Mechanisms of Perioperative Cerebral Infarction

ROBERT HART, M.D., AND BRAD HINDMAN, M.D.

SUMMARY  Perioperative cerebral infarction occurs in less than 1% of general surgical procedures; the mechanism is usually unknown. The clinical features of 12 consecutive perioperative strokes were retrospectively reviewed. Although intraoperative hypotension was frequent, onset of deficit occurred postoperatively in 83% and intraoperatively in 17%. Cardiogenic embolism was a common cause of stroke (42%), with atrial fibrillation present in 4 patients (33%) at the time of stroke. The potential roles of hypercoagulability, hypotension and carotid occlusive disease are discussed. Future reports concerning perioperative stroke should consider the multiple mechanisms and temporal relationship of stroke to the operative procedure.

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PERIOPERATIVE STROKE IS UNCOMMON, occurring in 0.38% of male general surgical patients over age 50 in one large survey.1 Even in elderly patients, who have a higher incidence of all types of perioperative complications, perioperative cerebral infarction occurs in only 1.0–2.5% of major general surgical procedures, the latter figure applying to octogenarians.2 3 Patients who undergo aortoiliac surgery, considered to be at special risk for perioperative stroke due to the relatively high prevalence of coexistent carotid atherosclerosis and intraoperative hypotension, have a perioperative stroke rate of only 1%.4–10

Despite the infrequency of perioperative stroke, prophylactic carotid endarterectomy has been recommended for preoperative patients with asymptomatic stenosis based on the assumption that perioperative stroke is related to untreated carotid occlusive disease.4,11–13 Presumably, intraoperative hypotension or hypoxemia, inconsequential in the absence of carotid stenosis, when combined with occlusive carotid atheroma, results in cerebral hyperperfusion and stroke.12,14 Such a recommendation requires a critical examination of the mechanism(s) of perioperative cerebral infarction.

It has been our impression that many perioperative cerebral infarctions occur in the postoperative period, not intraoperatively, and that the mechanism is often uncertain. Review of the literature of the temporal occurrence of perioperative stroke in 10 patients undergoing aortoiliac reconstruction showed that all 10 events had onset in the postoperative period.7,2,15 The mechanism(s) of perioperative stroke are thus not limited to intraoperative hypotension potentiated by carotid stenosis.

It is likely that mechanisms of perioperative cerebral...
infarction depend upon the type of surgical procedure. Factors contributing to cerebral ischemia after carotid endarterectomy or mitral valve replacement are likely to be different than factors etiologic in stroke following inguinal hernia repair. Based on differences in perioperative stroke prevalence and special surgical circumstances we propose to divide perioperative strokes into four general groups:

1. Related to procedures directly involving or adjacent to the carotid or cerebral arteries.
2. Related to cardiac surgery involving cardiopulmonary bypass.
3. Related to discontinuation of anticoagulants prior to elective surgery.
4. Related to general surgical (including urological, gynecological, orthopedic, etc.) and peripheral vascular procedures.

The first three categories have been extensively considered in the recent literature. We propose to divide perioperative strokes into four general groups:

Case histories of patients with perioperative cerebral infarction are sparse in the recent literature. We report 12 patients with perioperative cerebral infarction relating the occurrence of stroke to intraoperative and postoperative events in an attempt to identify the responsible mechanisms. Implications for prevention based on these mechanisms are discussed.

Methods

The hospital records of 48 consecutive patients with discharge diagnoses of nervous system complications of surgery (HICDS 998.9, ICD-9 997.0) at one hospital for the years 1975-1982 and a second hospital for 1979-1980 were reviewed. Patients with diffuse, nonfocal encephalopathies were excluded; only patients with focal neurologic deficits were further considered. The majority of perioperative strokes were related to carotid or cardiac surgery or cerebral arteriography; these are not further considered. Of the remaining patients, there were 12 who underwent general surgical procedures or peripheral vascular reconstructions (including abdominal aortic surgery) and who sustained cerebral infarction prior to hospital discharge. The records of these 12 patients were reviewed in detail, noting clinical, laboratory and radiographic features before and at the time of stroke. There were approximately 24,500 general surgical procedures, excluding carotid and cardiac surgery, performed during this interval (average age about 55), with about 30% of patients over age 65.

There were four additional patients with perioperative stroke related to discontinuation of chronic anticoagulants and another young woman who sustained a stroke due to carotid occlusion during elective tonsillectomy; these patients are not considered further in this report.

