Postoperative Stroke in Cardiac and Peripheral Vascular Disease

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The postoperative stroke rate in 330 patients requiring coronary artery (170) or peripheral vascular (160) surgery was compared with the presence of carotid bruits and the results of noninvasive screening (Doppler imaging and spectral analysis of flow) to determine prevalence and significance of carotid lesions and their relationship to perioperative stroke. Carotid lesions were suspected because of bruits in 70 patients with peripheral vascular disease (PVD) and in 28 patients with coronary artery disease (CAD). Noninvasive tests showed high grade stenosis or occlusion in 62 patients with PVD and in 14 with CAD. Forty-four patients with PVD and 101 patients with CAD had normal Doppler studies. The rest in both groups had plaquing without major stenosis. Noninvasive tests uncovered severe, occult lesions in only 13 patients (9 PVD, 4 CAD). Postoperative neurologic complications occurred in 16 patients (13 strokes: 5 PVD, 8 CAD and 3 TIAs: 2 PVD, 1 CAD). Thirteen neurologic complications occurred in patients having nonstenotic plaques or normal carotids without bruits. Only three of the strokes and 1 TIA occurred in patients with bruits and detectable carotid stenosis. Few of the postoperative strokes or TIAs were focal (2 PVD, 1 CAD), and the rest were nonfocal. None of the postoperative strokes or TIAs were associated with postoperative carotid occlusion. Physical examination is not an accurate method of determining severity of carotid disease. Severe carotid stenosis is more common in PVD patients than in CAD patients, but there is no significant difference in postoperative stroke rate. No direct relationship has been found between a bruit, severity of disease, and incidence of perioperative stroke.

MANY STUDIES HAVE SHOWN that the presence of a cervical bruit or a history of transient cerebral ischemia are associated with an increased incidence of subsequent stroke. However, little is known as to whether these same factors are associated with an increase in postoperative strokes. The purpose of this study is to determine the perioperative stroke rate in 330 high risk patients requiring elective coronary artery or peripheral vascular reconstructive surgery and to correlate these events with the prevalence and severity of pre-existing carotid artery disease.

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Materials and Methods

Between July of 1977 and December of 1979, all patients admitted for elective coronary artery bypass or peripheral vascular reconstruction were preoperatively screened for cervical carotid lesions. One hundred seventy patients with coronary artery disease (CAD) and 160 with peripheral vascular disease (PVD) were carefully questioned for symptoms of cerebrovascular insufficiency and examined for cervical bruits. The clinically defined prevalence of carotid disease based on these findings was compared with the incidence and hemodynamic characteristics of carotid disease as determined by noninvasive testing. Doppler imaging and spectral analysis of blood flow signals was used for screening these patients. These noninvasive tests were performed before surgery in order to determine the hemodynamic significance of suspected lesions and to detect clinically occult occlusive disease. This data was then correlated with the incidence of postoperative neurologic complications. Furthermore, postoperative noninvasive tests were performed on those patients with cerebrovascular complications in order to determine patency of the carotid vessels.

Results

There were significant risk factor differences between the coronary artery patients and peripheral vascular patients with regard to age, incidence of hypertension, diabetes mellitus, and hyperlipidemia. The peripheral vascular group was older, and more frequently hypertensive and diabetic. Hyperlipidemia, on the other hand, was more common in the coronary artery group (Table 1). Carotid artery lesions were suspected because of cervical bruits in 98 patients (30%:28 CAD, 70 PVD). Bruits were more prevalent in the PVD group (Table 2). The diagnosis of carotid disease was suspected on the basis of previous cerebral

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TABLE 1. *Incidence of Hypertension, Diabetes, and Hyperlipidemia in Cardiac and Peripheral Vascular Patients*

	Age	Hyper-tension	Diabetes	Hyper-lipidemia
CAD (170 Patients)	52	34	19	24
PVD (160 Patients)	60	90	37	10

TABLE 2. *Estimated Prevalence of Carotid Occlusive Disease Based on Cervical Bruit*

	Total	Unilateral	Bilateral
CAD (170 Patients)	28	14	14
PVD (160 Patients)	70	25	45

ischemic symptoms in 108 patients (33%:39 CAD, 69 PVD). A history of focal ischemic symptoms was more common in the peripheral vascular patients. Less than half of the clinically symptomatic patients had cervical bruits (Table 3). Based on a history of cerebral ischemia and/or the presence of a cervical bruit carotid artery disease was suspected in 155 patients (47%:57 CAD, 98 PVD).

