Outcomes of urgent carotid endarterectomy for stable and unstable acute neurologic deficits

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Objective: **The aim of the study was to assess the outcomes of carotid endarterectomy (CEA) performed in an urgent setting on acutely symptomatic patients selected through a very simple protocol.**

Methods: **From January 2002 to January 2012, 193 symptomatic patients underwent CEA. Of these, 90 presented with acute symptoms, and after a congruous carotid stenosis was identified, underwent urgent operations (group 1): 27 patients had transient ischemic attack (group 1A), 52 patients had mild to moderate stroke (group 1B), and 11 patients had stroke in evolution (group 1C). The remaining 103 patients with a nonrecent neurologic deficit were treated by elective surgery in the same period (group 2). End points were 30-day neurologic morbidity and mortality.**

Results: **The median delay of urgent CEA (U-CEA) from deficit onset was 48 hours (interquartile range, 13-117 hours). Groups 1 and 2 were comparable in demographics. Acute patients showed a higher rate of stroke at presentation (70% vs 37%; $P = .001$) and of history of coronary artery disease (30% vs 13.5%; $P = .007$). Acute patients sustained six postoperative strokes (6.6%). Neurologic outcomes were correlated to clinical presentation: no strokes occurred in group 1A patients, and 5.8% group 1B patients and 27.3% group 1C patients had postoperative stroke ($P < .01$). Postoperative mortality was 4.4% for U-CEA: one fatal myocardial infarction, one intracranial hemorrhage, and two thromboembolic strokes. Elective patients sustained four postoperative strokes (3.9%), with one death (0.9%) as a consequence of hyperperfusion cerebral edema. U-CEAs performed ≤48 hours from symptom onset had a lower postoperative stroke rate than those performed >48 hours (4.4% vs 8.8%; $P = .3$). Among patients presenting with a stroke (group 1B), the National Institutes of Health Stroke Scale (NIHSS) assessment at discharge showed improvement in 79% (although only 25% had ≥4 points in reduction), stability in 17%, and deterioration in 4%. Patients with moderate stroke were slightly better in NIHSS improvement than those with mild stroke (median NIHSS variation at discharge, −3 vs −1; $P = .001$).**

Conclusions: **Our results with U-CEA confirm that this population has a higher risk profile compared with elective surgery. The type of acute presentation is correlated with perioperative risk. U-CEA was safe when performed on patients presenting with transient ischemic attack. An acceptable complication rate was achieved for patients with minor to moderate strokes. The poorest outcomes occurred in patients presenting with stroke in evolution: U-CEA in these patients should be offered with extreme caution, although we are aware that a conservative treatment may not grant a better prognosis. (J Vasc Surg 2014;59:440-6.)**

At the beginning of the 1990s, large international randomized trials[1-3] demonstrated that elective carotid endarterectomy (CEA) is an effective intervention for patients with symptomatic carotid stenosis. In the mid-2000s, a pooled analysis of data from the North American Symptomatic Carotid Surgery Trial (NASCET) and European Carotid Surgery Trial showed that patients who underwent surgery ≤14 days after symptoms onset had better results than those operated on >14 days.[4-6]

Despite recent guidelines recommending CEA within this time frame,[7-9] debate on the timing of CEA is still open. There is the possibility that a more expedited approach (emergent/urgent CEA) might increase CEA effectiveness and minimize the rate of ischemic recurrence. However, definitive data are lacking on the risks of treating patients with cerebrovascular accidents (CVAs) in the hyperacute phase, sometimes before stabilization of symptoms. The aim of this study was to review our series of CEA performed on acute symptomatic patients since a Stroke Unit was opened in our hospital.

METHODS

Study design. We retrospectively reviewed a cohort of patients selected by a protocol aiming at reducing the risk of stroke[10-12] or stroke recurrence[13-15] for patients presenting in our hospital with an acute onset of a neurologic deficit. This protocol was set in 2002 when the Stroke Unit opened. We also retrospectively reviewed all patients who underwent elective CEA in the same period of time (January 2002 to January 2012).

