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Bilateral common carotid artery occlusion with minimal neurological deficit: Long term follow up in 3 patients

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Three patients, two women and one man, aged 56–70 year (mean 59 years) with modest neurological deficits and bilateral occlusion of the common carotid arteries initially identified by duplex scanning and angiography, were followed by repeated clinical and transcranial Doppler examination (TCD) over 6.5 years. Vasomotor reactivity (VMR) was tested by combined examination of TCD and xenon-133 cerebral blood flow (CBF) before and after intravenous administration of 1 g acetazolamide. At follow-up CBF was measured using single photon emission computerised tomography (SPECT). In two patients mean velocities in the middle cerebral artery (MCA) were within the normal range at repetitive examinations with good VMR ranging 30–111%, whereas CBF was reduced in MCA territories ranging 29–36 ml 100 g⁻¹ min⁻¹, but increasing 44–69% after acetazolamide, indicating good VMR. These 2 cases had anterograde flow in the ophthalmic artery and siphon. The third patient had very low MCA mean velocities of 21–27 cm sec⁻¹, increasing 26–33% after acetazolamide. This patient had retrograde flow in the ophthalmic artery and siphon connected with bilateral prolonged episodes of amaurosis fugax and transient ischemic attacks. In all three patients the posterior cerebral arteries were major supplying collaterals having high mean velocities of about 100 cm sec⁻¹ and high velocities in the basilar arteries of 85 cm sec⁻¹ as well. During follow-up no patient had a new stroke, but all experienced orthostatic dizziness and chronic fatigue. [Neurol Res 1996, 18: 109–116]

Keywords: Common carotid artery; transcranial Doppler; cerebral blood flow

INTRODUCTION

Occlusion of the common carotid artery (CCA) is very rare and obstruction of both CCA was previously considered to be incompatible with normal life. However, if the external carotid artery (ECA) acts as a collateral to the internal carotid artery (ICA), the flow in the ICA can be maintained in normal direction^{1–4}.

A reduction of regional perfusion pressure with depressed vasomotor reactivity (VMR) may follow extracranial obstructive disease⁵. We have previously described the hemodynamic extracranial and intracranial features as estimated by duplex scanning and transcranial Doppler (TCD) in 18 patients with unilateral and 3 patients with bilateral CCA occlusion⁶. Cerebral blood flow (CBF) and TCD examinations including a test of (VMR) was later performed in 12 of these patients⁷.

In this study we report the clinical course and the

hemodynamic consequences including assessment of VMR by repeated ultrasonic examination of extracranial and intracranial vessels and CBF in 3 patients with bilateral CCA occlusion.

PATIENTS

Three patients, 2 women and one man, aged 56–70 (mean 59 years) with long-standing bilateral CCA occlusion, initially identified by duplex scanning and angiography, were followed since 1987 (females) and 1989 (male) by repeated clinical and TCD examination for 38–88 months, in average 6.5 years. In 1989 a sequential examination of TCD and CBF using xenon-133 was done before and after intravenous administration of 1 g acetazolamide in patient no. 1 and 2⁷.

Subsequently, CBF was measured after intravenous administration of technetium-99m hexamethylpropylene amine oxime (^{99m}Tc-HMPAO) by single photon emission tomography (SPECT) in all three cases.

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Repetitive TCD examinations were initially performed by TC 64 B and since 1990 by 3-TCD scanner. The data were compared to age-matched healthy controls^{6,7}.

MATERIALS AND METHODS

Ultrasonic examination

The extracranial vessels were examined by duplex scanners with 7.5 MHz 2-D image and 5 MHz pulsed Doppler (ATL Mark 600 and Acuson XP, USA), a method described elsewhere^{8,9}.

Transorbital examination of the ophthalmic artery and siphon was performed with a handheld probe using a 2 MHz transducer. The same transducer was used for examination of intracranial vessels until 1990¹⁰. Since 1990 examination of intracranial vessels within the whole circle of Willis was performed by 3-D transcranial Doppler (TCD) scanner with a 2 MHz transducer (Trans-Scan, EMI Germany). In the 3-D TCD scanner each focal recording is stored as a combination of a directional spectral Doppler recording and a colored dot in three

planes (frontal, horizontal and sagittal). A composite display is built up by recording multiple spectra from major cerebral arteries¹¹.

