LONG TERM FOLLOWUP STUDY OF INTRACRANIAL AVM TREATED BY ENDOVASCULAR EMBOLIZATION

THESIS SUBMITTED TO THE SREE CHITRA TIRUNAL INSTITUTE OF MEDICAL SCIENCES AND TECHNOLOGY IN PARTIAL FULFILMENT OF THE RULES AND REGULATIONS FOR THE DM DEGREE EXAMINATION IN NEURORADIOLOGY

BY

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Certificate

Dr.Jayadevan.ER has been a DM neuroradiology postgraduate student in this department from January 2003 to December 2005

Certified that the materials for this thesis were obtained from the Sree Chitra Tirunal Institute of Medical Sciences and Technology, Thiruvananthapuram,based on bonafied cases investigated and studied by the candidate himself with technical assistance whenever required under my guidance.

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Contents

Long term followup study of intracranial AVM treated by endovascular embolization

Introduction

Brain AVMs are angioarchitecturally and hemodynamically complex systems of arteriovenous shunts with specific neurovascular relationships, a variable and unpredictable clinical presentation and a dynamic and only partially understood natural history, associated with a significant morbidity and mortality. Since their first clear description over a century ago (1), arteriovenous malformations (AVMs) of the brain have been increasingly recognized as an important cause of death and long-term morbidity, mostly due to intracranial hemorrhage and epilepsy. Technological advances in imaging the vasculature of the brain and the widespread availability of CT, MRI and intra-arterial digital subtraction angiography (IADSA) have augmented the rate of detection of AVMs (2), to the extent that they now pose a regular management problem. There is a growing interest in the frequency and clinical course of AVMs, although the pace of development of endovascular, surgical and radiation therapies seems to have overtaken the impetus to study their clinical course (3). The prognosis for any individual remains uncertain, as do the risks and benefits of the available treatments, leading to variation in practice and disagreement about the need for randomized trials

Background and purpose of the study

There are no systematic studies to addressing the natural history of the AVMs following endovascular embolisation. This study was designed to assess the efficacy of endovascular embolization and its after effects and also to understand more about the natural course of these AVM treated with endovascular embolization.

Aims of the Study

1. To describe clinical and radiological features in patients with brain AVM in relation to their location.
2. To formulate an effective reporting standard for endovascular treatment of AVM.
3. To formulate a set of diagnostic imaging criteria for the pre procedure assessment and for the follow up of Brain AVM patients.
4. To assess the effectiveness of embolization for brain AVM.
5. To find out difficulties and complications involved in the management of brain AVM and to recommend remedial measures.
6. To evaluate long term neurological status and outcome in patients treated with embolization.
7. To understand the natural history of Brain AVM treated with embolization.

Materials and Methods

This series consists of 103 patients with a brain AVM, which underwent endovascular treatment between January 2000 and October 2005.

Eighty-five patients were male and 18 female, 5 patients being children below the age of 16 years. The age ranged between 1 and 59 years, with a mean age of 34 years. Thirty nine patients (37%) presented with intracranial hemorrhage or had a history of previous hemorrhage. Forty six patients (44%) presented with epilepsy, 7(6%) with focal neurological deficits, 11(10%) with chronic headache. In 8 patients (7%) the AVM was detected incidentally by CT or MR performed for non-specific or vague complaints. All patients underwent embolizations at our institute.

A total of 170 embolization sessions were performed in the 103 patients, with an average of 1.6 sessions per patient. 58 patients received one session, 29 patients two sessions, 11 patients three sessions, 4 patients four sessions, 1 patient five sessions. All patients underwent endovascular treatment on a monoplane DSA unit with high – resolution live road mapping capability (ADVANTEX - LCV, GE medical system, Milwaukee, USA).

With the exception of four patients, which underwent emergency embolization in the presence of an acute hematoma, all other patients underwent preembolisation CT or MR and MR–angiography (MRA). Tri planar T1 –weighted MR was performed for topographic assessment of the AVM and axial T2 weighted MR

was performed for evaluatation of brain parenchyma. MRA was performed with 3D phase contrast and 3D time of flight techniques.

Majority of embolizations were performed general, and very few (13 sessions) were done under neurolept analgesia. All Children under the age of 15 years were embolized under general anesthesia

All procedures were performed via the transfemoral route using the Seldinger technique. The guiding catheters used during the procedure were continuously flushed with heparinized saline (2000 units /L) with systemic heparinisation of 5000 IU as bolus and 1000 IU every hour.

Superselective catheterizations and embolic injections were performed, using exclusively variable stiffness microcatheters of either Spinnaker or magic Microcatheter.

Decisions regarding injection of embolic material were taken following anatomic evaluation of superselective angiograms and preembolisation MR and with the conviction that all brain areas are eloquent.

Cyanoacrylate (NBCA), absolute alcohol and Bernstein liquid coils were the embolic material of choice used throughout this series. Cyanoacrylates were used as the sole embolic material in 80 (77%) cases of this series, 10 (9%) patients were embolized with absolute alcohol, 11 (10%) patients had both NBCA and alcohol. In two cases with high flow fistulae either Bernstein liquid coils used prior to cyanoacrylate embolization, because of angioarchitectural reasons.The techniques of catheterization and

embolisation used throughout this series are described in detail further below.

Immediate post embolisation brain CT was done in those cases with suspicion of complication. Patients with AVMs in which a complete obliteration was achieved, had follow up MR/CT or angiography at 6 months following the last embolization session and at variable intervals ranging between 1 and 4 years 6months. Follow up data from all patients were obtained from hospital case record, specially prepared questionnaire and also telephonic conversation with patients or their immediate relatives.

Results of Endovascular Treatment of Brain AVMs

Total no. of patients underwent embolization was 103, of this 85 were males and 55% were between 20-40yrs age group. Commonest presenting feature was seizures(46 patients, 44%) next was hemorrhage (39 patients, 37%), then head ache (11patients, 10%) and weakness (7 patients, 6%).

Age group(yrs)	Number of patients
11-20	22
21-30	32
31-40	24
41-50	17
51-60	6

Presentation	**Number of patients**
Seizure	46
Hemorrhage	39
Head ache	11
Weakness	7

Most commonly involved lobe was frontal, then parietal, occipital and temporal. AVMs involving 2 lobes commonly involves fronto parietal and parieto-occipital lobes. AVMs involving more than 2 lobes all cases were in the tempero parieto occopital areas.Of this 80% of AVMs were of high grade type(grade 3,4 and 5).Total stages of embolization were 170 about 28-32/year (562 feeder embolisations, 3.3feeder embolisation per stage), of this 4 patients had 4 times and 2 patients had 5 times embolization. Regarding the mode of embolization 80 patients had embolisation with NBCA alone, 11 patients had alcohol and NBCA, 10 patients had alcohol alone and 2 patients had embolization with NBCA and Bernstein coil.

AVM Grade	**Number of patients**
Grade 1	1
Grade 2	18
Grade 3	45
Grade 4	27
Grade 5	5

During each stages of embolisation with NBCA produced 20-70% nidal obliteration. Twelve cases showed complete nidal obliteration with NBCA.In 19 cases only minimal obliteration could be achieved, either because of angioarchitectural reasons or because the patients refused further embolization sessions.

In 22 patients treatment is not yet completed as additional stages of embolizations are remaining. In 8 patients with mainly large and angioarchitecturally complex AVMs or with diffuse type of nidus only palliative embolization either targeted on weak angioarchitectural elements or to palliate chronic severe headaches was performed.

Significant nidal obliteration rate was noted when alcohol beeing used as an embolizing agent. Of the 10 patients embolized with alcohol alone 7 had total angiographic obliteration of the nidus, two cases showed complete obliteration after radiotherapy and one showed tiny residual nidus (waiting for radiotherapy).

Nearly 84% of the patient had significant decrease in the symptoms resulted in the reduction of anti epileptic drugs in most, no change in symptoms in 11% and 5% showed increase of symptoms.

After the embolization 21 patients underwent stereotactic radiotherapy, after the radiotherapy significant improvement in the symptom noted in 18 patients, no change in 2 patients and increase in symptoms in one patient, and one patient showed rebleed also. Of this 21 patients check angiogram was done in 13 patients, 6monts to 2 years patients after radiotherapy, which showed complete nidal obliteration in 9 patients and residual small nidus noted in the rest of the 4 patients.

Post embolization surgery was done in 6 cases, of this post surgical weakness developed in 2 and rest of the 4 patients made

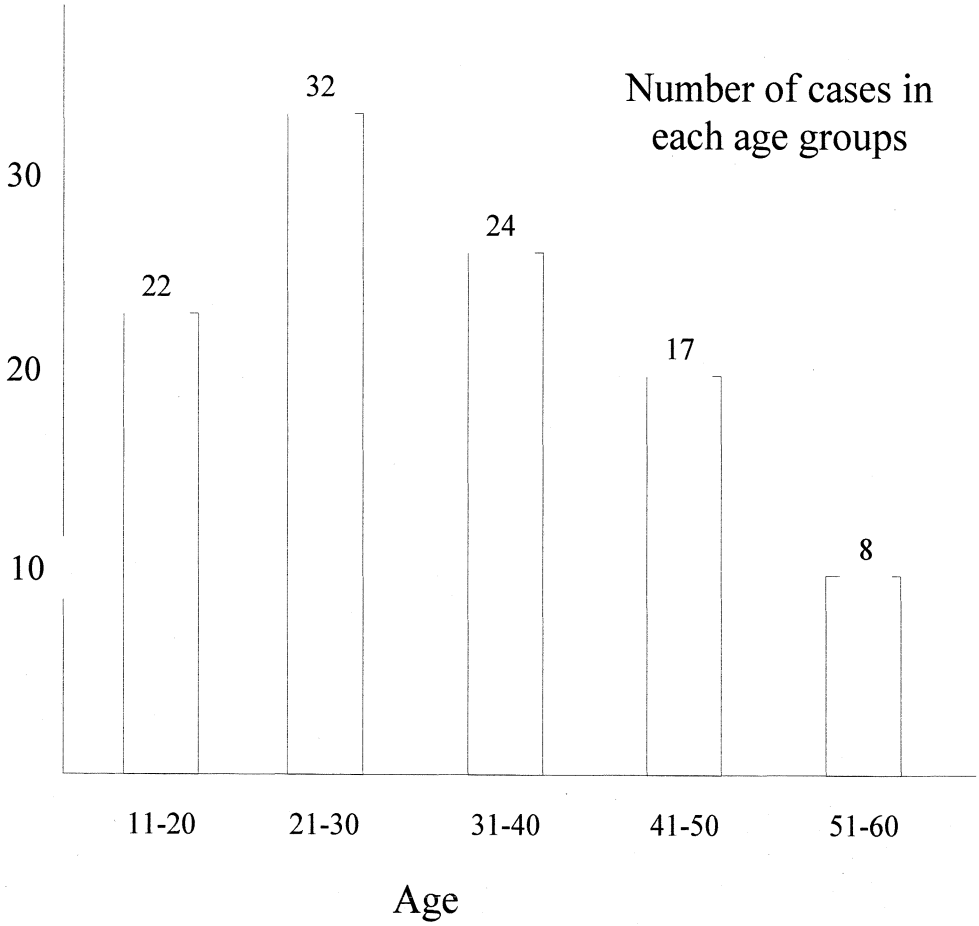
a good symptomatic recovery. Post surgical CT or DSA showed complete nidal obliteration in all the cases. Total nidal obliteration rate with embolisation alone was 18%, post embolisation stereotactic radiotherapy or surgery or both has increased the total occlusion rate to 36%.

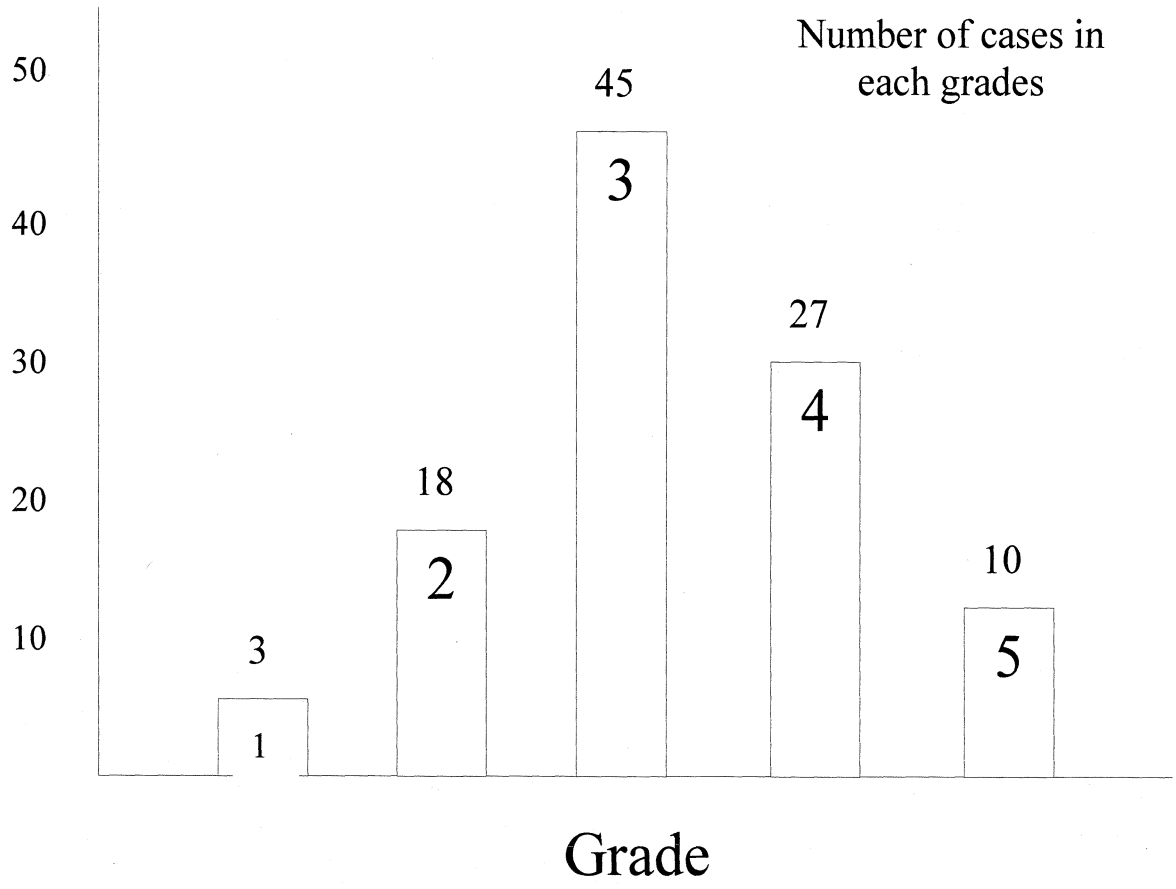
Of the 103 patients, 18 patients ((17.4%of total and 3.1% per year) suffered a hemorrhage during the follow up period. In 8 hemorrhages were immediately after the embolisation, of these 3 died due to massive hemorrhage. Other patients recovered completely from the hemorrhage.

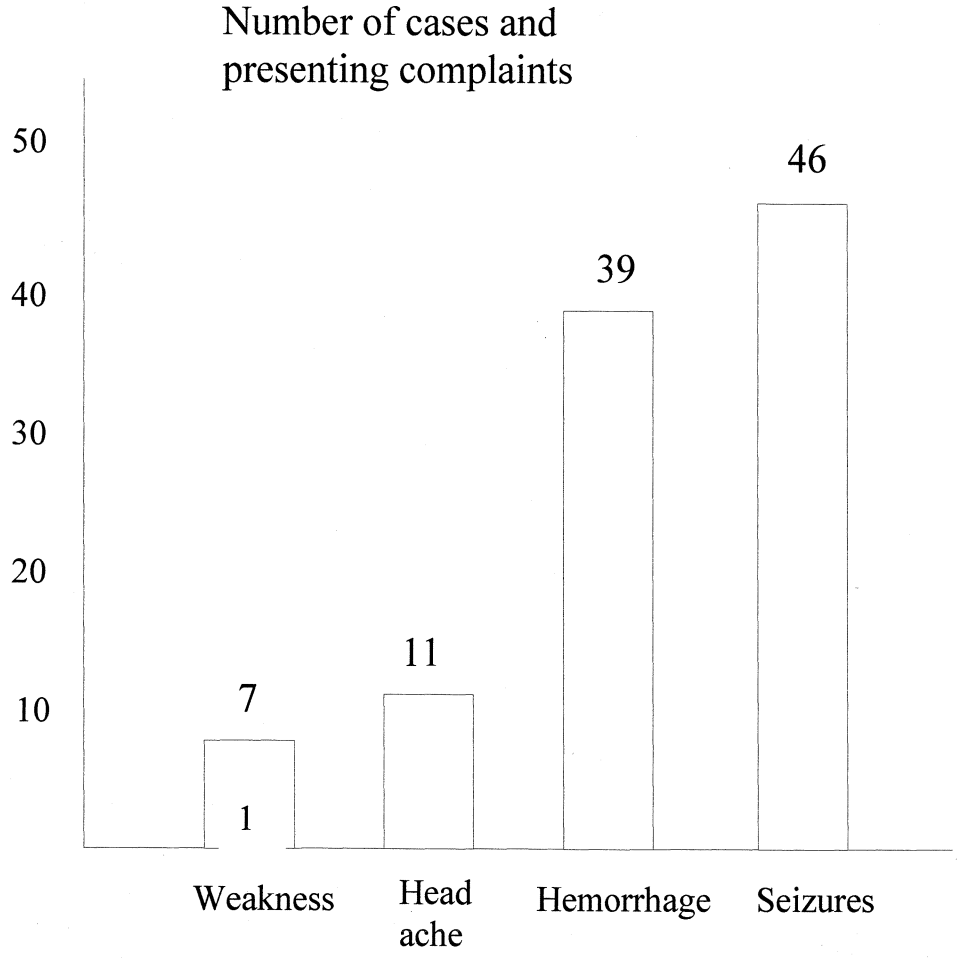
Immediately after embolisation 7 patients developed fresh neurological deficits, except one all recovered completely over a period of 1-6 months. One patient still has residual hemiplegia.

Catheter gluing was noticed in 5 cases; there was associated vasospasm in 2 cases, which were relieved off with papaverine and NTG. In one case intracerebral abscess with discharging sinus developed which later completely excised surgically. One patient had associated sigmoid venous sinus stenosis for which stenting was done.

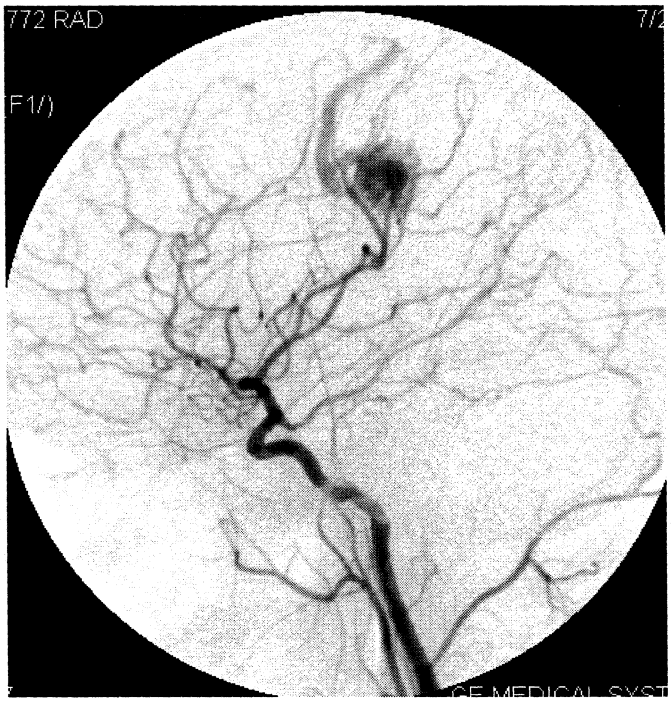
Out of these 103 patients, 3 died of post embolisation intracranial hemorrhage(within 30 days of embolisation), 2 died of ischemic heart disease, one died of post embolisation DVT and pulmonary embolisation. Morbidity rate was 8.7% and mortality rate was 2.9%


Number of cases in each age groups
30
20
10
32
24
22
17
8
11-20
21-30
31-40
41-50
51-60
Age

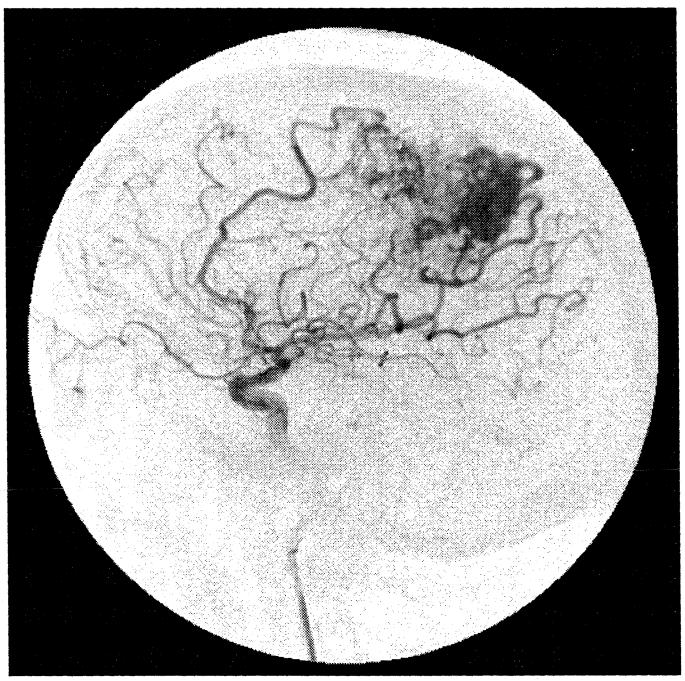

Number of cases in each grades
50
40
30
20
10
3
18
45
27
10
1
2
3
4
5
Grade


Number of cases and presenting complaints
50
40
30
20
10
46
39
11
7
1
Weakness
Head ache
Hemorrhage
Seizures