Noninvasive testing showed that 45 of the 98 patients with cervical bruits did not have significant carotid stenosis, and demonstrated unexpected complete occlusion of the internal carotid artery in 11 patients (7 with bruits, 4 without bruits). Severe occlusive disease in the absence of clinical signs or symptoms was uncovered by noninvasive testing in 17 patients (5%: CAD 2, PVD 15). On the basis of noninvasive test results alone, carotid occlusive lesions occurred in 103 patients (31%:CAD 20, PVD 83) (Table 4).

Postoperative cerebral ischemia occurred in 16 patients (CAD 9, PVD 7). This is 4.8% of the study group. There were 13 strokes and 3 TIAs. Six of these were focal suggesting carotid origin, the rest were diffuse neurologic deficits. Ten of the 16 patients with deficits did not have bruits, and only five had carotid stenosis (Tables 5 and 6). None of the patients that developed a neurologic complication had postoperative carotid occlusion by noninvasive testing. In no patients with a clinically occult stenosis or occlusion did a postoperative complication occur. Only three of the

TABLE 3. *Estimated Prevalence of Carotid Occlusive Disease Based on Previous History of Cerebral Ischemia*

	Bruits	No Bruits	Totals
CAD (170 Patients)	10	29	39
PVD (160 Patients)	41	28	69
			108

TABLE 4. *Prevalance of Carotid Occlusive Disease Based on Noninvasive Testing*

	Stenosis	Occlusion
CAD (20 Patients)	20	—
PVD (83 Patients)	72	11
Total (103 Patients)		

patients with perioperative stroke had a history of previous cerebral ischemic symptoms.

Discussion

Although the relationship between stroke and the presence of carotid occlusive disease has been firmly established, the concept that postoperative stroke is the result of surgically untreated carotid disease needs to be more adequately evaluated.[4,5,7] Indeed, there is evidence to suggest that the presence of cervical bruits and/or cerebral ischemic symptoms may not be accurate indicators of perioperative stroke risk. Postoperative strokes are rare events, and occurred in only 4.8% of our study group. This is consistent with the clinical experience of other investigators.[1-3,8,9] Most of these neurologic deficits were not lateralizing or focal suggesting diffuse cerebral ischemia. Ten of the 16 patients with postoperative cerebral ischemia (63%) did not have cervical bruits, and only five patients (31%) had carotid stenosis. Most of the patients with postoperative stroke or TIAs had normal or nonstenotic plaquing disease in the carotids. Three of the stroke victims had a previous history of cerebral ischemia. None of the neurologic complications were associated with postoperative carotid occlusion. The incidence of postoperative stroke was not appreciably different for the coronary artery or peripheral

TABLE 5. *Incidence of Cervical Bruit in Patients with Postoperative Cerebral Ischemia*

	Stroke		TIA	
	Bruit	No Bruit	Bruit	No Bruit
CAD (9 Patients)	2	6	—	1
PVD (7 Patients)	2	3	2	—

TABLE 6. *Incidence Carotid Occlusive Disease in Patients with Postoperative Cerebral Ischemia*

	Stroke		TIA	
	Normal/Wall Disease	Stenosis	Normal/Wall Disease	Stenosis
CAD (9 Patients)	6	2	1	—
PVD (7 Patients)	3	2	1	1

vascular patients despite the fact that the prevalence of carotid occlusive lesions was significantly higher in the PVD group. However, in the CAD group there was a higher incidence of diffuse neurologic deficits.

Noninvasive tests suggest that the prevalence of carotid occlusion is overestimated and that physical examination is not an accurate method of determining the severity of carotid disease. It shows that an appreciable number of cervical bruits are not related to carotid stenosis, that severe lesions can exist in the absence of clinical findings, that postoperative stroke may occur in the absence of occlusive lesions, and that perioperative neurologic deficits are rarely caused by carotid thrombosis.

No direct relationship has been found between cervical bruit, the severity of carotid disease, and the incidence of perioperative stroke. Because of this we do not recommend routine prophylactic carotid endarterectomy prior to coronary artery bypass or peripheral vascular reconstruction except possibly in the case where the patient is actively having transient cerebral ischemic symptoms.

