Revascularization of the Internal Carotid Artery for Isolated, Stenotic, and Symptomatic Kinking

Giulio Illuminati, MD; Francesco G. Caliò, MD; Vassilios Papaspyropoulos, MD; Giuseppe Montesano, MD; Antonio D’Urso, MD

Hypothesis: The operation for isolated, stenotic, and symptomatic kinking of the internal carotid artery is safe and effective in preventing stroke and relieving the symptoms of cerebral ischemia.

Design: A consecutive sample clinical study with a mean follow-up of 44 months.

Setting: The surgical department of an academic tertiary care center and an affiliated secondary care center.

Patients: Fifty-four patients with a mean age of 67 years underwent 55 revascularizations of the internal carotid artery. The surgical procedures consisted of the following: shortening and reimplantation in the common carotid artery in 36 cases, bypass grafting in 15 cases, and transposition into the external carotid artery in 4 cases.

Main Outcome Measures: Cumulative survival, primary patency, and stroke-free and neurologic symptom-free rates expressed by standard life-table analysis.

Results: No patients died in the postoperative period. The postoperative stroke rate was 1.8%. The cumulative rates (SEs) at 5 years were as follows: survival, 70% (10.2%); primary patency, 89% (7.8%); overall stroke free, 92% (6.8%); ipsilateral stroke free, 96% (5.3%); neurologic symptom free, 90% (7.5%); and ipsilateral symptom free, 93% (6.5%).

Conclusion: Revascularization of the internal carotid artery for the treatment of isolated, stenotic, and symptomatic kinking is safe and effective in preventing stroke and relieving symptoms of cerebrovascular insufficiency.

Arch Surg. 2003;138:192-197

Kinking of the internal carotid artery (ICA) is caused by elongation of the artery itself, with angulation of the vessel’s axis of 90° or less.1 This condition is detected in about 5% to 25% of the arteriograms of cervical arteries performed for cerebrovascular symptoms or for the further definition of an incidentally encountered, asymptomatic carotid artery stenosis.2-10

In adult patients with symptoms of cerebrovascular insufficiency, kinking of the ICA is often associated with significant atheroma of carotid bifurcation. Carotid endarterectomy has proven effective for the treatment of stenotic and symptomatic plaques.11 When stenotic kinking is associated with significant atherosclerotic stenosis of the carotid bulb in symptomatic patients, it is wise, and generally accepted, to treat both conditions simultaneously, to perform a fully satisfactory revascularization and not to leave either of the 2 potentially harmful lesions in place.12,13

The surgical indication for repair of stenotic kinking of the ICA, in the presence of neurologic symptoms but in the absence of atherosclerotic stenosis of the carotid bulb, is controversial. A few series reported that isolated kinks have a benign natural history and that their presumed correlation with neurologic symptoms rarely justifies surgical treatment.8,14 Other reports, based on the belief that ICA kinking is a potentially harmful condition, indicated its treatment to be surgical and reported good results with revascularization of the ICA.3,6,9,15-22

In patients with proven clinical cerebrovascular insufficiency associated with a stenotic kinking of the ICA, when any other cause of symptoms can be eliminated, surgical correction of the kinking would be likely to allow relief of symptoms and protection against ipsilateral cerebral ischemia. In this study, we systematically performed surgical revascularization in patients with isolated, stenotic, and symptomatic kinking of the ICA, and...
analyzed the outcome to determine whether such a hypothesis could be deemed correct.