Angioarchitecture



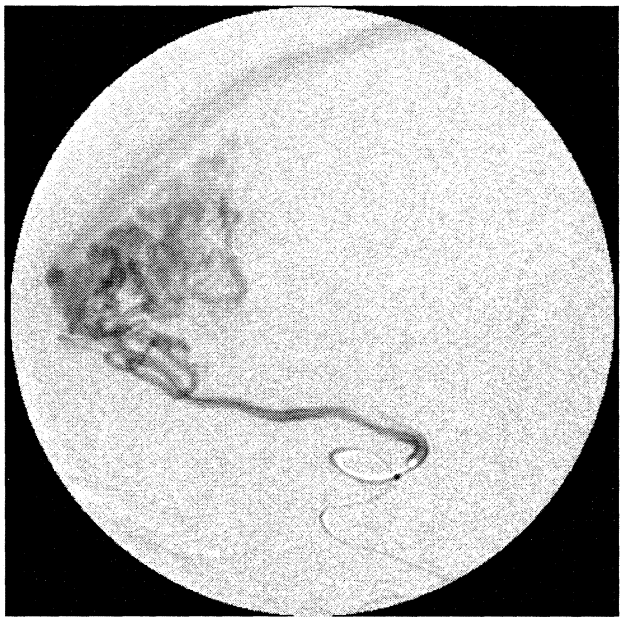
Feeding artery Nidus Draining vein

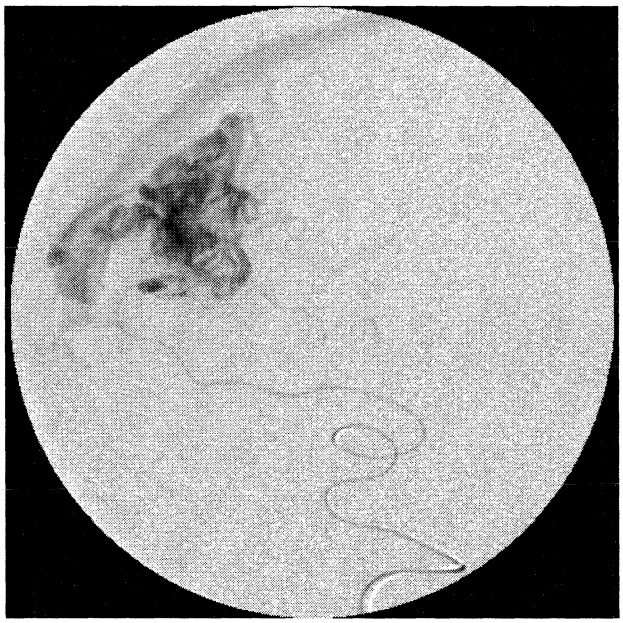


En-Passant Feeders Terminal Feeders

Angioarchitecture

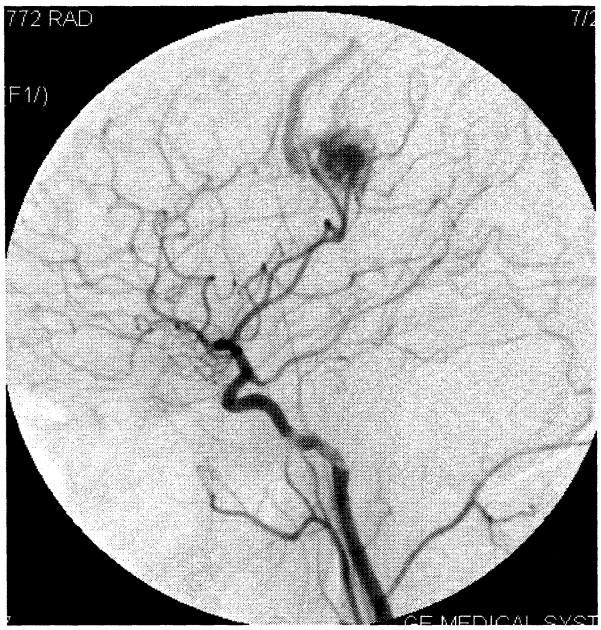
Feeder

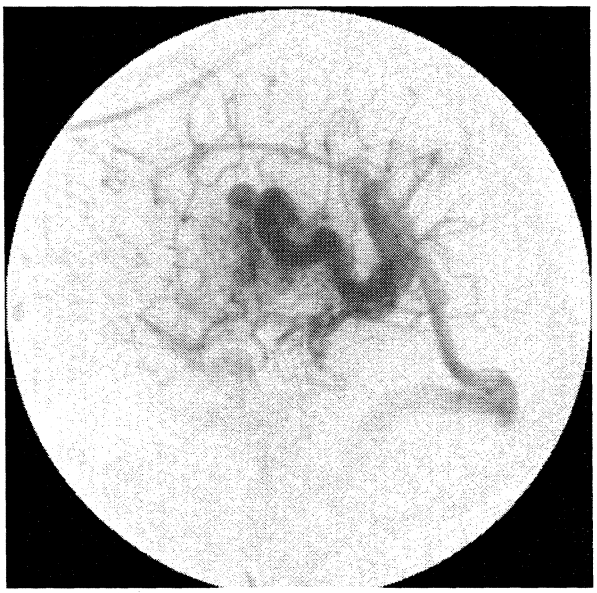




Nidus

Superficial Venous Drainage

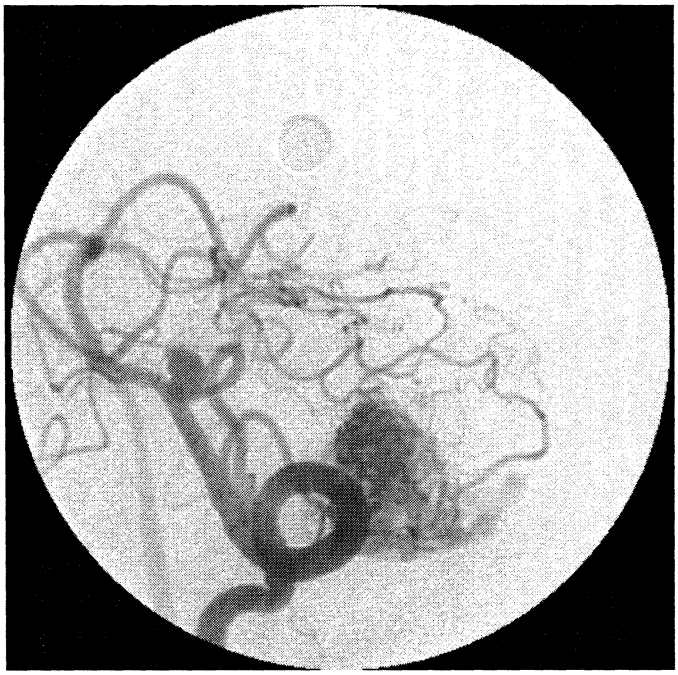


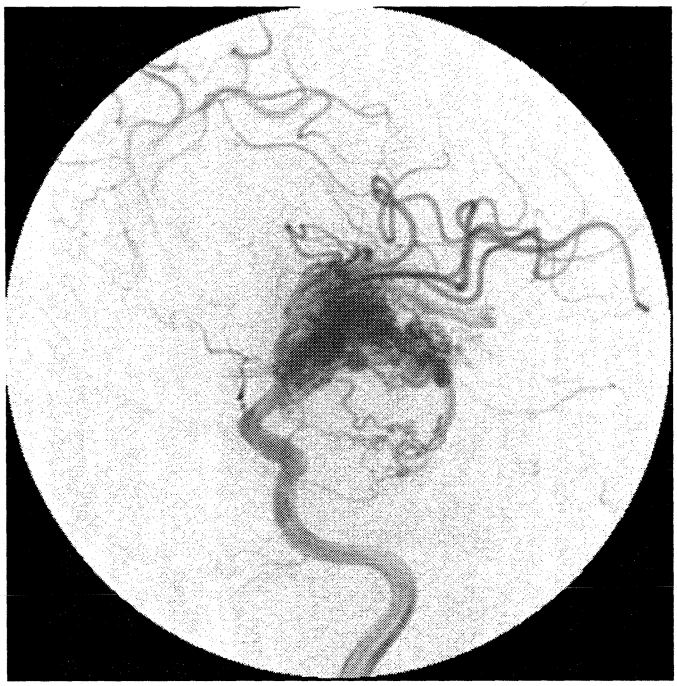


Deep venous Drainage

Angioarchitecture

Feeding Artery Aneurysm

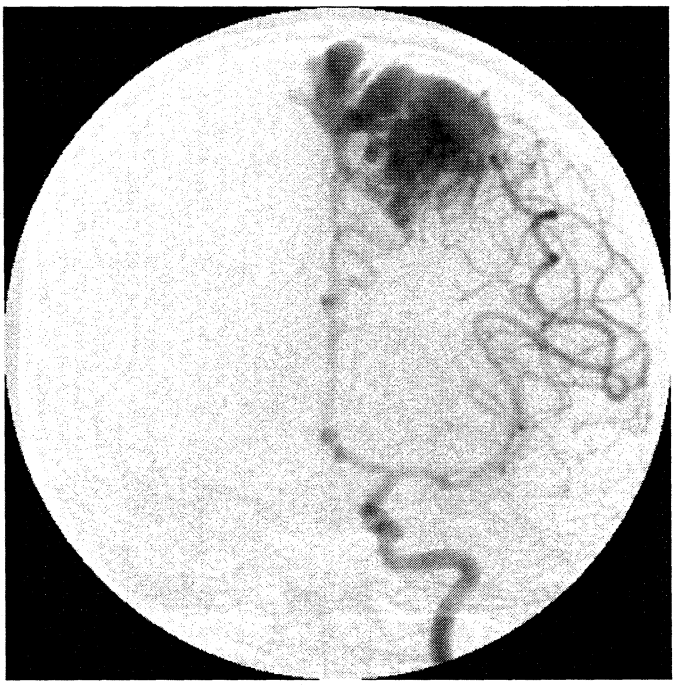


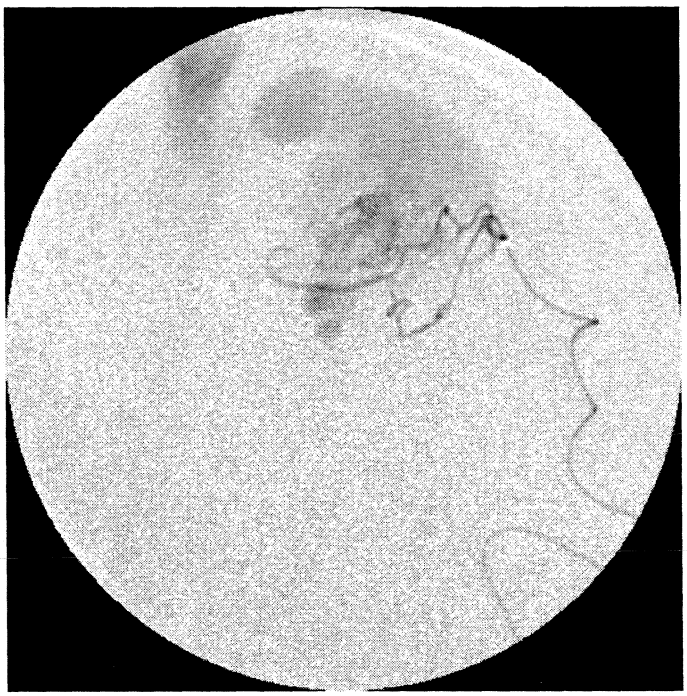


Intranidal Aneurysm

Angioarchitecture

Venous Stenosis

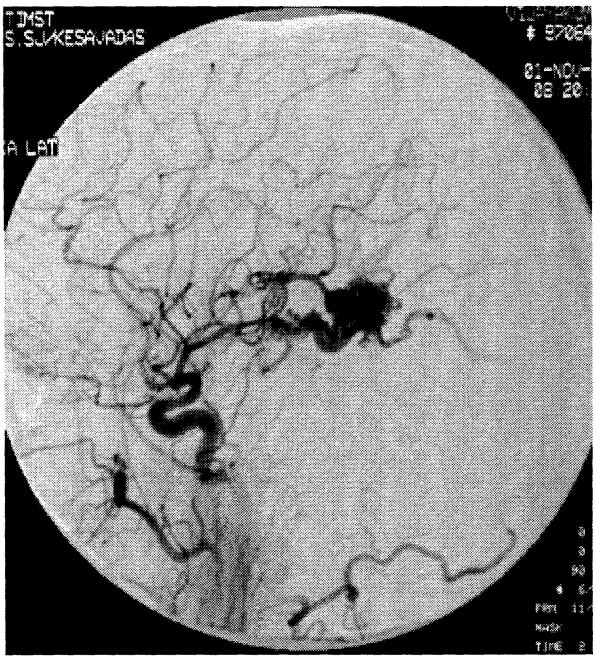


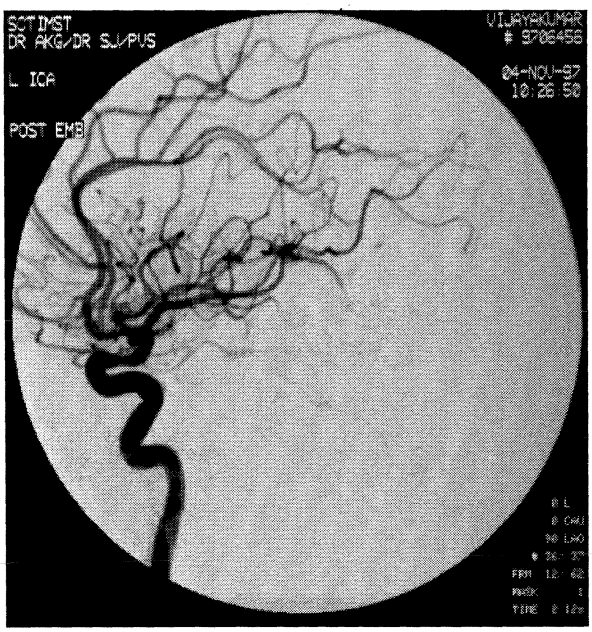


Venous Ectasia

Embolisation with Glue

Pre embolisation

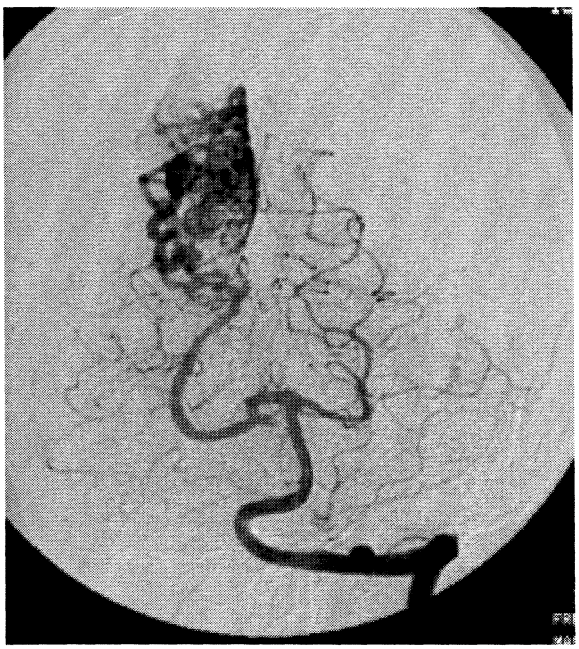


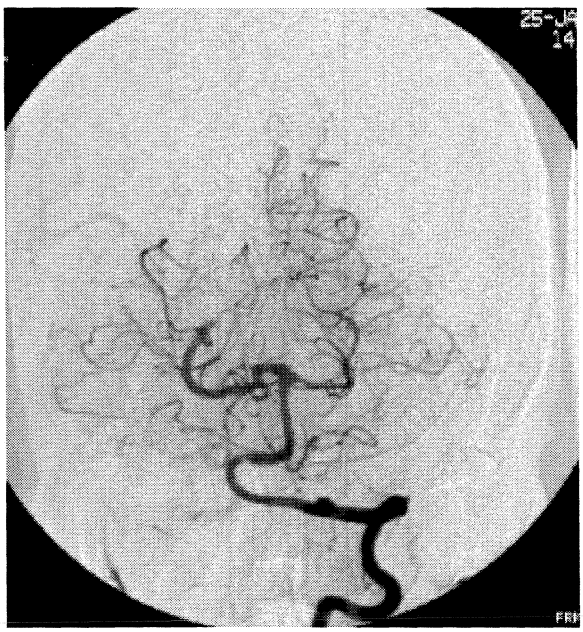


Post embolisation

Embolisation with Glue

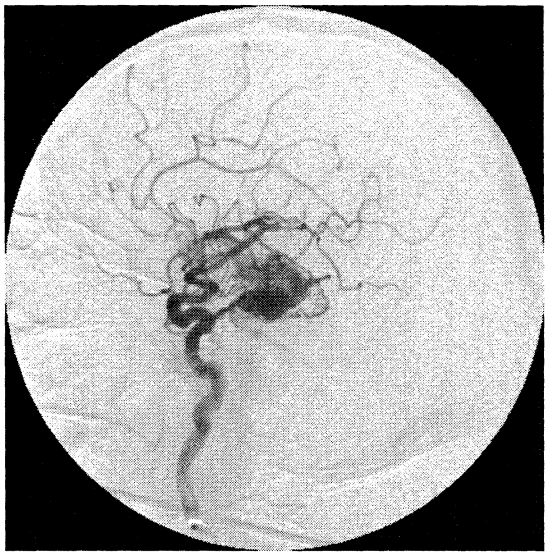
Pre embolisation

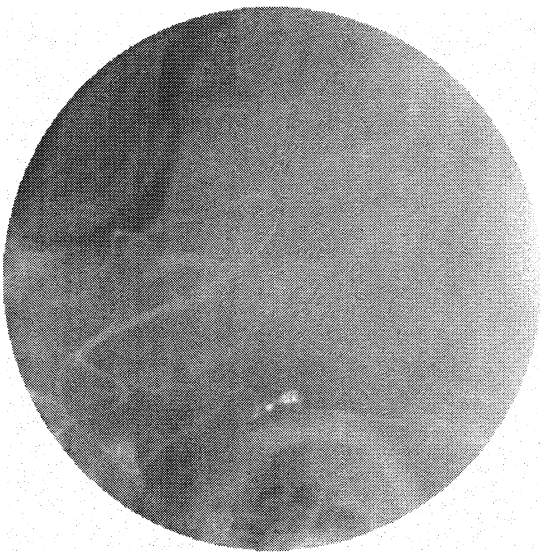


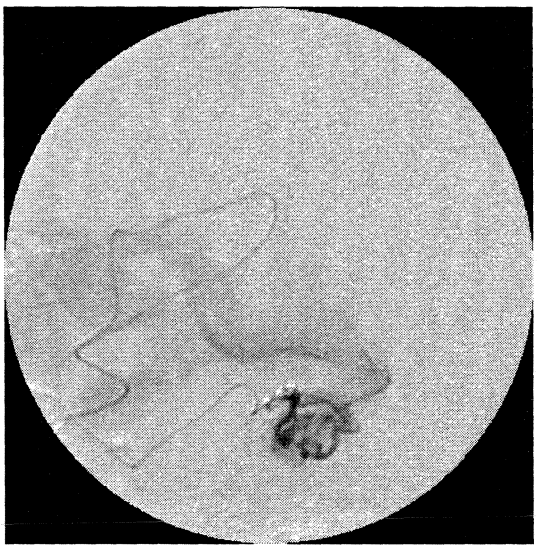


Post embolisation

Berenstein Liquid Coil for Flow Reduction

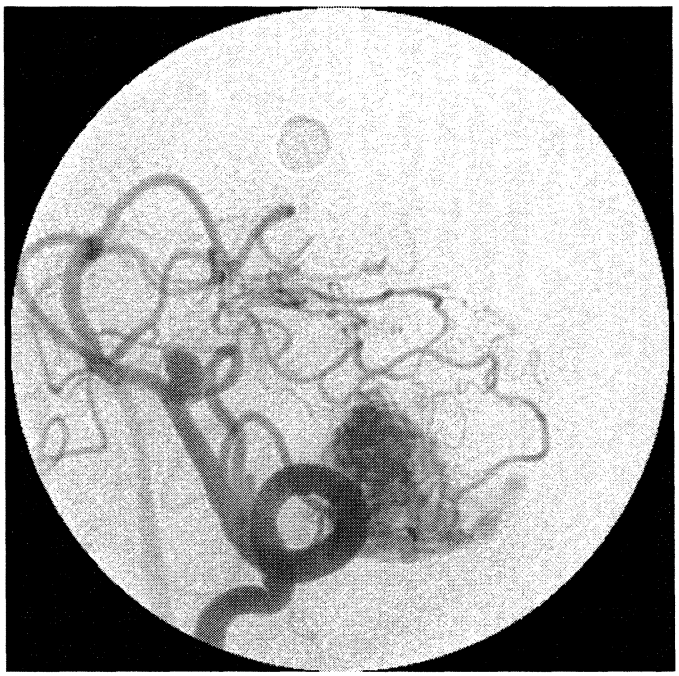


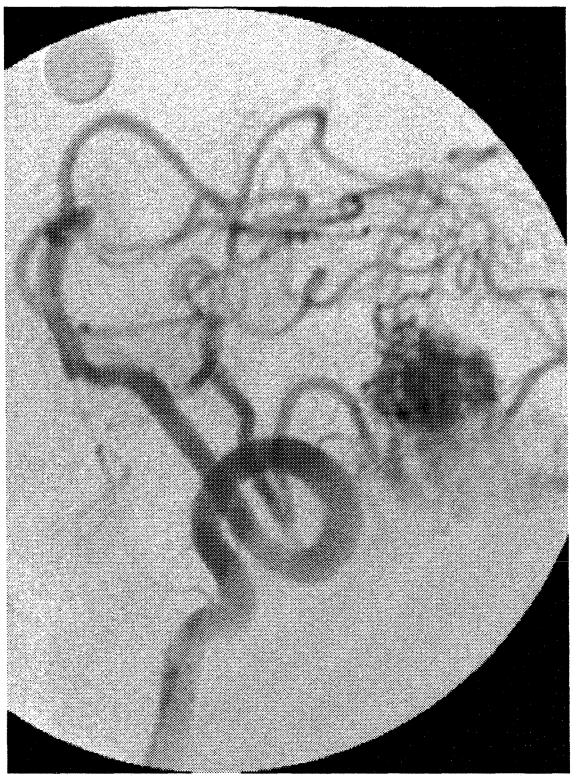




AVM with Feeding A. Aneurysm

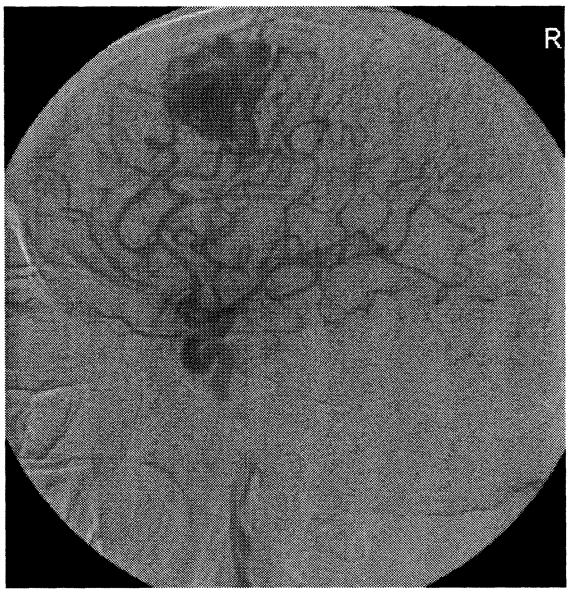
Pre and post coiling



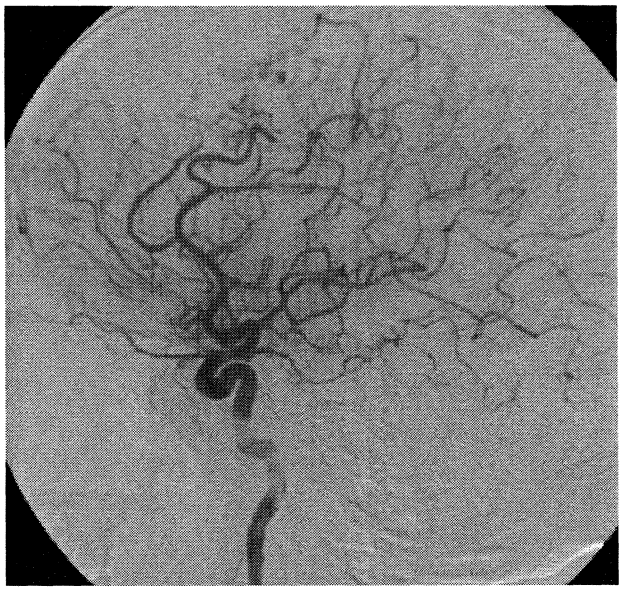


Ethanol embolisation

Right frontal AVM

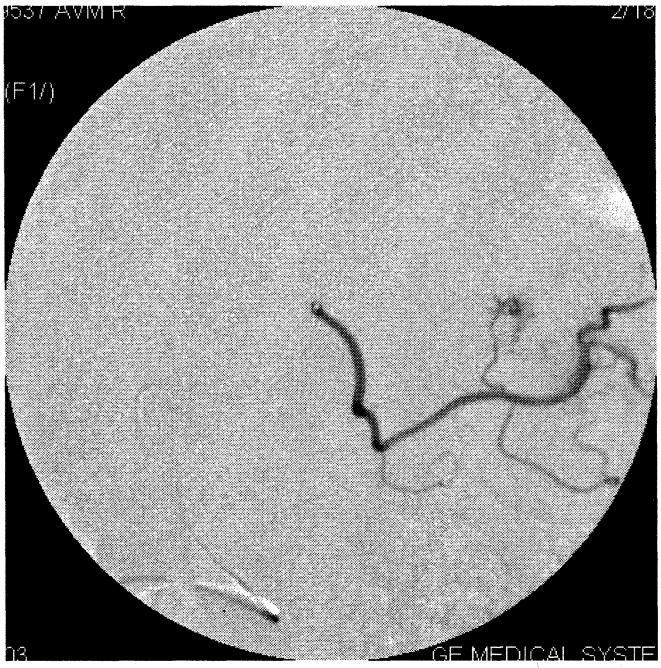


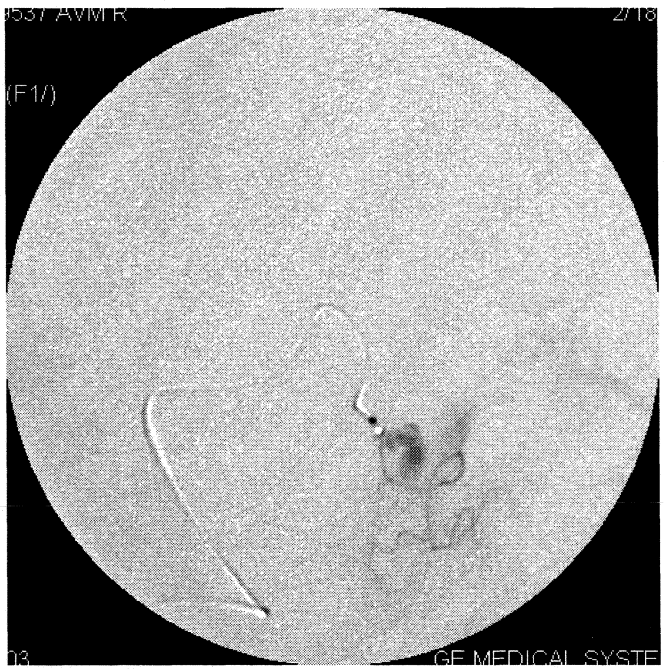
Pre-embolisation



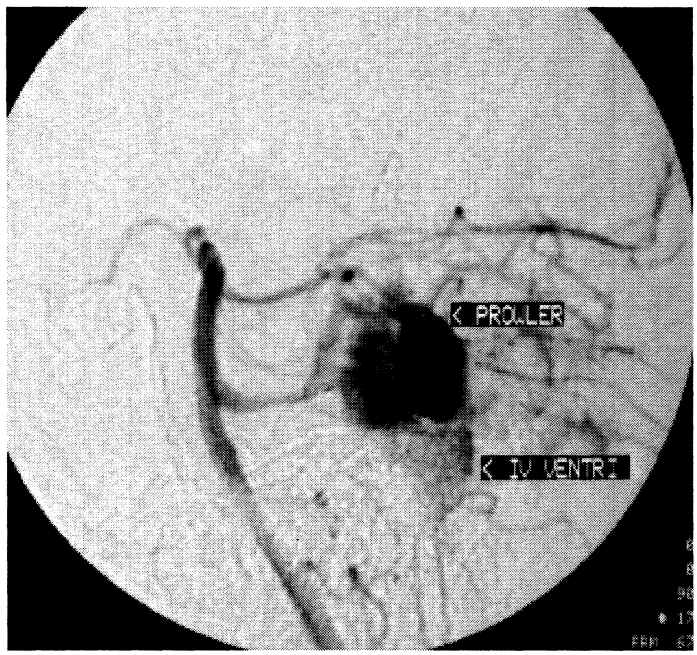
Post- embolisation

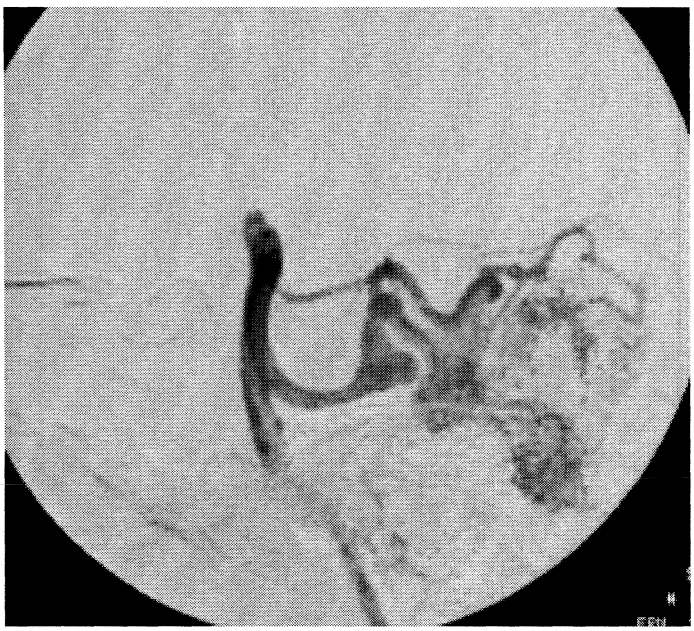
Rupture of the feeding artery during catheterization



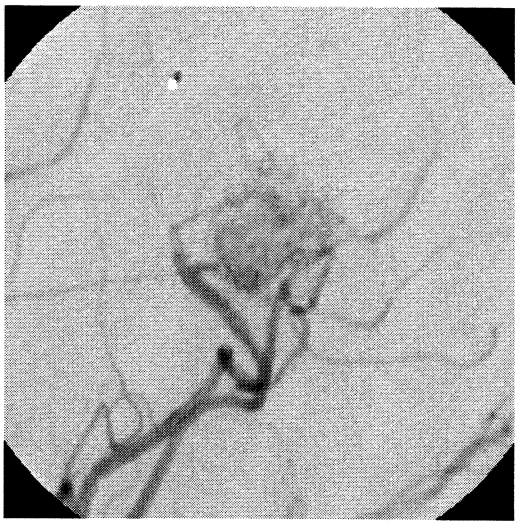


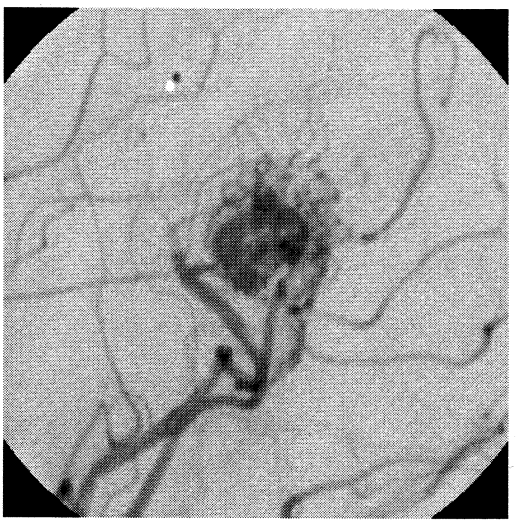
Rupture of the nidus into 4th ventricle during embolisation

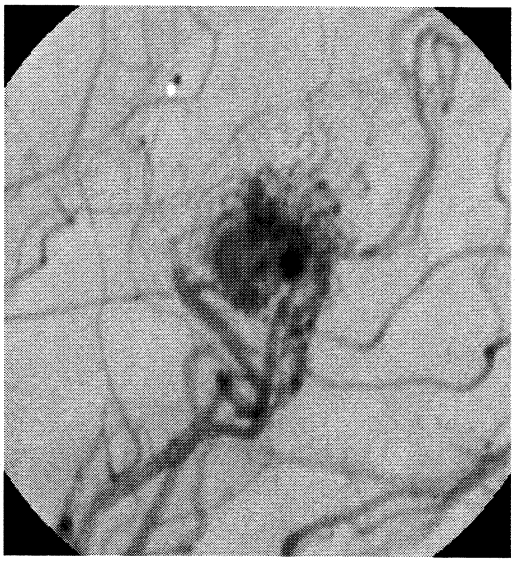


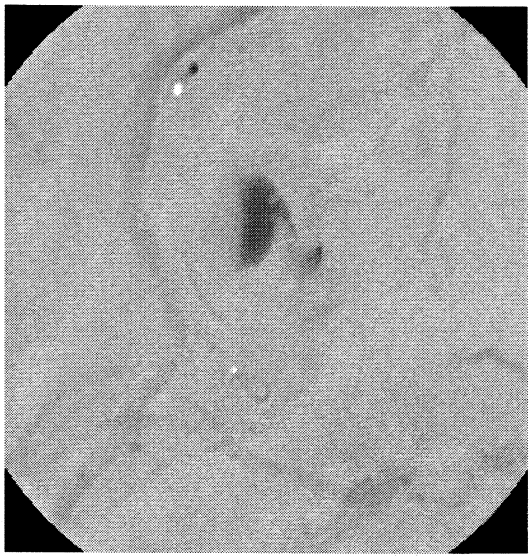


Rupture of the nidus during procedure



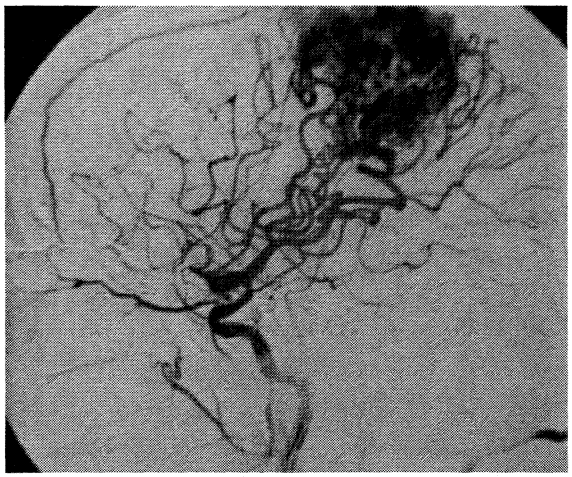




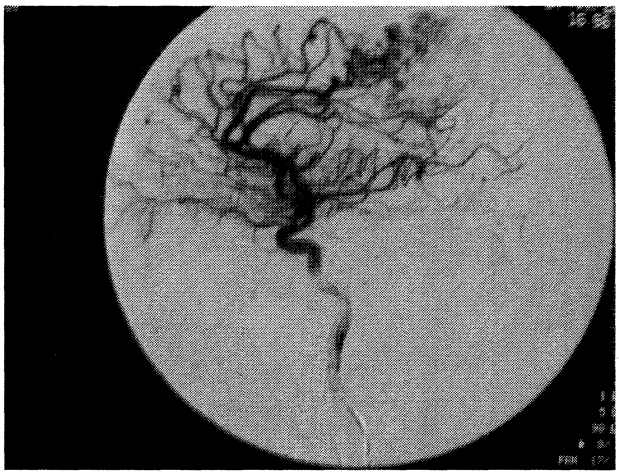


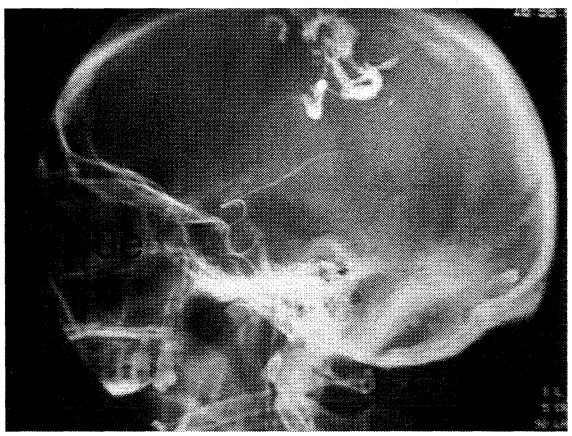
PYOGENIC INFECTION OF AN EMBOLISED AVM

Pre embolisation

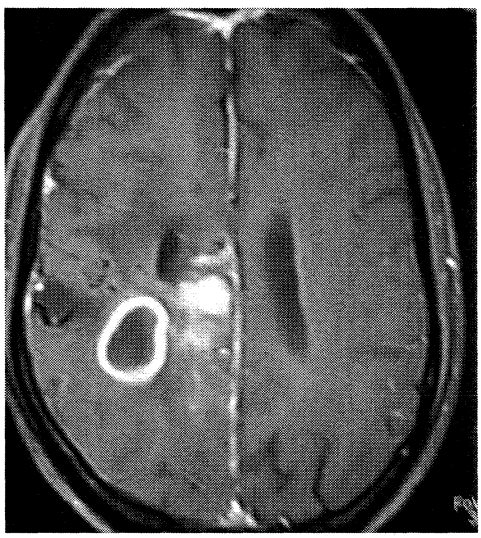


Post embolisation





Glue cast



CE MRI - Cerebral abscess

Review of literature and Discussion

Introduction and Historical Perspective

Brain AVMs are angioarchitecturally and hemodynamically complex systems of arteriovenous shunts with specific neurovascular relationships, a variable and unpredictable clinical presentation and a dynamic and only partially understood natural history, associated with a significant morbidity and mortality.

Brain AVMs are generally regarded as congenital lesions representing inborn errors of embryonic vascular morphogenesis caused by a defect or malfunction of the embryonal capillary maturation process and resulting in the formation or persistence of arteriovenous shunts (4).

There is increasing evidence that the majority of brain AVMs, with the exception of aneurysmal malformations of the Galenic vein, develop postnatally and represent a complex endothelial cell dysfunction, triggered by still unknown factors. In favor of this hypothesis are recent immuno-histochemical studies performed on surgically obtained specimens of human brain AVMs which demonstrated, that the pre pro endothelin-gene is locally repressed in brain AVMs (5). The repression of this gene is an intrinsic phenotype of endothelial cells of brain AVMs and is not due to factors in the microenvironment of the AVM. In addition, other recent studies demonstrated expression of vascular

endothelial growth factor predominantly in the subendothelial layer and in perivascular spaces of vessels composing brain AVMs (6).

Embolization represents one of the modalities currently available for the treatment of brain AVMs, the others being microneurosurgical removal and stereotactic radiotherapy. The theoretical advantage of the endovascular approach is, that it avoids any direct interference with the brain parenchyma such as cortical incisions, brain retraction, and cranial nerve manipulations. The limitations are mainly technical and include the in accessibility of certain AVMs or parts of AVMs with the currently available microcatheters and the occasional unpredictability of behavior of the injected embolic agent within the vascular spaces of the AVM.

During the years, embolization advanced from a simple technique initially conceived to block feeding arteries of AVMs to a sophisticated one with the aim to use the feeding arteries as vascular routes to reach and obliterate the core, so called nidus of the AVM.

The endovascular approach to brain AVMs dates back to Brooks (7), who injected muscle particles into the surgically exposed internal carotid artery in order to occlude endovascularly carotid-cavernous fistulas. Lussenhop and Spence (8) were the first to used this concept introduced thirty years earlier by Brooks to treat brain AVMs. They used steel particles (spheres) covered with methyl methacrylate introduced into the surgically exposed internal carotid-cavernous fistulas. Lussenhop and Spence (8) were the first to use this concept introduced thirty years earlier by Brooks (7) to treat brain AVMs. They used steel particles

(spheres) covered with methyl methacrylate introduced into the surgically a brain AVM, and called this technique "artificial embolization"(8). Using barium subsequently refined this technique – impregnated Silastic spheres introduced into the cerebral circulation through a catheter inserted surgically into the common carotid artery and advanced manually into the internal artery. (9, 10, 11).

In 1964 Dolce (12) reported on the first superselective catheterization of the cerebral arteries using a self-constructed 1.5 mm thin microcatheter tapering at its tip to 0.7 mm, inserted percutaneously with the Seldinge technique into the internal carotid artery and advanced into the proximal anterior and middle cerebral arteries.

In the late sixties, Yodh et al (13) and Hilal (14) in an attempt to improve intravascular navigation developed and applied independently magnetic control systems used with Silastic catheters, but without success regarding distal micro catheterization.

In the early seventies, the percutaneous transfemoral catheter embolization technique which enabled either the anterior or the posterior cerebral circulation to be approached. This nonselective endovascular technique using flow-directed particles was applied palliatively for embolization of large brain AVMs or preoperatively (14,16).

A major technical breakthrough in endovascular techniques occurred in 1974, when Serbinenko (17) reported for the first time on the introduction of detactable balloons mounted on the tip of

