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Cerebral arteriovenous malformations and movement disorders

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Abstract

A series of six patients with movement disorders associated with cerebral arteriovenous malformations (AVM) is reported. The AVMs were classified according to the Spetzler–Martin classification as grade V (one patient), grade IV (four patients), and as grade III (one patient). One patient had action-induced hemidystonia caused by a contralateral frontoparietal AVM which compressed the putamen and was supplied partially by enlarged lenticulostriate arteries. Two patients presented with unilateral cortical tremor associated with contralateral high-frontal cortical/subcortical AVMs sparing the basal ganglia. Another patient developed hemidystonia and hemichorea-hemiballism after bleeding of a contralateral temporooccipital AVM and subsequent ischemia. Two patients had focal dystonia after thalamic and basal ganglia hemorrhage from AVMs. Five patients were operated on. The movement disorder was abolished in one patient postoperatively. Different mechanisms were identified that are relevant for the development of AVM-related movement disorders: mass effect, diaschisis, local parenchymal altered cerebral blood flow, and hemorrhagic or ischemic structural lesions. © 1999 Elsevier Science B.V. All rights reserved.

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1. Introduction

Cerebral arteriovenous malformations (AVM) most frequently become manifest by spontaneous hemorrhage or epilepsy. The annual bleeding risk has been estimated to be 2-3% with an annual mortality rate of about 1% [1]. Seizures occur in 11-33% of cases [2,3]. Other presenting symptoms include headaches, progressive neurological deficit, and cardiac failure. Movement disorders secondary to cerebral AVMs have been reported infrequently. There have been incidental reports within the past three decades on patients with dystonia, chorea or tremors associated with AVMs. In only few instances magnetic resonance (MR) imaging studies were available to delineate the exact topography of the AVMs and, in general, there have been no studies on cerebral blood flow in such cases.

The purpose of the present report is to focus better the uncommon association between cerebral AVMs and

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movement disorders. Here, the clinical and radiological findings in a series of six patients are documented. So far, this represents the largest series on movement disorders secondary to cerebral AVMs. Possible pathophysiological mechanisms with regard to the imaging findings are analyzed.

2. Patients and methods

The patients reported here were seen over a period of 20 years, from 1976 to 1996. Only patients with an unequivocal cause-and-effect relationship between the cerebral AVM and the movement disorder were accepted in this series. Patients with venous angiomas or with cavernomas were excluded. Data were collected from the patients' charts and clinical notes. The movement disorders were documented by videotaping in four instances. Computed tomography (CT) studies and cerebral angiograms were available in all patients.

Imaging findings and clinical features of the patients are summarized in Table 1. There were four men and two women. The mean age at admission was 33 years, with a range from 22 to 45 years. All patients presented with AVMs located in eloquent areas. The AVMs were classified as grade V in one patient, as grade IV in four patients, and as grade III in one patient according to the Spetzler-Martin classification [3]. In three cases (patients 4, 5 and 6) the movement disorder became manifest only after spontaneous cerebral hemorrhage. Five patients underwent microsurgical interventions. Brief descriptions of the cases are given below. We concentrate primarily on the findings relevant to the movement disorders, but not on the complex issues of surgical or interventional treatment of these therapeutically challenging AVMs. The subheadings in the following denote the most probable pathomechanisms responsible for the development of the movement disorder.