**METHODS**

From January 1, 1994, to December 31, 2000, 54 consecutive patients underwent 55 surgical corrections of isolated, stenotic, and symptomatic kinking of the ICA at the “Francesco Durante” Department of Surgical Sciences and Applied Technologies, University of Rome “La Sapienza” Hospital, Rome, Italy, and 1 affiliated secondary care center. This figure represents 10% of the 525 revascularizations of the ICA performed during the same period. The study was approved by the local ethics committee, and informed consent was obtained from all of the patients. Descriptive terms, reporting practices, and outcome criteria were standardized according to the guidelines set by the ad hoc committee on reporting standards for cerebrovascular disease.23

Stenotic kinking was defined as an excess of length of the ICA, causing an angulation 90° or less of the vessel (elongation type 3, grade 1-3, according to the classification of Metz et al1) and a hemodynamically significant stenosis of the artery at duplex ultrasound and arteriography. At duplex ultrasound, kinkings causing stenoses of more than 60%, with a peak systolic velocity of more than 150 cm/s with spectral broadening, were considered significant,24 as well as those causing stenoses resulting in 80% diameter reduction at angiography, according to the criteria of the North American Symptomatic Carotid Endarterectomy Trial Collaborators. All patients were required to have angulation and flow reduction to qualify for the study. Thirty-one patients were men and 23 were women, with a mean age of 67 years (age range, 56-91 years).

Twelve patients (22%) had diabetes mellitus (risk factor category, 1-2); 24 (44%) were smokers (risk factor category, 2-3); 33 (61%) were receiving medical treatment for arterial hypertension (risk factor category, 1-2); all had cholesterol and triglyceride levels either within normal limits or with mild, diet-controlled, elevation (risk factor category, 0-1); 22 (41%) had evidence of coronary artery disease, defined as a medical history, clinical symptoms, or electrocardiographic signs of myocardial ischemia (risk factor category, 1-2); none had significant hyperlipidemia, renal, or pulmonary disease (risk factor category, 0-1);23, and 7 (13%) had aortoiliac occlusive or aneurysmal disease.

Thirty-seven patients (69%) had hemispheric symptoms, consisting of 29 (78%) transitory ischemic attacks (TIAs), and 8 (22%) strokes. Thirteen patients (24%) were initially seen with nonhemispheric TIAs, and 4 (7%) with mixed hemispheric and nonhemispheric TIAs. Nonhemispheric symptoms (dizziness, blurred vision, lateral homonymous hemianopsia, or drop attack) were so defined after ruling out any possible underlying cardiac, vestibular, untreated arterial hypertensive, and metabolic disease.

To indicate the need for surgery, more than 1 nonhemispheric symptom was required to cause, although transitory, an impairment of everyday activities corresponding to that caused by a deficit with a neurologic severity of at least 4,23 and to be resistant to at least 2 months of appropriate medical treatment; the occurrence of this condition was validated by a neurologist devoted to the study of cerebrovascular arterial insufficiency. Preoperatively all patients had a duplex scan of the carotid arteries, a computed tomographic (CT) scan of the brain, biplane carotid angiography, and a cardiac transsternal ultrasound study.

Beside ipsilateral kinking, 7 associated lesions of the contralateral ICA were apparent, including 3 kinkings, 2 occlusions, and 2 hemodynamically significant atherosclerotic stenoses. In 9 cases, associated lesions of the vertebral artery (VA) were also evident, as 8 VAs were unilaterally dominated or hypoplasic, without potentially embolizing plaques, and 1 ended in a posteroinferior cerebellar artery, not reaching the basilar artery. Overall, these lesions of the VAs were present in 6 (15%) of 39 patients with hemispheric symptoms, and in 3 (18%) of 17 patients with mixed or nonhemispheric symptoms. None of these lesions consisted of a localized, significant, or ulcerated stenosis of a dominant VA. Therefore, they were not considered symptomatic.

A CT scan of the brain was abnormal for ischemic lesions in 19 (35%) of 54 patients: 17 lesions were homolateral and 2 were contralateral. A hypodense area was present in the brain of 7 (13%) of 54 patients referred for stroke, in 9 (31%) of 29 referred for hemispheric TIA, and in 2 (12%) of 17 referred for mixed or nonhemispheric symptoms. None of the patients presented an active uptake of the contrast medium by the ischemic lesions.

All patients underwent surgery on an elective basis and under general anesthesia. Operations were performed without any specific means of cerebral protection other than radial artery pressure monitoring and its steady maintenance. An indwelling shunt was never inserted, and systemic heparin sodium administration (0.5 mg/kg) was not reversed at the end of the operation. In the latest 23 reconstructions, a completion angiography was performed to assess the technical result of arterial revascularization: at such control, a correction of the just performed revascularization was never required.

