Anesthesia for carotid endarterectomy

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Atherosclerosis is a systemic disorder that manifests itself as vascular occlusive or aneurysmal disease in one or more regional circulations. Patients with vascular occlusive disease of the carotid or vertebral arteries are therefore likely to have occlusive disease in other circulations, the most important of which is the coronary circulation (*Table 1*).[1, 2] In the development of carotid endarterectomy operations, innovations in anesthetic and surgical techniques naturally focused on the prevention of cerebrovascular accidents, the prime objective of the operation. However, it soon became apparent that almost half the mortality from this operation was the result of heart disease, particularly myocardial infarction. Despite this, it has been recognized only recently that some methods designed to protect the brain from ischemic damage during operation may in fact be inducing myocardial ischemia.[3] Therefore, a critical appraisal of methods of "cerebral protection" is warranted.

Cerebral protection during carotid endarterectomy in its most general sense included two different approaches (*Table 2*). The objective of the first was to increase the tolerance of the brain to ischemia during carotid cross-clamping, obviating the need for a shunt. The second consisted of devising methods for diagnosis of cerebral ischemia dur-

Table 1. Other manifestations of arterial disease in patients with carotid occlusive disease

	DeBakey[1] 1965	Ennix[2] 1979
No. of patients	804	1238
Systemic hypertension (>150/90)	52%	56%
Symptomatic heart disease	19%	17%
Stable angina	...	12.7%
Unstable angina	...	4.4%
Previous myocardial infarction	...	23.7%
Other occlusive or aneurysmal disease	52%	...

Table 2. Protection of the brain during carotid endarterectomy

Increasing tolerance to ischemia
General anesthesia
Hypercarbia
Hypocarbia
Induced hypertension
Hypothermia
Thiopental
Diagnosis of ischemia: indication for shunt
Local anesthesia
Jugular venous O_2 saturation
Jugular venous O_2 tension
Stump pressure
EEG
Regional cerebral blood flow

ing cross-clamping so that an intraluminal shunt could be inserted only when indicated. Some methods have never had an adequate clinical trial, e.g., large doses of thiopental. Other methods, e.g., shunting of all patients, stump pressures or electroencephalogram (EEG), have strong advocates whose positions are supported by their results.

The present situation is epitomized by the recent report of Kelly et al[4] and particularly the discussion that followed their presentation. That group used changes in the 10-lead EEG after carotid clamping as the criterion for insertion of an intraluminal shunt. Incidental to this they measured carotid stump pressures. Their data were interpreted to show the failure of the stump pressures below 50 torr to predict cerebral ischemia during carotid occlusion (*Table 3*). However, their data also clearly showed that use of the EEG or stump pressures or the insertion of a shunt did not prevent new postoperative neurologic deficits, and some patients with normal EEG and high stump pressures also developed postoperative neurologic deficits. Strokes occur postoperatively even when local or regional anesthesia is employed and frequent testing of neurologic status is used as the criterion for insertion of a shunt, since cerebral ischemia can develop as long as 45 minutes after carotid cross-clamping.[5]

The efficacy of any method of cerebral protection is difficult to determine, since it can be measured ultimately only by the incidence of new neurologic deficits appearing postoperatively. Such deficits result not only from cerebral ischemia consequent to occluding the carotid, but from embolization during mobilizing of the carotid bifurcation, from complications of shunt insertion, from dissection or thrombosis distal to the endarterectomy secondary to the repair, and from spontaneous distal

Table 3. Failure of carotid stump pressures; Kelly et al, 1979

Stump pressure	Normal EEG	No. of patients	Neurological deficits %
>50 torr	+	223	3.6
<50 torr	+	21	0
>50 torr†	0	17	12
<50 torr†	0	28	7
Total		289	4.2

* Transient *and* prolonged deficits included.
† Internal shunt employed.

