# Indications for Surgery and Prognosis in Patients with Cerebral Cavernous Angiomas

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#### Abstract

Seventy-three cerebral cavernous angiomas were removed microsurgically from a series of 71 patients between August, 1983 and December, 1989. This retrospective investigation assessed the current indications for surgery and determined the prognosis for patients with cerebral cavernous angioma. There were 38 males and 33 females with a mean age of 37 years. Analysis included clinical presentation and history, neuroradiological findings, indications for surgery, and postoperative course. After an average follow-up period of 15 months, 35 patients were symptom-free, 16 had improved preoperative complaints, six were unchanged, and eight had deteriorated. Microsurgical extirpation of the malformation is indicated in all symptomatic patients where neuroimaging demonstrates the presence of a readily accessible cerebral cavernoma. Surgery is recommended in cases with deep-seated lesions causing massive hemorrhage, repetitive minor bleeding, or significant long-standing and progressive neurological disabilities. Clinically silent cavernomas located in eloquent regions of the brain contraindicate surgery, but should be closely monitored. Patients presenting with convulsions or neurological deficits caused by easily accessible cavernomas of the hemispheres have the best prognosis and a negligible risk for surgical complications. Those with deep-seated lesions of eloquent regions of the brain that have bled or caused sustained neurological disorders face the highest risk for morbidity owing to the surgical intervention, requiring careful preoperative evaluation.

Key words: cavernoma, cavernous angioma, arteriovenous malformation, surgical indication, prognosis

## Introduction

Cavernous angiomas are a well known clinical and pathological entity.<sup>6,22,27,35</sup> Recently, the incidence of detected congenital vascular hamartomas during life, and the reports on successful surgical treatment of cerebral cavernous angiomas have continuously increased.<sup>1,2,5,7,9,11,14,21,26,29,30,33,34,36,39-42</sup> One of the main reasons is the high degree of sensitivity and specificity of high-field magnetic resonance (MR) imaging<sup>4,17,19,20</sup> which is now the method of choice for preoperative evaluation of these benign and in most cases surgically curable vascular malformations.

The present investigation included 71 patients who underwent surgery during a 6-year period at our institution. The objective of this retrospective study was to investigate: 1) the current appropriate indica-

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tions for surgery in cerebral cavernous angioma; 2) which patients definitely benefit from the surgical removal of a cavernous malformation; 3) which patients harboring a cerebral cavernous angioma must be considered at high risk for postoperative morbidity; 4) if surgery is clinically indicated, the optimal timing for the procedure in patients not presenting with massive hemorrhage. Special emphasis is placed on these questions because neurosurgeons are now dealing more frequently with cavernomas than in the past; and the incidence of hemorrhage and overall morbidity in the natural history of cavernous malformations are not entirely understood.27,38) Only recently have studies provided preliminary information about the risk of hemorrhage in patients with cavernous angioma.4,19,20)

## **Clinical Materials and Methods**

#### I. Patient population

Seventy-one consecutive patients underwent microsurgical removal of symptomatic and histologically confirmed cerebral cavernous angiomas between August, 1983 and December, 1989 at the Department of General Neurosurgery, University of Freiburg. Two individuals harbored two cavernomas each at different locations which were treated surgically at different times. There were 38 males (54%) and 33 females (46%), ranging in age from 5 to 63 years (mean, 37 yrs). Familial occurrence was seen in two patients who were siblings.

#### II. Clinical presentation

Epileptic seizures were the only preoperative complaints in 29 patients. Another 16 combined seizures with various neurological deficits. Among the patients presenting with seizures, 21 suffered from generalized convulsions, seven from each of focal and complex partial seizures, and 10 from combined seizures. The incidence was low (one or up to three episodes of seizures in history) in 17 individuals, moderate (four seizures in history or several per year) in 13, and high (several seizures per month or more) in 15. Twenty-six patients received anticonvulsants, but the incidence of seizure was satisfactorily reduced in only 11 patients. The age, sex, and location, side, or size of the malformation did not significantly influence the incidence or the pattern of epileptic seizures.

