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Intracranial Haemodynamics in Patients with Spontaneous Carotid Dissection

Transcranial Doppler Ultrasound Follow-up Studies

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Summary. In this study 11 patients aged between 34 and 57 years with clinical and angiographic findings typical of carotid dissection were thoroughly examined with transcranial Doppler ultrasound (TCD) repeatedly during a follow-up period of 1–6 months. Stagnating blood flow velocities in the downstream middle cerebral artery (MCA) were recorded initially as well as enhanced velocities due to postischaemic hyperperfusion syndrome. MCA embolism originating from the extracranial carotid artery with or without resolution could be detected by TCD in 5 cases. TCD findings in another 2 cases pointed to haemodynamic upset. In 3 cases, clinical as well as TCD data did not allow strict differentiation between embolic and haemodynamic complications, suggesting more complex pathophysiological mechanisms as the cause of infarction. Carotid recanalization was seen in 9 cases. The follow-up investigations moreover demonstrated that intracranial haemodynamics may change from day to day in patients suffering from internal carotid artery dissection. TCD data may thus improve the understanding of pathogenetics as well as rationales for individual therapeutic intervention in this particular disease.

Key words: Transcranial Doppler ultrasound – Intracranial haemodynamics – Carotid artery disease – Spontaneous dissection

Introduction

Spontaneous dissection of cerebral arteries as a cause of cerebral ischaemia has become the subject of increasing scientific interest. The characteristics of the disease have been discussed extensively (Hart and Easton 1983, 1986). One striking feature of the illness is a predilection for young patients without vascular risk factors. Dissection plays a role in approximately 5% of all cerebral ischaemic events in the younger age groups (Hart and Easton 1983). Of 1200 patients with a first ischaemic stroke, 2.5% were proven by angiography to have suffered carotid artery dissection (Bogousslavsky et al. 1987). In young patients symptoms of hemispheric ischaemia together with an ipsilateral Horner syndrome and unilateral head or neck pain suggest carotid stenosis or occlusion. Headache, frequently radiating unilaterally or into the orbit and oculosympathetic disorders often precede hemispheric symptoms and point to the time of onset of the illness. Common misdiagnoses of carotid occlusions must be kept in mind to avoid overestimation of individual clinical or paraclinical signs (Nespeca and Townsend 1987). The appearence of dissections on angiography differs from arteriosclerotic obstructions, but is usually not pathognomonic in the absence of unequivocal double lumen (Bradac et al. 1981).

Transcranial Doppler sonography (TCD) has recently provided an aid to the study of the effects of extracranial dissection on intracranial haemodynamics (Aaslid et al. 1982, Aaslid 1986). The method utilizes pulsed ultrasound for a non-invasive quantification of blood flow velocity in the cerebral arteries. Haemodynamic phenomena such as stagnation of blood flow, compensatory increase in flow velocity or recanalization of occluded vessels can be observed in a bedside examination, thus allowing a close follow-up. Prior to this study, TCD findings in carotid dissection have been the subject of only one case report (Rohr and Gauthier 1987). The aim of this TCD study was to throw light on the cerebral haemody-

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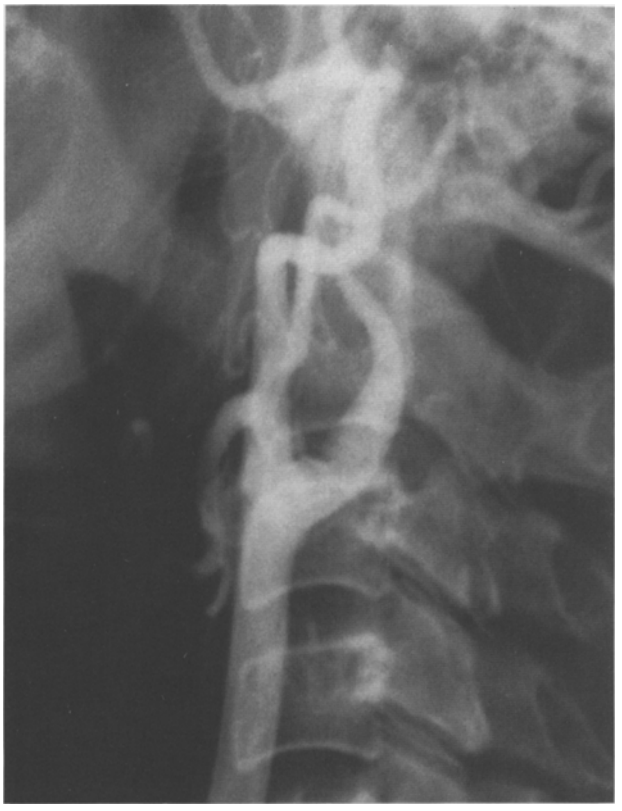

Fig. 1. Case 1: angiogram demonstrating a tapered narrowing of the internal carotid artery 2 cm distal of the carotid bifurcation

namics following dissections, and thus contribute to the understanding of individual cases as well as the general pathophysiology of ischaemia in this particular form of stroke.

Patients and Methods

Detailed TCD examinations were performed in 11 patients suffering from 12 carotid dissections. All patients conformed to the diagnostic criteria established by Heart and Easton (1983). Each clinical history was recorded and all cases were subjected to neurological examination, CT and angiography. Examination of extracranial cerebral arteries [common carotid artery (CCA), internal carotid artery (ICA), external carotid artery (ECA), ophthalmic artery (OA) vertebral artery (VA)] were carried out with a 4-MHz continuous-wave Doppler instrument. For TCD studies a range-gated pulsed wave Doppler device (TC2–64, EME, Überlingen, FRG) was used. Basal arteries [middle cerebral artery (MCA), posterior cerebral artery (PCA), posterior communicating artery (PcomA), anterior communicating artery (AComA)] were differentiated according to established criteria; our range of laboratory normals is consistent with published data (Aaslid 1986; Arnolds and von Reutern 1986; Hennerici et al. 1987; Spencer and Whisler 1986). The TCD follow-up studies were performed under well-defined conditions by the same investigator.

Case reports

Case 1. The 57-year-old patient first noticed weakness of his right hand shortly after breakfast and speech impairment 1 h later. Two days previously he had complained of pain above the left eye. There was no history of trauma, but instable hypertension had been noted earlier. On examination he was found to have a mild right sensory and motor hemiparesis and motor aphasia. Extracranial Doppler sonography indicated distal obstruction, with retrograde flow in the supratrochlear artery and a resistance profile in the ICA on the left. A stenotic signal, however, was not observed. The CT scan was normal. Tapered narrowing of the left ICA 2 cm distal to the carotid bifurcation was seen on angiography (Fig. 1). The intracranial blood vessels were unremarkable.

Ten days after the onset of hemispheric symptoms TCD showed a reduction of blood flow velocity in the left MCA of approximately 40% compared with the right. Pulsatility was severely decreased, but no cross-flow was demonstrable.

