Microneurosurgery

in 4 Volumes

M. G. Yaşargil

III B

AVM of the Brain, Clinical Considerations, General and Special Operative Techniques, Surgical Results, Nonoperated Cases, Cavernous and Venous Angiomas, Neuroanesthesia

IIIB

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M. G. Yaşargil

Contents

Introduction

Between January 1967 and end of April 1986, 414 patients with AVM underwent operation in Zurich with the help of microtechniques. With the addition of a further 86 patients who were reviewed but not operated upon, the total study group consisted of 500 AVM patients, thereby providing for simplified statistical analyses. These 500 AVM patients do not represent a particularly special or highly selected group of those harboring AVMs. The capacity of the department in Zurich has been such that some patients from abroad offered surgery with surgically favorable lesions died while awaiting admission; still others opted for operation elsewhere or underwent radiation treatment at various centers.

Patients both with and without neurological deficits were accepted. Some of these with overwhelming indications for surgery (such as massive hematoma) came to operation despite desperate looking pathology; others were operated upon at the absolute insistence of themselves and/or their relatives when doubts existed in my own mind as to their operability, particularly in those patients who were otherwise well. Included in the latter group are 3 medical doctors who had themselves scanned to evaluate rather vague symptoms. Each was totally unable to accept the non-operative risks of subsequent hemorrhage and disability, and each was found to have a resectable lesion.

Twenty eight patients had previously been treated at other hospitals by extracranial carotid ligation, intracranial partial or subtotal removal, and/or irradiation (conventional and proton beam).

The series includes many patients whose AVMs had bled previously and who were symptomatic from the associated hematomas. Some of these patients were initially comatose or had dense neurological deficits. A few required urgent operation but the remainder recovered without surgery either completely or at least sufficiently well to consider definitive surgery. Altogether in this series there were more patients operated upon with some degree of neurological impairment than those with no symptoms other than headache or epilepsy.

A meticulous full microtechnique was employed in each case starting and ending with the opening and closing of the dura.

Between 1967 and 1969, habit prompted the use of the prone position in 10 cases with infratentorial AVMs. From 1969 until the present, this uncomfortable state has been abandoned and all infratentorial, posterior interhemispheric and occipital lesions have been approached with the patient in the sitting position. There has not been a single instance of significant air embolism.

This surgically treated group of AVMs has not included any cases of pre- or perioperative embolization (with exception of seven cases in which only the dural-pial connections were preoperatively embolized). No stereotactic techniques have been used, and in no case has cardiac standstill with deep hypothermia been employed. Temporary hypotension has been used in some instances, although recently this technique has been employed less frequently. Excluding 2 cases of giant callosal AVM (in which 2 positions on the table were necessary) and 12 cases in which fragments of the AVM were missed at the initial operation, all procedures were carried out in one stage. Large volume and high flow AVMs have been treated in the same way, as the concept of normal perfusion pressure breakthrough has not been considered particularly relevant.

Many of the patients in this series underwent extensive pre- and postoperative electroencephalographic and neuropsychological evaluation. The detailed findings of these studies have been computerized and await final analysis by our neuropsychiatric colleagues. Unfortunately these results are unavailable at the present time and must await future separate publications.

The international cumulative experience with AVMs including all forms of treatment as reported in the world literature has been extensively and carefully studied, and it has been decided not to include in this text exhaustive analyses of these treatments or comparisons of the efficacy of these treatments with the results

achieved in Zurich. It seemed better merely to let the results stand on their own, simply as a representation of what can be achieved by careful anatomical study and the strict application of microtechnique in each case.

Artistic representations have been found to be more dynamic and informative than pictorial reproductions; for example the information from several photographs can be combined in a single drawing. This format has been adopted as the principle means of visually communicating operative techniques and findings in the current volumes.

At the same time, it was felt necessary to include as many pre- and postoperative angiographic studies as possible to document the findings and results and to show interesting hemodynamic problems, especially of the venous system. CT and MRI scans have been included, when available, as witnesses of the microsurgical findings and operative achievements. Altogether the pictures of 250 operated and 40 nonoperated patients are presented in Vol. IIIA and IIIB.

The analysis of results in this study involves the separation of patients into 2 groups: A) those patients with no preoperative neurological deficit, and B) those with some recognizable neurological deficit prior to operation. As will be evident later in this discussion, this separation permits a clearer definition of operative results and illustrates that deficits present before surgery were related to the hematoma and were reversible following operative intervention.