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Data collected included demographics, risk factors, medications, clinical presentation, radiologic findings at admission and at discharge, timing of surgery, operative details, complications, and follow-up. Primary outcomes were 30-day postoperative strokes, 30-day postoperative mortality, and combined 30-day death/stroke rate. Outcomes of urgently operated-on patients were further stratified according to their clinical presentation and to the timing of surgery from symptom onset.

Definitions. We defined a single transient ischemic attack (TIA) as a focal neurologic deficit lasting ≤24 hours, and recurrent TIAs were recurrent episodes of neurologic deficit all lasting ≤24 hours. Minor stroke was defined as stroke ranging from 1 to 4 on the National Institutes of Health Stroke Scale (NIHSS), moderate stroke was defined as a stroke ranging from 5 to 15 on the NHISS, and stroke in evolution (SIE) was defined as a stroke with progression or fluctuation of neurologic deficit while the patient was under observation.

Postoperative hemorrhagic stroke was defined as appearance of a new hemorrhagic lesion on brain computed tomography (bCT). Postoperative ischemic stroke was defined as expansion of a preoperative lesion or appearance of new ischemic lesions on bCT, when associated with nontransitory progression of NIHSS. Postoperative hyperperfusion syndrome was defined as the appearance of diffused brain edema on bCT associated with headache or seizures in presence of postoperative hypertension.

Emergency diagnostic workup. After blood tests and an electrocardiogram, a neurologic evaluation is immediately performed by a neurologist. In case of a TIA, the risk of progression to stroke by the ABCD2 assessment (age, blood pressure [BP], clinical features of the TIA, duration of symptoms, and diabetes mellitus)[16] is calculated. The grade of neurologic deficit is established by the NIHSS.[17] A bCT is routinely obtained to rule out intracranial hemorrhage. If no sign of hemorrhage or massive stroke is detected on bCT and the NIHSS is ≤15 without loss or severe alteration of conscience, patients are referred to the vascular surgeon for urgent carotid ultrasound imaging.

The carotid duplex scan is performed by vascular surgeons (case load ≥200 carotid duplex scans per physician per year) and it is available from 8:00 am to 8:00 pm every day. If a patient is admitted during the night, the screening carotid ultrasound assessment is performed by the neurologist on the ward. If the result is positive for echographic signs of plaque or stenosis, a confirmatory examination is immediately performed by the vascular surgeon on call, and if the result is negative, the examination is scheduled for the morning after. Evidence of an ipsilateral carotid stenosis ≥50% according to NASCET criteria is the main indication for urgent carotid surgery.

Second-level imaging of neck vessels, such as CT angiography or magnetic resonance angiography, is obtained when the accuracy of duplex is highly questionable (ie, hard calcific plaques or high lesions). When there is no agreement between the first-level and second-level examinations, carotid angiography is performed as the third-level assessment.

A preoperative assessment is immediately performed by an anesthesiologist. Timing of surgery is then discussed among the surgeon, neurologist, and anesthesiologist, with the aim of performing CEA as soon as possible after the diagnosis, given that appropriate medical treatment is started (ie, control of arterial hypertension).

Patients who are not already taking antiplatelet drugs are given acetylsalicylic acid (300 mg orally or 250 mg intravenously in case of difficult oral intake). Uncontrolled systolic hypertension (systolic BP ≥220 mm Hg) or moderate diastolic hypertension (diastolic BP 100-140 mm Hg) is treated by an intravenous infusion of labetalol (20-300 mg) and urapidil (50-250 mg). Uncontrolled severe diastolic hypertension (diastolic BP >140 mm Hg) is treated with slow infusion of sodium nitroprusside (0.5 mg/kg/min). Patients taking oral anticoagulants are reversed through vitamin K and preoperatively switched to low-molecular-weight heparin. Carotid surgery is deferred until normalization of coagulation tests.

