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Stroke 1988;19;1485-1490
Stroke is published by the American Heart Association. 7272 Greenville Avenue, Dallas, TX 72514
 Print ISSN: 0039-2499. Online ISSN: 1524-4628

Carotid Endarterectomy: To Shunt or Not to Shunt

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Because of controversies in the cerebrovascular literature regarding the use of an intraluminal shunt in carotid endarterectomy, we report a randomized prospective study of 118 consecutive symptomatic patients receiving surgery within a single neurosurgical practice. Over 4 years, 138 carotid endarterectomies were performed in the 118 patients, 63 operations with intraluminal shunting and 75 without. Standard rationale for surgery included ipsilateral cerebral infarction in 38% of the operations and ipsilateral transient ischemic attacks in 36%. Unilateral angiographic stenosis of >90% was seen in 58% of the operations; there were no ipsilateral occlusions. Surgery was performed under general anesthesia with barbiturate induction and mild blood pressure elevation. The 30-day complication rate included a mortality rate of 0.7% with a 5.1% incidence of postoperative neurologic deficit and a 1.4% rate of myocardial infarction. In the 24 hours after surgery there were no cerebral infarctions in the shunted group and six in the unshunted group. This 8% rate in the unshunted group compared with 0% in the shunted group was significant at $p=0.023$ with a power of 0.95 by Fisher's exact test and χ^2 analysis. This suggests that in our neurosurgical practice (resident training program) the use of an intraluminal shunt during carotid endarterectomy significantly reduces the risk of intraoperative neurologic deficit without increasing the incidence of other complications. (*Stroke* 1988; 19:1485–1490)

Carotid endarterectomy (CE), as the third most common operation performed in the United States, has recently come under close scrutiny in terms of both patient selection and surgical technique. A major controversy revolves around the use of an intraluminal shunt, be it selectively or routinely, with or without electroencephalography (EEG), stump pressure measurements, or cerebral blood flow (CBF) monitoring.[1,2]

Arguments against the use of a shunt include its unnecessary use in some 85% of cases, its attendant morbidity (risk of atheromatous or air emboli, intimal dissection, acute occlusion), its effect on lengthening procedure duration, or limiting plaque exposure.[1,3] One might also argue that the use of a shunt, because of the need for increased exposure, increases the risks of nerve damage, postoperative bleeding, or infection. Those supporting the use of a shunt cite its value in maintaining CBF, thus allowing unhurried CE, and in stenting the vessel during closure.[4–6] They note that once a surgeon becomes accustomed to it, a shunt is not a technical hindrance. A further concern is that for those surgeons who shunt selectively, relatively infrequent use of a shunt makes its use more hazardous in the hands of one less technically experienced just when a shunt is deemed most necessary.

A small, randomized, prospective study of consecutive patients undergoing CE has been performed to determine whether there is any significant difference within a single neurosurgical practice group in the postoperative complications of those patients undergoing surgery with and without intraluminal shunting.

Subjects and Methods

After preoperative neurosurgical evaluation, including bilateral carotid angiography and informed consent, consecutive patients underwent CE. All patients within our neurosurgical practice group (a residency training program) were randomized to either the shunted or unshunted group. All CEs were performed by either the attending neurosurgeon or by a senior neurosurgical resident under direct supervision. General endotracheal anesthesia after barbiturate induction was used, and the blood pressure was maintained at or slightly above the patient's preoperative baseline. During carotid clamping and/or shunting, the blood pressure was raised 10–20%. All patients received 5,000 units of

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Received March 7, 1988; accepted June 30, 1988.

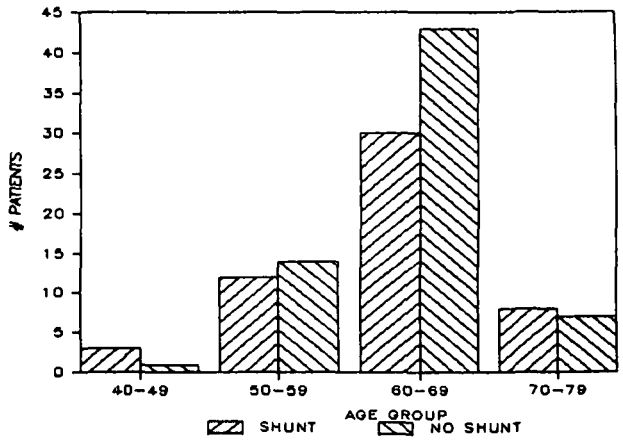

FIGURE 1. *Age distribution histogram indicates that 62% of 118 symptomatic patients undergoing carotid endarterectomy were in their seventh decade of life.*

heparin. In shunted patients a Javid shunt was held in place with Rummel tourniquets; vascular clamps occluded the vessels in unshunted patients. Primary arterotomy closure was with 6-0 Prolene suture. During the first half of the series (68 CEs), all patients were given protamine sulfate for heparin reversal; this practice was discontinued thereafter. Patients were discharged 3–7 days postoperatively, with outpatient follow-up at 1 month to evaluate any developments over the 30-day postoperative period.