Forty-nine patients presented with the clinical picture of a cerebral space-occupying lesion (Fig. 1). Progressive focal neurological deficits were manifest preoperatively in 44 individuals. The neurological disorders included impairment of cranial nerve function in 25 patients, sensory disturbances (chiefly hemihypesthesia and paresthesia) in 19, and motor deficits (predominantly hemiparesis) in 15. Severe headache, disorientation, dysphasia, dysarthria, trigeminal neuralgia, nystagmus, ataxia, and vertigo were additional signs and symptoms frequently encountered in these patients, depending upon the location of the malformation. Two patients with supratentorial cavernomas and three with lesions located in the quadrigeminal plate of the midbrain had symptoms of increased intracranial pressure. In the latter cases, this was due to obstructive hydrocephalus (Fig. 2), each requiring placement of a ventriculoatrial shunt prior to the removal of the vascular malformation. Cavernomas 2 cm in diameter or greater caused neurological deficits significantly more frequently than those less than 2



Fig. 1 Postcontrast CT scans of a 43-year-old male with a left insular cavernoma causing right hemiparesis, and generalized and focal seizures. Preoperative scan shows the hyperdense portion of the lesion extending close to the Sylvian fissure, and the hypodense cystic portion compressing the posterior limb of the internal capsule (*left*). The cavernoma was completely removed through a trans-Sylvian approach, as demonstrated on the postoperative scan (*right*). There were no additional neurological deficits postoperatively, and the seizures were significantly ameliorated.



Fig. 2 Preoperative postcontrast CT scans of a 13year-old boy, showing a hyperdense cavernous angioma of the quadrigeminal plate of the midbrain causing obstructive hydrocephalus. The malformation was completely removed by a supracerebellar infratentorial approach, and the postoperative course was uneventful.

cm in diameter.

Four individuals presented with intracerebral mass hemorrhage from the malformation (Fig. 3). In another 39, less severe bleeding from the cavernoma



Fig. 3 Postcontrast CT scans of a 30-year-old male. Preoperative scans show a ring-shaped lesion within the dorsal pons (*left*). During observation, he developed sudden onset of right hemi-hypesthesia, dysarthria, and abducens nerve paresis due to spontaneous bleeding from this pontine cavernoma (*right*). The lesion was completely extirpated through the fourth ventricle. At 3-year follow-up, he was symptom-free.

was indicated by an ictal episode, confirmed intraoperatively. Approximately half experienced recurrent episodes of symptom exacerbation during the preoperative course, most likely caused by repeated microhemorrhages.

## III. History

The period between first onset of complaints and surgery varied widely, ranging between 2 weeks and 26 years (mean, 49 mos). Seizures were of longstanding duration in most patients (mean, 65 mos). With one exception, patients harboring a pontine cavernoma had the shortest symptom duration (mean, 2.5 mos). Symptoms became manifest at a mean age of 33 years. Forty-four patients suffered a sudden onset of complaints. In the others, the symptoms developed gradually and most commonly progressed.

## IV. Neuroradiological findings

Table 1 lists the various findings from preoperative computed tomographic (CT) scans in each patient. There were seven midline lesions (located in the cerebellar vermis, pons, or quadrigeminal plate of the midbrain), and 34 involving the right and 32 the left hemisphere. Table 2 displays the distribution of malformations according to location. Obviously, 47 lesions (64%) were quite easily accessible, whereas 26 (36%) were located deeply, in functionally important regions of the brain. The greatest CT diameter of the lesions ranged from 0.5 to 4 cm (mean, 1.9 cm).