In 36 cases (67%) a standard correction of redundant ICA was achieved by oblique transection at its origin, distal, and proximal prolongation of the arteriotomy, respectively, in the ICA and in the common carotid artery (CCA), followed by reimplantation of the ICA itself more proximally in the CCA. In 15 cases (27%) the length of the diseased artery required a CCA to ICA bypass, performed with autologous vein in 9 patients and with a polytetrafluoroethylene (PTFE) graft (Gore-Tex stretch 7 mm; W. L. Gore & Associates, Flagstaff, Ariz) in 6 patients. In 4 patients (7%), the good quality and diameter of the external carotid artery (ECA) allowed the transposition of the ICA, transacted distally to the diseased area, into the ECA itself.

On awakening, the neurologic status of the patients was assessed; thereafter, they were monitored for 2 hours in the recovery room and finally returned to the ward. Postoperatively they received low-molecular-weight heparin for 30 days and were then prescribed 100 mg/d of oral aspirin. Before discharge from the hospital, all 54 patients had an assessment of the revascularization by arteriography (31 patients [57%]) and/or duplex scanning (28 patients [52%]). The postoperative period was defined as the first 30 postoperative days.

The clinical status of the patients and patency of arterial reconstruction were assessed at 4 weeks after the operation and twice every 12 months by clinical examination and duplex ultrasound scanning. Except for the introduction of completion angiography, there was no other significant change in the workup or treatment of the patients during the study period.

As main results, patients’ survival, primary patency of arterial reconstruction, freedom from neurologic symptoms, and freedom from stroke were considered. An arterial reconstruction was considered patent if free of restenosis or thrombosis at duplex ultrasound scanning. Restenosis was defined as the occurrence of a hemodynamically significant stenosis at the site of reconstructed ICA. Freedom from neurologic symptoms was defined as the absence of any TIA or stroke, that is, hemispheric, nonhemispheric, or both. Freedom from stroke was defined as the absence of ischemic, hemorrhagic, hemispheric, or nonhemispheric stroke from the postoperative period throughout the whole length of follow-up. Results were expressed by standard life-table analysis.25
No patients died in the postoperative period. One patient awoke from anesthesia with hemiparesis. Preoperatively this patient had sustained transitory monoparesis that prompted referral and diagnostic studies of the cervical vessels. At preoperative angiography the status of the contralateral ICA and both VAs was normal, as well as that of cerebral tissue at CT scanning. Arterial reconstruction consisted in a CCA to ICA saphenous bypass graft for a complex kinking of the ICA. Surgical reexploration was immediately performed, and the arterial reconstruction appeared to be normal. In the hours following return to the ward, the neurologic status of the patient improved and returned completely to normal within 7 weeks after the operation. No other central neurologic event occurred in the postoperative period. Thus, the postoperative stroke rate was 1.8%. Neither peripheral nerve palsy nor postoperative occlusion of arterial reconstruction occurred.

No early thrombosis of the reconstructions performed was observed in the whole series, irrespective of the introduction of completion angiography. Minor complications included 2 myocardial ischemic events, which were successfully managed with medical treatment.

The 54 patients, accounting for 55 reconstructions, were all followed up for a mean period of 44 months (range, 11-93 months). Nine late deaths occurred; 4 were due to myocardial infarction, 4 to metastatic cancer, and 1 to acute pancreatitis. Overall, cumulative survival (SE) rate was 70.7% (10.2%) at 5 years.

Of the 55 operated on ICAs, 5 (9.1%) developed restenosis during follow-up. No late thrombosis occurred. Restenoses involved 2 bypass grafts (1 saphenous vein and 1 PTFE) and 3 shortening angioplasties (4 of the revascularizations had been performed for hemispheric TIA and 1 for hemispheric stroke). All the restenoses occurred within 34 months from operation, were asymptomatic, and were not reoperated on. Currently, none has shown a rapid progression to preocclusive stenosis at subsequent controls. Overall, the cumulative patency (SE) rate was 89% (7.8%) at 5 years (Table 1 and Figure 1).