Interim biostatistical analyses were performed after each complication using standard significance levels ($p \leq 0.05$) and 90% confidence limits. Both the χ^2 test (analysis of categorical outcome) and Fisher's exact test (for small expected values) were used.

Results

Over 4 years (September 1, 1982–October 31, 1986), 138 CEs were performed in 118 patients (Figure 1), 53 (63 CEs) with intraluminal shunting and 65 (75 CEs) without shunting; 19 patients underwent bilateral CE, and one required reoperation 2.5 years later. One other patient operated on before the study had CE for recurrent stenosis during the study. The rationale for surgery is identified in Table 1; there were no asymptomatic patients in our series. Systemic disease was present in 92% of the patients (Table 1).

Carotid angiography was performed before CE in all patients (Table 2). No patient had angiographic evidence of ipsilateral carotid occlusion. However, one unshunted patient was found at surgery to have an occluded vessel (fresh thombus), which was reopened. Among the shunted CEs, 20 (32%) were performed in patients with *contralateral* stenosis of >90%, including nine with contralateral occlusion. In the unshunted group, 14 CEs (19%) were performed in patients with known *contralateral* stenosis of >90%, including six with contralateral occlusion.

TABLE 1. **Rationale for Surgery and Comorbidity for 138 Carotid Endarterectomies Performed in 118 Symptomatic Patients**

	Shunted	Unshunted
Rationale (N=138)		
TIA, ipsilateral	24	24
Infarction, ipsilateral	22	30
Amaurosis fugax	10	14
Vertebrobasilar insufficiency	5	4
Hollenhorst plaque	1	1
Positive computed tomogram	1	1
Infarction, contralateral	0	1
Total procedures	63	75
Comorbidity (N=118)		
Hypertension	33	39
Cardiac disease	30	40
Pulmonary disease	11	19
Peripheral vascular disease	10	17
Diabetes	13	11
Hyperlipidemia	5	2
Healthy	6	4

TIA, transient ischemic attack.

One patient in the shunted group had had previous neck irradiation, and the long segment of carotid atherosclerotic disease was presumed to be the result of accelerated atherosclerosis secondary to radiation.[7] With this exception, all patients in the study suffered primary atherosclerotic cerebrovascular disease.

In three patients in the shunted group, including two in whom the shunt was quickly removed just after insertion when atheromatous debris were seen in the shunt, a Javid shunt was not used. In the third patient, the distal internal carotid artery (ICA) would not easily accept the shunt. None of these patients had any difficulties postoperatively.

Postoperative complications (Table 3) in the shunted group included one death, two myocardial infarctions, and one patient who suffered a posterior cerebral artery infarction during coronary artery bypass grafting 12 days after uneventful CE.

Six patients in the unshunted group suffered new neurologic deficits postoperatively (Tables 3 and 4). Two of these six had delayed onset of neurologic deficit, which occurred 3 and 15 hours postopera-

TABLE 2. **Findings of Carotid Angiography Performed Before 138 Carotid Endarterectomies in 118 Symptomatic Patients**

	Shunted (*n*=63)		Unshunted (*n*=75)	
Stenosis	I	C	I	C
<60%	6	18	11	32
60–89%	22	11	19	14
90–95%	19	7	22	4
>95%	16	4	23	4
100%	0	9	0	6
Unavailable	0	8	0	10
CE	—	6	—	5

I, ipsilateral; C, contralateral; CE, previous angiogram and endarterectomy, patency documented by duplex scan, no repeat angiogram.

TABLE 3. Postoperative Morbidity and Mortality for 138 Carotid Endarterectomies Performed in 118 Symptomatic Patients

Complication	Shunted (n=63)	Unshunted (n=75)
Death	1	0
Cerebral infarction	1*	6†
Myocardial infarction	2	0
Nerve injury	3	2
Transient ischemic attack	2	2
Wound hematoma	1	1
Wound infection	0	1
Seizures	0	1
Symptomatic restenosis	1	0

Death due to myocardial infarction; cerebral infarction defined as any neurologic deficit lasting for >24 hours.
*12 days after carotid endarterectomy.
†All within first 24 hours after surgery.

tively; each had an occluded ICA at postoperative angiography. Both underwent emergent reoperation with intraluminal shunting and patch grafting. Four of the six patients awoke with immediate postoperative deficit; postoperative angiograms were normal in all four. One of the four patients had a small cortical hyperdensity on computed tomography (CT scan) (Figure 2). There were no deaths or myocardial infarctions in the unshunted group.