microcathetes and used for flow-guided intracranial navigation beyond the circle of Willis and for occlusion of major cerebral arteries as well as occlusion of feeding arteries related to brain AVMs and AV fistulate. Serbinenko's technique had major impact on the further refinement of cerebral endovascular techniques leading to the modern era of super selective microcatheterization and embolization, pioneered by Djindijan (18) in Europe and Kerber (19) and Pevsner (20) in the United States.

Kerber 1976 (19) introduced flow-guided, calibrated – leak microballon catheter systems consisting of very flexible and soft Silastic, pemiting supeselective microcatheterization of brain AVM feeding arteries and embolization with acrylics. Pevsner (20) introduced a pressure chamber to enhance the propulsion of the calibrated-leak microballoon – catheter and Berenstein (21) and Debrun et al. (22) introduced calibrated – leak latex balloons to improve small vessel microcatheterization and acrylic delivery. This technique was in routine use until 1986/87, when an era of superselective cerebral vascular navigation began with the introduction of the newest generation of variable stiffness microcathetes, used either in conjunction with microguidewires (23) or as flow-giuided systems (24) and permitting safer and more controllable super selective catheterization.

Epidemiology, Clinical Presentation and Natural History of Brain AVMs

Brain AVMs exhibit a wide variability in size, location composition and clinical presentation so that accurate determination of the natural history of an individual case is difficult and may even be impossible.

The prevalence of brain AVMs in the general population is uncertain and is probably influenced by geographical and racial factors. Jellinger (25) reported a prevalence of 0.11% calculated from general autopsy series on 41.395 cases and Karhunen (26) estimated the prevalence to the 0.06% based on a material of 8.038 consecutive medico-legal autopsies. In North America the prevalence has been estimated to be between 0.02% and 0.08% (27). The incidence, i.e. the frequency of newly diagnosed bain AVMs per year, has been estimated to be 0.001% - 0.01% (28, 29,30).

Hemorrhage is the most common presenting symptom of a brain AVM being reported to occur as the initial manifestation of the disease in approximately 50% of patients with an AVM (31,32, 33). There is increasing evidence, that the incidence of hemorrhage is higher. It is not unusual to find old hemorrhagic areas on MR images or to observe small chronic hemorrhage during microsurgical removal of AVMs in patients, in whom these events have not been detected clinically (34,35,36). It has been postulated, that episodes of acute headaches, seizures or other acute neurologic symptoms may represent hemorrhages.In our

study most of the patients presented with seizures then with hemorrhages.

In a prospective study of 166 patients with untreated symptomatic cerebral AVMs, Ondra et al. reported an annual bleeding rate of 4% regardless of the initial clinical presentation (30). This annual bleeding rate remained constant throughout the follow-up period of 24 years. The first hemorrhage is associated with a mortality of 10% reaching up to 20% for subsequent recurrent hemorrhages (37). In the prospective study of Ondra et al. (1990), the annual rate of mortality was 1% and that of severe morbidity 1.7%, this rates being constant over the course of the study, 85% of the patients who bled, corresponding to 34% of the patient population of the study, either died or suffered severe morbidity during the 24 years of the study. Our study showed a post embolisation bleed of 3.1% per year.

Several studies correlated the angiographic features with the clinical presentation of patients with brain AVMs (27, 38, 39,40,42,43,44, 45, 46, 47, 48). These studies clearly showed, that there is a statistically significant increased incidence of hemorrage in AVMs that are associated with flow-related aneurysms, stenoses or occlusion of draining veins, or that are associated with flow-related aneurysms, stenoses or occlusion of draining veins, or that are located in the deep parts of the brain and in the posterior fossa. It has been repeatedly postulated, that small and micro-AVMs are at higher risk of hemorrhage than larger AVMs because the pressures in the feeders and the nidus of small AVMs were found to be significantly higher than in larger AVMs (49, 50). Recent reports suggest, that there is no direct correlation

between the size of the AVM and the incidence of hemorrhage (27, 28, 30, 47, 51). Small AVMs are usually asymptomatic before hemorrhage and their most frequent manifestation is due to rupture. This is in contrast to larger AVMs, which more often present with seizures or neurological deficits.

Seizures are the second most frequent presenting symptom of cerebral AVMs following hemorrhage reported to occur in 28% to 67% of patients with an AVM (27, 32). The incidence of seizures is particularly high in AVMs of the temporal lobe and in those involving the motor sensory strip (52). Retrospective analysis of angiographic data of patients with AVM predicated venous hypertension may be a major cause of epilepsy. (27, 45). This evidence if further substantiated by the fact, that AVMs located in non-epileptogenic areas, such as the basal ganglia and the cerebellum, may cause seizures following venous thrombosis and rerouting of venous drainage (27). Seizures may also be a clinical manifestation of minor hemorrhage. The risk of hemorrhage in patients experiencing seizures related to their AVMs is lower than in those with previous hemorrhage, but higher than in patients without a history of epilepsy (49,52). In the prospective study of Ondra et al (30), patients with AVM related hemorrhage and patients with epilepsy had similar long-term morbidity and mortality.

Neurologic deficits not associated with previous hemorrhage occur less frequently than seizures or bleeding and may be caused by several mechanisms including steal effect of the AVM, decreased perfusion because of associated arterial stenoses,

venous hypertension or mass effect caused by compression of brain parenchyma by venous estasias or varices (27).

Headache not associated with an acute hemorrhage is a relatively frequent complaint of patients with AVMs. Angiographic –clinical correlations have shown, that dilated feeding arteries and draining veins, in topographic relationship with the meanings and the tentorium, may be responsible for chronic headaches in patients with brain AVMs. Further more, the incidence of headaches is particularly high in patients with additional dural supply of their AVM and in these with an occipital location of the lesion (27).In respect to the annual incidence post embolisation hemorrhage our study showed almost similar results compared to the natural history, but embolisation has resulted in significant reduction of patient's complains (84%).

