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Early Haemodynamic Changes in the Ophthalmic Artery, Siphon and Intracranial Arteries After Carotid Endarterectomy Estimated by Transcranial Doppler and Duplex Scanning

V. Zbornikova and L. Skoglund

Department of Neurology, University Hospital, Linköping, Sweden

Objectives: *To study early haemodynamic changes in connection with carotid endarterectomy (CE).*
Methods: *Sixty-three consecutive patients, average age 64, with symptomatic stenosis in the internal carotid artery (ICA) ≥70% were examined clinically and by transcranial Doppler (TCD) 1 day before and within 48 h after CE. Duplex scanning of extracranial vessels was performed within 1 week after CE.*
Results: *After CE, all retrograde systolic velocities (SV) in the ophthalmic artery (OA) and 9/10 retrograde mean velocities (MV) in the siphon changed to antegrade. Antegrade SV in the OA increased significantly (p<0.001) only on the operated side. SV in the OA on the operated side correlated (p<0.05) with MV in the siphon, and pulsatility index (PI) in the middle cerebral artery (MCA, p<0.001). MV in the MCA increased from 46±12 cm/s to 59±21 cm/s after CE and in the ACA with normal flow from 54±19 cm/s to 62±28 cm/s (p<0.001 and <0.05, respectively) only on the operated side. Stump pressure correlated (p<0.01) with SV in the OA and PI in the MCA and was higher (59±16 mmHg, p<0.01) in the group with antegrade flow in the OA compared to the group with retrograde flow in the OA (43±15 mmHg).*
Conclusion: *TCD and duplex gives important early information about patency of the ICA and haemodynamic intracranial changes after CE.*

Key Words: Thrombendarterectomy; Carotid artery; Transcranial Doppler; Duplex scan; Stump pressure.

Introduction

Carotid endarterectomy (CE) is accepted as preventing further embolisation from stenotic arteries in patients with symptoms, but the positive haemodynamic effect of operation is still questioned. Recurrent stenosis due to either myointimal hyperplasia or atherosclerosis has been reported in 12–30% of CE.[1–3] In 113 patients we found 14 occlusions and 13 restenoses diagnosed by duplex scanning during a long-term postoperative follow-up, with a significantly higher frequency of new neurological symptoms in comparison to vessels free from stenosis.[4] Most occlusions occurred in the early postoperative period.[5] As about half of the occlusions develop without symptoms,[6] clinical examination alone is unreliable in diagnosing occlusion. Transcranial Doppler (TCD) may help to give information about haemodynamic disturbances within the carotid territory and function of collaterals within the circle of Willis,[7] which is sometimes impossible by aortic arch angiography. Earlier reports, using TCD before and after carotid endarterectomy (CE), have concentrated on changes of mean velocity (MV) and pulsatility index (PI) in the middle cerebral artery (MCA).[8–11] Haemodynamic changes in the anterior cerebral artery (ACA), posterior cerebral artery (PCA), ophthalmic artery (OA) and siphon in combination with duplex scanning in the perioperative period have rarely been examined. The objective of the present investigation was to study the intracranial haemodynamics before and early after operation. Furthermore, the value of combined examination with TCD and duplex scanning in the diagnosis of postoperative occlusion was evaluated.

Patients and Methods

Sixty-three consecutive patients (46 men and 17 women; mean age 64, range 42–80), with symptomatic stenosis of ≥70% in the internal carotid artery (ICA) were operated on during the period June 1991–May 1994. Three patients underwent bilateral operation, and thus 66 arteries were available for evaluation. The indication for operation was hemispheric transient

* Please address all correspondence to: V. Zbornikova, Department of Neurology, University of Health Sciences, S-581 85 Linköping, Sweden.

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ischaemic attacks (TIA) in 20 patients, amaurosis fugax (AF) in 22, transient global amnesia in one and minor stroke in 19. One patient with bilateral asymptomatic stenoses was operated on because of a disturbing bruit. All patients with minor stroke underwent computed tomography (CT) scan preoperatively.

All patients underwent duplex scanning. Aortic arch angiography using a pig-tail catheter was performed before CE except in two patients, in whom magnetic resonance angiography was used. The degree of stenosis was estimated as described in European Carotid Surgery Trial.[12] The cervical arteries were visualised in four planes (anteroposterior, lateral, right and left oblique views).

Most patients were treated with oral anticoagulants until operation. Neurological examination and transcranial Doppler were performed one day before and within 48 h after operation. In two patients postoperative TCD examination was performed after 1 week. On the contralateral side stenosis of 55–69% was found in six, stenosis of ≥70% in seven, and occlusion in nine vessels. Thus, 16 patients with bilateral high degree lesions ≥70% (group B) were compared with patients having only a unilateral stenosis of ≥70% on the operated side (group A). All patients were followed-up for at least 12 months.

Ultrasonic examination

Duplex. Extracranial vessels were examined by duplex scanner with 5 MHz pulsed Doppler and 7.5–7 MHz echo image (ATL Mark 500 and Acuson XP, U.S.A.) preoperatively and within the first week after CE. Examination was repeated at 3 months, 6 months, and 1 year after operation. The degree of stenosis was calculated according to the equation:
% stenosis = 45.8 + 7.19* peak systolic velocity (m/s) + 4.8* end diastolic velocity (m/s) + 7.2 periorbital Doppler (coded as normal = 0 and 1 = abnormal).[13] Restenosis was defined as a stenosis of >50%.

