

Transient stenoses and occlusions of main cerebral arteries in children – diagnosis and control of therapy by transcranial Doppler sonography

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Abstract. Flow disturbances in main cerebral arteries may cause severe neurological symptoms. Using transcranial Doppler sonography (TCD) the blood flow velocities in the basal cerebral arteries (BCA) can be recorded at any age. Transient stenoses or occlusions of main cerebral arteries were detected in 11 children by this method and confirmed by other techniques. Vasospasm produced a marked increase in flow velocities in the affected arteries which was reduced by nimodipine, the calcium channel blocker. Vasospasm also occurred in severe bacterial meningitis. In acute hemiplegia due to cerebral arterial obstruction no flow velocities could be recorded at the corresponding site. If distal branches were obstructed reduced flow velocities were found proximally. Increased flow velocities or reversed flow in anastomoses indicated the collateralization. The transient nature of the occlusions was shown by repeated recordings. TCD is a reliable, noninvasive and rapidly available technique for diagnosing or excluding transient flow disturbances in the main cerebral arteries as the cause of neurological symptoms in children. It indicates the necessity and most advantageous stage for therapy.

Key words: Transcranial Doppler sonography – Children – Subarachnoid haemorrhage – Meningitis – Hemiplegia

Introduction

Dramatic neurological symptoms result from acute local disturbances of blood flow in main cerebral arteries. They occur after subarachnoid haemorrhage, in some cases of bacterial meningitis and in acute hemiplegia [3, 16, 17, 19].

Angiography is an established and reliable technique for diagnosing local involvement of cerebral arteries. Limited availability, invasiveness, the general anaesthesia required in younger children and possible side-effects restrict its application.

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Abbreviations: ACA = anterior cerebral artery; BCA = basal cerebral artery (-ies); CT = computed tomography; ICA = internal carotid artery; MCA = middle cerebral artery; MFV = mean peak flow velocity; PCA = posterior cerebral artery; TCD = transcranial Doppler sonography

In contrast, transcranial Doppler sonography (TCD) which records the flow velocities in the basal cerebral arteries (BCA) is rapidly available, noninvasive and has no known side-effects. TCD has been used mostly in adults [1, 2, 14, 22, 25]. In children the thinner bones facilitate the ultrasound waves to penetrate the skull and thus make the application of TCD easier [5].

TCD was used in this study to detect transient flow disturbances in the main cerebral arteries as the cause of certain acute neurological symptoms in children. We further applied TCD to monitor the course of both the diseases and therapy.

Methods

TCD was performed with an EME TC 2–64 system (EME, Überlingen, FRG) with a pulsed 2 MHz Doppler probe and an integrated Fast Fourier real time frequency analyser (sliding average technique, 64 frequency points, 132 spectral lines). Frequency to flow velocity conversions were made assuming an insonation angle of zero.

Middle cerebral artery (MCA), internal carotid artery (ICA), anterior cerebral artery (ACA) and posterior cerebral artery (PCA) were recorded through the temporal bone [1]. The basilar artery was insonated via the foramen magnum. In children under 1 year of age the ICA was recorded through the anterior fontanelle and the postcommunicating segment of the ACA from a high frontal position [5]. The measuring depth was age – dependent [5].

Systolic peak flow velocity, mean peak flow velocity (MFV), end-diastolic peak flow velocity and the ratio of systolic to end-diastolic peak flow velocity S/D were recorded. Since the behaviour of the flow velocities is similar in the reported cases, only quantitative values of MFV are given. The flow velocities were judged as elevated, normal or reduced in comparison with:

1. The age-dependent reference values and the reference range of $\pm 30\%$ [5].
2. The velocities in the contralateral artery, taking into account the variation coefficients of 2%–8% between both sides [5].
3. The flow velocities in the artery after recovery.

Computed tomography (CT), cerebral sonography, fontanometry, and angiography were performed with commercially available equipment using established techniques.

The study was approved by the hospital ethics committee.

