

The Combined Management of Cerebral Arteriovenous Malformations Experience with 100 Cases and Review of the Literature

R. Deruty¹, I. Pelissou-Guyotat¹, C. Mottolese¹, Y. Bascoulegue², and D. Amat¹

¹Department of Neurosurgery, ²Department of Neuroradiology, Hôpital Neurologique, Lyon, France

Summary

A series of 100 patients treated for a cerebral arteriovenous malformation (AVM) is presented. Patients were admitted between 1985 and April 1992. Two groups are considered: the first group including 52 patients treated before the availability of radiosurgery (1985–1988), and the second group including 48 patients treated after the availability of radiosurgery (1989–1992). AVM's were classified in five grades according to the Spetzler's Grading System.

Three techniques of treatment were used: surgical resection, intravascular embolization (with cyanoacrylate), and radiosurgery (linear accelerator). These three techniques were used either alone or in association, giving four types of management: surgical resection alone, embolization and resection, embolization alone, and radiosurgery (alone, or after embolization, or after surgical resection).

From 1989 on, the availability of radiosurgery was responsible for the decrease of the "embolization and resection" group, which until then was predominantly used as well for low-grade (I, II, III) as for high-grade AVM's (IV, V). Overall, for the low-grade AVM's, the treatment of choice was surgical resection (79% of cases), with pre-operative embolization in one-half of these cases; the other low-grade AVM's were irradiated, with various combinations. For the high-grade AVM's, the treatment of choice was intravascular embolization (95% of cases), either alone, or followed by resection (45%) or radiosurgery (9%).

Results were evaluated in terms of deterioration following treatment, in five groups: no deterioration (59%), minor deterioration (20%), long-lasting deficit (10%), major deterioration (5%), and death (6%). Overall, results improved after 1989: favourable outcome (no deterioration and minor deterioration) increased from 67% to 90%. Results were not related to the patients' age. More favourable results were obtained for low-grade AVM's (93%) than for high-grade AVM's (60%). For the low-grade AVM's the evolution from 1989 on (favourable outcomes increasing from 89% to 96%) occurred with the lowering of the mortality rate. For the high-grade AVM's, the evolution from 1989 onwards (favourable outcome increasing from 46% to 78%) occurred with the decrease of the cases with deficits.

The angiographic results were strongly related to the management: 95% of complete eradication after surgical resection and 5%

only after embolization alone. Concerning the results in irradiated cases, the follow-up is not long enough.

The review of the neurosurgical literature since 1972 demonstrates progressive modifications in the therapeutic attitude as regards AVM's. The surgical management which was predominantly used at the beginning gave way progressively to a combined management, with a combination of embolization, surgery, and lately radiosurgery. The authors' present attitude is in favour of combined treatment using the three techniques. Direct surgical resection is proposed for small and readily accessible AVM's. Direct radiosurgery is proposed for small but deep AVM's or those located in highly functional areas. Intravascular embolization is proposed in every other situation. After embolization has been completed, totally eradicated AVM's are left in place; no further treatment is proposed for AVM's which are still large with high surgical risk. AVM's which are sufficiently reduced in size are either operated on (if accessible) or irradiated (if deeply situated). When the results of radiosurgery are assessed with long enough follow-up (in terms of eradication and clinical outcome) the authors' attitude may either increase the role of radiosurgery or return towards surgical resection, depending of the quality of the results.

Keywords: Arteriovenous malformations; embolization; radiosurgery.

The therapeutic approach to Cerebral Arteriovenous Malformations (AVM's) has been modified lately, with the availability of new techniques, such as endovascular embolization and radiosurgery. The place of neurosurgery, which was predominant until the early eighties, happens to be reduced to narrower limits, and surgical resection should now be considered as one of several therapeutic methods.

Nowadays the treatment of cerebral AVM's consists of a combination of several techniques. What is the place of each of these techniques, and particularly that of surgical resection, remains to be assessed. In this

paper, we would like to report our experience in the combined management of a series of 100 cases and to study the evolution of ideas in this field, from the neurosurgical literature of the last two decades.

Patients and Techniques

Patients: From 1985 to April 1992, 100 patients harbouring a cerebral AVM were treated in our department. We do not consider in this study the patients whose AVM was only diagnosed but not treated. 1985 is the year during which we were able to use the endovascular embolization techniques. Another year, 1989, is a transition date, for from 1989 on we could use the radiosurgery technique (linear accelerator)*. So, our experience consists of two distinct periods which appear quite comparable as far as the number of patients is concerned: a first period (1985–1988), with 52 patients, and a second period (1989–April 1992) with 48 patients. Overall, our patients having been treated over a 7 years and 4 months period, the average of patients treated per year is 13.6 for the whole series, 13 for the first period (1985–1988), and 14.4 for the second period (1989–1992).

Age and sex: 66% of the patients were male and 33% were female. The age of the patients is shown in Table 1. Three groups were considered: 15–30 years old (young patients), 31–50 years old (middle-aged patients), and 51–65 years old (older patients). When we compare the two periods, we see a tendency towards ageing after 1989: decreasing number for the younger patients, and increasing number for the older patients (twice as many for the 51–65 years old group).

Symptomatology: Signs and symptoms at the time of diagnosis and at the time of treatment are presented in Table 2. Between diagnosis and treatment, some patients happened to bleed or to have a seizure for the first time, which modified the percentages of each group of signs and symptoms. The sub-group “others” includes headache, deficits, or casual discovery.

Table 1. *Patient's Age (Percentages)*

	15–30	31–50	51–65
Overall series	33	52	15
1985–1988	44	46	10
1989–1992	21	57	21

Table 2. *Signs and Symptoms (Percentages)*

	Haemorrhage	Epilepsy	Others
At the time of diagnosis	38	38	24
At the time of treatment	41	39	20

* Department of Radiotherapy. Prof. J. P. Gerard, Centre Hospitalier Lyon Sud, Lyon, France.

Site of AVM's: The AVM location is presented in Table 3. We have a large majority of hemispheric AVM's, and a greater number on the side of the dominant hemisphere than on the minor hemisphere.

Grading of AVM's: In this series, AVM's were graded according to the Spetzler's grading system²¹, with three variables: size of AVM, eloquence of brain area, venous drainage. The distribution of AVM's in 5 grades is given in Table 4. From 1989 onwards, we see the disappearance of grade V patients. This modification is probably due to the fact that, after 1985, with the availability of endovascular embolization, many of the grade V patients who had not yet been selected for treatment during the previous years were readmitted to undergo an embolization of their AVM, and so were numerous patients in the first period of our study. To simplify the study, we have considered together the low-grade AVM's (I, II, III) and the high-grade AVM's (IV, V), for the therapeutic problems are comparable for the AVM's included respectively in each of these two sub-groups. This simplified classification is presented in Table 5.