Transcranial Doppler. The examination was performed under resting conditions with patients lying supine with closed eyes. Blood pressure (BP) was measured before the examination, after 5 min rest. The OA and distal part of the ICA in the siphon were examined by handheld probe with 2 mHz transducer (TC 64 B, Uberlingen, Germany). Signals from the OA were registered at a depth of 40–45 mm and from the siphon at a depth of 60–70 mm just below the origin of the OA.[14]

Intracranial vessels were examined preoperatively and in most patients the first day postoperatively by three-dimensional transcranial Doppler (3-D TCD, EME, Uberlingen, Germany), each recording being displayed in frontal, horizontal, and lateral projections and colour coded for velocity and flow direction parallel with spectral curves. The position of the sample volume is indicated by a white circle on the xyz coordinates.[15] In only 10 patients before operation and in 21 bed-bound patients postoperative examination was performed by TC 64 B with the same 2 mHz transducer. Peak systolic velocity (SV) in the OA, and MV in the siphon and intracranial vessels were registered. The PI according to Gosling[16] was calculated manually.

Age-matched normal people, mean age 63 ± 7 years with normal angiogram (16 sides in MCA and 10 sides in ACA), served for comparison of the anterior circulation[17]; evaluation of the posterior circulation was based on values from 11 patients (eight healthy volunteers and three patients with normal aortic arch angiography, together 19 sides), mean age 40 ± 14 (S.D.). Normal values of MV in the MCA were of 56 ± 9 cm/s, in the ACA of 53 ± 19 cm/s and in the PCA of 39 ± 8 cm/s. Normal values for systolic velocities in the OA and MV in the siphon were obtained from 10 patients (20 sides), mean age 51 ± 12 years, who had earlier been successfully operated on for aneurysm and all showed normal extracranial and intracranial ICA on selective carotid angiography.[17] Examination was performed at least 2 months after the subarachnoid haemorrhage and operation.

The function of supplying collaterals was evaluated by brief compression of the ipsilateral and contralateral CCA low in the neck, as previously described.[18] Dominant external collaterals through the OA were considered in the presence of retrograde flow in the OA. A weak, undetectable signal in the OA was called isoelectric flow.[17] Supplying collaterals from the contralateral ACA were defined as reversed flow on the side to be operated on, which diminished or changed to normal on compression of the contralateral CCA.[17] If MV in the PCA on the operated side was >55 cm/s and increased by at least 10% at compression of ipsilateral CCA, the PCA was judged as supplying collateral.

The reconstruction was considered patent, if retrograde or isoelectric flow[17] in the OA changed to antegrade after CE and if a reversed flow in the ACA changed to normal direction. In patients with antegrade flow, the reconstruction was considered patent if flow direction did not change.

Operation

The operation was performed under general anaesthesia and with the use of a shunt. Vein patch angioplasty was performed in all except 11 patients. Stump pressure was measured using an electronic manometer (fy) proximal to the stenosis in the CCA after the cross-clamping.

Statistical methods

Student's two-tailed paired *t*-test was used for comparison of preoperative and postoperative mean values and differences between operated and non-operated sides. An unpaired two-tailed *t*-test was performed for comparison of MV and PI between groups A and B in different vessels; and MV differences in the MCA in patients with TIA, and minor stroke and normal people; for comparison of stump pressure in the group with antegrade and retrograde flow direction in the OA and for comparison of PI in the MCA ipsilateral to retrograde or antegrade flow in the OA. Correlation of stump pressure and SV in the OA with MV and PI in vessels within the same carotid territory and correlation of MV and PI in the open ICA with antegrade flow to the MCA on the operated and non-operated sides were studied by series of single regression analyses. Changes of flow direction in the siphon on the operated side were tested by the Chi-squared test.

Results

Clinical

One patient developed retinal embolisation, and three had a minor stroke without residual functional disability within 30 days of surgery. No patient with postoperative stroke had occlusion on the operated side. One minor stroke, with signs of dysphasia and central facial paresis on the operated side, developed in a patient with preoperative minor stroke within the same vascular territory. The operated ICA was patent, as it was also in the patient with retinal embolus. Two patients had minor stroke with hemilateral symptoms on the non-operated side referrable to preoperatively confirmed occlusion of the ICA. CT within 24 h after new signs showed an old infarction. Four patients had TIA. No patient had major ischaemic stroke. One patient, in whom the clinical state, postoperative duplex and TCD were normal, died at home of intracerebral haemorrhage 12 days after an uneventful operation. All other patients were free from symptoms. Thus, the perioperative morbidity was 6.3% and the mortality 1.6%.

Duplex scanning

Postoperative duplex scanning revealed three occlusions: one was associated with amaurosis fugax and two silent. Restenosis of >75% with retrograde ophthalmic flow was found 3 days after CEA in one woman. On examination 10 days after operation, the periorbital flow was antegrade and stenosis 75%. A decrease to 65–70% in lumen diameter was noted at 6 and 12 months.

Transcranial Doppler

Ophthalmic artery. Ophthalmic collaterals with reverse flow on the operated side were found in 17 patients before CE. Eleven patients had isoelectric flow with no detectable signals. These flow patterns were judged as pathological and were seen in 57% of patients. In 21 patients the flow direction was antegrade, but slower ($p<0.001$) than in normal subjects (Table 1). In the remainder, transorbital examination was not available. Postoperatively, all retrograde velocities changed to antegrade, and isoelectric velocities became measurable except for one postoperative occlusion with an undetectable signal (Table 1). Antegrade velocities increased significantly ($p<0.001$) after operation except in one patient with postoperative occlusion, in whom no signal could be registered. Another patient with occlusion after 5 weeks had antegrade SV of 20 cm/s in the OA before surgery, which increased to 70 cm/s in normal antegrade direction after CE, but the flow signal was unmeasurable at 5 weeks.

