Embolisation of Small (< 3 cm) Brain Arteriovenous Malformations

Correlation of Angiographic Results to a Proposed Angioarchitecture Grading System

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Summary

The role of embolisation in the treatment of small (< 3cm) brain arteriovenous malformations (AVMs) has not been elucidated. We reviewed our experience using embolisation in the treatment of small AVMs and correlated a proposed grading system based on the angioarchitecture to the percentage obliteration achieved by embolisation.

Eighty-one small AVMs in 80 patients were embolised from 1984 to 1999. The age range was from 3 to 72 years. The AVMs were given a score from 0 to 6 based on the angioarchitecture. The assigned scores were as follows: nidus (fistula = 0, < 1 cm = 1, 1-3 cm = 2), type of feeding arteries (cortical = 0, perforator or choroidal = 1), number of feeding arteries (single = 0, multiple = 2) and number of draining veins (single = 0, multiple = 1). Angiographic results based on percentage obliteration were grouped into three categories: complete, 66–99%, and 0–65%.

The goal of embolisation was cure in 27 AVMs, pre-surgical in 23, pre-radiosurgery in 26, and elimination of an aneurysm in five. Embolisation achieved complete obliteration in 22 (27%) of the 81 AVMs. In the AVMs where the goal was cure, 19 (70%) of 27 were completely obliterated. In the AVMs with angioarchitecture scores of 0-2, 12 (86%) of 14 were cured, with scores of 3-4, 8 (34%) of 24 were cured and with scores of 5-6, 2 (4%) of 44 were cured. Embolisation resulted in transient morbidity of 5.0%, permanent morbidity of 2.5%, and mortality of 1.2%. There were no complications in AVMs with scores of 0-2.

Embolisation is an effective treatment of small AVMs when the angioarchitecture is favourable (scores 0-2). This includes pure fistulas and AVMs with a single, pial, feeding artery.

Introduction

Brain AVMs are an important cause of haemorrhagic stroke and death in the population. Untreated AVMs bleed at an annual rate of 2-4% although the risk may be as high as 6% in the first year following a bleed [1-5]. The risk of death from a bleed has been estimated to be from 17 to 29% and 23 to 38% of survivors have a permanent disability [3,6]. In a review of 115 patients, Hartmann et Al found a lower morbidity from initial bleeds with only 16% of patients being left with moderate or severe morbidity [7].

Treatment strategies for small (<3 cm) AVMs have been a source of spirited controversy between advocates of surgery and radiosurgery (RS) [8-14]. Reports on embolisation have included predominantly larger AVMs and for most patients embolisation has been an adjunctive therapy followed by surgery or RS [15-20]. In this report our goal was to review our results of embolisation in small AVMs and test a proposed grading system based on the angioarchitecture that would predict percentage obliteration.

Material and Methods

We reviewed our clinical and imaging data on 80 consecutive patients with small brain AVMs who were treated by embolisation as part of the management strategy. The data were taken from our database of 674 patients with brain AVMs who have been assessed by our multidisciplinary team. The data were retrospectively gathered from 1984 to 1991 and prospectively collected from 1992 to 1999.

The cohort of 80 patients consisted of 41 males and 39 females ranging in age from 3 to 72 years (average age of 34.8 years). On presentation, 39 (49%) patients had haemorrhages, 32 (40%) had seizures, 4 (5%) had a neurological deficit and five (6%) were asymptomatic or had headaches. Thirty patients (38%) had neurological deficits at the time of embolisation. Embolisation was done in 81 small AVMs, in 80 patients. These 80 patients had a total of 99 embolisation sessions. Embolisation was the only treatment in 27 patients (28 AVMs), or done in combination with surgery in 20 patients, or in combination with RS in 30 patients. Three patients had a combination of embolisation, RS and surgery. Excluded from the analysis were patients with vein of Galen malformations and a single patient who had an embolisation with coils.