A final section on cavernous, venous, and occult malformations summarizes the findings in an additional 28 patients. As will be pointed out, our knowledge of these previously neglected and seldom recognized lesions is just beginning to develop with the advent of CT and MRI scanning.

1 Anatomical Location of AVMs from the Surgical Viewpoint

Localization in Planning Operative Approach

Before operating upon an AVM the surgeon needs to know:

1. The type of AVM; is it a fistulous, plexiform or mixed lesion?
2. The size and shape of the lesion.
3. The flow characteristics – high or low flow.
4. The localization of the AVM within the brain and its topographic relations.
5. The arterial territory in which it lies or whether it lies on the border between one or more arterial territories.
6. The arrangement and condition of the venous drainage system: – single vein, varicosities, stenosis or occlusion of the major sinuses, bizarre drainage patterns.
7. The position of the deep vessels in relation to the AVM as shown on angiography and of the ventricles as shown on CT scan, MRI and angiography.
8. Is there one nidus or more?
9. Are there normal areas of brain between the various parts of the lesion – particularly in cases of multiple AVM?

Bearing in mind these preoperative requirements for a well planned surgical approach, the pathological, radiological and operative (microsurgical) findings in 414 cases of intracranial AVM operated in Zurich have been studied. (Dural AVMs are not included in this series.)

There were 346 supratentorial and 68 infratentorial AVMs. 173 of these were right sided, 209 left sided and 32 midline. Of the supratentorial lesions 41.9% were on the right, 52% on the left and 6.1% midline. Cerebellar lesions comprised 41.2% right, 42.6% left and 16.2% midline.

Such data are of some general interest but are not of particular relevance to the special surgical considerations discussed later. Relating these findings to what is felt to be the most important aspects of intracranial surgical anatomy, it has been concluded that it is no longer appropriate to classify all cases of AVM simply as supra- or infratentorial, superficial or deep. On the other hand, to present every single AVM as a unique case would produce a hopelessly complicated surgically orientated classification.

Although there now exist many accounts of the anatomical relationships of and surgical approaches to AVMs at individual sites, there is as yet no accurate and comprehensive general guide to AVM surgical anatomy.

This causes great difficulty when trying to analyze data and understand operative techniques described in the published literature on AVM surgery. Commonly used terminologies and classifications of AVM location such as cortical, subcortical, white substance, grey matter, superficial, deep, para- and intraventricular, brainstem, basal ganglia, lenticuloinsular, striocapsular, capsulothalamic, incisural, ventriculocisternal, subtrigonal, juxtapeduncular, juxtathalamic and juxtatentorial mean little in isolation, do not relate readily to the 9 factors listed above, and are of little value in presenting results clearly and in describing technical problems encountered in AVM surgery in special areas of the brain.

For instance, AVMs of the hemispheric convexities may be simply cortical or subcortical and may or may not exhibit para- and intraventricular extensions. Some hemispheric AVMs cannot be described as superficial as they, in fact, lie deep – sometimes very deep – within the Sylvian fissure (insular AVM), interhemispheric fissure (median frontal, parietal and occipital AVM), transverse fissure (hippocampal AVM) or the calcarine fissure.

Almost 50 years ago Ask-Upmark (1938) attempted to localize gliomas and cerebral angiomas on the basis of evolution of the brain and its vascular system. Although he advanced only a short way along this path his basic concepts were

perhaps, more in keeping with those described here than much of what has been written since.

A tentative classification of AVMs is proposed for consideration. Although artificial from the purely anatomical standpoint in terms of functional anatomy and neuroradiological and surgical reasoning, it seems most appropriate to divide AVMs into two main groups.

1) Convexity (Pallial) AVMs
   a) Supratentorial
   b) Infratentorial

These may be cortical (surface or deep within sulci or fissures), subcortical or combined.

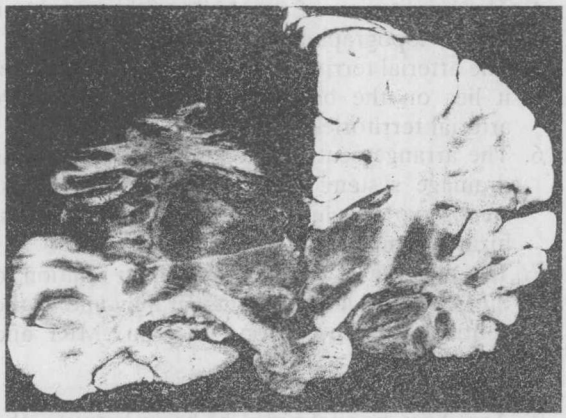
2) Deep Central AVMs
   a) Supratentorial
   b) Infratentorial

These may then be divided into various subgroups as described below.