According to regression analysis, significant correlations were found of SV in the OA to MV in the siphon ($r=0.474$, $p<0.05$) and PI in the MCA ($r=0.557$, $p<0.001$, Fig. 1) on the operated side.

On the non-operated side antegrade SV did not change, whilst the retrograde SV, corresponding to occlusion, slightly decreased (Table 1).

Siphon. In the distal part of the ICA (siphon) on both sides the preoperative antegrade MVs were slower ($p<0.01$) than at postoperative examination, when a bilateral increase in MV was registered (Table 1), except for a decrease in normal direction in a woman with postoperative intimal flap causing stenosis 75%. On the operated side in nine vessels, supplied by the OA, MV in the same retrograde direction were as in this artery and these changed to normal pattern after surgery (Table 1). In one patient with normal flow in the OA, flow direction in the siphon was retrograde pre- and postoperatively, because measurement was done

Table 1. Systolic velocities (SV) in the ophthalmic artery (OA), mean velocities (MV) in the siphon, and pulsatility index (PI) (a) before and (b) after operation. Antero = anterograde, retro = retrograde, iso = isolectric flow direction.

	Operated			Non-operated			Normal
SV in OA							
	Antero	Retro	Iso	Antero	Retro	Iso	Antero
n	21	17	10	32	4	2	20
	(*** Operated antero vs Normal)						
(a)	30.45 ±10.92	−38.17 ±20.01	0	37.55 ±10.70	−58.50 ±31.46	0	42.95 ±10.63
	***						
(b)	47.38 ±14.23	38.32 ±15.87	40.40# ±19.86	37.38 ±10.53	−52.25 ±18.41		
PI in OA							
(a)	1.35 ±0.51	0.97 ±0.47	0	1.53 ±0.64	0.79 ±0.20	0	1.31 ±0.47
	*	**					
(b)	1.89 ±0.94	1.94 ±0.63	1.74 ±0.68	1.55 ±0.50	0.93 ±0.13	0	
MV in siphon							
n	44	9	4	51	2	4	20
(a)	35.00 ±12.70	−34.62 ±22.40	0	36.08 ±14.34	40.63 ±16.89	0	42.35 ±13.23
	**			**			
(b)	41.14 ±16.18	36.53 ±15.00	42.60 ±17.50	±42.68 ±14.81	40.08 ±16.08	30.50 ±13.60	
PI in siphon							
	(* Operated antero vs Non-operated antero)						
(a)	0.91 ±0.39	0.80 ±0.27	0	1.08 ±0.27	0.71 ±0.13	0	1.00 ±0.26
	*						
(b)	1.04 ±0.30	1.14 ±0.40	1.16 ±0.36	1.08 ±0.33	1.00 ±0.40	0.77 ±0.07	

* = p 0.05; ** = p<0.01; *** = p<0.001. # = Except one patient with postoperative retrograde flow corresponding to stenosis of 75%.

above the genum of the elongated siphon. In four vessels with unmeasurable flow before operation, it was possible to register normal signals after operation. Changes in flow pattern differed significantly (p<0.01, Chi-squared = 7.45) from those on the non-operated side, although increase in antegrade flow, supplied by the same ICA, was observed on both sides.

On the non-operated side in two patients with retrograde flow in the siphon, supplied by the OA in the same direction ipsilateral to occlusion of the ICA, the flow direction remained unchanged. In four vessels with unmeasurable signals before CE, normal signals were recorded after operation. Two patients with early postoperative occlusion showed no change in antegrade velocities in the siphon. In the third patient, with occlusion after 5 weeks, postoperative increase in MV in the siphon was noted, which was supplied by the contralateral ACA. PI in the siphon was lower (p<0.05) preoperatively on the operated side than PI on the non-operated side, and increased significantly (p<0.05) only on the operated side. No difference in PI between sides was found after operation.

On the operated side, preoperative MV and PI in the siphon in an antegrade direction did not correlate with MV and PI in the ipsilateral MCA, but after CE a significant correlation was found according to linear regression analysis (r = 0.327, p<0.05 and r = 0.435, p<0.01 respectively, Figs 2(a,b) and 3(a,b). No correlation emerged between MVs in the siphon and the MCA on the non-operated side pre- or postoperatively (Fig

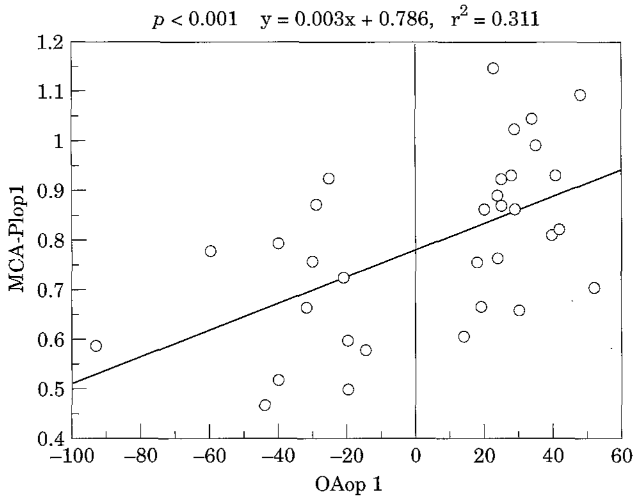

Fig. 1. Correlation of systolic velocity in the ophthalmic artery (OAop1, horizontal axis) to PI in the middle cerebral artery (MCA-Plop1, vertical axis0.

