

Carotid Endarterectomy for Symptomatic Complete Occlusion of the Internal Carotid Artery

Yong-Pil Cho^{a*}, Tae-Won Kwon^a, Sun U. Kwon^b, Won-Young Chae^a, and Geun-Eun Kim^a

Division of Vascular Surgery, Departments of ^aSurgery and ^bNeurology, University of Ulsan College of Medicine and Asan Medical Center, Seoul 138-736, Korea

We described 9 consecutive patients who underwent operative carotid artery exploration with attempted carotid endarterectomy (CEA) for symptomatic internal carotid artery (ICA) occlusion. Indications for this surgery based on vascular imaging included segmental occlusion of the proximal ICA and also extensive occlusion of the distal ICA in selected patients in whom color-flow duplex ultrasound showed a poorly echogenic or anechoic thrombus with a flow void, suggestive of an acute thrombus. CEA was performed successfully to restore blood flow in all 9 patients: CEA in 5 and CEA with Fogarty thrombectomy in 4. Postoperative magnetic resonance (MR) angiography confirmed that revascularization had been successful in all 9 patients, and MR imaging displayed improved perfusion in 4 patients. Despite the lack of a generalized efficacy of surgical revascularization for symptomatic ICA occlusion, our study demonstrated that preoperative vascular imaging allows the selection of patients who may benefit from CEA.

Key words: internal carotid artery, occlusion, carotid endarterectomy, vascular imaging

Carotid endarterectomy (CEA) is performed to prevent recurrent neurological symptoms and stroke in patients with significant internal carotid artery (ICA) stenosis. The efficacy of this operation has been established by several randomized clinical trials [1-4]. In high-grade, symptomatic ICA stenosis ($\geq 70\%$), the benefit from CEA is very significant and increases dramatically as the stenosis increases up to 95% [1-3]. However, the treatment of symptomatic ICA occlusion is still somewhat controversial. Despite optimal medical management, ICA occlusion is associated with increased ipsilateral recurrent stroke rates of 6% to 20% per year [5-7]. Although

the early attempts to restore blood flow after acute occlusion of the ICA often ended in catastrophic results, most likely due to postoperative intracranial hemorrhage [8, 9], some authors have recently reported beneficial effects of CEA in these patients [5, 10, 11].

In this small study, we retrospectively analyzed our results of CEA for the treatment of symptomatic ICA occlusion to evaluate its feasibility, safety, and outcomes and to determine the indications of this operation, based on imaging studies.

Materials and Methods

This is a retrospective study of medical records. From January 2000 to May 2009, 564 carotid surgeries were performed for ICA occlusive disease in our

Received November 16, 2010; accepted February 14, 2011.
*Corresponding author. Phone: +82-2-3010-5039; Fax: +82-2-474-9027
E-mail: ypcho@amc.seoul.kr (Cho YP)

department. Of these, 9 consecutive patients who underwent operative exploration with attempted CEA for symptomatic ICA occlusion were included in this study.

Preoperative diagnosis of the ICA occlusion was made by color-flow duplex ultrasound and magnetic resonance (MR) and/or conventional digital subtraction angiography. Patients were evaluated pre-operatively by a consultant neurologist using MR imaging to assess the presence and severity of stroke. Neurological status was based on the Modified Rankin Scale, and improvement or deterioration was defined as a change in the Modified Rankin Scale ± 1 [10]. In our department, the indications for operative carotid artery exploration based on neurological status and MR imaging were recurrent transient ischemic attacks, progressing stroke with ipsilateral cerebral infarction limited to not more than one-third of the middle cerebral artery territory, and also fixed, small neurological deficits with a severe perfusion deficit; the indications based on vascular imaging were segmental occlusion of the proximal ICA and extensive occlusion from the bulb to the distal ICA in selected patients in whom color-flow duplex ultrasound showed a poorly echogenic or anechoic thrombus with a flow void, suggestive of a recently formed thrombus. Patients with ICA occlusion after CEA; trauma- or vasculitis-related, severe neurological deficits; asymptomatic or chronic occlusions; or an ICA occlusion due to a tight stenosis of the intracranial portion of the ICA with a thrombus propagating from the skull base towards the bifurcation as suggested by preoperative imaging studies were excluded from this study. All patients with ICA occlusion had symptoms referable to the ipsilateral cerebral hemisphere and underwent operative carotid artery exploration within 1 week of the diagnosis (mean, 3 days, range, 0–7 days). Decision-making on elective or urgent surgery was made on a case-by-case basis according to the preoperative neurological status and imaging findings.

