions

1. Major brain infarction occurs in less than 2% of infants undergoing open heart surgery.

2. The cause of brain infarction during surgery is often embolic and may be related to dysrhythmias, ventricular mural thrombus, a brittle aorta, air in the system, microaggregate formation, or cerebrovascular disease.

3. There is no convincing evidence that asymptomatic extracranial internal carotid artery stenosis increases stroke risk or that prophylactic carotid endarterectomy in asymptomatic patients lowers stroke risk.

4. Clinically detectable encephalopathy occurs in at least 12% of patients undergoing open heart surgery and is usually multifactorial. More subtle encephalopathy detectable by psychometric testing occurs more frequently.

5. Lowering mean blood pressure to accepted levels for routine cardiopulmonary bypass may be a neglected cause of brain ischemia in patients with arteriolar disease and defective cerebral blood flow autoregulation.

6. Techniques for measuring cerebral blood flow and brain function should be used to identify patients at high risk for developing brain ischemia during surgery and for detecting the onset of brain dysfunction intraoperatively.

7. Stroke in the setting of open heart surgery provides a unique opportunity to study the pathogenesis and modification of both focal and global brain ischemia.

References


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