3. Summary of cases

3.1. Mass effect and hypoperfusion (diaschisis) of the basal ganglia

3.1.1. Patient 1

This 37-year-old man was admitted for treatment of a leftsided frontoparietal AVM. He suffered from generalized tonic-clonic seizures since childhood. When he was about 20 years old, he first noted 'spasms' of his right arm while he was writing. He had increasing difficulty to use his right hand. At age 34, he developed a mild gait disturbance which became worse over the next few years. On admission, general physical examination was unremarkable. Short-term memory deficits were present. Motor strength was normal. At rest, muscle tone of the right arm was mildly increased. Right hemidystonia became evident when he performed any daily routine activities with his right extremities and upon walking. Then, his arm adopted a dystonic posture with abduction in the shoulder, extension of the elbow, wrist and fingers, and radial abduction of the hand. In addition, there was plantar flexion and mild supination of his right foot accompanied by dorsal flexion of the toes. There were no sensory deficits. CT and MR studies showed a large AVM with a diameter of slightly more than 5 cm located in the left suprasylvian frontoparietal region (Fig. 1). The AVM involved the motor strip and the postcentral gyrus laterally. The nidus did not encroach the basal ganglia, but compression of the left putamen was evident. Digital subtraction angiography revealed that the AVM was fed by several large arteries branching from the middle cerebral artery and to a lesser degree from the anterior cerebral artery (Fig. 2). Small feeders branched from enlarged lenticulostriate arteries. There was both superficial and deep venous drainage. Cerebral blood flow (CBF) measurements with 99m-Tc HMPAO SPECT showed decreased parenchymal cerebral blood flow in the tissue adjacent to the AVM. The posterior and medial portions of the AVM were partially embolized via an endovascular approach in three subsequent sessions. The AVM was then considered accessible for a microsurgical approach. The postoperative course was complicated by a bleeding postoperatively. The patient died 3 weeks postoperatively due to pneumonia.

Table 1

Summary of imaging findings and clinical features in six patients with movement disorders secondary to cerebral arteriovenous malformations (AVM) ^a

| Patient/sex/age | Location of AVM | Spetzler–Martin grade | Movement disorder | Probable pathomechanism |
|-----------------|--|--------------------------|--|--|
| 1/M/37 | Left suprasylvian fronto- parietal cortex and white matter | IV | Action-induced hemidystonia (right extremities) | Mass effect and hypoperfusion (diaschisis) of the basal ganglia |
| 2/M/45 | Left high-frontal cortex and white matter | IV | Cortical tremor (right arm) | Cortical/ subcortical local parenchymal altered cerebral blood flow |
| 3/F/45 | Right high-frontal cortex and white matter | IV | Cortical tremor (left arm) | Cortical/ subcortical local parenchymal altered cerebral blood flow |
| 4/F/26 | Left medial temporooccipi- tal white matter | V | Hemidystonia, hemichorea- ballism (right extremities) | Structural thalamic/ subthalamic lesion due to posthemorrhagic vasospasm |
| 5/M/22 | Right thalamus | III | Focal dystonia (left hand) | Structural thalamic lesion |
| 6/M/22 | Right thalamus, internal capsule and basal ganglia | IV | Focal dystonia (left arm) | Structural thalamic and putaminal lesion after hemorrhage |

^a Age, age at admission; M, male; F, female.

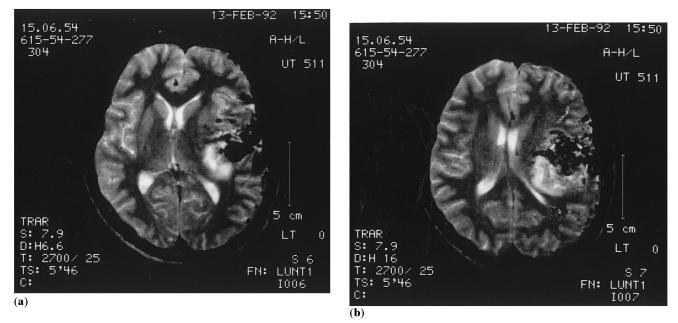


Fig. 1. Magnetic resonance (MR) images of a 37-year-old man with right action-induced hemidystonia (patient 1). (a and b) Axial 2700/25 scanning electron (SE) images after partial embolization. The arteriovenous malformations (AVM) is located in the left suprasylvian frontoparietal cortex and subcortex. The nidus does not directly involve the basal ganglia but there is compression of the left putamen.