Of the 17 patients referred for nonhemispheric symptoms, all were relieved after undergoing the operation. One (6%) of the 17 had a mild recurrence of symptoms about 2 years after the operation, despite a normally patent arterial reconstruction (ICA-ECA transposition) at duplex ultrasound, a normal brain CT scan, and normal findings on cardiac, metabolic, and otorhinolaryngology studies. This case was considered an ipsilateral recurrence of symptoms.

Two patients (3.7%) sustained a late stroke: one was ipsilateral and hemorrhagic, the other was associated with the occlusion of the contralateral ICA. No recurrence of hemispheric TIAs was observed. Overall, the cumulative stroke-free (SE) rate was 92.5% (6.8%) (Table 2 and Figure 2); the ipsilateral stroke-free rate, 95.7% (5.3%); the neurologic symptom-free rate, 90.4% (7.5%); and the ipsilateral symptom-free rate, 93.3% (6.5%) at 5 years. Adverse outcome linked to surgical procedure is summarized in Table 3.

### Table 1. Overall Cumulative Patency Rate for 55 Revascularizations of the Internal Carotid Artery (ICA) for Isolated, Stenotic, and Symptomatic Kinking

<table>
<thead>
<tr>
<th>Interval, mo</th>
<th>No. of ICA Revascularizations at Risk</th>
<th>No. of Arteries Occluded/Stenosed</th>
<th>No. of ICA Revascularizations Withdrawn During Interval</th>
<th>Interval Failure Rate</th>
<th>Cumulative Patency (SE) Rate, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-5</td>
<td>55</td>
<td>0</td>
<td>0</td>
<td>0.00</td>
<td>100.0 (0.00)</td>
</tr>
<tr>
<td>6-11</td>
<td>55</td>
<td>0</td>
<td>2</td>
<td>0.00</td>
<td>100.0 (0.00)</td>
</tr>
<tr>
<td>12-17</td>
<td>53</td>
<td>2</td>
<td>1</td>
<td>0.04</td>
<td>96.2 (2.58)</td>
</tr>
<tr>
<td>18-24</td>
<td>50</td>
<td>2</td>
<td>6</td>
<td>0.04</td>
<td>92.1 (3.66)</td>
</tr>
<tr>
<td>24-30</td>
<td>42</td>
<td>0</td>
<td>7</td>
<td>0.00</td>
<td>92.1 (3.99)</td>
</tr>
<tr>
<td>30-36</td>
<td>35</td>
<td>1</td>
<td>5</td>
<td>0.03</td>
<td>89.3 (4.94)</td>
</tr>
<tr>
<td>36-42</td>
<td>29</td>
<td>0</td>
<td>6</td>
<td>0.00</td>
<td>89.3 (5.43)</td>
</tr>
<tr>
<td>42-48</td>
<td>23</td>
<td>0</td>
<td>6</td>
<td>0.00</td>
<td>89.3 (6.10)</td>
</tr>
<tr>
<td>48-54</td>
<td>17</td>
<td>0</td>
<td>3</td>
<td>0.00</td>
<td>89.3 (7.09)</td>
</tr>
<tr>
<td>54-60</td>
<td>14</td>
<td>0</td>
<td>4</td>
<td>0.00</td>
<td>89.3 (7.82)</td>
</tr>
<tr>
<td>60-66</td>
<td>10</td>
<td>0</td>
<td>0</td>
<td>0.00</td>
<td>89.3 (9.25)</td>
</tr>
<tr>
<td>66-72</td>
<td>10</td>
<td>0</td>
<td>2</td>
<td>0.00</td>
<td>89.3 (9.25)</td>
</tr>
<tr>
<td>72-78</td>
<td>8</td>
<td>0</td>
<td>2</td>
<td>0.00</td>
<td>89.3 (10.34)</td>
</tr>
<tr>
<td>78-84</td>
<td>6</td>
<td>0</td>
<td>0</td>
<td>0.00</td>
<td>89.3 (11.94)</td>
</tr>
<tr>
<td>84-90</td>
<td>6</td>
<td>0</td>
<td>2</td>
<td>0.00</td>
<td>89.3 (11.94)</td>
</tr>
<tr>
<td>90-96</td>
<td>4</td>
<td>0</td>
<td>4</td>
<td>0.00</td>
<td>89.3 (14.62)</td>
</tr>
</tbody>
</table>