2(c–d)), in contrast to the case in PI, particularly after operation ($r = 0.512$, $p < 0.001$ and $r = 0.641$, $p < 0.001$, Figs 3c–d).

Intracranial vessels. In seven patients, who were free from symptoms postoperatively, no appropriate signal was received from the MCA before and after CE, because of lack of an acoustic window. MV in the MCA were lower (ns) on the operated side before operation than those on the non-operated side and in normal people, but were increased ($p < 0.001$) postoperatively (Table 2). The same was true of PI on the operated side. However, patients with ischaemic infarction had significantly ($p < 0.01$) slower MV (41.4 ± 10.16 cm/s) than normal people. In patients with preoperative hemisphere TIA and amaurosis fugax, with MV of 47.90 ± 12.32 cm/s, difference from normals was insignificant. PI in patients with infarction and TIA did not significantly differ from normal people, but showed wide variations, particularly patients with bilateral lesions.

In a patient, who had TIA before CE and who developed retinal embolisation from the operated ICA, the MCA was open with the same preoperative and postoperative MVs within the normal range; in the second patient with minor stroke before CE and with

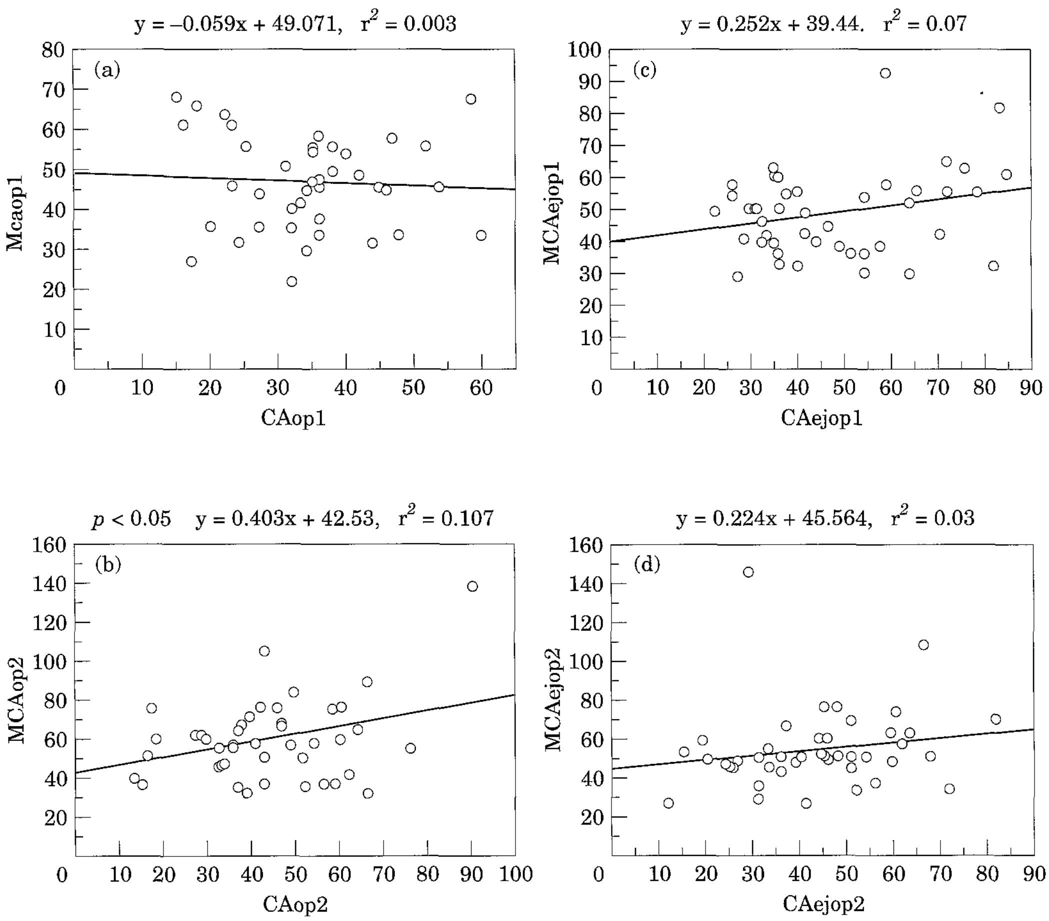

Fig. 2. Correlation between mean velocities (MV) in the internal carotid artery (CA, horizontal axis) and the middle cerebral artery (MCA, vertical axis) before and after carotid surgery on the operated (a) and (b) and non-operated sides (c) and (d).