Velocities from the middle cerebral artery (MCA) were recorded at a depth of 5.0–5.5 cm, anterior cerebral artery (ACA) at depth of 6.5–7.0 cm and posterior cerebral artery (PCA) at a depth of 6.0–6.5 cm¹⁰. Recording from the vertebrobasilar system was performed with the patient lying on the side with anteflexion of the head. One transducer was used through the foramen magnum acoustic window (Figure 1D). Maximal and mean velocities in cm sec^{-1} and pulsatility index (PI) defined as (peak systole–end diastole)/mean¹² were recorded. Pulse rise time (PRT) was estimated as time in msec from the end diastole to the peak systole⁶. The baseline TCD data were compared with results from an age-matched control-group ($n=8$), mean age 62 years.

CBF measurement

Three dimensional rCBF was measured by intravenous injection of approximately 2 GBq $^{133}\text{Xenon}$. The rCBF

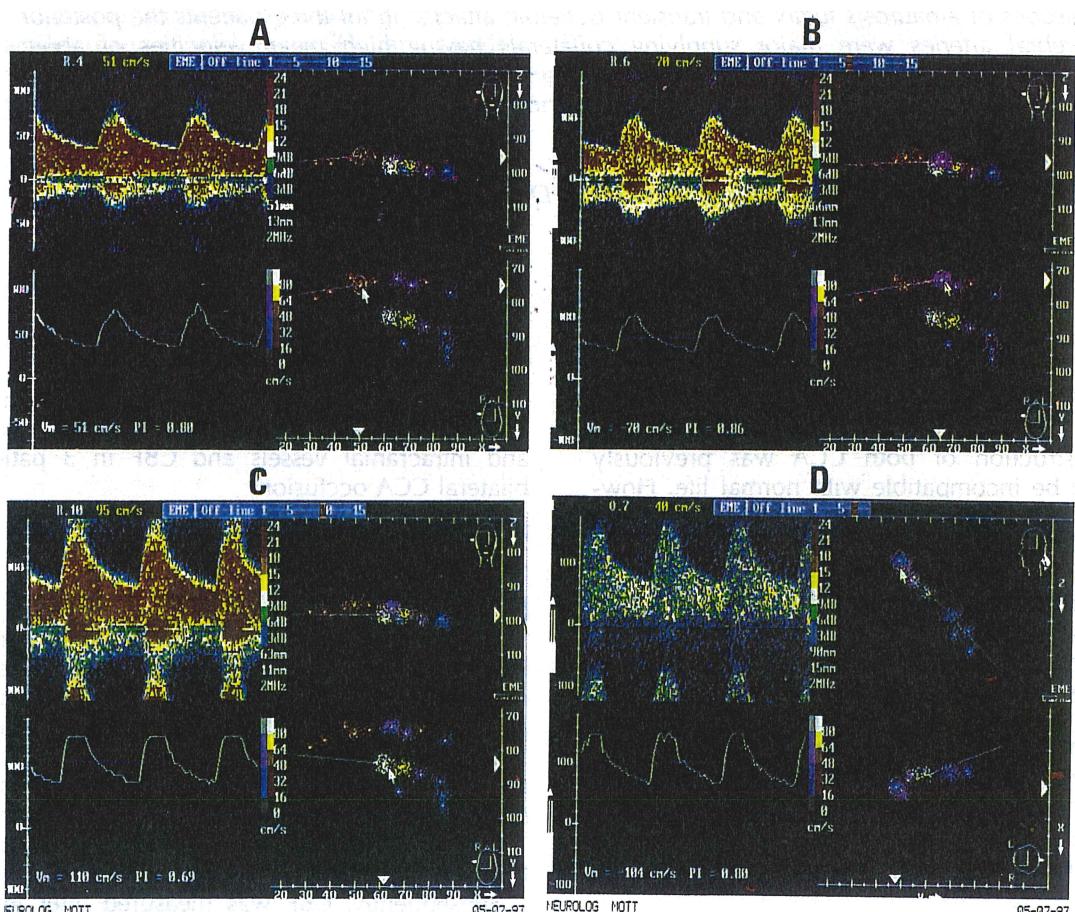


Figure 1: Transcranial scan of the middle cerebral artery (MCA, A), anterior cerebral artery (ACA, B), posterior cerebral artery (PCA, C), basilar artery (BA, D) in patient 2. The circular cursor indicates precisely location of the Doppler sample volume (white arrow). Observe the prolonged PRT in MCA and ACA and high velocities in PCA and BA