Table 3. *AVM Site (Percentages)*

	Major hemisphere	Minor hemisphere
Prefrontal	4	2
Fronto-Rolandic	17	17
Parietal	8	3
Occipital	5	3
Temporal	15	5
Basal ganglia	6	5
Splenium of c.c.	1	
Posterior fossa	9	
	55	35
That is:		
hemispheric	= 91%	
posterior fossa	= 9%	

Table 4. *Classification of AVM's (Spetzler Grading System) (Percentages)*

	I	II	III	IV	V
Overall	2	28	26	35	9
1985–1988	0	33	17	33	17
1989–1992	4	23	33	40	0

Table 5. *Simplified Classification of AVM's. Low grades and high grades (percentages)*

	I, II, III	IV, V
Overall	56	44
1985–1988	50	50
1989–1992	60	40

Techniques of treatment: Three techniques of treatment were used: surgical resection, endovascular embolization (cyanoacrylate), and after 1989 radiosurgery (linear accelerator). The overall utilization of each of these techniques is presented in Table 6.

AVM management: The three techniques above described were used either alone or in various combinations. So four therapeutic schemes may be described: 1) surgical resection alone, 2) embolization plus surgical resection, 3) embolization alone, 4) radiosurgery (either alone, or after embolization, or after incomplete surgical resection). The actual management is presented in Tables 7, 8, and 9. Radiosurgery cases appear only during the period 1989–1992, as this technique was not available beforehand in our department. For the low-grade AVM's the treatment of choice was surgical resection (79% of cases), performed in half of the cases after endovascular embolization; the remaining cases were treated by radiosurgery, with various combinations. For the high-grade AVM's, the treatment of choice was endovascular embolization (95% of cases) either alone, or followed by surgical resection (45% of cases) or sometimes by radiosurgery (9%). From 1989 on, the availability of radiosurgery modified our therapeutic approach: the sub-group "Embolization + Surgical resection" greatly decreased for the low-grade AVM's (from 59 to 21%) and for the high-grade AVM's as well

Table 6. *Techniques of Treatment*

Surgical resection	65%
Embolization	71%
Radiosurgery	14%

Table 7. *Overall Series.* Management according to the grading (percentages)

	I, II, III	IV, V
Surgical resection	38	2
Embolization – resection	39	45
Embolization alone	7	41
Radiosurgery	16	11
alone	9	2
after embolization	5	9
after resection	2	0

Table 8. *Grades I, II, III.* Management during the two periods (percentages)

	1985–1988	1989–1992
Surgical resection alone	30	45
Embolization – resection	59	21
Embolization alone	11	3
Radiosurgery		30
alone		17
after embolization		10
after resection		3

Table 9. *Grades IV, V.* Management during the two periods (percentages)

	1985–1988	1989–1992
Surgical resection alone	4	0
Embolization-resection	58	28
Embolization alone	38	44
Radiosurgery		28
alone		6
after embolization		22
after resection		0

(from 58 to 28%). For the low-grade AVM's this modification occurred to a lesser extent in favour of direct surgical resection (from 30 to 45%) but to a greater extent in favour of radiosurgical cases (up to 30% of cases). For the high-grade AVM's, this modification gave benefit partly to the "embolization alone" group (which increased from 38 to 44%) but particularly to the "radiosurgery" group (28%).

Results

Outcome Evaluation

Results were evaluated in terms of deterioration following treatment, in five groups: 1) No deterioration, 2) Minor deterioration (hemianopia, minor aphasia, minor hemiparesis . . .), 3) Long-lasting deficit created by treatment (hemiplegia, major aphasia), 4) Major deterioration (patients being dependent for self care), and 5) death. The sub-group "long-lasting deficit" seemed justified as it includes patients who, being hemiplegic, cannot be classified in the "minor deterioration" group, but, being independent in the daily life and sometimes being able to work, cannot be classified in the "major deterioration" group. Schematically these 5 groups can be considered in a simpler fashion in 3 categories: 1) favourable outcome (no or minor deterioration), 2) Long-lasting deficit, 3) unfavourable outcome (major deterioration and death). Such a simplification may render the results more understandable.

Outcome According to the Various Parameters

Results are presented in Tables 10 to 16. Overall, results improved after 1989: favourable outcome increasing from 67 to 90%, with a decrease of both the long-lasting deficits (from 15 to 4%) and the unfavourable results (from 18 to 6%). Concerning the age of the patients, no difference was found between the younger patients (15–30 years) and the middle-aged patients (31–50 years). Better results were seemingly

Table 10. *Overall Series*. Outcome in terms of deterioration

No deterioration	59%
Minor deterioration	20%
Long-lasting deficit	10%
Major deterioration	5%
Death	6%
That is:	
favourable	79%
deficit	10%
unfavourable	11%

Table 11. *Outcome During the Two Periods (Percentages)*

	1985–1988	1989–1992
No deterioration	50	69
Minor aggravation	17	21
Long-lasting deficit	15	4
Major deterioration	10	2
Death	8	4
That is:		
favourable	67	90
deficit	15	4
unfavourable	18	6

Table 12. *Outcome According to the Age (Percentages)*

	15–30	31–50	51–65
No deterioration	58	57	73
Minor deterioration	15	20	20
Long-lasting deficit	5	10	–
Major aggravation	3	10	–
Death	9	4	7
That is:			
favourable	73	77	93
deficit	15	10	–
unfavourable	12	14	7

Table 13. *Outcome According to the Grade (Percentages)*

	I-II-III	IV-V
No deterioration	75	38
Minor deterioration	18	22
Long-lasting deficit	4	18
Major deterioration	–	13
Death	4	9
That is:		
favourable	93	60
deficit	4	18
unfavourable	4	22

obtained in the oldest patients (51–65 years), but with probably a bias due to patient selection and the proposed management. Results are better for the low-grade AVM's (93% favourable) than for the high-grade ones (60% favourable). In the high-grade AVM's, the rate

Table 14. *Grades I, II, III*. Outcome during the two periods (percentages)

	1985–1988	1989–1992
No deterioration	77	72
Minor deterioration	12	24
Long-lasting deficit	4	3
Major deterioration	–	–
Death	8	–
That is:		
favourable	89	96
deficit	4	3
unfavourable	8	0

Table 15. *Grades IV, V*. Outcome during the two periods (percentages)

	1985–1988	1989–1992
No deterioration	23	61
Minor deterioration	23	17
Long-lasting deficit	27	6
Major deterioration	19	6
Death	8	11
That is:		
favourable	46	78
deficit	27	6
unfavourable	27	17

Table 16. *Outcome and Management (Percentages)*

	Resection alone	Embolization resection	Embolization alone
No deterioration	82	48	45
Minor deterioration	18	24	9
Long-lasting deficit	–	12	18
Major deterioration	–	10	14
Death	–	7	14
That is:			
favourable	100	72	54
deficit		12	18
unfavourable		17	28

Follow-up too short for the group "radiosurgery". No deterioration 86%, minor deterioration 14%.