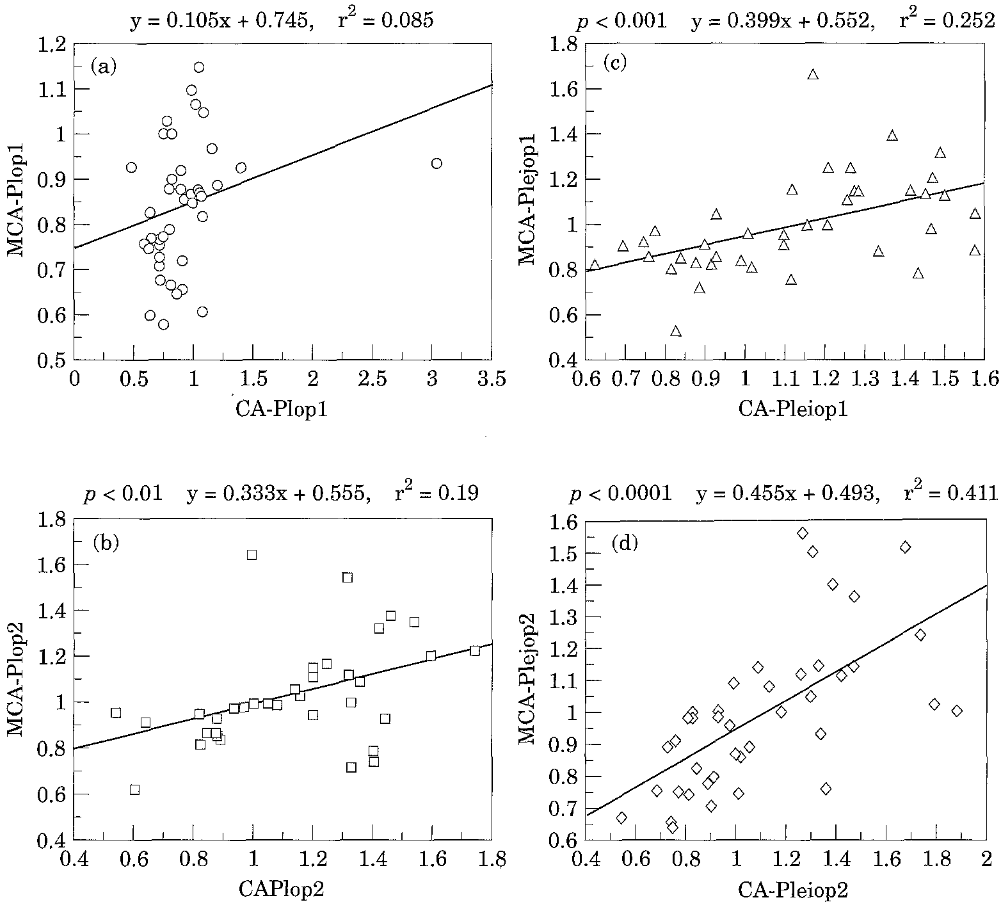

Fig. 3. Correlation of pulsatility index (PI) in the ICA vs. the MCA before and after carotid surgery on the operated (a) and (b) and non-operated sides (c) and (d).

Table 2. Mean velocities (MV) and pulsatility index (PI) in the middle cerebral artery (MCA), the anterior cerebral artery (ACA, only in preoperative normal direction, and the posterior cerebral artery (PCA) (a) before and (b) after operation.

	Non-operated			MV	Operated		
	MOA	ABA	PCA		MCA	ACA	PCA
N	59	30	36		59	34	41
(a)	48.85 ±14.30	58.50 ±20.50	46.50 ±21.20		46.27 ±12.07	53.81 ±18.63	44.39 ±18.11
					***	*	
(b)	52.91 ±19.10	53.31 ±20.05	44.86 ±18.73		59.17 ±20.75	62.49 ±27.68	44.47 ±14.85
				PI			
(a)	0.93 ±0.24	0.97 ±0.29	0.98 ±0.41		0.82 ±0.16	0.89 ±0.27	1.03 ±0.27
					***		
(b)	0.95 ±0.25	0.95 ±0.26	1.04 ±0.37		1.01 ±0.24	0.99 ±0.27	1.09 ±0.33

* = p<0.05; *** = p<0.001.

a recurrent stroke postoperatively, low MVs of 36 cm/s before and 38 cm/s after CE were found. In the third patient with minor stroke on the non-operated occluded side, the MV was low (41 cm/s) with increase after CE to 49 cm/s. The fourth patient with minor stroke corresponding to occluded side, showed unchanged MV of 33 cm/s after CE. PI increased significantly (p<0.01) only on the operated side. The PI in the MCA ipsilateral to the OA with retrograde flow direction was significantly (p<0.001) lower (0.68 ± 0.15) than PI (0.87 ± 0.15) in the MCA ipsilateral to the OA with antegrade flow.

Postoperatively, no signals could be detected in six ACAs, but were registered with normal power and direction after operation. ACA collaterals through the anterior communicating artery from the contralateral side, which was free from stenosis >70% were present in seven patients. The flow direction on the operated side was reversed (+53.46 ± 19.07 cm/s) and became normal pattern (−65.42 ± 15.94 cm/s) after operation. In the remainder, MV in the ACA had normal direction, with significant ($p<0.05$) postoperative increase (Table 2).

MV in the PCA remained unchanged, when all vessels were estimated (Table 2). Dominant PCA collaterals with MV of 73 ± 16.5 cm/s were found in seven patients; four of these showed retrograde flow and one isoelectric flow in the OA in addition. Four patients with PCA collaterals had high-degree contralateral lesions; (occlusion and stenosis >70%), respectively. The ipsilateral ICA supplied the anterior circulation in all other patients.

MV on the non-operated side increased insignificantly in the MCA and decreased in the ACA after CE (Table 2). In the ACA, ipsilateral to the occlusion on the non-operated side, flow direction changed from normal to reverse in two patients after CE, when the contralateral ACA on operated side became a main cross-over collateral. These vessels were excluded from postoperative analysis. The MV in the PCA did not change significantly.