Warfarin therapy was instituted, followed by a major improvement of the neurological deficits. Extracranial and transcranial Doppler sonography 6 months later showed normal cerebral circulation. Blood flow in the ophthalmic artery was orthograde, and MCA pulsatility was symmetrical.

Case 2. The 34-year-old patient was noted to be staring fixedly ahead and to be unresponsive to speech whilst driving. On leaving the car her husband saw she could neither walk nor stand and that her right arm was paralysed. During the previous 4 weeks she had complained of a left-sided headache. For 17 years she had been using oral contraceptives.

Neurological examination revealed anarthria and a severe right motor and sensory hemiparesis of both face and arm. Her comprehension of speech was unimpaired. Extracranial Doppler sonography suggested intracranial obstruction.

On CT scan an extensive left parietal infarction was seen. Angiography demonstrated a filiform narrowing of the left ICA proximal to the carotid canal extending for more than 2 cm (Fig. 2). The distal carotid syphon was also stenosed combined with slight annular stenoses. More distally the A_1 segment of the anterior cerebral artery (ACA) as well the MCA both contrasted poorly and both ACA were perfused by the right ICA. Extension of the carotid dissection into the M_1 segment was not visible.

Two weeks after onset the first TCD examination was carried out. Average flow velocity in the left MCA was approximately 30% less than on the right. Flow velocity in the right A_1 segment, feeding both ACA, was markedly increased due also to leptomeningeal collateralization. The follow-up examination 4 weeks after onset demonstrated a return to normal flow velocity in the right ACA with otherwise unchanged TCD findings. A strong residual flow in the left MCA on compression of the left CCA indicated good collateral compensation.

The neurological deficits improved well under warfarin. Five weeks after onset she had a mild clumsiness of the right hand and mild aphasia with infrequent paraphasia.

In summary, dissection of the left ICA caused left MCA branch infarction in this patient. Fibromuscular dysplasia of the involved arteries was suggested by angiography. The initial increase in flow observed in TCD indicated activated leptomeningeal anastomoses between ACA and MCA territories on the left.

A haemodynamic pathogenesis of the ischaemic event seems unlikely in view of the good compensatory capacity by the circle of Willis, as proved by TCD.

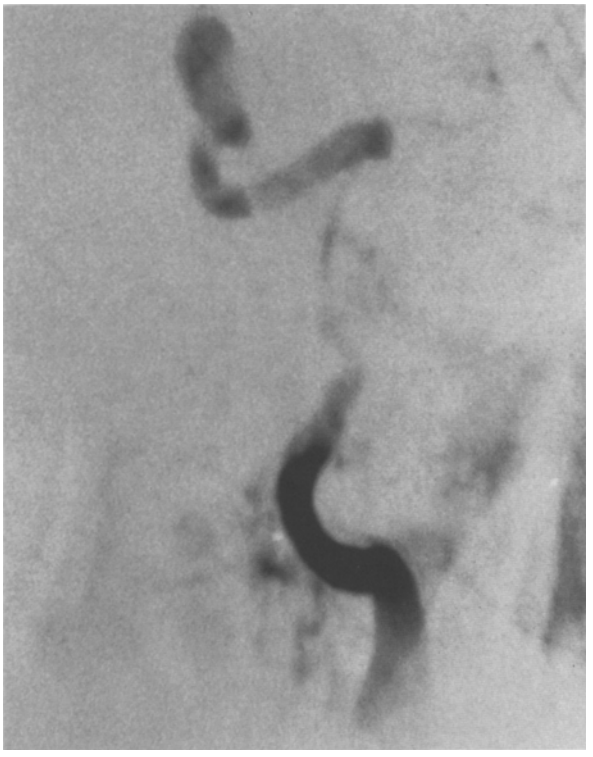

Fig. 2. Case 2: angiogram showing a filiform narrowing of over 2 cm in length ("string sign") of the internal carotid artery proximal to the base of the scull

Case 3. This previous healthy, 45-year-old patient developed a left-sided weakness one morning which resolved incompletely during the afternoon. Upon awaking the next morning, however, the weakness had returned with increased severity. Transient monocular blindness of the left eye had occured 2 weeks previously. No vascular risk factors were found.

The neurological examination revealed dysarthria and a severe left-sided weakness with pyramidal signs involving the left arm more than the leg. CT revealed infarction of the right lenticulostriate arteries with some compression of the right frontal horn.

Bilateral ICA occlusion was demonstrated on extracranial Doppler sonography. Angiography confirmed the complete left ICA occlusion distal to the bifurcation and demonstrated strong ophthalmic collaterals. An extensive filiform stenosis of the right ICA was seen. The right carotid syphon was filled by the ophthalmic artery as well as by the PcomA. An external carotid artery anastomosis perfused the distal right VA, which was occluded proximally. The left VA was somewhat hyperplastic. The ACA and MCA on both sides were perfused well via PcomA anastomoses.

During the following 4 weeks the symptoms resolved almost completely. Angiography 8 weeks after onset showed recanalization of the right ICA; the left ICA recanalized after 10 weeks.

TCD on the day of admission revealed markedly decreased flow velocity with reduced pulsatility in the right MCA (Fig. 3). The flow direction in the A_1 segment of the right ACA was orthograde, notwithstanding the severe ICA stenosis, indicating a sufficient PcomA anastomosis. On a follow-up TCD examination 3 days later the situation was unchanged. Eighteen days later flow conditions in both MCA were almost normal with only a slightly increased diastolic flow velocity on the left. Compression of the opposite CCA had no haemodynamic effect on either side owing to persisting bilateral ICA obstruction. Flow conditions in the MCAs were virtually symmetrical 2 months later even after recanalization of the right ICA (with persistent left ICA occlusion).

In summary, this patient suffered a right-sided MCA infarction induced by a haemodynamic upset with a very favourable clinical recovery under haemodilution. The cause is to be seen in the right ICA dissection; the simultaneous left ICA dissection remained asymptomatic except for the single attack of transient monocular blindness. TCD and angiography showed compensation via anastomoses from the left VA and both PcomAs. Recanalization took place in 8 weeks on the right and in 10 weeks on the left.

Dissection suggests itself as the cause of the asymptomatic right VA occlusion; however, recanalization of this artery was not observed. The fluctuating onset of the illness and the marked initial reduction of flow velocities in the MCA indicated a haemodynamic basis of infarction. We suspect that the ICA was initially totally occluded, and that recanalization had already begun at the time of angiography.

Case 4. This 38-year-old patient's wife had noted poverty of speech, misconstructed sentences and word-finding difficulties 2 days preceding admission. On the next day he remained in bed (contrary to normal habits), appeared to be confused, complaining of a severe headache. A consumption of 40 cigarettes daily was the only known vascular risk factor.