Table 17. *Eradication and Management (Percentages)*

	Resection alone	Embolization resection	Embolization alone
Total eradication	95	93	5
Partial eradication	5	7	95

Follow-up too short for the radiosurgery group. Total eradication 14%, partial eradication 86%.

Table 18. *Eradication and AVM Grade (Percentages)*

	I, II, III	IV, V
Total eradication	78	45
Partial eradication	7	45
Insufficient follow-up	15	9

of deficits and major deterioration is relatively high (18 and 13%). From 1989 onwards, the modification of outcome for the low-grade AVM's is clear (from 89 to 96% favourable), essentially due to the lowering of mortality rate. For the high grade AVM's the modification of outcome after 1989 is also obvious (from 46 to 78% favourable), mainly due to the decreasing of the deficit rate (from 27 to 6%). Concerning the type of management, the rate of favourable outcome decreases from the "resection alone" group (100%), to the "embolization resection" group (72%), and to the "embolization alone" group (54%). The latter group actually includes the less accessible AVM's and all the complications of the embolization.*

AVM Eradication

The AVM eradication may be complete or partial. The results in terms of eradication are presented in Tables 17 and 18. The rate of eradication is obviously satisfactory after an AVM resection, either alone or with pre-operative embolization. Conversely the rate of eradication is very low (5%) after embolization alone. According to the AVM grade, the rate of eradication is obviously more satisfactory in the low-grade group (78%) than in the high-grade group (45%).

Discussion

The review of the *neurosurgical literature* during the last twenty years demonstrates a progressive evolution in the therapeutic attitude as regards AVM's.

Surgery was once predominantly used. Several *consecutive surgical series* report the treatment of AVM's whatever be the location¹⁻²⁷. Length of treatment and observation in these series is long, from 8 to 35 years: (from 8 to 10 years for 5 series, from 11 to 20 years for 9 series, and from 21 to 35 years for 3 series). The number of patients reported per series ranges from 50 to 414 (from 50 to 100 for 7 series, from 101 to 200 for 8 series, and over 200 patients for 3 series only). However, whatever be the duration of the study and the number of patients, the average of patients reported per year remains low: from 3 to 5 patients per year (4 series), from 6 to 10 patients (8 series), from 11 to 15 (3 series), and from 16 to 20 (2 series only).

Concerning the type of management, 2 out of 3 of these series report both treated and untreated patients; the percentage of untreated patients may vary from 10 to 20% (6 series), from 20 to 40% (5 series), and even to 50% (1 series). The reason for conservative treatment rather than surgery is sometimes described^{1, 12}: first the operative risk, then the condition of the patient, the patient's refusal, and the insufficient symptomatology.

The *outcome* is variously evaluated: several series consider the classical evaluation with so-called Good, Fair, Poor Result, Death. However this evaluation does not take into account the previous state of the patients. Other series consider the existence of a post-operative deficit¹⁹, the working capacity¹⁵, the improvement or the deterioration after surgery^{12, 25}. Some authors^{4, 22} try to adapt these various evaluations, and respectively consider them together: Good Result and No Deterioration, Fair Result and Minor Deterioration, Poor Result and Major Deterioration. In such an evaluation one group seems to be missing: the group of those patients that Nornes¹⁵ calls "unable to work but independent"; actually these patients have a deficit but are independent for daily life and sometimes are able to work. In our own results we have considered this group under the denomination of "long-lasting deficit". When we try to evaluate the results reported in the consecutive series in the literature according to the above mentioned scale, we obtain the following figures: 1) Good (No Deterioration) 52 to 82%; 2) Fair (Minor Deterioration) 4 to 29%, 3) Poor (Major Deterioration) 2 to 8%, 4) Death 1 to 14%. To simplify these figures, we can consider a) Favourable outcome (1 + 2): 81 to 95% and b) Unfavourable outcome (3 + 4): 5 to 19%. The mortality varies from 1 to 5% (7 series), from 6 to 10% (3 series), and from 11 to 14% (6 series). The post-operative deficit may be important at first but is likely to improve later on^{10, 22} in 13 to 17% of patients.

The effect of surgical resection on *Epilepsy* is often studied. In cases with pre-operative seizures, epilepsy is reported to be cured in only 2 to 4% of cases, and to be unchanged in 11 to 33% of cases (average 25%); several series consider the results as "cured or improved" altogether, with very extreme figures varying from 4 to 89% (average 51%). In cases with no pre-operative seizures, surgery may precipitate epilepsy in 7 to 29% of cases (average 16%). Several authors discuss the indications for surgery on AVM's presenting with epilepsy. Surgery is sometimes considered as very effective for seizures²² or conversely as completely ineffective^{2,7,16}. For some authors, surgery is indicated only in cases with intractable epilepsy^{4,11,25} provided the AVM be readily accessible without excessive surgical risk. Davis⁴ proposes the following scheme for indications for surgery: considering that intractable epilepsy is sometimes cured, that moderate epilepsy is not always cured, and that surgery may precipitate epilepsy, surgery in AVM presenting with seizures is indicated only for epilepsy related to the temporal lobe; in other locations, the AVM resection is less justified, unless this AVM is readily accessible. A study has been especially devoted to the outcome regarding epilepsy in AVM cases²⁷ and demonstrates that a complete cure of epilepsy may be achieved in 70% of cases provided that surgery is directed not only towards the resection of the AVM but also towards the removal of epileptic foci remote from the AVM area.

As far as other symptoms are concerned, *haemorrhage* is, for every author, the primary indication provided the resection of the AVM is complete. Everybody agrees that partial resection does not protect against the risk of fresh haemorrhage.

For the other signs and symptoms, opinions may vary concerning the indications for resection of the AVM. The *ischaemic deficit* is described as reversible after surgery², or with little modification¹² or even increasing in large malformations¹³. The existence of a pre-operative deficit is often described as an unfavourable prognostic factor: for Drake⁵, the outcome is favourable in 99% of cases without deficit but only favourable in 81% in cases with deficit. Headache is usually little modified¹² or is a poor indication⁴. *Disturbances of consciousness* may be described as a good indication¹⁶ for they are due to the presence of haematoma. However for the majority of authors^{4,9,22,23}, these disturbances are considered as an unfavourable prognostic factor, and should favour a leaning conservative management. For Drake⁵ and Sundt²² the

outcome is favourable in respectively 99% and 89% of cases without disturbances of consciousness, and only in 76% and 36% of cases with disturbances of consciousness. Due to the prognostic value of the level of consciousness, the timing of surgery is sometimes discussed^{15,23} and late surgery is proposed, after improvement of the acute symptomatology.