3.2. Cortical | subcortical local parenchymal altered cerebral blood flow

3.2.1. Patient 2

This 45-year-old man developed focal and generalized epileptic seizures when he was 14 years old. He was treated with anticonvulsive medication. At the age of 21, angiography revealed a left frontal AVM. Twelve years later, he acutely developed a rightsided hemiparesis and aphasia. CT studies demonstrated a hemorrhage in the left frontal white matter. Repeat angiography showed the frontal AVM which had increased in size. In addition, a cherry-sized aneurysm of the anterior communicating artery was detected. The hemiparesis and the aphasia improved gradually. Over the next years, the patient noted occasional rhythmic involuntary shaking movements of his right arm. At the age of 44, he became more passive and withdrawn and developed a gait disturbance. CT imaging showed marked increase of the size of the anterior communicating artery aneurysm and bilateral occlusive hydrocephalus. He underwent bilateral shunting with subsequent im-

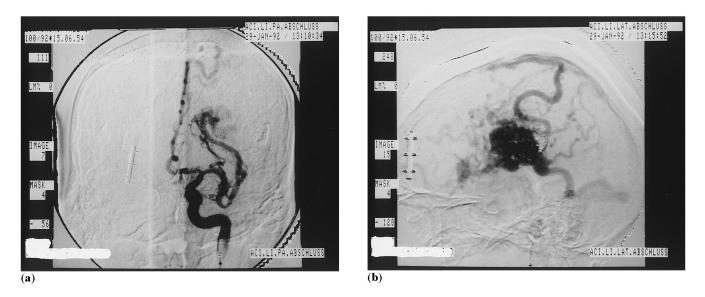


Fig. 2. Cerebral angiograms of patient 1 after partial embolization. (a) Anteroposterior, and (b) lateral views. There are feeding arteries from the left middle cerebral artery and also from enlarged lenticulostriate arteries. Venous drainage is both superficial and deep.

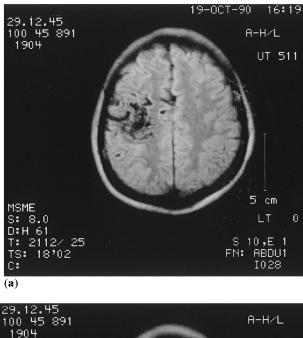




Fig. 3. Magnetic resonance (MR) images of a 45-year-old woman with cortical tremor of the left arm (patient 3). (a and b) Axial 2112/25 images. The arteriovenous malformations (AVM) is located in the rightsided frontal cortex and subcortical white matter just rostral to the precentral sulcus.

provement of his hydrocephalic symptoms. He was admitted at age 45 to our department for further diagnostic studies and treatment. He was alert and fully oriented, but had moderate memory deficits. Speech was hindered by a transcortical motor aphasia. The right eye was amaurotic. There was a moderate rightsided hemiparesis, and bilateral bradykinesia. There was no tremor at rest. When he performed any activities with his right arm a 9-Hz tremor appeared after a delay of several seconds. He had occasional focal motor seizures of his right arm despite combined anticonvulsive treatment. CT studies demonstrated an AVM located in the left frontal cortex and white matter extending just to the prefrontal sulcus. In addition, there was a partially thrombosed anterior communicating artery giant aneurysm causing distorsion and occlusion of the anterior ventricular system. Angiography demonstrated multiple arterial feeders from the left pericallosal and medial cerebral arteries to the AVM and superficial as well as deep venous drainage. The AVM had a maximal diameter of 5 cm. It was partially embolized. The lumen of the aneurysm was occluded with coils. It was decided to approach first the giant aneurysm operatively and remove the AVM in a subsequent operation. The aneurysm was clipped with multiple clips and resected via a left pterional craniotomy. The postoperative course was unremarkable. There was slight improvement of the aphasia and the hemiparesis. The postural and action tremor of the right arm was unchanged. One month postoperatively, the patient became comatose. CT scans showed a large left frontal hemorrhage in the region of the AVM and uncal herniation. The patient died several hours later.

