Continuing Controversies

Early Carotid Surgery for Recent Neurologic Deficit

CHRISTIAN SCHUNN, M.D., GUDRUN HETZEL, M.D.,
RALF LANGE, M.D., DIETMAR BÖCKLER, and DIETER RAITHEL, M.D.

ABSTRACT **Early carotid endarterectomy after recent neurologic deficit is controversial. Traditionally, an interval of at least 4 to 6 weeks has been recommended based on poor historical results of early surgical intervention after recent strokes and fear of hemorrhagic transformation of the infarct. On the other hand, recurrent strokes have been observed in up to 9.5% of patients while awaiting carotid repair. Since 1996 we have adopted a more aggressive approach to a highly selected group of patients with fresh ischemic deficits, prolonged reversible ischemic neurologic deficit (PRIND), or crescendo transient ischemic attacks (TIAs). Of 2824 patients who underwent carotid endarterectomy between August 1996 and December 1998, 112 patients (3.9%) underwent 123 carotid operations within less than 30 days (median interval: 8 days) after a nondisabling stroke, PRIND, or TIA. Postoperatively five patients (4.4%) suffered a stroke, one patient a transient worsening of his preoperative deficit, and one patient a TIA. Six patients died, three of which were due to their stroke (mortality 5.3%). One of these fatal strokes was due to an intracerebral hemorrhage in a patient with normal preoperative computed tomography. The combined 30-day permanent neurologic stroke and death rate was 7.1%. Functional neurologic outcome was excellent with 74.1% of patients suffering no or minimal deficit (Rankin scale 0–1). Patients with unstable or progressing neurologic deficit were found to have a significantly increased postoperative stroke rate (25%). Early carotid endarterectomy after nondisabling neurologic deficit may be performed with acceptable risk in carefully selected patients. The benefit of early versus delayed surgery, however, can only be determined in a prospective randomized study.**

C.S., Assistant Professor, Section of Vascular Surgery, Department of Surgery, Robert C. Byrd Health Sciences Center, West Virginia University, Morgantown, WV; G.H., R.L., D.B., Residents in Surgery; D.R., Professor of Surgery; Director, Department of Vascular and Endovascular Surgery, Hospital Nuremberg South; Teaching Hospital of the University of Erlangen, Nuremberg, Germany.
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Carotid endarterectomy has become a standard procedure for primary and secondary stroke prevention of patients with significant extracranial carotid atherosclerotic disease. Although large randomized multicenter studies[1–3] have clarified its role for both asymptomatic and symptomatic carotid stenosis, the role of carotid endarterectomy for the patient with acute neurologic deficit or unstable carotid plaque with repetitive or crescendo transient ischemic attacks (TIAs) remains to be defined. Historically, reluctance to correct carotid lesions acutely in patients with fresh stroke or unstable neurologic status was based on observations of high postoperative mortality and poor neurologic outcome.[4–9] Subsequent recommendations to postpone surgery for at least 4 to 6 weeks have been called into question because a substantial rate of recurrent neurologic events has been observed to occur within this waiting period.[1–12] Recently we have adopted a more aggressive approach to these patients and have performed early carotid surgery after acute neurologic deficit in selected patients.

MATERIALS AND METHODS

Between August 1996 and December 1998, 2,824 patients underwent carotid reconstructive procedures at our institution. Out of this group 123 carotid operations were performed on 112 patients within less than 30 days (0–29; median 8.0 days) after a nondisabling ischemic stroke (n = 99) or crescendo TIA (n = 13). The group consisted of 25 women and 87 men, with a median age of 68.9 years (range 45–91 years). Comorbidities are delineated in Table 1.