On examination the patient was somnolent and confused with global aphasia and a left-sided Horner syndrome. The left ICA appeared to be occluded on extracranial Doppler sonography, and angiography demonstrated a tapering occlusion of the vessel. Vertebrobasilar arteries filled the left MCA, whereas the left ACA was perfused from the right and contrasted poorly.

CT revealed a left-sided ischaemic infarction referrable to a postero-temporal branch of the MCA.

TCD (Fig. 4) on admission showed a reduction in MCA flow velocity by more than 55% on the left compared with the right (heart rate 60/min). A further decrease in left MCA flow on right CCA compression proved collateralization via the AComA. Correspondingly, retrograde flow in the A_1 segment of the left ACA with an accelerated orthograde flow in the A_1 segment of the right ACA was observed. Furthermore, flow in the P_1 segment of the left PCA and in the left PcomA was markedly accelerated with a further increase in velocity on right CCA compression. During the following 4 days the flow in both left and right MCA increased, but the asymmetry in flow velocity declined to 15% (heart rate 77/min).

In summary, the characteristic history and findings on angiography suggest left ICA dissection. No recanalization was demonstrable after a follow-up period of 6 months, but clinical deficits resolved satisfactorily, leaving mild residual dysphasia.

Case 5. Driving home after a vacation, this 47-year-old patient noted weakness and numbness of his right arm. The symptoms improved after 30 min, but progressive weakness and numbness of the right arm together with a speech disorder developed over the next 2 days. Five or six attacks of transient monocular blindness of the left eye occurred, each lasting several minutes. No causative event or vascular risk factor was known.

On admission he had a right-sided hemiparesis of the face and arm together with anarthria and facial apraxia.

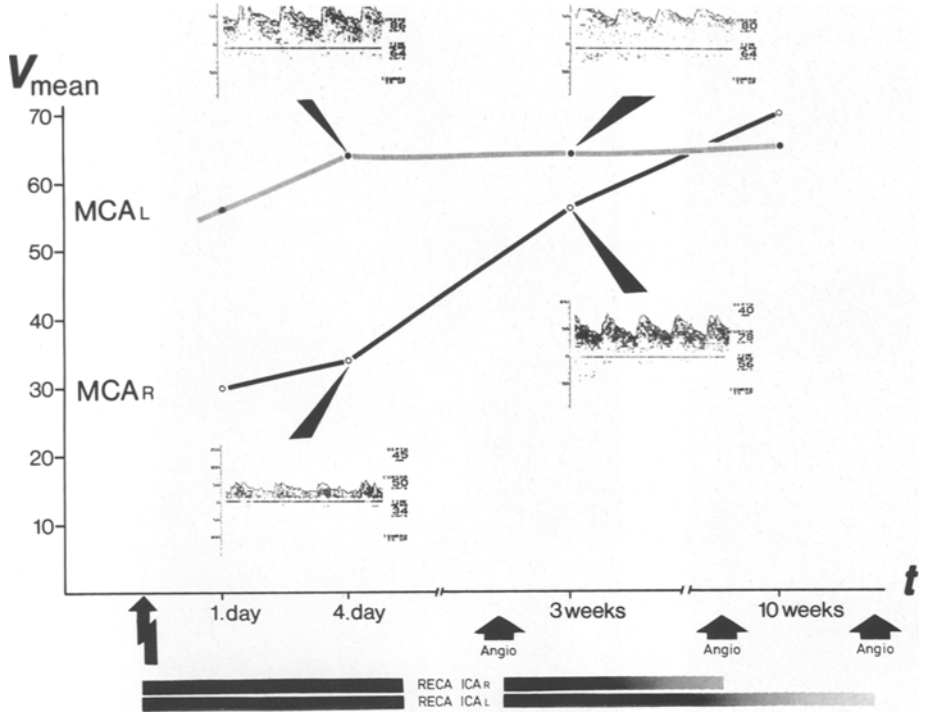

Fig. 3. Case 3: transcranial Doppler ultrasound (TCD) examinations initially revealing decreased flow velocity and pulsatility in the right middle cerebral artery *(MCA R)*. MCA flow velocity is virtually symmetrical 2 months later. Recanalization *(RECA)* of first right, then left internal carotid artery *(ICA)* was seen in angiographic follow-up examinations (*arrow* = onset of hemisphere symptoms)

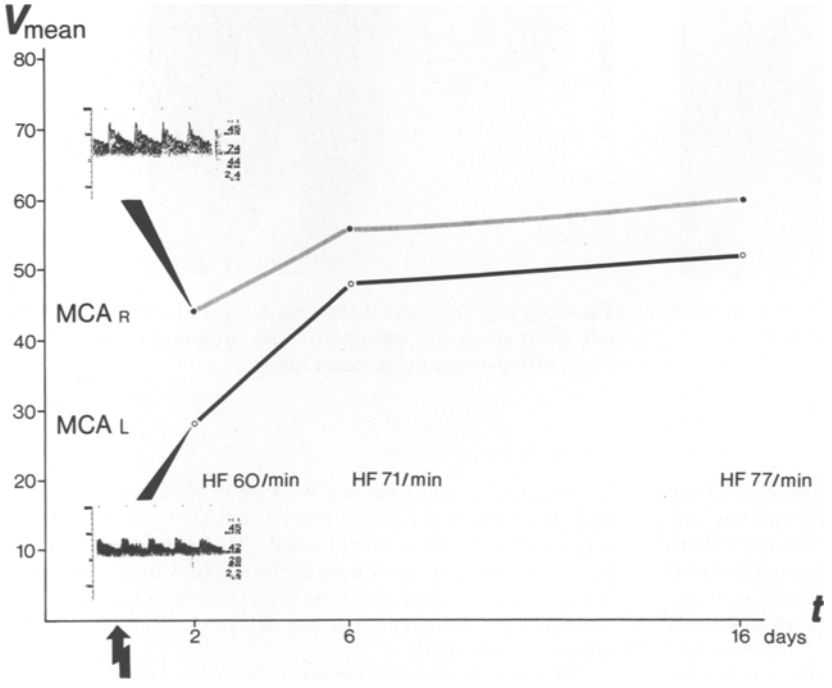

Fig. 4. Case 4: TCD on the 2nd day after the onset of symptoms showing a reduction of flow velocity in the left middle cerebral artery *(MCA L)* by over 55% compared with the right. A decline in asymmetry was seen over the following weeks (*arrow* = onset of hemisphere symptoms)

Extracranial Doppler sonography demonstrated occlusion of the left ICA with retrograde flow in the supratrochlear artery.

On TCD 10 days after the onset of symptoms flow conditions in the left and right MCA were found to be symmetrical. Compression of the left and right CCA had no effect on the flow profile on the left MCA. The perfusion of the left ACA was orthograde. Correspondingly, left PcomA flow was markedly accelerated with pulse curve irregularities suggesting turbulence.