3.2.2. Patient 3

This 45-year-old woman suffered epileptic seizures one year prior to admission. The seizures started with tingling sensations of the left arm, and turned into generalized tonic-clonic seizures. Gradually, progressive weakness of the left arm developed. After CT imaging had shown a



Fig. 4. Axial computed tomography (CT) scan of a 26-year-old woman with right hemidystonia and hemichorea-hemiballism 3 weeks after hemorrhage of a left medial temporooccipital arteriovenous malformations (AVM) (patient 4). The hemorrhage extends into the posterior thalamus. There is a hypodense lesion of the thalamus, the subthalamic region and the posterior internal capsule.

right frontal AVM the patient was referred for further evaluation and treatment. On admission, a mild left hemiparesis which was affecting the arm more than the leg was present. There was also leftsided hemihypesthesia. When the left arm adopted an antigravity posture or when the patient exerted forceful movements with the left hand a 10-Hz tremor of the left arm appeared after a delay of several seconds. The tremor stopped when she relaxed. Imaging studies revealed a right frontal subcortical and cortical AVM located just rostral to the precentral sulcus (Fig. 3). The nidus of the AVM had a diameter of about 4 cm. Angiography showed that it was fed by the right medial and anterior cerebral arteries. Both superficial and deep venous drainage was evident. Electroencephalographic studies showed a right frontal precentral focus but no interictal epileptic activity. The AVM was partially embolized in several sessions. After each embolization there was a slight increase of the leftsided hemiparesis which, however, recovered to baseline subsequently. The tremor was less notable when the hemiparesis was temporarily worse. One year later, the AVM was removed via a frontal approach. Postoperatively, the patient had a leftsided hemiplegia which recovered gradually over the next few weeks. On a follow-up examination 2 years postoperatively, the hemiparesis had considerably improved, although it was still more pronounced than preoperatively. No tremor was evoked with various manoeuvers. Angiography confirmed complete resection of the AVM. The patient was free of seizures without anticonvulsive medication.

3.3. Structural cerebral lesion due to posthemorrhagic vasospasm

3.3.1. Patient 4

This 26-year-old previously healthy woman collapsed and became comatose. She was intubated and admitted to the emergency room. CT studies demonstrated a left temporooccipital subcortical hematoma involving the thalamus and upper mesencephalon and adjacent subarachnoid hemorrhage. Angiography revealed a large AVM in the left medial temporooccipital white matter and two aneurysms of the proximal leftsided posterior cerebral artery. According to the distribution of the bleeding the AVM was considered responsible for the event. The AVM was fed by branches from the medial and posterior cerebral arteries with venous drainage to internal cerebral veins. The aneurysms were occluded with coils. In the further course severe vasospasm of the middle and posterior cerebral arteries on the left side were documented by transcranial Doppler sonography. When the patient regained consciousness 3 days later, complex oculomotor disturbances and a right hemiparesis were evident. In addition, the patient was aphasic and dysarthric. Over the next few days she developed

rightsided hemidystonia and hemichorea-hemiballism. Repeat CT scans 3 weeks after the bleeding demonstated an occlusive hydrocephalus and a hypodense lesion of the thalamus, subthalamic region and the posterior internal capsule on the left side (Fig. 4). After ventriculoperitoneal shunting she became more alert. Within 4 weeks the choreatic and ballistic movements became more disabling. Feeding branches to the AVM were embolized in three subsequent sessions. One year postoperatively, the hemidystonia was still present, but the hyperkinetic choreic and ballistic movements had subsided almost completely.

3.4. Structural posthemorrhagic cerebral lesions

3.4.1. Patient 5

This 22-year-old man had intermittent headaches since he was a child. At age 16, he noted double vision and left weakness. Emergency imaging studies showed a rightsided hematoma which was confined to the thalamus and the posterior internal capsule. Three weeks later he noted painful sensations of his left arm and leg. Over the next 4 months a dystonic posture of his left hand became evident. He was admitted for further evaluation and treatment at the age of 22. There was a marked leftsided hemiparesis, leftsided hemihypesthesia, and dystonia of his left hand with a 'bajonet' deformity and crawling of his left fingers. In addition, there was a spontaneous dorsal flexion of his left big toe. Angiography demonstrated an AVM of slightly less than 3 cm in the right thalamus which was supplied by the right posterior cerebral artery. The AVM was approached via a temporal approach. Postoperatively, there was a transient increase of the hemiparesis. The dystonia of the left hand was unchanged. Several months postoperatively the patient collapsed and died. Autopsy was refused.