Differences between group A and B

When patients were selected in group A (= without) and group B (= with contralateral high degree stenosis ≥70% or occlusion), those with bilateral high grade lesions differed haemodynamically from those with unilateral lesion ≥70%.

Siphon. MVs in the siphon on the operated side in group A were 37 ± 11 cm/s and differed significantly ($p<0.05$) from group B, where very slow MV of 28.26 ± 12.84 cm/s were registered.

Intracranial vessels. MV in the ACA on the operated side (Fig. 4a) and MV in the PCA on both sides (Fig. 4b) were faster in group B ($p<0.01$) than those in group A. On the non-operated side, MV in the MCA in the group B were lowest, in relation to normal people ($p<0.01$). Generally lower preoperative PI (0.73 ± 0.15) were measured in all vessels of group B than PI of

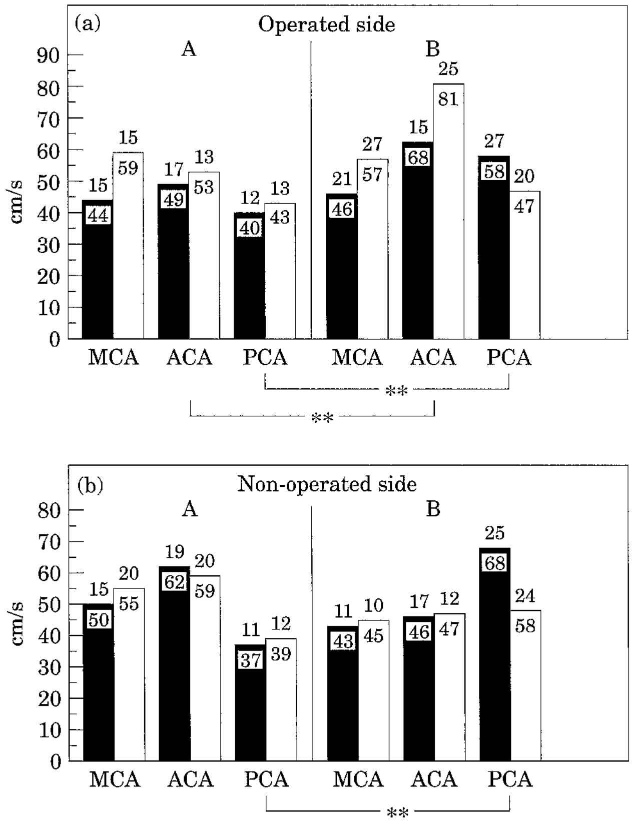

Fig. 4. Differences in MV between group A and group B on the operated (a) and non-operated (b) sides. Numbers above bars denote standard deviation. For explanation of statistical significances see text. (■) Preop; (□) postop.

1.03 ± 0.31 in group A ($p<0.001$). Postoperative PI values were 0.80 ± 0.20 in group B and 1.02 ± 0.29 in group A ($p<0.001$, Figs 5(a,b)). Significant ($p<0.001$), postoperative increase in PI emerged only in the MCA on the operated side in both groups (Figs 5(a,b)).

Blood pressure. On admission systolic blood pressure (BP) was 158 ± 26 mmHg and diastolic BP 85 ± 11 mmHg before CE and decreased postoperatively to 145 ± 21 mmHg in systole ($p<0.001$) and to 81 ± 10 mmHg in diastole ($p<0.05$). In groups A and B, a significant difference emerged between preoperative and postoperative BP only in group A with a fall in systolic BP from 161 ± 27 to 144 ± 23 mmHg ($p<0.001$) and in diastolic from 85 ± 12 to 80 ± 10 mmHg ($p<0.05$). In group B there was no significant difference in BP; 148 ± 21 mmHg in systole and 85 ± 9 mmHg in diastole before CE, and 146 ± 16 mmHg in systole and 83 ± 8 mmHg in diastole after CE.

Stump pressure. The stump pressure was 55 ± 17 mmHg (mean ± S.D.). In group A, higher values (55 ± 19 mmHg) were measured than in group B

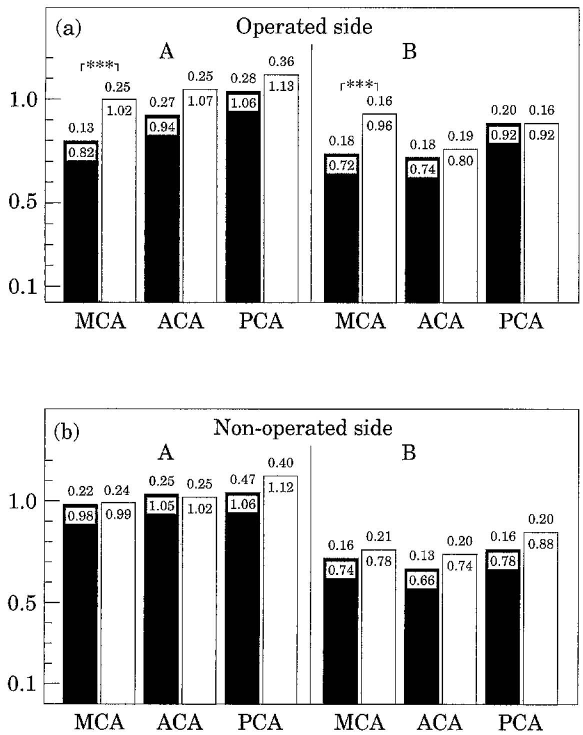

Fig. 5. PI changes in groups A and B on the operated (5a) and non-operated (5b) sides. (■) Preop; (□) postop. *** = $p<0.001$.