Topographic Classification of Brain AVMs

Yasrgil (36) introduced a refined classification of brain AVMs taking also in consideration the intrinsic arterial supply and the pattern of the venous drainage and incorporating the surgical approaches used for removal of brain. AVMs According to this topographic classification, brain AVMs are divided into two main groups, i.e. convexity or pallial AVMs with supra and infratentorial subgroups and in deep central AVMs with supra and infratentorial subgroups. The convexity or pallial includes the cortical and subcortical areas of the frontal, temporal, insular, parietal, occipital and cerebellar regions. The central of deep system includes the grey matter nuclei of the diencephalon, mesencephalon, metencephalon, connecting white matter fibers, the entire limbic system (amygdala and hippocampus, cingulated gyrus, corpus callosum, parasplenial area and fornix) and the choroids plexus of the lateral, 3rd and 4th ventricles (36). From the perspective of endovascular treatment of brain AVMs this classification system, although useful to a certain extent, fails to correlate the type of feeding arteries with the specific location of the AVM. For that purpose, amore detailed vascular anatomic topographic subdivision of brain AVMs was necessary. This was achieved with the routine application of MR imaging in the pre therapeutic evaluation of brain AVMs. For that purpose, a more detailed vascular anatomic topographic evaluation of brain AVMs was necessary. This was achieved with the routine application of MR imaging in the pre therapeutic evaluation of brain AVMs MR imaging proved essential for a precise localization of brain AVMs. MR imaging in the pre therapeutic evaluation of brain AVMs. Mr

imaging proved essential for a precise localization and topographic evaluation of brain AVMs. TI-weighted, triplanar MR imaging allows the evaluation of he AVM nidus with respect to adjacent gyri, sulci and subcortical white matter in cases of convexial or pallial (So-called cortical) AVMs and with respect to adjacent gyri, sulci and subcortical white matter nuclei and the white matter tracts in cases of deep or central AVMs. Correlation of the topographic information derived from MR imaging with th angioarchitectural information derived from cerebral angiography provides a better under standing of the particular vascularization and drainage of each AVM in relation to its specific location.

Based on MR imaging, both convexial and deep AVMs can be further and more precisely classified into district subtypes. Knowledge of the sulcal and gyral anatomy of the brain and application of MR criteria to correctly identify individual sulci, gyri and other structures are essential when analyzing an AVM with regard to location and topography (53,54).

Convexity of or so – called cortical AVMs. Within the group of convexity or so-called cortical AVMs three main types can be distinguished by MR imaging :

1) Sulcal AVMs

2) gyral AVMs and

3) mixed sulco gyral AVMs (55).

To understand the arterial supply of the sulcal and gyral AVMs and analyze superselective angiograms of these types of AVMs. Knowledge of the vascularization of the cerebral surface is

essential. The arterial vascularization of the pial surface of the brain the cortex and the subcortical white matter has been extensively studied by several authors (56, 57, 58, 59, 60, 61, 62, 63, 64).

Sulcal AVMs: Sulcal AVMs are primarily located within a specific sulcus. As was shown by Hutching and Weller (65) with electron micro scopic studies the pial arteries on the surface of gyri and sulci are located in the subpial space, which forms an intrinsic compartment of the subarachnoid space. Therefore, the nidus of sulcal AVMs occupies the subpial space of the sulcus and, depending on its size, it compresses the adjacent gyri to various degrees. Sulcal AVMs may be confined to a sulcus or may extend into the depths of the sulcus, through underlying cortex into the subcortical white matter and even to the ventricular wall. Depending on three subtypes: (1) pure sulcal (2) Sulcal with subcortical extension and (3) sulcal with subcortical and ventricular extension.

The nidus of sulcal AVMs adapts to the geometric space of the sulcus and assumes a pyramidal or conical shape. The base is covered by the dura-arachnoid layer whereas the apex is located in the depth of the sulcus, the subcortical white matter or in the ventricular wall, depending on the in depth extension of the AVM. The supply to sulcal AVMs mainly provided by pial arteries. The pail feeders end directly in the nidus after giving off cortical, medullary and cortico-medullary branches to the adjacent gyrus. Therefore, the main supply of sulcal AVMs is provided by direct type of feeders terminating into the sulcally located nidus and not participating into the supply of normal brain distal to the AVM. This

angioarchitectural feature white matter or near the ventricular wall receive additional supply from short or long medullary and cortico-medullary arteries arising from the pial arterial system as well as from basal perforating arteries. Unlike short and long modularly feeding arteries, the cortico medullary as well as the perforating arteries always participate in the supply of normal brain. In sulcal AVMs, these feeding arteries provide supplementary supply of normal brain. In sulcal AVMs, these feeding arteries provide supplementary supply to the AVM, the dominant supply being provided by the pial feeding arteries. The base of the pyramidally shaped sulcal AVMs is covered by dura-arachnoid layers and not by brain. This explains the frequent participation of additional supply of the more superficial portions of sulcal AVMs from meningeal arteries.

Gyral AVMs: Gyral AVMs are located within a specific gyrus. In contrast to sulcal AVMs, gyral AVMs are covered by cortex and have usually a round shape. Larger gyral AVMs expand the involved gyrus, compress the adjacent sulci and may extend into the subcortical white matter and toward the ventricular wall. The supply of gyral AVMs is provided by dilated cortical, cortico – medullary and medullary branches of the pial arteries of the involved gyrus that continue their course to supply normal brain distal to the AVM. Paraventricular extensions of gyral AVMs receive additional supply from basal perforating arteries. Since gyral AVMs are covered by cortex and therefore, have no direct contact with the dura or arachnoid layer, they lack additional meningeal supply.

Mixed sulcal and gyral AVMs: mixed sulcal and gyral AVMs are usually larger lesions than the pure sulcal or gyral type. They involve adjacent sulci and gyri and extend usually into the subcortical white matter and the ventricular wall. Their pattern of arterial supply combines the features of sulcal and gyral AVMs, i.e. the sulcal components of mixed AVMs are supplied by direct and terminal type pial arteries and in addition frequently by meningeal arteries whereas the gyra; and subcortical components are supplied by cortico medullary or medullary branches of pial arteries as well as in a supplementary fashion from basal perforating arteries.

"Diffuse cortical AVMs" :A particular type of AVM within the group of so-called "Cortical or convexity AVMs is the rare "diffuse" AVM. These lesions are characterized by numerous, only slightly dilated feeding arteries belonging to the pial cortical arterial system a moderately shortened shunt transit time and by numerous cortical draining veins of almost normal caliber. A nidus in the strictest sense cannot be identified angiographically and appears to be intermingled with normal brain tissue. The lesions are typically located cortico-subcortically and may involve small or large areas of a lobe, more than one lobe or even the surface of an entire cerebral hemisphere. Clinically they present commonly with seizures. They most probably correspond to a proliferative type of angiopathy of yet unknown etiology and not to a true AVM (27).

Subcortical AVMs: the term subcortical AVMs has been introduced to characterize a topographically with distinct type of AVM. These lesions have been always grouped together with deep brain AVMs. These AVMs are located in the deeper pats of

the subcortical white matter, which represents the arterial territory of the long medullary and conrtico medullary arteries arising from the pial arterial system and the venous territory of the deep transcerebral veins joining the subependymal venous system. Accordingly, subcortical AVMs receive their dominant supply from long medullary and corticomedullary arteries but may have supplementary supply from deep perforating arteries. The main venous drainage is towards the deep subependymal venous system through transcerebral veins but accessory veins may drain towards the cortical venous system. Subcortical AVMs are extremely rare and were found in 2% of cases in this series.

Deep (Central) brain AVMs: The topographic classification principle described above can also be used for further topographic classification of deep cerebral AVMs. Based on MR imaging and its correlation with angiography, deep brain AVMs are further subdivided into (1) subarachnoid AVMs (corresponding to the sulcal AVMs of the convexity) 2) Parenchymal AVMs(corresponding to the gyral AVMs of the convexity) (3)Plexal of intraventricular AVMs and (4) mixed deep AVMs.

Subarachnoid AVMs are located outside the brain parenchyma within basal cisterns and fissures. Their supply is provided by detailed branches of the proximal subarachnoid segment of perforating and choroidal arteries, which are more readily accessible for micro-cateterization than the distal parenchymal segments of these arteries.

Deep parenchymal AVMs are located within the deep brain structures such as basal ganglia, thalamus, hypothalamus, septum

pellucidum, corpus callosum, limbic system, internal capsule or other deep white matter tracts and nuclei. They are supplied in a dominant fashion by basal perforators. Choroidal and basal circumferential arteries and in a supplementary fashion by transcortical long medullary or corticomedullary branches of the pial arterial system.

Plexal AVMs arise from the vascular of the choroids plexus and are therefore extracerebral intraventricular AVMs. They are primarily and dominantly supplied by choroidal arteries in a direct, terminal fashion. Larger plexal AVMs in contact with the ventricular wall receive additional supply form the subependymal arteries arising from the circle of Willis.

Mixed type deep brains AVMs are usually large lesions and have angio architectural features of the subarachnoid, parenchymal and even plexal types.

Deep cerebral AVMs drain primarily and mainly into the venous collectors of the deep venous system, but may also use transcerebral veins joing the superficial venous system for drainage.

The topographic classification of brain AVMs (55) presented here is summarized in Table 1.

Table – 1 Topographic classification of Brain AVMs

A. Convexity or superficial AVMs (72%)

(So called Cortical AVMs)

1) Sulcal AVMs (28%)

(a) Pure sulcal

(b) Sulcal-csubcortical

(c) Sulcal-subcortical ventricular

(2) Gyral AVMs (12%)

a) Pure gyral

b) Gyral Subcortical

c) Gyral subcortical ventricular

(3) Mixed Sulco gyral AVMs (29%)

a) Sulco – gyral

b) Sulco gyral subcortical

c) Sulco gyral subcortical – ventricular

4) Diffuse AVMs (=Proloferative angiopathy) (3%)

B. Subcortical AVMs (2%)

C. Deep or central (26%)

1) Subarachnoid AVMs (12%)

2) parenchymal (Intrinsic, cisternal) 7%)

3) Plexal (intraventricular) (1%)

4) Mixed (6%)

Angioarchitecture of Brain AVMs

The term "angioarchitecture" refers to the angiographically demonstrate vascular elements composing a brain AVM and includes the feeding arteries, the nidus, the draining veins, any associated vascular anomalies and secondary vascular changes induced by the inherent high-flow of the AVM, subsumized under the term high-flow angiopathy.

Feeding Arteries

Proper angiographic analysis of the arteries supplying an AVM is essential for both understand in the individual construction of a given AVM and for treatment planning. The success of endovascular treatment of brain AVMs with respect to nidus obliteration and patient outcome clearly depends on the ability to super selectively catheterize the distal (prenidal) segments of feeding arteries and on the identification of their relationship with the nidus, their concomitant or absent participation into the supply of normal brain and of any associated high flow angiopathic changes.

Regarding the endovascular treatment of brain AVMs a classification system incorporating hemodynamic, geometric and anatomic criterial for each feeding artery proved useful.

Hemodynamic Types of Feeding Arteries

Hemodynamically, the feeding arteries of an AVM may be classified according to their contribution to nidus supply. Feeding arteries supplying.

Table 2. Classification system of Feeding Arteries of Brain AVMs

A. Direct types of feeding arteries

1) Monoterminal

a) Dominant

b) Supplementary

2) Multiterminal

a) Dominant

b) Supplementary

3) Pseudoterminal

a) Pseudoterminal

b) With flow reversal distally

b.i) Dominant

b.2) Supplementary

c) Induced by wedeged catheter

B) Indirect types of feeding arteries

(1) Transit arteries

a) With single or multiple supplementary feeding branches

b) Rarely, With dominant feeding branches

(2)Retrograde collateral feeding arteries

(a)Leptomeningeal

a.1) usually supplementary

a.2 Dominant following proximal occlusions of dominant feeders

(b) Subependymal

(b.1) Usually supplementary

(b.2) Dominant following proximal occlusions of dominant feeders.

Large portion or vascular compartment of the nidus is called dominant whereas feeding arteries supplying a small portion or compartment are called supplementary. Dominant type feeding arteries usually have a significantly larger diameter and higher flow than supplementary type feeding arteries.

Most AVMs, 82% in this series, were supplied by both dominant and supplementary type of feeders, I various combinations. In the majority of cases with dominant and supplementary supply, multiple dominant and multiple supplementary type of feeding arteries are identified. The next frequently observed combination is that of a single dominant and multiple supplementary feeding arteries. Single or multiple

dominant feeding arteries in combination with a single supplementary artery is rarely observed.

Some AVMs, 13% in this series, were supplied exclusively by multiple supplementary type feeding arteries. This mode of supply is particularly observed with insular, some gyral and brain stem AVMs.

Finally, a few AVMs, 5% in this series, were exclusively supplied by one or few dominant type feeding arteries. This pattern of supply is mainly seen with purely fistulous AVMs, such as pial AVMs and some vein of Galen aneurysmal malformations.

Obviously, the participation of dominant and/or supplementary type of feeding arteries and their relative number in a given AVM influence the achievable degree of obliteration. As a general rule, AVMs supplied either exclusively by dominant feeding arteries or by more dominant than supplementary feeding arteries have a higher chance for complete or subtotal obliteration, than AVMs supplied either exclusively by supplementary or by more supplementary than dominant feeding arteries.

Geometric Types of Feeding Arteries

The term geometric refers to the angiographically demonstrable relationship between a feeding artery with the nidus and the supply of normal brain parenchyma. This relationship is primarily guided by the underlying normal vascular anatomy and arterial disposition of the area involved by the nudus of the AVM. Generally two types of supply of an AVM can be distinguished, i.e. direct and indirect.

Direct Type

Direct Type of feeding arteries end directly in the nidus without continuation towards normal brain distal to it. This type of feeder has been called Terminal artery" by Yasargil (66). The main trunk of a direct type feeding artery may terminate I the nidus either as a single artery, i.e. monoterminal type or may divide into two or even more branches all terminating in the nidus, i.e. multiterminal type. Regardless of their termination pattern, direct type feeding arteries give off branches to normal brain before entering the nidus and therefore participate into the supply of normal brain proximal to the AVM. Because of the high flow within direct type feeders and the concomitant sump effect of the nidus, these normal branches are usually not opacified on selective angiography carried out from the internal carotid or vertebral arteries but may become visible on superselective studies.

On their course towards the nidus direct type feeding arteries may give off other direct or indirect feeding branches to the nidus.

Depending on their contribution to nidus, direct type feeding arteries may be hemodynamically either dominant or supplementary. According to the authors' observation direct, multiterminal type feeding arteries are usually dominant feeders, whereas direct, monoterminal type feeding arteries may be either dominant or supplementary feeders.

Pseudoterminal type

The term Pseudoterminal feeder" is introduced to describe particular types of feeders, which angiographically appear to end directly in the nidus but because of their known anatomic disposition a continuation of their course distal to the nidus must be assumed. The angiographic nonvisualization of their distal portion is caused by the sump effect of the nidus. This particular pattern of AVM supply is encountered with feeders, which give off a terminal branch to the AV but continue their course distal to it to supply normal brain. Due to the sump effect of the nidus the feeding terminal branch dilates up to the size of the parent trunk and flow reversal occurs in the distal portion of the parent trunk. These hemodynamic, flow induced changes finally create the erroneous angiographic impression of a direct, monoterminal type of feeder.

A pseudoterminal feeder appearance may be caused by a microcatheter wedged into the main trunk. The pseudoterminal feeder appearance may also be induced by spam of the main trunk caused by the microcatheter. These iatrogenically induced pseudoterminal feeder appearances have to be distinguished from the naturally occurring pseudoterminal type of feeders.

Embolization performed through a pseudoterminal type of feeder carries a risk for an ischemic complication occurring in the brain parenchyma located immediately distal to the AVM. Because of the changing hemodynamic conditions taking place during the injection of NBCA, migration of the embolic material into the invisible distal segment of the feeding artery may occur.

Obviously, this situation cannot be simulated with amobarbital injection, thus making function testing of pseudoterminal type of feeders highly unreliable.

Indirect Types of feeding Arteries

The term "indirect" supply refers to the main participation of the feeding artery into the supply of normal brain and to its secondary participation into the supply of the nidus of the AVM. Two types of indirect supply of an AVM can be distinguished i.e. transit arteries with feeding branches to the nidus and collateral arteries feeding the nidus in a retrograde fashion.

Transit arteries. The transit artery refers to an arterial trunk coursing in close topographic proximity to the nidus of an AVM, giving off one or several branches to the nidus, but continuing its course distal to the nidus to supply normal brain parenchyma. This arrangement of supply to an AVM has been called "feeder en passage" "indirect feeder", "transit artery with participation", and perforating type" (27,55,66), Feeding branches arising from a transit artery are usually smaller and shorter than direct type of feeding arteries and they usually originate in a right or sharp angle from the parent artery. Depending on their origin from the transit artery in relation to the nidus, they may have either a short, more or less direct course ending in the nidus or, if they arise more toward the nidus, Typically, the transit artery is usually moderately dilated up to the point of origin of the most distal feeding branch and assumed distal to it a normal size. The majority of transit type feeding arteries provide supplementary supply to an AVM. Some

AVM are exclusively supplied by supplementary feeding arteries arising from transit arteries.

The anatomic characteristics of feeding branches arising from transit arteries make superselective catheterization difficult and represent an important limiting factor regarding complete obliteration of brain AVMs. The recent introduction of hydrophilically coated microcatheter and the ability to appropriately pre shape the tip of th microcatheter (Fas Tracker 10 or Magic) made is recently possible to safely catheterize and embolize such feeders in several cases.

Retrograde collateral feeding arteries. This type of feeding arteries is observed with AVMs located proximal to a watershed area between two or three arterial territories. Anatomically, the feeding arteries belong to the distal arterial system of the vascular territory, within which the AVM is located. Their anterograde perfusion distal to the AVM is compromised by the sump effect of the nidus. Reconstitution of flow is provided through compensatory recruitment of leptomeningeal collaterals developing at the watershed area and being supplied by the distal branches of the adjacent vascular territory. Therefore, flow in the artery or arteries distal to the AVM is reversed. This phenomenon has been called "Watershed transfer" by Berenstein and Lajaunias (27) and represents one of the manifestations of the high-flow angiopathy occurring with brain AVMs.

Anatomic Types of Feeding Arteries

The anatomic types of arteries participating into the supply of AVMs depend primarily on the specific location, the topography and the vascular territory of the AVM anatomically, the following types of feeding arteries can be distinguished:

1. Pial Feeding Arteries

2. Dural (Meningeal) feeding Arteries

3. Retrograde Collateral Feeding Arteries

4. Choroidal Feeding Arteries

1. Pial Feeding Arteries

The pial arteries participate in the supply of AVMs either by their extracortical sub pial trunks or by their cortical, medullary or cortico meduallary branches.

a) The pial artery trunks typically supply in a dominant fashion so called pial arterivenous fistulate (AVFs) and distal pial arteriovenous fistulate (AVFs) and distal pial arteries of the periocallosal and posterior cerebral arteries typically supply vein of Galen aneurismal malformations. The trunks of the pial arteries coursing in the subpial space over gyri and within sulci supply in a dominant fashion sulcal type of AVMs and in either a dominant or supplementary fashion mixed sulcal – gyral AVMs.

(b) The cortical branches of the pail arteries may become involved in the supply of purely gyral AVMs

c) The medullary and the corticomedullary branches of the pail arteries may be dominant feeders of gyral, mixed gyral sulcal and subcortical AVMs . They may be supplementary feeders of deep brain AVMs as well as subcortical extension of sulcal, gyral or mixed sulcal gyral AVMs.

2. Dural (Meningeal) feeding Arteries

Three instances of participation of he meaningful arterial system into the supply of AVMs can be recognized.

a) Direct supply of the nidus in a supplementary fashion. Such direct nidus supply is reported to occur in approximately 30% of cases (67). Direct dural supply is observed with AVMs being in contact with h dural arachnoid pial layers, as with sulcal mixed sulcal and gyral but also some deep but subarachnoidally located AVMs with direct contact to the tentorium.

b) Transdural anastomoses with normal pial arteries distal to the AVM. This, most probably represents an angiogenic reaction t brain tissue ischemia distal to the AVM (68).

c) An associated secondarily induced, dural sinus arteriovenous shunt. Such acquired, secondary arteriovenous shunts are nor related to the AVM and should not be confused with a second AVM. They arise from the high sump effect of he dural sinus downstream to the AVM on dural arteries, are asymptomatic, represent incidental angiographic findings, and regress spontaneously following treatment of the AVM.

d) It seems that subarachnoid hemorrhage represents an additional factor that may secondarily increase dural supply to an AVM (69).

3. Retrograde Collateral Feeding Arteries

These types of feeding arteries involve the leptomeningeal and rarely the subependymal systems, develop in association with watershed transfer as described earlier and provide supplementary supply to the nidus of the AVM.

The perforating arteries provide the main supply to the majority of deep brain AVMs. Depending on the size and location of a deep brain AVM, perforating type-feeding arteries may provide supply in a dominant, combined dominant and supplementary or exclusively supplementary fashion. Perforating type arteries and particularly lenticulostriate arteries provide supplementary supply to the subcortical and paraventricular extensions of superficial AVMs.

4. Choroidal Feeding Arteries

The anterior, posterior medial and posterior lateral choroidal arteries participate into the supply of deep brain AVMs, deep brain AVMs with intraventricular extensions or purely intraventricular (plexal) AVMs. Parenchymal and intraventricular branches of the choroidal arteries supply in a dominant or supplementary fashion these AVMs Intraventricular AVMs are supplied in a dominant way by the choroidal arteries.

Arterial High Flow Angiopathy in Bran AVMs

The term high flow angiopathy describes secondary changes of the arterial and venous circulation developing over a period of time and being induced by the av shunt of the AVM. The concept of high flow angiopathy has been proven experimentally (70) and best explained as the response of endothelial cells of the proximal arterial and distal venous system upon stress-triggers generated by the av-shunt and acting upon the normal functioning endothelial cells. The arterial high flow angiopathy includes the following:

1.Arterial Enlargement and Variations

Development of extra-and intracranial dolichoarteries is a relatively frequent observation in brain AVMs. Such vessels and loops, if intracranial, may cause cranial nerve symptoms or focal neurological deficits due to mechanical compression.

Arterial variations are reported to occur more frequently in patients with brain AVMs than in patients evaluated for other pathology or in autopsy series (48) Anatomical variations of he internal carotid and vertebral arteries such as persistence of the trigeminal or hypoglossal arteries, are only moderately increased in patients with brain AVMs. More frequently, anatomical variations are observed in the circle of Willis and include such rare variations as an accessory middle cerebral artery, a duplicated posterior communicating artery, an artery of the corpus callosum etc(27).

2.Arterial Stenosis

Angiographically detectable stenotic changes in the feeding arteries or in the more proximal arteries of the vascular territory, in which the AVM is located are reported to occur in up to 20% of cases. Angiographically, such stenoses may be isolated or multifocal.

3.Associated Aneurysms

The overall incidence of angiographically demonstrable arterial aneurysms associated with brain AVMs has been reported to vary from 2.7% (27,42,71, 72, 73,74,75). The rate of detection of such AVM associated arterial aneurysms depends on complete, high quality selective angiography for visualization of more proximally located aneurysms, and superselective angiography of the feeding arteries for detection of more distally located aneurysms. Unfortunately, this data are derived from angiographic studies suing additional superselective angiography, the incidence of such aneurysms additional superselective angiography, the incidence of such aneurysms has ben reported to be 58% (76), a rate close to that found in pathologic specimens, reported as 55.5% (77).