Figure 1. Overall cumulative primary patency rate after revascularization of the internal carotid artery for symptomatic kinking. The numbers above the dotted line indicate the number of revascularizations at risk for each interval.
The association between kinking of the ICA and cerebrovascular insufficiency has been reported for 5 decades.26 From then until now, the available reports describe relief of neurologic symptoms by revascularization of a kinked carotid artery.3,6,9,10,15-22 The rationale behind revascularization of the ICA for symptomatic kinking is to relieve symptoms, prevent carotid thrombosis and stroke, and improve cerebral perfusion.

However, the relationship between kinking of the ICA and cerebrovascular insufficiency and, thus, the indication for surgical treatment have been sometimes questioned, particularly when nonhemispheric symptoms are involved.8,14 The lesser frequency of kinking, compared with atherosclerotic stenosis of carotid bifurcation, accounts for incompletely standardized indications to its surgical treatment as an isolated lesion.

Symptomatic kinking is usually seen in an elderly population. It is most likely an acquired condition, due to a degenerative process, that permits a greater elongation of the muscular layer compared with the adventitia, which determines a bending of the arterial lumen.6 The kinking causes a reduction of flow within the vessel that may be exacerbated by progressive head rotation up to the point that causes complete cessation of flow.16 Chronic arterial hypertension, causing a progressive moving upward of the aortic arch, may favor the process of kinking and is often associated with the condition.27,28 Intimal damage at the site of arterial bending has been experimentally obtained in rabbits.13 Turbulence and flow reduction may lead to intimal ulceration at the point of maximal angulation, which may result in distal embolization.17 Consequently, a kink may cause cerebrovascular symptoms with either a hemodynamic or thromboembolic mechanism, like an atherosclerotic plaque of carotid bifurcation.12,16

Different techniques have been proposed for the treatment of kinking of the ICA, essentially including resection with end-to-end reanastomosis of the ICA, lower reimplantation with shortening of the vessel in the CCA, resection and anastomosis with shortening of the CCA, and bypass grafting.10,17,29-32 The reconstruction of a kinked...
ICA requires an individualized approach, based on the complexity of the lesion itself.

Sectioning of the ICA at its origin and shortening by proximal reimplantation on the CCA, after gentle liberation of adventitial adhesions, was the standard technique used in most of the patients in this series. Resectioning and shortening of the CCA is indicated when both ICAs and ECAs are kinked, which was never the case in this study. When the kinking is complex, for example, double and extended on a significant length of the ICA, not allowing shortening by reimplantation, transposition on the ECA and bypass grafting are 2 alternative options. Transposition to the ECA terminolaterally is anatomical, elegant, and avoids insertion of grafts. This reconstruction could be performed in a few patients with good anatomic and clinical results and can be considered whenever the ECA is suitable as an inflow site (Figure 3). Bypass grafting can be performed with either autogenous saphenous vein or PTFE as conduit materials. In this series, no difference could be detected between the 2 types of grafts in terms of early and late complications, probably owing to the few patients involved: our policy remains to reserve PTFE grafts for those patients whose saphenous vein is, for whatever reason, unsuitable for grafting.

In our opinion, shortening angioplasty remains the first-choice operative procedure, suitable for treatment of most of the simply kinked ICAs. When kinking is complex, as may be anticipated at preoperative angiography, so that complete straightening of ICA cannot be accomplished, either transposition or bypass is the possible alternative. Transposition of ICA to ECA is more technically demanding than a bypass. We would prefer the former method if the ECA is of good quality and size, and if a convenient length of distal ICA can be safely mobilized. If this is not the case, bypass grafting remains the best option.