The locations of the AVMs are summarized in table 1. Forty-three were left sided, 30 right sided and eight were midline either corpus callosum (six AVMs) or cerebellar vermis (two AVMs). Fifteen (19%) of 81 AVMs were Spetzler-Martin grade I, 35 (44%) were grade II and 31 (39%) were grade III. Fourteen patients had 16 aneurysms: ten were pre-nidal, five were intra-nidal, and one was remote. One patient with Osler-Weber-Rendu disease had two separate arteriovenous fistulas (AVFs) treated by embolisation.

The goal of embolisation for each patient was available from the database and was categorized as follows: cure in 27 AVMs, pre-surgical in 23 AVMs, pre-radiosurgery in 26 AVMs and elimination of an aneurysm in five AVMs. The pre-radiosurgery embolisations were done for size reduction since the chance of cure by radiosurgery increases as the AVM size decreases. Three of the five patients who were embolised to occlude an aneurysm went on to surgery to treat the AVM.

All AVMs were given a score based on features of their angioarchitecture. The score was based on the size of the nidus, the number of feeding arteries, the presence of feeding perforating arteries and the number of draining veins (table 2). We established this grading system to determine its predictive value in embolisation. The assigned scores were as follows: nidus (fistula = 0, < 1 cm = 1, 1-3 cm = 2), type of feeding arteries (cortical = 0, perforator or choroidal = 1), number of feeding arteries (single= 0, multiple= 2) and number of draining veins (single = 0, multiple = 1). The weighting of the score for the number of feeding arteries was based on an analysis of previous reports on embolisation [15,16]. These reports had suggested that as the number of feeding arteries increased the percentage of angiographic obliteration decreased. For analysis the angioarchitecture scores were totaled and separated into three categories: 0-2, 3-4 and 5-6.

The angiographic results based on follow-up angiograms were grouped into three categories: complete obliteration, 66-99% obliteration and 0-65% obliteration. In 67 AVMs the results were based on angiograms done between two months and five years after embolisation. In the remaining 14 AVMs the results were based on angiograms done immediately following the embolisation. These angiographic results were correlated to the angioarchitecture scores (table 3). These correlations were statistically analyzed using Fisher's exact test (two-tailed). The relationship between angiographic results and the individual features of the angioarchitecture were also reviewed (table 4). The cure rates and complications of embolisation were reviewed in relationship to the Spetzler-Martin Grade (table 5).

Up until 1995 most embolisations were done

under neuroleptic anaesthesia. Since then most have been done under general anaesthesia. Systemic heparinization (5,000-10,000 units intravenously) was used in all cases. Protamine was used to reverse the heparin at the end of the study. Provocative testing with sodium amytal was done in a few early cases. Up until 1987 a calibrated leak balloon catheter (Ingenor, Paris, France) was used with the propulsion chamber. Since 1987, flow directed microcatheters (Magic, Balt Co., Montmorency, France; Spinnikar, Target Therapeutics, Fremont, CA, U.S.A.) have been used for most cases. In a few patients variable stiffness guidewire-directed microcatheters (Tracker, Target Therapeutics, Fremont, CA, U.S.A.) were used. Isobutyl-2 cyanocrylate (IBCA) was the embolic agent used until 1988 and thereafter N-butyl cyanoacrylate (NBCA, Histoacryl Bleu, Melsungen AG, Germany) was used. The liquid adhesive was mixed with lipiodol and tantalum powder in varying proportions according to the flow. From 1993 to 1994 tantalum was not available and tungsten powder was substituted. In the last few years when concentration of the lipiodol was greater than 50% tantalum was not added. The patients were given dexamethasone (8 mgm three times daily for 2 days) beginning at the time of the procedure.

Results

Embolisation achieved complete angiographic obliteration in 22 (27%) of the 81 AVMs. Follow-up angiography, from two months to five years later, confirmed the obliteration in 19 of the 22 AVMs (table 3). In three of the 22 AVMs complete obliteration was evident at the time of the embolisation but follow-up angiography was not done. Nine of the 22 AVMs had residual shunting immediately after embolisation but the follow-up angiogram showed complete occlusion. In the 27 AVMs where the goal of embolisation was cure, 19 (70%) were completely obliterated and six (22%) had 66-99% obliteration. Table 5 shows the number of cures in relation to Spetzler-Martin grades. Embolisation was successful in all five patients who were embolised to eliminate prenidal aneurysms.