Results

Between September 1985 and December 1987, 1,074 TCD examinations were performed on 588 children. Eleven patients showed either transiently increased flow velocities indicating acute stenosis or reduced/absent flow velocities indicating partial or complete occlusion in at least one BCA. Transiently increased velocities were found in five cases with vasospasm after subarachnoid haemorrhage from a ruptured aneurysm (2) or after intracranial bleeding due to other causes (3). In 3 out of 14 cases with bacterial meningitis and in 3 out of 12 patients with acute hemiparesis, transiently increased flow velocities were found. Eleven cases of bacterial meningitis showed increased flow velocities (130%–150% of the reference values) in all BCA. Five patients with transient hemipareses [aetiology unknown (3) Coxsackie B4 encephalitis (1), complicated migraine (1)] had no significant change of flow velocities on the affected side. Four patients with acute hemiplegia [Moya-Moya-syndrome (2), angioma of the right thalamus (1), radiation therapy (1)] showed permanent changes of flow velocities in one or more BCA. They have been presented in part elsewhere [7].

Case reports

Vasospasm

Case 1. This 9-year-old girl presented with acute signs of subarachnoid haemorrhage. Lumbar puncture revealed blood-stained cerebrospinal fluid. CT performed on the same day showed haemorrhage more into the left than into the right ventricle. Intravenous nimodipine was given continuously (1 mg/h). An increase in flow velocities was recorded only in both MCAs (Fig. 1). Five weeks later, a 3 mm aneurysm of the left PCA was detected by angiography and surgically removed. Recovery was complete.

Case 2. The 16-year-old girl presented with acute signs of subarachnoid haemorrhage, which was confirmed by lumbar puncture. CT showed haemorrhage within the right frontal subarachnoid space. Arteriography on day 15 is shown in Fig. 2. After clipping the aneurysm of the anterior communicating artery on the same day, she received nimodipine (2 mg/h) for 7 days. TCD on day 22 and 28 showed increased flow velocities only in the right precommunicating segment of the ACA (MFV 72 and 104 cm/sec). Five months later the MFVs in the right/left ACA were 44/40 cm/sec. The girl made a complete recovery.

Case 3. This 14-year-old boy developed a right frontal chronic subdural haematoma, which was confirmed by CT and removed surgically. TCD showed increased flow velocities only in the right MCA for 2 weeks (Fig. 3a). Angiography on day 10 is shown in Fig. 3b. Eleven weeks after the operation, normal flow velocities in both MCAs were seen. Recovery was complete.

Case 4. A 10-year-old boy suffered from a large astrocytoma of the right temporobasal lobe. TCD carried out 18 days after surgery revealed an increased MFV of 160 cm/sec (twice the norm) only in the right MCA, which decreased under nimodipine (1.5 mg/h) to 120 cm/sec. After discontinuing nimodipine, a short increase to 160 cm/sec was followed by a spontaneous reduction to normal (85 cm/sec). Left facial weakness and focal seizures persisted.

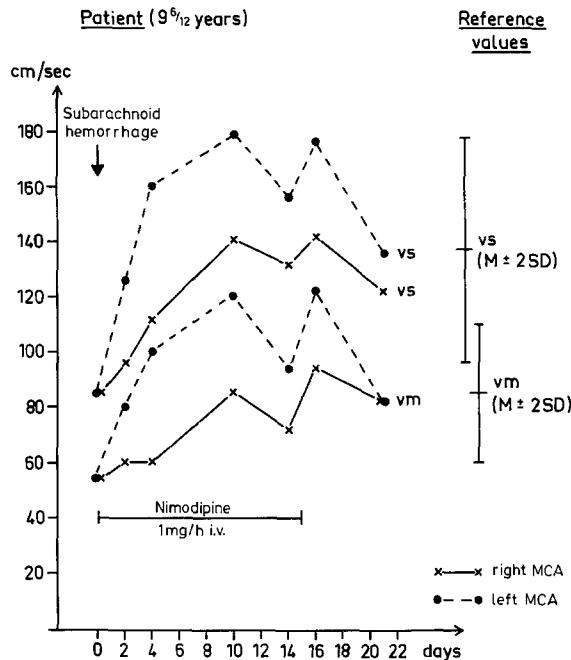


Fig. 1. TCD recordings of systolic (vs) and mean (vm) peak flow velocities in the left and right MCA after subarachnoid haemorrhage from an aneurysm of the left PCA (case 1)