The surgical procedures were preferably carried out under regional cervical block (superficial and deep), and general anesthesia with endotracheal intubation was selectively used in patients who were neurologically unstable and may not have tolerated cervical block. Intra-operative blood pressure was monitored by arterial line as well as by pneumatic cuff. Systemic heparinization was given to all patients

(5,000 IU, i.v.) prior to carotid clamping. In patients with regional cervical block, the ability to tolerate cross-clamping was assessed by evaluating the level of consciousness and motor function such as by counting numbers and squeezing a squeaking rubber doll placed in the contralateral hand. The administration of all sedatives was withheld until an assessment of tolerance to carotid cross-clamping was completed. When the patients developed speech or motor dysfunction, a Javid™ carotid shunt (Bard, Inc., Murray Hill, NJ, USA) was inserted. In selected patients with general anesthesia, a Javid™ carotid shunt was used routinely. Routine patch angioplasty was performed with optical $\times 3.5$ power magnification and Prolene 5/0 and 6/0 continuous sutures. Postoperatively, all patients were given antiplatelet therapy (cilostazol or aspirin) in combination with stringent control of blood pressure and close observation in an intensive care unit for at least 24 h, and they were followed up both clinically and by color-flow duplex ultrasound and/or MR imaging with angiography before discharge, and then by color-flow duplex ultrasound every 6 months.

In this study, ICA occlusion was confirmed at surgical exploration, and CEA was attempted with the standard technique. After CEA, when thrombus was present in the distal ICA or ICA back-bleeding was not observed, a No. 3 Fogarty embolectomy catheter was carefully used for sequential ICA thrombectomy.

Results

The clinical details of the 9 consecutive patients receiving CEA for symptomatic complete occlusion of the ICA are summarized in Table 1.

Patients. We retrospectively reviewed the medical records of the 9 consecutive patients who underwent operative carotid artery exploration with attempted CEA for symptomatic ICA occlusion. These patients included 7 males and 2 females with a mean age of 66 years (range, 55–73 years). Comorbid risk factors in these patients included prior cerebral vascular events in 5 patients, hypertension in 8, diabetes mellitus in 2, smoking in 6, and hyperlipidemia in 3. Presenting symptoms, referable to the ipsilateral cerebral hemisphere, at the time of admission included recurrent transient ischemic attacks in 1 patient, progressing stroke in 5, and fixed, small neurological deficits in 3. The Modified Rankin Scales

Table 1 Clinical details of 9 consecutive patients receiving carotid endarterectomy for symptomatic complete occlusion of the internal carotid artery

Case	Sex/Age	Symptom	Duration [†] (days)	Imaging findings	Fogarty thrombectomy	Changes of MRS [‡]
1	M/60	transient ischemic attacks	10	segmental occlusion	not done	improved
2	M/62	fixed, small neurological deficits	17	segmental occlusion	not done	unchanged
3	F/67	fixed, small neurological deficits	2	extensive occlusion	done	improved
4	M/64	progressing stroke	7	segmental occlusion	done	unchanged
5	M/69	fixed, small neurological deficits	5	extensive occlusion	done	improved
6	M/71	progressing stroke	1	segmental occlusion	not done	improved
7	M/55	progressing stroke	13	segmental occlusion	not done	improved
8	M/73	progressing stroke	1	extensive occlusion	done	improved
9	F/73	progressing stroke	0	segmental occlusion	not done	unchanged

[†]Duration means the time interval from the onset of the last neurological symptoms to the carotid artery exploration.

[‡]Modified Rankin Scales evaluated at 3 to 6 months postoperatively.

Segmental occlusion, segmental occlusion of the proximal internal carotid artery; Extensive occlusion, thrombus propagating from the bulb to the distal internal carotid artery.

were 0 to 2 in 3 patients and 3 to 5 in the other 6 patients.