3.4.2. Patient 6

This 20-year-old man had an unremarkable medical history until the age of 19. During work he had noted paresthesias and progressive weakness of his left arm. Within hours he became comatose. Imaging studies revealed a subarachnoid hemorrhage and a hematoma in the right thalamus, the posterior limb of the internal capsule and the posterior putamen. Angiography showed a thalamic AVM which was fed by thalamoperforating branches from the right posterior communicating artery and drained into the internal cerebral veins. Marked left hemiparesis and hemihypesthesia were evident. During the next few weeks the hemiparesis gradually improved. Parallel to the amelioration of the hemiparesis the patient noted increased muscle tone of his left arm. On admission 6 months after the bleeding, there was a moderate left spastic hemiparesis and a hemihypesthesia. In addition, hemianopsia to the left side was noted. The left arm was held in a dystonic

posture with outward rotation in the shoulder, extension in the elbow, dorsal flexion and abduction of the wrist, and hyperextension of the fingers. The dystonia became worse with various activities. Treatment with a variety of drugs did not result in improvement of the movement disorder. The AVM was resected via a posterior parietal approach. The early postoperative course was unremarkable. Within the next year the hemiparesis improved while the dystonia of the left arm was unchanged. A follow-up angiography confirmed complete removal of the AVM.

4. Discussion

Movement disorders may be secondary to a variety of diseases affecting the brain including intraaxial and extraaxial tumors, perinatal trauma, head injury, neurodegenerative disorders, neuronal storage diseases, ischemic infarctions, hemorrhage, and infectious diseases [4-10]. In patients with dystonia and chorea focal thalamic or basal ganglia lesions or compression of these structures may be identified [7,9–11]. AVMs have been described only exceptionally to cause movement disorders. The frequency of AVM-related movement disorders remains unclear. Remarkably, hyperkinesias or tremors have not been reported as a presenting symptom in large historic and contemporary series focusing on different aspects. Lobato et al. did not describe any movement disorders when he compared the clinical presentation of 133 patients with symptomatic AVMs and 55 patients with cavenomas [12]. Studies that investigated the mass effect of AVMs or the effect on perfusion of adjacent brain tissue were also unrevealing [13,14]. In addition, no descriptions of AVM-related movement disorders were found in multipatient series with thalamic or basal ganglia AVMs [15-17]. On the other hand, it is notable that in a study on 28 patients with symptomatic dystonia four were found to have a cerebral AVM [10].

Movement disorders that have been described earlier to be associated with AVMs include dystonic syndromes such as cervical dystonia, hemidystonia, hemiathetosis, and paroxysmal hemichoreoathetosis, various tremors, and single instances of hemichorea, hemiballism, action myoclonus, and palatal myoclonus (Table 2) [10,18–32]. A total number of 23 cases reported previously were identified. Patients with cavernomas or venous anomalies were not included in this review. It appears that dystonic syndromes are the most common AVM-related movement disorders. In almost all of these patients the thalamus or the basal ganglia, in particular, the caudate and the putamen were involved.