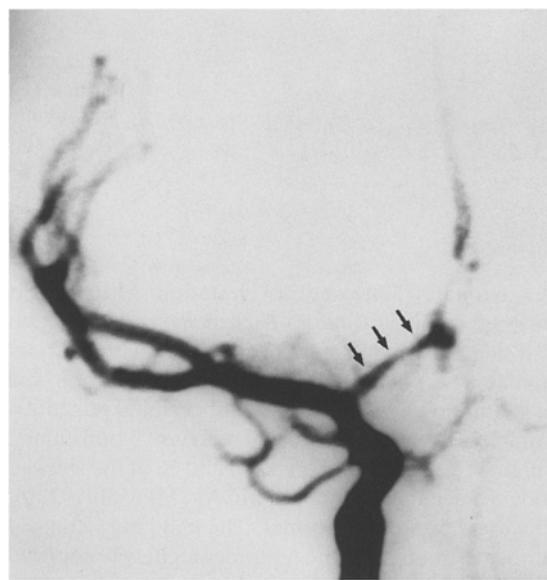


Fig. 2. Subtraction angiography showing spasm of the precommunicating segment of the right ACA after subarachnoid haemorrhage from an aneurysm of the right anterior communicating artery (case 2)

dipine, a short increase to 160 cm/sec was followed by a spontaneous reduction to normal (85 cm/sec). Left facial weakness and focal seizures persisted.

Case 5. This 6-year-old boy showed multiple intracerebral haemorrhages due to severe head injury. When he was transferred to our hospital on day 11, the flow velocities only in both MCAs and ICAs (MFV 160 cm/sec) were twice normal levels (MFV of 84 cm/sec). Normal velocities were recorded 11 days later. During the following months, the boy remained in a vegetative state.