was calculated from the recorded ^{133}Xe on clearance by the method of Kanno and Lassen¹³. Measurements were done with the patient resting supine in a laboratory that was quiet except for ambient noise at 1. rest, and 2. 20 min after i.v. administration of 1 g acetazolamide. SPECT camera (Tomomatic 564), following $^{99\text{m}}\text{Tc}$ -HMPAO i.v. administration, was used for later flow estimations.

Aortic arch angiography was performed at four different angles, the sagittal and lateral view and at 45° right and left oblique views.

Case reports

1. Female, born 1920, who at the age of 22 had tuberculosis with lymphadenitis and was treated by x-ray radiation directed to her neck. In 1951 she awoke with right-sided hemiparesis which resolved completely within 2–3 days. Angiography 3 years later showed bilateral CCA occlusion, probably caused by the previous radiation therapy, although pulseless disease was considered as an alternative diagnosis at that time. Hypertension was diagnosed in 1974 and treated with betablockers.

Duplex examination in 1984 confirmed the occlusion of both CCA, but revealed patent bifurcations on both sides, with the ICA supplied in cephalic direction by the ECA. The patient was admitted to hospital in 1985 because of intermittent right-sided hypoesthesia. Over the following years the patient complained of intermittent dizziness and some impairment of memory. Neurological examination and CT were normal. Current medication consists of Amilorid, Atenolol, Furosemid retard, Allopurinol, Naproxen, Felodipin and Acetylsalicyl acid.

2. Female, born 1935, had a family history of cardiovascular disease with two brothers having myocardial infarction. The patient was diagnosed as having arterial hypertension, claudication of the lower extremities, angina pectoris and hyperlipidemia. In 1984 'Y graft' surgery was performed due to occlusion of the distal aorta. In 1986 the patient was admitted with vertigo and blurred vision provoked by head movement. These symptoms were later provoked also by physical work, particularly when raising her arms above the horizontal line, and by postural changes. The neurological status revealed a rightsided hemianopia. CT scan showed a left occipital infarct 4.4 × 3 cm.

Duplex scanning revealed bilateral CCA occlusion with patent left bifurcation where flow was directed from ECA to ICA. On digital subtraction angiogram the right vertebral artery was seen to be occluded as well. Her neurologic symptoms gradually worsened over time, but repeated aortic arch angiography and CT in 1992 were unchanged.

She is now treated by Isradipin, Simvastin, Spironolacton and Dicoumarol.

3. Male, born 1921, smoker, with hyperlipidemia since 1978, treated by diet. Hypertension was diagnosed 1989. The same year the patient suffered from

vertigo and had a minor stroke with right-sided hemiparesis.

CT showed a small subcortical ischemic infarct (1.5 × 1.5 cm) in the left carotid territory. Primarily, duplex scanning showed bilateral occlusion of CCA with patent bifurcation bilaterally. The aortic arch angiography showed occlusion of both CCA. Right ICA was filled downstream to the siphon but the left ICA was patent and filled by ECA. Subsequently, the patient complained of blurred vision of the right eye and 2 years later observed prolonged amaurosis fugax of the right eye lasting about 30 min. In 1992 he was admitted with blurred vision of the left eye and vertigo provoked by physical work and postural changes and the same year left-sided transient ischemic attack.

A by-pass anastomosing the left subclavian artery to the patent ICA was attempted, but was not feasible due to deep location of the subclavian artery. He is treated by 75 mg dose acetylsalicyl acid.