Five patients had partial obliteration by embolisation and no further treatment. Two had bled and their prenidal aneurysms were occluded. Both were in their eighth decade of life and no further treatment was advised. Three patients were reluctant to proceed with further treatment.

In the AVMs with angioarchitecture scores of 0-2, 12 (86%) of 14 were cured (table 3). In the 23 AVMs with angioarchitecture scores of 3-4, eight (34%) were cured, five (22%) were 66-99% obliterated and ten (43%) were 0-65% obliterated. In the 44 AVMs with angioarchitecture scores of 5-6, two (4%) were cured, ten (23%) were 66-99% obliterated and 32 (73%)

Table 1 **Location of the small AVMs embolised (N=81 AVMs)**

Location	No (%)
Frontal	15 (19%)
Temporal	18 (22%)
Parietal	13 (16%)
Occipital	12 (15%)
Corpus Callosum	6 (7%)
Thalamic	6 (7%)
Cerebellum	11 (14%)

Table 2 **Determination of angioarchitecture score**

Angioarchitecture	Points Assigned
Nidus	
Pure Arteriovenous Fistula	0
< 1 cm	1
1 - 3 cm	2
No. of Feeding Arteries	
Single	0
Multiple	2
Type of Feeding Artery	
Cortical	0
Perforator / choroidal	1
No. of Draining Veins	
Single	0
Multiple	1

were 0-65% obliterated. The correlation between the angiographic scores and the angiographic obliteration achieved by embolisation was found to be statistically significant using Fisher's exact test (P<0.0001). Table 4 shows the percentage of cures in relationship to the specific features of the angioarchitecture.

Seven (8.8%) of the 80 patients had complications as a result of embolisation (table 6). Two of these patients had intracerebral haemorrhages: one ten hours and the other four days after the embolisations. One died as a result of the bleed accounting for the mortality of 1.2% and the other patient completely recovered. Five patients developed non-haemorrhagic neurological deficits either immediately following the embolisation or up to 12 hours after the procedure. Four of the six patients made a complete recovery. Two patients were left with a disability accounting for a permanent morbidity of 2.5%. One was minor and she was able to return to work. The other was left with a moderate disability. Four patients had "glued" catheters that could not be removed from the vascular system but none caused any morbidity.

There were no complications related to embolisation of AVMs with angioarchitecture scores of 0-2. Five of the seven patients who had complications had AVMs with high (5-6) angioarchitecture scores (table 6).

Five of the seven patients who had complications had Spetzler-Martin grade III AVMs and two were grade II (table 5). There were no complications in the grade I patients. There was no permanent morbidity or mortality in the Spetzler-Martin grades I or II AVMs. Three of seven complications occurred in thalamic AVMs, including the two patients with permanent morbidity and mortality. This represented 50% of the thalamic AVMs in our series. Two were due to inadvertent occlusion of perforators to the internal capsule resulting in strokes. One resulted from venous penetration of the embolic material into the vein of Galen followed by an early post embolisation haemorrhage.