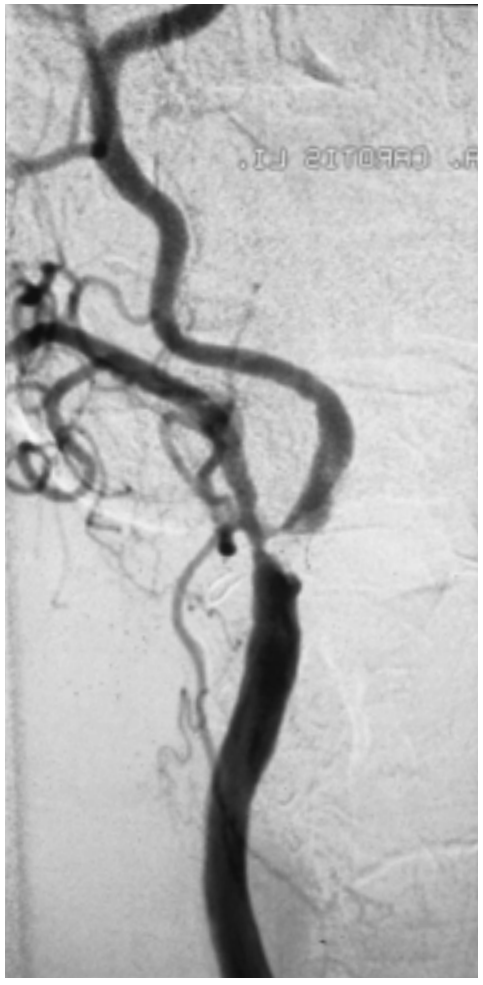
The selection criteria for early carotid intervention after an ischemic cerebral event included a limited neurologic deficit (nondisabling stroke) ipsilateral to a high-grade internal carotid artery stenosis, or occlusion with contralateral significant carotid artery stenosis. Patients with mental status changes or severe disabling hemiplegia were excluded from early repair.

A further selection criterion was cerebral appearance on computed tomography (CT) scan or magnetic resonance imaging. Large hypodense lesions (>2–3 cm), significant periinfarct edema or mass effect, and any evidence of hemorrhagic transformation were considered criteria to exclude the patients from early vascular repair.

Patients with evidence of fresh ischemic lesions on admission CT scan were reevaluated 3 to 5 days later with a second CT scan using i.v. contrast to document significant enhancement, which also served as exclusion criteria. Small patchy enhancement at the rim of a previously hypodense lesion however was not considered an absolute contraindication for early carotid repair (Fig. 1).

Table 1 Comorbidities

Diabetes	27%	Hypertension	79%
Coronary Disease	40%	CABG	6%
Obesity	33%	COPD	20%
Smoking	57%	PAOD	25%
Hyperlipidemia	45%	Amputations	4%
Renal Insufficiency	9%		



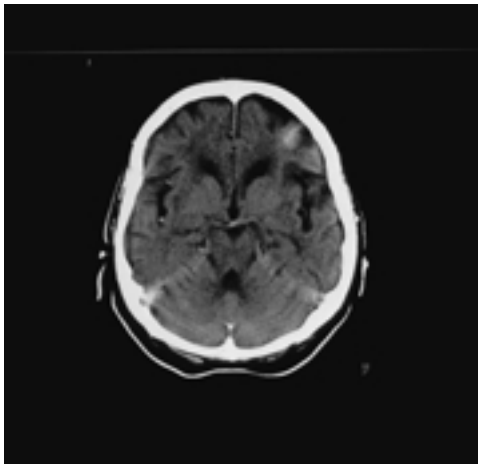


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Fig. 1 Seventy-seven year-old, right-handed female with right arm weakness and dysarthria. **(A)** Selective digital subtraction arteriogram (DSA): filiform left internal carotid stenosis. **(B)** Cranial computed tomography (CT) with i.v. contrast. Left frontoparietal ischemic defect with enhancement.

The exact interval between onset of neurologic symptoms and surgical intervention was determined by the overall clinical situation, the CT appearance of the infarct as well as by referral patterns beyond our control.

The neurologic deficit was evaluated prospectively in its evolution at admission to our service, postoperatively at discharge, and at follow-up. Neurologic deficits were classified according to the modified Rankin scale.[13] At admission patients were classified as asymptomatic, improved, stable, or showing progressive neurologic deficit compared with their initial symptoms at onset of neurologic deficit. Follow-up consisted of personal interview with the patient and, when appropriate, with family members and/or private physicians. Median follow-up was 10.0 months (range 0–28 months).

OPERATIVE STRATEGY

Once patients were selected for early carotid repair this was considered an urgent procedure and generally performed within 12 to 24 hours after the initial evaluation. The operative strategy included the use of general anesthesia, intraoperative controlled hypertension for cerebral protection during clamping, and heparinization (80–125 units per kg body weight). Cerebral monitoring was performed using somatosensory evoked potentials with shunting in only highly selected patients. Operative approach was chosen according to the individual anatomy; however, eversion endarterectomy was favored whenever feasible to minimized clamping time.

Postoperative management was focused on tight blood pressure control with aggressive treatment of any blood pressure spikes beyond the patients' normal preoperative blood pressure range. Routine ICU services, however, were not used.

