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Immediate reexploration for the perioperative neurologic event after carotid endarterectomy: Is it worthwhile?

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Purpose: **When managing a new neurologic deficit after carotid endarterectomy (CEA), the surgeon is often preoccupied with determining the cause of the problem, requesting diagnostics tests, and deciding whether the patient should be surgically reexplored. The goal of this study was to analyze a series of perioperative neurologic events and to determine if careful analysis of their timing and mechanisms can predict which cases are likely to improve with reoperation.**

Methods: **A review of 2024 CEAs performed from 1985 to 1997 revealed 38 patients who manifested a neurologic deficit in the perioperative period (1.9%). These cases form the focus of this analysis.**

Results: **The causes of the events included intraoperative clamping ischemia in 5 patients (13.2%); thromboembolic events in 24 (63.2%); intracerebral hemorrhage in 5 (13.2%); and deficits unrelated to the operated artery in 4 (10.5%). Neurologic events manifesting in the first 24 hours after surgery were significantly more likely to be caused by thromboembolic events than by other causes of stroke (88.0% vs 12.0%, $P < .002$); deficits manifesting after the first 24 hours were significantly more likely to be related to other causes. Of 25 deficits manifesting in the first 24 hours after surgery, 18 underwent immediate surgical reexploration. Intraluminal thrombus was noted in 15 of the 18 reexplorations (83.3%); any technical defects were corrected. After the 18 reexplorations, in 12 cases there was either complete resolution of or significant improvement in the neurologic deficit that had been present (66.7%).**

Conclusions: **Careful analysis of the timing and presentation of perioperative neurologic events after CEA can predict which cases are likely to improve with reoperation. Neurologic deficits that present during the first 24 hours after CEA are likely to be related to intraluminal thrombus formation and embolization. Unless another etiology for stroke has clearly been established, we think immediate reexploration of the artery without other confirmatory tests is mandatory to remove the embolic source and correct any technical problems. This will likely improve the neurologic outcome in these patients, because an uncorrected situation would lead to continued embolization and compromise. (J Vasc Surg 2000;32:1062-70.)**

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The management of the perioperative neurologic event (PNE) after carotid endarterectomy (CEA) is an area where many controversies remain. Disagreement remains regarding the severity of the deficit that requires reoperation, the role of noninvasive testing and angiography, and the window of opportunity when reoperation should be accomplished. Although many surgeons recommend reexploration, there are few outcome data to support this opinion.

In previous work at our institution, investigators have looked at the causes of perioperative strokes

Table I. Comparison of patient demographics between patients who had PNE and those who did not

	PNE (n = 38)	*No PNE (n = 1996)*	P *value*
Coronary disease	46.5%	45.0%	NS
Hypertension	69.8%	56.4%	NS
Diabetes	25.0%	22.1%	NS
Smoker	47.6%	35.4%	NS
Sex (% male)	63.8%	59.9%	NS
Mean age	68.7 y	69.2 y	NS

NS, Not significant; *PNE,* perioperative neurologic event.

Table II. Comparison of indications for surgery between patients who had a PNE and those who did not

	PNE	*No PNE*	P *value*
TIA	22.7%	42.3%	.009
CVA	38.6%	23.0%	< .02
Asymptomatic	27.3%	34.4%	NS
Crescendo TIA or stroke-in-evolution	11.4%	0.4%	< .001

CVA, Cerebrovascular accident; *NS,* not significant; *PNE,* perioperative neurologic event; *TIA,* transient ischemic attack.

after CEA.[1] The most common cause is technical error resulting in postoperative thrombosis, embolization, or both. Presumably then, immediate correction through reexploration will afford the opportunity to correct errors and reduce the chance of further ischemia. The goal of this study was to analyze a series of PNEs and to determine if analysis of their timing and mechanisms can predict which cases are likely to improve with reoperation.

PATIENTS AND METHODS

A review of a prospectively compiled database of CEAs performed at our institution was conducted. All primary CEAs performed between 1985 and 1997 were included (N = 2024). A PNE was defined as a new or worsening deficit, transient or permanent, that manifested within 30 days after surgery. A new deficit occurred in 38 patients (1.9%). The course, management, and outcome of these cases were investigated through the database and charts. Determination of the etiology of the PNE was made on the basis of clinical findings, the findings of reexploration, and the results of any other test used to evaluate the patient, including duplex ultrasound scan, computerized tomographic scanning, magnetic resonance imaging, and arteriography.