Discussion

The controversy regarding treatment of small AVMs has been between surgery and RS. Surgical cure rates range from 94-98% [10,11]. Schaller et Al reported that 27.4% had neurological morbidity related to surgery and permanent neurological deficits persisted in 3.2% of patients [10]. The proportion of patients with non-neurological morbidity from this series was 9.7%. RS results in a progressive obliteration of the AVM over a two to three year period. However, during this period, the AVM can still bleed. The annual bleeding rate during this latent period following RS varies from 3.0 to 4.8%, although in Pollock et Al's series, 7.7% bled in the first eight months [9,16,21]. Cure rates from RS, confirmed by angiography, range from 55-65% [22,23]. The most important factor for a cure is the size of the AVM 22. Higher cure rates from RS, in the range of 80%, are frequently quoted, however only 38 to 55% of the patients in these series have had angiographic follow-up [9,24,25]. Delayed radiation necrosis after RS can result in neurological deficits in three to 13% of patients [5,22,25,26]. Most of these deficits occur between six months and two years following RS, although Yamamoto et al. reported two patients (5.3%) who had symptomatic radiation necrosis five and seven years post RS [22]. Porter et Al used a decision analysis model to conclude that surgery of small AVMs achieved a better clinical outcome, compared to RS, as long as the rate of permanent neurological morbidity from surgery did not exceed 12% and surgical mortality did not exceed 4% [13].

Embolisation of brain AVMs is often an adjunctive therapy to be followed by surgery or RS [15-20]. In most series, cure rates from embolisation have ranged from 11-15% with a permanent procedural morbidity from 6-14% and a mortality from 0-1.6% [15-20,27-29]. In Valavanis et Al's review of their own series, 158 (41%) of 387 AVMs were completely obliterated by embolisation [29]. In the series by Wickholm et Al ten out of 14 AVMs with a volume of less than 4 cc were completely obliterated by embolisation [20]. In Gobin et Al's series 31% of the small AVMs were cured by embolisation [16].

In our series, the goals of embolisation were cure, presurgical, preradiosurgery and elimination of an aneurysm. In 27 AVMs the goal of embolisation was cure. Nineteen (70%) of the 27 AVMs were completely obliterated by embolisation alone. In six of the remaining eight AVMs we achieved a 66-99% size reduction. The cure rate from embolisation when the goal was cure is comparable to RS but has the advantage of providing an immediate protection from bleeding.

In theory, size reduction of an AVM should be a useful adjunct to RS since smaller volume AVMs are more likely to be obliterated by RS [23,25]. Friedman et Al achieved a complete obliteration with RS in 81% of AVMs between 1 and 4 cc in volume, 89% of AVMs between 4 and 10 cc in volume and 69% of AVMs greater than 10 cc in volume [25]. Pollock et Al found that smaller AVM size correlated with successful RS but found that embolisation before RS was a negative predictor for successful RS [23]. This apparent dichotomy can be explained by the fact that their patients had been embolised with particles that are well known to recanalize. Recanalization can occur with liquid adhesives (NBCA). The apparent "cure" results from proximal occlusion of the feeding arteries with little or no penetration into the nidus and its junction with the draining veins [30]. At present there is no proof indicating that AVMs made smaller by embolisation are more likely to be cured by RS compared to AVMs treated only by RS.

Retrospective studies have correlated the presence of aneurysms and venous stenosis to presentation with haemorrhage [31-34]. Prospectively, patients with arterial aneurysms associated with AVMs have been found to have a higher annual risk of bleeding, from 5% to 9%, irrespective of their presentation [35]. In this report, embolisation was successful in eliminating prenidal aneurysms in all five patients where this was the primary goal.

The angioarchitecture of an AVM has been an important factor in deciding whether embolisation should be considered. Embolisation of AVMs with direct dominant feeders and the pure arteriovenous fistulas have had favorable outcomes [29]. Outcome from embolisation of AVMs with "en passage" arterial feeders and

Table 3 **Relationship of angioarchitecture score* and % obliteration by embolisation alone (N=81 AVMs)****

Angioarchitecture Score*	No. of AVMs	Results of Embolisation		
		Cure***	67-99%	0-66%
0 - 2	14	12	1	1
3 - 4	23	8	5	10
5 - 6	44	2	10	32
Total	81	22	16	43

** See table 2 for angiographic scoring system*
*** Correlations were statistically significant using Fisher's exact test (2 tailed) (P < 0.0001)*
**** On follow-up angiography in 19 of the 22 AVMs and on immediate post embolisation angiography in the remaining 3 AVMs*