Surgery is scheduled as soon as possible, often before admission to the ward. When necessary, an emergency operating room (OR) is used, and an emergency anesthesiologist is called. Especially in recent years, patients with TIAs with a low ABCD2 score admitted during the evening or night are deferred until the next morning if an elective OR is available. Sometimes this option is also chosen if the emergency OR is expected to be unavailable for a long time. In these cases, the patient is admitted to the Stroke Unit.

None of our patients urgently admitted for a neurologic deficit are deferred or postponed on purpose until a predetermined period of time.

Operative procedure. Seven surgeons performed 193 CEAs during the 10-year period. Surgical technique and periprocedural and intraoperative monitoring were the same for patients who underwent urgent and nonurgent operations.

Routine intraoperative physiologic monitoring included pulse oximetry, electrocardiogram, and invasive BP measurement. In case of general anesthesia, cortical blood oxygen saturation was continuously monitored through an INVOS Cerebral Oximeter (Somanetics, Troy, Mich). In case of locoregional anesthesia, motor and cognitive functions were clinically monitored. A temporary indwelling shunt was selectively used in case of persistent ≥20% fall of cortical oxygenation at INVOS under general anesthesia, whereas for locoregional anesthesia, when motor or cognitive deterioration appeared. Heparin (5000 IU) was administered intravenously before cross-clamping, and its efficacy was assessed by the activated clotting time test (target ≥220 seconds).

The surgical technique consisted of patch angioplasty in most patients, and eversion endarterectomy was chosen when the carotid artery was redundant or kinked. Routine intraoperative carotid duplex scanning was performed in all

Table I. Patient groups by clinical presentation

Group	*Presentation*	*Patients, No.*
Total		193
Group 1	Acute neurologic deficit	90
Group 1A	TIA	27
	Single	16
	Recurrent	11
Group 1B	Stroke	52
	Minor	33
	Moderate	19
Group 1C	SIE	11
Group 2	Nonrecent TIA or stroke	103
Group 2A	TIA	65
Group 2B	Stroke	38

SIE, Stroke in evolution; *TIA,* transient ischemic attack.

patients to assess the patency of the internal carotid artery and to detect residual end-plaque frond/flap or other defects. At the end of the procedure, heparin was selectively reversed after a second activated clotting time test.

During the postoperative period, patients who underwent urgent operations were admitted to the Stroke Unit, where evolution of their clinical status was strictly monitored: any deterioration of a previous deficit or appearance of a new deficit was recorded and quantified through NIHSS variation and immediately investigated through bCT. In patients with no postoperative deterioration, bCT was routinely repeated 48 to 72 hours after surgery.

Postoperative hospitalization of elective CEA patients was conducted in a vascular surgery ward. Neurologic evaluation was routinely repeated after surgery, but bCT was not routinely repeated.

After discharge, all patients were scheduled for a duplex scan at 6 and 12 months and yearly thereafter. The presence of a >50% contralateral carotid stenosis was sometimes the reason for a shorter interval after the first year. A vascular neurologist performed a clinical follow-up of all urgently operated-on patients at 6 and 12 months and as needed by each patient thereafter.

Statistical analysis. Results are expressed by means and standard deviations or by median and interquartile range (IQR) as appropriate. Statistical analysis was performed using the Pearson test and, when appropriate, the Fisher exact test for nominal variables. Comparison of continuous measures was performed using the Student *t*-test or, when appropriate, the Wilcoxon signed rank test. Significance was assumed at $P < .05$.

RESULTS

From January 2002 to January 2012, 193 symptomatic patients were treated for symptomatic carotid disease (Table I). Ninety patients were urgently operated on after admission to our emergency department (group 1). Among these 90 patients, clinical presentation was TIA in 27 (30%, group 1A), stable stroke in 52 (57.8%, group 1B), and SIE in 11 (12.2%, group 1C).