Overall, *the indication for surgical resection* in AVM patients is widely discussed. During the seventies, the schemes for indications for surgery were very aggressive^{2,14,17}: every AVM should be operated on, whatever the situation (even deeply located) or the symptoms (even epilepsy alone). Mingrino¹⁴ however considers some contraindications, related either to the size (diffuse AVM's) or the location (basal ganglia); in later years, some contraindications are progressively recognized. The *patient's age* is the first factor of restriction: admittedly a greater surgical risk may be accepted in younger patients, in whom the spontaneous AVM risk is higher; conversely in older patients conservative management is more and more considered, as the surgical prognosis is less favourable^{18,23}, the spontaneous risk of AVM is lower, especially in cases with no haemorrhage¹², and surgery actually gives little benefit to the life expectancy⁹. The *anatomy of the AVM* is the second limiting factor, particularly the size and the location. The indication for surgical resection is usually accepted for the small malformations, in which the spontaneous risk exceeds the surgical risk¹³ and in which good results may be obtained even in functional areas¹¹; conversely, conservative management is more and more discussed for large AVM's, in which the surgical risk exceeds the natural risk after 40 or 50 years of age^{4,10,11}. According to Herniesnemi¹¹, large AVM's located in central, thalamic, and mesencephalic areas, are probably inoperable with the techniques available in 1990. Eventually, in the eighties, more realistic schemes of indications for surgery are proposed. Guidetti⁹ discusses first conservative management in the following situations: epilepsy associated with a large AVM and well controlled with antiepileptic drugs, elderly patients, patients presenting with disturbances of consciousness; in every other situation, surgery is indicated to prevent the haemorrhagic risk. Luessenhop¹³ considers surgery in small AVM's (diameter under 4 cm) in which the spontaneous risk is higher than the surgical risk, and recommends conservative management in large AVM's (over 4 cm in diameter) in which the surgical risk after the fifth or sixth decade exceeds the natural risk. Furthermore, an ischaemic deficit or

epilepsy associated with such a large AVM is a contra-indication, as the post-operative deficit will probably be greater than the deficit due to the natural history; overall for Luessenhop, the surgical risk is better than the natural risk in about 65 to 70% of all cases.

Two studies deal with a *decision analysis*^{3, 6} concerning unruptured AVM's the aim of these studies being to compare the respective benefit of either surgical resection or conservative management. In Fisher's study, the parameters are not only the patient's age, the natural history of unruptured AVM's, but also the results obtained by the neurosurgeon and the patient's wishes. The conclusions are as follows: in a young patient (from 25 to 35 years), in order to obtain an effective surgical benefit, the outcome should stay inside the limits of 5.5% for the mortality and 9% for morbidity; in an older patient (55 years) the respective limits are 2% and 10%. Beyond these figures, no surgical benefit is given to the patient. Auger's conclusions are similar: given the present standards of surgical results (as published in the literature), the resection of an AVM with a size and a location favourable is justified up to the age of 44 years; surgery in older patients (up to 60 years) would not be justified without an improvement of the present standards, in terms of mortality and morbidity.

More and more papers deal with *embolization and combined treatments* (combining embolization and surgical resection)²⁸⁻⁷². However, the study usually consists of a limited number of patients: less than 50 patients for most of the series, 4 series include respectively 50 to 136 patients^{30, 52, 61, 71}. Four techniques of embolization are proposed: the intra-operative embolization of cervical arteries, the intra-operative embolization of intracranial arteries, the endovascular embolization through femoral catheterisation, and the intra-operative arterial occlusion with a balloon, either temporarily or permanently. This latter technique described by Suzuki⁶⁹ and used also by Halbach⁴⁵ actually does not seem very often utilized, compared to the other three. There is a general agreement to acknowledge that Luessenhop was the first to describe the embolization technique in the management of cerebral AVM's, from 1960 onwards^{50, 51}. The technique of Luessenhop was an *intra-operative embolization* of the cervical arteries (Common, internal and external carotid arteries, sub-clavian artery): the artery was dissected in the neck and embolized with silastic spheres. Several authors used this technique of Luessenhop later on^{49, 68}. George⁴³ described a similar method to em-

bolize the vertebral artery at the C1-C2 level. The intra-operative embolization of the intracranial arteries which is the second possibility was described during the early eighties^{30, 31, 33-36, 61, 62, 65-67, 72}; in this technique, the feeders of the AVM are approached through a bone flap, they are dissected, catheterized, and injected either with a polymer (cyanoacrylate) or with particles (Ivalon). Per-operative angiography is performed to control the filling of the AVM. However, at the present time, most authors favour *pre-operative embolization* by femoral catheterization^{30, 32, 33, 39, 40, 41, 45, 61, 64-67, 70, 71}. Historically many embolizing substances were advocated and used: detachable balloons, particles (Silastic, Latex, PVA), polymers (cyanoacrylate IBCA or NBCA, EVAL), metallic coils, threads. Many questions arise and are discussed concerning the embolization technique: when the embolization should be stopped^{56, 68}? What is the toxicity of the various embolizing materials^{59, 61}? The possibility of *revascularization* of the AVM after embolization is often discussed. Such re-irrigation may occur through a collateral blood supply, when the occlusion of the nidus was not complete^{32, 39, 46, 52, 68}, and so the conclusion arises that the embolization should concern the nidus itself and not only the feeders; Debrun³³ and Purdy⁶¹ point out that the detachable balloons are not able to achieve this goal, and that a tissue adhesive or particles should be used for embolization of an AVM. However even in those cases in which the nidus itself was properly occluded, a canalisation of this nidus may occur, such as is described in cases with cyanoacrylate occlusion^{35, 36, 48} or in cases with PVA particles occlusion⁴⁴. In some cases, the pathological examination of the resected AVM demonstrated the presence, inside the occluded nidus, of newly formed capillaries and normal red blood cells; such a repermeation is described to take place early, within 4 weeks after embolization, in about 18% of cases⁴⁴. The *extension of embolization* is often considered. In most cases the occlusion of the nidus after embolization is only partial. The occlusion is described as complete in only 0 to 17% of cases^{33, 39, 49, 52}. When the occlusion is partial, as most often acknowledged, the shrinkage rate of the nidus may vary inside wide limits: sometimes the reduction rate is low, with an average of 37% for Pasqualin⁵⁸, sometimes the rate is higher: from 50 to 75% in $\frac{2}{3}$ of the cases, and from 75 to 90% in $\frac{1}{3}$ of the cases for Vinuela⁷¹. However, for Fournier³⁹ the occlusion rate cannot reach over 60% when the AVM nidus is supplied through more than 2 arterial groups. The *complications of the*