A prospective study recently compared a group of patients with ICA kinking undergoing arterial revascularization with a similar group of patients who were unoperated on.20 Late strokes and thromboses of the kinked arteries were observed only in patients who were unoperated on, whereas patients who were operated on remained free from neurologic events. Although this difference did not reach statistical significance, probably owing to the small sample size, and despite the fact that a few patients either asymptomatic or with associated carotid plaques were also included, the study supports the surgical indication of symptomatic kinking of the ICA.

We share this conviction, and deem that a crucial point is the evidence that kinking is the cause of preoperative neurologic symptoms. This evidence is usually apparent when dealing with hemispheric TIAs and strokes, but it may be not equally sound when nonhemispheric symptoms are involved. In the present series, more than 40 (75%) of the 54 patients were operated on for true hemispheric symptoms, whereas one fourth of the whole study population was operated on for nonhemispheric symptoms alone. In this latter group, criteria for inclusion in the study were rather strict and always validated by a neurologist. One could argue that, overall, 12 (22%) of the 54 patients had diabetes mellitus and that 33 patients (61%) were treated for arterial hypertension; both of these conditions may mimic a nonhemispheric neurologic syndrome. However, their incidence was comparable to that of any series of patients with vascular conditions, and in all of the cases these conditions were well controlled by medical treatment. Finally, 49 (91%) of the 54 patients, and all but 1 of those with nonhemispheric symptoms, were relieved by revascularization of the ICA.

When nonhemispheric symptoms are involved, an association between ICA kinking and significant stenoses of the VAs would be expected. This was not the case in the present study: all of the lesions consisted of hypoplasias of a single VA, which did not require surgical treatment, whereas no stenotic or embolizing lesions of a dominating VA were present. Furthermore, none of the patients either had a revascularization of a VA before revascularization of the ICA, or required a subsequent VA operation. When a significant or embolizing stenosis of a dominating VA is associated with a significant kinking and/or stenosis of the ICA in a patient with cerebral nonhemispheric symptoms, the ICA should be revascularized first. However, as revascularization of the ICA alone may be followed by a recurrence of symptoms, both ICA and VA should be simultaneously revascularized if their lesions are ipsilateral.34 If the stenosed, dominating VA is contralateral to the diseased ICA, we would revascularize the ICA first, and would operate on the VA later on, only if symptoms persist. These conditions did not occur in any of the patients of the present series.

The restenosis rate of 9% (5 patients) in this series is comparable to that experienced in other studies.32 No statistical correlation between the technique of revascularization and recurrence rate could be established. Restenoses were approached with the same policy deserved to those occurring after standard carotid endarterectomy: in the absence of symptoms, of preocclusive narrowing, and of contralateral carotid occlusion, they are controlled with duplex ultrasound study twice a year, but none has been considered yet either for reoperation or angioplasty and stenting.

For the single patient who experienced recurrence of preoperative, nonhemispheric symptoms, despite a patent revascularization, no satisfactory explanation could
be found: we can either hypothesize some progressive reduction of intracranial perfusion or simply admit that in this isolated case the operative indication was not correct.

As experienced in other studies,\textsuperscript{12,13,33} surgery for kinking of the ICA was accomplished in this series with a low postoperative neurologic morbidity, whose incidence is comparable to that of standard carotid endarterectomy.

The results of this series strongly support the assumption that surgery for isolated, stenotic, and symptomatic kinking of the ICA is safe and effective in preventing stroke and relieving symptoms of cerebrovascular insufficiency.

Accepted for publication October 5, 2002.

Corresponding author and reprints: Giulio Illuminati, MD, Dipartimento di Scienze Chirurgiche e Tecnologie Mediche Applicate, "Francesco Durante," Universita degli Studi di Roma “La Sapienza,” Via Vincenzo Bellini 14, 00198 Roma, Italia (e-mail: giulio.illuminati@uniroma1.it).

REFERENCES