Preoperative MR imaging in 60 patients was partic-

Table 1	СТ	findings	of	73	cerebral	cavernous
	angi	omas				

CT finding	No. of cases
Hyperdensity	49
Mixed hypo- and hyperdensity	18
Ring enhancement	9
Hypodensity	2
Calcifications within the lesion	29
Perilesional edema	10

Table 2 Location of 73 cavernous angiomas

Location	No. of cases		
Supratentorial			
frontal	21		
temporal	14		
parietal	8		
occipital	3		
insula	6		
basal ganglia	4		
thalamus	2		
Infratentorial			
pons	8		
midbrain	5		
brachium pontis	1		
cerebellum	1		



Fig. 4 T<sub>1</sub>-weighted MR images of a 30-year-old male, demonstrating a cavernous angioma extending from the left thalamus and third ventricle to the left peduncle. The malformation was removed through an interhemispheric transcallosal approach. Postoperatively, he experienced right upper extremity paralysis and recent memory deficits which were markedly improved at 1-year follow-up.



Fig. 5 Preoperative  $T_1$ -weighted MR images of a 50year-old male, demonstrating a hyperintense lesion within the dorsolateral pons. There is no contact between the lesion and the surface of the brainstem. The malformation was totally removed *via* the fourth ventricle. Postoperatively, a transient sensory disturbance occurred in the right leg. At 1-year follow-up, he was symptom-free.

ularly useful in confirming the suspected diagnosis of cavernous angioma and in delineating the relationship of the malformation to the surrounding brain structures (Figs. 4 and 5).

Cerebral angiography was performed in each case. A normal angiogram was found in 50 cases. A subtle but definite vascular stain was evident in only two pontine cavernomas. Filling of prominent draining veins adjacent to the cavernoma was seen in four frontal, and one each of temporal, occipital, insular,

Table 3	Surgical approaches selected for removal of
	the 73 cavernous angiomas

Approach	Craniotomy	Location of cavernoma	No. of cases
Trans-sulcal	convexity	frontal, parietal, temporal, or occipital convexity	21
Transcortical (superficial)	convexity	frontal, parietal, or temporal convexity	16
Trans-Sylvian	pterional*	insula; basal ganglia	10
Via floor of fourth ventricle	suboccipital (median)	dorsal pons	7
<i>Via</i> interhemi- spheric fissure	(parasagittal)	frontal and occipital midline cortex	6
Via cerebello- pontine angle	suboccipital (lateral)	lateral pons	1
Supracerebellar, infratentorial	suboccipital (paramedian)	midbrain; brachium pontis	5
Subtemporal	temporal (basal)	temporal lobe; peduncle	4
Interhemispheric transcallosal (via third ventricle)	frontal parasagittal	thalamus	2
Direct cerebellar	suboccipital (median)	cerebellum	1

\*In one case extended towards the parieto-occipital region.

and pontine malformations, suggesting a venous angioma.

#### V. Indications for surgery

The indications for surgical intervention in this series included treatment of convulsions, improve-

ment of focal neurological disorders, correction of cerebrospinal fluid pathway obstruction, evacuation of hematoma, and prevention of further bleeding. The latter was a prophylactic surgical indication. Eliminating the risk of a potentially life-threatening massive bleeding was appropriate mainly in patients with repetitive hemorrhages or with lesions of eloquent areas of the brain, where even minor bleeding had already caused substantial morbidity.

Superficially located malformations were reached by exposing the convexity of the brain. More deeply placed lesions were generally approached by dissecting the cerebral sulci or fissures, and removed from the closest point to the surface of the brain or ventricles.<sup>1,25)</sup> The surgical approaches used in this series are presented in Table 3.

#### Results

#### I. Early postoperative course

Table 4 summarizes the overall surgical outcome by discharge (10-14 days after surgery in 61 cases). Of the patients not presenting with neurological disorders, only one experienced transient deficits following the procedure which had resolved by followup examination. The surgical complications are listed in Table 5. A detailed analysis of the complications encountered in patients with deep-seated malformations has been presented elsewhere.<sup>1)</sup>

#### II. Follow-up

Follow-up data were available from 65 individuals (92%), obtained from questionnaires, telephone contact, or re-examination. The mean follow-up period was 15 months, ranging from 8 months to 5 years.