Results

Table 1 summarizes the clinical features of these 12 patients. There were 7 males and 5 females; mean age was 69 years. There were 4 patients (33%) undergoing peripheral vascular procedures. Two patients (17%) underwent procedures as primary treatment of malignancy (squamous cell carcinoma of vulva, adenocarcinoma of colon). Atrial fibrillation was present in 4 patients (33%) at onset of stroke; another patient had a history of atrial fibrillation. Onset of deficit occurred in the postoperative period in 10 patients (83%). Some degree of intraoperative hypotension occurred in almost half of patients, without apparent sequelae (table 1). Cases 1 and 9 had prior cerebrovascular symptoms. Case histories follow, with four described in detail.

Case 1

The patient was a 65-year-old male with severe peripheral vascular disease. On the day of admission he awakened with right limb weakness which resolved over 1–2 hours. Later that day, examination was remarkable for blood pressure (BP) of 208/88 mm Hg, bilateral cervical bruits and a right Babinski sign. Ad-

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**TABLE 1 Clinical Features of Perioperative Stroke**

<table>
<thead>
<tr>
<th>Case #</th>
<th>Age</th>
<th>Sex</th>
<th>Procedure</th>
<th>Bruits</th>
<th>Intra-op BP*Δ</th>
<th>Time of onset</th>
<th>Associated events</th>
<th>Possible mechanisms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>65</td>
<td>M</td>
<td>PVR</td>
<td>bilat</td>
<td>180/80-120/—</td>
<td>3 days post-op</td>
<td>relative ↓BP</td>
<td>hypotension</td>
</tr>
<tr>
<td>2</td>
<td>79</td>
<td>F</td>
<td>hip surgery</td>
<td>none</td>
<td>no Δ</td>
<td>6 days post-op</td>
<td>—</td>
<td>thrombotic</td>
</tr>
<tr>
<td>3</td>
<td>69</td>
<td>F</td>
<td>retinal repair</td>
<td>none</td>
<td>130/90-60/—</td>
<td>? intra-op</td>
<td>A-fib</td>
<td>hypotension</td>
</tr>
<tr>
<td>4</td>
<td>57</td>
<td>M</td>
<td>appendectomy</td>
<td>none</td>
<td>140/80-60/—</td>
<td>5 days post-op</td>
<td>atelectasis</td>
<td>embolic</td>
</tr>
<tr>
<td>5</td>
<td>78</td>
<td>F</td>
<td>vulvectomy</td>
<td>none</td>
<td>140/74-80/—</td>
<td>5 days post-op</td>
<td>—</td>
<td>?</td>
</tr>
<tr>
<td>6</td>
<td>68</td>
<td>M</td>
<td>laparotomy</td>
<td>none</td>
<td>140/90-110/70</td>
<td>7 days post-op</td>
<td>A-fib</td>
<td>embolic</td>
</tr>
<tr>
<td>7</td>
<td>90</td>
<td>M</td>
<td>PVR</td>
<td>none</td>
<td>130/70-90/50</td>
<td>18 days post-op</td>
<td>M.I.</td>
<td>embolic</td>
</tr>
<tr>
<td>8</td>
<td>80</td>
<td>M</td>
<td>lens surgery</td>
<td>?</td>
<td>no Δ</td>
<td>2 days post-op</td>
<td>A-fib</td>
<td>embolic</td>
</tr>
<tr>
<td>9</td>
<td>69</td>
<td>F</td>
<td>PVR</td>
<td>none</td>
<td>150/90-80/—</td>
<td>13 days post-op</td>
<td>M.I., ↓BP</td>
<td>?</td>
</tr>
<tr>
<td>10</td>
<td>80</td>
<td>M</td>
<td>cecumectomy</td>
<td>none</td>
<td>170/90-140/—</td>
<td>36 hrs post-op</td>
<td>—</td>
<td>?</td>
</tr>
<tr>
<td>11</td>
<td>62</td>
<td>F</td>
<td>laminectomy</td>
<td>?</td>
<td>115/70-90/60</td>
<td>intra-op</td>
<td>endocarditis</td>
<td>embolic</td>
</tr>
<tr>
<td>12</td>
<td>69</td>
<td>M</td>
<td>PVR</td>
<td>none</td>
<td>170/90-100/—</td>
<td>1 day post-op</td>
<td>A-fib</td>
<td>embolic</td>
</tr>
</tbody>
</table>

Abbreviations: PVR = peripheral vascular reconstruction (including abdominal aortic surgery); A-fib = atrial fibrillation; M.I. = myocardial infarction; p-op = postoperative; intra-op = intraoperative; BP = blood pressure.