Angiographically to types of aneurysms can be distinguished in patients with AVMs. (1) those occurring on the arteries related hemodynamically to the supply of the AVM, so called flow related aneurysms and (2) those located on arteries not related hemodynamically to the supply of the AVM that are called remote, dysplastic or not flow related aneurysms .

Flow related aneurysms represent 37% to 69% of all aneurysms associated with brain AVMs (32,78, 73,79). Depending on their location with respect to the nidus of the AVM, flow related

aneurysms are classified with respect to the nidus of the AVM, flow related aneurysms are classified into proximal and distal types. Distal flow related aneurysms may be located on the distal segments of feeding arteries (extranidal type) or may be located within the nidus itself (intranidal type). Each type of flow related aneurysm may occur singly or with additional flow related or not flow related aneurysms.

Types of Associated Aneurysms in Brain AVMs (50-70%)

a) Flow related aneurysms (40-70%)

1) Proximal

(a) Single

(b) Multiple

(2) Distal extranidal

(a) Single

(b) Multiple

(3) Distal Intranidal

(a) Single

(b) Multiple

e) Non Flow related aneurysms (10-20%)

(Remote or dysplastic aneurysms)

(a) Single

(b) Multiple

In a recent report based on superselective angiographic evaluation of brain AVMs, intranidal aneurysms have been found in 42% and multiple flow related aneurysms in 57% of patients (76),. In previous reports based mainly on selective and not on superselective angiographic studies, intranidal aneurysms were reported in 12% to 90% (80,81) and multiple aneurysms in 12% to 84% of patients (76, 81).

The angiographic demonstration and the number of identified flow related aneurysms correlate significantly with a clinical presentation or history of hemorrhage. In patients with AVMs presenting with hemorrhage. 75% to 80% are found to harbour an aneurysm (47,76) This association appears to be even stronger in the presence of multiple aneurysms (76). Therefore, angiographic demonstration of a flow related aneurysm in a patient with a brain AVM worsens the overall prognosis of that AVM (27,39).

The angiographic visualization of a flow related aneurysm significantly influences the therapeutic endovascular approach to the brain AVM. As a general rule, the primary endovascular target is the AVM, since it represents the causative factor of the flow-related aneurysms. Distal intra-and extranidal aneurysms will usually be obliterated together with the obliteration of the brain AVM. More proximally located aneurysms frequently regress spontaneously following successful obliteration of the brain AVM. If on follow up angiograms performed after 6 months no regression of the aneurysm is visible, they surgical clipping must be considered.

Endovascular navigation of the microcatheter or micro guidewire in the presence of an associated aneurysm carries a certain risk like aneurysm perforation and hemorrhage. Such a complication has been occasionally reported (82). The use of biplane road mapping technique is of critical importance, when navigating through an artery carrying an associated aneurysm.

4. Watershed Transfer

The phenomenon of "watershed-transfer" as one of the angiographically demonstrate manifestations of arterial high flow angiopathy, refers to compensatory recruitment of leptomeningeal collaterals and associated pial arteries to reconstitute the arterial territory distal to an AVM (27, 55). This may occur as a congenital disposition to provide supply of brain distal to the nidus or secondarily to provide also supply to the nidus. This secondary watershed transfer may be associated with non-sprouting angiogenesis developing as a response to brain tissue ischemia.

The Nidus o Brain AVMs and Is Angioarchitecture

The term "nidus" was introduced by Doppman (83) based upon his angiographic observations on spinal cord arteriovenous malformations. The term nidus is derived from latin (Plural nidi) and means a nest. Other terms which could also be used include epicenter. Nucleus, or focus of the AVM. The nidus of an AVM represents that area of the entire AVM angioarchitecture, interposed between the readily identifiable distal segments of feeding arteries and emerging proximal segments of draining

veins, where arteriovenous shunting occurs (27,55,66,83). The nidus is the source of all hemodynamic changes observed up and down stream of the AVM, of the secondarily induced high flow angiopathy and of he majority of hemorrhages occurring with AVMs. It represents the actual target of any therapeutic approach because its radical elimination or complete obliteration results in cure of the AVM.

Side of Nidus

The size of the nidus of brain AVMs varies from very small to giant. Various systems of size classification of brain AVMs have been introduced of size determination of a nidus of an AVM lies mainly in its surgical resectability or stereotactic radiotherapy. From the perspective of endovascular treatment, size is not a parameter of major or critical importance as it does not affect directly neither the endovascular accessibility nor the degree of achievable obliteration of the AVM. The primary practical importance of size determination from the endovascular view point lies primarily in the estimation of the length of a procedure, its technical complexity and the number of sessions to be performed.

Size of the nidus represents a major component of most surgical AVM grading systems (84, 85, 86,87,88). Based on the angiographically measured maximum diameter of the nidus.

AVMs are classified according to Yasargil (66) into:

- **Micro AVMs (0.5-1.0cm)**
- **Small (1-2 cm)**
- **Moderate (2-4cm)**
- **Large (4-6cm)**
- **Giant AVMs (more than 6 cm)**

Shape of Nidus

Correlation of MR Imaging with cerebral angiography has shown that the shape of an AVM nidus is primarily determined by the anatomic structure or space within which it is located. AVMs located within a sulcus, cistern or fissure tend to adapt their shape to the overall geometry of that particular space. This explains the shape of sulcal AVM exhibits a pyramidal shape or a fissure AVM a more linear, elongated shape. In AVMs located within the brain parenchyma, the shape of the nidus is determined by the intrinsic parenchymal structure involved and its anatomic boundaries. A subcortical white matter AVM typically exhibits a spherical shape whereas a callosal AVM adapts to the shape of that part of the corpus callosum involved by the nidus. Obviously, larger AVMs involving several structures and spaces exhibit complex or even undefinable shapes.

Vascular Composition of Nidus

Hamby (89) in examining grossly and histopathologically brain containing AVMs,considered the nidus to be a complex vascular system of coiling and intercommunicating vascular composition and intrinsic angioarchitecture of the nidus cannot be evaluated adequately by selective internal carotid or vertebral angiography. Simultaneous visualization of different, hemodynamically independent compartments, over projection of large venous structures of the nidus and flow or pressure related poor or non visualization of various vascular elements limit precise nidus evaluation. This information, however, often may be obtained from superselective angiography of the individual, feeding arteries that enables architectural and compositional mapping of the nidus. Based on the experience obtained from routine application and systematic interpretation of superselective feeder injections performed before or during the course of embolization procedures of brain AVMs, new insights and concepts regarding the nidus of the AVM have been developed and progressively incorporated in the endovascular treatment.

Angiographically, three main patterns of arteriovenous shunting can be distinguished within an AVM. The nidus may consist of atriovenous shunting across a plexiform network of vascular channels (i.e. pure plexiform nidus), large arteriovenous fistulae (i.e. pure fistulous nidus), or of both plexiform and fistulous parts (i.e. mixed plexiform and fistulous nidus). In this series pure plexiform nidi were observed in 36% of cases mixed plexiform and fistulous nidi in 53% of cases and pure fistulous nidi in 11% of cases.

In the plexiform type of nidus one or several arterial feeders end in vascular conglomerate of multiple arteriovenous macro communications from which one or multiple venous channels emerge as draining veins. In the mixed plexiform and fistulous type of nidus additional single or multiple direct arteriovenous communications (fistulae) are observed. In some of the mixed plexiform nidi, the plexiform portion may clearly dominate, in other there may be clear dominance of the fistulous components. In the pure fistulous type of nidus, dilated feeding arteries end directly into dilated venous channels. Such direct fistulous communications may be composed of single or multiple feeding arteries ending directly either in an end to end or in an end to side fashion along a draining vein or veins.

In the majority of cases, the nidus of the AVMs appears angiographically as a single, compact, vascular structure. Rarely, multiple but compact nidi are observed. These multifocal nidi are observed mainly In children and may be distinct with interposed normal brain tissue or be adjacent without interposed normal brain tissue simulating a larger single nidus. Also rarely the nidus may have a diffuse appearance without clearly defined borders, characterized by a nidus like network of vessels and only slightly enlarged draining veins. The nidus-like network does not have a compact and homogenous appearance, as it contains normal brain tissue. There is increasing evidence, that this “diffuse type of AVM” most probably represents a proliferative form angiopathy. It can be localized involving only one or a few gyri or be extensive, involving one or more lobes or even an entire hemisphere. While in the majority of the cases of single compact nidi there was clearly

discernible boundaries.In approximately 23% of cases, the nidus is surrounded by perinidal angiogenesis. The perinidal angiogenesis most probably represents a secondary angiogenic response to chronic hypoperfusion or ischemia induced by the arteriovenous shunt of the nidus. Morphologically it can be localized, moderate or even extensive, simulating a diffuse nidus (pseudo-diffuse nidus). Failure of recognition of perinidal angiogenesis may lead to overestimation of the true size of the nidus of the AVM.

Nidus Composition According to the Type of Arteriovenous Shunts

1. Pure plexiform (36%)
2. Mixed Plexiform and Fistulous (53%)
    a) Plexiform > Fistulous
    b) Plexiform ~ Fistulous
    c) Plexiform < Fistulous
3. Pure fistulous (11%)
    (a) Monofistulous
    (b) Multifistulous

The nidus may be composed of a single or multiple vascular compartments. The concept of vascular compartments was introduced by More et al. in 1980(90) to describe the vascular composition of glomus tumors as seen on selective angiography. Applied to the nidus of the AVM, a compartment is a purely angiographic term, defined as an intranidal vascular unit,

characterized by one or more feeding arteries, arteriovenous shunting and a draining vein (i.e. compartmental vein). In mono compartmental nidi, this compartmental vein is the draining vein of the AVM (i.e. nidal draining vein). It can exit the nidus as a solitary draining vein or can divide early into two or more veins, sometimes simulating the presence of multiple draining veins emerging from the single compartment. In multi compartmental AVM's, compartmental veins may converge towards one or more nidal draining veins or exit the nidus individually.

Based on superselective angiographic observations, it can be concluded that the compartments of the AVM nidus are nor rigid, well-defined anatomic vascular units, but rather hemodynamic units may even intercommunicate. The feature of communication between compartments becomes evident following occlusion of a compartmental feeder without obliteration of its compartment. This compartment may still be perfused through communication with a neighboring compartment or through collateral supply. The feature of intracompartmental communication may be used to reach otherwise inaccessible compartments of nidi during embolization of AVMs.

Intranidal Vascular Cavities

Superselective angiography may demonstrate intranidal vascular cavities (27). Such vascular cavities or pouches represent focal, aneurysmal dilatations originating from intranidal vascular channels. They may be small and hardly discernible, medium – sized or even rarely large. Intranidal vascular cavities may be located anywhere within the nidus, involving the arterial inlet, the

central or peripheral part of the nidus or the venous outlet. They may be single or multiple and may be categorized as (1) Intranidal arterial aneurysms. (2) arterial pseudoaneurysms, (3) venous pseudoaneurysms and (4) Intranidal venous ectasias. Intranidal aneurysms represent the most common type of an intranidal vascular cavity and are reported to occur in 12%-42% of cases (55,76). On superselective angiograms, arterial intranidal aneurysms are visualized in the early phase and are located on the nidal ramifications of the feeding arteries. They may be single or more frequently multiple and are usually small (less than 4 mm in size). Coiled intranidal arterial segments and closed intranidal arterial aneurysm. Therefore, multiple projections may be needed to reliably differentiate a true intranidal aneurysm from an arterial loop.

intranidal arterial aneurysms represent a week angioarchitectural element of AVMs and an angioarchitectural risk factor for AVM rupture. Recent studies have confirmed a statistically significant correlation between prior hemorrhage and the frequency of intranidal aneurysms in patients with AVMs. The association of intranidal aneurysms and hemorrhage has been found in 41% to 100% of patients (47,76,80). Two mechanisms are probably implicated in rupture of intranidal aneurysms. First, intranidal aneurysms are exposed to nearly the same arterial pressures as the arterial components of the AVM. Because intranidal aneurysms have a thinner and weaker wall than the other anterior elements of the AVM they represent the most likely site of rupture following intraarterial pressure rise, particularly if it occurs suddenly. Following embolization of a nidus compartment,

a sudden increase in intraarterial pressure involving the non-occluded vessels supplying the remaining AVM may occur predisposing unprotected intranidal aneurysms to rupture. Therefore, embolization of AVMs should be first performed through feeding arteries either carrying flow-related aneurysms or supplying compartments containing intranidal arterial aneurysms (76,80). Second, venous hypertension, as it occurs with stenosis or obstruction of the venous drainage, if severe enough may cause retrograde propagation of increased pressure towards the arterial side of the nidus and lead to rupture of intranidal arterial aneurysms (27,80).

Intranidal pseudoaneurysms develop following nidus rupture and result from the unclotted portion of the hematoma that still communicates with the lumen of the reptured vessel (91). Intranidal pseudoaneurysms are usually irregularly shaped, vascular cavities lacking true walls. They are located within or at the margin of a recent hematoma and originate from the arterial or venous part of a recent hematoma and originate from the arterial or venous part of the nidus, depending on the site of the ruptured vessel. Pseudoaneurysms represent an important angioarchitectural characteristic of an AVM because their angiographic demonstration indicates the exact site of AVM rupture and bleeding. They must be differentiated from intranidal arterial aneurysms. Angiographically, pseudoaneurysms appear as irregular, oval or round aneurysmal cavities frequently associated with some stagnation of contrast material. Arterial pseudoaneurysms usually fill before any visualization of draining veins and can be related to an arterial pedicle, whereas venous

pseudoaneurysms usually fill simultaneously with the draining veins. Because intranidal pseudoaneurysms represent an acquired angioarchitectural feature encountered only in the posthemorrhagic period, the most pathognomonic diagnostic sign is their absence on previous angiograms or MR – images, if available for comparison.

The incidence of angiographically detected intranidal pseudoaneurysms in patients presenting clinically with a recent AVM rupture and bleeding has been origin (60%) with the remaining involving the venous side the nidus (91).

Because intranidal pseudoaneurysms have only recently been recognized as a specific entry of ruptured AVMs and because they are usually not studied by repeated angiography, their natural history is not yet known. Based on data from small series of patients, three outcomes for pseudoaneurysms have been already identified. Intranidal pseudoaneurysms may undergo (1) spontaneous thrombosis, observed in most cases, (2) incorporation into a draining vein producing an intranidal eccentric venous ectasia not necessarily associated with distal stenosis or obstruction of the draining vein, observed in approximately 10% of cases (91) Evidence of an enlarging pseudoaneurysm on repeat angiography in a patient with an acutely ruptured AVM represents a rare indication for emergency microsurgical or endovascular treatment of an AVM. Embolization of an AVM in the presence of an intranidal pseudoaneurysm should be performed under special precautions to avoid preprocedural rupture of the fragile pseudoaneurysm.

Intranidal venous ectasias or varices represent a separate entity of intranidal vascular pouches. In most cases, such venous ectasia are observed extranidally on a draining vein. However, venous ectasias may occur intranidally on the emerging segment of the draining vein. Their pathogenesis is identical to the extranidal venous ectasias in that they are produced following a distal stenosis or obstruction of a draining vein and indicate venous hypertension within the nidus.

Two types of intranidal venous ectasias can be distinguished by superselective angiography, namely those with an **open exit** occur more frequently than the **closed exit** variant and are almost constantly located proximal to a mechanical obstruction such as a kinking or a stenosis of the draining vein. Venous ectasias with a closed exit are caused by thrombosis of a draining vein and occur in association with an acute hemorrhage of a draining vein and occur in association with an acute hemorrhage. They therefore indicate clinically relevant intranidal hypertension and may also represent one of the few indications for emergency microsurgical or endovascular treatment (27).

Draining Veins

The anatomic type of vein draining an AVM can usually be predicted from the location of the lesion. Superficial AVMs drain through cortical veins into the adjacent dural sinus. Superficial AVM with subcortical or ventricular extensions usually drain into both superficial cortical and deep subependymal system. In up to 30% of cases, however, an unexpected venous drainage pattern is observed angiographically. A deep brain AVM or the deep

paraventricular extension of a superficial AVM may not drain ventriculofugaly into the subependymal venous system as would be expected from nidus location, but unexpectedly through a dilated trans cerebral vein in a ventriculofugal direction toward the cortical venous system. Inversely, a superficial AVM without subcortical extension may not addition to exclusively through one or several transcerebral veins in a ventriculopetal direction toward the cortical venous system. Inversely, a superficial AVM without subcortical extension may not drain exclusively through cortical veins, but empty unexpectedly veins in a ventriculopetal direction towards the subependymal venous system. Angiographic evidence of unexpected venous drainage most probably represents a secondary event following thrombosis of the anatomically expected draining vein and, therefore, corresponds to the development of venous collateral circulation (27). Selective angiographic studies, however, have several limitations regarding the precise analysis of the draining veins of AVMs. Small, so-called accessory draining veins may be missed on selective angiography, but become visible upon superselective angiography of individual feeders. Furthermore, because of superimposition of various venous channels, both the actual number and the relationship of the draining veins to the nidus may be obscured or even impossible to depict. On the other hand, selective angiography is essential in providing information on the venous drainage of normal brain parenchyma and its relation to the draining veins of the AVM. For precise evaluation of the nidal draining veins, superselective angiography of the individual feeding arteries is mandatory. Based on such studies, several

observations on the architecture and arrangement of nidal draining veins have been made.

The draining veins of AVMs may be single or multiple. A single short draining vein may sometimes divide early into several channels simulating the presence of multiple veins.

Hemodynamically, the draining veins may be classified into (1) **main** and (2) **accessory** types. Main draining veins are characterized angiographically by larger caliber and usually higher flow than accessory draining veins. A high variability in the venous drainage of individual intranidal compartments have been observed on superselective angiographic studies. Typically, an individual compartment has a single draining vein. This compartmental vein may exit the nidus as a single isolated vein corresponding to either a Main or an accessory nidal draining vein. Larger compartments drain usually into main veins, whereas small compartments usually drain into accessory veins. However separate compartmental veins may converge intranidally into a single main nidal draining vein. Larger AVMs may show both draining patterns with isolated compartmental veins and converging compartmental veins. The incidence of these draining patterns of AVMs has not been yet elaborated.

Associated Venous Findings and Venous High Flow Angiopathy

Important features regarding the venous drainage of an AVM are the presence of anatomic variations, the development of high flow angiopathic changes resulting in stenoses or ectasias, the

development of collateral venous circulation and competition between the venous drainage of the AVM and the normal brain. Angiographic recognition of these associated venous findings is important because they help to understand the clinical symptomatology as well as the natural history of a particular AVM and contribute to decision making regarding treatment and its risks (27, 66).

Anatomic variations of the venous system occur in 30% - 32% of cases of cerebral AVMs (47,66,92). They are of developmental disturbance caused by the venous hypertension associated with the arteriovenous shunt. They include anatomic variations of the cerebral veins, the dural sinuses (such as persistence of the occipital or falcine sinus) as well as persistence of embryonic veins (27,93). Presence of venous obstacles or obstructions represent an important angiographic finding, because they indicate increased venous pressure proximally. Such a venous obstacle or obstruction may have several causes that may be identified angiographically and include extramural, mural and intraluminal causes. Venous obstacles of extramural origin include a mechanical compression of the dilated draining vein by rigid extracerebral structures such as the tentorial edge. The sphenoid ridge or the falco tentorial junction and b) compression by pial arteries bridging over draining cortical veins. Venous obstacles of mural origin are focal stenoses of the wall of the draining vein occurring as an endothelial reaction and wall remodeling because of high flow.

Venous obstructions of intraluminal origin are caused by spontaneous thrombosis of the venous lumen. Irrespective of the cause, such obstruction and obstacles increase the venous pressure proximally and induce collateral circulation to bypass the venous hyperpressure. Therefore, venous collateral circulation represents an acquired, secondary response of the venous system aimed to distribute the increased pressure of the veins draining the AVM into normal veins. Depending on the location of the AVM with respect to the venous system, such collateral rerouting may occur by way of ipsilateral, contralateral or transcerebral veins (27). Sufficient venous collateralization decreases the risk of rupture and prevents the development of neurologic symptoms. Angiographically, it is recognized by the redistribution of venous flow in the ipsilateral, contralateral, or transcerebral veins without evidence of venous congestion of the brain and by the absence of significant venous ectasia or varix formation proximal to the obstacle (27, 94,94).

Failure of the venous collateral circulation to compensate for the venous hyper pressure results in the formation of focal venous ectasia or varix proximal to the obstacle and in the development of acute or progressive, transient or permanent clinical symptoms. The clinical manifestations of an insufficient or failing venous collateral circulation include **(1)** symptoms of mass effect or cranial nerve palsies caused by direct, mechanical compression of the brain or cranial nerves by venous varix, **(2)** seizures or progressive neurologic deficits caused by venous congestion of normal brain parenchyma (3) hemorrhage because of AVM rupture: AVM rupture caused by venous hypertension may occur

at the venous side of the nidus, at the veno nidal junction or from the rupture of intranidal or distal extranidal arterial aneurysms due to retrograde propagation of the increased pressure. Therefore such venous ectasias or varies represent weak elements of AVM angioarchitecture and their angiographic demonstration indicates an increased risk for hemorrhage.

Venous thrombosis may cause congestion of pial veins that may lead to a focal cerebral ischemia or infarction. Over time, focal ischemia will cause focal cortical atrophy that may be associated clinically with seizures or a neurologic deficit. Venous infarction will create a porencephalic cavity that may also be associated with a neurologic deficit.

An extreme high flow condition in a dural sinus or major cerebral vein (such as the Galenic Vein) down- stream from an AVM may exert a sump effect on a remote vein or on adjacent dural arteries, which in turn may induce a secondary arteriovenous shunt. Such acquired, secondary arteriovenous shunts are not related to the AVM and should not be confused with a second AVM. They are asymptomatic and have been shown to regress spontaneously following treatment of the AVM and normalization of flow in the involved dural sinus or major draining vein (27,55).

Hydro venous Disorders

In infants and children with AVMs dural sinus high flow may impair cerebrospinal fluid reabsorption leading to macrocrania, hydrocephalus or tonsillar herniation. If long lasting such a

hydrovenous disorder leads to subependymal atrophy with exvacuo hydrocephalus, subcortical atrophy, and white matter calcifications or syringomyelia. Clinically these patients develop mental retardation, complex neurologic deficits or epilepsy . The hydrovenous disorders induced by brain AVMs in the pediatric population have been extensively studied by Lasjaunias (27).

Indications for Endovascular Treatment

The primary goal of treatment of a brain AVM is to prevent new or recurrent hemorrhage. Other goals may include improving or stabilizing neurological deficits, to treat intractable epilepsy or to reduce the severity and frequency of chronic headaches. An incidentally detected brain AVM does not automatically represent an indication for treatment. In general the indication for active versus conservative treatment is derived from the estimated natural history of a given AVM. The anticipated risks of the various treatment modalities the patients neurologic and general condition, the patients past history, the age and the patients attitude toward conservative or active treatment (36,96, 97, 98). In the attempt to improve selection criteria for active treatment various decision analysis schemes (99, 100) Classification systems (85) and grading systems (86,84,87,88,101) have been introduced and applied. These systems use various criteria, such as the number of feeding arteries, the size, the age, the sex, neurological deficits, the location, eloquence of adjacent brain pattern of venous drainage and they range in complexity from simple and easy to apply to complex and difficult to use. The scope of these grading systems is to predict the risk of neurological morbidity resulting

from treatment especially surgery. They therefore are based mainly on criteria directly related to anticipated surgical difficulties, like deep venous drainage large size or close proximity to so called eloquent brain tissue (88).