The *convexial (pallial) system* covers the cortical areas of frontal, temporal, insular, parietal, occipital and cerebellar regions. The *central system* represents the grey matter (nuclei) of the diencephalon, mesencephalon, metencephalon and the connecting fibers, the limbic system (amygdalohippocampus, cingulate gyrus, corpus callosum and fornix) and the choroid plexus of the lateral, third and fourth ventricles (Figs 1.**1A–E** and 1.**2A–B**).

The concept of classifying AVMs in such a manner is based not only upon consideration of the arterial supply but also upon the pattern of the often neglected venous drainage (Tables 1.**1** and 1.**2**):

1. Convexity AVMs are supplied mainly by long circumferential convexity arteries such as ACA, MCA, PCA, PICA, AICA and SCA, whereas the supply from perforating vessels is secondary.
2. The venous drainage of convexity AVMs is mainly to the superficial cortical (ascending or descending) venous system – superior and inferior sagittal sinus, sphenoparietal sinus, transverse, petrosal and sigmoid sinuses.
3. The central supratentorial AVMs are supplied only by perforating branches of ACA, MCA, PCA, anterior and posterior choroidal arteries; the infratentorial, by perforating vessels from PICA, AICA, SCA and the vertebrobasilar arteries.
4. The venous drainage in central AVMs is in almost every case (a few rare exceptions have been observed), through subependymal vessels such as the septal, internal cerebral, basilar, inferotemporal, diverse atrial and internal occipital veins in the supratentorial compartment and through lateral recess veins, lateral and dorsal mesencephalic and precentral cerebellar veins in the infratentorial region. All these veins drain via the vein of Galen to the straight sinus.
5. In cases with convexity AVM (especially occipital and cerebellar) some stenosis or occlusion of the major venous sinuses has been observed and there is also a bizarre pattern of venous drainage. With central AVMs, however, stenosis and occlusion of venous sinuses with development of large collateral channels and bizarre (sometimes embryological) drainage patterns and various sizes of varices is the rule rather than the exception. Obstruction of the straight, transverse and sigmoid sinuses has been observed in about 60–80% of this series (see also Table 9.**2**).



A

Fig 1.**1A** Frontal and horizontal section, showing three-dimensionally the convexial (pallial) and central areas and also showing that such a separation is artificial but useful as a working hypothesis.