Outcome evaluation included

1. Postoperative neurologic deficit
2. Thirty-day postoperative death rate
3. Functional neurologic status (Rankin criteria) at late follow-up
4. Stroke-free survival at late follow up using life-table analysis by the Kaplan-Meier method

MATERIALS

Neurologic Presentation

Of 112 patients 99 (88.4%) presented with prolonged reversible ischemic neurologic deficit (PRIND) or completed minor stroke. Thirteen (11.6%)

were operated on for repetitive/crescendo TIAs. Ninety-nine patients (88.4%) presented with ipsilateral hemispheric deficits in relation to a significant carotid stenosis, whereas 7 (6.2%) developed hemispheric deficits contralateral to a significant carotid lesion with ipsilateral carotid occlusion. Six patients (5.4%) presented with nonspecific symptoms that could not be attributed to either hemisphere. At preoperative evaluation 20% of the patients were considered asymptomatic, 48% showed marked improvement of their neurologic deficit, 20% showed no significant change since onset of their deficit, and 12% presented with unstable or progressive neurologic deficits. Functional neurologic impairment was minimal or none according to the modified Rankin scale (0/1) in 31.2%, 67% were able to ambulate with minor or moderate neurologic deficit (Rankin 2/3), and 1.8% had a moderate deficit and required assistance with walking (Rankin 4, Table 2).

Vascular morphology included preocclusive filiform stenoses in 72 (59%) patients, high-grade (60–90%) stenoses in 29 (23.8%) patients, internal carotid artery occlusion in 8 (6.6%) patients, and nonstenosing ulcers or fresh thrombus at the bifurcation in 3 (2.5%) patients. Carotid status of the contralateral side included 10 patients (8.9%) with ICA occlusion, 14 patients (12.5%) with preocclusive lesions, and 15 patients (13.3%) with high-grade stenoses.

Four of 13 patients (31%) presenting with TIAs displayed a fresh ischemic ipsilateral defect on preoperative CT scan. Of 99 patients presenting with PRIND or stable neurologic deficit 42 (42%) were found to have an ipsilateral ischemic defect.

According to their clinical status and CT findings patients were operated at a median interval of 8.0 days after onset of their neurologic deficit (range 0–29 days).

Eleven patients also underwent carotid reconstruction of the contralateral side within 2 to 11 days after their initial carotid surgery. Two of these 11 patients first underwent repair of a more high-grade carotid artery stenosis contralateral to the symptomatic side before undergoing reconstruction of their symptomatic carotid lesion.

Table 2 Functional Neurologic Presentation and Outcome

Rankin Classification	0	1	2	3	4	5	6
	n (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)	*n* (%)
Preoperative	20 (17.8)	15 (13.4)	34 (30.4)	41 (36.6)	2 (1.8)	0	0
Postoperative	61 (54.5)	22 (19.6)	8 (7.1)	13 (11.6)	1 (0.9)	1 (0.9)	6 (5.4)

Table 3 Operative Technique

Operative Technique	*n* = 123
EEA	92
TEA/Patch	10
PTFE Interposition Graft	14
Embolectomy	2
Overpatching	2
External Carotid Patchplasty	3

Operative technique is delineated in Table 3. Two patients with fresh cardiac emboli to the carotid bifurcation were treated with embolectomy only. Two fibrous stenoses were treated with overpatching. Three hypoplastic internal carotid arteries were sacrificed, and an external artery patchplasty was performed.

RESULTS

Postoperative results are outlined in Table 4.

A postoperative stroke occurred in five patients (4.4%), three of which subsequently had a fatal outcome. One of these fatal strokes resulted from an intracerebral hemorrhage and will be described below. In one patient a transient worsening of a preexisting hemiplegia resolved after several days (PRIND); one patient had a brief hemiparesis that resolved in less than an hour (TIA). This patient's immediate postoperative angiography confirmed a technically adequate repair and unveiled a previously undocumented high-grade intracranial carotid stenosis.

Table 4 Postoperative Complications

	n			*n*
Stroke nonfatal	2		Pneumonia	2
Stroke fatal	3		GI bleed	1
PRIND	1		Neck hematoma	4
TIA	1		Pulmonary edema	1
Stroke (after 2nd operation)*	2		Arrhythmia	1
			Cardiac death	3
Combined Neurologic Morbidity/Mortality	**8**	**(7.1%)**		

*Not included in overall permanent morbidity/mortality.

Two further strokes occurred among the 11 patients who underwent repair of a significant contralateral internal carotid artery stenosis during the same hospital stay. Both patients had undergone uneventful repair of the asymptomatic contralateral carotid first because of a higher degree of stenosis on that side; after repair of the second and initially symptomatic side, however, both suffered neurologic sequelae. One developed a minor stroke involving a left arm paresis on postoperative day 1. The second patient suffered an ipsilateral stroke on postoperative day 4, resulting in an incomplete left hemiparesis.

