



Complete Summary

GUIDELINE TITLE

Recommendations for the management of intracranial arteriovenous malformations: a statement for healthcare professionals from a special writing group of the Stroke Council, American Stroke Association.

BIBLIOGRAPHIC SOURCE(S)

Ogilvy CS, Stieg PE, Awad I, Brown RD Jr, Kondziolka D, Rosenwasser R, Young WL, Hademenos G. AHA Scientific Statement: Recommendations for the management of intracranial arteriovenous malformations: a statement for healthcare professionals from a special writing group of the Stroke Council, American Stroke Association. Stroke 2001 Jun; 32(6):1458-71. [189 references] [PubMed](#)

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COMPLETE SUMMARY CONTENT

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SCOPE

DISEASE/CONDITION(S)

Intracranial (parenchymal or pial) arteriovenous malformations (AVMs)

Note: This document does not cover recommendations for angiographically occult AVMs, cavernous malformations, dural AVMs or fistulae (including vein of Galen AVM), or spinal AVMs.

GUIDELINE CATEGORY

Assessment of Therapeutic Effectiveness
Evaluation
Management
Risk Assessment
Treatment

CLINICAL SPECIALTY

Neurological Surgery
Neurology
Obstetrics and Gynecology
Pediatrics

INTENDED USERS

Physicians

GUIDELINE OBJECTIVE(S)

- To review published data for intracranial arteriovenous malformations (AVMs) to develop practice recommendations regarding epidemiology, natural history, potential treatment strategies, and outcomes
- To serve as a basis for the development of treatment strategies for AVMs, which overall represent a fairly heterogeneous group of cerebrovascular lesions and which may demonstrate different natural histories

TARGET POPULATION

Patients with intracranial arteriovenous malformations (AVMs)

INTERVENTIONS AND PRACTICES CONSIDERED

Evaluation

1. Computed tomography (CT) with or without contrast
2. Magnetic resonance imaging (MRI)
3. Magnetic resonance angiography (MRA)
4. Arteriography
5. Superselective angiography

Management/Treatment

1. Direct surgical treatment (surgical extirpation)
2. Endovascular treatment
 - Presurgical embolization
 - Preradiosurgical embolization
 - Palliative embolization
3. Radiosurgery
4. Multimodality treatment
5. Anesthetic and perioperative considerations
 - Blood pressure control

- Control of patient temperature
- Choice of anesthetic agent

Interventions for pregnant patients and the pediatric population are also considered.

MAJOR OUTCOMES CONSIDERED

- Sensitivity and specificity of diagnostic tests
- Predictive value of diagnostic tests
- Morbidity and mortality
- Incidence of hemorrhage
- Total/permanent angiographic obliteration of the lesion

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

The reports reviewed for this synthesis were selected on the basis of study design, sample size, and relevance to a particular topic.

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Weighting According to a Rating Scheme (Scheme Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Levels of Evidence

Level I: Data from randomized trials with low false-positive (alpha) and low false-negative (beta) errors

Level II: Data from randomized trials with high false-positive (alpha) and high false-negative (beta) errors

Level III: Data from nonrandomized concurrent cohort studies

Level IV: Data from nonrandomized cohort studies using historical controls

Level V: Data from anecdotal case series

METHODS USED TO ANALYZE THE EVIDENCE

Review

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Expert Consensus

DESCRIPTION OF METHODS USED TO FORMULATE THE RECOMMENDATIONS

After review of the available literature, recommendations for current practice were made according to 3 separate grades (see "Rating Scheme for the Strength of the Recommendations").

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Strength of Cumulative Data

Grade A: Supported by level I evidence

Grade B: Supported by level II evidence

Grade C: Supported by level III, IV, or V evidence

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

External Peer Review

Internal Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

This statement was approved by the American Heart Association Science Advisory and Coordinating Committee in February 2001. It was published in *Stroke* 2001;32:1458-1471.

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

Definitions of the levels of evidence (Levels I-V) and grades of recommendation (Grades A-C) are provided at the end of the Major Recommendations field.