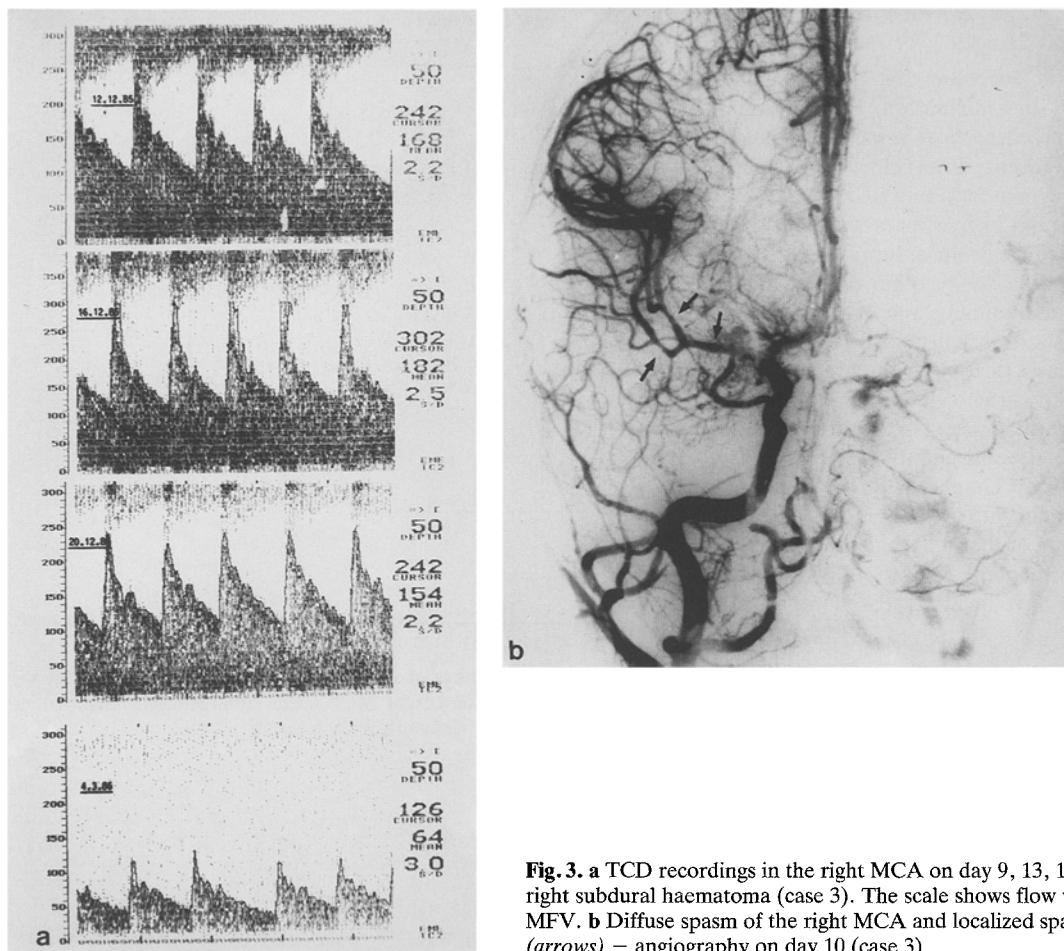


Fig. 3. a TCD recordings in the right MCA on day 9, 13, 17 and 101 after operation of a right subdural haematoma (case 3). The scale shows flow velocities in cm/s; MEAN = MFV. b Diffuse spasm of the right MCA and localized spasms of its two main branches (arrows) – angiography on day 10 (case 3)

Bacterial meningitis

Case 6. This neonate (37th week of gestation, birth weight 3150g) developed meningitis due to *Escherichia coli* on the 5th day of life, which persisted in spite of treatment with ceftriaxone. Treatment with phenobarbitone, muscle relaxation and mechanical ventilation were necessary. Suboccipital punctures on day 3 and 7 of treatment revealed viscous, bloodstained yellowish fluid. TCD performed on day 6 and 12 of life showed increased flow velocities only in both MCAs (MFV 80/192 cm/sec), which were 2.5–5 times normal. The child died at age 4 weeks. Autopsy showed subacute leptomeningitis pronounced in the basal cisterns, an old haemorrhage at the meninges, and a multicystic leucomalacia of both hemispheres with diffuse hypoxic-ischaemic damage to gangliocytes.

Patient 7. This 8-month-old girl presented with meningitis due to *Diplococcus pneumoniae*. Despite therapy with penicillin G and chloramphenicol she was comatose for ten days with convulsions and respiratory insufficiency. Phenobarbitone and mechanical ventilation were applied. TCD showed increased flow velocities only in both MCAs (Fig. 4), whilst fontanometry revealed normal pressure. Cerebral sonography on day 5 of the disease showed infarctions of both lateral thalamic regions. Six months later, the child presented with severe mental retardation and hypotonic cerebral palsy.

Case 8. This 1-year-old boy (31st week of gestation, posthaemorrhagic hydrocephalus with normal pressure) presented

with meningitis due to *Diplococcus pneumoniae*. Despite treatment with penicillin G and chloramphenicol he developed coma, respiratory insufficiency and left hemiparesis. The CT of day 3 is shown in Fig. 5. TCD of the right/left MCA on day 2, 3, 6 and 47 of the disease showed MFVs of 128/116, 168/120, 184/114 and 70/66 cm/sec (reference value 79 cm/sec). Normal pressure was determined in repeated ventricular punctures between day 3 and 25. One year later the child presented with severe psychomotor retardation.

Thromboembolism

Case 9. This 8-year-old boy developed right hemiplegia and motor aphasia. CT performed on day 2 showed infarction of the area supplied by the left lenticulostriate artery. TCD carried out on day 2 showed reduced flow velocities in the left (MFV 64 cm/sec) compared with the right (MFV 92 cm/sec) MCA and increased velocities in the left PCA (MFV 152 cm/sec; reference value 57 cm/sec). On the left an atypical vessel above the MCA with high retrograde flow velocities (MFV 152 cm/sec) was identified. The angiography performed on day 30 is shown in Fig. 6. One year later a slight hemiparesis, concentration and reading difficulties persisted. TCD showed normal velocities in both MCAs and PCAs.

Case 10. This 10-year-old boy developed right sided hemiplegia, confusion and speech problems. TCD performed on day 4 was unable to detect any flow in the left MCA. In the