Standard technique for CEA at our institution includes a preference for regional anesthesia with selective shunting, routine shunting for patients under general anesthesia, and patch angioplasty. We do not routinely use intraoperative imaging techniques.

Statistical analysis was performed with the software package SPSS (SPSS, Inc, Chicago, Ill). A result was considered statistically significant with a P value less than .05. Comparisons were performed with the χ^2 test or the Fisher exact test, where appropriate.

Table III. Comparison of additional surgical factors between patients who had a PNE and those who did not

	PNE	*No PNE*	P *value*
Contralateral occlusion	10.6%	15.3%	NS
Contralateral stenosis > 50%	70.2%	74.6%	NS
Regional anesthesia	68.4%	80.9%	< .01
Tolerated clamping	42.1%	87.6%	< .01

NS, Not significant; *PNE,* perioperative neurologic event.

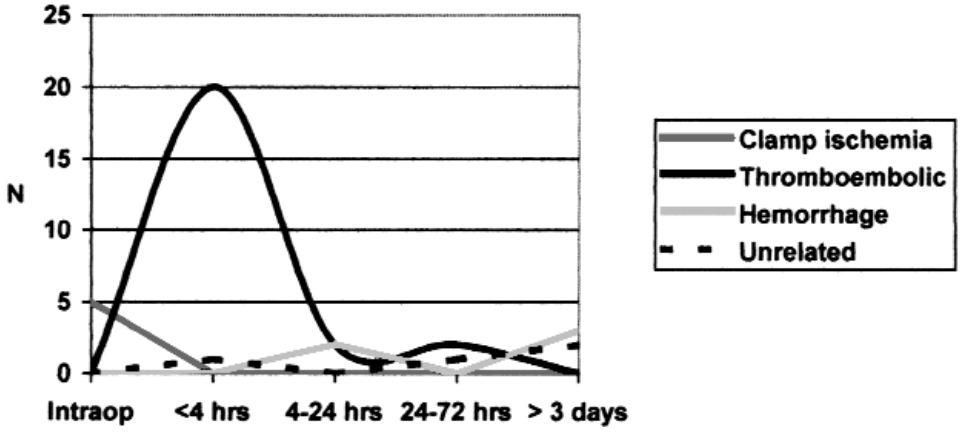
RESULTS

Comparison between PNE and non-PNE patients

Patient demographics. Patient demographics were compared between patients who sustained a PNE and those who did not (Table I). No statistically significant differences were found.

Indications for surgery. The indications for surgery were compared between patients who sustained a PNE and those who did not (Table II). PNE patients were significantly more likely to have had a preoperative stroke (38.6% vs 23.0%, $P < .02$). PNE patients were significantly less likely to have had a transient ischemic attack (TIA) or amaurosis fugax (22.7% vs 42.3%, $P = .009$). There was a significantly higher proportion of PNE patients who underwent emergency CEA for either crescendo TIAs or a stroke-in-evolution (11.4% vs 0.4%, $P < .001$).

Additional comparison and analysis of patient and surgical factors. There were no significant differences in the percentages of patients in the two groups who had contralateral carotid occlusion or significant contralateral stenosis (> 50%) (Table III). Significantly fewer PNE patients completed surgery while under regional anesthesia (68.4% vs 80.9%, $P < .01$). PNE patients were significantly less likely to tolerate clamping of the carotid artery under regional



Relationship between timing and etiology of the PNE. *Intraop,* Intraoperative.

anesthesia (42.1% vs 87.6%, $P < .01$). Therefore, among PNE patients, shunts were used in 42.1% of cases where patients were under regional anesthesia.

Of the 38 PNE cases, patch angioplasty was performed with saphenous vein in 10 patients (26.3%), with Dacron in 23 (60.5%), with polytetrafluoroethylene in 2 (5.3%), and with jugular vein in 1 (2.6%). Two cases involved interposition grafting, one with vein and one with polytetrafluoroethylene. There was no correlation between the type of patch and the occurrence of postoperative thrombosis or platelet aggregation.