In a retrospective comparative study of the grading systems introduced to predict postoperative morbidity and mortality, the Spetzler and Martin system showed the highest overall correlation with surgical difficulty and postoperative neurological outcome (100). This system is currently in wide use for prediction of surgical risk and for operative decision making in patients with AVMS. This system is based on only three graded variables derived from cerebral angiography CT or MRI

Size of the nidus	**Scores**
small <3 cm	1
medium 3-6 cm	2
large > 6cm	3

eloquence of adjacent brain	
Non eloquent	0
Eloquent	1

Pattern of venous drainage

Superficial	0
Deep	1

The sum of the three scores provides the AVM grade, which may range between Grade I and V. In addition large AVMs that involve extensive areas of eloquent cortex or smaller AVMs located within the brain stem or hypothalamus are classified as Grade VI and are regarded as inoperable (103). Regarding the endovascular treatment of brain AVMs, these grading systems fail to take into consideration the specific angio architecture of the AVM including high flow angiopathic changes, the intrinsic angioarchitecture of the nidus and the relationship between topography of the lesion and its vascular supply and drainage. In contrast to surgical morbidity morbidity of the ,endovascular approach is directly related to the capacity of reaching safely and super selectively the nidus and remaining strictly within it. Therefore the specific indications for endovascular treatment and the particular goals of endovascular treatment in a given case, are derived from comprehensive MR and angiographic evaluation of the lesion including precise topographic evaluation, identification of the type of feeding arteries, depiction of the angioarchitecture of the nidus and its particular vascular composition, delineation of the venous drainage, detection of any associated lesions (flow related aneurysms, venous stenoses, venous varix, venous hypertension , pseudoaneurysms) and evaluation of the status of collateral circulation and of the circulation of the remaining brain.

For these reasons, the Spetzler Martin grading system does not correlate with the difficulty of treating patients with AVMs by the endovascular approach and cannot be applied to predict the risk of neurological impairment resulting from endovascular treatment.

In the majority of cases, the specific goal of endovascular treatment in a particular patient with brain AVM can be defined from the above mentioned parameters. These goals include the curative application of embolization aiming at complete obliteration, the preoperative or pre stereotactic radiotherapy application of embolization aiming at size reduction and hemodynamic improvement and the palliative application of embolization aiming at partial and targeted elimination of angio architecturally weak elements or at elimination of vascular elements responsible for venous hypertension or at elimination of vascular elements responsible for venous hypertension or tissue hypoperfusion . In some cases, however a predefined goal of endovascular treatment has to be changed respectively adapted to the observations made during the initial endovascular procedure. For example, during preoperative embolization it may become clear to the interventional neuroradiologist, that complete obliteration of the AVM is possible without an increase of the predetermined risks of the combined embolization and surgical treatment. During embolization of an AVM with the goal of complete obliteration it may become evident, that subtotal obliteration followed pre stereotactic radiotherapy is preferable, Ideally a multidisciplinary approach, including a neurosurgeon, an interventional neuroradiologist a radiotherapist and a neuro

anesthesiologist should be adopted for therapeutic decision making (34, 104).

In the majority of cases endovascular treatment of brain AVMs is carried out electively under optimal clinical conditions and after adequate neuroradiologic evaluation .In contrast to spontaneous intracerebral hematomas, patients presenting with even large acute hematomas following rupture of an AVM usually recover rapidly and spontaneously or in response of an AVM usually recover rapidly and spontaneously or in response to steroid and diuretic therapy without the need of early decompressive surgery (36) Emergency surgery is obviously required in patients with acute hematomas with rapid clinical deterioration. If possible emergency operation should aim at removing the hematoma and not eliminating the AVM. CT is the imaging modality of choice to detect acute intracranial hemorrhage from a ruptured cerebral AVM. Most commonly this occurs in the brain parenchyma adjacent to the AVM, with subarachnoid or intraventricular hemorrhage is most probably caused by rupture of an associated proximal aneurysm and not be AVM rupture. In the acute phase of parenchymal hemorrhage the hematoma compressed the nidus of the AVM. Cerebral angiography performed early may therefore not demonstrate the entire AVM giving the incorrect impression of reduced flow through the AVM or a small AVM as a result of self and nidus compression. Depending on the relative sized of the hematoma and the AVM, the nidus may be fully compressed by the hematoma in the early acute phase of hemorrhage and be undetectable angiographically. Exceptionally nidus compression by an acute hematoma may lead to thrombosis and spontaneous

obliteration of the AVM. Unless there is an indication for emergency, surgical evacuation of the hematoma in the acute phase, cerebral angiography should be repeated after hematoma resorption to demonstrate the true size, extension and flow conditions of the AVM (34, 105). Emergency embolization in a patient being investigated angiographically in the acute phase of intracerebral hemorrhage is indicated if a pseudoaneurysm is being identified Pseudoaneurysms carry a relatively high risk of early rapture of the AVM (91). Otherwise, the incidence of early rebleeding of an AVM is considered to be far less that for aneurysms, so that it is preferable to delay definitive treatment until the patient has fully recovered and the lesion has been comprehensively investigated (36).

The incidence of angiographically visible vasospasm following subarachnoid hemorrhage from a ruptured AVM is very low and symptomatic vasospasm because of ischemia is extremely rare in patients with ruptured AVM (106).

Technical Aspects

Patient preparation

All patients scheduled for endovascular treatment are placed on corticosteroids, beginning the day before the procedure with a dose of 4x4 mg decadron per day. The dose is tapered off after the 3rd day following the embolization.

Antiepileptic medication is used in patients who have had previous seizures and in those patients already on antiepileptic therapy or with a recent intracranial haemorrhagia. The endovascular treatment neither induces seizures nor increases their frequency.

General Versus Local Anesthesia

In the initial period of the application of embolization techniques the great majority of patients with brain AVMs were treated under local anesthesia, sedation and continuous monitoring of ECG blood pressure and oxygen saturation. General anesthesia was reserved for children unto 16 years uncooperative adult patients and in a few patients with intracerebral hematoma .It was thought, tat repeated neurological examination during the procedure with or without functional testing was essential in detecting beginning deficits and discontinuing the procedure before the establishment of major deficits.

With increasing experience in the application of endovascular techniques and with an improved understanding of the angioarchitecture and topography of brain AVMs it became apparent that local anesthesia was an important limiting factor regarding the degree of AVM obliteration achievable in one session. Since 1993 the majority of patients with a brain AVM underwent embolization under general anesthesia.

The advantages of general over local anesthesia in the embolization of brain AVMs include:

(1)Better working conditions for the interventional neuroradiologist. There is no distraction of the interventional neuroradiologist by the patient and there is not need to communicate verbally with the patient during the procedure. If it is performed under general anesthesia.

Selection of microcatheter for catheterization individual feeder, the fluoroscopically guided superselective catheterization itself, performance of superselective angiograms and their immediate analysis, decisions regarding choice, amount and rate of injection of embolic material, the fluoroscopically guided deposition of the embolic agent as well as the immediate fluoroscopic or angiographic recognition of any technical complication occurring during catheterization or embolization require the full concentration and undistracted attention of the interventional neuroradiologist in a quite environment. These conditions are better achieved with general than local anesthesia.

(3) General anesthesia is also preferable for the patient, because eliminates any patient anxiety and its associated cardiovascular and neuro vegetative reactions.

(3) In case of a major complication occurring during the procedure, such as hemorrhage or occlusion of a normal cerebral artery, immediate endovascular treatment, or patient transfer to the CT unit and performance of an emergency CT with optimal quality or if indicated immediate transfer of the patient to the operation room for hematoma evacuation and / or ventricular drainage having the patient already under general anesthesia obviates the need for intubation under difficult conditions saves important time and may be life saving.

Because of these significant advantages of general over local anesthesia both the total number of sessions required to complete endovascular treatment of larger AVMs can be reduced and the rate of complete obliteration of smaller and medium side AVMs achievable in one or two sessions can be increased.

Obviously, this benefit is achieved by prolonging the duration of individual sessions, because general anesthesia eliminates patient discomfort, which usually increased in relation to the length of the procedure.

Premedication is performed approximately 45 to 60 minutes before induction of general anesthesia with a hypnotic preferably benzodiazepine (Midazolam). General anesthesia is induced with Thiopental. For muscle relaxation Pancuronium or a short acting

non-depolarizing relaxant is used. For analgesia and maintaining narcosis, fentanyl is administered Ventilation is performed with a mixture of nitrous oxide and oxygen. The use of nitrous oxide is contraindicated in patients with increased intracranial pressure.

In adults and cooperative patients we prefer neurolept analgesia instead of general anesthesia.

During the procedure there is routing continuous monitoring of systemic blood pressures, oxygen saturation and ECG.

If induced hypertension is required for better penetration of the nidus with an acrylic embolic agent or for occlusion of larger, high flow fistulas, this is usually achieved with nitroprusside drip infusion.

Neuroangiography Suite and Equipment

The neurangiographic investigation and embolization of brain AVMs is carried out in the neuro endovascular therapy suite on biplane digital subtraction angiography (DSA) equipment with improved resolution capabilities, live high quality subtraction road mapping magnification and high frame rate capabilities.

Advantages of the biplane DSA system include improved contrast sensitivity, reduced volume of contrast agent used throughout the procedure reduction of radiation exposure and reduced study time.

Non ionic iodinated contrast agents are used exclusively for the endovascular investigation and treatment of brain AVMs because of their decreased neurotoxicity compared with ionic agents. A 30% iodine concentration is usually used for DSA of brain AVMs. On average a volume of 8 ml is injected for selective internal carotid artery, 6 ml for selective vertebral artery, 4 ml for selective external carotid artery and 1to 2 ml for super selective AVM feeder angiography. Furthermore, this newer generation DSA system provides the ability to rapidly acquire and continually interpret DSA images while endovascular treatment is in progress. Additional post processing capabilities of this neurangiographic equipment such as delayed masking techniques, pixel shift, zooming techniques , etc. further enhance overall AVM and nidus visualization.

Neurangiographic Investigation

Usually the procedure is started with a complete neurangiographic investigation guided by the location and extension of the AVM as derived from MRI and MRA with the goal to reveal the complete angioarchitecture of the AVM and the circulation of the brain.

Since the initial angiographic work up is followed by superselective catheterization and embolization of the AVM nidus and angiographic catheter which will be subsequently used as a guiding catheter is selected for the neurangiographic investigation. At our institute we routinely use 6 French polyethylene catheter (Vista brite tip-Cordis) for that purpose.

This initial comprehensive neurangiographic investigation of a brain AVM should provide the following information (107):

(1) The arterial territory or territories involved into the supply of the AVM.

(2) The individual feeding arteries.

(3) The arterial supply of normal brain proximal around and distal to the AVM.

(4) Arterial highflow angiographic changes: (a) arterial enlargement dolicho- ecstatic arteries, loops and kinking: extra and /or ntra cranial; (b) isolated or multiple arterial stenosis moya moya like changes: proximal and / or distal flow related aneurysms.

(5) Gross assessment of the AVM nidus: (a) size and shape (b) compact or diffuse: (c) plexiform and / or fistulous composition: (d) intraidal vascular cavities: (e) flow conditions.

(6)Venous territory or territories involved in the drainage of the AVM

(7)Individual draining veins

(8)Venous high flow angiopathic changes (a) Venous stenosis with or without venous collateral circulation (b) venous varices: (c) venous occlusion: (d) dural sinus high flow with secondary cortical arteriovenous shut.

(9)Venous anatomic variations; persisting embryonic veins

(10) Venous drainage of the normal brain.

Despite the limitations inherent to selective neurangiographic investigation of brain AVMs regarding the reliable depiction of intrinsic angioarchitectural details of the AVM as opposed to superselective neuroangiography, this information is essential in planning the superselective endovascular exploration and embolization of the AVM.

Selection of Cervical Artery or arteries for Intracranial Navigation

Based on the information obtained from the preliminary neuro angiographic investigation on the anatomy and geometry of the involved and not involved extra and intra cranial arteries in relation to the vascular territory or territories of the AVM and on the type number and contribution of the feeding arteries, the cervical artery or arteries through which the endovascular microcatheter approach to the nidus of the AVM will be performed is selected. Selection of the best-suited cervical artery and in case of involvement of more than one vascular territory into the supply of the AVM, determination of the sequence of the multiple approaches through the most appropriate cervical arteries is essential for the successful micro catheterization of the AVM nidus.

AVMs located fully or partially in the territory of one anterior cerebral artery may be approached through either the ipsilateral or contralateral internal carotid artery, depending on the angulation of the carotid siphon, on the angulation of the A1 segment in relation to the distal internal carotid artery and on the potency of anterior communicating artery.

In AVMs located in the vascular territory of both anterior cerebral arteries such as is frequently the case with callosal AVMs, the approach to both anterior cerebral arteries may be performed through one internal carotid artery or through each ipsilateral internal carotid artery or even through each contralateral internal carotid artery, depending on the siphon angulation, A1 segment angulation in relation to the distal internal carotid artery and on the presence or absence of loops or kin kings in the internal carotid arteries.

In AVMs located fully or in part in the middle cerebral artery territory. The approach is usually through the ipsilateral internal carotid artery. Rarely in cases with extreme looping or kinking of the ipsilateral extracranial internal carotid artery or in cases with associated high grade stenoses or occlusions of the ipsilateral internal carotid artery and approach through the contralateral internal carotid artery and through anterior communicating artery has to be selected. In these cases a special, long microcatheter version has to be used.

In AVMs located in the posterior cerebral artery territory (perforator, choroidal or pial posterior cerebral artery branches),

the approach is usually through the dominant vertebral artery. In several cases, however, with a dilated Pcom artery, and approach through the ipsilateral internal carotid artery proved very useful.

In AVMs located in the vascular territory of the anterior choroidal artery the approach is through the ipsilateral internal carotid artery.

In AVMs located in the vascular territory of the anterior choroidal artery the approach is through the ipsilateral internal carotid artery.

In AVMs located in the vascular territory of the posterior communicated artery perforators the approach is through he ipsilateral internal carotid artery or through the dominant vertebral artery depending on the angle and orientation of the proximal segments of these perforators in relation to the posterior communicating artery on flow direction.

In AVMs supplied by proximal and / or distal M1 perforators the approach is usually through the ipsilateral internal carotid artery.

In AVMs supplied by A1 perforators and particularly by Heubners artery the optimal approach in the majority of cases proved to be through the contralateral internal carotid artery because of the recurrent orientation of the origin of these arteries with respect to the A1- segment.

AVMs supplied by anterior communicating artery perforators are approached through one of the internal carotid arteries depending on the most suitable geometry.

Posterior fossa AVMs supplied by posterior inferior (PICA) anterior inferior (AICA) or superiors cerebellar arteries (SCA) uni or bilaterally are usually approached through the dominant vertebral artery. However PICA catheterization is usually performed through the ipsilateral vertebral artery. In rare instances, with extreme tortuosity of the vertebral arteries a retrograde approach through one of the internal carotid artery's and through posterior communicating artery has to be performed.

In AVMs supplied by feeding arteries arising from two or three vascular geometry two guiding catheters inserted through a bifemoral approach may be placed in the appropriate cervical arteries for simultaneous microcatherization of two feeding arteries with two microcatheters.

Endovascular Micro Instrumentation for Catheterization of Brain AVMs

The current generation of microcatheter systems available and applied in the super selective endovascular exploration and embolization of brain AVMs consists exclusively of the variable stiffness microcatheters. The shaft of these microcatheters consists of a more rigid proximal segment, a flexible mid segment and a soft distal part including the microcatheter tip. Usually a radiopaque marker is positioned at the distal tip for fluoroscopic

visualization. This tapered design of the microcatheter diameter is a key design component of all currently available microcatheter systems used for distal superselective cerebral catheterizations. The stiffness of the proximal part of the microcatheter improves the ability to push and advance the catheter while the softer distal segment prevents trauma to the vessel wall, catheter induced arterial vasospasm or vessel wall perforation.

Two types of variable stiffness microcatheters are available and used in brain AVM embolization. The first type is used exclusively in combination with steerable microguidewires allowing for distal catheterization by torque control (20) Several type of variable stiffness microguidewires are available to assist in catheter advancement and guiding direction into the desired arteries or veins. The second type are flow guided microcatheters which have a slightly dilated radiopaque tip and a softer distal segment permitting distal catheterization of tortuous vessels (108).

Superselective Exploration of Brain AVMs

Despite its usefulness in demonstrating the general vascular features of an AVM, selective angiography of the internal carotid, external carotid, and vertebral arteries has significant limitations. Overprotection of early draining veins on arterial feeders may obscure visualization of small feeding arteries as well as small flow related aneurysms located in proximity to the nidus. The nidus itself frequently obscures the origin of the draining veins and particularly intranidal division of a single draining vein into multiple veins. Frequently the intranidal angioarchitecture can not be

reliable recognized and important angioarchitectural features such as intranidal aneurysms, pseudoaneurysms or smaller direct arteriovenous fistulae may remain undetected on selective angiographic studies. In addition because of different hemodynamic conditions in different parts (compartments) of an AVM some small accessory draining veins may not be visualized at all. Most of the currently used classification and grading systems for brain AVMs as well as the available statistical data on the angioarchitecture and vascular characteristics of brain AVMs are mainly based on selective angiographic studies, Obviously this data will differ from similar data derived from superselective angiographic studies.

Selective angiographic studies are used for planning the superselective endovascular microcatheterization of the nidus of a given AVM. Usually several feeding arteries are superselectively catheterized and embolized in one session. Appropriate selection of feeding arteries for superselective microcatheterization and the microcatheterization sequence of several feeding arteries in a single session are important factors for successful embolization of a given brain AVM.

Superselective angiographic exploration of an AVM should provide the following information:

1. Distal prenidal, segments of feeding arteries (a) anatomic type: (b) geometric features (c) hemodynamic characteristics

2. Arterio nidal junction

3. Assessment of nidus:

(a) compartmental composition;(b) Intracompartmental communications (c) intranidal vascular composition (plexiform and / or fistulous); (d) intranidal vascular cavities or ectasias (aneurysms, pseudoaneurysms venous varices); (e) arteriovenous transit time.

4. Veno nidal junction

5. Proximal segments of individual draining veins

This information influences the final decision proceeding with embolization the selection of the appropriate embolic material, its amount, mixture and mode of injection.

The sequence of superselective microcatheterization and subsequent embolization of individual feeding arteries in the usual multipedicular AVM is mainly determined by the hemodynamic and geometric types of feeding arteries in relation to the major arterial territories involved into the supply of the AVM. If several feeding arteries in a single arterial territory supply the AVM, then catheterization and embolization starts with the dominant and direct type of feeder(s) and proceeds with the supplementary and direct feeder(s) and finally with the indirect type of feeders if present and technically accessible. If more than one arterial territories participate in the supply of the AVM the territory

providing the dominant supply is being catheterized first. If dominant feeding arteries arise from two or three arterial territories, as is the case with larger AVMs located directly within a watershed area, catheterization usually starts with that feeding artery supplying the larger compartment of the nidus. Obviously this approach has to be adapted to the predefined goals of the embolization procedure, ie complete versus partial obliteration or embolization or embolization targeted to a particular component or weak angioarchitectural element.

In AVMs receiving supply from more than one arterial territories simultaneous catheterization of two feeding arteries arising from two territories through a bifemoral approach proved very useful in terms of nidus evaluation and embolization. Simultaneous and alternate super selective angiograms obtained during injection of contrast material through both microcatheters allows a better evaluation of two adjacent nidus compartments and their individual draining veins and permits immediate continuation with acrylic embolization of the second compartment following the glue deposition in the first compartment.

Embolic Materials used for embolization of Brain AVMs

A wide variety of embolic materials, including particulate and liquid agents, have been used for the embolization of brain AVMs. Although the ideal material for endovascular therapy of brain. AVMs has yet to be discovered, cyanoacrylates represent currently the primary material used in the endovascular management of brain AVMs.

Cyanoacrylates

Cyanoacrylates have been used for embolization of brain AVMs for more than 20 years. The exposure of the cyanoacrylate monomer solution to an ionic environment, such as blood, initiates the process of polymerization by adding a negative ion to open the carbon double bond. Among the cyanoacrylates, isobutyl cyanoacrylate (IBCA) and n butyl cyanoacrylate (NBCA) have been used for embolization of brain AVMs. Presently NBCA (Histoacryl or Avacryl), Tri-point Medical Braun Melsungen, Germany) is the preferred embolic agent as it has significant advantages over IBCA (27, 82,109). NBCA has a lower bonding strength, higher surface tension and higher viscosity than NBCA. Its lower bonding strength significantly reduced the possibility of gluing the catheter tip in the arterial feeder immediately after embolization. Thanks to its higher surface tension and viscosity. NBCA produces a more uniform column and respectively a more compact cast of the nidus with less fragmentation. To prevent recanalization of the embolized nidus a complete, homogeneous and compact acrylic cast is necessary. If blood clot is left within or around the cast this can be reabsorbed resulting in partial recanalization of the nidus. This phenomenon occurs in less than 2% of cases.

In order to permit fluoroscopic visualization it is essential to add opacifying agents to the acrylic mixture. As opacifying agent Tantalum powder is being added to the mixture. Tantalum is a biocompatible and inert metal with an atomic number of 73 and is used in powder form with particle sized of 1 to 2 um in size.

Addition of Tantalum powder slightly increases the viscosity of the mixture. High-resolution subtraction fluoroscopy increases the visibility of the mixture at lower concentrations of Tantalum powder. Usually 0.5-1g/ml NBCA mixture is used. The polymerization time of the cyanoacrylate must be adjusted to the flow conditions by the addition of Lipiodol. Lipiodol retards polymerization time and allows better penetration of plexiform nidi. Before injection of the acrylic, the micro catheter is irrigated with a non-ionic solution such as 5% dextrose. Contact of the acrylic mixture with an ionic solution will initiate the polymerization process. Therefore, precautions must be taken to avoid premature occurrence of polymerization. In order to determined the appropriate polymerization time, the transit time of the AVM nidus is assessed by determining the time from injection to the appearance of the earliest nidal draining vein or veins.

NBCA embolization was shown to facilitate the technical aspects of surgical resection. Vessels and niduses embolized with NBCA are found to be spongy and easily compressible during surgery. They can be easily cut with microscissors because they are softer and more pliable than those embolized with IBCA. In addition, the NBCA embolized vessels are easily distinguished from arteries supplying normal parenchyma, which therefore can be readily spared (110, 111).

Histologically NBCA was shown to provoke a moderately intense foreign body reaction over the first weeks followed by a lymphocytic infiltration .After 4 weeks focal necrosis of the vessel

wall (angionecrosis) and occasional migration of the NBCA into the extravascular space have been observed histologically (109, 112).