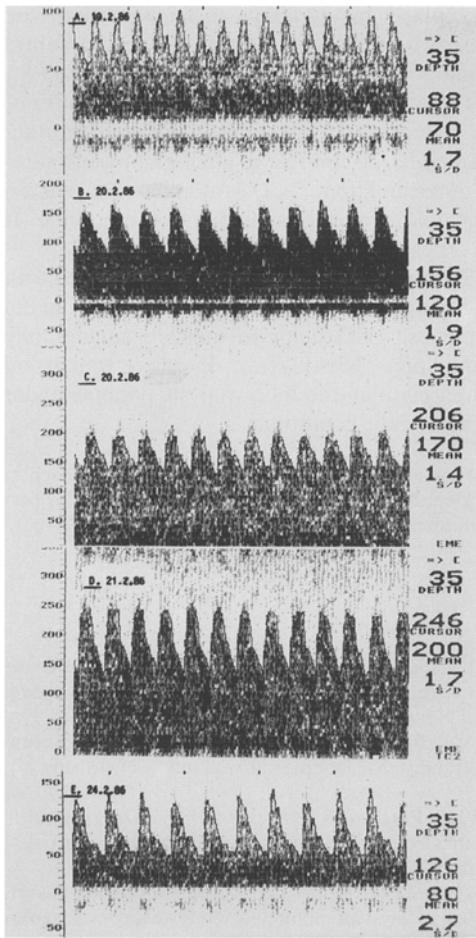


Fig. 4. TCD recordings of left MCA on day 3, 4, 5 and 8 of bacterial meningitis (case 7). The scale shows flow velocities in cm/sec; MEAN = MFV



Fig. 5. Ischaemic infarction of the right basal ganglia in CT on day 3 of bacterial meningitis (case 8)

left ACA a retrograde flow was found, the MFVs in the left/right PCA were 70/50 cm/sec. 3 days later the flow velocities in the left/right MCA were almost identical (MFV 52/58 cm/sec). CT performed on day 5 showed infarction of the region supplied by the left MCA. Angiography done on day 19 revealed no significant anomaly. Hemiparesis and dysarthria persisted.

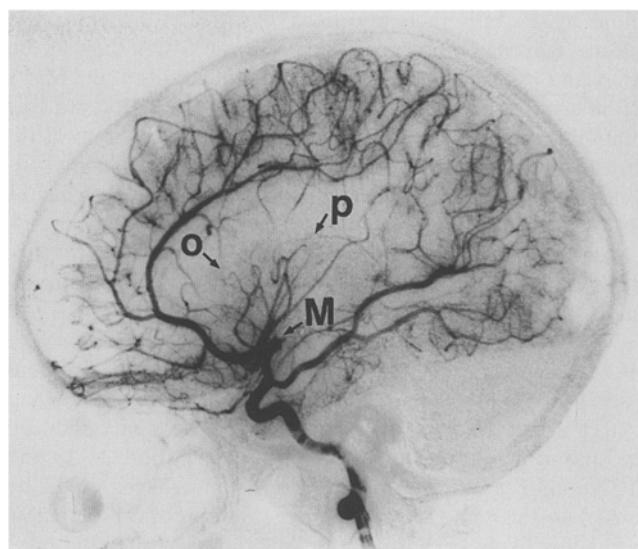


Fig. 6. Angiography showing subtotal occlusion of the left MCA (M), orthograde perfusion of the opercular (O) and parietal (P) branches and retrograde filling of the other branches of the MCA by cortical anastomoses (case 9)

Case 11. This term neonate (birth weight 2520 g) with perinatal asphyxia developed persistent fetal circulation. Cerebral sonography performed on day 2 of life showed infarction of the upper part of the right parietal region. TCD recorded MFVs of 8/20/34 cm/sec in the right and of 10/32/50 cm/sec in the left MCA on day 2, 4 and 7 of life. Widespread hypodense areas in the right hemisphere were seen in CT on day 20. The child had developed normally until the age of 15 months.

Discussion

The inverse relationship between blood flow velocities and cross-sectional area of a vessel is a principle of haemodynamics. Vessel narrowing causes an increase in flow velocities. In BCA, this can be studied noninvasively using TCD. The principle and examination technique have been discussed elsewhere [1, 5, 14].

Different diseases can cause stenoses and occlusions of the BCA [6, 7, 10, 11, 16, 20, 24, 28]. Until now the number of paediatric patients is too small to calculate the percentage of affected children in distinct diseases. Multicentre studies are required for this purpose.

Transient and permanent alterations of the cross sectional area of the BCA can be distinguished by repeated TCD recordings. This paper only deals with transient changes.