embolization technique are often reported⁵⁴ and may be of ischaemic origin (proximal arterial thrombosis, spasm, embolus), or due to haemorrhage (rupture of a residual nidus, associated aneurysm). The incidence of these complications is not easy to assess: haemorrhage is reported in 7 to 11% of cases^{29,59}. Fournier³⁹ reports permanent complications in 8% of cases, and transient complications in 16%. Vinuela⁷¹ describes the following complications: minor and moderate 11%, severe and death 3%. And recently Benati reports 7% of transient deficits, 7% of permanent deficits, and 1.4% of death. The embolization technique may be used either alone or combined with other techniques of treatment. Out of 28 series, the embolization technique was used as sole method of treatment in five series (18%), was combined with surgical resection in 20 series (71%), and combined with surgery or radiosurgery three times (11%). The *embolization alone* was used either in every case^{49,57} or only in malformations thought as "inoperable"^{30,31,33,35,36,38,52,53}. The outcome of such an isolated treatment with embolization are variously reported. Kusske⁴⁹ and Luessenhop⁵² reported an improvement of deficits, of seizures (in 40% of cases) and of headache. Benati³⁰ found also an improvement in the frequency and severity of headache, a moderate effect on the frequency of seizures, and a poor result on the progression of neurological deficit. However, the main point in this discussion is the prevention of haemorrhage: Is the embolization technique, used as sole treatment, able to prevent the rupture, or the re-rupture, of the AVM? Possibly for some authors³¹, certainly not for others^{35,36,49}, and in any case, a very long follow-up will be necessary to confirm the opinions^{33,56}. Actually, the haemorrhagic risk after embolization alone has been studied. Luessenhop⁵² reported a low risk (5% within 4 years) for the AVM's without previous haemorrhage, and a high risk (50% within 4 years) for the AVM's having already bled. Fournier³⁹, with a follow-up of 2 to 6 years, and Benati³⁰ with a follow-up from 4 to 7 years, found that after embolization, the risk rate is identical to the risk rate of the natural history.

In most papers however, *the embolization technique is combined with the surgical resection* of the AVM^{29,30,33,35-37,40,41,46,48,54,57,61,65-68,71}. Several ideas gave way to this combined management: embolization does not protect against the recurrence of haemorrhage; a complete occlusion of the nidus by means of embolization alone may be obtained in AVM's of low-grade with few feeders^{33,39}, but usually is not obtained in

high grade malformations fed by numerous arteries. But these low-grade AVM's have a low surgical risk which is not improved by the embolization technique⁷¹ and Luessenhop⁵⁴ states that the best indications for embolization are also the best indications for surgery. Eventually^{39,54} the main role of the embolization technique should be the conversion of a large and inoperable AVM into a smaller AVM, accessible either to surgery, or to radiosurgery. In any case, a multidisciplinary approach appears indispensable^{42,60,71}, with cooperation between the various specialists dealing with the management of AVM's (Neurosurgeon, Neurologist, Neuroradiologist, Radiotherapist).

Various schemes of combined management have been proposed for the treatment of AVM's. The Spetzler's scheme^{66,67} was designed to occlude and resect the high-grade AVM's: Endovascular embolization of feeders coming from the external carotid artery, intra-operative embolization of feeders coming from the internal carotid artery, then surgical resection, in several stages if necessary. Albert's scheme²⁸ is based upon the size and the location of the AVM. Small and middle-size AVM's in non-functional areas are operated upon, as radiosurgery does not protect against haemorrhage during the first year; small and middle size AVM's in highly functional areas are proposed for embolization or radiosurgery; the large AVM's with numerous draining veins are not an indication for direct surgery (their spontaneous haemorrhagic risk is low); radiosurgery for these large AVM's is, at the present time ineffective; so these large AVM's should be embolized, in order to make them accessible either to resection or to radiosurgery. Recently, Gentili⁴² and Benati³⁰ proposed very similar schemes. Overall for Luessenhop⁵³ 50% of AVM's are accessible to direct surgical resection, 25% can be treated with embolization or combined management, and for the remaining 25%, no treatment is, of now, strictly established. Vinuela⁷¹ advises endovascular embolization rather than intra-operative embolization, to avoid multiple craniotomies.

The *interval between embolization and surgery* is still under discussion: either a short interval, within 24 hours⁴⁶, or several days to several weeks⁵². For Stein⁶⁸ and Pasqualin⁵⁸ an interval of a few days is too short and as it favours the complications; a long interval (several weeks) may give way to the development of a collateral blood supply; the ideal interval would be from 1 to 2 weeks. Is an embolized AVM easier or more difficult to resect? Various opinions have been expressed: the surgical resection was described as more

difficult when the AVM has been transformed into a hard mass by the cyanoacrylate^{61, 62, 66, 67}. Conversely, the resection was also described as easier under the same circumstances^{35, 36, 71}, the hardness of the tumour-like AVM being not a disadvantage, but on the contrary an advantage. Other authors^{58, 68} without considering the nature of the embolizing material, found the AVM resection easier after embolization.

Results of the combined treatment are sometimes difficult to assess from the published series. It is not easy to find what are the complications either of the embolization procedures, or of the surgical resection, or of both. Overall, the following figures can be accepted: mortality between 0 and 6%, major morbidity between 2 and 11%^{29, 35, 36, 39, 68, 71}. Originally, the aim of these combined treatments was to suppress or to diminish the complications (Breakthrough theory^{66, 67}). Is this aim achieved? The answer is yes for some authors^{63, 66, 67}. For others, this goal is only partially reached: Pasqualin⁵⁸ noted a lower rate of intra-operative haemorrhages, a lower rate of post-operative deficits, but the same rate of post-operative haemorrhages. And for some authors, the answer is categorically "No"^{47, 56}: for these authors, combined and multiple treatments are dangerous for several reasons: they increase the ischaemic risk (adding an acute ischaemia to the chronic ischaemia due to the AVM); the optimal chronology between the various procedures is not really known and the haemorrhagic risk may be higher in between; last of all, the surgical difficulties are increased, as the delay between the various procedures may favour the development of a collateral blood supply, particularly where there are deep feeders.

Radiosurgery is the most recent technique applied to the treatment of cerebral AVM's. Radiosurgery may be used either alone or combined with other techniques (embolization and surgery). Publications in the neurosurgical literature are still small in number⁷³⁻⁸⁷ since Steiner's initial paper⁸³. Recently, Steiner⁸⁴⁻⁸⁵ summarized his experience with the Gamma-Knife technique, reporting 247 cases over a 14 years period, 94% of whom having suffered with at least one haemorrhage. He studied the protective effect against haemorrhage in incompletely obliterated AVM's and found 1) with the person/year method, a risk identical to that of the natural history (2-3% per year) and 2) with the Kaplan-Meier life-table a risk of 3.7% until 60 months after radiosurgery. An overview was established in 1990 by Ogilvy⁸² who compared the outcome of the various techniques of radiosurgery (Gamma-Knife, Linear Ac-

celerator, Heavy Particles): all techniques included and with a follow-up of 2 years, the haemorrhagic risk after radiosurgery was from 2 to 2.6% per year (similar to the risk of the natural history); the risk of deficit after irradiation was 1.7% to 4% taking all techniques together, 2 to 3% for the Gamma-Knife and 3% for the Linear Accelerator. Ogilvy concluded that 1) the efficacy of radiosurgery is proven; 2) There are failures with incomplete occlusion of the nidus and then subsequent haemorrhages; 3) Complete angiographic destruction occurs for small malformations (from 2.5 to 3 cm in diameter), but for the large AVM's the technique is still under evaluation; 4) For the present time radiosurgery should be reserved for truly inoperable malformations and requires excellent collaboration between neurosurgeons and radiotherapists.