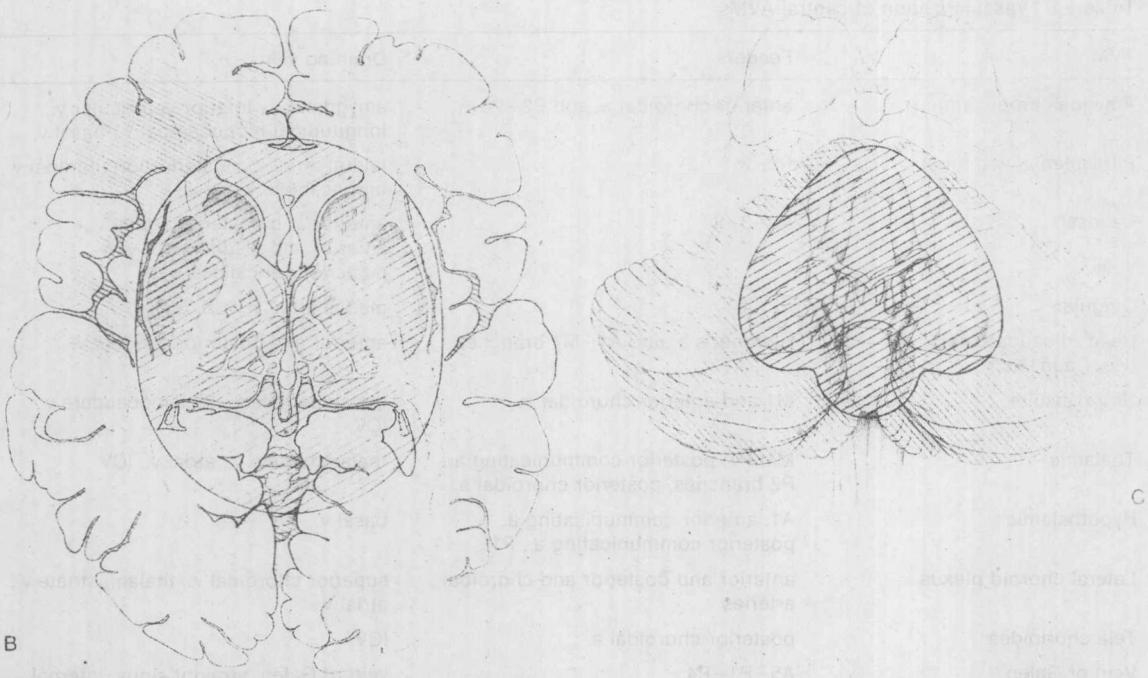
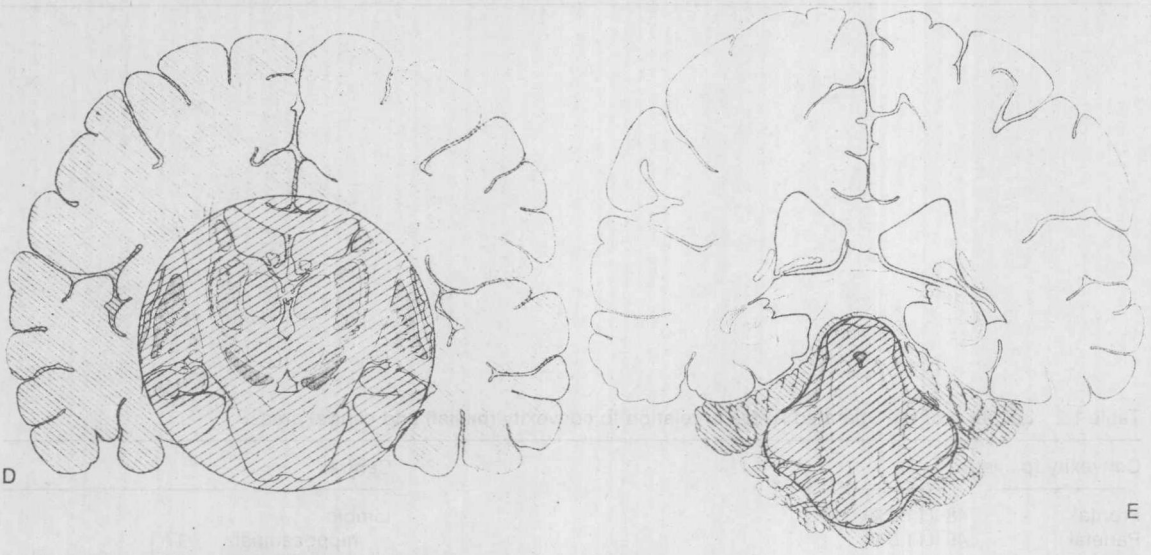


Fig 1.1B–C Diagrammatic representation in horizontal section of 2 groups of supra- and infratentorial areas, in which AVMs occur. Yellow: convexial (pallial), red: central AVMs.



D–E Frontal section. Yellow: convexial (pallial), red: central AVMs.

Table 1.1 Vascularization of central AVMs

AVM	Feeders	Draining vein
Amygdalohippocampal	anterior choroidal a. and P2–P3	amygdalar v., inferior ventricular v., longitudinal hippocampal v., basal v.
Parasplenial (isthmus)	P2–3	lateral atrial v., posterior longitudinal v., hippocampal v., basal v.
Callosal	A2–3–4	anterior and posterior septal v., anterior and medial atrial v., posterior pericallosal v.
Cingular	A2–4	medial atrial v., ISS, SSS
Head of caudate nucleus (Hd. Caud. Nc.)	Heubner's a. and A1–M1 branches	anterior and posterior caudate v.
Striocapsular	M1 and anterior choroidal a.	thalamostriate v., thalamocaudate v., ICV
Thalamic	M1, P1, posterior communicating a. P2 branches, posterior choroidal a.	thalamostriate v., atrial v., ICV
Hypothalamic	A1, anterior communicating a., posterior communicating a., P1	basal v.
Lateral choroid plexus	anterior and posterior and choroidal arteries	superior choroidal v., thalamostriate v., atrial v.
Tela chorioidea	posterior choroidal a.	ICV
Vein of Galen	A5–P1–P4	vein of Galen, straight sinus, internal occipital v., collaterals
Mesencephalic	collicular a., SCA	dorsal mesencephalic v.
Pontine	paramedian branches of basilar a.	lateral mesencephalic v.
Cerebellar central	branches of SCA, PICA, AICA	lateral rec. v., precentral cerebellar v.