TCD studies in adults with subarachnoid haemorrhage revealed an incidence of BCA spasms of 70%–80% [2, 3, 14, 25]. They start at day 2, reach their maximum on day 10–17 and decrease until day 40 after the haemorrhage [17, 25]. An inverse relationship between vessel diameter in angiography and flow velocities in TCD was found [2]. The spasm and additional disturbances at the microvascular level cause a decrease of cerebral blood flow [21, 23]. Ischaemic deficits may result [3, 14]. The diagnosis of vasospasm is important for the timing of angiography and for treatment [3, 4, 8, 12, 14, 25].

We studied five children with subarachnoid haemorrhage. The time course of vasospasm was similar to that in adults. We

found significantly increased flow velocities at the site of the spasm. This was more pronounced in the MCA than in the ACA and PCA because of the lack of collaterals in the MCA [2]. A significant velocity reduction in the BCA after starting nimodipine and a rebound effect after discontinuation was recorded. With prophylactic nimodipine the flow velocities increased, but the MFV never reached a critical level, which is assumed to be between 140 and 250 cm/sec [2, 14, 25]. The effect of nimodipine on the flow velocities in the BCA after subarachnoid haemorrhage in children is, as in adults, presumably due to a reduction of vasospasm.

We recorded transiently increased flow velocities in one MCA indicating vasospasm not only after rupture of an aneurysm, but also after operations for a subdural haematoma and a large astrocytoma. Subarachnoid blood may have induced the spasm. The transient velocity increase in both ICAs and MCAs after multiple intracerebral haemorrhages could be caused in the same way. An increased cerebral blood flow seems less likely because of the long duration after the trauma, the involvement of only some arteries and the neurological sequelae. Moreover, vasospasm is a known complication after severe head injury [18]. Even traumatic occlusions of BCA may occur [10, 20, 28]. Thus we propose, that all children with severe head injury should be examined for spasm or occlusion of the BCA.

A constriction of the BCA in bacterial meningitis is known from angiographic [13, 26], intraoperative [14] and postmortem studies [15, 26]. In 3 of 14 children with bacterial meningitis we found markedly increased flow velocities in some BCA. These patients suffered from a severe acute disease and developed ischaemic encephalopathy as demonstrated by ultrasound, CT and autopsy. The survivors showed cerebral palsy and severe mental retardation. Thus the increased flow velocities are assumed to be a sign of vessel narrowing causing cerebral ischaemia and additional damage to the brain. The high risk of angiography made confirmation impossible. Increased intracranial pressure as a cause of the increased flow velocities was excluded by pressure measurements. The moderately increased flow velocities in the 11 cases of bacterial meningitis with a favourable outcome are ascribed to increased cerebral blood flow.

Acute hemiplegia in childhood can be due to haemodynamic disturbances in the main cerebral arteries. The clinical outcome not only depends on the affected vessels, but also on the capacity of the collaterals.

With TCD no flow velocities were recorded in an occluded artery. Spontaneous recanalization was documented. Increased flow velocities and/or a change of flow direction were found in typical or atypical collaterals. The obstruction of large branches distal to the recorded artery could be suspected if a significant reduction of flow velocities was found in the preceding artery of one side. Due to the small physiological difference of flow velocities in the BCA of both sides [5] TCD is suitable for detecting obstructions of large branches of BCA.

The ischaemic origin of acute hemiplegia can be recognized by TCD. However the cerebral blood flow of the affected area cannot be quantified. Thus TCD is unable to give prognostic criteria. In acute hemiplegia TCD should be complemented by Duplex-sonography of the extracranial brain supplying arteries [9]. TCD will be helpful in prospective studies of the haemodynamics in neonatal cerebral infarction [6, 20]. Continuous recording facilities have already been developed for this purpose (Bode H, unpublished).

TCD cannot replace angiography, as knowledge of the morphology of the cerebral vessels is often needed. Cerebral blood flow determination may be helpful but has a limited availability. TCD is very valuable when angiography is contraindicated or entails high risk as in high grade vasospasm or in severe bacterial meningitis. In contrast to angiography and cerebral blood flow determination, TCD is noninvasive, has no known side-effects and is quickly available at the bedside. It thus can be used repeatedly.

In summary, TCD should be used to examine children with focal neurological symptoms for a local flow disturbance in the main cerebral arteries. TCD helps to indicate angiography and medical (nimodipine, lysis [27]) or operative (aneurysm surgery [4, 14]) treatment and to follow up the patients under spontaneous or therapeutic conditions.

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