These findings indicated good compensation of a left ICA occlusion via the PcomA with an insufficient ACoA anastomosis. The TCD was confirmed by angiography, which also showed an irregular right VA calibre at the C1 level suggesting fibromuscular dysplasia (Fig. 5). CT demonstrated a contrast-enhancing left cortical zone of infarction. The left ICA recanalized spontaneously during the following 2 weeks. Follow-up using Doppler sonography and digital subtraction angiography (DSA) documented normalization of the haemodynamics in the carotid circulation.

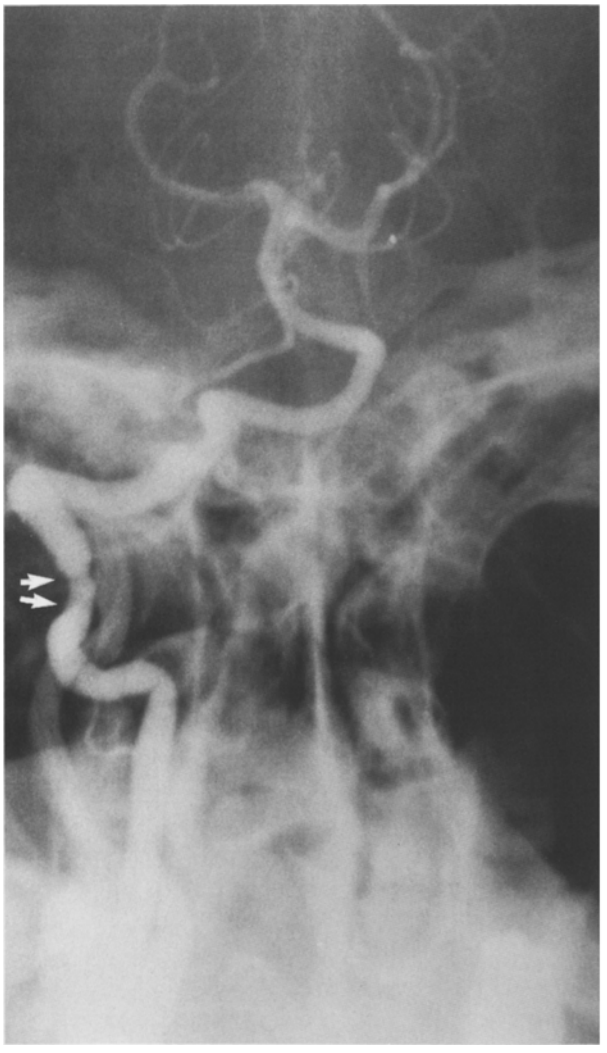

Fig. 5. Case 5: angiogram showing irregular right vertebral artery caliber *(arrows)* suggesting fibromuscular dysplasia

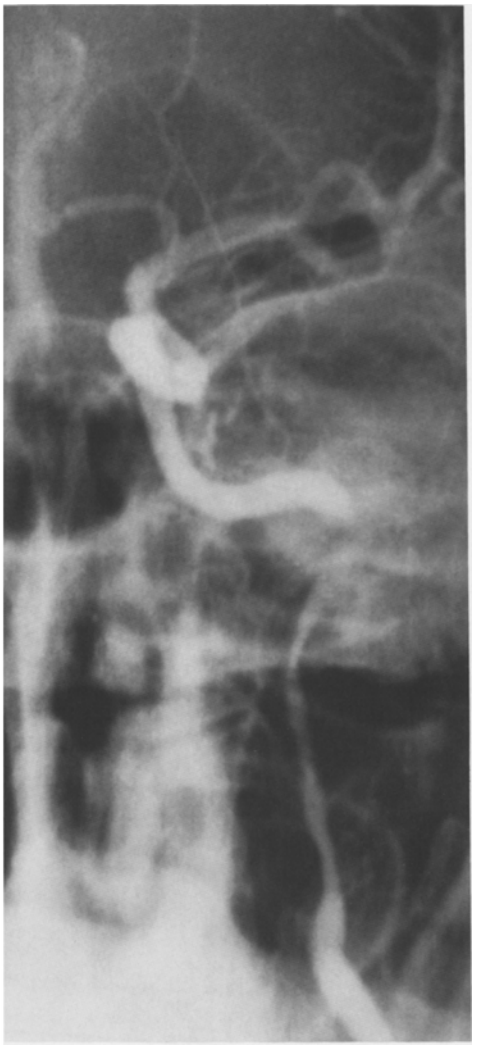

Fig. 6. Case 6: angiogram showing a tapered narrowing of the left internal carotid artery for 6 cm proximal to the carotid canal with delayed intracranial filling

In summary, the patient suffered a left-sided carotid occlusion manifesting as a progressive stroke. A TCD examination, carried out 11 days after the onset of symptoms, was not able to demonstrate any effects of the carotid occlusion on intracranial haemodynamics. The spontaneous recanalization in conjunction with the abnormal right VA suggest a diagnosis of arterial dissection. The neurological deficits resolved incompletely during the following 5 weeks under haemodilution and thrombocyte aggregation inhibitors leaving moderate dysarthria and mild facial weakness.

Case 6. This 57-year-old patient first noticed blurred vision of the left eye, followed by right-sided weakness and speech disorder lasting 4–5 h. Severe right-sided weakness occurred the next day in conjunction with bradycardia. The symptoms progressed further after admission to hospital, resulting in a severe right hemiparesis and global aphasia with a Horner syndrome on the left. No vascular risk factors or traumatic events could be found on questioning.

On the day of admission CW Doppler sonography was normal, but later a resistance profile and flow reversal in the supratrochlear artery were noted. The angiogram demonstrated a concentric narrowing of the left ICA over a stretch of 6 cm proximal to the base of the skull (Fig. 6). Filling of the intracranial carotid circulation was delayed compared with the extracranial branches.

The first TCD examinations (Fig. 7) showed a severe decrease in left MCA flow velocity and pulsatility.

The left ICA recanalized during the next 4 weeks, and the left MCA and supratrochlear artery flow returned to normal. An early CT scan did not demonstrate a definite zone of infarction, but magnetic resonance imaging (MRI) showed a sharply delineated ischaemic lesion in the left MCA – ACA watershed zone.

A slow recovery was made under haemodilution and treatment with salicylates. On discharge 8 weeks later the patient had a right spastic hemiparesis mainly of the arm as well as residual expressive aphasia.