Diagnosis and Clinical Manifestations: Natural History of Arteriovenous Malformations (AVMs)

Intracranial AVMs may be diagnosed with a variety of diagnostic imaging studies. Computed tomography (CT) without contrast has a low sensitivity, but calcification and hypointensity may be noted; enhancement is seen after contrast administration. Magnetic resonance imaging (MRI) is very sensitive, showing an inhomogeneous signal void on T1- and T2-weighted sequences, commonly with hemosiderin suggesting prior hemorrhage. MRI can also provide critical information detailing the localization and topography of an AVM as intervention is being considered. Magnetic resonance angiography can provide some data noninvasively, without detailing factors such as presence of intranidal or feeding artery aneurysms, comprehensive data on venous drainage patterns, or subtle AVM nidus characterization. Arteriography is the "gold standard" for defining the arterial and venous anatomy. In addition, superselective angiography can provide functional and physiological data important to clinical decision analysis. On the basis of available information, it is strongly recommended that an MRI study and a 4-vessel angiogram be obtained to delineate the anatomy of an AVM.

Refer to the original guideline document for additional discussion of the natural history of AVMs, risk for hemorrhage, and other clinical considerations.

Direct Surgical Treatment

Timing of Surgery

The recommendation for surgery for AVMs should generally be elective. Occasionally, one must operate emergently to remove a large, life-threatening hematoma. Under these conditions, only superficial AVMs that are readily controllable are removed with the hematoma. When the hematoma is caused by a complicated AVM, the blood clot can be removed and the patient allowed to recover until further details are known regarding the exact angiographic AVM architecture. In a nonemergent situation, the lesion is approached as are other elective intracranial operations.

Lesions are typically excised by standard microsurgical techniques with the operating microscope. The arterial feeders are generally attacked first, followed by excision of the nidus of the lesion and finally resection of the draining vein. In general, the veins are preserved until the very end of the operation. When a brain AVM is resected, the goal should be complete obliteration. To this end, intraoperative or postoperative angiography is usually recommended. If there is residual lesion, immediate resection should be considered to avoid subsequent hemorrhage from the remaining vessels. Another treatment consideration for the residual lesion may include stereotactic radiosurgery, although there remains a risk of hemorrhage during the intervening period until lesion obliteration.

Treatment Options

At present, there are 4 major treatment options available for patients with an AVM of the brain. The lesion can be monitored expectantly with the understanding that the patient would have some risk of hemorrhage or other neurological symptoms such as seizures or focal deficit. Alternatively, intervention can be undertaken with the goal of complete AVM obliteration, because subtotal therapy does not confer protection from hemorrhage. Management strategies include single or combined

therapy applying microsurgery, endovascular techniques, or radiosurgery (focused radiation). Each treatment option has associated risks and benefits.

Anesthetic and Perioperative Considerations for Microsurgical Resection

Recommendations for anesthetic management are based primarily on level V evidence. In general, conduct of anesthesia for AVM resection follows the same recommendations for neuroanesthetic management for any intracranial lesion regarding choice of monitoring, vascular access, anesthetic agents, vasoactive drugs, and muscle relaxants.

Because AVM resection is usually not emergent, preexisting medical conditions should be optimized, and neurological dysfunction, either as a result of presenting hemorrhage, presumed effect of the AVM, or preoperative embolization (infarction or edema), should be factored into the intraoperative and postoperative management plan. An important consideration throughout the operative period is the potential for massive, rapid, and persistent blood loss. Choice of intraoperative monitoring is tempered by this eventuality, and adequate amounts of blood, along with access for its administration, must be readily available.

The risk of AVM rupture during induction is probably low based on inferential evidence. Nevertheless, blood pressure control that approximates the patient's normal range is sound anesthetic practice in the absence of mitigating circumstances. However, it should be borne in mind that approximately 10% of AVM patients harbor intracranial aneurysms that may increase the risk of rupture during increases in arterial blood pressure.

Although intracranial pressure control is rarely a problem with the AVM patient who presents for elective resection, intracranial compliance may be abnormal. Therefore, the usual caveats about avoidance of anesthetics and vasoactive agents that cause cerebral vasodilation seem prudent (i.e., high inspired concentration of volatile anesthetics and high doses of vasodilators that directly relax vascular smooth muscle).