*Intra-op BPΔ compares the average preoperative BP to the lowest recorded intra-op BP.
mission laboratory studies, EKG and computed tomo
graphy (CT) were normal. Cerebral arteriography showed
total occlusion of the left internal carotid artery at the com-
mon carotid bifurcation with the left middle and anterior
cerebral vessels supplied by ocular collaterals. There was
only minimal irregularity of the right common carotid
bifurcation and no distal lesions. Intervening hospital
course was unremarkable.

Twelve days after admission, elective aorto-femoral
bypass was carried out under general anesthesia with
nitrous oxide, enflurane, pancuronium and num-
ophan. The lowest intraoperative BP was 120 mm Hg
systolic. Aspirin was started on the first postoperative
day.

At 2:00 A.M. on the third postoperative day, BP
rose to 210 mm Hg systolic and treatment with 10 mg
of I.V. hydralazine was initiated. The lowest BP sub-
sequently recorded was 150/76 mm Hg.

At 9:00 A.M. later that day, his wife found him
speechless. Exam showed moderate, nonfluent dys-
phasia and a mild right hemiparesis. His BP was 160/
80 mm Hg. For the next three hours his deficit varied
from mild to severe aphasia and hemiparesis despite a
stable blood pressure. At noon he was densely aphasic
with a right hemiplegia. Hematocrit was 30% (47% on
admission). EKG revealed lateral T-wave inversion
which subsequently resolved without other evidence
suggestive of myocardial infarction. A CT scan was
again normal. Beta-thromboglobulin was 50 ng/ml
(normal less than 48) and platelet factor 4 was 7 ng/ml
(normal 1–7) when obtained during the period of fluc-
tuation of his deficit.

He was treated with heparin, with subsequent im-
provement of his deficit to mild dysphasia and hemi-
paresis within 24 hours of onset.

Comment: Cerebral vascular dysautoregulation of
the left hemisphere following the ischemic event that
occurred the day of admission could have made the left
hemisphere especially vulnerable to relative hypoten-
sion (further, this area relied on collateral circulation
due to ipsilateral carotid occlusion). However, the pa-
tient tolerated lower BPs intraoperatively without se-
quelae. A postoperative fall in hematocrit could have
potentiated the effect of a relative decrease in BP. The
fluctuation of the deficit for the initial three hours
before stabilizing at a severe degree despite a stable BP
(carefully monitored during that interval) suggests a
more complex mechanism of stroke than straight-for-
adward hypoperfusion. Alternatively, emboli originating
from the recently occluded ipsilateral carotid could
have caused stroke.

Case 2
The patient was a 79-year-old woman admitted for
internal fixation of a left hip fracture. There was a
history of labile hypertension and paroxysmal, non-
valvular atrial fibrillation, but no prior cerebrovascular
symptoms. On examination, there were no carotid
bruits. Routine admission laboratory studies were nor-
mal; EKG showed an intra-atrial conduction defect and
sinus rhythm.

The intraoperative and immediate postoperative pe-
riods were entirely uneventful with halothane general
anesthesia. No antiplatelet agents or heparin were ad-
ministered postoperatively. On the third postoperative
day, BP increased to 230/100 mm Hg and hydrochlor-
othiazide and oral hydralazine were begun.

Because of persistent BP elevation, she was moni-
tored in the intensive care unit (ICU) with continuous
cardiac monitoring. On the sixth postoperative day she
developed dysartria. Vital signs were stable with no
episodes of hypotension or cardiac dysrhythmia. Over
the following 6–12 hours her deficit fluctuated and
finally stabilized with a marked right hemiparesis and
mild aphasia. A CT showed only mild, generalized
atrophy without infarction. Neurological deficits slow-
ly improved during hospitalization. Several 24-hour
cardiac monitors were carried out; all showed sinus
rhythm. No assessment of carotid patency was done.