In our personal experience, the advent of radiosurgery, in 1989, definitely modified our therapeutic approach for cerebral AVM's, as has been outlined above under "AVM management".

The overall improvement of our results is clear, as the favourable outcome rate increased from 67% to 90% all grades included, from 89% to 96% for the low-grade AVM's, and from 46% to 78% for the high-grade group. Such an improvement is for us a reason to maintain our attitude. In our approach *our management at the present time* is not far from that proposed by Albert²⁹ and Gentili⁴²: the small and readily accessible AVM's are directly operated upon. The small and deeply situated AVM's are directly irradiated. In every other case, we propose first endovascular embolization, with as many procedures as it is necessary to reduce the nidus. The interval between each embolization sitting may vary according to the size of the AVM, the disposition of the patient, the possible onset of a complication; the longer the interval, the greater the risk of development of new collateral channels. The usual interval is 1 or 2 weeks, but in some very large AVM's the embolization procedures may be performed over several months. After the embolization has been completed to its maximum, three situations may occur: 1) the AVM is completely obliterated (this eventuality is infrequent, 5% of cases in our experience); we do not usually propose surgical resection, although we are aware of the possibility of further recanalisation of the nidus. 2) The AVM is still too large to be irradiated (more than 3 cm in diameter), and the surgical resection still appears hazardous; we stop the therapeutic procedures, being well aware of the persistent haemorrhagic risk. 3) The AVM nidus is sufficiently reduced

to allow a further therapeutic approach: for accessible malformations, the choice is between radiosurgery (with a diameter less than 3 centimeters) and direct surgical resection (more and more patients, nowadays, are asking for radiosurgery); for deep AVM's we propose radiosurgery.

So, overall we favour now the radiosurgery technique rather than surgical resection, either on the basis of the patient's wishes, or with the idea of an evaluation of the method. So far, our follow-up is quite inadequate and it is not possible for us to evaluate either the clinical outcome (in terms of deterioration), or the angiographic eradication rate. In the future, should the evaluation of the radiosurgery technique be positive according to these criteria (clinical and angiographic outcome), we will maintain this approach. Conversely, should we see the occurrence of complications either radionecrosis or subsequent haemorrhage of an unacceptable rate, or should the angiographic eradication rate remain low, we will return to surgery, the place of which will then appear less limited.

Acknowledgements

The irradiation of our cases with AVM is performed in the department of Radiotherapy, by Prof. J. P. Gerard[†] and members of his team. We thank them for the quality of their collaboration.

References

Consecutive Surgical Series

1. Albert P (1982) Personal experience in the treatment of 178 cases of AVM's of the Brain. *Acta Neurochir (Wien)* 61: 207–226
2. Amacher AL, Allock JM, Drake CG (1972) Cerebral angiomata. Sequellae of surgical treatment. *J Neurosurg* 37: 571–575
3. Auger RG, Wiebers DO (1992) Management of unruptured intracranial arteriovenous malformations: A decision analysis. *Neurosurgery* 30: 561–569
4. Davis Ch, Symon L (1985) The management of cerebral AVM's. *Acta Neurochir (Wien)* 74: 4–11
5. Drake CG (1978) Cerebral AVM's. Consideration for and experience with 166 cases. *Clin Neurosurg* 26: 145–207
6. Fisher WS III (1989) Decision analysis. A tool of the future. An application to unruptured AVM's. *Neurosurgery* 24: 129–135
7. Forster DM, Steiner L, Hakanson S (1972) AVM's of the brain. Longterm clinical study. *J Neurosurg* 37: 562
8. French LA (1976) Surgical treatment of AVM's. A history. *Clin Neurosurg* 24: 22–33
9. Guidetti B, Delitala A (1980) Intracranial AVM's. Conservative and surgical treatment. *J Neurosurg* 53: 149–152
10. Heros RC, Korosue K, Diebold PM (1990) Surgical excision of cerebral arteriovenous malformations: Late results. *Neurosurgery* 26: 570–578
11. Hernesniemi J, Keranen T (1990) Microsurgical treatment of AVM's of the brain in defined population. *Surg Neurol* 33: 384–390

12. Jomin M, Lesoin F, Lozes G (1985) Prognosis for AVM's of the brain in adults based on 150 cases. *Surg Neurol* 23: 362–367
13. Luessenhop AJ, Rosa L (1984) Cerebral AVM's. Indication and results for surgery and the role of intravascular techniques. *J Neurosurg* 60: 14–22
14. Mingrino S (1978) Supratentorial AVM's of the brain. In: Krayenbühl H (ed) *Advances and technical standards in neurosurgery*, Vol 5. Springer, Wien New York, pp 93–126
15. Nornes H, Lundar T, Wikeby P (1979) Cerebral AVM's. Results of microsurgical management. *Acta Neurochir (Wien)* 50: 243–258
16. * Parkinson D, Bachers G (1980) AVM's. Summary of 100 consecutive supratentorial case *J Neurosurg* 53: 285–299
17. Patterson RH Jr, Voorhies RM (1977) Surgical approaches to intracranial and intraspinal AVM's. *Clin Neurosurg* 25: 412–423
18. * Pelletieri L, Carlsson CA, Grevstens, Norlen G, Uhlemann C (1980) Surgical versus conservative treatment of intracranial arteriovenous malformations. A study in surgical decision making. *Acta Neurochir (Wien)* [Suppl] 29: 1–86
19. * Pertuiset B, Ancrì D, Sichez JP, Chauvin M, Guilly E, Metzger J, Gardeur D, Basset JY (1983) Radical surgery in cerebral AVM's. Tactical procedure based upon haemodynamic factors. In: Krayenbühl H (ed) *Advances and technical standards in neurosurgery*, Vol 10. Springer, Wien New York, pp 81–146
20. * Pertuiset B, Ancrì D, Kinuia Y, Haisa T, Bordini L, Lin C, Makdi M, Arthuis F (1991) Classification of supratentorial arteriovenous malformations. A score system for evaluation of operability and surgical strategy. Based on an analysis of 66 cases. *Acta Neurochir (Wien)* 110: 6–16
21. * Spetzler RF, Martin NA (1986) A proposed grading for AVM's. *J Neurosurg* 65: 476–483
22. Sundt ThM, Piepgras DG, Stevens LN (1989) Surgery for supratentorial AVM's. *Clin Neurosurg* 37: 49–115
23. * Tamaki N, Ehara K, Lin Tin-Kai, Kuwamura K, Obora Y, Kanazawa Y, Yamashita H, Matsumoto S (1991) Cerebral arteriovenous malformations: Factors influencing the surgical difficulty and outcome. *Neurosurgery* 29: 856–863
24. Trumpy JH, Eldevik P (1977) Intracranial AVM's. Conservative or surgical treatment. *Surg Neurol* 8: 171–175
25. Wilson CB, U HS, Domingue J (1979) Microsurgical treatment of intracranial vascular malformations. *J Neurosurg* 51: 446–454
26. Yaşargil MG (1988) *Microneurosurgery*, Vol IIIB. AVM of the brain. Summary of operative results. Thieme, Stuttgart, pp 369–395
27. Yeh HS, Kashiwai S, Tew JM jr, Berger TS (1990) Surgical management of epilepsy associated with cerebral AVM's. *J Neurosurg* 72: 216–223