Preoperative imaging study. In this study, diagnostic imaging varied over the 9-year study period. Preoperative MR imaging was obtained in all 9 patients. MR imaging exclusion criteria for CEA include evidence of cerebral hemorrhage, significant cerebral edema, hemispheric asymmetry, or cerebral infarction involving more than one-third of the middle cerebral artery territory. All patients had evidence of acute or subacute infarction on MR imaging. Preoperative vascular imaging was obtained by means of MR angiography in all 9 patients, color-flow duplex ultrasound in 8, and conventional digital subtraction angiography in 5. Vascular imaging exclusion criteria for CEA include evidence of supraclinoid ICA occlusion and chronic occlusion. Thrombus that appears retracted and markedly echogenic is undoubtedly chronic, and patients with a small cord-like ICA, in which the ICA is disproportionately smaller than the common carotid artery or external carotid artery, are likely to have a chronic occluded ICA. In this study, vascular imaging showed segmental occlusion of the proximal ICA in 6 patients (Fig. 1) and extensive occlusion from the bulb to the distal ICA in 3 patients, in whom color-flow duplex ultrasound showed a poorly echogenic or anechoic thrombus with a flow void, suggestive of an acute thrombus (Fig. 2). Recently, MR imaging with diffusion-weighted and perfusion-weighted imaging was performed to evaluate the potential benefit of CEA in 5 patients with symptomatic ICA occlusion. The perfusion-weighted imag-

ing/diffusion-weighted imaging volume mismatch is one of the most important criteria for selecting patients for CEA in symptomatic ICA occlusion and was noted in 4 patients. According to the degree of the contralateral ICA stenosis, 1 patient showed complete occlusion, 2 severe (70–99%), 5 moderate (50–69%), and 1 mild (30–49%) stenosis.

Surgery. Urgent surgery within 72h after the onset of the last neurological symptoms was done in 4 patients and elective surgery in 5. The mean time interval from the onset of the last neurological symptoms to carotid artery exploration was 6 days (range, 0–17 days): within 3 days in 4 patients, 1 week in 2, 2 weeks in 2, and 1 month in 1. Four patients underwent operative carotid artery exploration under regional cervical block; the remainder required general anesthesia. After surgical exposure and opening of the carotid bifurcation, complete ICA occlusion was confirmed in all 9 patients. Carotid shunting was routinely used in 5 patients with general anesthesia after the removal of the distal thrombus and the restoration of sufficient ICA back bleeding. In all 9 patients, routine patch angioplasty was performed with an autogenous patch from the great saphenous vein in 2 patients and a bovine pericardium patch (Bio-Vascular Inc., Saint Paul, Minn, USA) in 7. The use of bovine pericardium for carotid patch angioplasty proved to be safe and highly durable and can reduce the total operating time in comparison to an autogenous patch in these critical patients [12, 13]. CEA was performed successfully to restore blood flow in all 9 patients: CEA only in 5 and CEA with Fogarty