Table 1: Systolic and mean velocities and pulsatility index in ophthalmic, siphon, vertebral and basilar artery at repetitive examinations 1987–1993 in 3 patients

No	Artery	Right			Left		
		S	M	PI	S	M	PI
1	Ophthalmic	32	16	2.00	32	12	2.6
		50	27	1.78	23	12	3.33
		60	35	1.05	36	27	0.96
		83	61	0.89	48	32	0.90
		61	40	0.80	-36	23	0.80
		-33	23	0.75	27	12	1.15
		-20	10	1.15	-48	28	0.98
		-38	26	0.47	-23	19	0.57
		-26	18	0.43	-36	24	0.63
		-54	35	0.67	-44	30	0.75
2	Siphon	36	27	0.47	-40	29	0.79
		-38	24	0.80	-58	31	0.75
		49	15	2.00	23	12	3.33
		60	54	1.14	71	36	1.14
		56	32	1.09	-	-	-
		61	40	0.80	-54	26	0.88
		56	36	0.90	56	36	0.93
		52	29	0.63	35	25	0.69
		-27	17	0.65	-25	17	0.76
		-28	19	0.53	-42	30	0.58
3	Vertebral	-35	21	0.85	-24	20	0.57
		-46	33	0.65	-38	27	0.55
		-23	17	0.60	-15	11	0.55
		96	50	1.36	165	103	1.00
		68	42	1.00	103	65	1.06
		160	106	0.88	-	-	-
		56	40	0.77	-	-	-
		65	36	1.08	56	46	0.60
		51	29	0.78	46	36	0.63
					S	M	PI
1	Basilar				132	72	1.27
					160	114	0.83
					102	60	0.81
					179	104	0.80
					51	31	1.00
					109	83	0.72

S, systolic; M, mean velocity; PI, pulsatility index

Table 2: Velocities and pulsatility index in the middle cerebral artery, anterior cerebral artery and posterior cerebral artery at repetitive examinations during 1987–1993

No	Artery	Right			Left		
		S	M	PI	S	M	PI
1	MCA	98	60	0.93	100	64	0.93
		100	65	0.85	82	60	0.85
		100	59	1.04	87	56	1.00
		96	51	1.13	113	67	1.10
		88	62	0.70	74	56	0.59
		95	72	0.61	69	50	0.56
2		105	61	1.08	77	56	0.64
		77	51	0.80	79	52	0.73
		96	64	0.80	72	51	0.58
		42	28	1.61	54	38	0.68
		61	38	0.89	36	26	0.69
		46	30	1.38	41	27	1.13
3		40	24	1.04	29	23	0.60
		—	—	—	41	35	0.42
		59	24	1.16	—	—	—
		100	60	1.15	100	59	1.15
		128	83	1.22	124	72	1.15
		124	83	0.67	51	31	1.00
1	ACA	115	77	0.83	—	—	—
		110	70	0.86	—	—	—
		102	64	0.89	—	—	—
		42	28	1.61	54	38	0.61
		56	31	1.16	—	—	—
		—	—	—	160	100	0.98
2	PCA	160	96	0.78	191	150	0.76
		—	—	—	118	63	1.10
		148	100	0.73	127	72	0.94
		206	150	0.76	113	77	0.73
		186	114	0.86	100	62	0.67
		182	110	0.69	150	98	0.79
3		150	99	0.61	140	83	0.96
		86	54	1.01	112	70	0.90
		165	119	0.69	100	72	0.66
		160	114	0.66	106	76	0.75
		—77	54	0.54	—150	109	0.55
		64	45	0.71	108	74	0.72
		160	107	0.62	—	—	—

RESULTS

Transcranial Doppler

Five patent bifurcations were found. In 3, the flow direction was from the ECA to the ICA, whilst in the remaining 2, flow was reverted, from ICA to ECA. TCD results are described in Tables 1 and 2, VMR in Tables 3 and 4.

In the ophthalmic artery and the siphon (Table 1), velocities were in anterograde direction in patient 1 and 2. PI in patient 2 was < 1.0 , which suggested low perfusion pressure in the ICA bed (Figure 2b). In patient 3 the velocities in the ophthalmic artery and the siphon were in retrograde direction with a blunted profile of the pulse curve, i.e. delayed pulse rise time (PRT) as compared to normals (0.113 s, Figure 2a)⁶.

MCA velocities at repetitive examinations (Table 2) were within the normal range⁶ and did not change significantly during follow up in patient 1 and 2, and both showed considerable increase after acetazolamide administration (Table 2). In the third patient lower mean velocities were observed with average values of $29 \pm 6 \text{ cm sec}^{-1}$ and $30 \pm 6 \text{ cm sec}^{-1}$, on the right and left side respectively. After acetazolamide, increases in mean velocities of about 26% and 33%, were seen, but these values were still below the normal range (Table 3).