Embolization: Combined Treatments

28. Albert P, Salgado H, Polaina M, Trujillo F, Ponce De Leon A, Durand F (1990) A study of the venous drainage of 150 cerebral AVM's as related to haemorrhage risks and size of the lesion. *Acta Neurochir (Wien)* 103: 30–34
29. Andrew BT, Wilson CB (1987) Staged treatment of AVM's of the brain. *Neurosurgery* 21: 314–323
30. Benati A (1992) Interventional neuroradiology for the treatment of inaccessible arteriovenous malformations. *Acta Neurochir (Wien)* 118: 76–79

31. Cromwell LD, Harris AB (1980) Treatment of cerebral AVM's. A combined neurosurgical and neuroradiological approach. *J Neurosurg* 52: 705-708
32. Debrun G, Lacour P, Caron JP, Hurth M, Comoy J, Keravel Y (1978) Detachable balloon and calibrated leak balloon techniques in the treatment of cerebral vascular lesions. *J Neurosurg* 49: 635-649
33. Debrun G, Vinuela F, Fox A, Drake CG (1982) Embolization of cerebral AVM's with bucrylate. Experience in 46 cases. *J Neurosurg* 56: 615-627
34. Deruty R, Lapras C, Bret P, Taboada F, Duthel R (1981) Embolization per opératoire des MAV cérébrales inextirpables. Tentative d'oblitération par un mélange à polymérisation retardée. *Neurochirurgie* 27: 5-14
35. Deruty R, Lapras C, Pierluca P, Patet JD, Pialat J, Bascoulergues Y, Garcia C (1985) Embolization per opératoire des M.A.V. Cérébrales par le Butyl-Cyanoacrylate (18 cas). *Neurochirurgie* 31: 21, 29
36. Deruty R, Lapras C, Patet JD, Bascoulergues Y, Pialat J, Honorato D (1986) Intra-operative embolization of cerebral arteriovenous malformations by means of isobutylcyanoacrylate (experience in 20 cases). *Neurol Res* 8: 109-113
37. * Drake CG (1978) Cerebral AVM's. Consideration for and experience with 166 cases. *Clin Neurosurg* 26: 145-207
38. Fleisher AS, Kricheff I, Ransohoff J (1972) Postmortem findings following the embolization of an AVM. Case report. *J Neurosurg* 37: 606-609
39. Fournier D, Terbrugge KG, Willinsky R, Lasjaunias P, Montanera W (1991) Endovascular treatment of intracerebral arteriovenous malformations. Experience in 49 cases. *J Neurosurg* 75: 228-233
40. Fox J, Al-Mefty O (1977) Embolization of an AVM of the Brain stem. *Surg Neurol* 8: 7-9
41. Friedman P, Salazar JL, Sugar O (1978) Embolization and surgical excision of giant AVM's. *Surg Neurol* 9: 149-152
42. Gentili F, Schwartz M, Terbrugge K, Wallace MC, Willinski R, Young C (1992) A multidisciplinary approach to the treatment of brain vascular malformations. In: Symon L (ed) *Advances and technical standards in neurosurgery*, Vol 19. Springer, Wien New York, pp 179-208
43. George B, Riche MC, Gaston A, Laurian C (1984) Embolization peropératoire des MAV à partir de l'artère vertébrale. *Neurochirurgie* 30: 269-273.
44. Germano IM, Davis RL, Wilson CB, Hieshima GB (1992) Histopathological follow-up study of 66 cerebral arteriovenous malformations after therapeutic embolization with polyvinyl-alcohol. *J Neurosurg* 76: 607-614
45. Halbach VV, Higashida RT, Yang P, Barnwell S, Wilson CB, Hieshima GB (1988) Preoperative balloon occlusion of AVM's. *Neurosurgery* 22: 301-308
46. Hieshima GB, Mehringer CM, Grinnell VS, Landau B, Sage DJ, Goodman SJ, Beresini D, Pribram HF (1982) Preoperative balloon occlusion. "The intra-vascular ligature". *Surg Neurol* 17: 191-195
47. Jones FD, Boone SC, Whaley RA (1982) Intracranial hemorrhage following attempted embolization and removal of large AVM's. *Surg Neurol* 18: 278-283
48. Klara PM, George ED, Mc Donnell DE, Pevsner PH (1985) Morphological studies of human AVM's. Effect of IBCA. Embolization. *J Neurosurg* 63: 421-425
49. Kusske JA, Kelly WA (1974) Embolization and reduction of the steal syndrome in cerebral AVM's. *J Neurosurg* 40: 313-321
50. Luessenhop AJ, Spence WT (1960) Artificial embolization of cerebral arteries. Report of use in a case of AVM. *JAMA* 172: 1153-1155
51. Luessenhop AJ, Kachman RJ, Shevlin W (1965) Clinical evaluation of artificial embolization in the management of large cerebral AVM's. *J Neurosurg* 23: 460-471
52. Luessenhop AJ, Presper JH (1975) Surgical embolization of cerebral AVM's through internal carotid and vertebral arteries. Longterm results. *J Neurosurg* 42: 443-451
53. Luessenhop AJ, Mujica SH (1981) Embolization of segments of circle of willis and adjacent branches for management of certain inoperable cerebral AVM's. *J Neurosurg* 54: 573-582
54. * Luessenhop AJ, Rosa L (1984) Cerebral AVM's. Indication and results for surgery, and the role of intravascular techniques. *J Neurosurg* 60: 14-22
55. Mizoi K, Takahashi A, Yoshimoto T, Sugawara T, Saito K (1992) Surgical excision of giant cerebellar hemispheric arteriovenous malformations following preoperative embolization. *J Neurosurg* 76: 1008-1011
56. Morgan MK, Sundt ThM Jr (1989) The case against staged operative resection of cerebral AVM's. *Neurosurgery* 25: 429-435
57. Mullan S, Kawanaga H, Patronas NJ (1979) Microvascular embolization of cerebral AVM's. A technical variation. *J Neurosurg* 51: 621-627
58. Pasqualin A, Scienza R, Cioffi F, Barone G, Benati A, Beltramello A, Da Pian R (1991) Treatment of cerebral AVM's with a combination of preoperative embolization and surgery. *Neurosurg* 29: 358-368
59. Pevsner SH, George ED, Doppman JL (1982) Interventional radiology polymer update: Acrylic. *Neurosurgery* 10: 314-316
60. Purdy PD, Batjer HH, Samson D (1991) Management of hemorrhagic complications from preoperative embolization of arteriovenous malformations. *J Neurosurg* 74: 205-211
61. Purdy PD, Batjer HH, Risser RC, Samson D (1992) Arteriovenous malformations of the brain: Choosing embolic materials to enhance safety and ease of resection. *J Neurosurg* 77: 217-222
62. Samson D, Ditmore M, Beyer CW (1981) Intravascular use of IBCA. Part 1. Treatment of intracranial AVM's. *Neurosurgery* 8: 43-51
63. Schell G, Hodge CJ, Cacayorin E (1986) Transient neurological deficit after therapeutic embolization of the arteries supplying the medial wall of the hemisphere including the supplementary motor area. *Neurosurgery* 18: 353-356
64. Serbinenko FA (1974) Balloon catheterization and occlusion of major cerebral vessels. *J Neurosurg* 41: 125-145
65. Spetzler RF, Martin NA, Carter LP, Flom RA, Raudzens PA, Wilkinson E (1987) Surgical management of large AVM's by staged embolization and operative excision. *J Neurosurg* 67: 17-28
66. Spetzler RF, Zabramski JM (1988) Grading and stages resection of cerebral AVM's. *Clin Neurosurg* 36: 318-337
67. Spetzler RF, Zabramski JM (1988) Surgical management of large AVM's. *Acta Neurochir Wien [Suppl]* 42: 93-97
68. Stein BM, Wolpert SM (1977) Surgical and embolic treatment of cerebral AVM's. *Surg Neurol* 7: 359-369
69. Suzuki J, Takahashi A, Yoshimoto T, Mirobumi S (1985) Use of balloon occlusion and substances to protect ischemic brain