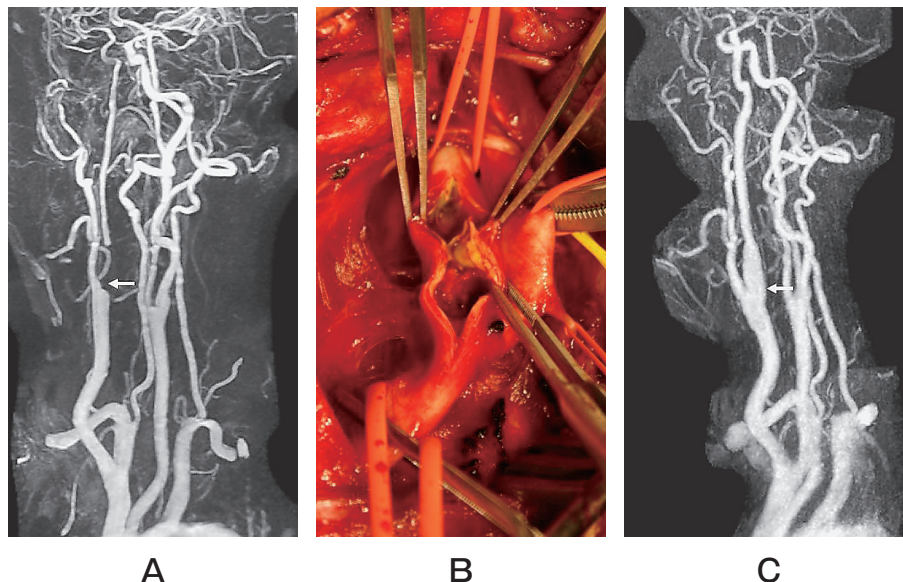


Fig. 1 A 55-year-old man presented with a 1-month history of gradually worsening neurological deficit. **A**, Preoperative magnetic resonance (MR) angiography showed segmental occlusion (arrow) of the right proximal internal carotid artery (ICA); **B**, Complete ICA occlusion was confirmed after surgical exposure and opening of the carotid bifurcation. Note that there was no backflow from the ICA without cross-clamping; **C**, Postoperative MR angiography showed restoration of blood flow (arrow) in the right ICA.

thrombectomy in 4. Staged bilateral CEAs were performed in 2 patients with contralateral high-grade ICA stenosis.

Follow-up data. The immediate postoperative Modified Rankin Scales showed improvement in 4 patients, no change in 4, and deterioration in 1. One patient with failed carotid artery stent placement due to the failure of the delivery of a distal anti-embolic device beyond the filling defect who received CEA with Fogarty thrombectomy successfully deteriorated because of hyperperfusion syndrome. Postoperative MR angiography confirmed that revascularization had been successful in all 9 patients who received CEA, and MR imaging displayed improved perfusion in 4 patients and hemorrhagic transformation of the pre-existing infarction without clinical consequences in 1. At 3 to 6 months postoperatively, 6 of 9 patients showed improvement in Modified Rankin Scales and 3 of 9 were unchanged. There was no postoperative major stroke or mortality in these patients during a mean follow-up of 54 months (range, 15–117 months).

Discussion

The pattern of progression of carotid stenosis is unpredictable, and the disease may progress swiftly

or slowly or remain stable for many years. Even though modern medical treatment can diminish the progress of the disease and protect against stroke to a certain extent, CEA is still the standard revascularization therapy in carotid occlusive disease. The efficacy of CEA has been proven in high-grade ICA stenosis by several randomized clinical trials [1–3]. Recently, the European Society for Vascular Surgery produced updated guidelines for the invasive treatment of carotid disease [14]. According to these updated guidelines, CEA is recommended in symptomatic patients with > 50% stenosis if the perioperative stroke/death rate is < 6%, preferably within 2 weeks of the patient's last symptoms. However, optimal treatment of symptomatic ICA occlusion is still somewhat controversial.

Extracranial ICA occlusion resulting in ischemic stroke is different from other forms of occlusion of the intracranial blood vessels [15]. The occluded segments of the intracranial arteries, such as the middle cerebral artery, usually consist of an occlusive embolus or a short length of thrombus in a normal vessel, whereas the occlusion of the extracranial ICA consists of predominantly atherosclerotic plaque at the carotid bifurcation and a superimposed thrombus [11, 15]. The pathophysiology of ischemic stroke due to