Comment: Cardiac monitoring in the 72 hours prior to
stroke and admission EKG excluded atrial fibrillation
as an embologenic cause of her stroke, suspected be-
cause of her prior history of this dysrhythmia. Despite
treatment of postoperative hypertension, blood pres-
sure remained above admission levels in the hours
prior to cerebral infarction. The mechanism of this
stroke is uncertain, although the well-defined stutter-
ing evolution favors a thrombotic event.

Case 3
The patient was a 69-year-old female who was ad-
mitted for repair of a left retinal detachment. There
were no prior cerebrovascular symptoms; no carotid
bruits were present on admission. Routine preopera-
tive laboratory studies were normal. EKG revealed
previously unrecognized atrial fibrillation and digoxin
was begun. After preoperative morphine sulfate, gen-
eral anesthesia with halothane, sodium thiopental and
succinyl choline was complicated by a 10-minute epi-
sode of hypotension early in the procedure with systo-
lic BP falling from 130 to 60 mm Hg. Hypotension
responded to decreasing halothane, fluids and phenyl-
ephrine infusion. BP was subsequently stable for the
duration of the 1½ hour procedure. Phenylephrine was
tapered without difficulty in the recovery room. She
was monitored in the ICU for the initial 48 hours due to
rapid heart rate and mild S-T depression on EKG.
Serial cardiac enzymes and EKGs did not reveal evi-
dence of myocardial infarction. No heparin or anti-
platelet agents were administered postoperatively.

She remained mildly somnolent during the initial 24
hours following surgery. About 30 hours postopera-
tively, a left facial droop, mild left hemiparesis and
hemianopsia were appreciated. Vital signs had been
stable during the entire postoperative interval. Hema-
tologic, coagulation and blood gas studies were normal
at the time of stroke recognition. CT scan showed a
right parietal infarction. An echocardiogram revealed
mild left atrial enlargement and mitral valve prolapse
with no intracardiac thrombus. She was treated with
intravenous heparin with good functional recovery.

Comment: Due to postoperative lethargy that may
have delayed recognition of hemiparesis, it is uncertain if stroke occurred intraoperatively, related to hypotension, or postoperatively, related to embolism. No pre- or post-stroke assessment of carotid patency was done. It is not possible in this instance to distinguish with certainty between intraoperative hypotension, postoperative embolism due to atrial fibrillation, mitral valve prolapse or other factors in the genesis of stroke.

Case 4

The patient was a 57-year-old man with chronic obstructive pulmonary disease who underwent appendectomy. Two years previously, bilateral carotid arteriograms were performed for post-traumatic headache and were normal. There were no prior cerebrovascular symptoms and no cervical bruits. Admission laboratory studies and EKG were within normal limits. General anesthesia with halothane was complicated by a brief fall in systolic BP to 60 mm Hg. No heparin or antiplatelet agents were given postoperatively.

The immediate postoperative course was complicated by total atelectasis of the right lower lobe with an arterial oxygen of 59 mm Hg. On the fourth postoperative day, thick mucous plugs were removed at bronchoscopy with radiographic re-expansion of the atelectatic lobe.

On the fifth postoperative day he awoke with a right hemiparesis, dysphasia and loss of vision in the left eye. Ophthalmologic exam revealed Marcus-Gunn pupil and edematous retina. Hematocrit, platelet count and EKG were unchanged. Arterial oxygen partial pressure was 76 mm Hg.

Hemiparesis and visual loss only minimally improved over the following month.

Comment: The unusual occurrence of simultaneous ischemia to the eye and hemisphere suggests ipsilateral common carotid occlusion. In the presence of a normal arteriogram two years before and an absence of cerebrovascular risk factors, embolic occlusion is suggested. The temporal relationship of stroke to re-inflation of a totally atelectatic pulmonary lobe suggests the possibility of embolism due to pulmonary vein thrombosis in the atelectatic lobe, followed by clot fragmentation and embolization after expansion.

Case 5

The patient was a 78-year-old woman who underwent radical vulvectomy for squamous cell carcinoma. There were no cervical bruits. During the 6½ hour procedure, BP transiently decreased to 80 mm Hg systolic and later was 100 mm Hg for one hour (baseline BP was 140/75 mm Hg). Initial post-operative course was uneventful; she was treated with low-dose heparin (5000 units subcutaneously twice daily) for the entire postoperative period. On the fifth postoperative day, she developed a moderate left hemiparesis. CT was normal; EKG was unchanged. Neurological deficits slowly resolved over several weeks with no recurrence of stroke in the subsequent two years of follow-up.