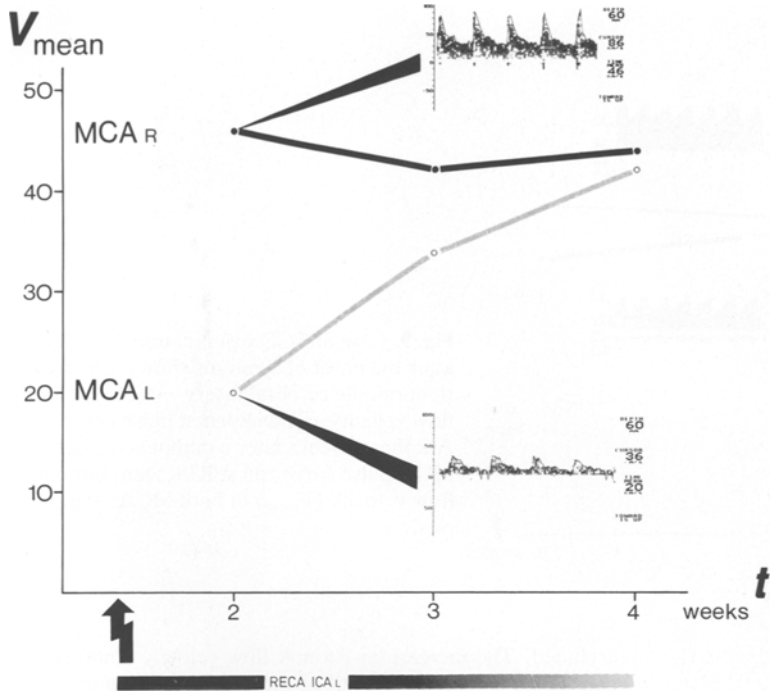

Fig. 7. Case 6: TCD examination initially showing a decrease in left middle cerebral artery *(MCA L)* flow velocity and pulsatility compared with the right *(MCA R)*. Normalization is seen during the following 4 weeks, paralleled by recanalization of the left internal carotid dissection *(RECA ICA L)*

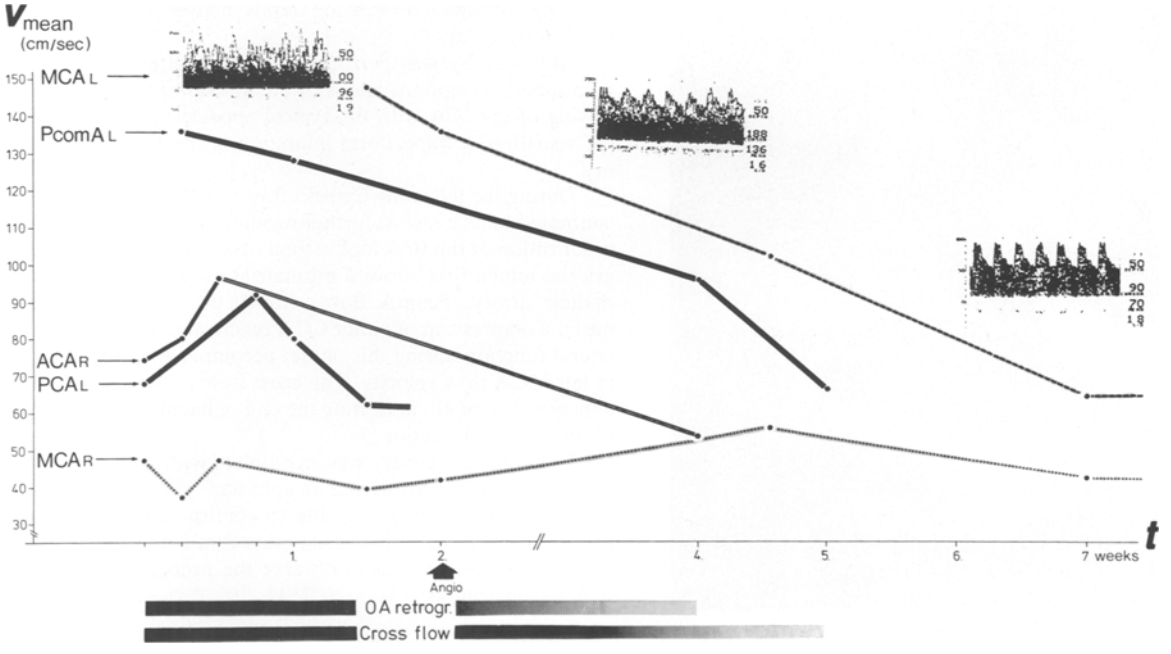

Fig. 8. Case 7: TCD examination on the day of admission showing high flow velocity in both the left posterior cerebral artery *(PCA)* and right anterior cerebral artery *(ACA)*; the left middle cerebral artery *(MCA L)* is not demonstrable. Twenty-four hours later a stenotic signal is recorded from the left MCA; the signal from the left posterior communicating artery *(PcomA L)* is increased. Collateral flow *(ACA_R, PCA_L)* increases during the first few days. Normalization of cerebral blood flow takes place during weeks 2–7. *OA*, Ophthalmic artery

This case demonstrates the typical constellation of infarction due to haemodynamic upset.

Case 7. While driving home from a skiing vacation, this 40-year-old patient complained of a severe left-sided headache; her left eye was reddened. The complaints improved temporarily, but she then collapsed at work 2 days later. No vascular risk factors were found.

On admission she had a severe right sensory and motor hemiparesis, global aphasia, right hemianopia and a left Horner syndrome. A left ICA occlusion with retrograde supratrochlear flow was seen on CW Doppler sonography. TCD on the

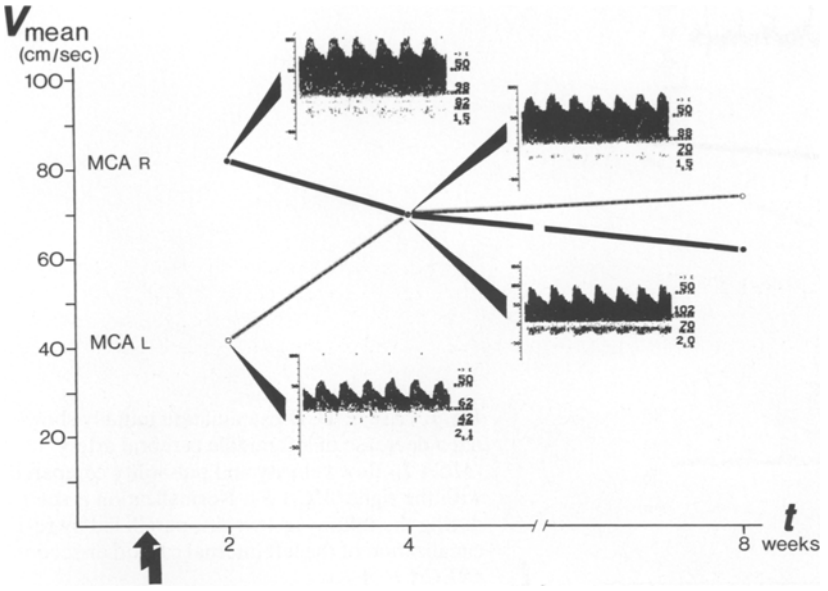

Fig. 9. Case 8: TCD examination 2 weeks after the onset of symtoms shows increased right middle cerebral artery *(MCA R)* blood flow velocity and dampened pulse curve. Another 2 weeks later a dampened right MCA pulse curve can still be seen, but mean flow velocity (V_{mean}) in both MCAs symmetrical