Three patients died secondary to cardiac reasons. Not accounting for sequelae of carotid surgery on contralateral carotid lesions, this resulted in a combined 30-day perioperative permanent neurologic morbidity and mortality rate of 7.1 % ($n = 8$).

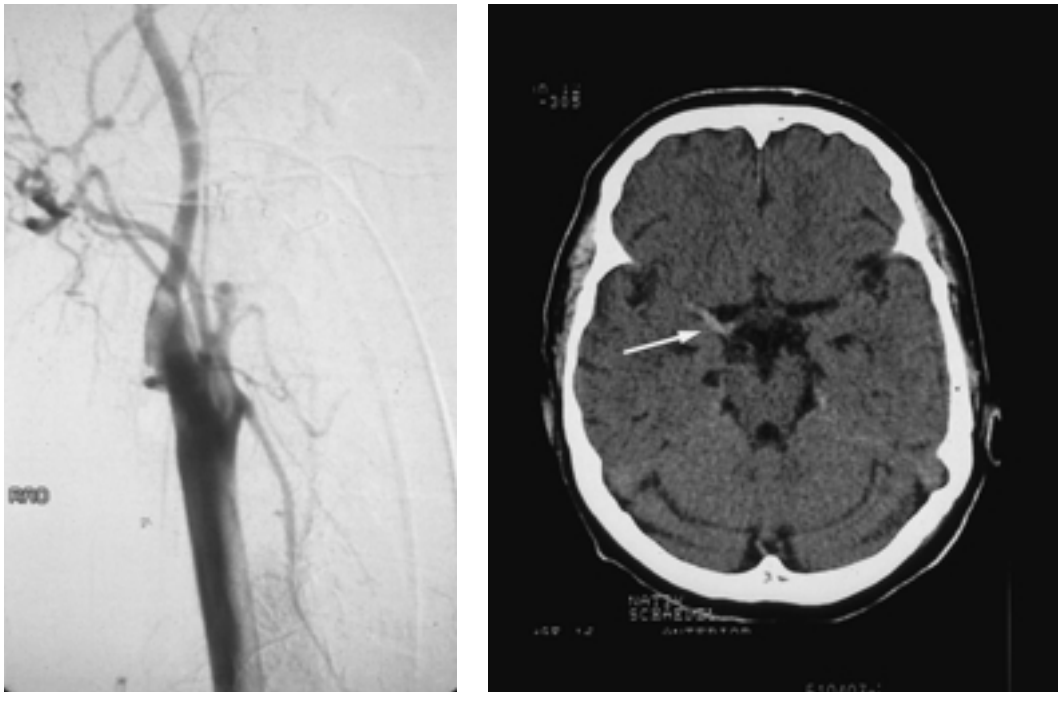
Nonfatal complications occurred in nine patients (8.0%). Four patients required operative revision of a neck hematoma, and CPR was necessary for one patient for intraoperative asystole; two patients were treated for pneumonia, one for pulmonary edema and one for UGI bleed.

FATAL STROKES

An 89-year-old patient presented with postprandial attacks of dizziness, presyncope, and bilateral high-grade internal carotid stenosis. He underwent an uneventful right carotid eversion endarterectomy but developed right arm weakness on postoperative day 1. His postoperative angiogram showed no technical problems, and his CT scan failed to show any fresh ischemic lesions. He was therefore operated on urgently, correcting a high-grade stenosis of his left internal carotid artery. Postoperatively his right arm paresis resolved, but 2 days later he developed a progressive right hemiparesis, aphasia, and loss of consciousness. A CT scan showed a large left frontal cerebral hemorrhage to which he succumbed 2 days later.

The second patient was an 80-year-old man who was operated 24 hours after the onset of slurred speech, confusion, and weakness of his left arm. He had suffered an ipsilateral stroke 3 years previously. His preoperative CT failed to show a fresh ischemic focus but showed signs of advanced diffuse cerebral vasculopathy. His angiogram demonstrated a high-grade preocclusive stenosis of the left internal carotid artery (ICA) with a high-grade but less severe ICA stenosis on the right. He underwent an uneventful left eversion endarterectomy but developed a dense left hemiplegia with progressive clouding of consciousness on postoperative day 1 to which he succumbed on postoperative day 24. His postoperative CT documented multiple fresh ischemic right hemispheric lesions.

The third patient was a 46-year-old male who presented with an acute embolic occlusion of his right carotid bifurcation due to a cardiac embolus which could be operated on within 4 hours after onset of his left hemiplegia.