during resection of posterior fossa AVM. *J Neurosurg* 63: 626–629

70. Terada T, Nakamura Y, Nakai K, Tsuura M, Nishiguchi T, Hayashi S, Kido T, Takiw, Iwata H, Komai N (1991) Embolization of arterio-venous malformations with peripheral aneurysms using ethylene vinyl alcohol copolymer. Report of three cases. *J Neurosurg* 75: 655–660
 71. Vinuela F, Dion JE, Duckwiler G, Martin NA, Lylyk P, Fox A, Pelz D, Drake CG, Girvin JJ, Debrun G (1991) Combined endovascular embolization and surgery in the management of cerebral arteriovenous malformations. Experience with 101 cases. *J Neurosurg* 75: 856–864
 72. Vlahovitch B, Fuentes JM (1976) Embolization of cerebral aneurysms by catheterization of cortical arteries. *Neuroradiology* 11: 243–248
- Radiosurgery*
73. Backlund EO (1979) Stereotactic radiosurgery in intracranial tumours and vascular malformations. In: Krayenbuhl H (ed) *Advances and technical standards in neurosurgery*, Vol 6. Springer, Wien New York, pp 3–38
 74. Betti O, Derechinski V (1983) Irradiation stéréotaxique multi-faisceaux. *Neurochirurgie* 29: 295–298
 75. Betti O, Munari C, Rosler R (1989) Stereotactic radiosurgery with the linear accelerator: Treatment of arteriovenous malformations. *Neurosurgery* 24: 311–321
 76. Betti O, Munari C (1992) Traitement radiochirurgical avec accélérateur linéaire des “petites” malformations artério-veineuses intracranienne. *Neurochirurgie* 38: 27–34
 77. Colombo F, Benedetti A, Pozza F, Marcetti C, Chierigo G (1989) Linear accelerator radiosurgery for cerebral AVM's. *Neurosurgery* 24: 833–840
 78. Levy RP, Fabrikant JI, Frankel KA, Phillips MH, Lyman JT (1989) Stereotactic heavy charged particle bragg peak. Radiosurgery for the treatment of intracranial AVM's in childhood and adolescence. *Neurosurgery* 24: 841–852
 79. Lunsford LD, Kondsiolka D, Flickinger V, Bissonette DJ, Jungreis CA, Maitz AH, Horton JA, Coffey RJ (1991) Stereotactic radiosurgery for arteriovenous malformations of the brain. *J Neurosurg* 75: 512–524
 80. Mehdorn HM, Grote W (1988) Noninvasive follow-up of patients with intracranial AVM after proton-beam radiation therapy. *Acta Neurochir (Wien) [Suppl]* 42: 98–102
 81. Merienne L, Laurent A, Meder JF, Lefkopoulos D (1991) Irradiation stéréotaxique de 46 angiomes cérébraux. Analyse des résultats angiographiques deux ans et demi après traitement. *Neurochirurgie* 37: 185–195
 82. Ogilvy C (1990) Radiation therapy for arteriovenous malformations. A review. *Neurosurgery* 26: 725–735
 83. Steiner L, Leksell L, Greitz T, Forster DMC, Backlund EO (1974) Stereotactic radiosurgery in intracranial arteriovenous malformations. *Acta Neurochir (Wien) [Suppl]* 21: 195–209
 84. Steiner L, Lindquist C, Adler JR, Torner JC, Alves W, Steiner M (1992) Clinical outcome of radiosurgery for arteriovenous malformations. *J Neurosurg* 77: 1–8
 85. Steiner L, Lindquist C, Steiner M (1992) Radiosurgery. In: Symon L (ed) *Advances and technical standards in neurosurgery*, Vol 19. Springer, Wien New York, pp 19–102
 86. Tognetti F, Andreoli A, Cuscini A, Testa C (1985) Successful management of an intracranial AVM by conventional irradiation. *J Neurosurg* 63: 193–195
 87. Yamamoto M, Jimbo M, Kobayaski M, Toyoda C, Ide M, Tanaka N, Lindquist C, Steiner L (1992) Long-term results of radiosurgery for arterio-venous malformation: Neurodiagnostic imaging and histological studies of angiographically confirmed nidus obliteration. *Surg Neurol* 37: 219–230

* References marked with an asterisk are quoted in two groups (surgical series and embolization).

Correspondence: R. Deruty, M.D., Hôpital Neurologique, 59 Bvd. Pinel, F-69394 Lyon, France.