In the same period of time, 103 patients (group 2) underwent elective surgery because of a nonrecent CVA (Table I); 65 (63.1%) had sustained a TIA, and 38 (36.9%) were diagnosed with an ischemic stroke (Table II). This difference between group 1 and group 2 in the proportion of transient symptoms was highly significant ($P = .0001$).

Study population was composed of 129 men (66.8%) and 64 women (33.2%), and their mean age at presentation was 71 years (range, 37-92 years). The two groups did not significantly differ in age and sex (Table II). The prevalence of classic risk factors for CVAs, such as systemic hypertension, smoking habit, and dyslipidemia, was similar across groups. Group 1 showed a significantly higher proportion of patients with vascular comorbidities, such as a history of coronary heart disease (30% vs 13.5%; $P = .01$), a history of peripheral artery disease (15.5% vs 6.8%; $P = .4$), and a trend for a higher prevalence of diabetes mellitus (27.7% vs 18.4%; $P = .1$).

We always performed an urgent bCT in group 1 patients, and ischemic lesions were recorded in 22 (24.4%) before surgery. Magnetic resonance imaging with diffusion-weighted imaging sequence was also performed in eight patients to confirm CT results. Group 2 patients also had a preoperative bCT, which was repeated only in seven patients where preoperative symptoms worsened or new symptoms developed after surgery (Table III).

In 131 patients (67.9%), the sole diagnostic imaging modality was carotid duplex. Second-level imaging of carotid arteries was necessary for 56 (29%), comprising 48 CT angiographies and eight magnetic resonance angiographies. Only six (3.1%) underwent an angiogram. The proportion of second-level imaging was higher for group 2 (20% vs 53.4%; $P = .001$).

We then looked specifically at the timing of our operations in group 1 patients. The median time from symptom onset to surgery was 48 hours (IQR, 17-113 hours; Table III); all 90 patients were diagnosed with carotid stenosis in the acute phase of a CVA and underwent operations as soon as possible. Median time from symptoms to observation was 4 hours (IQR, 2-24 hours) and from observation to surgery was 18 hours (IQR, 8-64 hours).

A Vascu-Guard bovine pericardium patch (Synovis, St. Paul, Minn) was used to close the arteriotomy in 150 patients (77.2%), direct closure of the arteriotomy was chosen in 26 (11.1%), and eversion CEA was the technique for 24 (10.3%). One procedure from group 1 was an urgent standard embolectomy with Fogarty catheter. This patient had embolic carotid bulb occlusion with patency of the distal vessel. After surgery, he was found to have a deep vein thrombosis and a patent foramen ovale. Mean duration of CEA was 119 minutes (range, 40-190 minutes), with a mean carotid clamping of 40 minutes (range, 14-100 minutes).

On the basis of intraoperative monitoring, 14 patients (7.2%) required shunting. An intraoperative ultrasound control after suture of the arteriotomy is a standard procedure in our division and was performed during all interventions. This led to three surgical revisions (1.5%) because of

Table II. Comparison of urgently and nonurgently operated-on patients: Demographics, clinical presentation, treatment, and outcomes