In the first 24 hours after CE, we observed an 8% (six of 75) stroke rate in the unshunted group. Compared with no infarctions in the shunted group, this finding was significant at p=0.023 with a power of 0.95 using the χ^2 and Fisher's exact tests. With the exception of these cerebral infarcts, no significant difference in morbidity and mortality between the two groups (Table 3) and no increased incidence of complications attributable to the use of a shunt were identified.

Discussion

The use of an intraluminal shunt during CE is controversial. Our study is an attempt to answer cogently this question using a randomized design. The groups were comparable in general health, presenting signs and symptoms, and angiographic findings; surgical technique varied only in the use of a shunt. We assumed the null hypothesis of no difference in outcome between CE with a shunt and CE without.

In reviewing the literature it is difficult to determine the true incidence of postoperative neurologic deficit lasting >24 hours (the formal definition of infarction) because of the liberal use of the term "transient" to describe neurologic deficits.[3-5,8-21] The formal definition of a transient ischemic attack (TIA) is any neurologic deficit that clears within 24 hours. In reviewing surgical series throughout the literature, one finds transient used to describe a deficit lasting from <24 hours to one clearing "prior to discharge," certainly an indeterminate time.[3,11,12,19,22] Other series use transient to mean as long as 21 days, thus including resolving ischemic neurologic deficits (RINDs).[22,23] Where possible, we have attempted to define the rates of stroke as any neurologic deficit lasting for >24 hours. Therefore, we have made assumptions in preparing Table 5 that may slightly overestimate or underestimate neurologic deficits lasting for >24 hours. It was not possible to assess each series for 30 days since this was rarely the reported length of follow-up. The literature reports major morbidity and mortality ranging from 0% to 21%. The 2% mortality in the multicenter combined retrospective review comprises stroke and myocardial infarction.[22] Our single death (0.7%) was of cardiac etiology.

The etiology of post-CE neurologic deficit is postulated to be the result of arterial thrombosis, embolization, cerebral hyperperfusion, or intraoperative ischemia. Diagnosing the specific cause in each patient is difficult and often speculative. Our shunted patient who died had a posterior cerebral artery territory infarct following coronary artery bypass grafting 12 days after an uneventful CE; the posterior cerebral artery filled from the basilar artery on angiography. Of the six unshunted patients who suffered postoperative neurologic deficit before discharge, four deficits occurred immediately and two occurred 3 and 15 hours after CE; all occurred within the first 24 hours after surgery.

Three of the six unshunted patients who suffered postoperative neurologic deficit had abnormal preoperative CT scans. Graber et al[24] noted that positive CT scans correlated with a higher incidence of

TABLE 4. Postoperative New Neurologic Deficit After Carotid Endarterectomy in Six Patients in Unshunted Group

		Preoperative			Postoperative	
			% stenosis			
Patient	Deficit onset (hr)	Computed tomogram	I	C	Computed tomogram	Angiogram
19	15	Normal	95	100	MCA infarct	Occlusion
26	0	L parietal infarct	35	<50	No change	Negative
68	3	R parietal infarct	80	100	No change	Occlusion
87	0	Normal	>95	>95	Negative	Negative
98	0	Normal	>95	<50	Small cortical hemorrhage	Negative
138	0	L posterior infarct	>95	60	No change	Negative

I, ipsilateral; C, contralateral; L, left; R, right; MCA, middle cerebral artery.