Two of the PNE cases (5.3%) involved interposition grafting for the primary operation. This was due to the nature of disease found by the surgeon. In one of these cases, reexploration revealed thrombosis of the bypass graft. In the other case, the patient underwent postoperative duplex scanning and arteriography as evaluation for the neurologic deficit. On the basis of these test results, which revealed a patent graft without any abnormalities, the patient was not reexplored.

Two PNE cases involved thrombectomy of an acutely occluded internal carotid artery (5.3%). One of these cases involved a patient who was experiencing crescendo TIAs. The other case involved a patient with a preocclusive (99%) stenosis and a recent ipsilateral infarct. During surgery, the patient became hemiparetic with carotid clamping. Function did not return despite the insertion of a shunt. Intraoperative arteriography revealed thrombosis of the distal internal carotid artery.

Detailed analysis of PNE cases

Manifestations of the PNE. The manifestations of the PNE included hemiparesis, hemiplegia, or aphasia in 31 cases (81.6%); obtundation in 1 case (2.6%); seizure in 3 cases (7.9%); headache in 1 case (2.6%); and global cerebral ischemia in 2 cases (5.3%). Obtundation, seizures, and headaches were considered manifestations of a PNE when they were the only symptoms that led to a computerized tomographic or magnetic resonance imaging diagnosis of either intracerebral hemorrhage or new infarct. Global cerebral ischemia was defined as bilateral waxing and waning motor or sensory deficits along with depressed mental status.

A deficit related to the contralateral hemisphere occurred in five cases (13.2%). A bilateral stroke occurred in one case; the underlying cause appeared to be related to bilateral hemispheric ischemia during clamping, despite the use of a shunt.

Timing of the PNE. In five cases the PNE manifested intraoperatively in patients who were under regional anesthesia (13.2%). The remaining cases occurred at the following intervals in the postoperative period: less than 4 hours, 21 cases (55.3%); from 4 to 24 hours, 4 cases (10.5%); from 24 to 72 hours, 3 cases (7.9%); and more than 3 days postoperatively, 5 cases (13.2%). Deficits manifesting in the first 24 hours after surgery have been designated as *early PNEs*, whereas deficits manifesting later in the perioperative period have been designated as *late PNEs.*

Etiology and management of the PNE. We determined that PNEs were the result of clamping ischemia in 5 cases (13.2%); thromboembolization in 24 cases (63.2%); intracerebral hemorrhage in 5 cases (13.2%); and events not directly related to the operated artery in 4 cases (10.5%).

PNEs due to clamping ischemia. The five cases that manifested intraoperatively were diagnosed with the

Table IV. Reasons for deferring immediate reexploration in seven early PNE cases (< 24 hours)

Reason	*N*	*Final determined etiology of PNE*	*Decision correct?*
Intracerebral hemorrhage or reperfusion injury suspected/confirmed	3		
Lethargy without focal deficit	1	Hemorrhage	Yes
Severe headache	1	Hemorrhage	Yes
S/P urgent ICA thrombectomy	1	?Thromboembolic	No
Contralateral hemispheric stroke	2	Thromboembolic	No
Hypoxia/premature extubation	1	Unrelated	Yes
Preoperative deficit that became worse postoperatively (uncertainty in the immediate postoperative period whether this resulted from general anesthesia)	1	Thromboembolic	No

ICA, Internal carotid artery; *PNE,* perioperative neurologic event; *S/P,* status, post.

patient under regional anesthesia, and all were thought to be the result of clamping ischemia. These cases were managed intraoperatively with routine procedures, including the insertion of a shunt. Unfortunately, these were patients whose deficit did not improve with shunting. Of note, two of these cases of intraoperative ischemia were being performed emergently on neurologically unstable patients. Two of the remaining cases of intraoperative ischemia had PNEs related to the contralateral hemisphere; one of these patients had a contralateral occlusion.

Although it could be argued that these cases were due to intraoperative thromboembolism, they were thought to be the result of clamping ischemia, according to the observation of the neurologic status of the patients under regional anesthesia. All of these patients demonstrated deficits with test clamping of the artery, which then reversed when the artery was unclamped. Shunts were inserted in the routine fashion and were found to be functioning well. There were no cases of clamp or shunt injury. However, the neurologic status of these patients deteriorated despite the restoration of flow with a shunt. As detailed in the previous paragraph, two of these cases of intraoperative ischemia were performed on neurologically unstable patients. Two additional patients manifested strokes related to the contralateral hemisphere. It is presumed that these patients had a neurologic situation so tenuous that even a functioning shunt was unable to adequately perfuse "watershed" areas of the brain.