Anterior cerebral artery (ACA) velocities were high in case 1 and 2, (72 ± 16 and 74 ± 8) although flow signals were very weak in patient 2, except for the first study in 1987. In the case 3 ACA velocities were reduced and weak (Table 2).

In all patients the PCA were the main supplying collaterals with high average of mean velocities. In patient 2 very high velocities were detected in both posterior communicating arteries with turbulence (Figure 3). In patient 3 velocities varied between different examinations, but during the second, third and the last

Table 3: Systolic and mean velocities and pulsatility index in the middle cerebral artery before (a) and after (b) acetazolamide, in three patients

No	Right					Left				
	S	M	Change	PI	PRT	S	M	Change	PI	PRT
1a	98	56		1.12	272	86	38		1.26	242
b	136	90	61%	0.88	272	122	80	111%	0.90	181
2a	64	40		0.75	180	64	40		0.65	180
b	79	56	40%	0.55	180	74	58	45%	0.76	181
3a	42	27		0.89	200	30	21		0.67	290
b	58	35	30%	0.86	210	37	28	33%	0.57	250

Change = % increase of M from resting values. Pulse rise time (PRT) = time in msec from enddiastole to peak systole. S, systolic; M, mean; PI,

Table 4: Regional CBF expressed in $\text{ml } 100 \text{ g}^{-1} \text{ min}^{-1}$ in ACA, MCA and PCA at baseline (a) and after acetazolamide in patient 1 and 2

No	Right						Left					
	ACA	Change	MCA	Change	PCA	Change	ACA	Change	MCA	Change	PCA	Change
1a	28		29		34		27		29		33	
b	48	71	49	69	57	68	45	67	49	69	56	70
2a	36		36		41		34		35		36	
b	54	50	52	44	62	51	52	53	53	51	58	61