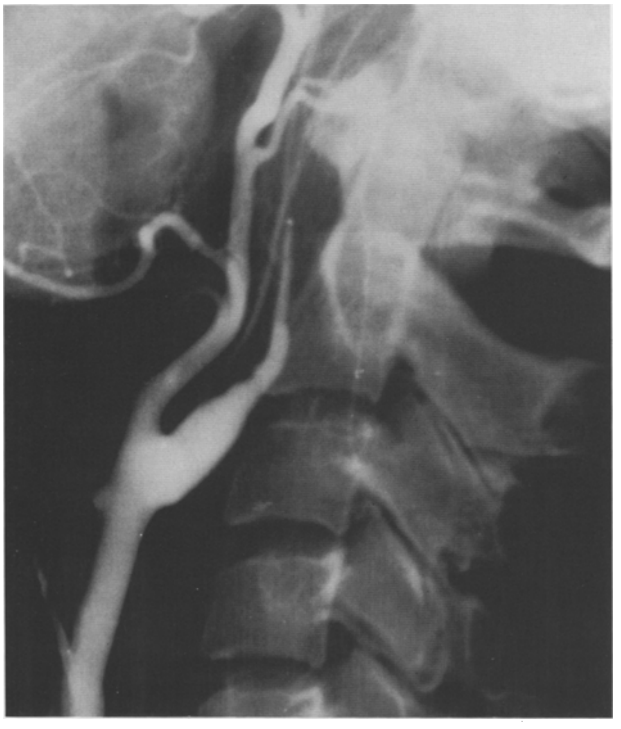

Fig. 10. Case 9: left carotid angiogram showing typical tapered occlusion

day of admission (Fig. 8) failed to demonstrate the left MCA notwithstanding a sufficient "acoustic window" of the temporal bone, whereas increased flow velocity in the left PCA and right ACA suggested activation of leptomenigeal anastomoses. Twenty-four hours later a high-frequency stenotic signal referrable to the main stem of the left MCA was recorded. A PcomA with greatly increased flow was identified at a depth of 65 mm. The stenotic MCA signal disappeared during compression of the right CCA; simultaneously, flow velocity in the left PcomA increased further. The left ICA was now completely occluded. The increase in PcomA flow velocity could be explained by a "suction effect" of the ischaemic, hypoperfused MCA territory, increasing with the progressive recanalization of the MCA. Correspondingly, flow velocity in the right ACA (A_1 segment) and left PCA collaterals increased further during the following days.

Angiography was performed 2 weeks after the onset of hemispheric symptoms and showed an extensive subtotal narrowing of the ICA with the typical appearance of dissection. CT visualized a trapeziform infarction in the left MCA territory.

During the following 6 weeks flow velocity in the left MCA continued to decrease as further recanalization took place. Recanalization of the ICA took a total of 8 weeks; the increase in arterial lumen first allowed normalization of flow in the ophthalmic artery, PcomA flow velocity decreasing simultaneously. Compression of either CCA reactivated the PcomA collateral function during this phase, accompanied by a decrease in left MCA flow velocity. The cross-flow in the AComA disappeared last of all, indicating the end of haemodynamic effect of the carotid dissection.

The clinical recovery was incomplete with persistent moderate spastic hemiparesis and an aphasia.

In summary, TCD was able to confirm the clinical diagnosis of cerebral arterio-arterial embolism without delay, and subsequently enabled us to observe the process of MCA and ICA recanalization.

Case 8. This 51-year-old patient first complained of a severe right frontal headache with nausea and vomiting. Shortly afterwards he noticed drooping of his right eyelid. Two days later a TIA with weakness of his left arm lasting 10 min occurred. He had no vascular risk factors and no history of trauma.

On examination a right Horner syndrome was observed. The right carotid artery was found to be occluded on extracranial Doppler sonography; the angiogram showed the tapered occlusion indicating carotid dissection. MRI demonstrated a small right-sided subcortical ischaemic lesion of the centrum semiovale.

The pulse curve of the left MCA was normal on TCD (Fig. 9); ACA perfusion was orthograde but accelerated. MCA flow velocity on the right (the side of the carotid occlusion) was

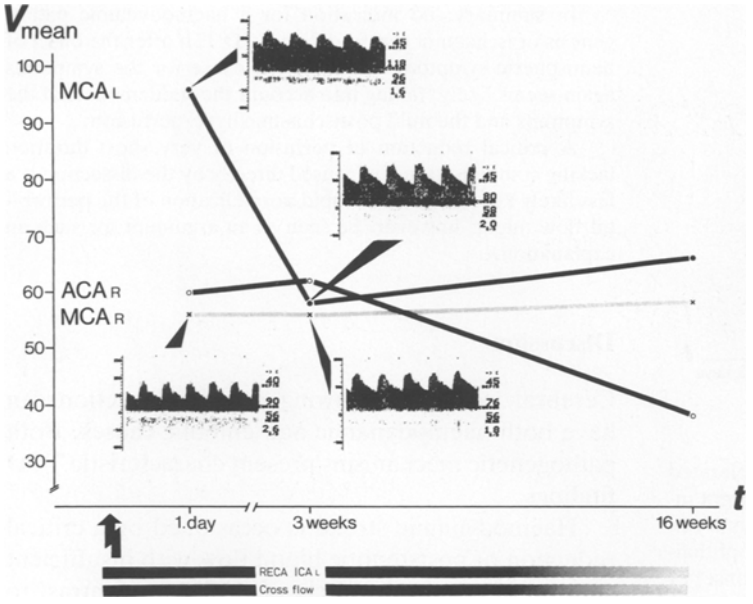

Fig. 11. Case 9: TCD examination on the 1st day of onset shows increased left middle cerebral artery *(MCA L)* blood flow velocity and reduced pulsatility with compensatory increased right anterior cerebral artery *(ACA R)* flow velocity. Three weeks later symmetrical MCA flow velocity is restored. Cross-flow is demonstrable for 16 weeks until recanalization of the left internal carotid artery *(RECA ICA L)* (*arrow* = onset of hemisphere symptoms)

markedly increased. Diastolic flow was increased, causing a dampened pulse curve. Compression of the left CCA led to an impressive decrease in right MCA flow velocity, the remaining flow indicating additional right PcomA collaterals. The turbulent signal from the retrograde right ACA disappeared on left CCA compression, confirming cross-flow.

The occluded CCA did not recanalize during 6 months under warfarin, but flow in both MCAs normalized in 4 weeks except for a slightly dampened pulse curve amplitude on the right.

Case 9. This 42-year-old patient was admitted to hospital having collapsed during the morning, subsequently developing a weakness of the right arm. During the preceding days she had complained of pain in the left angle of the jaw and left side of the neck, for which she had consulted a dentist; also her left eye had "grown smaller". She had no vascular risk factors, but had been on contraceptives for years.