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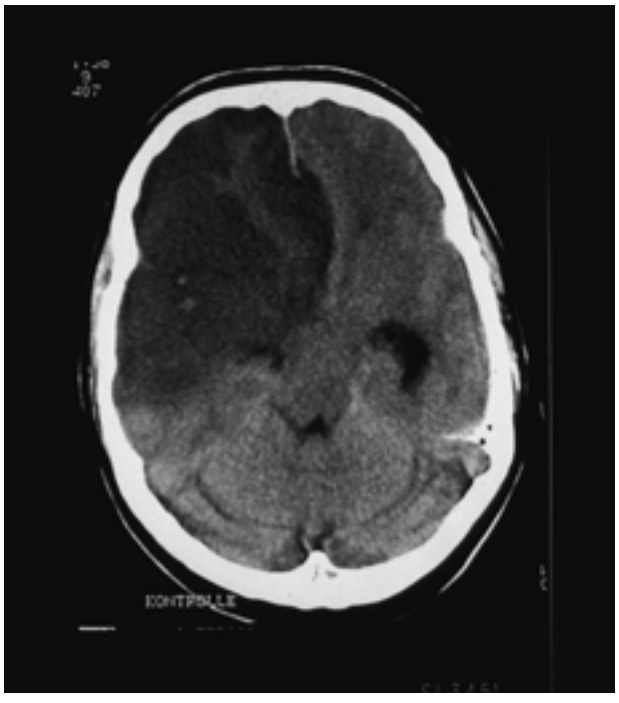

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Fig. 2 Forty-six-year-old male with subtotal acute embolic right internal carotid artery (ICA) occlusion. **(A)** DSA demonstrates fresh embolus in right carotid bulb and ICA. **(B)** Early cranial CT (without contrast) shows dense right middle cerebral artery (MCA) (arrow) and discrete signs of early edema in MCA territory. **(C)** Postoperative day 1 cranial CT with massive MCA territory edema and midline shift.

He had remained conscious and oriented preoperatively. Native CT scan had documented early signs of a middle and anterior cerebral artery territory insult with discrete edema. Transcranial Doppler failed to demonstrate a patent right middle cerebral artery. Angiography documented embolic occlusion of the carotid bifurcation with some evidence of embolic material at the level of the carotid siphon. Postoperatively he suffered an ipsilateral stroke with massive edema documented on repeat CT to which he succumbed 2 days postop (Fig. 2).

FOLLOW-UP

Neurologic outcome at follow-up is delineated in Table 2. Functional neurologic impairment was minimal or none according to the modified Rankin scale (0/1) in 74.1% of patients, 18.7% were able to ambulate with minor or moderate neurologic deficit (Rankin 2/3), 0.9% had a moderate deficit and required assistance with walking (Rankin 4), 0.9% were severely disabled and nonambulatory (Rankin 5), and 5.4% had died within 30 days after the intervention (Rankin 6).

During follow-up 10 patients died (Table 5), one of them secondary to a contralateral stroke. There was no ipsilateral stroke. One patient on coumadin died due to an intracranial hemorrhage after suffering minor head trauma. One- and 2.5-year stroke-free survival rates (all strokes any hemisphere) according to life-table analysis were 87 and 82%, respectively (Fig. 3).

DISCUSSION

Surgical repair of carotid lesions in patients with a fresh ischemic deficit has long been regarded as contraindicated. This opinion was based on historic series reporting unfavorable mortality and neurologic outcome after early surgical intervention.[4–7] On review many of these studies reported on small series of patients with profound deficits and unclear cerebral morphology, as CT evaluation was not yet available. The only randomized study quoted most frequently in this context, the Joint Study of Extracranial Arterial Occlu-

Table 5 Cause of Death During Follow-Up

Cardiac	2	Stroke (ipsilateral)	0
Suicide	2	Stroke (contralateral)	1
Renal failure	1	Intracranial hemorrhage	1
Malignancy	1	Unclear	1
Sepsis	1		