Change = % increase from baseline values after acetazolamide

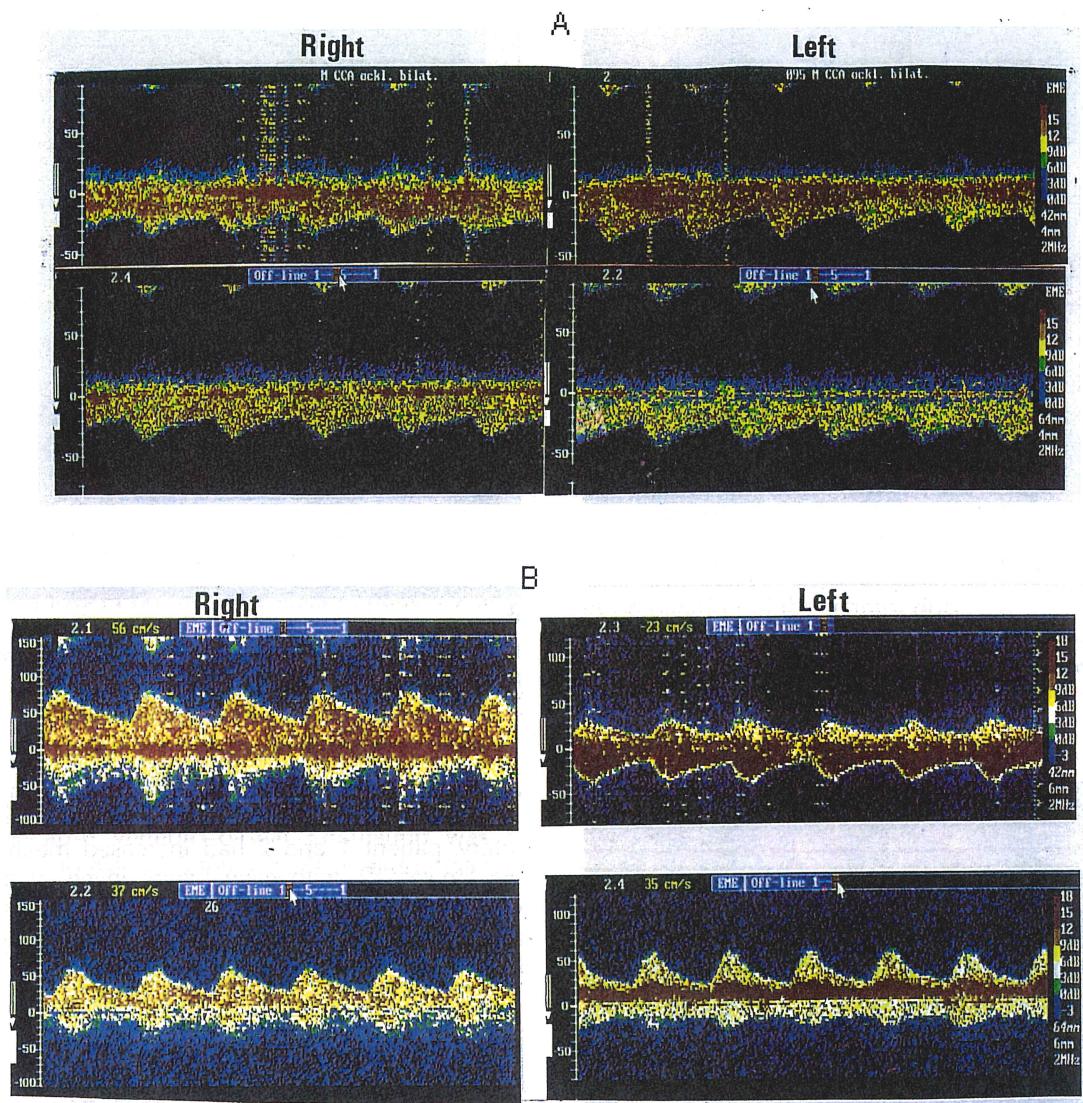


Figure 2A-B: Records of spectra from ophthalmic artery (upper) and siphon (lower) in patient 3 (1A) and 2 (1B). Note reverse (1A) and bidirectional flow pattern of the pulse curves (left 1B) in the ophthalmic artery. PRT is delayed in all recordings

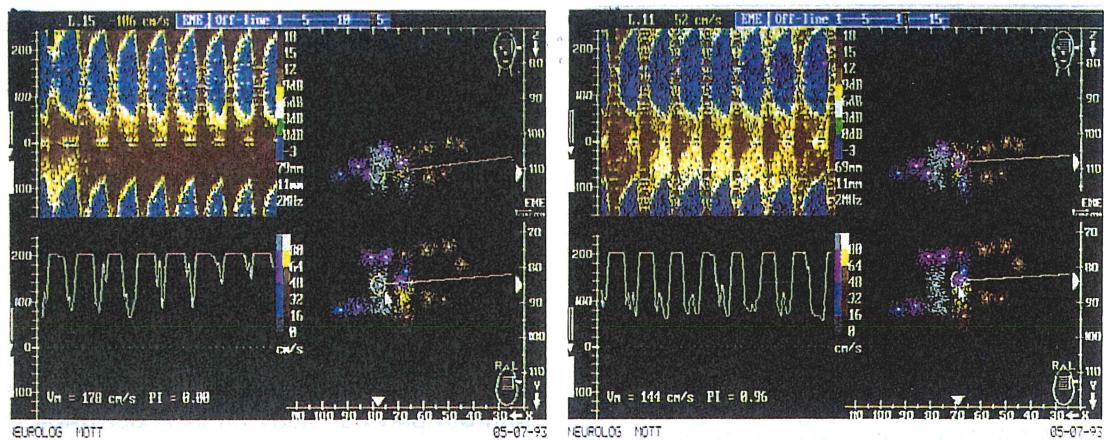
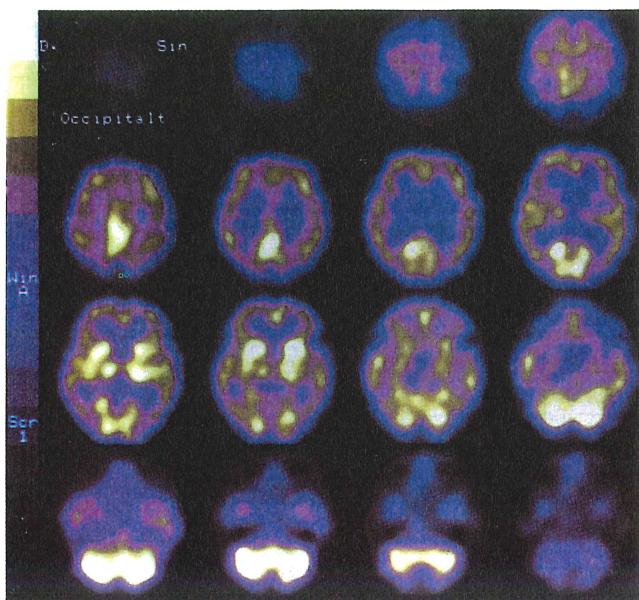
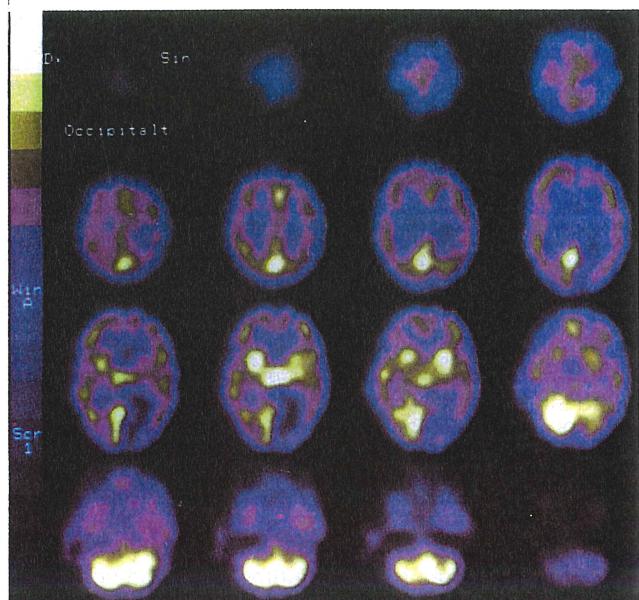