Onyx

It is a nonadhesive liquid, first used by Taki et al for AVM embolization in 1989. It is an ethylene vinyl alcohol copolymer dissolved in the organic solvent dimethyl sulfoxide (DMSO). When the material comes into contact with an aqueous solution, it precipitates and forms a soft spongy polymer cast, initially with an outer layer, remaining semi-liquid centrally. As further material is injected into the cast, it fills the space into which it is injected, then additional material breaks out through the outer layer of the existing cast. It has the advantage of long setting time compared to the NBCA wich will significantly reduces the gluing of the catheter(150). Tantalum powder is used as the radiopaque agent along with this.Its disadvantage is the adverse reactions like infarctions and cerebral necrosis produced by DMSO and also the necessity of DMSO compatible catheters.

Polyvinyl Alcohol Foam

Polyvinyl alcohol foam (PVA) is a particulate embolic material used extensively in brain AVM embolization (113,114). PVA is available in particle sizes ranging from 45-1250 micro m. They are injected in a suspension of contrast material. PVA has been used extensively for preoperative embolization of brain AVMs. The main advantage of preoperative PVA embolization is that the AVM is easily compressed and retracted at surgery (115).

However the main disadvantage of PVA embolization is its high recanalization rate. PVA produces vessel occlusion by mixing with stagnant blood that coagulates. This explains the high recanalization rate of vessels embolized with PVA. Histologically a moderate foreign body reaction with areas of focal angionecrosis is observed following PVA embolization (116). Recent studies demonstrated the superiority of NBCA over PVA as embolic agent in the endovascular treatment of brains AVMs (117,118).

Coils

Soft platinum microcoils are available in different sizes and configurations and with or without Dacron fibers attached. Their application is restricted to certain high flow arteriovenous fistulae, such as vein of Galen aneurysmal malformations, pial arteriovenous, pial arteriovenous fistulae, such as vein of Galen aneurysmal malformations, pial arteriovenous fistulae, or larger av-fistulas within plexiform AVMs in order to decrease flow prior to injection of NBCA, thus avoiding distal migration of NBCA. Coils can also be used through the transvenous approach to obliterate venous pouches of large arteriovenous fistulae. Similar results are achieved with Guglielmi detachable microcoils (GDC) in the obliteration of venous pouches of arteriovenous fistulae, through either the arterial or venous route.

No other embolic materials such as silicon sphere (119), silk (120), ethylene vinyl alcohol copolymer (121) or estrogen-alcohol and polyvinyl acetate (122) have been used in this series.

Absolute alcohol

Absolute alcohol can be effectively used as an embolizing agent ina brain AVMs. This can be used especially in cases of AVMs involving the non eloquent areas after studying the angioarchitecture by superselective catheterization of the AVM. Alcohol can be used at a dose of 1ml/Kg body weight. It acts mainly by denuding endothelial cells and precipitates their protoplasm.It also produces coagulative necrosis of the vessel wall especially the tunica media. Its main complications are intra vascular hemolysis and pulmonary vasoconstriction.

Applications and Goals of Endovascular treatment of Brain AVMs

Preoperative embolization

Preoperative embolization represents of the most important applications of this technique in the treatment of brain AVMs. According to the data available in the literature, it appears that preoperative embolization is the most frequently applied form of embolization in the overall treatment of brain AVMs. Preoperative embolization is performed in order to facilitate surgical removal of large, or angioarchitecturally complex but operable AVMs or in order to convert an inoperable into an operable AVM (123).

In order to be efficient, preoperative embolization must be appropriately planned taking into consideration the particular surgical approach and the anticipated potential difficulties to be encountered during dissection and removal.

The specific goals of preoperative embolization include (1) the size reduction of the nidus, (2) the occlusion of deep feeding arteries such as perforators or choroidal supply, (3) the obliteration of intranidal aneurysms or other intranidal vascular cavities representing weak angioarchitectural elements, and (4) the occlusion of intranidal arteriovenous fistulae.

To have a favorable impact upon subsequent microsurgical removal in terms of radically and postoperative morbidity, preoperative embolization should efficiently and significantly decrease the size of the nidus and the degree of arteriovenous shunting by intranidal deposition of the embolic material. For that purpose, an appropriate mixture of NBCA and lipiodol instead of PVA particles should be used, because NBCA has a superior nidal penetration capacity and a significantly lower, if not absent, recanalization rate compared to PVA.

Preoperative embolization with NBCA was shown not to interfere with or adversely affect the technical aspects of microneurosurgical resection of partially or subtotally embolized brain AVMs. NBCA – embolized vessels and nidi are rather soft and therefore easily compressible and can be easily cut with microscissors, without any or with only minimal bleeding occurring form the transected vessels. This in turn minimize preoperative

blood loss and reduced overall operative time by decreasing the use of bipolar coagulation. Jafar et al. (124) showed that the achieved results of microneurosurgical removal and the observed long-term outcome in their 20 patients with larger sized (mean size 3.9cm), higher grade (mean Spetzler Martin grade 3.2) AVMs who received preoperative transfemoral embolization with NBCA were comparable to those for previously non embolized 13 patients with smaller – sized (mean size 2.3cm), lower grade (mean Spetzler – Martin grade 2.5) AVMs.

Occlusion of feeding arteries without concomitant nidus obliteration or with insufficient nidus obliteration will rapidly induce collateral supply creating significant technical difficulties during subsequent surgical removal of the AVM. This is particularly true for large cortico-subcortical, pail feeders without sufficient nidus obliteration will lead to compensatory recruitment and enlargement of deep, perforating feeding arteries, either directly or through subependymal collaterals. These deep perforating feeding arteries which lie on the far side of the nidus, can only be reached after coagulation and mobilization of the main, more superficial part of the AVM, are buried in the brain parenchyma, have a different consistency and higher fragility than pial feeders and their elimination may be associated with extreme operative difficulties.

Proximal occlusion of pial feeding arteries may also lead to early post embolization development of leptomeningeal collateral arteries on the surface of the brain. Such superficial collateral neovascularity represents non-sprouting angiogenesis and may mask the plane of cleavage between the nidus and the adjacent

brain and may also be the source of significant bleeding. Therefore, feeding artery occlusion without nidus obliteration, with the exception of av-fistulae, should be regarded as technical failure of embolization and the neurosurgeon should be made aware of the anticipated surgical difficulties excepted to arise from either recruitment of deep perforating arteries and / or the development of collateral leptomeningeal vessels.

In order to achieve significant size reduction and diminution of the degree of AV – shunting, one or more embolization sessions may be required preoperatively, depending on the size, number of feeders and angioarchitecture. Endovascular therapy in conjunction with surgery resulted in 9% - 34% savings per treated patient as compared to patients treated with surgery alone (125).

Embolization Before Stereotactic radiotherapy

The role of embolization embolization before stereotactic found little attention in the literature, as opposed to its role as a preoperative method, which has been extensively covered and discussed. Only since 1990 reports addressing specifically the role and efficacy of pre stereotactic radiotherapy embolization and the obliteration rates achieved by radiosurgery of previously embolized AVMs appeared in the literature (108,126,127,128,129). In the most recent study on the subject including 125 patients who underwent embolization to reduce the size of the AVM and subsequently underwent stereotactic radiotherapy total occlusion of the AVM by embolization alone was achieved in 11.2% of the cases obviating the need for radiosurgery and by radiosurgery in

65% of the remaining, partially embolized AVMs (128). This study also concludes that the risk of hemorrhage following partial embolization with ABCA is comparable to the natural history of the AVMs and that the results of stereotactic radiotherapy of the residual nidus are almost as for previously untreated AVMs of the same size (128).

The main goals of pre stereotactic radiotherapy

embolization are :

1. To decrease the target size to less than 3 cm in diameter or the residual nidus volume to less than 10cm, because all radiosurgical series using either the gamma unit or the linear accelerator show a strong correlation between the size/volume of the AVM and the achieved rate of complete obliteration with a significant drop in the radiosurgical cure rate in AVMs exceeding a nidus volume of 10cm (130, 131,133).

2. To occlude selectively angioarchitectural weak elements, particularly intranidal aneurysms and venous pouches in order to reduce the risk of bleeding in the latency period between radiosurgery and AVM thrombosis, which may last up to 24 months.

3.To occlude large arteriovanous fistulae located within an otherwise plexifrom nidus, because it is believed that these high flow, rather large diameter, slower flow coiled and intermingled vascular channel of the plexiform part of the nidus, which are more

prone to develop endothelial proliferation and subsequent thrombosis following application of appropriate stereotactic radiotherapy dosages.

4.To obliterate large AVMs in such a way that multiple compact smaller targets, each one less than 10cm in volume are left, so that each of which can be treated individually by stereotactic radiotherapy.

5.To occlude the dural supply of an AVM, if present, because this represents elimitation of stereotactic radiotherapy due to the proximity of the meaningeal feeding arteries to the bone and the scalp (27).

6. Pre stereotactic radiotherapy embolisation with microparticles of PVA is less effective than NBCA embolization, because of the high recanalization rate of PVA and its inability to effectively occlude intranidal aneurysms and large arteriovenous fistulae. Therefore, for preradiosurgical embolization the best results in terms of stability of obliteration until radiosurgical obliteration is reached are achieved with the use of NBCA as an embolic material (gobin et al, 1996, Berenstein and Lasjaunias 1991, Hurst et al 1995).

Pre stereotactic radiotherapy I embolization does not have any influence on the incidence of postradiosurgical complications, other than contributing to a smaller radiation field by reducing overall nidus size. The incidence if symptomatic radiation necrosis usually observed between 4 and 56 months is reported to be 3.5% to 12.5% (134, 135,136). In addition, Yamamotot et al (137) reported a

delayed morbidity of 5.2% and delayed asymptomatic cyst formation in 7.8% of cases, occurring more than five and up to ten years following radiosurgery.

Since the long-term effects on the brain following, radiosurgical treatment of brain AVMs, with or without previous embolization is not known we follow a cautious policy regarding the indication for radiosurgery, particularly in children and young adult patients.

Palliative Embolization

The role of embolization as a palliatively applied technique in the management of brain AVMs has been underestimated in the literature, Palliative embolization is reserved for large or giant AVMs, which are regarded as inoperable and which because of their extensive arterial supply and complex angioarchitecture cannot be completely obliterated by embolization. The goals of palliative embolization include.

1.The targeted endovascular elimination of weak elements of angioarchitecture associated with a known increased risk for hemorrhage or rehemorrhage, such as flow related extra and/or intranidal aneurysms, intranidal venous varix proximal to an obvious obstruction of the venous drainage, or the presence of an arterial or venous intranidal pseudoaneurysm.

In these cases, catheterization and embolization is restricted to the feeder or feeders carrying the distal flow related aneurysm or supplying a compartment containing the above mentioned weak

angioarchitectural elements. In order to achieve a complete and permanent occlusion. NBCA is used is these cases.

2.Targeted obliteration of compartments draining into the subependymal venous system and having an intraventricularly prolapsing venous varies. This may represent a significant risk factor for intraventricular bleeding an is observed with large cortico ventricular as well as with deep, mainly strio-capsulo –thalamic AVMs. Targeted obliteration of the compartment draining into the subependymal varicously dilated vein, will usually result in flow and pressure decrease as well as shrinkage of the venous varix, eventually eliminating the risk of future intraventricular hemorrhage.

3.Partial embolization of extensive "convexity" AVMs in patients presenting, with severe epilepsy resistant to antiepileptic medication. Such AVMs are usually large and located in two to three vascular territories (MCA, ACA and /or PCA), drain into both the superficial and deep venous system and are associated with regional hypofusion and various degrees of venous congestion. Significant improvement of the frequency and severity of seizures may require multiple sessions of embolization.

4.The relief or amelioration of chronic headaches in patients with large AVMs and additional supply by dural arteries. Selective embolization of the dural supply proved to be very effective as a palliative treatment of severe, chronic headache, in an otherwise large, unresectable AVM.

Postoperative and Post Stereotactic radiotherapy embolization

Post operative embolization is rarely being performed, because surgery in well selected cases usually results in complete AVM removal.

Post operative embolization is considered in the following situations : 1) Following emergency surgical evacuation of hematoma, caused by rupture of an underlying AVM. Unless the underlying AVM is small and readily accessible and removable with the hematoma, larger AVMs are usually not manipulated and not removed. Embolization or other treatment is being performed following patient recovery and after repeat postoperative angiography has demonstrated the full size and construction of the AVM.

2) Following incomplete surgical removal of an AVM. If following surgery of an AVM a residual portion has been left intentionally or unintentionally, embolization or radiosurgery in order to obliterate this residual part may be performed. Obviously, endovascular accessibility will depend on the location of the residual nidus and the type of feeding arteries involved. Proximal clips on feeding arteries may render embolization technically impossible.

Embolization can be also applied after stereotactic radiotherapy either to treat or to assist in the multimodality management of those brain AVMs that have not responded to initial radiosurgical treatment. If there has been no change inAVM size and angioarchitecture at the 24 months angiographic follow up, or if

obliteration has not been observed at 36 months, then the radiosurgical treatment should be regarded as having failed (138). Theoretically, embolization performed after radiosurgery may be associated with an increased risk of arterial dissection caused by microcatheter manipulations, because of radiation induced vessel with changes such as fissuring and endothelial cell necrosis (138). However, in one of the 15 cases of postradiosurgical embolization of this series did such a complication occur.

Curative Embolization

Curative embolization refers to the complete and persisting endovascular obliteration of brain AVMs. Unfortunately the application of endovascular techniques with the defined goal of achieving a complete obliteration of brain AVMs has not found up to now wide acceptance and in most series reported in the literature, curative embolization represents the least frequent type of application of endovascular techniques in the overall treatment of brain AVMs.

This limited role of embolization as a single modality curative method for brain AVMs is clearly reflected in the literature, where reports addressing specifically the issue of complete obliteration of brain AVMs form a part of data on the application of embolization as a preoperative or pre stereotactic radiotherapy adjunct.

Therefore, it seems that in most series reporting on embolization results in brain AVMs, complete obliteration was neither the intention nor the primary goal of the endovascular

treatment in the majority of cases. This might explain the reportedly low rate of achievable complete obliteration of brain AVMs, which is reported to range between 10% and 20%, with the discrepancies between individual series being mainly related to different referral patterns and patient selection criteria. Retrospective analysis of the characteristics of the AVMs in which a complete obliteration was achieved shows a relation with the size, number of feeding arteries and Spetzler Martin grade. In the series of Wickholm et al. (139) complete obliteration was achieved I 10 out of 14 AVMs (71%) with a volume less than 4cc, but in only 3 out of 20 lesions (15%) with a volume of 4 to 8cc for an overall complete obliteration rate of 13.3% in their series of 150 cases.

In the series of Gobin et al. (140) a complete obliteration by embolization alone was achieved in 42% of grade V to VI AVMs Relating the complete obliteration rate with the maximum diameter of the AVM, showed a 31% complete obliteration rate in AVMs with a size of 2 to 3cm, 7% in AVMs 3 to 4cm in size, 13% in AVMs 4 to 6 cm in size and 0% in AVM with a volume of 4 to 10cc, 8% in AVMs with a volume of 10 to 50cc. Finally, the complete obliteration rate was 33% in AVMs with one feeding arteries and 3% in AVMs with more than three feeding arteries. The overall complete obliteration rate in their series of 125 patients with a brain AVM was 11.2% (140).

Similar results arrive Vinuela et al, (141) in their retrospective analysis of 405 patients, with a reported long-term complete obliteration by embolization alone in 40 patients (9.9%) with small and medium-sized AVMs and with fewer than four feeding arteries.

Although both size of the AVM and number of feeders certainly may have an impact on the achievable obliteration rate they didn't prove to be the major determinants for achieving a complete obliteration in a given case of AVM in this series. With larger lesions usually having a higher number of feeding arteries. Both parameters were shown to influence more the technical challenge and complexity of the endovascular procedure, the number of required superselective microcatheterizations, the number of sessions and the length of sessions but not necessarily the achieved degree of obliteration.

In this series, with a clearly predefined subgroup of patients undergoing embolization with the goal of complete, several other parameters were identified, which proved to be more important than size of nidus and total number of feeding arteries in influencing in a positive or negative manner the obliteration rate. In addition, parameters were identified, which didn't seem to have a major influence on obliteration rate.

Complete obliteration rate was clearly related to the topography of the AVM as derived from pre embolization multiplanar MRI, according to the classification described above. Since the topography of AVMs is related to the type of feeding arteries, it becomes evident that AVMs supplied by direct and dominant types of feeding arteries, as it the case with sulcal and mixed sulcal-gyral convexity supratentorial and infratentorial AVMs, pail AV-fistulae and mainly extrinsic deep brain AVMs and AVFs have a significantly higher chance of complete obliteration than AVMs supplied mainly or exclusively by indirect and

supplementary types of feeders, as is the case with many gyral convexity, with deep parenchymal and especially with diffuse AVMs.

Accordingly in this series, a complete obliteration was achieved in 19 patients,of this 12 were achieved with NBCA and 7 were with absolute alcohol alone.

The type of feeding arteries supplying a given AVM is far more important is determining the possibility for complete obliteration than their total number. Pial, choroidal or perforating direct and dominant type feeding arteries, direct and supplementary type feeding arteries and in some cases transit arteries with dominant feeding branches have a positive influence on achieving a complete obliteration that transit type arteries with supplementary feeding branches and retrograde collateral type feeding arteries thus facilitating the endovascular access and the deposition of NBCA.

Angiographic evidence of secondary perinidal angiogenesis was found to be an angioarchitectural influencing strongly and negatively the degree of achievable nidus obliteration. This phenomenon was very frequently associated with transit type arteries giving off multiple supplementary feeding branches but also branches connected with the perinidal angiogenic zone, making differentiation difficult and superselective catheterization impossible.

The compartmental composition of the nidus also influence the obliteration rate. As can be expected, the probability of achieving a complete obliteration is higher with mono compartmentally than with multi compartmentally composed niduses.

In several instances, penetration of the NBCA through intracompartmental communicating channels were observed fluoroscopically resulting in additional obliteration of that compartment and thus obviating the need of a subsequent separate catheterization and embolization. Although precise statistical data on the incidence of intracompartmental communications in multi compartmental AVMs are still lacking, it seems that their presence influences positively the degree of achievable obliteration rate, provided that the embolization is carried out with NBCA.

The pattern of venous drainage rather than the anatomic type of draining veins (cortical, subependymal, both) is an additional parameter influencing the obliteration rate. In this series, a complete obliteration was more frequently achieved in AVMs with converging compartmental draining veins or single draining nidal veins than in AVMs with isolated compartmental draining veins or mixed isolated and converging compartmental draining veins.

It is overall constellation and the relative representation of these parameters in a given case of brain AVM, which influence

positively, negatively or not essentially the achievable degree of obliteration.

Many of the completely obliterated AVMs in this series were surgically well accessible and removable, even without preoperative embolization. The indication for embolization in these cases was influenced either by the patients themselves, explicitly wishing the endovascular treatment or by the referring neurosurgeon.

The mechanisms underlying the recurrence of an obliterated brain AVM after embolization are recanalization and revascularization. Recurrence through recanalization refers to the restoration of the lumen in a feeding artery and / or in vascular channels of the nidus. The use of a reabsorbable material, like PVA, predisposes for recanalization of the occluded channels. Such a recanalization may occur as early as a few weeks following embolization. Recanalization may also occur with permanently occluding agents, such as NBCA. If the cast produced is incomplete, thrombus will form in the residual lumen, leading initially to complete occlusion. In the process of thrombus organization, new channels lined with endothelium may form reconstituting flow to the nidus (142).

Recurrence through revascularization refers to the reestablishment of blood supply to the nidus of the AVM through development of collateral feeding arteries. Such revascularization will occur following proximal occlusion of feeding arteries without appropriate obliteration of the nidus and represents the vascular

response to a technical failure. Angiographically, the venous drainage of the revascularized AVM is always identical to the initial venous drainage of the AVM, emphasizing the need to include the emerging segment of the draining vein in the nidus obliteration. Depending on the arterial territories involved in the supply of the AVM and the pattern of its arterial supply, the following types of collateral revascularization following proximal occlusion may develop, singly or in combination.

Development of newly formed collateral supply : (a) leptomaningeal; (b) subependymal; (c) indirect retrograde, leptomeningeal or subependymal : (d) transdural. (2) Accentuation of preexisting (not embolized)supply with transformation of supplementary into dominant supply.

Fournier et al (143) reported on two occipital AVMs out of a series of 52 brain AVMs embolized with cyanoacrylate. Which showed revascularization at 6 months and two years respectively and suggested that the occipital lobe, because of its rich vascularity, is more prone than other parts of the brain to produce intense collateralization leading indirectly to resupply of embolized AVMs.

Recanalization with formation of capillary structures within the lumen of vessels embolized with NBCA has been shown histologically to occur later than 3 months in partially embolized AVMs (144).

The present series showed the total nidal obliteration rate with embolisation alone was 18%, post embolisation radiotherapy and

surgery has increased the total occlusion rate to 36%.These figures are almost similar to the various data in various publications. Still some of our patients are waiting for other stages of embolization and some are still under followup. that will definitely increase our total occlusion rates by continuing this study.

Complications of endovascular embolisation of cerebral AVMs

Ischemic compilations of embolization may occur either during catheterization and navigation or following injection of embolic material. ischemic complications due to catheterization and navigation represent technical failures and include dissections of major arteries, formation of blood clot around catheters or inadvertent injection of blood clot. The chance for such complications is higher with small than with larger, multipedicular, high flow AVMs. Use of meticulous technique, full and constant attention during fluoroscopically guided catheterization and careful manipulation of microcatheters and micro guidewires are essential for endovascular treatment of the brain AVMs.

Ischemic complications occurring in conjunction with the embolization, i.e. following the injection of he embolic material is invariably due to occlusion of normal arteries. Two mechanisms of normal artery occlusion due to embolization of brain AVMs are distinguished, i.e. anterograde and retrograde (due to reflux). Anterograde occlusion of normal arteries may occur in the

presence of a pseudoterminal type of feeding artery. Pressure rise I the pseudoterminal feeder occurring during the injection of the embolic material may suddenly open its anatomically present but angiographically invisible connection to its distal to the nidus. Anterograde occlusion of normal arteries may also occur, if injection of the embolic material is performed from a proximal position of the catheter tip, within a direct type of feeding artery. Failure to distinguish between an angiogenic zone in relation to watershed transfer an nidus of the AVM, may be another cause of occlusion of arteries supplying normal brain. Retrogenic occlusion of normal arteries is caused by reflux of embolic material proximal to the tip of the microcatheter. This may occur either within a direct type of feeder with occlusion of normal branches proximal to the nidus or within a feeding branch of an indirect feeder with reflux into the trunk of the indirect feeder and occlusion of normal arteries distal to the AVM.

Ischemic compilations occurred in 5 patients (4%). Four of these patients a focal infarction or ischemic zone was detected on post embolization CT.In one patient a transient neurological deficit for about 12 hrs was observed.

The other type of serious complication of endovascular treatment of brain AVMs is hemorrhage, occurring wither during the procedure (perprocedural hemorrhage) or during the early (<72 hours) postembolization period. Perprocedural hemorrhage may be caused by mechanical perforation of a cerebral artery during microcatheterization or due to hemodynamic changes induced by particle embolization of the nidus. Early postembolization hemorrhage is invariably caused by hemodynamic changes

occurring in the hours following partial or subtotal embolization of the AVM.