(47 ± 20 mmHg). Stump pressure on the operated side correlated ($p<0.01$) with retrograde-antegrade SV in the OA (Fig. 6a) and PI in MCA (Fig. 6b) measured before operation, but did not correlate with MV in the MCA, using single regression analysis. Stump pressure connected with preoperative retrograde flow in the OA was 43 ± 15 mmHg, and stump pressure associated with antegrade flow was 59 ± 16 mmHg. This difference was significant ($p<0.01$).

Discussion

The present study confirms a further decrease in postoperative morbidity and mortality in comparison to our earlier report.[4] Postoperative complications are slightly lower than interim results from European Carotid Surgery Trialists' Collaborative Group (ECST)[12] and the North American Symptomatic Carotid Endarterectomy Trial (NASCET).[19] However, our duplex criteria[13] and the angiographic measurement[12] used by ECST resulted in a calculated degree of stenosis that is greater for any given stenosis than those derived by NASCET.[20] Stenosis of 70% in NASCET is equivalent with stenosis of 82% in ECST. No patient had persistent functional disability related to the operated vessel. Introduction of new operative technique using vein patch resulted in a decrease in postoperative occlusions/high grade lesions >75%, which accords with other reports using the same operative approach.[21]

The most impressive features were changes in flow direction in the OA, the siphon, and the ACA, and significant MV increase within the carotid territory only on the operated side; this differed significantly from the flow pattern on the contralateral side. After operation, MV in the MCA and ACA were within the normal range, which accords with the normal values in the same age groups, reported by us[17] and other researchers.[22]

Unilateral increase in MV in the anterior circulation postoperatively may depend on increase in mean blood volume. This finding accords with a previous report of Blohme and co-workers,[9] who found a 43% increase in MV in the MCA compared to preoperative values. Regression analysis confirmed correlation between velocities in the siphon and the MCA only on the operated side after CE, which probably reflects a haemodynamic improvement within the same vessel territory. Lindegård and co-workers have reported that a relationship exists between MV in the MCA and MV in the extracranial part of the ICA, measured by pulsed Doppler, and expressed as MCA/ICA index, which was about 1.7 in normal subjects.[23] The volume of blood in the distal part of the extracranial ICA might be equivalent to volume flow in the siphon behind the orbit. We recently found a significant correlation ($r=0.60$, $p<0.01$) between MV in the siphon and MV in the MCA on the side contraindicated to the unilateral ICA occlusion.[17] Thus, the hypothesis that the blood flow is redistributed in favour of the operated side is supported by improved correlation of the ICA vs. MCA, increased MV in the MCA and the ACA on the operated side after CE and lack of significant changes in the posterior circulation and on the contralateral, non-operated side. High-grade stenosis/occlusion in the ICA on the contralateral side and ischaemic infarcts in the MCA territory with lowering of MV,[24] which we also found in the current work, impair such close correlation.

Positive correlation between SV in the OA and stump pressure seems to be a sensitive indicator of perfusion within the carotid territory and function of collaterals, which is supported by the finding that the group with retrograde ophthalmic collaterals had significantly lower stump pressure than the group with antegrade velocities. In spite of an open anterior communicating artery (ACoA) the ophthalmic flow may be reversed;[17] thus, findings of an open ACoA alone in preoperative compression tests and at angiography do not provide sufficient information about a