thrombosis as part of the natural course of the disease. Since postoperative neurologic deficits have occurred in every reasonably large series of operations reported, including those in which shunts were used in all patients, it is impossible at the moment to determine what proportion of patients are at risk of postoperative neurologic damage from carotid cross-clamping alone compared with the proportion at risk from manipulation and the disease process. It follows as a corollary that the role of anesthetics or anesthetic management in preventing or producing new neurologic deficits must be equally unknown. Nor is it likely that its role will be delineated, since the incidence of mortality and postoperative neurologic deficit is generally less than 5%. As a further consequence of this low incidence, few factors that increase postoperative neurologic deficits have been as yet identified (*Table 4*). The likelihood of obtaining several series of patients balanced for even the known risk factors in order to provide a basis for testing the various hypotheses regarding cerebral protection seems remote.

Table 4. Factors affecting incidence of postoperative neurological deficits

Patients with prior stroke
Patients with contralateral carotid occlusion
Extent of collateral circulation and duration of clamping
Experience of surgeon and/or institution

Although the role of anesthetics in cerebral protection during carotid endarterectomy is generally agreed to be uncertain, there is only unanimity regarding the role of high blood pressure in preventing strokes. This opinion persists despite the lack of evidence that hypotension is a precipating factor in the genesis of strokes and even some evidence that it is not.[6, 7] The wide use of stump pressures as the criterion for insertion of intraluminal shunts and the use of vasopressors to increase stump pressures apparently strengthened this long-standing bias and has led to the common recommendation that blood pressure be kept at the "preoperative level" or even slightly higher throughout the operation, with the use of vasopressors when necessary. Considering the patient population undergoing carotid endarterectomy, half with hypertension and one quarter with previous myocardial infarction (*Table 1*), it was not surprising that focus suddenly shifted from postoperative neurological deficits to the mortality of myocardial infarction in these patients. Riles et al[3] indicted the use of metaraminol for increasing stump pressure as responsible for the high incidence of postoperative myocardial infarction in patients with preexisting heart disease and recommended increased use of shunts rather than vasopressors (*Table 5*). Ennix et al[2] were alarmed by the high incidence of postoperative myocardial infarction following carotid endarterectomy in pa-

Table 5. Myocardial infarction following carotid endarterectomy; Riles et al, 1979

	No heart disease	Heart disease*
No. of patients	288	203
Postoperative myocardial infarction	0.7%	6.9%
	No aramine—2.0%	
	With aramine—8.1%	
Deaths from myocardial infarction	0%	2.5%

* Angina, congestive heart failure, arrhythmias, previous myocardial infarction.

Table 6. Coronary artery disease in 1238 patients who underwent carotid endarterectomy (1967–1977); Ennix et al, 1979

	I	II	III*
Coronary artery disease symptoms (angina)	0	+	+
No. of patients	1026	77	135
Unstable angina	0	22%	28%
Previous myocardial infarction	18%	55%	50%
Myocardial infarction	1.0%	13%	2.6%
Cardiac/cerebral deaths	2/10	11/3	1/1
In-hospital mortality	1.5%	18.2%	3.0%

Forty-two percent of all deaths were from myocardial infarction.
* Prior or simultaneous coronary artery bypass.

tients with preoperative angina (*Table 6, Group II*). They therefore treated patients with angina by coronary artery bypass before carotid endarterectomy as a staged procedure, or performed both operations simultaneously, with improved mortality (*Table 6, Group III*).

Because of the unknown incidence of postoperative stroke from ischemia of carotid cross-clamping alone, the uncertain benefits of any method of cerebral protection and the well-known mortality rate of postoperative myocardial infarction, it would seem wise to avoid any method of cerebral protection that might increase the likelihood of perioperative myocardial infarction, such as inducing hypertension in patients with symptomatic coronary artery disease.

Anesthetic management should be directed toward avoidance of both hypertension and hypotension, maintenance of sleeping blood pressure, avoiding tachycardia and bradycardia and utilizing anesthetic agents and adjuvants that would be selected if one assumed all patients undergoing carotid endarterectomy had symptomatic coronary artery disease.

References

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