The evolution of the seizure disorder by follow-up examination is presented in Table 6. Excluding the first 5 postoperative days, 27 patients presenting with epileptic seizures remained seizure-free, either with or without antiepileptic medication. The prevalence of seizures clearly decreased from 60% preoperative-

Table 6	Evolution	of	seizures	bv	foll	low-un	in	45	natients
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Seizure type	No. of patients*	Free without anticonvulsants	Free with anticonvulsants	Frequency reduced	Frequency unchanged	Unknown
Generalized	21	5	10	4	0	2
Focal	7	2	2	1	0	2
Complex partial	7	3	3	1	0	0
Combined	10	0	2	3	3	2
Total	45	10	17	9	3	6

\*Preoperatively.

Table 4	Early postoperative	outcome	in	73	caver-
	nous angiomas				

Outcome	No. of cases	%
Neurologically intact pre- and postoperatively	37	50.7
Neurologically intact preoperatively, postoperative deficits	1	1.3
Preoperative neurological deficits improved at discharge	14	19.2
Preoperative deficits unchanged at discharge	7	9.6
Preoperative deficits worsened or new deficits	14	19.2

Table 5 Surgical complications

Complication	No. of patients
Intraoperative paradoxical air embolism	1
Ischemic infarction within internal capsule	1
Hemorrhagic infarction within thalamo- striate region	2
Mechanical damage to internal capsule	2
Wound infection*	3
Recurrent hemorrhage from residual cavernoma*	2
Subdural hygroma*	1

\*Surgical treatment.

ly to 18% postoperatively. Additionally, the incidence was markedly reduced in 75% of the 12 patients still suffering from convulsions. These 12 individuals had a significantly longer duration of symptoms (mean, 124 mos) compared with the seizure-free patients (mean, 34 mos). The size of the lesion did not influence the outcome.

Table 7 summarizes the evolution of the main neurological disorders. In 15 of the 22 patients with

Neurological deficits No	o. of patients*	Cured	Improved	Unchanged	Worsened	Unknown
Motor	15	5	3	3	3	1
Sensory	19	7	5	3	2	2
Cranial nerves	25	10	5	6	3	1

 Table 7 Evolution of main neurological deficits by follow-up

\*Preoperatively.

unsatisfactory neurological condition in the early postoperative stage, deficits had resolved or markedly improved by follow-up examination.

Overall, by follow-up examination, 35 individuals (54%) were symptom-free, 16 (25%) had improved preoperative complaints, six (9%) remained unchanged, and eight (12%) had deteriorated.

## Discussion

Age and sex distribution, history, and clinical and neuroradiological findings in this series compare favorably with details given in other reports.<sup>24,27,35,39</sup> The MR imaging features of cavernomas may not be pathognomonic,<sup>16,17</sup> but a correct diagnosis was possible in virtually all our patients for whom preoperative MR images were obtained. As in other reports,<sup>4,5,7,11,16,17,19,23,36</sup> MR imaging offered several advantages: a high degree of specificity; detection of even very small lesions, particularly within the posterior fossa; and sufficient accuracy of triplanar sections to determine the exact location of the malformation and the relationship with the surrounding cerebral structures.

Cerebral angiography is not primarily a diagnostic tool, but can eliminate coincidental vascular malformations, such as cavernous angioma associated with venous angioma or capillary teleangiectasia, both rare but possible occurrences.<sup>1,18,39,42)</sup> Cavernoma was associated with a venous angioma in at least five cases in our series. Angiography was also important in localizing and delineating the required craniotomy in superficial lesions of the convexity, or in those approached through the interhemispheric fissure. In such cases, precise knowledge of the superficial vascular pattern was indispensable for adequate planning of the surgical approach. Since angiography complements CT and MR imaging, we believe that this study is not obsolete for preoperative evaluation of cerebral cavernous angiomas.