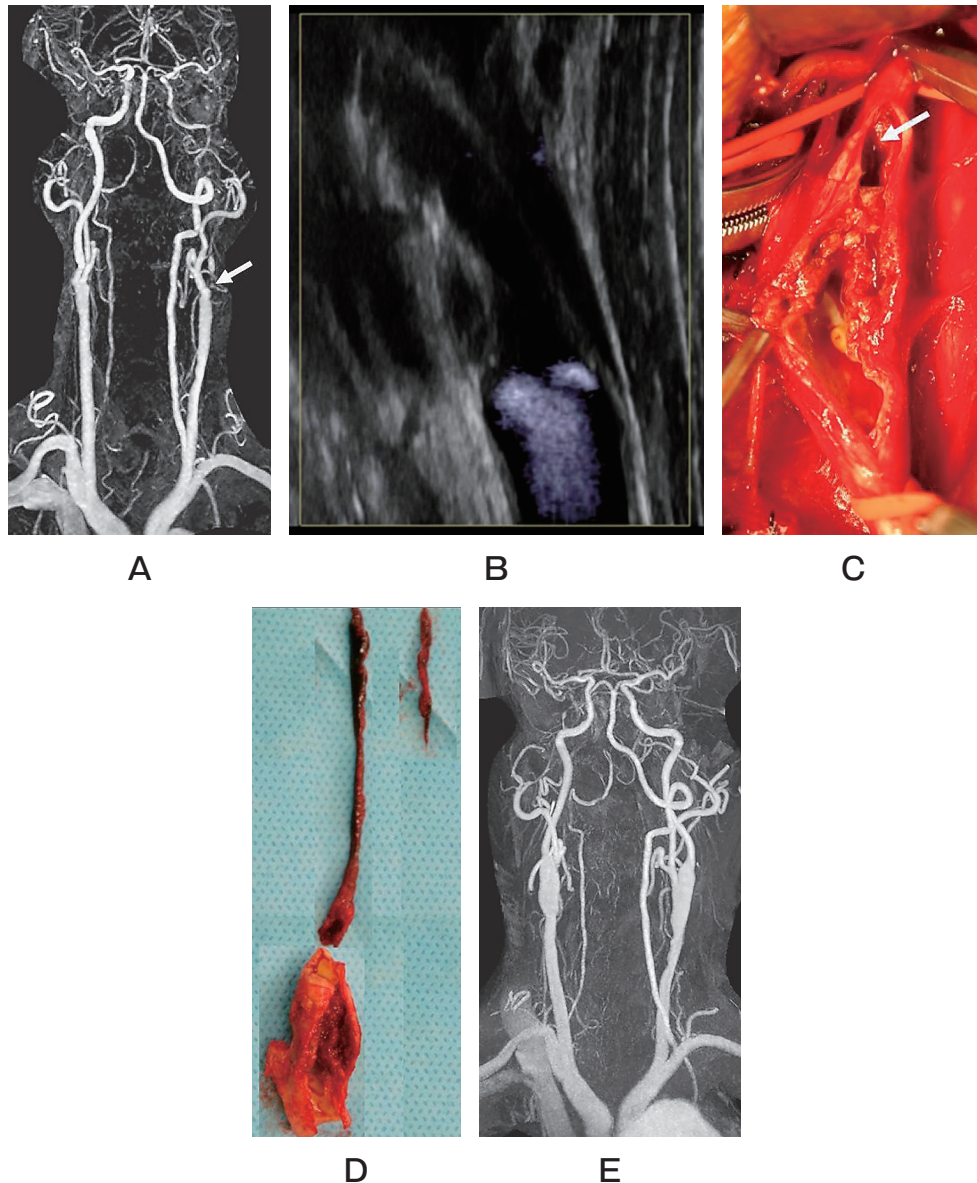


Fig. 2 A 69-year-old man presented with recurrent transient ischemic attacks. **A**, Preoperative magnetic resonance (MR) angiography showed extensive occlusion from the bulb to the distal internal carotid artery (ICA) (arrow) of the left ICA; **B**, Preoperative color-flow duplex ultrasound showed a poorly echogenic thrombus with a flow void, suggestive of a recently formed thrombus; **C**, Intraoperative photograph showed complete ICA occlusion with a superimposed distal ICA thrombus (arrow); **D**, Note the surgical specimen after carotid endarterectomy (CEA) with Fogarty thrombectomy; **E**, After staged bilateral CEAs were performed, postoperative MR angiography showed restoration of blood flow in both ICAs.

acute and subacute ICA occlusion depends not only on cerebral hypoperfusion but also on distal embolization from an occluded ICA. Although the best medical treatments with anti-platelet and anticoagulant therapy might help reduce the propagation of thromboemboli, persistent cerebral hypoperfusion and atherosclerotic

plaques can lead to increased risk of recurrent stroke without surgical revascularization [5-7]. However, the early attempts to restore blood flow after occlusion of the ICA have shown dismal results, most likely due to postoperative intracranial hemorrhage [8, 9], and a randomized clinical trial of extracranial-intra-

ranial bypass surgery failed to document any benefit in symptomatic ICA occlusions [16, 17]. According to the guidelines for CEA from the *ad hoc* committee, AHA, most patients who have a neurological deficit associated with acute ICA occlusion are not candidates for CEA because of the severity of their deficits and/or a delay in diagnosis, but decisions must be made on a case-by-case basis; there may be an opportunity to save ICA patency and neurological function with CEA in carefully selected patients [18].