Case 6

The patient was a 68-year-old chronic alcoholic man with longstanding atrial fibrillation who was evaluated for acute abdominal pain, nausea and vomiting. Laparotomy revealed infarction of the ileum and right colon, and was complicated by a transient fall in BP to 100/70 mm Hg (baseline BP was 140/90 mm Hg). Postoperatively, BP remained in the range of 110/70 mm Hg. On the seventh postoperative day, confusion and left-sided weakness were noted which progressed over 12 hours to severe hemiparesis. BP, hematocrit and EKG were unchanged during the onset of stroke. CT showed a right parietal infarction; echocardiogram was technically inadequate.

Case 7

The patient was a 90-year-old man admitted for repair of large (10 cm by 4 cm), asymptomatic popliteal aneurysms. During general anesthesia with ketamine and nitrous oxide, BP transiently decreased to 90/50 mm Hg (Baseline BP was 130/70 mm Hg) during surgery. Two days postoperatively, heparin 5000 units twice daily was initiated, but was decreased to 3500 units twice daily after partial thromboplastin time increased from 34.2 seconds to 50.6 seconds. On the eighth postoperative day, severe chest pain heralded inferior myocardial infarction. On the 13th postoperative day, low-dose heparin was discontinued as he was fully ambulatory. On the 18th postoperative day he awakened with moderate dysphasia and right facial weakness. BP and hematocrit were unchanged. Neurologic deficits resolved over the next 72 hours. He was treated with aspirin and subsequently died of pneumonia three years later without further cerebrovascular symptoms.

Case 8

The patient was an 80-year-old man with chronic, nonvalvular atrial fibrillation who underwent repair of ocular lens prolapse which followed earlier prosthetic lens implantation. Intraoperative course was uncomplicated; he returned to normal activity status on the first postoperative day. On the second postoperative day he awakened with dysarthria and a mild right hemiparesis. Vital signs and EKG were unchanged. A CT showed a left parietal infarction. On the sixth postoperative day, the right foot became suddenly cyanotic. Thrombectomy was successfully carried out after arteriography revealed occlusion of the common iliac artery.

Case 9

The patient was a 69-year-old woman with longstanding hypertension who underwent abdominal aortic aneurysm resection. Three weeks before, bilateral carotid endarterectomies had been carried out for a central retinal artery occlusion and contralateral, asymptomatic carotid stenosis. Aortic surgery was complicated by intraoperative myocardial infarction. On the third postoperative day, BP elevation of 160/120 mm Hg was followed by mild stupor and right hemiparesis. CT showed an area of increased density.
Discussion

Ten of 12 (83%) cases of perioperative cerebral infarction had onset in the postoperative period, remote from the influences of intraoperative hypotension or hypoxemia. Although the extent of carotid occlusive disease was unknown in most of our patients, these data seriously question the role of intraoperative hypotension combined with carotid occlusive disease in the genesis of perioperative cerebral infarction.

There are several caveats inherent to retrospective studies that may particularly apply to these data. Minimal focal deficits may have gone unrecognized or unrecorded; patient selection based on discharge diagnosis codes may not represent consecutive patients with all degrees and locations of focal cerebral infarction. Hypotension in a setting of bilateral carotid stenosis could produce borderzone ischemic damage mimicking nonfocal encephalopathy; all patients in this review with ischemia-related encephalopathy had undergone cardiac surgery and, thus, were not further considered. Undoubtedly some number of preoperative patients underwent prophylactic carotid surgery on the basis of carotid bruits or noninvasive carotid screening. If such surgery is beneficial in reducing perioperative stroke, this would result in underrepresentation of such stroke mechanisms in our sample. Despite these potential limitations, these case reports illustrate that many different mechanisms may be operable in patients with perioperative stroke (table 2).

These 12 instances of perioperative stroke, plus the five specific exclusions related to anticoagulants and tonsillectomy, represented a prevalence of 0.07% of all general surgical procedures, excluding cardiac and carotid surgery. This low prevalence may suggest that case finding was incomplete; however, the series reporting a higher prevalence (0.38%) included only male patients over age 50 and was also retrospective. The perioperative stroke rate in older patients (65 or older) in this study was comparable: 0.22%.