There is no anesthetic regimen that has been rigorously shown to confer "cerebral protection" in neurosurgical patients. The choice of anesthetic agent must be consistent with safe conduct of intracranial surgery, including brain relaxation, excellent blood pressure control, and rapid emergence. Euvolemia, normotension, isotonicity, normoglycemia, and mild hypocapnia are recommended. Profound hypocapnia is not recommended unless indicated for control of brain swelling or surgical exposure.

An ongoing randomized, controlled study (Intraoperative Hypothermia in Aneurysm Surgery Trial 2 [IHAST2]) is evaluating the use of mild induced hypothermia (33 degrees F) for cerebral protection during craniotomy for aneurysm clipping. If successfully completed, this study will provide the first opportunity to gain level I evidence of intraoperative cerebral protection. The induction of general anesthesia results in an obligatory core temperature decrease as peripheral vasodilation redistributes heat to the periphery. The current recommendation is to maintain normothermia or accept the mild decrease in body temperature that results from general anesthesia and not aggressively rewarm

patients until timing for emergence is planned. This recommendation is based only on level V data.

Induced hypotension is frequently useful during AVM resection, especially in large AVMs that have a deep arterial supply. Bleeding from these small, deep feeding vessels may be difficult to control, and decreasing arterial pressure facilitates surgical hemostasis. The subject of induced hypotension is discussed extensively in the anesthesiology literature. There is no compelling evidence to use one particular agent. Choice of agent must be placed in the context of the clinical situation (e.g., avoidance of beta-adrenergic blockers with bronchospastic airway disease or use of nitroglycerin with coronary artery disease) and the experience of the practitioner.

The intraoperative appearance of diffuse bleeding from the operative site or brain swelling and the postoperative occurrence of hemorrhage or swelling have been attributed to normal perfusion pressure breakthrough (NPPB) or "hyperemic" complications. There is no universally accepted definition of what constitutes a hyperemic state, and it should be a diagnosis of exclusion after all other correctable causes for malignant brain swelling or bleeding have been considered. Alpha-adrenergic blockade may be of use in preventing and treating this syndrome, based on anecdotal information and suggestive observations. Emergence hypertension is frequently encountered after AVM resection. Data suggest that elevated plasma renin and norepinephrine levels are associated with this phenomenon.

The upper and lower limits of blood pressure control have potential opposing effects. Ischemic deficits due to intraoperative sacrifice of an en passage feeding vessel (a vessel feeding an AVM and also sending distal branches to normal brain), for example, may result in a deficit ascribed to brain retraction or to the resection itself. Marginally perfused areas may be critically dependent on collateral perfusion pressure. Maintenance of low or even normal blood pressure may be inadequate and may result in infarction if hypoperfusion is unrecognized. Verification of potential borderline perfusion states may require imaging modalities such as intraoperative or immediate postoperative angiography.

Postoperative hyperthermia may be detrimental and may even be exacerbated by mild, intraoperative-induced hypothermia. Therefore, careful attention should be paid to control of patient temperature in the intensive care unit.

Associated Aneurysms

Intracranial aneurysms are found in approximately 7 to 17% of patients. Intracranial aneurysms can occur on the feeding artery to the AVM. These may involute after resection or obliteration of the brain AVM. Alternatively, patients may also harbor more saccular intracranial aneurysms at typical locations in the circle of Willis. It is recommended that these be approached during the same surgery if the operative field is adequate or that they be treated separately with endovascular or open surgical obliteration. There are no natural history data regarding this point in the literature, and therefore the rationale for treatment of aneurysms that are not associated with AVMs is used.

Brain Edema/Hemorrhage

Two hypotheses for the cause of brain edema and hemorrhage during or after surgery have been proposed: NPPB or occlusive hyperemia. The NPPB theory suggests that postoperative hemorrhage and edema are caused by a failure in autoregulation in the ischemic brain around the AVM. Chronic hypoperfusion in brain surrounding an AVM may cause maximal chronic vasodilation, which results in an inability of these vessels to vasoconstrict in response to the resumption of normal perfusion pressure after the AVM has been resected. According to this theory, the key to prevention of malignant postoperative hemorrhage and edema is staged reduction of blood supply to the malformation. This can be accomplished by staged surgical ligation of the feeders or by endovascular embolization. With the technological advance of interarterial embolization, this is the current recommended route, although admittedly this recommendation is based on apparent safety without statistical documentation in the literature. Surgical resection of the AVM should occur shortly (i.e., several days) after the final feeding artery embolization to prevent development of new collateral flow.