PNEs due to thromboembolic causes. Most PNEs were due to thromboembolic events (63.2%). These were diagnosed either on the basis of reexploration or on the basis of tests that revealed a technical problem at the endarterectomy site or evidence of embolization in the intracerebral circulation.

Of the 24 PNEs now thought to be related to thromboembolic causes, 18 underwent reexploration. The remaining eight cases underwent other diagnostic testing. Although the operating surgeon at the time thought that reexploration was unwarranted, in reviewing these cases we have determined that thromboembolism was the likely cause of the event. Of the 24 PNEs now thought to be related to a thromboembolic event, 22 (91.7%) occurred within the first 24 hours after surgery.

Table V. Relationship between etiology and timing of the PNEs occurring in the postoperative period

	Thromboembolic	*Other cause*	*Total*
Early PNE (< 24 h)	22 (88%)	3 (12%)	25
Late (> 24 h)	2 (25%)	6 (75%)	8
Total	24	9	33

PNEs caused by intracerebral hemorrhage. PNEs caused by cerebral hemorrhage most often manifested as obtundation, seizure, or headache. They occurred at varying times in the postoperative period: two within 24 hours after surgery, and three later than 72 hours (Figure). None of these patients underwent reexploration.

PNEs unrelated to the operated artery. Cases deemed to be unrelated to the operated artery included the following: 1 case of early extubation and global hypoxia, 2 cases of documented cardioarterial embolization, and 1 case of a late contralateral stroke associated with contralateral occlusion.

Management of early versus late PNEs. Twenty-five cases (65.8%) occurred within 24 hours after surgery (early PNE). These were patients who arrived in the recovery room neurologically intact and subsequently had a new deficit. Unless another cause was clearly established, these were generally assumed to be due to a thromboembolic event. Of these early PNEs, immediate reexploration was performed in 18 patients. Reexplorations were performed with patients under general anesthesia, with rapid rehep-

arinization, and with the insertion of a shunt. In the remaining seven early PNE cases, management was based on the judgment of the surgeon, and various other diagnostic tests were performed. The reasons for deferring reexploration are presented in Table IV.

The remaining eight cases (21.1%) occurred more than 24 hours after surgery (late PNE). None of these cases underwent reexploration. Individualized management was again performed according to the specific scenario and results of other tests.

Results of surgical reexploration. Of 18 reexplorations, 15 (83.3%) had positive intraoperative findings. These included the presence of thrombus or platelet aggregates at the endarterectomy site, with or without an obvious technical defect. The technical defects discovered at reexploration included irregularities at the suture lines, at the proximal or distal extent of the endarterectomy site, or at the site of a plication. Most commonly, platelet aggregates rather than frank thrombus were found. Occasionally, platelets were found to be carpeting the entire endarterectomy site without any obvious technical irregularity. The repair was completely thrombosed in only two cases; in the others the artery remained at least partially patent.

In the remaining three cases the endarterectomy site was found to be without any technical defects or obvious thromboembolic source. Of these three patients, one was clinically improved after reexploration, and the other two were had no change in their neurologic status.

Procedures performed during the 18 reexplorations included simple thrombectomy, 7 cases (38.9%); redo patch angioplasty with Dacron, 5 cases (27.8%); redo patch angioplasty with saphenous vein, 4 cases (22.2%); and carotid bypass grafting, 2 cases (11.1%).

Outcome of surgical reexploration. After surgical reexploration, the clinical assessments of the patients were resolution of the preexploration deficit, 3 cases (16.7%); significant improvement in the deficit, 9 cases (50.0%); no change in the deficit, 5 cases (27.8%); and a severe stroke resulting in death in 1 case (5.6%). Overall, 66.7% of patients who underwent reexploration for an early PNE had either resolution or significant improvement in their neurologic status. Reexploration did not worsen any patient. No subsequent intracerebral hemorrhages occurred in patients who underwent one reexploration.