Variables[a]	*Total (N = 193)*	*U-CEA (n = 90)*	*E-CEA (n = 103)*	P
Demographics				
Age, years	71 (37-92)	70 (37-89)	72 (47-92)	.6
Male sex	129 (66.8)	64 (71.1)	65 (63.1)	.2
Hypertension	167 (86.5)	75 (83.3)	92 (89.3)	.3
Smoking	115 (59.6)	56 (62.2)	59 (57.2)	.5
Dyslipidemia	98 (50.8)	48 (53.3)	50 (48.5)	.5
Diabetes	44 (22.8)	25 (27.7)	19 (18.4)	.1
Coronary artery disease	41 (21.2)	27 (30.0)	14 (13.5)	.007
Peripheral artery disease	21 (10.9)	14 (15.5)	7 (6.8)	.06
Presentation				
Stroke	101 (52.3)	63 (70.0)	38 (36.9)	.0001
TIA	92 (47.7)	27 (30.0)	65 (63.1)	
Surgery				
Duration, minutes	119 (40-190)	119 (60-180)	119 (40-190)	.5
Clamp time, minutes	40 (14-100)	40 (16-80)	39 (14-100)	.5
Patch angioplasty	150 (77.7)	71 (78.8)	79 (76.6)	.6
Shunting	14 (7.2)	5 (5.6)	9 (8.7)	.4
30-day outcomes				
Stroke	10 (5.2)	6 (6.6)	4 (3.9)	.5
Mortality	5 (2.6)	4 (4.4)	1 (0.9)	.1
Stroke/mortality	11 (5.7)	7 (7.7)	4 (3.9)	.3

E-CEA, Elective carotid endarterectomy; *TIA,* transient ischemic attack; *U-CEA,* urgent carotid endarterectomy.
[a]Categoric data are shown as number (%) and continuous data as mean (range).

Table III. Comparison of timing of surgery, radiologic findings, and outcomes of urgently operated-on patients (group 1) by clinical presentation

Variables[a]	*Total (N = 90)*	*TIA (n = 27)*	*Stroke (n = 52)*	*SIE (n = 11)*	P
Timing of surgery					
Onset-surgery, hours	47.5 (17-113)	29 (18-95)	49.5 (16-117)	53 (17-168)	.7
Preoperative					
Ischemic lesion at bCT/MRI	27 (30.0)	0	24 (46.1)	3 (27.3)	
Postoperative					
New ischemic lesion at bCT	35 (38.9)	0	27 (51.9)	8 (72.7)	
Hemorrhagic lesion at bCT	1 (1.1)	0	1 (1.9)	0	
30-day outcomes					
Stroke	6 (6.6)	0 (0)	3 (5.8)	3 (27.3)	.008
Mortality	4 (4.4)	1 (3.4)	1 (1.9)	2 (18.2)	.05
Stroke/mortality	7 (7.7)	1 (3.4)	3 (5.8)	3 (27.3)	.03

bCT, Brain computed tomography; *IQR,* interquartile range; *MRI,* magnetic resonance imaging; *SIE,* stroke in evolution; *TIA,* transient ischemic attack.
[a]Categoric data are shown as number (%) and continuous data as median (IQR).

residual stenosis of the distal internal carotid, one in group 1 (1.1%) and two in group 2 (1.9%).

Outcomes and clinical presentation. Overall, ten 30-day postoperative strokes (5.2%) were recorded, and the 30-day mortality rate was 2.6% (five patients). The combined death/stroke rate was 5.7% (11 patients). Six of the 90 urgent CEAs were complicated by periprocedural stroke (6.6%), and four deaths (4.4%) were recorded. Four postoperative strokes were recorded among the 103 elective CEAs (3.9%), with only one death (0.9%), which was caused by postoperative hyperperfusion cerebral edema (Table II).

The postoperative stroke rate of urgently operated-on patients was significantly correlated with clinical presentation (Table III). None of the 27 patients with a TIA (group 1A) had clinical or radiologic signs of brain ischemia. Among 52 patients from group 1B (mild to moderate stroke), three (5.8%) sustained a postoperative stroke, namely, one fatal intracranial hemorrhage on postoperative day 4 and two thromboembolic lesions (postoperative appearance of multiple ipsilateral ischemic lesions on bCT) associated with significant NIHSS deterioration (+5 points in both patients). Group 1C patients had the highest rate of complications, with three of 11 patients (27.3%) with a postoperative stroke: two with a fatal massive hemispheric ischemic lesion with cerebral edema and herniation and one thromboembolic stroke associated with significant NIHSS deterioration (Table IV). Mortality was similarly unevenly distributed: one death in 27 group 1A

Table IV. Detailed description of postoperative neurologic complications within urgently operated-on patients (group 1)