A number of observations suggest that the details of this theory are not applicable to most cases of malignant postoperative hemorrhage and edema. Intraoperative studies demonstrate maintained autoregulation in the region surrounding an AVM both before and immediately after its resection, even in cases subsequently complicated by edema and hemorrhage. This observation argues against the value of staged operation or embolization in the resection of AVMs. It has also led to the proposal of an alternative hypothesis regarding the cause of malignant postoperative edema and hemorrhage termed "occlusive hyperemia."

This theory postulates that malignant postoperative hemorrhage and edema are caused by either arterial stagnation and obstruction or venous outflow obstruction, which are in turn direct results of resection of the AVM. Evidence for the role of outlet obstruction in spontaneous hemorrhage presented above tends to support this hypothesis, as does the observation that long feeding arteries correlate with a greater risk of postoperative deterioration than do short vessels of similar diameter and flow. Moreover, given this theory, indications for staged resection would be limited to those cases necessitated by technical factors, and hypotensive therapy in the management of postoperative edema may prove more deleterious than beneficial. All of the data presented regarding these theories are level V, and therefore, their impact on AVM management is only moderate.

Postoperative Care

The recommendations for postoperative care include neurological intensive care monitoring for at least 24 hours. Blood pressure is monitored with an arterial catheter and urine output with an indwelling catheter. Typically, normotensive and euvolemic conditions are maintained; however, tight blood pressure control with agents that do not act in the central nervous system may be appropriate for selected individuals. Perioperative antibiotics, steroids, and seizure medication are used variably. After being monitored in the intensive care unit, the patient is transferred to a standard surgical floor, where mobilization occurs. An angiogram is also performed to confirm complete resection of the AVM during the immediate postoperative period. A new neurological deficit after surgery is usually investigated with a CT scan to rule out a hemorrhage or hydrocephalus. MRI scanning with diffusion-weighted imaging may be appropriate if an infarction is entertained.

In summary, AVM surgery is usually elective and frequently preceded by preoperative embolization. The surgical approach allows complete resection of the nidus, resecting the feeding vessels and subsequently the draining veins. Management of associated aneurysms is determined on an individual basis.

Recommendations

In general, surgical extirpation should be strongly considered as the primary mode of therapy for Spetzler-Martin grade I and II lesions. For patients with small lesions, where surgery offers some increased risk based on location or feeding vessel anatomy, radiosurgery should be strongly considered. For grade III lesions, a combined modality approach with embolization followed by surgery is often feasible (see section below titled "Multimodality treatment of arteriovenous malformation [AVM]"). Surgical treatment only is often not recommended for grade IV and V lesions because it confers a high risk.

Endovascular Treatment

Technical advances in interventional neuroradiology/endovascular neurosurgery have afforded new alternatives in the treatment of cerebral AVMs. Flow-directed and flow-assisted microcatheters have made navigation of intracranial vessels safer and have allowed more accurate delivery of embolic materials. Current embolic materials are divided into solid or liquid agents. Solid agents consist of polyvinyl alcohol particles, fibers, microcoils, and microballoons. Liquid agents consist of cyanoacrylate monomers such as l-butyl cyanoacrylate (IBCA) and n-butyl cyanoacrylate (NBCA), as well as polymer solutions such as ethylene vinyl alcohol (EVAL copolymer). Other liquid agents include absolute ethanol, with and without the use of contrast agents for visualization under digital subtraction fluoroscopy. NBCA has recently been officially approved by the Food and Drug Administration for use in brain AVMs.

Embolization of cerebral AVMs is only one aspect of a multimodality approach to these lesions. Current indications for embolization can be divided into presurgical embolization in large or giant cortical AVMs and embolization before radiosurgical intervention to reduce nidus size. In addition, palliative embolization may be used in large nonsurgical or nonradiosurgical AVMs in patients presenting with progressive neurological deficit secondary to high flow or venous hypertension. In this group of patients, the goal is flow reduction in an attempt to minimize or halt symptom progression. Finally, embolization of a pseudoaneurysm that seems to be related to a hemorrhage is also possible.