Table 1.2 Location of the operated AVMs in relation to convexity (pallial) and central areas

Convexity (pallial)			Central			
Frontal	48	(11.6%)	Limbic			
Parietal	49	(11.8%)	hippocampal	17		
Insular	23	(5.6%)	parasplenial	40	98	(23.7%)
Temporal	53	(12.8%)	cingular	7		
Occipital	30	(7.2%)	callosal	34		
Cerebellar	58	(14.0%)	Hd. Caud. Nc.	11		(2.7%)
			Strio-cap-thal.	15		(3.6%)
	261	(63.0%)	Vein of Galen	16		(3.9%)
			Mesencephalic	6		(1.5%)
			Pontine	4		(1.0%)
			Choroid plexus	3		(0.7%)
				153		(37.1%)

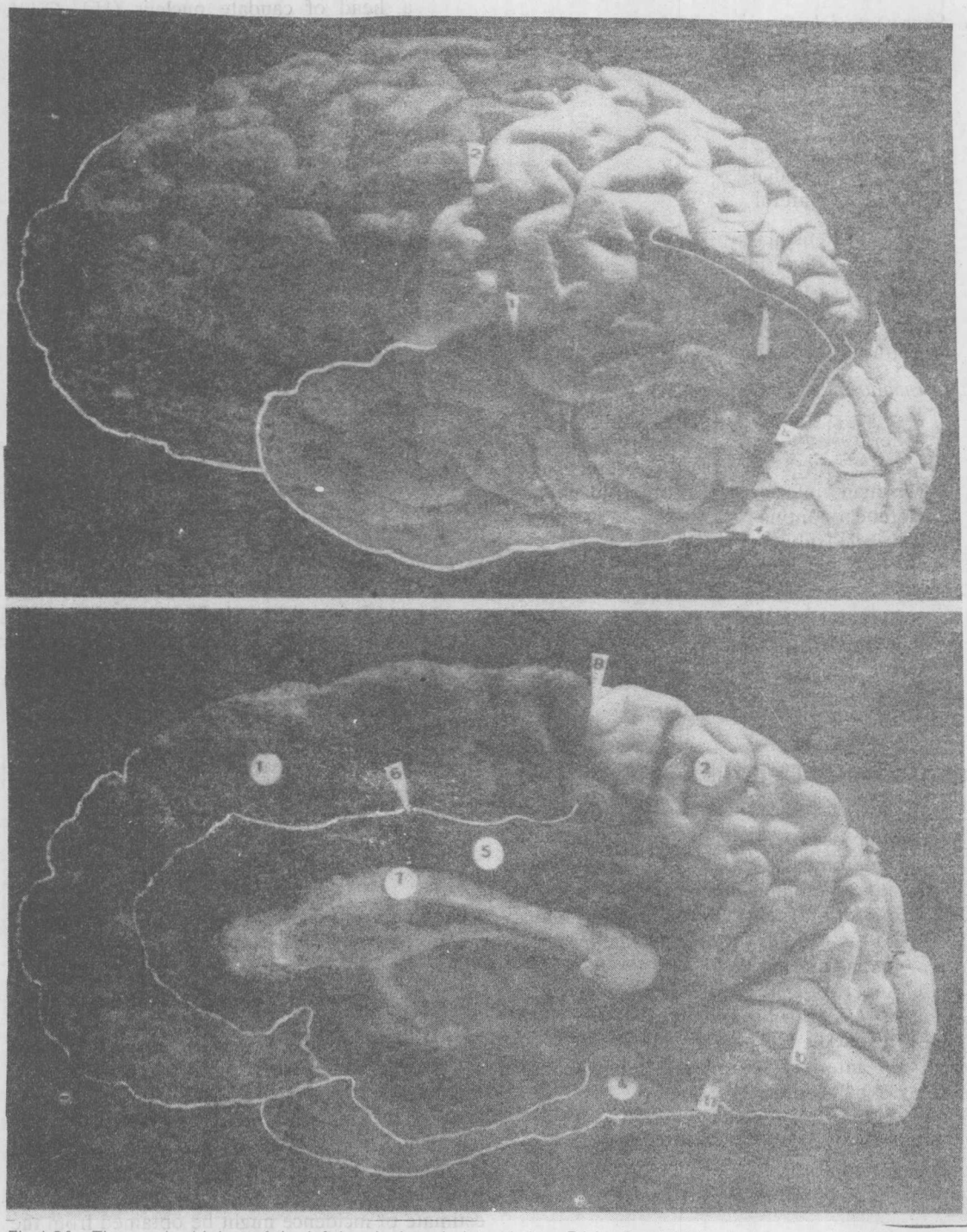


Fig 1.**2A** The cerebral lobes of the left hemisphere in lateral view. Red = frontal lobe, beige = parietal lobe, yellow = temporal lobe, white = occipital lobe.

1 Lateral sulcus
2 Central sulcus
3 Parietooccipital sulcus
4 Preoccipital incision
5 Parietooccipital line
(6) Parietotemporal line

B Medial aspect of right hemisphere.
1 Frontal lobe (red)
2 Parietal lobe (beige)
(3) Occipital lobe (white)
4 Temporal lobe (yellow)
5 Limbic lobe (olive green)
6 Cingulate sulcus
7 Corpus callosum
8 Central sulcus
9 Parietooccipital sulcus
10 Calcarine sulcus
11 Collateral sulcus

Subgroups of Convexity AVMs

A. *Supratentorial*

1. Frontal
2. Parietal
3. Temporal
4. Occipital

- fistula
- giant
- dorsal
- basal
- polar
- medial
- lateral

5. Insular
   - fistula
   - anterior
   - middle
   - posterior

6. Diffuse, whole hemisphere

B. *Infratentorial*

1. Hemispheric — superior
2. Vermian — inferior

(superior and inferior groups are related to the great cerebellar horizontal sulcus)

3. Cerebellopontine
   Extrinsic (subarachnoidal-epipial)
   Intrinsic (intraparenchymatous around the foramen of Luschka)
   a. superior
   b. inferior

(related to sulcus horizontalis)

4. Giant
5. Fistula

Subgroups of Central AVMs

A. *Supratentorial*

1. *Limbic system*
   a. anteromedial (amygdalohippocampal)
   b. posterior (connection of hippocampus to the isthmus of the cingulate gyrus) = parasplenial
   c. superior: callosocingular area
      α posterior (splenium)