Patient sex, years	*Group*	*NIHSS*	*Timing of surgery*	*Postoperative bCT*	*Time of onset*	*Outcome*
F, 68	1C	8-12	168 hours	Vast hemispheric infarction with brain edema	POD 1	Death
M, 77	1B	5	240 hours	New discrete ischemic lesion	OD	NIHSS 10 (+5)
M, 51	1C	2-6	40 hours	Extension of previous ischemic lesion	POD 3	NIHSS 15 (+9)
M, 68	1B	3	102 hours	Intraparenchymal hemorrhage	POD 4	Death
M, 72	1C	6-13	56 hours	Vast hemispheric infarction with brain edema	POD 2	Death
F, 71	1B	2	34 hours	2 new ischemic lesions	OD	NIHSS 7 (+5)

bCT, Brain computed tomography; *F,* female; *M,* male; *NIHSS,* National Institutes of Health Stroke Scale; *OD,* operative day; *POD,* postoperative day.

Table V. Comparison of outcomes of urgently operated-on patients (group 1) by timing of surgery

Variable	*≤48 hours (median 17 hours), No. (%)*	*>48 hours (median 110 hours), No. (%)*	P
Group 1 (n = 90)	45	45	
30-day stroke	2 (4.4)	4 (8.8)	.6
30-day stroke/mortality	2 (4.4)	5 (11.1)	.4
Group 1A (n = 27)	16	11	
30-day stroke	0 (0)	0 (0)	1
30-day stroke/mortality	0 (0)	1 (9)	.4
Group 1B (n = 52)	25	27	
30-day stroke	1 (4)	2 (7.4)	.9
30-day stroke/mortality	1 (4)	2 (7.4)	.9
Group 1C (n = 11)	4	7	
30-day stroke	1 (25.0)	2 (28.6)	.7
30-day stroke/mortality	1 (25.0)	2 (28.6)	.7

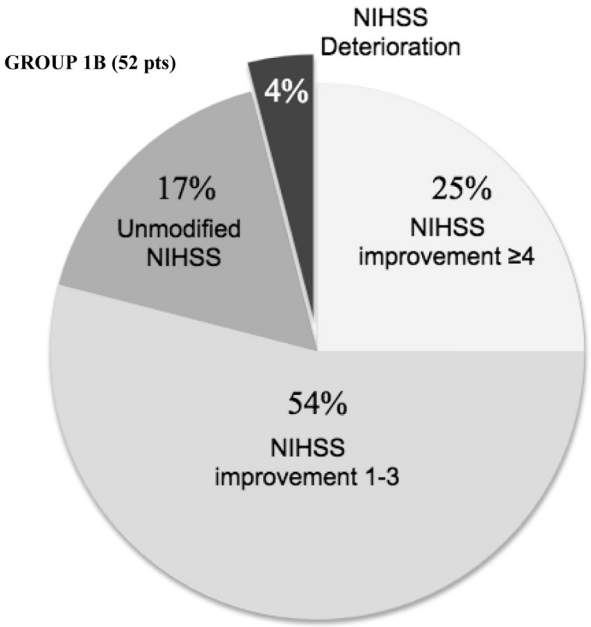

Fig. Distribution of National Institutes of Health Stroke Scale (*NIHSS*) variation after carotid surgery in group 1B patients.

patients (3.7%) because of a fatal myocardial infarction, one among 52 in group 1B (1.9%), and 2 of 11 in group 1C (18.2%).

Combined mortality/stroke rate was 7.7% (seven of 90) for group 1, 3.7% (one of 27) for group 1A, 5.7% (three of 52) for group 1B, and 27.3% (three of 11) for group 1C, respectively (Table III). SIE patients had a significantly higher 30-day postoperative stroke rate ($P = .008$), 30-day mortality rate ($P = .05$), and 30-day combined mortality/stroke rate ($P = .03$) than patients presenting after TIA or stable stroke.