Median 10 months

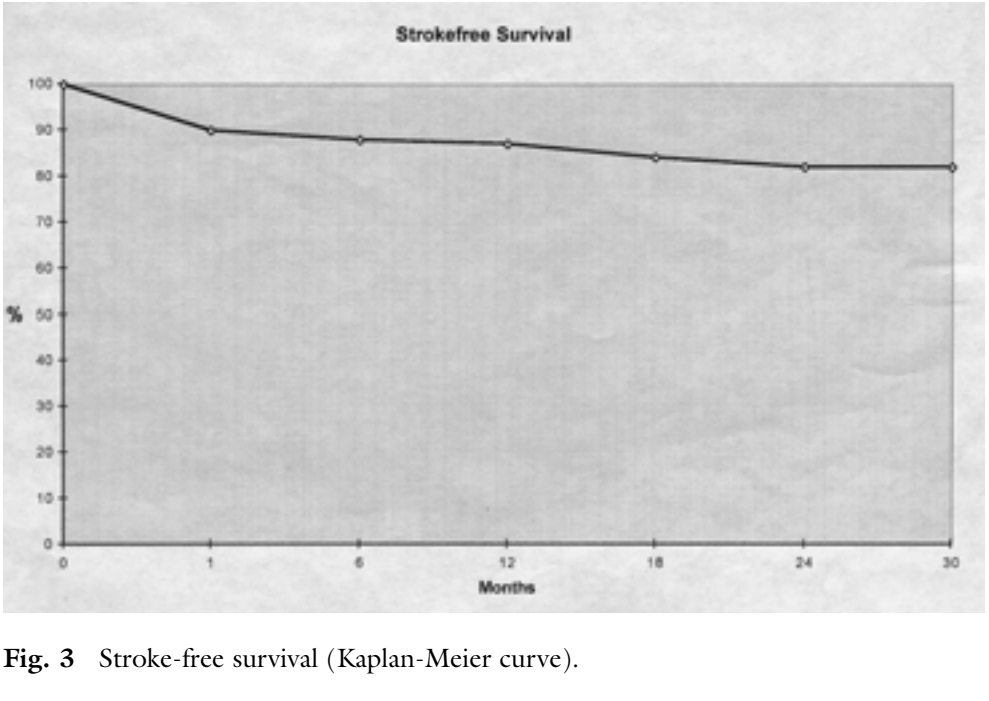

Fig. 3 Stroke-free survival (Kaplan-Meier curve).

sion[4,5] compared 187 patients with stroke and mental status changes raging from stupor to frank coma treated medically to 50 patients with similar deficits treated within 2 weeks of their neurologic deficit. Mortality and neurologic improvement in the surgical group were 42 and 34%, respectively, versus 20 and 53% in the medical group. A large part of these patients had severe deficits and included impairment of mental status. In contrast, among 18 patients operated after an interval of more than 2 weeks, a mortality of 17% was reported. In a more contemporary report Giordano et al. describe a perioperative stroke in 5 (18.5%) of 27 patients who underwent surgery within 5 weeks after an ischemic stroke versus 0% in 22 patients who were operated after more than 5 weeks. The level of preoperative neurologic deficit remained unclear however, and CT findings were not recorded or performed in all patients. These observations resulted in the somewhat arbitrary recommendation of a minimum waiting period of 5 to 6 weeks, which has been widely respected for several decades.

On the other hand, several reports have since documented that even good risk patients—with minor initial deficits—incur a significant risk of recurrent stroke within this waiting period. In the NASCET trial 4.9% of 103 patients with stroke and severe carotid stenosis randomized to medical treatment experienced a recurrent ipsilateral stroke within 30 days after entering the trial.[11] Sacco et al.[12] reported a 14% recurrent stroke rate within 2 years in 1273 patients with acute cerebral infarction, 30% of which occurred in the first 30

days. Dosick et al.[10] observed a 9.8% recurrence of neurologic events in 74 patients within 4 to 6 weeks after an ischemic stroke. In the ECST trial 71 patients randomized to medical care suffered an ipsilateral ischemic stroke, 24% of which were observed within 4 months of randomization.[2]

Several authors have since reported favorable results with a more aggressive approach to selected patients with recent neurologic deficit reporting combined stroke/mortality rates between 0.9 and 7.4%.[10,11,14–19]

Although selection criteria have varied individually, several general principles are beginning to emerge.

A preoperative assessment of cerebral morphology by CT is mandatory to exclude the approximately 15% of patients with primary hemorrhagic infarct or 5% with tumor as a priori contraindications to carotid endarterectomy.[20] A significant amount of cerebral edema or a large area of fresh ischemia is felt to increase the risk of postoperative reperfusion injury and secondary hemorrhage. These guidelines were not followed in early surgical approaches to patients with acute stroke and severe carotid stenosis or occlusion with the known catastrophic results. Accordingly, in modern series these high-risk patients with large defects generally have been excluded.[10,11,16]

The value of sequential CT scans however remains unclear. In our series as well as others the presence or absence of a new ipsilateral ischemic defect on CT did not appreciably influence outcome (Table 6).[11,18,19] One of the limitations of this study is that due to referral patterns we are unable to report the outcome of patients who failed the morphological CT criteria and were excluded, which might have helped to clarify this issue.