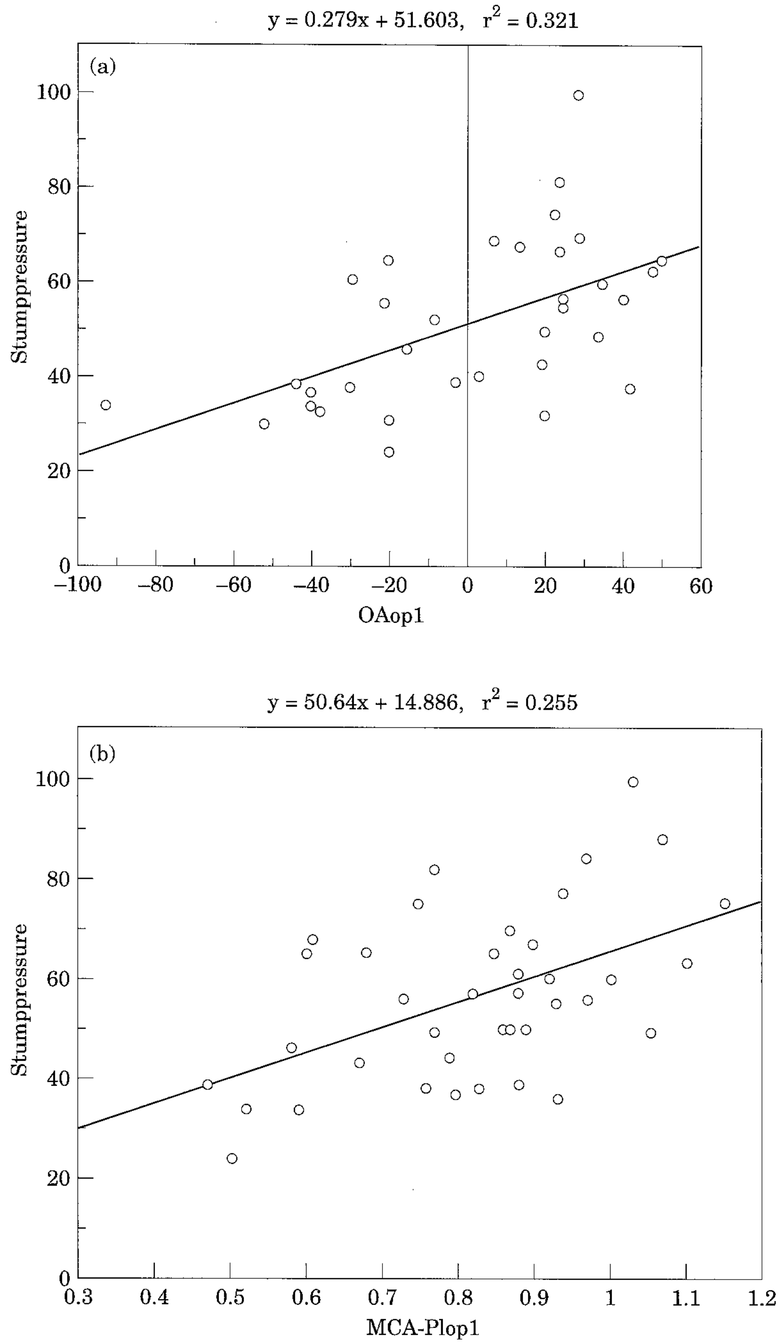

Fig. 6. Correlation of stump pressure (vertical axis) to systolic velocity in the ophthalmic artery on the operated side (OAop1, a) and to pulsatility index (PI) in the middle cerebral artery (MCAop1, b).

potential risk of peroperative hypoperfusion. Change of pressure in the carotid territory after surgery causes change of flow direction to normal or significant increase in systolic velocity ($p<0.001$) in antegrade direction in the OA, whilst flow direction and velocities on the contralateral side remain unchanged. In the OA, Kerty and Horven[25] found low SV values in an antegrade direction in 13 patients with high degree lesion (10 occlusions and three stenoses >75%) in comparison to normal subjects with SV of 42.3±9.8 cm/s; this tallies with our findings. When ophthalmic collaterals lose a supplying function to the brain circulation a change of flow direction is connected with change in low resistivity flow profile to the high resistivity profile with decrease in diastolic velocity and significant increase of PI; a velocity profile resembling that found in the peripheral vessels such as, for example, in the external carotid artery (ECA).[13]

Previously, attempts at correlating stump pressure back to changes in consciousness estimated a critical value of 25 mmHg in patients operated on under local anaesthesia.[26] In a later study Boysen and co-workers[27] reported a higher critical level of 55 mmHg. They found that CBF variation followed variation in stump pressure and suggested measuring stump pressure as a simpler procedure than CBF, needing less technical assistance. After operation, the CBF remained unchanged, but ICA flow increased significantly, which is in accordance with our findings. The insignificant correlation of stump pressure to preoperative MV in the MCA in our report is in agreement with the study carried out by Padayachee,[28] who showed significant correlation only with peroperatively measured MV, where 50% reduction of the MCA velocity identified whether the stump pressure was greater or less than 50 mmHg. Later, others reported[8,11] an inverse correlation of stump pressure with decrease in MV in the MCA during cross-clamping. Surprisingly, the function of the posterior communicating artery did not influence the stump pressure values.[11]

Low stump pressure[10] was described in patients with signs of postoperative cerebral hyperfusion lasting for 12 days. These researchers also observed changes in MCA velocities over time; the greatest increase being noted during the first 2 days after CE. Correlation of low ICA/CCA pressure ratio to CBF increase was shown by Schroeder and co-workers,[29] who postulated that more pronounced and long lasting hyperaemia occurred in patients with ICA/CCA pressure ratio below <0.7, in whom a lower stump pressure was also found. The state of hyperperfusion in combination with hypertension may lead to breakthrough perfusion resulting in hypertensive encephalopathy as a cause of cerebral haemorrhage.[30]

Difference in preoperative and postoperative BP was found only in group A, which may reflect impaired vasomotor reactivity in group B patients with bilateral lesions. Differences in MV and low PI in group B with decrease in MCA/PCA gradient, which normally is 1.55 ± 13[22], particularly on the non-operated side and corresponding to a high-grade stenosis/occlusion, imply a fall in perfusion pressure and pulsatility within the anterior circulation and a dominating function of posterior collaterals.[8,31] However, if the anterior communicating artery is patent, rise in perfusion pressure in the ACA on the operated side after CE may cause a change of flow direction on the opposite side, previously haemodynamically compromised by occlusion,[32] as we observed in two patients. Hopefully, our present results could support a decision to operate, in patients with bilateral lesions and also contralateral high-grade stenoses, in order to bilaterally enhance perfusion within the anterior circulation and to relieve the posterior circulation from increasing haemodynamic demands. Haemodynamic improvement may lead to metabolic normalisation, expressed as an increase in the N-acetyl-aspartate/choline ratio, which has been reported to be suppressed before CE.[33]

In conclusion, TCD provides important information about the risk of preoperative and peroperative hypoperfusion, particularly if a combination of retrograde flow in the OA and low pulsatility in the MCA is present. After operation, velocity changes in the OA and the siphon confirm postoperative normalisation. TCD also facilitates the diagnosis of postoperative patency/occlusion of the ICA before performing duplex scanning.

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