Before the advent variable stiffness microcatheters, overdistention of an artery by a calibrated leak balloon was the most common cause of intraprocedural hemorrhage (27). With the newer generation variable stiffness microcathers used for brain AVM embolization, mechanically induced hemorrhage became a rare complication of micoratheter has been reported by (145) and anecdotally by others (146). Immediate recognition of arterial perforation is crucial in avoiding morbidity. Perforation of flow – related aneurysm during microcatheter navigation or placement has also been occasionally reported (82). Arterial perforation is diagnosed by contrast material extravasation. Depending on the type of artery perforated, this extravasation will occur either into the subarachnoid space or into the brain parenchyma. It is imperative not to withdraw or pull back the microcatheter, once extravasation of contrast material and thus an arterial perforation has been recognized. The spam induced by the trauma to the arterial wall may lead to cessation of bleeding, if the microcatheter is left in place and thus blocks blood flow. If bleeding does not stop rapidly, they it is advisable to proceed with occlusion of the rupture area using either coils or cyanoacrylate. In this series, arterial rupture occurred in three cases during NBCA injection. In all three cases bleeding stopped by further NBCA injection.None of the patients had a neurologic deficit and all three patients had an excellent outcome.

Perprocedural hemorrhage not associated with mechanical arterial perforation and early postprocedural hemorrhage occurring

within 72 hours following embolization is usually caused by compromise of the venous drainage of AVMs, but in some cases no obvious cause can be identified. In this series, perprocedural hemorrhage in seven cases ie. 6%. Two of these patients died because of massive hemorrhage. Other patients recovered completely without significant residual deficits.

When a patient demonstrates rapid neurologic deterioration following embolization of an AVM or if AVM rupture occurs during the procedure, an emergency CT should be immediately performed. If a cerebral hematoma with mass effect, intraventricular extension, hyperventilation and administration of an intravenous bolus of 100 mg mannitol should be performed and the patient transferred to the operating room for emergency craniotomy and hematoma evacuation under barbiturate anesthesia. Following hematoma evacuation, the patient preferably remains in a pentobarbital induced coma, with continuous monitoring of blood pressure, pulmonary capillary wedge pressure, central venous pressure and cardiac output through arterial and pulmonary artery catheters, until the intracranial pressure returns to normal, i.e. less than 15mmHg for 24 hours. This policy proved life saving, the only poor outcome was most probably caused by a delay of more than one hour between beginning of clinical deterioration and surgery.

Five patients were treated conservatively, because of a stable neurologic condition without or with only slight decline of the level of consciousness. The overall outcome in this group of 7 patients with a hemorrhagic complication was 5 good (72%) with a mortality of 3 cases (28%).

Retrospective evaluation of these cases with per or early post procedural hemorrhage not associated with mechanical arterial perforation revealed the following causes:

1) Embolization of an intranidal AV-Fistula including its draining vein in a mixed plexiform-fistulous AVM, in which the plexiform portion drains also through the same vein.

2) Compromise of drainage of a main nidal draining vein in the presence of multiple converging compartmental draining veins.

3) Occlusion of the main nidal draining cortical vein in convexity AVM with intra ventricular extension into the subependymal venous system, but unexpectedly into the cortical, superficial draining vein through transcerebral veins.

4) Passage of cyanoacrylate into the venous side with occlusion of already stenotic veins at a distance from the nidus, in AVMs with a nidal vein dividing early into multiple draining veins and associated with significant venous high flow angiopathic changes.

5) Presence of intranidal or distal extranidal arterial flow related aneurysms in the residual (not embolized) portion of the AVM.

6) Progressive thrombosis leading to delayed occlusion of draining veins (147).

In no instance could the phenomenon of normal perfusion pressure breakthrough, as described by Spetzler et al. (148), be identified as a cause of a hemorrhagic complication. Maintaining the mean arterial blood pressure to approximately 10 to 15% below baseline for 24 hours after embolization may minimize the risk of hemorrhage in cases in which partial occlusion of the major venous outlet occurred.

The overall morbidity in this series of 103 patients with brain AVMs which underwent a total of 170 sessions of embolization treatment, was 8.7% (9 patients).

The overall mortality rate was 2.9% (3 patients), three patients died of a massive postembolization hematoma.

Summary and Conclusions

Advances in superselective microcatheterization techniques, which took place in the past decade, established superselective endovascular exploration as an integral an indispensable tool in the pre therapeutic evaluation of brain AVMs. The strict and routine application of superselective angiography furthered our knowledge on the angioarchitecture of brain AVMs, including vascular composition of the nidus, types of feeding arteries and types and patterns of venous drainage. In addition, various types of weak angioarchitectural elements, such as flow related aneurysms, intranidal vascular cavities and varix formation proximal to high grade stenosis of draining veins, could be identified as factors predisposing for AVM rupture. A wide spectrum of secondary angiomorphological changes induced by the arteriovenous shunt of the nidus and occurring up and downstream of the nidus have been identified as manifestations of high flow angiopathy. These data help to better predict the natural history, understand the widely variable clinical presentation and to define therapeutic targets of brain AVMs.

Correlation of the topography of the AVM as demonstrated by MR with the angioarchitecture as demonstrated by super selective angiography provided a system for topographic vascular classification of brain AVMs which proved very useful for patient selection and definition of therapeutic goals.

This study showed that 18% of patients with brain AVMs can be cured by embolization alone with a morbidity of 8.7% and a mortality of 2.7%. Part of these patients can, however, be cured

equally effective by microsurgery or stereotactic radiotherapy. Which modality will be chosen for a particular patient will mainly depend on the locally available expertise and experience, but also on the preference of the patient following its comprehensive information about the chances for cure and the risks associated with each of these therapeutic modalities. Post embolization radiotherapy or surgery has increased the cure rate up to 36% in this case.

Embolization has a significant role in the multimodality treatment of brain AVMs, by either enabling or facilitating subsequent microsurgical or radiosurgical treatment. Appropriately targeted embolization in otherwise untreatable AVMs represents a reasonable form of palliative treatment of either ameliorating the clinical condition of the patient or reducing the potential risk of hemorrhage.

Regarding the practical aspects of the endovascular treatment the following conclusions could be drawn from the experience obtained with this series of 103 patients with a brain AVM:

1. The goal of endovascular treatment should be defined prior to the procedure. This does not preclude a change in the goal, if additional information obtained during the procedure make this necessary.

2.The result of endovascular treatment of a brain AVM in terms of the degree of obliteration achieved and complication rate depends mainly on the endovascular strategy developed and the technique applied. These depend on the specific angioarchitecture and topography of the individual AVM, on the past history and clinical

presentation of he patient and on the predefined goal of embolization. The strategy should include the definition of embolization targets, the selection of the most appropriate approach for endovascular navigation, the determination of the sequence of catheterization of individual feeding arteries, the selection of the type of catheters and microcatheters, the selection of the appropriate embolic material as well as the site and mode of their delivery. Therefore, every endovascular move should be, a in a chess game, the result of a logical plan.

3.Atraumatic superselective micro catheterization is a key point in the endovascular treatment of brain AVMs. It requires manual skills, knowledge of anatomy and respect for the vascular wall.

4.All locations of brain AVMs should be regarded as eloquent, and no distinction should be made between eloquent and non-eloquent areas of the brain when deciding on the execution of embolization or the selection of embolic materials.

5.Embolization should be performed only after the particular angioarchitecture has been fully appreciated and the particular compartment to the embolized has been precisely localized with angiographic MR correlation.

6.The technical goal of embolization is the stable obliteration of the nidus of the AVM with preservation of the normal arterial supply of the adjacent and remote brain parenchyma and without compromise of the venous drainage of the brain. Cyanoacrylate is, currently, the best available embolic agent to achieve that goal. It is not more dangerous than the other available embolic materials, but it use requires appropriate training. Other embolic

materials can be used in selected cases with particular angioarchitectural features, either to enable appropriate delivery of cyanoacrylate (e.g. use of coil in large intranidal arterio – venous fistulae to slow down the flow before injecting the cyanoacrylate) or to enhance progressive nidus obliteration following cyanoacrylate embolization (e.g. Supplementary embolization of a small remaining part of nidus fed by indirect type of feeders with microparticles of PVA, following subtotal nidus obliteration with cyanoacrylate).

7.Performing the endovascular treatment of brain AVMs under general anesthesia was shown in this series to improve the working conditions for the interventional neuroradiologist and its team, to increase the obliteration rate, to improve the overall patient outcome and to decrease the number of sessions required to achieve the defined goal of treatment.

Our understanding of the topographic-vascular relationships and of the angioarchitecture of brain AVMs as well as out ability to resolve angiographically small feeding arteries and intranidal vascular micro channels are more advanced than the currently available endovascular micro instrumentation in safely entering these vessels and precisely obliterating them. Unless smaller, less traumatic microcatheter-micro guide wire systems and additional endovascular obliteration techniques will be developed, it is hardly expectable that the currently achievable complete obliteration rate will significantly increase.

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Appendix

Patients included in this study

Age	Sex	Presentation	Diagnosis	Stage	Sitting
17	F	Focal with sec generalisation	Lt.frontal	3	2
28	F	Sensory seizures	Rt.basal ganglion	4	2
41	M	Focal Seizures	Rt.temporal	4	3
57	M	Haematoma	Rt.parietal	2	1
18	M	Head ache	Lt.parietal	3	1
14	M	Haemorrhage,Head ache	Rt.frontal	1	1
45	M	ICH	Rt.occipital	4	2
52	M	IVH	Post.callosal	5	4
29	M	ICH	Lt.Frontal		1
38	M	Haematoma,Focal Seizures	Rt.frontal	3	4
23	M	ICH,IVH	Rt.parieto occipital	3	1
42	M	Focal Seizures	Rt.fronto parietal	4	3
50	M	Occipital head ache	Rt.occipital	4	3
36	M	GCTS	Rt.frontoparietal	4	2
49	M	ICH	Rt.frontoparietal	3	3
17	M	GCTS	Lt.Frontal	2	1
16	m	ICH	Lt.parieto occipital	4	4
29	M	ICH	Rt.parieto occipital	4	3
41	M	GCTS	Lt.temporal	3	2
31	M	Focal Seizures	Rt.temporal	3	2
42	M	Focal Seizures	Lt.insular	3	2
36	M	Focal Seizures	Rt.frontal	3	2
29	M	ICH	Corpus callosum	3	1
23	M	GCTS	Lt.Frontal	3	1
40	M	GCTS	Rt.fronto parietal	3	2
27	M	Seizures,abnormal movements of limbs and face	Rt.thalamic	4	2
36	M	ICH	Rt.parieto occipital	4	2
22	M	GCTS	Rt.motor	3	3
57	M	ICH	Rt.parietal	3	1
18	M	Focal Seizures	Rt.frontoparietal	3	1
36	M	Head ache	Rt.parieto occipital	4	3
30	M	ICH	Rt.parietal	2	1
43	m	IVH	Rt.frontoparietal	3	2
49	M	LOC	Lt.temporal	2	1
22	M	Focal Seizures	Rt.frontoparietal	3	1
40	M	Dysphasia	Lt.temporal	3	2
34	M	GCTS	Lt.temporal	3	2
17	M	IVH	Rt.choroidal	3	1
24	M	ICH	Rt.frontal	3	2
26	M	Focal Seizures	Lt.insular	3	1
39	M	GCTS	Rt.parieto occipital	3	1
18	m	Visual blurring	Lt.parieto occipital	3	1
20	M	GCTS , head ache	Lt.fronto parietal	3	2

35	M	Focal Seizures	Lt.insular	4	2
33	M	ICH	Rt.motor	2	1
29	M	GCTS	Rt.Parieto tempero occipital	5	1
59	M	Focal Seizures	Lt.frontal	3	2
36	M	ICH +IVH	Rt.thalamic	3	1
47	M	GCTS	Rt.tempero parieto occipital	5	2
34	M	ICH	Cerebellar	3	1
53	M	Focal Seizures	Lt.parietal	3	1
33	M	ICH	Lt.parieto occipital	3	1
30	M	ICH	Rt.frontoparietal	2	1
36	M	Head ache	Rt.parieto occipital	3	2
30	M	Focal Seizures	Rt.frontal	2	1
52	M	ICH	Rt.parietal	3	1
38	M	ICH	Lt.parietal	3	1
20	M	Focal Seizures	Lt.temporal	2	1
22	m	focal lt UL seizures	Rt. frontal	2	4
20	M	Gen. seizures,head ache	Lt temporal	3	1
18	M	Focal with sec generalisation	Rt. Fronto-parietal	3	3
41	m	Gen. seizures,IVH	Rt.temporal	5	5
51	M	IVH,Head ache	Vermian		1
17	M	LTemporalVisual fied cut,head ache	Lt.occipital	1	1
45	M	IVH	Posterior callosal	4	1
24	M	IVH	Lt.peritrigonal	2	1
36	F	Focal seizures	Lt.frontal	4	1
28	F	SAH	Rt.temporal	4	1
25	M	Gen.seizures	Rt.perisylvian	4	2
15	F	Gen.Seizures	Rt.Thalamic	4	2
39	F	ICH	Lt.tempero-parietal	3	2
22	M	Sizures	Rt.Motor strip	3	3
47	M	Gen.seizures	Lt.frontal	2	1
20	F	Haemorrhage	Rt.frontal	1	1
28	F	Haemorrhage	Rt.basal ganglia	5	3
23	F	Gradual weakness rt.hand	Lt.frontal	4	1
42	M	Haemorrhage	Lt.insular	4	1
48	M	Focal seizures	Rt.frontal	2	2
31	M	Haemorrhage,Lt.homo.hemi anopia	Rt.tempero parieto occipital	5	3
36	M	Haemorrhage	Rt.parieto occipital	5	2
26y	M	Gen.seizures	Rt frontotemporal	4	1
19	M	Focal seizures	Rt.parieto occipital	5	1
40	M	Gen.seizures	Rt.fronto parietal	4	2
29	F	Head ache,Lt.inf.quadrantanopia	Rt.parieto occipital	4	1
27y	F	IVH,gen.seizures,head ache	Lt.capsuloganglionic	5	1
22	f	Focal seizures,mild Rt.hemiplegia Gr.4	Lt.thalamic	4	2

26	M	Head ache	Lt.occipital	3	1
31	M	Focal with sec generalisation	Rt.frontoparietal	5	1
12	M	IVH,post surgical residul AVM	Rt.temporal	2	1
27	M	Head ache	Rt.occipital	2	1
34	M	Focal with sec generalisation	Rt.fronto parietal	4	1
19	F	Haemorrhage	Rt.tempero parieto occipital	3	1
52	M	Focal seizures after surgery for AVM	Rt.parietal	3	1
16	M	Hemorrhage	Corpus callosum	3	2
29	M	Hemorrhage	Cerebellar	4	1
23	M	GCTS	Rt.Frontal	2	1
13	F	IVH	Corpus callosum	2	1
50	M	GCTS	Lt parietal	3	2
21	F	Head ache	Corpus callosum	2	1
32	F	GCTS	Lt.Insular	3	1
25	F	Head ache	Lt.occipital	3	1
47	F	ICH	Lt.parieto occipital	4	1
18	M	ICH	Lt parietal	2	1

CEREBRAL AVMs – NATURAL HISTORY AND MANAGEMENT

Hospital No [ ] Category [ ]

Name: Age: Sex:

Referred by: Dr.....
Address:

Patients Address:

DIAGNOSIS :

Size of the AVM at the time of initial diagnosis:

DO Regn. [ ]

Management Type

Medical Mx ○ Embolized ○ Operated ○ Radiotherapy ○

No.	MRD No	Date Adm	Date Dis	DSA	CT	CTA	MRI	MRA	Surgery	RT	Embo
Study No Date											

Hospital No [ ] Name:

PRE PROCEDURE EVALUATION

HISTORY:

Date of first symptom

Date / Duration Description

Hemorrhage

- Loss of consciousness
- Sudden Headache
- Vomiting
- Altered Sensorium
- Sudden Neuro deficits

Seizures

- Focal
- Generalized

- Medications

- Response to Rx

Progressive deficits

Chronic Headache

Smoking

Others

Hospital No [ ] Name:

Past History
Hypertension Y / N Diabetes Y / N Body Weight :

Family History
Hypertension Y / N Diabetes Y / N Intracerebral Hge. Y / N Seizures Y / N

GENERAL EXAMINATION:

Baseline Pulse rate: Baseline B.P.: Neurocutaneous markers:

CNS EXAMINATION :

Handedness Rt / Lt / Ambi

GCS at presentation:

Fundoscopy : Atrophy
Papilloedema
Haemorrhages

Preprocedural deficits

Cranial Nerve

I	VII
II	VIII
III	IX
IV	X
V	XI
VI	XII

Motor GR I II III IV V

UL

BICEPS
TRICEPS
SUPINATORS
PRONATORS
FOREARM FLEXORS
EXTENSORS

Hospital No [ ] Name:

PREPROCEDURE IMAGING: CT Date : CT No.:

CT Findings: Site
Side
Size
Depth
Associated haemorrhage
Atrophy

MR Date : MR No.:

Location

- **Side** Right Left Midline
- **Site** Frontal Parietal Temporal Occipital Deep nucleii Cerebellar Brainstem
- **Depth** Cortical Subcortical Ventricular surf. Intraventricular Basal ganglia Internal capsule Corpus callosum Cerebell Hemisph Vermian Deep cerebell Brainstem

Eloquence Sensorimotor Visual Language Thalamus Hypothalamus Basal ganglia Int. Capsule Cerebell. peduncle Deep cerebell. nuclei Brainstem

Size Max diameter in Axial Coronal Sagittal

Border Compact Diffuse (islands of normal parenchyma within AVM)

Edema

Mass Effect

Hospital No [ ] Name:

LL

HIP FLEXORS
EXTENSORS
KNEE FLEXORS
EXTENSORS
PLANTAR FLEXORS
EXTENSORS

Sensory	UL	LL
Rt		
Lt		

Reflexes	Rt	Lt
SUPFL		
DEEP		

Hospital No [ ] Name:

Hemorrhage

Compartment

Location

Size

Age

Other Findings

Hospital No | | Name:

DIAGNOSTIC ANGIOGRAM

DSA Date | Time after ictus | DSA No.

Arterial :

Feeding arteries

ICA

ACA

MCA

PCA

VERTEBRAL/ BASILAR

Cortical

Perforator

Dural

Aneurysms

Intranidal | Distal | Proximal | Unrelated

Hospital No [ ] Name:

Nidus :

Size

AV Communication

Flow characteristics Low
Mild
Moderate
High

Venous :

Draining veins

Cortical

Deep

Veins also draining normal parenchyma

No. of Veins

- Draining the nidus
- Reaching the sinus

Stenosis

Present Absent

Kinks

Present Absent

Ectasias

Present Absent

Venous sac communicating directly with sinus
Venous sac giving rise to veins
Feeders to venous sac – through fistulae

Hospital No [ ] Name:

Sinus thrombosis / Occlusion

Present Absent

Venous reflux

Present Absent

SPETZLER MARTIN GRADING

Size:
Venous drainage:
Eloquence:

GRADE I II III IV V VI

Constriction/ stenosis of the draining vein (Type A) : Yes / No

Presence of incidental, feeder or intranidal aneurysms (Type B) : Yes / No

Periventricular location (Type C) : Yes / No

OTHER INVESTIGATIONS :

Hospital No [ ] Name:

EMBOLIZATION: Anesthesia : GA Sedation LA

Stage of embolization: 1st / 2nd / 3rd / 4th / 5th

Interval since last embolization:

Indication for Embolization

- Once / multiple bleed
- Increasing neurological deficit
- Inoperable
- Patient's wish
- Intractable seizures

Aim of embolisation

- Definitive
- Pre operative
- Pre Stereotactic Radiotherapy
- Palliative

Hospital No [ ] Name:

	Feeder # 1	Feeder # 2	Feeder # 3	Feeder # 4
PROCEDURE				
Location in nidus				
Feeder artery				
Location of guidecath				
Microcatheter				
Microcath Len / soft				
Microguidewire				
Pressures				
- Guidecath tip				
- C1 ICA / Bas				
- Proximal Art				
- Mid Way				
- Distal Art				
- Feeder				
Final Location (Nidal / Proximal)				
Embolic material (%)				
Volume				
BP at embo (S / D / M)				
End point of embo.				
- Good percol.				
- Venous embo				
- Feeder reflux				

Hospital No [ ] Name:

Technical complications :

Check angiogram

- Percentage of reduction of nidus
- Feeder
- Venous stasis
- Others

END OF PROCEDURE :

- Extubation : Cath lab extubation Elective ventilation
- Reason for elective ventilation
- Post procedural vitals
  - Pulse
  - BP
- Post procedural neurological status

OTHER PROCEDURAL DATA :

- Duration of procedure
- Fluoroscopy time

Heparin

Contrast

Other Drugs

Hospital No [ ] Name:

POST PROCEDURAL ORDERS

- Blood Pressure Target BP

Body weight
SNP Reconstitn : mg in ml → ml / hr = 1μ / Kg / min

NTG Reconstitn : mg in ml → ml / hr = 1μ / Kg / min

Time of withdrawal of vasodilators (from end of procedure) - hrs

- Heparin Infusion : Units / hour

Bolus : Units hourly

LMW Heparin ml hourly

- Mannitol ml hourly
- Dexona mg hourly
- IV Fluids Negative / Equilibrium / Positive → ml

DETAILS OF ICU STAY Time of arrival at ICU :

(Neurological status) (Hemodynamic status)

First 6 hours :

6 – 12 hours

12 – 48 hours

Hospital No [ ] Name:

Neurological status (At time of discharge)

PLAN : Review after months for
- Check up
- Imaging studies – CT / MR
- Admission and angiogram
- Repeat embolization

Follow Up

3 months

6 months

12 months

2 years

3 years

4 years

5 years

6 years

7 years

8 years

9 years

10 years

QUESTIONNAIRE

Name : Age: Sex: Hopital No:

Address:

Instruction

1.	**Read all questions clearly and answer**
2.	**Write your asnwers in the space provided**
3.	**Tick ☑ the appropriate boxes**

1. How many times you have had embolisation ? .....
2. Sepcify the date of last embolisation .....
3. Was there any episode of bleeding in the brain after any embolisation ?

   Yes ☐ No ☐

   If yes when .....

   If yes how many days/ months/ years after embolization.....
4. Were there any symptoms (head ache/ fits/ weakness or any other) related to this disease before emboization

   Yes ☐ No ☐

   (a) If yes what were the symptoms. .....

   (b) Status of the symptoms after embolization

   Increased ☐ Decreased ☐ No change ☐
5. Have you been advised sterotactic radiation treatment after embolization

   Yes ☐ No ☐

   (a) if yes when and where did you have radiation treatment..... Date of treatment

   (b) If no state reasons for not doing the advised radiation treatement .....
6. Have you been advised surgery after embolization?

   Yes ☐ No ☐

   (a) If yes date of surgery and where .....

   (b) If no state reasons for notdoing the advised surgery. .....
7. Status of the symptoms after radiation treatment or surgery

   Increased ☐ Decreased ☐ No change ☐
8. For the same disease have you been taking any medicnes before embolization

   Yes ☐ No ☐

(a) If yes which Medicines .....