The same applies to the role of contrast enhancement. Although contrast enhancement may carry a worse prognosis, it varies over time and with the technique of administration.[21–23] It is rarely seen within the first week but may be observed in 60 to 70% of patients by 2 to 4 weeks after the acute event.[23,24] It is also seen more commonly with larger infarcts and infarcts with mass effect, possibly accounting for the observed poor prognosis of enhancing lesions.[22] Possibly patients with stroke and no ischemic lesions on CT are a favorable selection for early surgery. Dosick et al.[10] reported one stroke (0.9%) and no death in 110 patients operated within 14 days of a nondisabling stroke who had negative CT scans on days 1 and 5.

A preoperative assessment of MCA patency and collateralization in patients with acute or subacute occlusion of the ICA is necessary to exclude from surgery the unfortunate patient with occlusion of both vessels where carotid desobliteration will likely be inadequate to salvage neurologic function. Judging by our experience the patient with a "hyperdense MCA sign" (see Fig. 2B) on cerebral CT will likely perform poorly after carotid recanalization, even when performed within several hours of occlusion. This is in line with observations of Bone et al.,[25] who observed 7 cerebral infarcts in 32 patients who underwent surgical revascularization within hours of stroke resulting from acute carotid occlusion. Three of these infarcts became hemorrhagic

and were also found to have middle cerebral artery in addition to carotid occlusion.

The appropriate time interval for surgery after acute stroke, although apparently of lesser importance than initially believed,[19] remains poorly defined. Giordano[9] reported an 18.5% perioperative stroke rate in 27 patients operated within 5 weeks after a stroke. It is noteworthy however, that 9 of these 27 patients were operated within 0 to 14 days after the acute event, all without neurologic sequelae. Furthermore, at least one of these perioperative strokes was due to early carotid thrombosis as assessed by OPG.

Paty et al.[19] reported 3 strokes (2%) in 149 patients operated within 4 weeks after stroke. Of these 1 occurred in 31 patients operated in the first week (3.2%), 1 in 21 patients operated in the 3rd week (4.8%), and 1 in 36 patients operated in the 5th week (2.8%) after stroke. In our series the stroke rate 1 to 4 weeks after the neurologic event varied between 5.5 and 9.1% without appreciable statistical significance (Table 6).

The severity of initial neurologic deficit and level of consciousness to a large degree determines outcome.[26,27] Therefore, it is prudent to exclude patients with mental status changes or severe hemiparesis from surgical intervention.[11,14,16,19] That severity of neurologic deficit as classified by the modified Rankin score did not significantly influence outcome in our series (Table 6), does not refute this experience but underscores the importance of patient selection, excluding from immediate surgery patients with severely debilitating deficits.

Patients with recent neurologic event and unstable or progressive deficit belong to the highest risk group.[28] In our series 3 of 12 patients (25%) with this constellation suffered postoperative neurologic sequelae (Table 6). Early surgery in these patients needs to be approached with greatest caution. However, therapeutic nihilism would be the wrong conclusion as conservative treatment carries an even worse prognosis. Millikan[29] reported on 204 patients with progressive stroke treated conservatively. After 2 weeks only 12% had recovered, 69% were hemiparetic, 5% were monoparetic, and 14% had died. Toni et al.[30] found a 36% mortality in patients with progressing neurologic deficit due to ischemic stroke treated medically. Mentzer et al.[31] reported on 55 patients with crescendo TIAs or progressive neurologic deficit who were treated either conservatively ($n = 31$) or surgically ($n = 24$). Among the surgical group 79.8% had a complete or near complete recovery, whereas this was seen in only 28.6% of the nonoperative group. Mortality was 4.2 versus 14.7%, respectively.

The influence of contralateral high-grade stenosis or occlusion on outcome in patients with acute stroke is unclear. Although there seemed to be a trend toward a worse outcome for patients with contralateral occlusion or greater than 80% stenosis, this was not statistically significant (Table 6) and contrasts with our previously observed combined stroke/mortality rate of only 2.2% in 554 patients with contralateral occlusion.[32] Although we have

Table 6 Factors Influencing Outcome

	Postoperative stroke/PRIND			
No stroke	***n***	***n***	**%**	***p***
Ipsilateral stroke on preoperative CT*,#				
Yes	44	3	6.5	
No	60	5	7.7	n.s.
Time interval (days)*				
0–7	51	3	5.5	
8–14	30	3	9.1	
15–21	25	2	9.1	
22–30	9	0	7.4	n.s.
Neurologic deficit##				
Asymptomatic	20	0	0	
Improving	50	0	0	
Stable	18	3	14.3	
Progressing	9	3	25	$p < 0.005$
Preoperative ranking classification				
0	20	0	0	
1	14	1	6.7	
2	31	3	8.8	
3	39	2	4.9	
4	2	0	0	n.s.
Contralateral stenosis (%)				
<60	68	3	4.2	
60–80	11	0	0	
≥80	18	2	10.0	
Occluded	9	1	10.0	n.s.
Correction contralateral ICA stenosis*				
Yes	19	3	13.6	
No	96	5	5.0	n.s.