Table 4 **Specific angioarchitecture features and relationship to angiographic cure by embolisation alone (N = 81 AVMs)**

Angioarchitecture feature	No. of AVMs with this feature	Cure
pure AVF*	5	5 (100%)
single artery**	18	15 (83%)
< 1 cm nidus	15	7 (47%)
single vein	37	12 (32%)
multiple veins	44	10 (23%)
1-3 cm nidus	61	10 (16%)
multiple arteries	63	7 (11%)
perforators/ choroidal feeders	20	2 (10%)

** arteriovenous fistula; ** all pial*

Table 5 **Summary of the results of embolisation in small arteriovenous malformations (N = 81) in relationship to Spetzler-Martin Grade**

Spetzler-Martin Grade	No. of AVMs	Cure*	Minor Morbidity	Major Morbidity & Mortality
I	15	6 (40%)	0	0
II	35	13 (37%)	2 (5.7%)	0
III	31	3 (10%)	3 (9.6%)	2 (6.4%)
Total	81	22 (27%)	5 (6.2%)	1 (2.4%)

** includes 3 patients who did not have follow-up angiography*

Table 6 **Summary of the results of embolisation in small arteriovenous malformations (N = 81) in relationship to Spetzler-Martin Grade**

Age / Sex	Lesion Location	Grade *	Score **	Complication	Time Post Embolisation	Etiology of Complication	Outcome
29 / F	occipital	III	5	incomplete hemianopsia	1-6 hours	reflux of liquid adhesive	complete recovery
56 / F	thalamic	III	3	ICH, IVH	4 days	venous penetration	died
49 / F	thalamic	III	5	hemi-paraesthesias, memory deficit	immediate	perforator occlusion	mild disability at 6 months, back to work
30 / F	corpus callosum	III	5	hemiparesis, IC infarct	immediate	perforator injury by guidewire	complete recovery at 7 months
43 / M	temporal	II	4	seizures, aphasia	12 hours	venous penetration	complete recovery at 12 hours
32 / M	thalamic	III	5	hemiplegia, hemi-sensory deficit	1-6 hours	perforator occlusion	moderate disability, ambulates with a cane
47 / M	temporal	II	5	ICH, IVH, mild hemiparesis	6-24 hours	venous penetration	complete recovery at 72 hours

** = Spetzler-Martin grade; ** = angioarchitecture score; ICH = intracerebral hemorrhage; IVH = intraventricular hemorrhage; IC = internal capsule*

leptomeningeal collateral supply has been less favorable [29].

In this report, we graded the AVMs based on the angioarchitecture and correlated the angioarchitecture score to the angiographic obliteration achieved by embolisation (table 3). We found a statistically significant correlation ($P<0.0001$) between the angioarchitecture grade and the angiographic obliteration. In AVMs with angioarchitecture scores of 0-2, we had a cure rate of 86%, which is better than RS and begins to compare favorably to the high cure rates of surgery.

Analysis of the individual features of the angioarchitecture gives us further insight into which cases could be selected for embolisation (table 4). All five pure AVFs were cured. Fifteen (83%) of the 18 AVMs with a dominant, single, pial arterial feeder were cured by embolisation. Previous reports on embolisation have found a positive correlation between a higher percentage of angiographic obliteration and a smaller number of feeding arteries [15,16]. In Fournier et Al's report, cures were limited to AVMs with supply from only one arterial group (i.e. middle cerebral artery) [15]. In Gobin et Al's series complete occlusion was obtained by embolisation in 33% of AVMs fed by a single artery decreasing in a linear fashion to 3% if there were more than three feeding arteries [16].

Table 4 highlights the steep decline in cure rates in AVMs with angioarchitecture features other than the pure AVFs and the single, pial arterial feeders. In those AVMs with multiple feeding arteries the cure rate was only 11%. The presence of feeding arterial perforators or choroidal supply also correlated with a low cure rate (10%). The cure rate of micro-arteriovenous malformations (< 1 cm) was 47% compared to the low cure rate in 1-3 cm AVMs. There was a minimal difference in the cure rates between those AVMs with a single vein

compared to those with multiple draining veins.