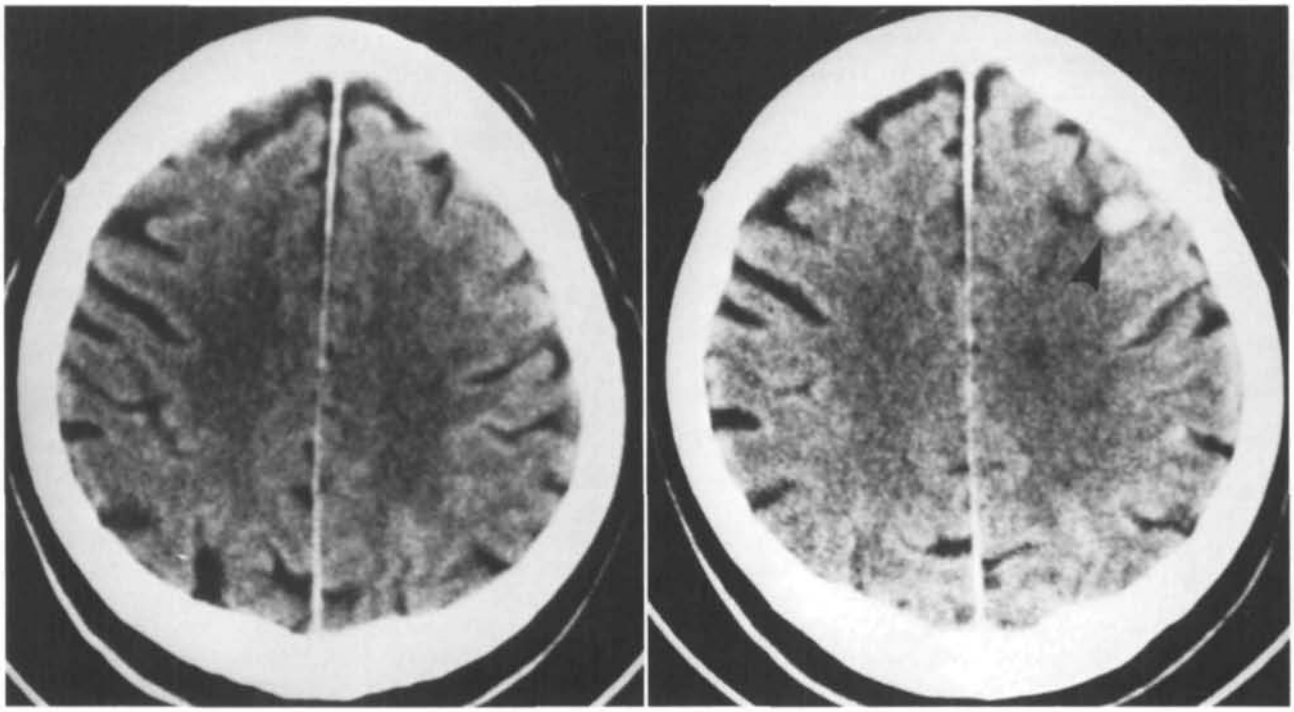

FIGURE 2. *Computed tomograms (CT scans) in Patient 98.* Left: *Preoperative CT scan is unremarkable.* Right: *CT scan after carotid endarterectomy without intraluminal shunt shows small left cortical hemorrhage at gray–white matter junction (arrowhead).*

complications. However, in our series, of the remaining 123 preoperative CT scans in patients who did not suffer postoperative neurologic deficit, 69 (56%) were abnormal.

Onset of the neurologic deficit may be helpful in determining etiology as delayed deficit is more frequently the result of carotid thrombosis, with or without secondary embolization.[20,25,26] Such neurologic deficit following a "lucid interval" has been associated with a high (44%) mortality.[19] Novick et al[26] report delayed symptomatic thrombosis 1–72 hours after CE in five patients. Rosenthal et al[20] discuss postoperative neurologic deficits in terms of onset within 6 hours. All three patients in that series with late-onset (>6 hours) deficit had thrombosis at the operative site as did seven with early-onset deficit; the remaining 17 patients with early-onset deficit had normal angiograms. Unfortunately, the authors did not distinguish between true "immediate" deficits and those within 1–6 hours.

TABLE 5. Carotid Endarterectomy Series From the Literature

Reference	CEs	Deficit		Death	
		No.	%	No.	%
Without shunt					
Allen and Preziosi[8]	154	2	1.3	1	0.6
Bland and Lazar[9]	280	3	1.1	0	0
Ferguson[1,10]	147	7	4.8	1	0.7
Morawetz et al[11,12]	142	11	7.7	3	2.1
Ott et al[13]	309	10	3.2	2	0.6
Whitney et al[14]	1917	64	3.3	37	1.9
With shunt					
Browse and Ross-Russell[3]	215	7	3.3	1	0.5
Giannota et al[4]	163	8	4.9	1	0.6
Javid et al[5]	2075	55	2.7	32	1.5
Thompson[15]	516	11	2.1	3	0.6
Selective use of shunt					
Collice et al[16]	141	6	4.3	1	0.7
Crowell and Ojemann[17]	408	12	2.9	3	0.7
Hunter et al[18]	139	2	1.4	1	0.7
Prioleau et al[19]	789	48	6.1	12	1.5
Rosenthal et al[20]	818	37	4.5	4	0.5
Sundt[21]	1992	92	4.6	25	1.3

CEs, carotid endarterectomies.