There are 2 important prerequisites for successful CEA in patients with symptomatic ICA occlusion: (1) visualization of the distal ICA and (2) viability and magnitude of the brain tissue at risk. ICA occlusions usually occur at a point of critical stenosis in the carotid bifurcation and proximal ICA. However, it should be noted that there is frequently an additional occlusion of the distal ICA, in particular in the supraclinoid segment. If an ICA occlusion develops from a tight stenosis of the intracranial portion of the ICA with a thrombus propagating from the skull base towards the bifurcation, CEA cannot be successful. Surgical revascularization can only be achieved if the occlusion is limited to the extracranial ICA [11]; therefore, visualization to confirm the patency of the distal vessel is essential before performing surgical revascularization. In patients with a superimposed distal ICA thrombus, the age of the distal ICA thrombus is another important factor in performing successful CEA. The occluding thrombus usually originates from the carotid bifurcation and propagates cephalad to where patency of the ICA can be maintained by collateral flow, commonly occurring at the level of the ophthalmic artery [10]. The thrombus causes inflammation of the arterial wall, and organization of the thrombus begins within days after arterial occlusion. Color-flow duplex ultrasound has become the method of investigation of choice for the assessment and monitoring of ICA stenosis. Its role in identifying complete occlusion is well documented. Although the ability of duplex ultrasound to detect arterial thrombus and to differentiate between thrombus and plaque has not been investigated, it has been determined based on duplex ultrasound experience with venous thrombosis that the echogenicity of the thrombus varies greatly depending on its age. A recently formed thrombus (hours to a few days old) is not very echogenic and may be difficult to identify if a flow void

cannot be appreciated on the color-flow duplex ultrasound image. The thrombus becomes more echogenic over time as it retracts, is invaded by fibroblasts, and becomes organized into fibrous tissue, and this change in echogenicity permits the age of thrombus to be approximated. Therefore, if vascular imaging studies show segmental occlusion of the proximal ICA or extensive occlusion from the bulb to the distal ICA with a recently formed distal ICA thrombus on a duplex ultrasound image, internal CEA will be feasible and is associated with limited morbidity and mortality. Patients with ICA occlusions can have a variable presentation ranging from asymptomatic to profound neurological impairment and death, according to the collateral flow filling the cerebral arteries of the affected hemisphere. In addition, long-term patient neurological outcome is unpredictable. In acute ICA occlusion, neurological disorder occurs either due to thromboembolism while the occluding thrombus is propagating or to hypoperfusion of the dependent hemisphere in patients with insufficient collateral blood flow [11]. MR imaging with diffusion-weighted and perfusion-weighted imaging may help to differentiate between ischemic brain tissue that is definitively and irreversibly damaged and "the brain tissue at risk," hypoperfused but still viable, which may be salvaged by early revascularization. The clinical outcome after CEA in patients with symptomatic ICA occlusion is also dependent on the viability and magnitude of the brain tissue at risk.

Recently, the endovascular interventional approach was found to be an effective treatment modality for acute or subacute ICA occlusion and deteriorating neurological symptoms in selected patients [19]. In contrast to the surgical approach, the endovascular approach affords the opportunity to define the length of the occluded ICA. Additionally, it also allows treatment of the occluded intracranial segment in patients with tandem occlusions. However, presently there is no evidence that the endovascular interventional approach provides better results in the treatment of symptomatic complete occlusion of the ICA compared with the surgical approach, and prospective, randomized studies may be required to confirm the efficacy of the endovascular approach in these difficult cases.

Although this study was a small-volume, retrospective study, our results indicate that the use of

internal CEA for the treatment of acute and subacute symptomatic ICA occlusion is feasible with limited morbidity and mortality if strict selection criteria, on the basis of neurological status and modern cerebral imaging techniques, are used, and successful surgical revascularization can improve cerebral perfusion and prevent recurrent stroke due to distal embolization in patients with symptomatic complete occlusion of the ICA. Despite the lack of a generalized efficacy of surgical revascularization for symptomatic ICA occlusion, the present report provides the initial data required to pursue and optimize a new strategy for the treatment of these patients. A randomized prospective trial is needed to define the true natural history of symptomatic ICA occlusion and the potential benefits of CEA in these patients.