The patients described in the present report illustrate the spectrum of movement disorders that can be secondary to AVMs and emphasize the variety of possible

Table 2

Reported cases of movement disorders secondary to cerebral arteriovenous malformations

| Author | Movement disorder | No. patients |
|--------------------------------|--------------------------------------|--------------|
| Cushing and Bai- ley [19] | Hemichorea | 1 |
| Mackenzie [20] | Hemichorea | 1 |
| Paterson and McKissock [21] | Hemiathetosis | 1 |
| Avenarius et al. [22] | Hemiballism | 1 |
| Shaw et al. [23] | Cervical dystonia | 1 |
| Lobo-Antunes et al. [24] | Cervical dystonia | 2 |
| | Focal dystonia and tremor | 2 |
| Avanzini et al. [25] | Intention/action myoclonus | 1 |
| Podlas et al. [26] | Unilateral tremor | 1 |
| Diamond et al. [27] | Unilateral action tremor | 1 |
| | Hemichoreoathetosis | 1 |
| Marsden et al. [10] | Cervical dystonia | 2 |
| | Hemidystonia | 2 |
| Friedman et al. [28] | Hemidystonia | 2 |
| Vincent [29] | Transient hemichoreo- athetosis | 1 |
| Chalk et al. [30] | Palatal myoclonus | 1 |
| Shintani et al. [31] | Paroxysmal choreoathetosis | 1 |
| Ezura et al. [32] | Hemichorea | 1 |
| Krauss et al. | Action-induced hemidysto- nia | 1 |
| | Hemidystonia/ hemichorea- ballism | 1 |
| | Unilateral cortical tremor | 2 |
| | Focal dystonia (arm/hand) | 2 |

pathomechanisms that may be associated with their development. Basically, four different underlying causes were identified: parenchymal altered cerebral blood flow at the site of the location of the AVM, diaschisis, mass effect and hemorrhagic or ischemic structural lesions. We think that it is important to differentiate between these different possible mechanisms. Unusual movement disorders should encourage a more thorough investigation for the underlying causes. On the other hand, with regard to the delayed onset of movement disorders after structural lesions in the basal ganglia the causal association might be overlooked. Furthermore, the mechanism by which the movement disorder is caused has also prognostic implications.

Local parenchymal altered cerebral blood flow is the most likely cause in the two patients who presented with cortical tremor secondary to cortical/subcortical high-frontal AVMs. There was no indication for functional basal ganglia involvement in these cases. The term 'cortical tremor' has been coined only recently by Ikeda et al. who described high-frequency postural and action tremors in patients with electrophysiologic evidence of cortical myoclonus [33]. Cortical tremor is considered a variant of cortical reflex myoclonus [34]. The occurrence of both focal motor seizures and cortical tremor has been reported to be associated with subcortical infarction [35]. Remarkably, the seizures and the tremor were alleviated after resection of the AVM in patient 3 of this series.

It is thought that the action-induced hemidystonia in patient 1 is related both to mass effect caused by the unruptured AVM and by diaschisis. It has been generally considered that mass effect is evidence for rupture of AVMs. Miyasaki et al., however, have shown that mass effect is not uncommon in AVMs without previous bleeding [13]. Factors that were associated with mass effect were large size of AVMs, marked venous drainage and brain edema around the AVM. All these factors were present in the patient and resulted in compression of the left putamen. Since the hemidystonia was markedly pronounced by prolonged activity of the right arm a dynamic element most likely is also involved. The putamen is supplied by lenticulostriate ateries branching directly from the middle cerebral artery. In this case the AVM was partially supplied by enlarged lenticulostriate arteries. As shown by blood flow studies hypoperfusion was found in the parenchyma adjacent to the AVM. It is conceivable that prolonged activity of the right arm resulted in further decrease of putaminal blood flow triggering thus the appearance of the movement disorder.

Different aspects are relevant in patients 4, 5 and 6 of the series. Patient 4 developed both hemidystonia as well as hemichorea-hemiballism contralateral to the left medial temporooccipital AVM after a delay of few days. Most likely vasospasm resulting in hypodense lesions of the thalamus, subthalamus and internal capsule is responsible for the movement disorders. Ischemic infarction is the most frequent cause for hemichorea-hemiballism [6,36]. Usually vascular hemiballism is self-limiting and improves after days to weeks. The structural posthemorrhagic lesions in the thalamus and the putamen in patients 5 and 6 correspond to well known localizations that may cause focal dystonia.