Anesthetic and Perioperative Considerations for Endovascular Therapy

Although many of the risks and responses are for the most part conceptually the same, there are also many important differences in the working environment. There are generally two schools of thought on how to manage the patient undergoing AVM embolization. One is to rely on knowledge of neuroanatomy and vascular architecture to ascertain the likelihood of neurological damage after embolization. The "anatomy school," therefore, will prefer to embolize under general anesthesia. Arguments for this approach include improved visualization of structures with the absence of patient movement, especially with temporary apnea or when the ventilator is correlated with digital subtraction angiography

contrast injection. Furthermore, it can be argued that if the embolic material is placed intranidally, then by definition, no normal brain is threatened.

The "physiological school" trades off the potential for patient movement against the increased knowledge of the true functional anatomy of a given patient, given the wide variability described in these patients. At the present time, the physiological approach demands deep intravenous sedation to render the patient comfortable during catheter placement and yet keep the patient appropriately responsive for selective neurological testing.

There is no evidence that either general endotracheal anesthesia or intravenous sedation is associated with a lower rate of complications (level IV evidence). Recommendations for premedication with corticosteroids, anticonvulsants, aspirin, calcium channel blockers, and antibiotics have been made, but none have rigorous support for their use.

Direct transduction of arterial pressure is indicated for intracranial embolization procedures, especially with manipulation of systemic pressure with vasoactive agents. The femoral artery introducer sheath is easily used to monitor arterial pressure. Intravascular pressures may also be monitored from the coaxial (guiding) catheter, as well as via the superselective catheter.

In addition to the recommended American Society of Anesthesiology monitors, additional considerations include placement of an additional pulse oximeter on the foot of the leg that will receive the femoral introducer catheter as an early warning of femoral artery obstruction or distal thromboembolism and overly vigorous compression for postprocedure hemostasis. Bladder catheters assist in fluid management as well as patient comfort. Supplemental oxygen should be given to all patients who have received sedative-hypnotic agents.

General endotracheal anesthesia considerations are conceptually similar to those for open craniotomy. Primary goals of anesthetic choice for intravenous sedation include alleviation of pain or discomfort, anxiety, and patient immobility, but at the same time, the anesthetic must allow for a rapid decrease in the level of sedation when neurological testing is required. There is no evidence one regimen is superior to any other; propofol and midazolam have been directly compared and found to be similarly effective (level II evidence). Choice should be based on the experience of the practitioner and the aforementioned goals of anesthetic management. Common to all intravenous sedation techniques is the potential for upper airway obstruction. Placement of nasopharyngeal airways may cause troublesome bleeding; it may be prudent to place them before anticoagulation. Careful management of coagulation is required to prevent thromboembolic complications during and after the procedures, although algorithms for anticoagulation remain controversial.

Profound deliberate systemic hypotension may be induced while the interventionist prepares the glue for injection. Hypotension slows the flow through the fistula and provides for a more controlled deposition of embolic material, particularly the glues. Blood pressure reduction can be achieved with vasoactive agents, general anesthetics, or even by brief, adenosine-induced cardiac pause.

Complications during endovascular navigation of the cerebral vasculature can be rapid and dramatic and require interdisciplinary collaboration. The primary responsibility of the anesthesia team is to preserve cardiovascular function and gas exchange and, if indicated, secure the airway. If emergent endotracheal intubation is necessary, a thiopental and relaxant induction should not be avoided because of the possibility of a transient decrease in perfusion pressure.

In the setting of inadvertent vascular occlusion, a method to increase distal perfusion is blood pressure augmentation with or without direct thrombolysis. The systemic blood pressure may be increased to drive adequate flow via collaterals to the area of ischemia as a temporizing measure. Given the best available evidence, deliberate hypertension in the face of symptomatic cerebral ischemia from vascular occlusion during AVM embolization should not be avoided because of fear of rupturing the malformation. If the problem is hemorrhagic, immediate reversal of heparin is indicated. Protamine is given as rapidly as possible to reverse heparin without undue regard for systemic blood pressure.

Recommendations for endovascular management of AVMs

Recommendations for endovascular management of AVMs can be divided into presurgical, preradiosurgical, or palliative management for focal neurological symptoms or uncontrolled seizures. The decision to perform embolization of an AVM should take into consideration Spetzler-Martin grade as well as the combined surgical and endovascular risk for a particular patient. The risks of embolization must be weighed against other risks in terms of combined morbidity and mortality for surgery and/or radiosurgery. Currently, all data available are either level III or IV, because no prospective randomized trials exist concerning embolization therapy.