Figure 3: Posterior communicating arteries (PCoA) in the same patient recorded from the left temporal window. Left scan corresponds to the right and right to the left PCoA



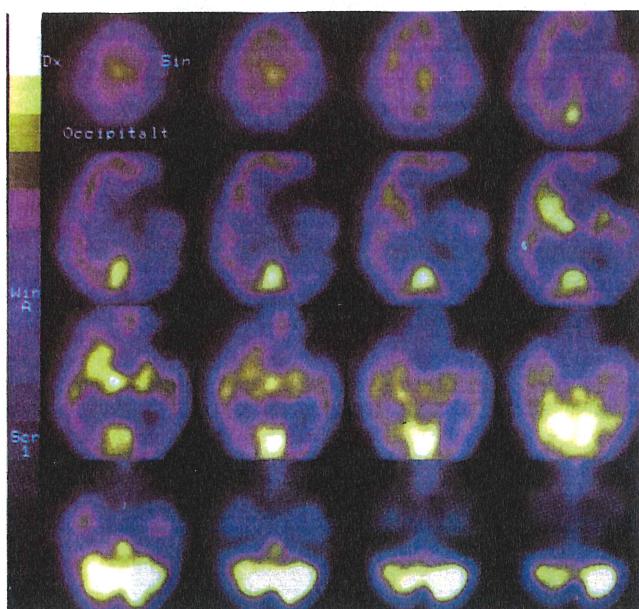
A

Figure 4: SPECT results in patients 1,2,3. **A:** Patient 1. Hypoperfusion parasagittally on the both sides in frontoparietal region, consistent with cortical atrophy



B

B: Patient 2. Lack of perfusion in the left occipital lobe, related to previously described infarction



C: Patient 3. Severely reduced flow level in the entire MCA territory on the left side. On the right centrally in the MCA territory is seen a small, well demarcated area of hypoperfusion

recording the velocities in the right PCA were >100 cm sec. The patient suffered headache during these registrations.

Increase of mean velocities in vertebrobasilar territory was observed. Patient 1 and 2 had values of 84 ± 27 cm sec $^{-1}$ and 79 ± 32 cm sec $^{-1}$, in the left

and right vertebral arteries, respectively. In patient 3 velocities are within the normal range. In the basilar artery, patient 1 and 2 had increased mean velocities (66 cm sec $^{-1}$), with even higher levels at the second examination (109 cm sec $^{-1}$, *Figure 1*). Patient 3 had a low level of 31 cm sec $^{-1}$ initially, which increased to 83 cm sec $^{-1}$ at follow up examination.