TABLE 6. Unmonitored Carotid Endarterectomies

	Shunted					Unshunted				
		Deficit		Death			Deficit		Death	
Reference	*N*	No.	%	No.	%	*N*	No.	%	No.	%
Fode et al[22]	953	68	7.1	33	3.5	955	48	5.0	15	1.6
Rosenthal[20,25]	318	14	4.4	—	—	274	14	5.1	—	—
Current study	63	0	0	1	2	75	6	8	0	0

CEs, carotid endarterectomies.

Divergent opinions exist regarding operative risk in the face of contralateral carotid occlusion.[15,27-30] In our series there were 14 known contralateral carotid occlusions (eight in the shunted, six in the unshunted group). Our two unshunted patients with delayed neurologic deficits 3 and 15 hours after CE both had contralateral ICA occlusions, and postoperative angiography showed ipsilateral ICA occlusion as well. No other patient with contralateral ICA occlusion suffered postoperative morbidity. Sachs et al[29] compared 54 CEs in patients having contralateral ICA occlusion with 503 CEs in patients having patent contralateral ICAs and determined no significant difference in morbidity and mortality between these two groups using Fisher's exact test, although the group with contralateral occlusion had a 9.3% neurologic deficit rate and no mortality while the other group had a 4.0% neurologic deficit rate and 0.8% mortality. Baker et al[30] found no increased risk with contralateral occlusion in unshunted patients, and these authors describe one patient with contralateral occlusion suffering a delayed (4 hours) postoperative neurologic deficit as a result of ipsilateral carotid thrombotic occlusion. Patterson[27] reported 23 shunted patients with contralateral occlusion and no morbidity or mortality. Phillips et al[28] reported 37 patients with contralateral occlusion, 21 operated on with intraluminal shunting and 16 without; there were no new neurologic deficits in the postoperative period.

The etiology of cerebral infarction in our four unshunted patients with immediate postoperative neurologic deficit is unclear (Table 4). Each patient had a patent carotid artery without evidence of thrombosis or cerebral emboli; three patients showed no change on postoperative CT scan. No patient had hemodynamically significant contralateral ICA stenosis. The single patient with a small superficial cortical hemorrhage on postoperative CT scan had presented with TIAs and no neurologic deficit. While clamp time was long, back-bleeding at surgery was excellent. It is unlikely that the etiology of this patient's deficit is cerebral hyperperfusion.[31] One cannot differentiate ischemic from embolic damage although the site of hemorrhage (Figure 2) suggests embolic causation. Of the remaining three unshunted patients with immediate postoperative neurologic deficit, angiograms and CT scans were not helpful; back-bleeding at surgery was good, and blood pressure stability/control was never a problem. We cannot differentiate between embolic, ischemic, or other etiology.

In the unshunted group median clamp time was 55 minutes, with the majority of clamp times <1 hour, which is relatively long but reflects the experience of others.[32,33] Littooy et al[32] noted that 24 of their patients had clamp times of >1 hour, four of whom suffered postoperative neurologic deficit. In our series, of 28 patients with clamp times of >1 hour, two patients had immediate neurologic deficit and one suffered delayed ICA thrombosis.

CEs were performed by attending staff or closely supervised neurosurgical residents. Objection may be raised regarding the fact that this study was performed at a training institution, but other studies have indicated that the results when residents are involved compare favorably with community hospital and attending staff experience.[34,35]

We did not monitor EEG, stump pressure, CBF, oculoplethysmogram, or oxygen utilization intraoperatively (Table 6). It is unclear from the literature that any correlation can be made between the results of these monitoring techniques and the necessity of shunting.[1,10,18,21-23,35,36-41] Sundt and colleagues,[21,23,36,37,41-43] with an ongoing experience using EEG and CBF measurements, advocate routinely shunting all patients with CBF of <20 ml/100 g/min. They note that two thirds of these patients will have EEG changes. It is *assumed* that EEG changes during carotid clamping are the result of neuronal dysfunction secondary to ischemia.[21]

While it is difficult to determine the etiology of neurologic deficit in all our patients, our randomized series suggests that the use of a shunt results in fewer postoperative neurologic deficits and no increased morbidity.

Acknowledgments

The patient record-keeping by our residents, Drs. Michael Malos, Michael Lawrence, and David Baker, was invaluable. We would like to acknowledge the expert advice of our biostatistician, Dr. Ron Weigel, in performing the several interim analyses as well as the final statistical evaluation. We also wish to thank Ms. Penny Joiner and Ms. Janet Sapp for their secretarial assistance in the tabulation of patient data and preparation of this manuscript.

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KEY WORDS • arteriovenous shunt, surgical • endarterectomy