It appears that the natural history of AVM-related movement disorders is characterized most frequently by persistence of the hyperkinesias in patients with unruptured AVMs. There is only one report on spontaneous improvement of AVM-related cervical dystonia [23] and one report on transient hemichoreoathetosis associated with hyperglycemia [29]. Medical therapy has rarely been reported to be effective. Limited benefit has been achieved with high-dose anticholinergic medication in a patient with hemidystonia [28]. Carbamazepine therapy resulted in amelioration of paroxysmal choreoathetosis in a patient with a parietal

AVM [31]. As seen in patient 3 of the study, however, such movement disorders may disappear after surgical removal of the AVM. Podlas et al. described abolition of appendicular tremor in their patient after embolization of a thalamic AVM [26].

Overall, AVM-related movement disorders appear to be more frequent in patients with large AVMs corresponding to Spetzler–Martin grades IV and V. Surgical removal of such AVMs remains difficult and may require multistage surgery. The optimal treatment of such patients is still under debate.

References

- Wilkins RH. Natural history of intracranial vascular malformations: a review. Neurosurgery 1985;16:421–30.
- [2] Camarata PJ, Heros RC. Arteriovenous malformations of the brain. In: Youmans JR, editor. Neurological Surgery, 4th edition. Philadelphia: Saunders, 1996:1372–404.
- [3] Spetzler RF, Martin NA. A proposed grading system for arteriovenous malformations. J Neurosurg 1986;65:476–83.
- [4] Krauss JK, Nobbe F, Wakhloo AK, Mohadjer M, Vach W, Mundinger F. Movement disorders in astrocytomas of the basal ganglia and the thalamus. J Neurol Neurosurg Psychiatry 1992;55:1162–7.
- [5] Krauss JK, Paduch Th, Mundinger F, Seeger W. Parkinsonism and rest tremor secondary to supratentorial tumours sparing the basal ganglia. Acta Neurochir 1995;133:22–9.
- [6] Dewey RB Jr., Jankovic J. Hemiballism-hemichorea: clinical and pharmacologic findings in 21 patients. Arch Neurol 1989;46:862–7.
- [7] Krauss JK, Borremans JJ, Nobbe F, Mundinger F. Ballism not related to vascular disease: a report of 16 patients and review of the literature. Park Rel Disord 1996;2:35–45.
- [8] Krauss JK, Tränkle R, Kopp KH. Post-traumatic movement disorders in survivors of severe head injury. Neurology 1996;47:1488–92.
- [9] Pettigrew LC, Jankovic J. Hemidystonia: a report of 22 patients and a review of the literature. J Neurol Neurosurg Psychiatry 1985;48:650-7.
- [10] Marsden CD, Obeso JA, Zarranz JJ, Lang AE. The anatomical basis of symptomatic hemidystonia. Brain 1985;108:463–83.
- [11] Krauss JK, Mohadjer M, Braus DF, Wakhloo AK, Nobbe F, Mundinger F. Dystonia following head trauma-a report of nine patients and review of the literature. Mov Disord 1992;7:263–72.
- [12] Lobato RD, Rivas JJ, Gomez PA, Cabrera A, Sarabia R, Lamas E. Comparison of the clinical presentation of symptomatic arteriovenous malformations (angiographically visualized) and occult vascular malformations. Neurosurgery 1992;31:391–7.
- [13] Miyasaka Y, Kurata A, Tanaka R, et al. Mass effect caused by clinically unruptured cerebral arteriovenous malformations. Neurosurgery 1997;41:1060–4.
- [14] Tanaka K, Yonekawa Y, Kaku Y, Kazekawa K. Arteriovenous malformation and diaschisis. Acta Neurochir 1993;120:26–32.
- [15] Lee JP. Surgical treatment of thalamic arteriovenous malformations. Neurosurgery 1993;32:498–504.
- [16] Malik GM, Umansky F, Patel S, Ausman JI. Microsurgical removal of arteriovenous malformations of the basal ganglia. Neurosurgery 1988;23:209–17.
- [17] Sasaki T, Kurita H, Saito I, et al. Arteriovenous malformations in the basal ganglia and thalamus: management and results in 101 cases. J Neurosurg 1998;88:285–92.