Table 5 shows some correlation between the Spetzler-Martin grade and cure by embolisation with a higher percentage of cures in the lower grades (40% for grade I, 37% for grade II, 10% for grade III). This correlation was much less striking than the correlation between cure rates and angiographic scores (86% in scores 0-2, 34% in scores 3-4, 4% in scores 5-6) (table 3). Even with the pure arteriovenous fistula removed from our analysis there was still a cure rate of 78% (7 of 9 AVMs) in AVMs with angioarchitecture scores of 0-2. In this review, the angioarchitecture score turned out to be a better predictor of angiographic obliteration than the Spetzler-Martin grading system.

In this report embolisation resulted in a 5.0% transient morbidity, 2.5% permanent morbidity and 1.2% mortality (table 5). This degree of morbidity and mortality is similar to the series reported by Valavanis et Al[29]. We had no morbidity or mortality related to embolisation of AVMs with angioarchitecture scores of 0-2. Our complications occurred in AVMs with high angioarchitecture scores (table 6). We had no morbidity or mortality in Spetzler-Martin grade I AVMs and no permanent morbidity in the Spetzler-Martin grade II AVMs. The only permanent morbidity and mortality in this series occurred in three patients with Spetzler-Martin grade III thalamic AVMs (table 6). It is likely that surgical morbidity in Spetzler-Martin grade III thalamic AVMs may be higher than the morbidity quoted for small AVMs in general[8,10,11].

Reports by Gobin et Al and Wikholm et al. on embolisation of brain AVMs had a high percentage (85-90%) of Spetzler-Martin grades III – VI and therefore cure rates by embolisation alone in these series are low (10-13%) [16,20]. Many of these patients were embolised for size reduction before RS and therefore nidus penetration was the goal in most cases. Embolisation in these series resulted in a 1.3 to 1.6% mortality and 6.7 to 7.2% moderate-to-severe morbidity[16,20]. In contrast, presurgical embolisation requires only closure of the feeding arteries. Closure of the feeding pedicle only reduces the risk of embolisation by avoiding venous penetration, which is a risk factor for early post embolisation haemorrhages. Venous penetration of the embolic material accounted for three of our seven complications and two of these were bleeds (table 6). In our 23 patients where the goal was a presurgical embolisation, we had one complication that was related to venous penetration. This complication occurred in 1993. Since that time we have realized that presurgical embolisation should be simple arterial "ligations" to avoid the risk of venous penetration. In addition, our surgeons prefer not to have the embolic material in the nidus since it interferes with their ability to do the dissection.

Limitations of the Study

This series spans one and a half decades and thus reflects a number of philosophies as our management strategy evolved. Twenty-three patients had pre-surgical embolisation targeting specific feeding arteries that were not easily accessible during the operation. Twenty-six patients had pre-radiosurgery embolisation for size reduction. The first four years of this study included 12 patients, whereas 61 patients were treated from 1989 to 1994. From 1995 to 1999, only seven patients with small AVMs were embolised. The increased activity in the second period reflected the burgeoning number of referrals and to some extent the development of flow-guided microcatheters. By 1995 our treatment paradigm shifted towards achieving a predictable cure. Early in our experience we had hoped that partial treatment would have some protective effect against bleeding. In Porter et Al's review of partially treated AVMs the annual bleeding rate after any type of treatment was similar, at approximately 3.4%, comparable to the natural history of AVMs[36].

Our Present Treatment Strategy

Our embolisation results have helped focus our decision-making for management of small AVMs. For AVMs with angioarchitecture scores of 0-2, embolisation has advantages over RS. The cure rate with embolisation is competitive to RS or even better if we compare to RS series with proven angiographic cures[23,25]. Embolisation offers the advantage of immediate protection. Compared to surgery, the cure rates are lower with a similar morbidity[8,10,11]. In small AVMs that are favorable for surgery, surgery is currently our preferred treatment except in the pure arteriovenous fistula where embolisation has proven to be effective.