      β middle (corpus callosum)
      γ anterior (subcallosal, genu, septum pellucidum)
      δ superior (cingulate gyrus)
2. *Strio-capsulo-thalamic* (Strio-cap-thal.)
   a. head of caudate nucleus (Hd. Caud. Nc.)
   b. claustrum + putamen + pallidum + internal capsule
   c. thalamic
      superior (palliothalamic)
      anterior
      posterior (pulvinar)
      inferior (truncothalamic)
   d. hypothalamic
3. *Choroid plexus*
   a. trigone
   b. tela chorioidea of III ventricle
4. *AVM of vein of Galen*
   a. fistula
   b. mixed form (plexiform + fistulous)
5. *Mesencephalon*
   a. ventral
   b. lateral
   c. dorsal
   d. entire

(extrinsic or intrinsic)

B. *Infratentorial central AVMs*

1. *Cerebellar*
   a. AVM of cerebellar nuclei (dentate and others) and cerebellar peduncles
   (No cases of isolated AVM of the nuclei or peduncles were observed but in 28 out of 58 cerebellar malformations the AVMs extended into the central areas and down to the pons)
   b. choroid plexus AVM of IV ventricle
2. *Pontine*
   a. ventrolateral
   b. dorsolateral
   c. entire pons

(extrinsic or intrinsic)

The main locations of AVM found in the current series, together with the various subgroups, and the frequency of their presentation are given in Table 1.3**a–b**, and the central AVMs are represented diagrammatically in Fig 4.**86**, page 204.

It should be noted that the incidence both of left sided supratentorial (dominant hemisphere) lesions and of central AVMs (37%) in this series, is probably artificially high in relation to natural prevalence. The cause of this is the pattern of referral and the choice of cases considered for surgery in Zurich (Tables 1.4–1.**5**). A more accurate estimate of incidence might be obtained from the figures of Laine et al. (1981), who found 46 cases (9%) of what would be termed deep central lesions, among 500 consecutive cases of AVM. Laine's figure of 27 good or excellent results after operating upon these deep central lesions is, incidentally, highly commendable.