Outcome of patients not reexplored. Patients who did not undergo reexploration were composed of three groups: those who had intraoperative strokes (5 cases), early PNEs (7 cases), and late PNEs (8 cases). Of the cases in which intraoperative strokes occurred, two patients died and three remained severely hemiplegic, aphasic, or both. Five patients with intracerebral hemorrhage were not reexplored: two occurred early, and three occurred after 72 hours. Two of the patients with intracerebral hemorrhage died, two significantly improved, and one remained alive with severe deficits. Four patients with PNEs unrelated to the operated artery were not reexplored; one occurred early, and three occurred late. One of these patients died and the remaining three remained hemiplegic, aphasic, or both. The remaining six cases not reexplored were finally thought to result from thromboembolic events. Four of these occurred early, and two occurred late. None of these patients died, and five of six gradually improved but had mild or moderate deficits.

Relationship of etiology and timing of the PNE. Intraoperative strokes were all presumed to be a result of clamping ischemia and were therefore excluded from the following analysis. PNEs manifesting in the first 24 hours after surgery were significantly more likely to be caused by thromboembolic events rather than by other causes (88.0% vs 12.0%, $P = .002$, Fisher exact test). Similarly, PNEs occurring after the first 24 hours were significantly more likely to result from causes other than thromboembolism. Likewise, thromboembolic strokes were significantly more likely to occur during the first 24 hours after surgery than at a later period of time (91.7% vs 8.3%, $P = .002$, Table V, Figure).

DISCUSSION

The approach to the perioperative stroke after CEA has been a matter of debate for several decades. Since that time, many surgeons have continued to advocate immediate exploration for the post-CEA neurologic deficit.[2-4] However, other surgeons have placed qualifications on this approach.[5-13] Several have argued that only patients with suspected thrombosis of the artery should be reexplored.[6-8] The rationale for this approach is that only patients with actual thrombosis of the artery will benefit from surgical correction. Other authors have mentioned the severity of the deficit as being the determining factor. These authors state that a postoperative "TIA" may be observed.[9-11] Still others have advocated selective reexploration according to whether the patient awakens from general anesthesia with a deficit or has a deficit after a neurologically intact "lucent" period.[12-14] We think that the patient who awakens from anesthesia with a new deficit is unlikely to benefit from reexploration. Additionally, the window of opportunity for reexploration and the correction of any technical problems has not been established.

Finally, it can be argued that even reexploration and correction of any technical defects do nothing to correct embolization once it has already occurred. Comerota and Eze[15] have reported successful management with reexploration and thrombolytic agents. Our experience with this management has been limited to two recent cases with poor outcomes.

The recommendations of many authors are based on albeit extensive experience without the presence of extensive supportive data. Because of the low incidence of perioperative stroke after CEA, there are no large series in which the results of post-CEA reexploration are examined. However, if patients with perioperative stroke can be salvaged with reoperation, this is a critical area to explore and one that may reduce the final stroke rate after CEA. Unfortunately, this is not an area in which a randomized, prospective trial is feasible.

One of the unique advantages of performing CEA with patients under regional anesthesia is the ability to distinguish strokes caused by clamping ischemia during the intraoperative period from other etiologies. Analysis has consistently found that the most common causes of postoperative stroke are related to technical errors that result in postoperative thromboembolism.[1] It would appear intuitive that reexploration to correct such a problem would likely improve the neurologic outcome, because an uncorrected situation would lead to continued embolization and compromise.

In this series, we discovered that PNEs were more likely to occur in patients who had experienced a preoperative stroke, in those who underwent urgent surgery, and in those who received general anesthesia. At our institution patients who receive general anesthesia are often those in whom the surgeon would feel uncomfortable in performing the operation without a shunt. If a shunt is deemed necessary, observation under regional anesthesia is thought to be superfluous. Patients operated on while under general anesthesia are likely to be those who have sustained a recent infarct or are neurologically unstable. We think that patients who have sustained a preoperative completed stroke or are neurologically unstable likely have a watershed area of brain at risk and are more likely to clinically manifest a stroke from a given technical problem and thromboembolic event. Previous data from our institution support this conclusion.[1] The current series has revealed several useful pieces of information. Excluding intraoperative strokes, perioperative strokes are most likely to occur during the first 24 hours after surgery (65.8%), and most will occur in the first 4 hours. PNEs manifesting in the first 24 hours are significantly more likely to be caused by thromboembolic events rather than other causes, and these are presumably related to technical error. These "correctable" lesions may continue to embolize or cause complete thrombosis if not corrected. If the artery has already completely thrombosed, the patient will likely benefit from early restoration of flow in an expedient manner.