In general, Spetzler-Martin grade II or III lesions may be embolized before surgery or radiosurgery. Grade IV or V lesions should not be embolized unless this is to be done in conjunction with other treatment modalities (surgery or radiosurgery) for the goal of complete care. The only exception to this may be in a patient with a grade IV or V lesion with venous outflow obstruction, in whom embolization is used to reduce arterial inflow to control edema, or in a patient with true "steal," in whom embolization is used to relieve the amount of shunt through the AVM.

Radiosurgery

Recommendations

Radiosurgery can be considered in lesions thought to be at high risk from a surgical or endovascular standpoint. The overall efficacy of radiosurgery is higher for small lesions, and therefore, this modality may be considered as a primary treatment for smaller as opposed to larger lesions. However, size is not the only factor in the final determination of treatment. The exact location, patient age, symptoms, and angiographic anatomy must be considered in this decision process. For small, surgically accessible lesions (Spetzler-Martin grade I or II), surgery has fewer risks than radiosurgery. Radiosurgery may be considered in larger lesions (Spetzler-Martin grade II through V) only if the overall goal is complete obliteration of the lesion. Partial treatment of a larger lesion with

radiosurgery or embolization subjects the patient to the risks of the procedure without eliminating the risk of hemorrhage.

Multimodality Treatment of AVM

Recommendations

Multimodality therapy should be performed only if it is part of a total treatment plan to eradicate an AVM. The goals of the different modalities should be clear at the outset. Because of the variability of resources available in any one area of the country or world, some patients are offered partial treatment with a single technique. Such treatment is unjustified. Although it is difficult to make generalizations about specific uses of multimodality treatment, such treatment does appear to play a helpful role in larger lesions (Spetzler-Martin grade III or V) for which complete obliteration is the goal. The hope is that with combined techniques, the overall risk of therapy will be reduced, although this is yet to be proven statistically.

Specific Considerations

Pregnant Patients

If a woman anticipates pregnancy and has a known AVM, treatment should be considered before the pregnancy. If the lesion is discovered during pregnancy, a decision should be made regarding the treatment risks versus the risk of hemorrhage during the remainder of the pregnancy if the lesion is left untreated. This also must include the potential risk to the fetus during intervention, whether it be by embolotherapy, surgical extirpation, or radiation and the associated diagnostic tests. In most cases, such risk-benefit analysis will not support elective treatment of AVMs during pregnancy.

Pediatric Lesions

The younger the patient, the more conclusively treatment is warranted. More aggressive treatment strategies can be justified in dealing with pediatric patients, whereas only low-risk strategies should be offered to elderly patients.

Management of Complications

Hydrocephalus

Hydrocephalus may occur as a result of intraventricular hemorrhage secondary to an AVM. When this occurs soon after hemorrhage, urgent insertion of ventricular drainage catheters may be necessary. These catheters can also be used to monitor intracranial pressure in patients in the intensive care unit setting. As the ventricular blood is cleared, patients may have chronic hydrocephalus and thus may warrant ventriculoperitoneal shunting. This decision should be made on an individual basis, based on the size of the ventricles and the cerebrospinal fluid pressure. In rare instances, hydrocephalus can result from compression of the aqueduct of Sylvius by large draining veins of AVMs.

Seizures

Although all of the reports available are level V data, it can generally be expected that surgical or radiosurgical obliteration of an AVM will reduce seizure activity. No studies exist from which recommendations can be made in terms of duration or type of anticonvulsant prophylaxis after treatment.

Definitions:

Levels of Evidence

Level I: Data from randomized trials with low false-positive (alpha) and low false-negative (beta) errors

Level II: Data from randomized trials with high false-positive (alpha) and high false-negative (beta) errors

Level III: Data from nonrandomized concurrent cohort studies

Level IV: Data from nonrandomized cohort studies using historical controls

Level V: Data from anecdotal case series

Strength of Cumulative Data

Grade A: Supported by level I evidence

Grade B: Supported by level II evidence

Grade C: Supported by level III, IV, or V evidence

CLINICAL ALGORITHM(S)

None provided

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The type of supporting evidence is identified and graded for some of the recommendations (see "Major Recommendations").