The potential role of carotid occlusive disease in the genesis of perioperative stroke can be further defined from several recent reports involving patients undergoing peripheral vascular reconstruction. Carotid occlusive disease was relatively common in these patients: asymptomatic bruits were present in about 15% (table 3) and asymptomatic stenosis by noninvasive vascular testing were present in 10–18%. The overall rate of perioperative cerebral infarction was low and was not increased in subgroups of patients with asymptomatic bruits or evidence of carotid stenosis by noninvasive vascular testing (table 3). Further, all 10 patients with perioperative stroke from these series in whom the temporal relationship of stroke to surgery was given had stroke two or more days after surgery, often in association with postoperative myocardial infarction or atrial fibrillation. Thus, there is no convincing evidence from these reports linking perioperative stroke to the presence of bruits or noninvasive evidence of carotid stenosis in asymptomatic patients undergoing peripheral vascular reconstruction.
operative stroke is contributed to by misclassification of hemodynamically insignificant degrees of carotid stenosis by noninvasive testing. Thus, there may indeed be a subset of perioperative stroke patients in whom severe carotid occlusive disease coupled with intraoperative hypotension are the crucial determinants of cerebral ischemia. However, the uncertain adequacy of collateral circulation makes prediction of the patient at high risk of perioperative stroke in the setting of asymptomatic carotid occlusive disease currently unreliable. In many institutions, the complication rates of cerebral arteriography and prophylactic endarterectomy are not negligible and are comparable to the risk of perioperative stroke. Thus, special precautions to avoid intraoperative cerebral hypoperfusion may be a more judicious approach to protecting most patients with asymptomatic carotid occlusive disease from perioperative stroke.

Although most inhalational anesthetics decrease cerebral oxygen utilization and increase cerebral blood flow, blood flow to ischemic brain during general anesthesia has been shown to sometimes decrease, both in experimental animals and in humans, presumably by vasodilation in nonischemic areas resulting in a "steal" phenomena. Impairment of cerebral autoregulation and iatrogenic hypocoagulability with resultant decreased cerebral blood flow during general anesthesia offer further potential threats to cerebral tissues. Therefore, intraoperative control of BP and oxygen and carbon dioxide tensions is critically important in maintaining cerebral perfusion in these patients.

The role of postoperative hypercoagulability in perioperative stroke is ill-defined. A hypercoagulable state in the postoperative period is supported by the common occurrence of venous thrombosis and measurable alterations in clotting factors, platelet number and function and fibrinolytic activity. Additionally, postoperative increases in blood viscosity have been demonstrated, related to increased fibrinogen and decreased red cell deformability. Microcirculatory impairment due to increased viscosity could enhance, if not directly initiate, cerebral ischemia. However, laboratory abnormalities have not been reliable predictors of perioperative arterial or venous thrombosis and correlation of laboratory indices of a prothrombotic state and postoperative stroke has not yet been attempted.

Despite the paucity of direct evidence, it is tempting to speculate on potential contributions of a postoperative prothrombotic state to perioperative stroke. A large fraction of our patients had concomitant atrial fibrillation. Nonvalvular atrial fibrillation in elderly patients is often related to coronary atherosclerosis; such patients might be expected to harbor carotid atherosclerosis as well. However, it is conceivable that a mild prothrombotic state in the postoperative period might favor formation of left atrial thrombus and lead to embolic stroke. Similarly, a prothrombotic state combined with ulcerative atheroma may possibly initiate thrombosis in a previously asymptomatic lesion.