References

1. Barnes RW, Marszalek PB, Rittgers SE. Asymptomatic carotid disease in preoperative patients. Stroke 1980; 11(1): 136.
2. Bernhard VM, Johnson WD, Peterson JJ. Carotid artery stenosis association with surgery for coronary artery disease. Arch Surg 1972; 105:837.
3. Evans WE. Cooperman M. The significance of asymptomatic unilateral carotid bruits in preoperative patients. Surgery 1978; 83:521.
4. Haas WK, et al. Joint study of extracranial occlusion: II. Arteriography, techniques, sites, and complications. JAMA 1968; 203:159.
5. Fields WS, Maslenikov V, Meyer JS, et al. Joint study of extracranial occlusive disease: V. Progress report of prognosis following surgery or nonsurgical treatment for transient cerebral ischemic attacks and cervical carotid artery lesions. JAMA 1970; 211:1993.
6. Michigan JT, Buch WS, Pipkin RD, Fogarty TJ. A planned approach to coexistent cerebrovascular disease in coronary artery bypass candidates. Arch Surg 1977; 112:1403.
7. Thompson JE, Austin DJ, Patman RD. Carotid and arteriotomy for cerebrovascular insufficiency: long term results in 592 patients followed for thirteen years. Ann Surg 1970; 172:663.
8. Treiman RL, Foran RF, Shore EH, Levin PM. Carotid bruit significance in patients undergoing an abdominal aortic operation. Arch Surg 1973; 106:803.
9. Tufo HM, Ostfeld, AM, Shekelle R. Central nervous system dysfunction following open heart surgery. JAMA 1970; 212:1333.

Discussion

Dr. Victor Bernhard (Milwaukee, Wisconsin): Many surgeons have embraced the prejudice that the presence of a carotid bruit in a patient who is to undergo a major surgical procedure should be evaluated to rule out a significant stenosis which may produce a perioperative stroke. However, three previous studies in addition to the authors' investigation consistently contradict that thesis. Whereas previous studies have relied on a carotid bruit to identify carotid stenosis, Dr. Turnipseed and his colleague have used a noninvasive test more precisely to identify patients presumed to be at risk. In their hands, ultrasonic mapping and spectral analysis seem to be highly accurate, although in the hands of others these methods have not been quite as reliable.

The broader issue raised by the authors is how to identify a patient who, during some major operative procedure, may be in danger of having a stroke. It is possible that by merely looking for stenosis, either by listening for a bruit or by noninvasive testing, we have all been looking at the wrong thing.

(slide) This slide, which summarizes previous retrospective studies, demonstrates that postoperative stroke is infrequent and that there is no difference in the incidence of stroke regardless of whether the patient did or did not have a bruit, or even whether the patient did or did not have symptoms.

Furthermore, it is important to note that those patients who did have strokes, and in the aggregate there were only six, invariably had this complication after the third postoperative day. This would seem to reduce our concern that a stenosis is a threat merely on the basis of hypoperfusion during the operative procedure. However, hypoperfusion may be a factor after the third postoperative day when we are monitoring the patient much less closely, at a time when the patient may also be in some poorly defined hypercoagulable state. I would think that a clearer understanding of the exact cause of postoperative stroke would help us to identify those patients at risk. In this regard, I would like to ask the authors if they could more carefully define the cause of the strokes that did occur in their patients in the postoperative period.

The aggressive use of standard angiography as a preoperative screen may not only increase stroke morbidity but also lead to confusion. (slide) The patient whose angiogram is depicted on this slide entered the hospital for the repair of an asymptomatic abdominal aortic aneurysm. He had no neurologic symptoms but had bilateral carotid bruits. Because the patient was reluctant to have carotid angiography, we proceeded to repair his aneurysm forthwith. On the seventh postoperative day left hemispheric symptoms developed —transient ischemic attacks, and the patient underwent angiography. On the right side the bruit was caused by a rather menacing 60% stenosis with ulceration, whereas the left sided murmur was produced by an external carotid lesion. If we had seen this angiogram preoperatively, we would have operated on his right side, but I will remind you that his symptoms were related to the left-sided cerebral circulation.

This anecdote highlights the problem of identifying patients undergoing major surgical procedures who are at risk for a cerebrovascular complication. Another point to be considered, therefore, is whether or not other noninvasive techniques may be helpful to us in identifying these patients. Specifically, will the use of computerized intravenous angiography under investigation at the authors' institution be able to define patients who have extracranial or intracranial lesions that may be significant?

Dr. Jesse E. Thompson (Dallas, Texas): As Dr. Shires stated, one of the unsolved problems in carotid surgery has been the risk posed by an asymptomatic carotid bruit in patients undergoing operation in areas other than the carotid artery. The incidence of stroke after any operation is really quite low, but accidents continue to occur in patients with and without carotid bruits.

We have performed prophylactic carotid endarterectomy when we thought patients were at risk, but not very often. In a consecutive series of 108 abdominal aortic aneurysms, we did carotid endarterec-