CBF

Patient 1 and 2 had both low global resting flow levels, 30 and 37 ml 100 g $^{-1}$ min $^{-1}$, as compared to a level of 55 ± 5 ml 100 g $^{-1}$ min $^{-1}$ in age-matched controls. In the MCA territories CBF ranged from 29 – 36 ml 100 g $^{-1}$ min $^{-1}$. Case 2 in addition had a marked CBF reduction in the left occipital lobe, related to the infarct seen on CT.

Adequate global flow increases ranging from 44–69% were seen following acetazolamide, without change in regional flow distribution (*Table 4*). SPECT examinations are described in *Figures 4a–c*.

DISCUSSION

All three patients had only modest neurological deficits when referred to neurologic-evaluation, despite severe obstructive extracranial disease. Transient neurological symptoms, including dizziness related to orthostatic changes, occurred during follow up, without residual functional disability. This outcome is in concordance with our previous study on CCA occlusion, where the best prognosis was seen in the patients having patent bifurcation and/or good collateralization 6 . However orthostatic dizziness may be a consequence of flow

redistribution as well as following ischemia and development of low perfusion infarction.

Retinal ischemia as prolonged amaurosis fugax or blurred vision may depend on temporary insufficiency of ophthalmic collateral during increased hemodynamic demands within the anterior circulation¹⁴. Dominating ophthalmic collaterals with retrograde flow direction is generally not sufficient in occlusive carotid disease and often accompanied by severe neurological deficits¹⁵. This pattern was found in patient 3, who had ischemic infarct in the left MCA territory.

Velocities in MCA did not change significantly during the observations time. In patient 1 and 2 the values were within the normal range reflecting adequate flow increase in the collaterals as well functioning circle of Willis. In the third patient the values were very low as a possible consequence of inadequate function of the collaterals and low regional perfusion pressure with following permanent tissue damage.

Changes of wave form observed, with delayed PRT generally and low PI on the both sides in patient 2 and on the left side in patient 3 may reflect a post-occlusive phenomena and a fall in pressure gradient as we found in our previous study of patients with CCA occlusion. These changes were reported also by others^{16,17}.

ACA flow signals in patient 3 were difficult to estimate because of a low Doppler shift.

Velocities in PCA and basilar artery were very high, probably because vertebro-basilar circulation was a feeding source to the MCA territory. PCA as dominating collateral in extracranial bilateral severe atherosclerotic lesion, eg. carotid stenosis > 75% lumen reduction or occlusion was confirmed by us⁷. In normals the mean velocity in BA is lower than in MCA, values between 31–40 cm sec⁻¹¹⁸. Blood volume in BA is around 300 ml min⁻¹, eg smaller than in the ICA with amount in each artery about 350 ml min⁻¹¹⁹. The basilar artery is probably too small to maintain adequate amount of the blood in bilaterally severely stenosed or occluded internal carotid arteries. Increased velocities in BA as well as reversed periorbital flow are signals of ineffective collateral circulation with subnormal perfusion of MCA²⁰.

During follow up we observed the difference between the first and second examination of BA, with higher values at the last recording, which may be explained by reduced peripheral cerebral resistance with precapillary vessel dilatation and increased demands in brain tissue within the MCA vascular territory.

Ischemic defects were presented in all patients, but low perfusion was the possible cause of infarction in patient 3, where we could not record Doppler signals from the depth < 4.5 cm on the left side. Low flow infarction occurs mostly in the border zone between posterior and anterior circulation²¹. VMR was excellent in both female patients but impaired in the male no. 3, where very low values in MCA were seen in resting condition.

CBF measurement showed very low global flow and in spite of very good response after acetazolamide, the

reached values were still below those for age related normal population. This state can lead to development of ischemic lesion e.g. chronic penumbra, defined as reversible ischemic paralysis with impaired function of neurons, which occurs, if flow decreases to interval 18–23 ml 100 g⁻¹ min⁻¹²².

Our study suggests that despite good vasomotor response these patients are chronically and increasingly hypoperfused. Bypass operation with an anastomosis between subclavian–patent ICA and in special selected cases direct thrombendarterectomy in CCA, may possibly prevent retinal ischemia and enhance perfusion within anterior circulation^{23,24}.

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