Fear of exacerbating an intracerebral hemorrhage has led to concern about recommending reexploration. Although in this series the numbers were small, it appears that intracerebral hemorrhages tend to occur later in the perioperative period and are more often manifested clinically by headache, seizure, or lethargy than hemiparesis or other focal deficits. Patients with strokes resulting from intracerebral hemorrhage or intraoperative cerebral ischemia appeared to have worse outcomes overall than those with thromboembolic strokes. Lessons can be learned from the patients who did not undergo immediate reexploration for an early PNE (Table IV). The most probable cause of stroke was thromboembolism in four of these seven cases. Two of these patients had contralateral strokes and were not reexplored on this basis; however, arteriography revealed embolic debris in the contralateral hemisphere, without an obvious technical problem or thromboembolic source at the endarterectomy site. It was presumed that these cases represented episodes of "crossover" embolization. Although no problem with the endarterectomy site was identified by arteriography in these instances, it was further hypothesized that there must have previously been a thrombotic source at the endarterectomy site that had subsequently embolized. A recent case of contralateral perioperative stroke at our institution (not included in this series) definitively demonstrated both an intraluminal defect at the ipsilateral endarterectomy site and crossover embolization to the contralateral hemisphere on arteriography. Therefore, thromboembolic events resulting from technical errors during CEA can be the cause of stroke related to the contralateral hemisphere. On the basis of the reevaluations performed in this review, four of the seven early PNE patients not reexplored actually may have benefitted from reexploration.

The goal of reexploration is to prevent further embolization and to restore normal flow to the area of ischemic injury. Of 18 reexplorations performed for an early neurologic deficit, 83.3% clearly showed signs of thrombus or platelet aggregation. Nearly 70% of patients reexplored had either complete resolution of or significant improvement in the deficit that had been present. No patient was worsened by reexploration. Therefore, the high probability that

an early PNE is due to thrombus formation suggests that immediate reexploration is mandatory and indicated even without confirmatory tests. Of course, the individualized judgment of the operating surgeons is paramount. Although patients with presumed thromboembolic events who were not reexplored had relatively good clinical outcomes, we do not think that a direct comparison with patients who were reexplored is possible. It is likely that the patients with more severe events underwent reexploration, whereas those with less severe deficits were more likely to have been managed expectantly.

The best form of treatment of perioperative stroke is prevention. We do not routinely use intraoperative imaging techniques. Instead, we have chosen to rely on extensive exposure of the internal carotid artery well above the level of the plaque, long arteriotomies, meticulous endarterectomy technique, and patch angioplasty to reduce technical errors.

CONCLUSIONS

Careful analysis of the timing and presentation of PNEs after CEA can predict which cases are likely to improve with reoperation. Neurologic deficits that present during the first 24 hours after CEA are likely to be related to intraluminal thrombus formation and embolization. Unless another cause for stroke has clearly been established, we think immediate reexploration of the artery without other confirmatory tests is mandatory to remove the embolic source and correct any technical problems. This will likely improve the neurologic outcome in these patients, because an uncorrected situation would lead to continued embolization and compromise.

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DISCUSSION

Dr Anthony J. Comerota (Philadelphia, Pa). Good morning, ladies and gentlemen, Dr O'Donnell, members, and guests. I congratulate Dr Riles and his colleagues for another important contribution in the management of patients with carotid disease. If one looks at the manuscript and then looks at the numbers, the 1.9% stroke rate is impressive. Excluding intracranial hemorrhage, late strokes due to cardioembolic sources, aortic valve replacement and so forth, which they included as a post-op neurologic deficit, their true stroke rate is approximately 1.1%, which was truly impressive.

They focus on these patients who manifested the early post-op neurologic deficit and their approach of immediate reexploration in correcting a technical defect by repair and patching, and carotid bypass was used in two patients. The authors present a strong argument for this approach and one that I would agree with wholeheartedly. In their manuscript, and a bit in this presentation, it is implied that this approach will improve the neurologic deficit in most patients. While this approach corrects a source for additional potential cerebral emboli, I would submit that it does not correct any ischemic neurologic event but rather

allows the natural history of that event to take its course without additional ischemic insult, unless, of course, the carotid was occluded and that was picked up and fixed.