On examination she was somnolent, with a left Horner syndrome, expressive aphasia and a severe right hemiparesis with pyramidal signs.

Extracranial Doppler sonography proved occlusion of the left ICA; on angiography the occlusion showed the features of dissection (Fig. 10).

CT revealed a left-sided subcortical infarction in the territory of the lenticulostriate arteries.

The initial TCD (Fig. 11) showed a marked increase in flow velocity and a decrease of pulsatility in the left MCA, together with typical signs of AcomA collateralization. Three weeks later velocity and pulsatility in the main stems of the left and right MCA were equal. Cross-flow owing to the persisting ICA occlusion could still be demonstrated for another 16 weeks; DSA then showed ICA recanalization. TCD findings were normal from then on. The patient's recovery on anticoagulant medication was excellent, with a discrete clumsiness on the right persisting after 16 weeks.

In conclusion, the course of the disease was explained by MCA embolism with subsequent spontaneous resolution and postischaemic hyperaemia.

Case 10. The family of this 56-year-old patient heard him fall and found him lying on the floor paralysed. Two days previously he had complained of pain over the right eye. There was no injury in the history.

On examination he was somnolent with deviation of head and eyes to the right. He had a severe left motor and sensory hemiparesis with a complete paralysis of the left arm. A right Horner syndrome with anhydrosis of the right side of the face was observed.

On extracranial Doppler sonography the right ICA was seen to be occluded and the perfusion of the periorbital arteries was retrograde.

The angiogram showed a narrowing of the contrast column distal to the carotid bifurcation, tapering to complete occlusion below the base of the skull. The right MCA could not be demonstrated, and the right PCA originated from the carotid syphon but filled via a tenuous PcomA.

The right MCA could not be located by TCD either. Flow velocity and pulsatility in the terminal right carotid was markedly reduced in comparison with the left. The PcomA showed increased flow velocity as well as turbulence. Perfusion conditions in the left carotid circulation were normal except for increased flow velocity in the A_1 segment of the ACA, suggesting leptomenigeal collateralization. Five days after the onset of symptoms the occlusion recanalized and a steady increase in flow velocity was seen in the terminal right ICA, supplying only the ACA. Eight days later extracranial Doppler sonography demonstrated recanalization of the ICA with orthograde flow in the supratrochlear arteries. This was confirmed by DAS. Correspondingly, flow velocity in the terminal right ICA and in the orthograde A_1 segment of the right ACA had increased, whereas the MCA remained occluded.

An extensive lenticular infarction was seen on the CT scan.

In summary, considering the prodromal headache together with the angiological findings, we concluded that the right ICA dissection was complicated by a periocclusive embolism to the right MCA. Recanalization of the extracranial cerebral arteries was observed within 1 week, but the MCA occlusion persisted during a 6-week follow-up. The patient was treated with

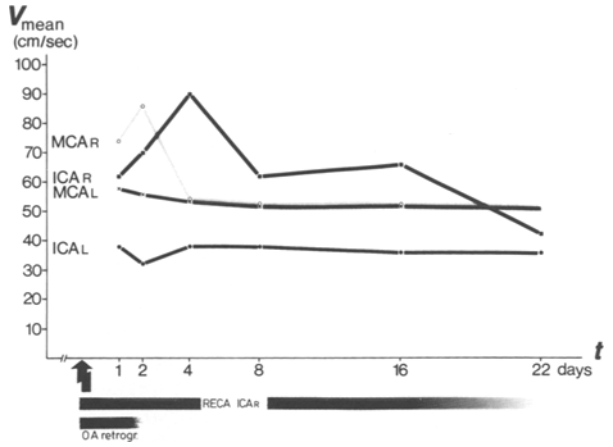

Fig. 12. Case 11: initial TCD examinations shows accelerated flow in the right middle cerebral artery *(MCA R)* and right interal carotid artery *(ICA R)* compared with the left. This asymmetry increases during the first 3 days, but the right ophthalmic artery *(OA)* is orthograde already 12 h after the onset of hemisphere symptoms. Normal intracranial flow conditions are restored 4 days after the onset of symptoms. The right ICA is completely recanalized approximately 3 weeks later

haemodilution for 2 weeks followed by anticoagulation, but his recovery was incomplete.

Case 11. During the morning the patient had worked in his garden carrying heavy weights. Around midday he noticed sudden pain over his right eye immediately followed by paralysis of his left side.

On admission to hospital he had a left sensory and motor hemiplegia with anosognosia.

The findings on CW Doppler sonography were flow reversal in the right ophthalmic artery and flow acceleration in the ICA segment near the bifurcation. The further course of the artery was evaluated with a transcranial ultrasound probe, which showed an acceleration of flow to more than 1.5 times the normal velocity and a marked decrease in pulsatility. Angiography revealed a stenosis of the right ICA tapering over a stretch of 8 cm length. The intracranial vessel contrasted poorly and could not be evaluated with certainty. Repeated CT scans were normal.

On TCD examination flow velocity in the right MCA was accelerated in comparison with the opposite side owing to an increased diastolic flow component (Fig. 12). The flow in the right ACA was orthograde, and nothing indicated compensating acceleration of flow was seen in the posterior circle of Willis.

Blood flow in the right MCA was stopped by compression of the right CCA; this disproved a complete carotid occlusion, but also indicated an insufficient collateralizing potential of the circle of Willis.

Neurological deficits resolved completely during the following 12 h and the flow direction in the right ophthalmic artery normalized. There was a transient increase of flow velocity in the dissected ICA segment and MCA at first due to reduced peripheral resistance. TCD findings were normal 4 days after the first signs of cerebral ischaemia. Doppler sonographic follow up showed complete resolution of the dissection during the subsequent 3 weeks.

In summary, no indication for a haemodynamic pathogenesis of ischaemia was found on TCD 12 h after the onset of hemispheric symptoms. An embolic cause for the symptoms again seems likely, taking into account the sudden onset of the symptoms and the mild postischaemic hyperperfusion.

A critical reduction of perfusion of very short duration lacking compensation and caused directly by the dissection is a less likely alternative. The rapid normalization of the periorbital flow might, however, be seen as an argument for such an explanation.

Discussion

Cerebral ischemia following carotid dissection can have both haemodynamic and embolic causes. Both pathogenetic mechanisms present characteristic TCD findings.

Haemodynamic stroke is occasioned by a critical reduction of poststenotic blood flow with insufficient compensation via the circle of Willis. In contrast to atherosclerotic arterial stenosis, dissection narrows the lumen of the vessel rapidly, leaving little time for an intracranial collateral system to adapt to altered haemodynamic conditions. Moreover, dissection of an extensive segment of the vessel is of especial relevance, since haemodynamic impairment depends not only on the degree of extracranial stenosis, but also (as defined by the law of Hagen-Poiseulle) on its length and on the reduction of pressure gradient. TCD accordingly shows severely reduced flow velocity and pulsatility in the main MCA stem (cases 3, 6). Conversely, flow velocity in the communicating arteries (as far as they may exist) is markedly increased with pulse curve irregularity indicating exhausted collateral transport capacity. If neurological deficits have not appeared at this point, bradycardia may finally trigger clinical symptoms (case 6).