*Includes data from 11 patients with repair of both carotid arteries during same hospitalization (122 procedures in 112 patients).
#CT data from 9 patients (12 procedures) unavailable.
##9 patients with unknown status.

generally avoided the use of shunts a more liberal use may be of potential benefit in this highly selected group of patients.

In patients presenting with significant bilateral carotid lesions the symptomatic lesion should be corrected first even in the presence of a more high-grade lesion on the contralateral, asymptomatic side. Both patients in our series who first underwent surgical correction of the asymptomatic contralateral carotid stenosis suffered neurologic sequelae after the second operation.

CONCLUSION

This study demonstrates that correction of symptomatic carotid lesions in carefully selected patients may be performed with acceptable morbidity and mortality within less than 4 weeks after a nondisabling stroke. The net benefit of early compared with delayed surgery however can only be determined in prospective randomized studies, two of which are currently under way in Europe.[16,20]

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Expert Commentary

Richard M. Green, M.D.

One of the most difficult decisions a vascular surgeon faces is the proper timing of carotid endarterectomy after a stroke. Conventional teaching mandates waiting four to six weeks based on data that suggest early operation leads to intracranial hemorrhage and death. Our conundrum is that there are also data that show that delay is associated with an unacceptably high incidence of additional neurologic events (4.9% of the 103 medically treated patients with stroke had an ipsilateral stroke within 30 days after entry into the NASCET trial). Most of us have seen both complications but many of us have seen gratifying results with early endarterectomy. Selecting patients for each approach is therefore the critical element in the management of these high-risk patients. The authors present a vast experience over a short period of time that provides many useful guidelines when faced with the difficult choice of whether to operate or wait or decline.

I think it is helpful to consider three elements when making the decision about operative timing: the status of the patient, the status of the brain, and the status of the carotid artery. Most would agree that certain findings preclude an early operation—a dense hemiplegia or altered state of consciousness, a large defect on CT scan, and an occluded or minimally stenotic carotid artery. On the other hand, the patient with a nondisabling stroke, a negative CT scan or one with a small defect, and a critical ipsilateral carotid stenosis will likely benefit from early operation.

Our policy has been to wait for maximum neurologic improvement while maintaining the patient on heparin anticoagulation and then to proceed with endarterectomy usually within the first two weeks following the event providing the carotid lesion is critical. This is the only clinical situation that mandates a preoperative angiogram in our practice. We certainly agree with the authors that patients with tandem occlusions of the MCA do poorly after early endarterectomy. Our treatment algorithm is flexible, as we will delay operation if neurologic recovery takes longer than two weeks or the carotid stenosis is moderate rather than severe. We do anticoagulate these patients with coumadin until their operation.

The role of the CT scan in determining the timing of operation is an unsettled one. The NASCET data show clearly that a positive preoperative CT scan is not a predictor of an adverse outcome after early endarterectomy. Furthermore, there were no instances of intracranial bleeding in the subset of patients operated upon early after nondisabling strokes. Some believe that

R.M.G., Professor of Surgery, Chair, Division of Vascular Surgery, University of Rochester Medical Center, Rochester, NY.
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contrast-enhanced CT scans may offer some value in predicting which patient is likely to have a postoperative intracranial hemorrhage. Data are lacking to prove this hypothesis but it makes little intuitive sense, as early strokes have a high incidence of contrast enhancement and this and other studies clearly show that the incidence of bleeding is low after early operation.

I agree with the treatment priorities as outlined by the authors and recommend serious consideration of early operation after a nondisabling stroke in the patient with a critical carotid stenosis. Even though a rare patient may develop an intracranial hemorrhage, an unnecessary four to six week postponement of operation is a more dangerous course to recommend.

The Last Word

Schunn et al.

It is gratifying to see that scientific evidence is able to change therapeutic prejudice, and we are reassured by Dr. Green's supportive remarks of our aggressive approach towards patients with severe carotid disease and recent stroke. In contrast to his practice of mandatory angiography for patients with fresh stroke, however, we reserve preoperative angiography for the rare patient with witnessed acute internal carotid artery occlusion.