I would point out that one third of their patients did not improve after reexploration. So I would submit that with all that is known about the treatment of acute ischemic stroke, we, as vascular surgeons, have an ideal opportunity to reverse ischemic operative neurologic deficits with the use of thrombolytic agents simply because we see them so early in their course. Of course, the key to treating these ischemic events is, indeed, that they are ischemic and not hemorrhagic; however, as Dr Riles so nicely explained, the intracranial hemorrhage patients present late and have a much different clinical scenario.

Tom, you mentioned briefly in the paper that two patients of yours were treated with intraoperative lytic therapy without a good outcome, and I was wondering if you could expand for us on those patients, the timing of the use of the lytic agents, the technique, the dose, and so forth.

I would strongly suggest that the approach in these patients be immediate reexploration, as Dr Riles has explained, but that we insert a Pruitt-Inahara shunt and infuse a plasminogen activator through the side infusion port. It's important to give enough of the lytic agent to make a difference. I have arbitrarily chosen a 1-hour infusion, have used 1 million units of urokinase in our first patient and 20 mg of recombinant tissue plasminogen activator in our last patient, diluted in a solution that's large enough to give over an hour, about 100 cc. Both of these patients had good outcomes.

On-table arteriography and selective intracerebral catheterization, of course, would be the ideal, assuming you can get good intracranial films, but few institutions have that capability in the OR and we certainly do not.

I have several questions for you, Tom.

Would you consider treating your next patient with intra-arterial lytic therapy in order to actively treat the embolic or thrombotic ischemic event?

You do not mention in your manuscript or in your presentation how you evaluate the technical adequacy of your carotid endarterectomy before the patient leaves the operating room. Do you use routine intraoperative carotid duplex? And if so, if the duplex was normal, would you change your approach if your patient had an early neurologic deficit? And if you do not use an intraoperative completion carotid duplex, would you consider starting that since you found 15 of 18 patients with a correctable problem at the time of reexploration?

You mention that you found platelet adherence in some of these patients, and did this represent a form of HIT? Did any of your patients experience a profound platelet drop at the time of their diagnosis of the ischemic event?

You answered a couple of my questions in your presentation, but I would also submit that I got the impression that the patients operated on under regional anesthesia seemed to have fewer neurologic events; but those are also the patients in whom you used selective shunting. In your series, all patients undergoing general anesthesia, I believe, had an obligate shunt. The aggregate literature strongly suggests that selective shunting reduces operative neurologic events, and do you think this may be a bias against general anesthesia?

Once again, I commend the authors for bringing this important information to our attention, and I thank the Society for the opportunity to discuss this paper.

Dr Thomas S. Riles. Thank you very much, Tony, you've asked some very important questions.

First about the thrombolytic agents. The two patients that had this type of treatment were actually taken to the neuroradiology suite for thrombolysis. One patient developed intracranial hemorrhage and became much worse; the other had no improvement at all. By the time the procedure was completed it was at least 4 hours after the initial event. I think that your idea of giving thrombolytic therapy intraoperatively is a great idea and I would like to try it. I compliment you and your group for having tried it.

Regarding the evaluation of technical adequacy, no, we do not do routine ultrasounds in the operating room. Those who have done this have usually found problems such as kinking of the artery, occlusion of the external carotid, or some gross defect in the operating room after restoring flow. A lot of the techniques that we routinely use, including frequent using of plication of the artery and patching, are typically the corrective measures that many authors use once the defect is detected. Since we do these routinely, we feel the ultrasound is of limited benefit.

Would I not reoperate on someone who had a stroke if he had a normal ultrasound before leaving the operating room? Absolutely. The thrombus often takes 3 or 4 hours to form. You may have had a perfectly normal ultrasound at one point in time but later see a flow defect. In fact, we've had ultrasounds that look normal while the patient is having the stroke and taken them back and found the platelet defect. So I don't think that you can rely upon a normal ultrasound at any time to tell you that you shouldn't go back.

I have not observed the platelet drop.

And finally, about the selective shunting, there are two points to that question. First, there was definitely a bias for using general anesthesia and, therefore, shunts for higher risk patients. Patients that had massive strokes were often given general anesthesia because they couldn't be adequately monitored for new neurologic deficits.