In three patients with postoperative stroke (#1, 2,

<table>
<thead>
<tr>
<th>Table 2 Possible Mechanisms of Perioperative Stroke</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>I. Intraoperative</strong></td>
</tr>
<tr>
<td>A. hypotension</td>
</tr>
<tr>
<td>1. diffuse or borderzone ischemia</td>
</tr>
<tr>
<td>2. focal ischemia potentiated by vascular occlusive disease</td>
</tr>
<tr>
<td><strong>B. contributing factors</strong></td>
</tr>
<tr>
<td>1. anemia</td>
</tr>
<tr>
<td>2. hypoxemia</td>
</tr>
<tr>
<td>3. hypocarbia</td>
</tr>
<tr>
<td>4. hypercarbia</td>
</tr>
<tr>
<td><strong>II. Postoperative</strong></td>
</tr>
<tr>
<td>A. hypotension of any cause (see I.A. above)</td>
</tr>
<tr>
<td>B. embolic</td>
</tr>
<tr>
<td>1. perioperative myocardial infarction or atrial fibrillation</td>
</tr>
<tr>
<td>2. postoperative bacterial endocarditis</td>
</tr>
<tr>
<td>3. paradoxical embolism from crural venous thrombosis</td>
</tr>
<tr>
<td>C. hypercoagulability</td>
</tr>
<tr>
<td>1. potentiating cerebrovascular atherosclerosis</td>
</tr>
<tr>
<td>2. potentiating atrial fibrillation</td>
</tr>
<tr>
<td>a. reduced fibrinolytic activity</td>
</tr>
<tr>
<td>b. increased coagulation factors</td>
</tr>
<tr>
<td>c. reduced plasma coagulation inhibitors</td>
</tr>
<tr>
<td>d. increased platelet activation and number</td>
</tr>
<tr>
<td>D. contributing factors</td>
</tr>
<tr>
<td>1. anemia</td>
</tr>
<tr>
<td>2. iatrogenic polycythemia and hyperviscosity</td>
</tr>
<tr>
<td>3. hypoxemia</td>
</tr>
<tr>
<td>E. cortical vein and sinus thrombosis</td>
</tr>
</tbody>
</table>

This apparent lack of relationship between carotid occlusive disease and perioperative stroke in such patients is surprising. However, impairment of cerebral blood flow requires a high degree of carotid stenosis and collateral circulation is often adequate. It is conceivable that the lack of correlation of noninvasive evidence of carotid stenosis and the occurrence of perioperative stroke is contributed to by misclassification.

<table>
<thead>
<tr>
<th>Table 3 Perioperative Stroke and Cervical Bruit in PVR*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No.</strong></td>
</tr>
<tr>
<td>-----------------</td>
</tr>
<tr>
<td>All patients</td>
</tr>
<tr>
<td>( + ) Bruits†</td>
</tr>
<tr>
<td>No bruises</td>
</tr>
</tbody>
</table>

*Aggregate data from 4 recent reports comprised of patients with no cerebrovascular symptoms undergoing peripheral vascular reconstruction (PVR) including abdominal aortic surgery. Stroke includes focal deficits only. Data are not included because 43% of their patients had cerebrovascular symptoms.

†Thompson additionally reported a 3.6% perioperative stroke rate in 138 general surgical and PVR patients with asymptomatic bruits. Intraoperative versus postoperative onset of stroke and stroke rate in patients without bruits were not given.
9), stroke followed treatment of hypertension with initial, transient fall in blood pressure, although the patients were not hypotensive at the onset of neurologic deficit. Whether transient hypotension initiated thrombotic occlusion which progressed over subsequent hours is uncertain.

It is unlikely that the occurrence of stroke in the postoperative period was random, unrelated to the surgical procedure, in these relatively elderly patients. If an average hospital stay of 10 days is estimated and a random, predicted stroke incidence of 539 per 100,000 per year in ages 65–74 is assumed,40 then the expected occurrence of “random” stroke in our population would account for about one cerebral infarction.

Of related interest, aggregate data from 5 reports on perioperative stroke in coronary artery bypass procedures, with the associated intraoperative hypotensive and nonpulsatile flow, shows postoperative onset of deficit in 45% of 38 patients with focal deficits.17,22,41,43

In our series most perioperative strokes occurred in the postoperative period, apparently unrelated to hypotensive episodes. Treatment of a postoperative prothrombotic state, albeit ill-defined, may well be beneficial.22 However, the efficacy of short-term heparin and/or platelet antiaggregation agents in prevention of postoperative stroke is unknown.

Conclusions

Most perioperative cerebral infarctions in general surgical and peripheral vascular patients occur in the postoperative period and are unrelated to hypotension. Cardiogenic embolism is common. The presence of cervical bruits or evidence of carotid occlusive disease by noninvasive testing are not predictive of perioperative strokes in these patients, based on review of the literature. Failure of these two indicators to separate the very tight stenosis required to reduce flow from lesser degrees of stenosis may contribute to this apparent lack of relationship. Several mechanisms contribute to perioperative stroke. The role of altered, albeit ill-defined, coagulability in the postoperative period is of potential major importance. Future reports describing perioperative stroke should consider the multiple mechanisms and report the temporal relationship to the surgical procedure.

Acknowledgments

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References

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