Outcomes and timing of surgery. Further analysis of outcomes of group 1 patients was performed after stratification by delay of surgery from onset of symptoms (Table V). We used the median timing of surgery within this population as a reference: outcomes of patients operated on ≤48 hours from onset of symptoms (median delay, 17 hours; IQR, 9-26 hours) were compared with outcomes of patients operated on after >48 hours (median delay, 110 hours; IQR, 55-210 hours). CEAs performed ≤48 hours resulted in two postoperative strokes, whereas CEAs performed >48 hours resulted in four postoperative strokes (4.4% vs 8.8%; $P = .6$). This difference, although not statistically significant, was confirmed in group 1B (4% vs 7.4% postoperative stroke; $P = .9$) and in group 1C (14.3% vs 50% postoperative stroke; $P = .4$).

NIHSS variation in group 1B patients. Group 1B patients (minor to moderate stroke) had a median NIHSS of 4 (IQR, 2-6.2) on admission and of 2 (IQR, 1-4) at discharge ($P < .001$). Median absolute NIHSS variation was −2 (IQR, 0 to −4), whereas its median relative decrease was −50% (IQR, −25% to −75%). NIHSS improved by ≥4 points in 13 of 52 patients (25%), by <4 points in 28 patients (54%), and showed no improvement in the remaining nine (17%). In two patients (4%), we observed persistent NIHSS deterioration; in both patients, the NIHSS gained 5 points: from 5 to 10 in one and from 2 to 7 in the other (Fig).

Absolute value of NIHSS variation was significantly higher for patients with moderate stroke than for patients with minor stroke ($P = .001$). The former subgroup had a median score of 8 (IQR, 5-11) at presentation and a median score of 5 (IQR, 2-8) at discharge, with a median variation of −3 (IQR, 0 to −5). The median NIHSS in the latter subgroup was 3 (IQR, 2-3.5) on admission and 1

(IQR, 0-2) at discharge, with a median variation of −1 (IQR, −0.75 to −2).

DISCUSSION

This study is the result of a retrospective analysis of data from prospectively identified patients who were treated with urgent CEA in our hospital from 2002 to 2012. Performing CEA in an urgent setting requires strong clinical and logistic collaboration between vascular surgeons and neurologists, and this multidisciplinary collaboration started in our center in 2002. In this series, the median delay of surgery from onset of symptoms was 48 hours (to our knowledge, one of the shortest in the literature), and we regard this as a sign of our efforts to avoid any unnecessary delay. Besides, the number of urgently operated-on patients was very close to that of electively operated-on patients (90 vs 103), and in the future, this proportion will possibly reverse because all acutely ill patients are offered urgent treatment whenever possible.

Our diagnostic flow chart is very simple and was applied, regardless of different clinical presentation, to patients with TIAs, strokes with minor to moderate neurologic deficit, and even in patients with rapidly deteriorating brain infarctions. Beyond obvious exclusion of patients with hemorrhage and major neurologic disability, population selection was minimal compared with other studies. Brain imaging was done by plain CT in all patients, with complementary use of diffusion-weighted magnetic resonance imaging in a small minority of patients.

The surgical protocol is exactly the same as with elective CEA: cerebral monitoring by near-infrared spectroscopy and intraoperative control by ultrasound imaging are both easily managed by the operating surgeon, without the need of specialized personnel (ie, electrophysiologist for electroencephalogram or radiology technician for angiography).

Performing urgent CEA on this "unselected" population yielded acceptable 30-day rates of stroke, death, and combined stroke/death. Not surprisingly, a higher rate of postoperative complications and death resulted from comparison with patients operated on for a nonacute disease, although the difference was not statistically significant. These data have already been reported[18,19] but may be misleading if taken alone: a bigger proportion of patients presenting with stroke and a higher prevalence of vascular comorbidities in the group of urgent CEA may at least partly account for worse outcomes and make the two populations not perfectly comparable.