Secondly, I believe that selective shunting is probably better than routine shunting. Only last week, I saw an embolus pass through the shunt, resulting in a terrible stroke. I believe that shunting is not the wherewithal to prevent strokes. In fact, it can, in some cases, be the cause of stroke.

Thank you very much.

Dr Enrico Ascher (Brooklyn, NY). Tom, thank you very much for sharing your experience with us. It's always a pleasure to listen to this very careful analysis.

I just want to add a little comment regarding the intraoperative ultrasound, because I think it's very important. Not only is the B mode important, because that's easy to

see: you see a couple flaps and you remove and fix them. But what I think is also important is to measure volume flows in the internal carotid artery right after your procedure is completed. The reason I'm saying that is because in two patients of 40 that had flow volumes less than 100 cc/min, both patients had perioperative strokes within 6 hours. And there was no B mode abnormality, which is perfect repair, yet they had it. And I really don't know what the cause for this is, maybe because we don't do perioperative arteriograms or MRAs in all the patients; we base solely on duplex scan. But for those who are based on duplex scans preoperatively, I think at this point it would be very important to get a completion duplex scan, not only with the B mode but also look at the hemodynamics. Could you comment on this?

Dr Riles. I cannot perfectly defend the fact that we don't do it. We've tried to work around it. I guess I can't get my hospital administrators to buy me an extra duplex scan to roll in the operating room these days, so I'll use that as an excuse. Of course, we also see that our stroke rate overall is no higher than those groups who routinely employ these procedures.

Dr Richard M. Green (Rochester, NY). I have two questions, one very practical and one philosophical.

For those folks who are doing carotid stenting, the first step and last steps, in most places, are to do an intracranial angiogram looking for an embolus. If you're going to do neurosalvage, it's really at that time that you must do it. So my philosophical question to you, recognizing that most strokes in most experienced places are related to emboli, should we be doing intraoperative arteriography of the head before and after we do carotid endarterectomy?

And my second question relates to what you do when you find these platelets. We've been impressed that when the platelets are there, they're not on the patch; they're really on the endarterectomized surface. So repatching that makes no sense, and we've gone to using bypass. And I see that you had two cases of doing that. Is that a later trend recognizing the same thing?

Dr Riles. First of all, the first question is about the intraoperative angiography. We did that for a while. I must say that it's difficult when you're doing local anesthesia to do a lot of intraoperative angiography. It's just difficult for me, it's difficult for the patient, and that's the reason we never were very satisfied with it. Patients are moving around, and the films were just never worthwhile. So my answer would be no, I don't have any particular use for that.

The second point was the platelets. I think it depends on what the problem is. It's a little misleading saying we simply repatched it because it was often more than that. Usually the problem was something like a kink or a fold in the artery. And so the artery would be opened up, that part of it would be repaired, and then the new patch would be put on. And the second patch was sort of incidental to trying to fix it. Sometimes you will see platelets over the entire endarterectomy site. Those are the ones that worry me, and those are the ones I definitely would do an interposition graft on. I would get rid of that site, because that's not one simple defect: the whole surface has become thrombogenic.

Dr Krishan Gulati (Plattsburgh, NY). What I have done is, obviously, if a patient has a stroke on the table or in the recovery room or some kind of a TIA, you bring the patient to the operating room; instead of exploring it, do an angio right on the table. Once you have done the angio, you may explore it, you may not explore it, but at least you have opened the incision and you see if there is flow and make sure everything looks good. You do an angiogram instead of just opening up the incision and going forward, and it works out very well. At least I have saved some patients opening the arterial suture line.

Dr Riles. I think that's admirable. I guess I don't have the patience to wait to see what the angiogram looks like. Usually if I'm there that close to the artery, my tendency is more just to open it up and get the shunt in right away.

Dr Larry A. Scher (Manhasset, NY). We had a patient at our institution that was taken back to the operating room after a postoperative neurologic deficit and had t-PA used, and the patient had a cerebral hemorrhage. We looked at the literature and found Dr Comerota's article and a small series from Canada, which reported pretty good results with this treatment. I'm wondering if adverse outcomes from this treatment ever get reported. If somebody is going to try to tackle a trial as you'd mentioned, Tom, does anybody know of any adverse outcomes related to intraoperative use of t-PA used to treat postoperative stroke after carotid endarterectomy?

Dr Riles. That's a good point. I don't have much experience other than these two cases.