For AVMs with angioarchitecture scores of 3-6 that are not favorable for surgery due to location or angioarchitecture, targeted embolisation to treat aneurysms can be justified before RS [17]. Presurgical embolisation of small AVMs is recommended in a limited number of patients where specific arteries will not be encountered till late in the dissection.

Conclusions

Embolisation is an effective treatment of small AVMs when the angioarchitecture is favorable (scores 0-2). Favorable angioarchitecture includes the pure fistulas and AVMs with a single, dominant, pial, feeding artery. In surgically accessible small AVMs we currently favor surgery due to its high cure rate and immediate protection. Targeted embolisation has a role in the treatment of aneurysms before surgery or RS. With further experience, better embolic materials, improved catheter technology and the currently available biplane angiographic imaging, we expect that the results from embolisation will improve. Use of the angioarchitecture score in predicting embolisation results needs validation by a prospective study that includes clinical outcome measures.

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References

1 Graf CJ, Perret GE, Torner JC: Bleeding from cerebral arteriovenous malformations as part of their natural history. J Neurosurg 58: 331-37, 1983.
2 Brown RD Jr, Wiebers DO et Al: Frequency of intracranial hemorrhage as a presenting symptom and subtype analysis: a population-based study of intracranial vascular malformations in Olmsted Country, Minnesota. J Neurosurg 85: 29-32, 1996.
3 Brown RD Jr, Wiebers DO et Al: The natural history of unruptured intracranial arteriovenous malformations. J Neurosurg 68: 352-57, 1988.
4 Ondra SL, Troupp H et Al: The natural history of symptomatic arteriovenous malformations of the brain: a 24-year follow-up assessment. J Neurosurg 73: 387-391, 1990.
5 Zakhary R, Williams J et Al: Stereotactic radiosurgery for AVMs: LINAC vs. gamma knife. J Neurosurg 82: 361A, 1995.
6 Porter PJ, terbrugge K et Al: Bleeding from brain arteriovenous malformations: how well do patients recover? Proceedings American Society of Neuroradiology 36th Annual meeting: 140-141, 1999.
7 Hartmann A, Mast H et Al: Morbidity of intracranial hemorrhage in patients with cerebral arteriovenous malformation. Stroke 29: 931-934, 1998.
8 Hamilton MG, Spetzler RF: The prospective application of a grading system for arteriovenous malformations. Neurosurgery 34: 2-6, 1994.
9 Pollock BE, Lunsford LD et Al: Patient outcomes after stereotactic radiosurgery for "operable" arteriovenous malformations. Neurosurgery 35: 1-7, 1994.
10 Schaller C, Schramm J: Microsurgical results for small arteriovenous malformations accessible for radiosurgical or embolization treatment. Neurosurgery 40: 664-672, 1994.
11 Sisti MB, Kader A, Stein BM: Microsurgery for 67 intracranial arteriovenous malformations less than 3 cm in diameter. J Neurosurg 79: 653-60, 1993.
12 Steiner L, Lindquist C et Al: Microsurgery and radiosurgery in brain arteriovenous malformations [editorial]. J Neurosurg 79: 647-52, 1993.
13 Porter PJ, Shin AY et Al: Surgery versus stereotactic radiosurgery for small, operable cerebral arteriovenous malformations: a clinical and cost comparison. Neurosurgery 41: 757-764, 1997.
14 Pikus HJ, Beach ML, Harbaugh RE: Microsurgical treatment of arteriovenous malformations: analysis and comparison with stereotactic radiosurgery. J Neurosurg 88: 641-646, 1998.
15 Fournier D, terBrugge KG et Al: Endovascular treatment of intracerebral arteriovenous malformations: experience in 49 cases. J Neurosurg 75: 228-233, 1991.
16 Gobin YP, Laurent A et Al: Treatment of brain arteriovenous malformations by embolization and radiosurgery. J Neurosurg 85: 19-28, 1996.
17 Hurst RW, Berenstein A et Al: Deep central arteriovenous malformations of the brain: the role of endovascular treatment. J Neurosurg 82: 190-195, 1995.
18 Jafar JJ, Davis AJ et Al: The effect of embolization with N-butyl cyanoacrylate prior to surgical resection of cerebral arteriovenous malformations. J Neurosurg 78: 60-69, 1993.
19 Wikholm G, Lundqvist C, Svendsen P: Transarterial embolization of cerebral arteriovenous malformations: improvement of results with experience. Am J Neuroradiol 16: 1811-1817, 1995.
20 Wikholm G, Lundqvist C, Svendsen P: Embolization of cerebral arteriovenous malformations: Part I - Technique, morphology, and complications. Neurosurgery 39: 448-457, 1996.
21 Friedman WA, Blatt DL et Al: The risk of hemorrhage after radiosurgery for arteriovenous malformations. J Neurosurg 84: 912-919, 1996.
22 Yamamoto M, Jimbo M et Al: Gamma knife radiosurgery for arteriovenous malformations: long-term follow-up results focusing on complications occurring more than 5 years after irradiation. Neurosurgery 38: 906-914, 1996.