References

1. North American Symptomatic Carotid Endarterectomy Trial Collaborators: Beneficial effects of carotid endarterectomy in symptomatic patients with high-grade stenosis. *N Engl J Med* (1991) 325: 445-453.
2. European Carotid Surgery Trialists' Collaborative Group: MRC European Carotid Surgical Trial: interim results for symptomatic patients with severe (70-99%) or with mild (0-29%) carotid stenosis. *Lancet* (1991) 337: 1235-1243.
3. Ferguson GG, Eliasziw M, Barr HWK, Clagett GP, Barnes RW, Wallace MC, Taylor DW, Haynes RB, Finan JW, Hachinski VC and Barnett HJM: The North American Symptomatic Carotid Endarterectomy Trial: surgical results in 1415 patients. *Stroke* (1999) 30: 1751-1758.
4. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study: Endarterectomy for asymptomatic carotid artery stenosis. *JAMA* (1995) 273: 1421-1428.
5. Paty PSK, Adeniyi JA, Mehta M, Darling III RC, Chang BB, Kreienberg PB, Ozsvath KJ, Roddy SP and Shah DM: Surgical treatment of internal carotid artery occlusion. *J Vasc Surg* (2003) 37: 785-788.
6. Grubb RL and Powers WJ: Risk of stroke and current indications for cerebral revascularization in patients with carotid occlusion. *Neurosurg Clin N Am* (2001) 12: 473-487.
7. Klijn CJ, Van Buren PA, Kappelle LJ, Tulleken CA, Eikelboom BC, Algara A and van Gijn J: Outcome in patients with symptomatic occlusion of the internal carotid artery. *Eur J Vasc Endovasc Surg* (2000) 19: 579-586.
8. Rob CG: Operation for acute completed stroke due to thrombosis of the internal carotid artery. *Surgery* (1969) 65: 862-865.
9. Wylie EJ, Hein MF and Adams JE: Intracranial hemorrhage following surgical revascularization for treatment of acute stroke. *J Neurosurg* (1964) 21: 212-215.
10. Kasper GC, Wladis AR, Lohr JM, Roedersheimer LR, Reed RL, Miller TJ and Welling RE: Carotid thromboarterectomy for recent total occlusion of the internal carotid artery. *J Vasc Surg* (2001) 33: 242-249.
11. Weis-Müller BT, Huber R, Spivak-Dats A, Turowski B, Siebler M and Sandmann W: Symptomatic acute occlusion of the internal carotid artery: Reappraisal of urgent vascular reconstruction based on current stroke imaging. *J Vasc Surg* (2008) 47: 752-759.
12. Biasi GM, Mingazzini P, Baronio L and Sampaola A: Processed bovine pericardium as patch angioplasty for carotid endarterectomy: a preliminary report. *Cardiovasc Surg* (1996) 4: 848-852.
13. Kim GE, Kwon TW, Cho YP, Kim DK and Kim HS: Carotid endarterectomy with bovine patch angioplasty: a preliminary report. *Cardiovasc Surg* (2001) 9: 458-462.
14. Liapis CD, Bell PRF, Mikhailidis D, Sivenius J, Nicolaidis A, Fernandes e Fernandes J, Biasi G and Norgren L, on behalf of the ESVS Guidelines Collaborators: ESVS guidelines. Invasive treatment for carotid stenosis: indications, techniques. *Eur J Vasc Endovasc Surg* (2009) 37: S1-S19.
15. Qureshi AI: Endovascular revascularization of symptomatic internal carotid artery occlusion. *Stroke* (2005) 36: 2335-2336.
16. EC/IC Bypass Study Group: Failure of extracranial-intracranial arterial bypass to reduce the risk of ischemic stroke. Results of an international randomized trial. *N Engl J Med* (1985) 313: 1191-1200.
17. Fluri F, Engelter S and Lyrer P: Extracranial-intracranial arterial bypass surgery for occlusive carotid artery disease (Review). *Cochrane Database Syst Rev* (2010) 17: CD005953.
18. Moore WS, Barnett HJM, Beebe HG, Bernstein EF, Brener BJ, Brott T, Caplan LR, Day A, Goldstone J, Hobson RW 2nd, et al.: Guidelines for carotid endarterectomy. A multidisciplinary consensus statement from the ad hoc Committee, American Heart Association. *Stroke* (1995) 26: 188-201.
19. Jovin TG, Gupta R, Uchino K, Jungreis CA, Wechsler LR, Hammer MD, Tayal A and Horowitz MB: Endovascular stenting of extracranial internal carotid artery occlusion in acute stroke has a high revascularization rate. *Stroke* (2005) 36: 2426-2430.