Table 1.3a Location of the AVM in 414 operated patients

I. *Convexity AVM (261 = 63%)*

A. *Supratentorial* (203 cases)

	Operated			Non-operated
	Right	Left	Total	
Frontal				
Giant (whole lobe)	1	–	1	–
Dorsal	9	15	24	2
Basal	7	6	13	2
Polar	2	–	2	–
Paramedian (marginal)	2	3	5	–
Median	–	3	3	2
	21	27	48	6
Parietal				
Giant (whole lobe)	–	2	2	2
Dorsal	10	12	22	12
Paramedian	8	13	21	6
Median	2	2	4	4
	20	29	49	24
Insular				
Fistula	2	1	3	–
Anterior	1	2	3	–
Middle	3	3	6	4
Posterior	4	7	13	1
	10	13	23	5
Temporal				
Fistula	2	1	3	–
Giant (whole lobe)	1	1	2	1
Polar	7	3	10	–
Dorsal	10	7	17	4
Laterobasal				
anterior	3	1	4	–
posterior	3	14	17	–
	26	27	53	5
Occipital				
Giant (whole lobe)	3	–	3	1
Laterobasal	–	6	6	–
Dorsal	7	2	9	3
Paramedian	2	1	3	–
Median	1	4	5	–
Polar	1	3	4	–
	14	16	30	4
Whole hemisphere	–	–	–	2

B. *Infratentorial (cerebellar) convexity AVM* (58 cases)

	Right	Middle	Left	Total	Non-operated
Fistula	1	–	1	2	1
Giant (whole hemisphere)	3	–	3	6	1
Superior hemisphere	13	–	11	24	1
Inferior hemisphere	2	–	4	6	–
Vermis					
superior	–	8	–	8	2
inferior	–	2	–	2	–
Cerebellopontine (foramen of Luschka, flocculonodular)	4	–	6	10	1
	23	10	25	58	6

Table 1.3b Location of the AVM in 414 operated patients

II. *Central AVMs (153 = 37%)*

A. *Supratentorial* (149 cases)

	Right	Middle	Left	Total	Non-operated
1. *Limbic system* (98 cases)					
a. amygdalohippocampal	6	–	11	17	
b. parasplenial (post. hippocampal – isthmus gyri cing.)	18	–	22	40	
c. superior (callosocingular)					
1. cingular	3	–	4	7	1
2. splenium	1	1	4	6	
3. middle callosal	2	1	6	9 } 34	3
4. anterior callosal	6	–	6	12	
5. entire callosal	–	4	3	7	
2. *Strio-capsulo-thalamic* (26 cases)					
a. Hd. Caud. Nc.	7	–	4	11	–
b. putamen + pallidum + internal capsule	3	–	1	4	–
c. thalamic (palliothal.)					
superoanterior	–		2	2	–
superoposterior (pulvinar)	3		5	8	–
truncothalamic	–		1	1	12
d. hypothalamic	–		–	–	–
3. *Choroid plexus* (3 cases)					
a. lateral ventricle	1	–	2	3	1
b. III ventricle	–	(2)*	–	–	–
4. *Vein of Galen* (16 cases)					
a. fistulous	–	2	–	2	–
b. plexiform + fistulous	–	14	–	14	3

* combined with multiple callosal AVMs

B. *Infratentorial*

	Right	Middle	Left	Total	Non-operated
1. *Mesencephalic* (6 cases)					
a. extrinsic (subarachnoidal-epipial)	–	–	–	–	–
ventral	–	–	–	–	–
lateral	–	–	–	–	–
dorsal	(1)*	–	–	1	–
b. intrinsic					
ventral	–	–	–	–	1
lateral	2	–	–	2	–
dorsal	–	1	2	3	3
entire	–	–	–	–	4
2. *Cerebellar*					
a. dentate nucleus	?	?	?	?	–
cerebellar peduncles	?	?	?	?	–
b. choroid plexus of IV ventricle	–	(2)**	–	2	–
3. *Pontine* (4 cases)					
Extrinsic					
ventral	–	–	–	–	–
lateral	–	–	(2)***	–	–
dorsal	–	–	1	1	–
Intrinsic					
ventral	2	–	1	3	–
lateral	–	–	–	–	–
dorsal	–	–	–	–	–

( )* combined with orbital AVM
( )** combined with nodulus AVM
( )*** calculated under cerebellopontine