- [18] Bruyn GW, Ferrari MD. Chorea and migraine: 'hemicrania choreatica'? Cephalalgia 1984;4:119–24.
- [19] Cushing H, Bailey P. Tumors Arising from the Blood Vessels of the Brain. London: Bailliere, Tindall & Co, 1928.
- [20] Mackenzie I. Clinical presentation of cerebral angioma. Brain 1953;76:184–214.
- [21] Paterson JH, McKissock W. A clinical survey of intracranial angiomas with special reference to their mode of progression and surgical treatment: a report of 110 cases. Brain 1956;79:233–66.
- [22] Avenarius HJ, Gerstenbrand F, Jellinger K. Beitrag zur Pathophysiologie des Ballismus. Spontanremission der Hyperkinese bei einem Fall von arteriovenösem Angiom im Gebiet der A. vertebralis. Acta Neurochir 1964;12:408–24.
- [23] Shaw KM, Hunter KR, Stern GM. Medical treatment of spasmodic torticollis. Lancet 1972;1:1399.
- [24] Lobo-Antunes J, Yahr MD, Hilal SK. Extrapyramidal dysfunction with cerebral arteriovenous malformations. J Neurol Neurosurg Psychiatry 1974;37:259–68.
- [25] Avanzini G, Broggi G, Caraceni T. Intention and action myoclonus from thalamic angioma: report of a case. Eur Neurol 1977;15:194–202.
- [26] Podlas H, Lipschitz R, Allen ML. Selective arterial embolization of the brain in an inoperable arteriovenous malformation in the thalamus: a case report. S Afr Med J 1979;55:1000–2.
- [27] Diamond MS, Huang YP, Yahr MD. Sudden onset of involuntary movement disorders with arteriovenous malformations of the basal ganglia. Mt Sinai J Med 1982;49:438–42.

- [28] Friedman DI, Jankovic J, Rolak LA. Arteriovenous malformation presenting as hemidystonia. Neurology 1986;36:1590–3.
- [29] Vincent FM. Hyperglycemia-induced hemichoreoathetosis: the presenting manifestation of a vascular malformation of the lenticular nucleus. Neurosurgery 1986;18:787–90.
- [30] Chalk JB, Patterson MC, Pender MP. An intracranial arteriovenous malformation and palatal myoclonus related to pseudoxanthoma elasticum. Aust N Z J Med 1989;19:141–3.
- [31] Shintani S, Shiozawa Z, Tsunoda S, Shiigai T. Paroxysmal choreoathetosis precipitated by movement, sound and photic stimulation in a case of arterio-venous malformation in the parietal lobe. Clin Neurol Neurosurg 1991;93:237–9.
- [32] Ezura M, Takahashi A, Yoshimoto T. Successful treatment of an arteriovenous malformation by chemical embolization with estrogen followed by conventional radiotherapy. Neurosurgery 1992;31:1105–7.
- [33] Ikeda A, Kakigi R, Funai N, Neshige R, Kuroda Y, Shibasaki H. Cortical tremor: a variant of cortical reflex myoclonus. Neurology 1990;40:1561–5.
- [34] Toro C, Pascual-Leone A, Deuschl G, Tate E, Pranzatelli MR, Hallett M. Cortical tremor: a common manifestation of cortical myoclonus. Neurology 1993;43:2346–53.
- [35] Schulze-Bonhage A, Ferbert A. Cortical action tremor and focal motor seizures after parietal infarction. Mov Disord 1998;13:356–8.
- [36] Vidakovic A, Dragasevic N, Kostic VS. Hemiballism: report of 25 cases. J Neurol Neurosurg Psychiatry 1994;57:945–9.