Authors have been unanimous for more than a decade in considering surgery the therapy of choice in readily accessible cavernous angiomas causing clinical symptoms and/or recurrent hemor-

rhages.<sup>2,6,10,13,15,27,35,39,40)</sup> Although the follow-up period is not long enough to allow definitive inferences, the vast majority of our patients presenting with convulsions experienced a significant amelioration of their seizure disorder postoperatively. Similarly, those presenting with neurological disabilities caused by cavernous angiomas located in noneloquent regions of the brain unequivocally benefited from surgery. Surgical therapy was less effective, however, in patients suffering from uncontrollable long-standing seizures.

Assessment of appropriate indications for surgery upon cavernous angiomas located within functionally important regions of the brain, however, remains a constant challenge. Such lesions may remain clinically silent, and are now usually detected by chance on MR images.<sup>4,19</sup> They may cause various symptoms due to repeated microhemorrhage or to gradual enlargement,<sup>1,13,14,27</sup>) or they may suddenly cause the patient's condition to become critical and life-threatening when massive hemorrhage occurs.<sup>6,22,27)</sup> Controversy continues over whether surgical or conservative treatment is preferable in such cases.<sup>3,4,9,19,21,28,32,37,42)</sup> Previously, surgical removal of deep-seated lesions, particularly of those located within the brainstem, has been carried out mainly in patients with local massive bleeding<sup>5,8,11,13,21,36,41</sup> or with recurrent subarachnoid hemorrhage.<sup>31)</sup> We also consider that extirpation of the malformation is the therapy of choice in such cases, in order to avoid a second, potentially fatal bleeding.

Apart from cases with major bleeding, we operated on patients with cavernomas in sensitive regions of the brain presenting with various symptoms other than massive hemorrhage from the malformation. Only a few similar cases have been published.<sup>26,40,42)</sup> In the initial period, our results were sometimes disappointing. Later, our surgical success rate has clearly increased, and serious complications have become a rarity since 1988.<sup>1)</sup> Encouraged by these results, and like several authors,<sup>5,10,26,42)</sup> we now prefer a rather aggressive surgical policy in patients presenting with either repetitive minor bleeding (and correlating symptoms) or with sustained

neurological deficits, provided that the malformation extends close to or reaches the surface of the brainstem or ventricles, including the surface of the third ventricle. We must be aware, however, that these individuals face the highest risk for transient and even permanent morbidity attributable to the surgical intervention. A single episode of minor bleeding, or less severe symptoms which rapidly resolved, is not necessarily a surgical indication upon deep-seated lesions.

In contrast to arteriovenous malformations whose natural history is now well defined, 12,38) the yearly risk for massive hemorrhage from cavernoma is difficult or impossible to precisely predict. Del Curling et al.4) recently estimated the annualized bleeding rate at 0.25%/person-year of exposure, and Robinson et al.<sup>19)</sup> 0.7%. The risk of rebleeding after an initial hemorrhage, however, remains unknown.<sup>4)</sup> Nevertheless, there is no doubt that these lesions can be the source of major hemorrhage, and the consequence of such an episode in a patient harboring a deep-seated cavernoma may be devastating.<sup>6,22,27,42)</sup> A careful evaluation of the optimal therapy is therefore mandatory in patients with a symptomatic deep-seated cavernoma not presenting with massive bleeding. There is no indication for surgery in patients with clinically silent cavernous angiomas located in critical regions of the brain, particularly when approaching these lesions would require traversing healthy and functionally important cerebral structures. Certainly, such patients need to be carefully followed clinically and neuroradiologically at least once per year.