No level 1 or level 2 evidence (i.e., data from randomized trials) was available in the literature for management of arteriovenous malformations. All evidence was level III (data from nonrandomized concurrent cohort studies), level IV (data from nonrandomized cohort studies using historical controls), or level V (data from anecdotal case series).

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

- Appropriate management of patients with intracranial arteriovenous malformations (AVMs)
- Numerous studies describe the beneficial effect of presurgical embolization in reducing operative time and blood loss, as well as converting high Spetzler-

Martin grade lesions to lower-grade lesions, with a concurrent reduction in morbidity and mortality.

- Obliteration of AVMs may reduce the incidence of seizures.

POTENTIAL HARMS

- Surgical treatment only is often not recommended for grade IV and V lesions because it confers a high risk.
- Partial treatment of a larger lesion with radiosurgery or embolization subjects the patient to the risks of the procedure without eliminating the risk of hemorrhage.
- Complications during endovascular navigation of the cerebral vasculature can be rapid and dramatic and require interdisciplinary collaboration.
- The intraoperative appearance of diffuse bleeding from the operative site or brain swelling and the postoperative occurrence of hemorrhage or swelling have been attributed to normal perfusion pressure breakthrough (NPPB) or "hyperemic" complications.
- There is a 5 to 7% risk of treatment-related complications with radiosurgery. In addition, symptomatic patients are exposed to a 3 to 4% risk per year of hemorrhage during the time to obliteration. Therefore, over a 3-year period, the patient has a 14 to 19% risk of complication or hemorrhage in addition to possible incomplete obliteration.

QUALIFYING STATEMENTS

QUALIFYING STATEMENTS

- By the design of this type of review, the recommendations in this report represent an overview of existing treatment protocols that may vary considerably. These guidelines were developed to serve as a basis for the development of treatment strategies for arteriovenous malformations (AVMs), which overall represent a fairly heterogeneous group of cerebrovascular lesions and which may demonstrate different natural histories. In addition, for brain AVMs, no level I or II data are available in the literature. Because of the heterogeneity of these lesions and their relatively infrequent occurrence, strictly defined subcategories for comparison of the efficacy of various treatment modalities are difficult. Therefore, the recommendations presented here are potentially open to a wide interpretation.
- This document specifically addresses intracranial parenchymal or pial AVMs and does not cover recommendations for angiographically occult AVMs, cavernous malformations, dural AVMs or fistulae (including vein of Galen AVM), or spinal AVMs. These other lesions reflect unique considerations of epidemiology, diagnostic evaluation, natural history, risk-benefit analysis, and therapeutic strategies. Other special considerations in rare familial AVMs and those associated with hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu disease), including vascular malformations affecting multiple organ systems, are also beyond the scope of this report.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

An implementation strategy was not provided.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better
Living with Illness

IOM DOMAIN

Effectiveness

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

Ogilvy CS, Stieg PE, Awad I, Brown RD Jr, Kondziolka D, Rosenwasser R, Young WL, Hademenos G. AHA Scientific Statement: Recommendations for the management of intracranial arteriovenous malformations: a statement for healthcare professionals from a special writing group of the Stroke Council, American Stroke Association. Stroke 2001 Jun; 32(6):1458-71. [189 references] [PubMed](#)

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ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

2001 Jun

GUIDELINE DEVELOPER(S)

American Heart Association - Professional Association
American Stroke Association - Disease Specific Society

SOURCE(S) OF FUNDING

American Heart Association

GUIDELINE COMMITTEE

Not stated

COMPOSITION OF GROUP THAT AUTHORED THE GUIDELINE

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FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

Not stated

GUIDELINE STATUS

This is the current release of the guideline.

GUIDELINE AVAILABILITY

Electronic copies: Available from the American Heart Association Web site:

- [HTML Format](#)
- [Portable Document Format \(PDF\)](#)

Print copies: Available from the American Heart Association, Public Information, 7272 Greenville Ave, Dallas, TX 75231-4596; Phone: 800-242-8721.

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

This summary was completed by ECRI on September 13, 2004. The information was verified by the guideline developer on October 13, 2004.

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