Within group 1, results of urgent surgery were not uniform across clinical subgroups. The postoperative course was neurologically uneventful for patients presenting with a single or recurrent TIA (group 1A). An aggressive surgical approach is safe in this population and is of particular value when the risk of recurrence is high, such as in patients with an ABCD2 score ≥4.

Results were particularly poor in the patients presenting with SIE (group 1C). This is historically the clinical presentation associated with the worst postoperative outcomes,[20] although the natural history of SIE does not indicate medical treatment as a safe and effective alternative to surgery. Leseche et al[21] published a report in 2012 documenting excellent outcome of CEA performed in the acute phase of SIE (median delay of 6 days from onset of symptoms to surgery). Their experience was quite different from ours and from most series. In the light of our results, we have gradually shifted to a more conservative approach.

Group 1B is the most interesting because it was composed of a number of urgently operated-on patients with stable stroke who showed a low rate of postoperative complications. This was the less predictable population because there is a striking lack of published data on this specific issue. Besides the relatively low rate of postoperative strokes, we observed that a very high percentage of patients (79%) experienced postoperative NIHSS improvement, and we consider it a promising result. We do not know if the beneficial effects associated with early intervention should be attributed to prevention of ischemic recurrence (possible episodes of plaque re-embolization) or precocious blood flow restoration on a still vital ischemic brain tissue (brain penumbra), with prevention of perilesional edema (a mechanism of blood flow limitation itself), or even to both.

The only study on U-CEA reporting the outcome of postoperative neurologic improvement of minor and moderate stroke was published by Capoccia et al.[22] Our data support some of their conclusion: minimizing the time of intervention reduces the risk of recurrence and can be considered safe even for patients with an NIHSS score >8 and ≤15. Overall, we did not find a statistical correlation between the timing of CEA and the rate of postoperative neurologic complications. Using our median time interval from symptoms to surgery as a threshold for comparison, we did not find a higher risk of stroke or death in the patients who received surgery ≤48 hours from onset of symptoms.

These data are in contrast with those reported by Stromberg et al[23] in a recent analysis of the Swedish Vascular Registry. After stratifying for timing of surgery for >2500 symptomatic patients who underwent CEA in Sweden, they concluded that CEA performed <48 hours from onset of symptoms has a higher rate of complications than if the procedure is delayed by >48 hours (combined stroke/death rate of 11.5% vs 3.6%-5.7%).

We believe that urgency alone does not increase risks of postoperative complications of CEA and that, as stated by Naylor[24] in a letter to Stromberg et al, high risks deriving from a surgical procedure should not be considered without comparison with the natural history of the specific CVA during the hyperacute period. This is not possible unless a clinical stratification is adopted when reporting the outcome of urgent CEA.

CONCLUSIONS

Our results with U-CEA confirm that this population has a higher risk profile compared with elective CEA. There is a correlation between the type of acute

presentation and perioperative risk. U-CEA was safe when performed on patients presenting with TIA. In our opinion, the nonelective procedure should be available at least in case of an ABCD2 score ≥4. An acceptable complication rate was achieved for patients with minor to moderate strokes, and we believe it is justified by the significant risk of early ischemic recurrence in the natural history of the disease. Treatment in the hyperacute phase can also improve neurologic symptoms. We had the poorest outcomes on patients presenting with SIE: U-CEA on these patients should be offered with extreme caution, although we are aware that a conservative treatment may not grant a better prognosis.

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AUTHOR CONTRIBUTIONS

Conception and design: MC, PL, RD
Analysis and interpretation: IB, MC, GM, PL, RD
Data collection: IB, GM, PL, DM, AS
Writing the article: IB, MC
Critical revision of the article: IB, MC, PL
Final approval of the article: IB, MC, GM, PL, DM, AS, RD, PS
Statistical analysis: IB, MC, DM
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Overall responsibility: MC
IB and MC contributed equally to this work.

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