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### References

- Bertalanffy H, Gilsbach JM, Eggert H-R, Seeger W: Microsurgery of deep-seated cavernous angiomas: Report of 26 cases. *Acta Neurochir (Wien)* 108: 91– 99, 1991
- Buckingham MJ, Crone KR, Ball WS, Berger TS: Management of cerebral cavernous angiomas in children presenting with seizures. *Childs Nerv Syst* 5: 347-349, 1985
- Cappabianca P, Spaziante R, De Divitiis E, Villanacci R: Thalamic cavernous malformations. J Neurosurg 75: 169, 1991 (letter)

- Del Curling O Jr, Kelly DL, Elster AD, Craven TE: An analysis of the natural history of cavernous angiomas. J Neurosurg 75: 702-708, 1991
- Fahlbusch R, Strauss C, Huk W, Röckelein G, Kömpf D, Ruprecht KW: Surgical removal of pontomesencephalic cavernous hemangiomas. *Neuro*surgery 26: 449-457, 1990
- Giombini S, Morello G: Cavernous angiomas of the brain. Account of fourteen personal cases and review of the literature. *Acta Neurochir* (*Wien*) 40: 61-82, 1978
- Hassler W, Zentner J, Petersen D: Cavernous angioma of the optic nerve. Case report. Surg Neurol 31: 444-447, 1989
- Kashiwagi S, van Loveren HR, Tew JM Jr, Wiot JG, Weil SM, Lukin RA: Diagnosis and treatment of vascular brain-stem malformations. J Neurosurg 72: 27-34, 1990
- 9) Lobato RD, Perez C, Rivas JJ, Cordobes F: Clinical, radiological, and pathological spectrum of angiographically occult intracranial vascular malformations. Analysis of 21 cases and review of the literature. J Neurosurg 68: 518–531, 1988
- Martin NA, Wilson CB, Stein BM: Venous and cavernous malformations, in Wilson CB, Stein BM (eds): Intracranial Arteriovenous Malformations. Baltimore, Williams & Wilkins, 1984, pp 241-245
- Ondra SL, Doty JR, Mahla ME, George ED: Surgical excision of a cavernous hemangioma of the rostral brain stem: Case report. *Neurosurgery* 23: 490-493, 1988
- Ondra SL, Troupp H, George ED, Schwab K: The natural history of symptomatic arteriovenous malformations of the brain: A 24-year follow-up assessment. J Neurosurg 73: 387-391, 1990
- Pozzati E, Gaist G, Poppi M, Morrone B, Padovani R: Microsurgical removal of paraventricular cavernous angiomas. J Neurosurg 55: 308-311, 1981
- Pozzati E, Giuliani G, Nuzzo G, Poppi M: The growth of cerebral cavernous angiomas. *Neurosur*gery 25: 92-97, 1989
- Pozzati E, Padovani R, Morrone B, Finizio F, Gaist
   G: Cerebral cavernous angiomas in children. J Neurosurg 53: 826-832, 1980
- 16) Rapacki TFX, Brantley MJ, Furlow TW Jr, Geyer CA, Toro VE, George ED: Heterogeneity of cerebral cavernous hemangiomas diagnosed by MR imaging. J Comput Assist Tomogr 14: 18-25, 1990
- Rigamonti D, Drayer BP, Johnson PC, Hadley MN, Zabramski J, Spetzler RF: The MRI appearance of cavernous malformations (angiomas). J Neurosurg 67: 518-524, 1987
- Rigamonti D, Spetzler RF: The association of venous and cavernous malformations. Report of four cases and discussion of the pathophysiological, diagnostic and therapeutic implications. Acta Neurochir (Wien) 92: 100-105, 1988
- 19) Robinson JR, Awad IA, Little JR: Natural history of the cavernous angioma. J Neurosurg 75: 709-714,