During the further course flow velocity in the main stem of the MCA increases again owing the recruitment of collateral sources of perfusion. The clinical manifestations of haemodynamic infarction evolve progressively, with a watershed pattern seen on CT.

The majority of cerebral ischaemic events in conjunction with carotid dissection had an embolic basis (cases 2, 7, 8–10). The diagnosis of embolic MCA occlusion by TCD is justified if the acoustic window allows recording of ACA and PCA signals, but the MCA cannot be discovered (cases 7, 10) (Mattle et al. 1988). In a typical case the blood flow in the nonembolized basal cerebral arteries is accelerated owing to activation of leptomeningeal anastomoses. Occlusion of individual branches of the MCA eludes Doppler sonographic demonstration especially with proximal extracranial stenosis. In follow up TCD studies persisting MCA occlusion (case 10) or recanalization (case 7) can be documented.

Two patients exhibited markedly increased flow velocity in the postocclusive MCA compared with the opposite side. Sonographic follow-up and angiography excluded stenosis of the MCA stem as the explanation; neither could a retrograde ACA with high flow velocity owing to collateral perfusion be confused with the vessel in question. The most plausible explanation for an accelerated systolic and diastolic blood flow in the MCA is dilatation of peripheral arterioles causing reduction in peripheral resistance. A parallel phenomenon is the so-called luxury perfusion syndrome seen after cerebral ischaemia especially following rapid recanalization of arterial obstruction, due to resolving embolism (Lassen 1966; Olsen et al. 1981). The prerequisite is a sufficient collateral supply, which was the case with both patients. The characteristic history in the context of carotid dissection suggested an embolic complication. In two cases cranial CT demonstrated lenticular infarction (cases 9, 10) indicating transitory obstruction of the lenticulostriate arteries. In two other cases MCA branch occlusion occurred (cases 7, 8).

The history and findings in diagnostic imaging did not enable definite separation of a haemodynamic from an embolic cause of infarction in four cases (cases 1, 4, 5, 11). Clinical circumstances suggested haemodynamic causes, but CT was normal or featured MCA branch occlusion. TCD led to only limited clarification of the pathogenesis, as the examination was carried out several days after the onset. Therefore only guarded statements concerning the initial haemodynamic situation were possible. At the time of examination a significant alteration in flow velocity was not demonstrable in either MCA main stem. It is remarkable that in case 5 the situation of perfusion in both MCAs 11 days after the onset of hemispheric symptoms was quite symmetrical despite unilateral carotid occlusion, reflecting adequate collateral capacity of the circle of Willis.

Neurological symptoms in case 4, too, evolved progessively during 24 h. On the 2nd day of illness reduced flow velocity compared with the normal side was seen; however, this was not in the critical range (below 30 cm/s; Halsey 1988). The zone of infarction demonstrated by CT represented the area supplied by a posterotemporal MCA branch. Case 11 shows how rapidly recanalization of the carotid stenosis can take place following the ischaemic event: Flow in the ophthalmic artery, which initially was retrograde, normalized by the 2nd day of illness.

Infarction restricted to the territory of individual MCA branches in patients without cerebral atherosclerosis as an additional local factor cannot be explained simply by haemodynamic failure following carotid dissection. In such cases watershed infarction (case 6) and severely reduced flow velocity in the MCA are to be expected. Since a progessive or initially fluctuating course is atypical for cerebral embolism, more complex explanations must be taken into consideration.

Seemingly contradictory findings in clinical examination and in diagnostic imaging are most readily explained by a combination of haemodynamic and embolic mechanisms (Torvik 1984). Microscopic emboli originating from dissection can constitute a point of origin for thrombi. A poststenotic decrease in flow velocity visible on TCD favours propagation of such "stagnation thrombi" in a retrograde direction. Thus, proximal arterial branches would be successively involved in the ischaemic process. Another possibility is that perfusion in the territory of an MCA branch occluded by embolism can initially be maintained via leptomeningeal anastomoses. With progressive dissection reducing perfusion pressure, the system of collateral compensation may finally collapse stepwise, thus producing branch infarction with the clinical appearance of progressive stroke.

Recent therapeutic concepts have been influenced by increasing recognition of the embolic component in ischaemic deficits following dissection (Hart and Easton 1986). For this reason anticoagulants and thrombocyte inhibitors are advised on a defined time base depending on recanalization in spite of the haemorrhage into the arterial wall in dissection (Chapleau and Robertson 1981; McNeill et al. 1980). In extensive cerebral infarction it is necessary to wait for a decrease in the risk of secondary haemorrhage. Therapeutic efforts aiming at an improvement of microcirculation are called for immediately following the first manifestations of infarction.

TCD shows that the haemodynamic situation during the course of carotid dissection can change from day to day. In the acute phase the basis for therapeutic procedures aiming at amelioration of cerebral perfusion changes correspondingly. For example, an attempt to increase cerebral perfusion is promising with a stagnating blood flow. The rationale for such measures is questionable if the vessel has already recanalized or if state of hyperaemia has supervened (Grotta 1987). Haemodilution may enhance postischaemic oedema in this cases (Lassen 1966).

In contrast to angiography, TCD can be performed as often as required and permits insight into the state of perfusion in the basal cerebral arteries at any chosen time. It can also help to improve the rationales of differential therapy which have until now had a mainly empirical basis.

TCD helps to obtain an opinion of the cerebral arteries if judgement using angiography alone is difficult owing to poor contrast distal to a carotid ob-

struction. Compared with TCD, angiography usually features only a single "action-shot". Repeated invasive investigations may not be feasible because of considerable risk to the patient.

Sonographic serial studies, starting immediately after onset of ischaemia, therefore provide the most valuable information. Relative alterations in flow velocity can be monitored accurately on repeated follow-up studies. Conclusions drawn from singular examination are of less certain validity owing to considerable individual variability of TCD parameters.

In this study the patients came to admission after varying intervals after the presumed onset of carotid dissection.

Regular intervals of investigation are difficult to establish, because individual haemodynamic changes during the course of the disease are obviously highly variable.

The prognosis of carotid dissection varies widely in the literature according to differing criteria of selection (Bogousslavsky et al. 1987; Shuster et al. 1985).

In our series a diagnosis of TIA was made in only one case. Nine patients suffered cerebral infarction, with severe neurological deficits in all cases excepting one. The satisfactory reversal of symptoms in five of the eight severe cases despite extensive infarction demonstrated on the CT scan is a notable point. This might be explained by the low age of our patients compared with other stroke victims and the consequent lack of additional atherosclerotic changes.

Recanalization of the ICA did not influence the prognosis, as the patients' fate is decided much earlier.

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