23 Pollock BE, Flickinger JC et Al: Factors associated with successful arteriovenous malformation radiosurgery. Neurosurgery 42: 1239-1244, 1998.
24 Colombo F, Pozza F et Al: Linear acceleratorradiosurgery of cerebral arteriovenous malformations: An update. Neurosurgery 34: 14-21, 1994.
25 Friedman WA, Bova FJ, Mendenhall WM: Linear accelerator radiosurgery for arteriovenous malformations: the relationship of size to outcome. J Neurosurg 82: 180-189, 1995.
26 Lunsford LD, Kondziolka D et Al: Stereotactic radiosurgery for arteriovenous malformations of the brain. J Neurosurg 75: 512-524, 1991.
27 Lundqvist C, Wikholm G, Svendsen P: Embolization of cerebral arteriovenous malformations: Part II - Aspects of complications and late outcome. Neurosurgery 39: 460-467, 1996.
28 Wikholm G: Occlusion of cerebral arteriovenous malformations with N-butyl cyano- acrylate is permanent. Am J Neuroradiol 16: 479-482, 1995.
29 Valavanis A, Yasargil MG: The endovascular treatment of brain arteriovenous malformations. Advances and Technical Standards in Neurosurgery 24: 131-214, 1998.
30 Fournier D, terBrugge K et Al: Revascularization of brain arteriovenous malformations after embolization with bucrylate. Neuroradiology 32: 497-501, 1990.
31 Mansmann U, Meisil J et AL: Factors associated with intracranial hemorrhage in cases of cerebral arteriovenous malformation. Neurosurgery 46: 272-279, 2000.
32 Meisel HJ, Mansmann U et AL: Cerebral arteriovenous malformations and associated aneurysms: analysis of 305 cases from a series of 662 patients. Neurosurgery 46: 793-800, 2000.
33 Marks MP, Lane B et Al: Intranidal aneurysms in cerebral arteriovenous malformations: evaluation and endovascular treatment. Radiology 183: 355-360, 1992.
34 Willinsky R, Lasjaunias P et Al: Brain arteriovenous malformations: analysis of the angio-architecture in relationship to hemorrhage (based on 152 patients explored and/or treated at the hopital de Bicetre between 1981 and 1986). J Neuroradiol 15: 225-237, 1988.
35 Redekop G, terBrugge K et Al: Arterial aneurysms associated with cerebral arteriovenous malformations: classification, incidence, and risk of hemorrhage. J Neurosurg 89: 539-546, 1998.
36 Porter PJ, terBrugge K et Al: Hemorrhage rates in untreated and partially treated brain arteriovenous malformations: is Partial therapy protective? Proceedings American Society of Neuroradiology 36th Annual meeting: 92, 1999.

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