Neurol Med Chir (Tokyo) 32, August, 1992

1991

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- 20) Robinson JR Jr, Little JR, Awad IA: The natural history of cavernous angiomas. J Neurosurg 72: 333A, 1990 (abstract)
- 21) Roda JM, Alvarez F, Isla A, Blázquez G: Thalamic cavernous malformation. Case report. J Neurosurg 72: 647-649, 1990
- 22) Russell DS, Rubinstein LJ: Pathology of Tumours of the Nervous System. London, Edward Arnold, 1989, pp 730-736
- 23) Rutka JT, Brant-Zawadzki M, Wilson CB, Rosenblum ML: Familial cavernous malformations. Diagnostic potential of magnetic resonance imaging. Surg Neurol 29: 467-474, 1988
- 24) Savoiardo M, Strada L, Passerini A: Intracranial cavernous hemangiomas: Neuroradiologic review of 36 operated cases. AJNR 4: 945-950, 1983
- 25) Seeger W: Strategies of Microsurgery in Problematic Brain Areas. Wien, Springer-Verlag, 1990, pp 137-214
- 26) Seifert V, Gaab MR: Laser-assisted microsurgical extirpation of a brain stem cavernoma: Case report. Neurosurgery 25: 986-990, 1989
- 27) Simard JM, Garcia-Bengochea F, Ballinger WE Jr, Mickle JP, Quisling RG: Cavernous angioma: A review of 126 collected and 12 new clinical cases. Neurosurgery 18: 162-172, 1986
- 28) Solomon RA: Comment on Ondra SL, Doty JR, Mahla ME, George ED: Surgical excision of a cavernous hemangioma of the rostral brain stem: Case report. Neurosurgery 23: 493, 1988
- 29) Tagle P, Huete I, Méndez J, del Villar S: Intracranial cavernous angioma: Presentation and management. J Neurosurg 64: 720-723, 1986
- 30) Tatagiba M, Schönmayr R, Samii M: Intraventricular cavernous angioma. A survey. Acta Neurochir (Wien) 110: 140-145, 1991
- 31) Terao H, Hori T, Matsutani M, Okeda R: Detection of cryptic vascular malformations by computerized tomography. Report of two cases. J Neurosurg 51: 546-551, 1979
- Tung H, Giannotta SL, Chandrasoma PT, Zee C-S: 32)

Recurrent intraparenchymal hemorrhages from angiographically occult vascular malformations. J Neurosurg 73: 174-180, 1990

- 33) Vaquero J, Leunda G, Martinez R, Bravo G: Cavernomas of the brain. Neurosurgery 12: 208-210, 1983
- 34) Vaquero J, Salazar J, Martinez R, Martinez P, Bravo G: Cavernomas of the central nervous system: Clinical syndromes, CT scan diagnosis, and prognosis after surgical treatment in 25 cases. Acta Neurochir (Wien) 85: 29-33, 1987
- 35) Voigt K, Yasargil MG: Cerebral cavernous haemangiomas or cavernomas. Neurochirurgia (Stuttg) 19: 59-68, 1976
- 36) Weil SM, Tew JM Jr: Surgical management of brain stem vascular malformations. Acta Neurochir (Wien) 105: 14-23, 1990
- 37) Weil SM, Tew JM Jr, Steiner L: Comparison of radiosurgery and microsurgery for treatment of cavernous malformations of the brain stem. J Neurosurg 72: 336A, 1990 (abstract)
- 38) Wilkins RH: Natural history of intracranial vascular malformations: A review. Neurosurgery 16: 421-430, 1985
- 39) Yamasaki T, Handa H, Yamashita J, Moritake K, Nagasawa S: Intracranial cavernous angioma angiographically mimicking venous angioma in an infant. Surg Neurol 22: 461-466, 1984
- 40) Yasargil MG: Microneurosurgery, vol IIIB. Stuttgart, Thieme, 1988, pp 419-434
- 41) Yoshimoto T, Suzuki J: Radical surgery on cavernous angioma of the brainstem. Surg Neurol 26: 72-78, 1986
- 42) Zimmerman RS, Spetzler RF, Lee KS, Zabramski JM, Hargraves RW: Cavernous malformations of the brain stem. J Neurosurg 75: 32-39, 1991
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