# Endovascular Treatment of Cerebral Arteriovenous Malformations

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## Abstract

Brain arteriovenous malformations (AVMs) are rare and heterogeneous vascular abnormalities. AVMs are formed of a tangled anastomosis of arteries and veins without intervening capillaries located within the brain parenchyma (90 % supratentorial) with pathologic shunting of blood from the arterial to the venous side. Prevalence is approximately 18 per 100,000 adults with an incidence of approximately 1.3 per 100,000 adults per year. Most cases are sporadic – cause unknown and probably multifactorial with genetic and environmental factors.

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## Clinical Features

- Brain arteriovenous malformations (AVMs) are rare and heterogeneous vascular abnormalities.
- Formed of a tangled anastomosis of arteries and veins without intervening capillaries located within the brain parenchyma (90 % supratentorial) with pathologic shunting of blood from the arterial to the venous side.
- Prevalence approx. 18 per 100,000 adults and incidence approx 1.3 per 100,000 adults per year.
- Most cases are sporadic – cause unknown and probably multifactorial with genetic (>900 genes are involved in the pathogenesis) and environmental factors. Minority associated with congenital and hereditary syndromes: Rendu-Osler-Weber, Wyburn-Mason, and Sturge-Weber. Rare familial cases not associated with syndromes are also described.
- Biologically active lesions can grow or regress. Following obliteration they can rarely recur – most recurrences in children.

## Clinical Presentation

- Intracranial hemorrhage (ICH) parenchymal subarachnoid or intraventricular bleed (incidence of AVM-related ICH is approx. 0.50 per 100,000 person-years).
- Seizures (focal or generalized – most respond well to antiepileptic drugs – intractable seizures rare).
- Headaches (no distinctive features – the yield of AVMs in patients investigated for headache is very low).
- Progressive neurological deficit, arterial steal, or venous hypertension.
- Pulsatile tinnitus.
- Typical presentation is ICH in young adult, but many now detected incidentally and majority of cerebral AVMs now present unruptured.

## Natural History

### Ruptured AVM

- The Columbia database suggests for patients with recent AVM-related ICH that 47 % of those have no deficit, an additional 37 % remain independent, 13 % are moderately

disabled, and 3 % are severely disabled. The long-term crude fatality rate is around 1–1.5 % per annum and the annual rates of severe morbidity around 1.4 %.

## Unruptured AVM

- Unclear – some data suggests that interventions may lead to worse outcomes than natural history – ARUBA trial is underway.

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# Diagnostic Evaluation

## Clinical

- Comprehensive medical, neurological, cognitive, and radiological assessment is essential. Where relevant, consider other specific assessments, i.e., ophthalmological and psychiatric.

## Laboratory

- Basic blood screening includes full blood count, urea and electrolytes, coagulation profiles, and blood group and save; electrocardiography (ECG), chest radiograph etc., as required.

## Imaging

### Magnetic Resonance Imaging (MRI)

MRI is vital to establish AVM location, relationship to eloquent cortex, evidence of old ICH, and parenchymal changes of gliosis or edema (latter often reflects venous hypertension).

### Cerebral Angiography

Cerebral angiography is the gold standard to evaluate arterial and venous anatomy and is crucial for planning treatment: 6-vessel angiography should always be considered, particularly when there has been a previous cerebral intervention, as unexpected (i.e., dural) collaterals are more likely.

**Table 1** Major risk factors for hemorrhage and annual bleeding risk (Stapf 2006 from columbia database)

| Major risk factors for hemorrhage | Annual bleeding risk (%) |
| --- | --- |
| 1. Previous ICH | 4.5 |
| 2. Deep location | 3.1 |
| 3. Exclusively deep venous drainage | 2.5 |

## Indications

Management is often challenging and controversial, particularly in unruptured AVMs. Treatment decisions should be made on case-by-case basis with multidisciplinary input with all treatment modalities represented. Risks of invasive treatment must be balanced against natural history, but unbiased natural history data is currently limited.

### Suggested Indications for Treatment

- Intracranial hemorrhage (ICH)
- Progressive neurological deficit attributable to the AVM
- Intractable seizures (surgical excision renders 80 % seizure-free)
- Other disabling symptoms likely to be improved by treatment (i.e., dural headache)

### Risk Stratification for Future Hemorrhage

Annual bleeding risks: 0/3 risk factors are 0.9 %; 3/3 factors may be as high as 34.4 % (Table 1).

Other risk factors often considered to predict future hemorrhage:

- Stenotic/occlusive changes in the draining vein
  - Single draining vein
  - Intranidal aneurysms
  - Small nidal size
- Some estimate risk the lifetime of bleeding by subtracting age from 105.

## Contraindications

- Difficult access – including tortuous/occluded proximal arterial anatomy
- Iodinated contrast allergy
- Severe renal impairment

## Anatomy

Thorough angiographic assessment should consider each of the following:

### Arteries

- Type and number of feeding arteries
- Aneurysms (remote, feeding vessel, or intranidal)
- Induced collaterals from adjacent vascular territories (may be misinterpreted as part of the nidus)

### Shunt

- Nidus compact or diffuse
- Presence of fistula – fast/slow?

### Veins

- Numbers – deep/superficial
- Stenosis/ectasia
- Degree of congestion

## Spetzler-Martin Grading (Grades I–V) (Table 2 and 3)

- Widely used – primarily reflects surgical risk but broadly reflects embolization; risk large complex AVMs require multiple procedures
- Higher risk with those related to eloquent cortex, deep feeders, and deep venous drainage
- BUT does NOT consider perforator arterial supply, compact on diffuse nidus, associated aneurysms, and, importantly, the experience of the neurosurgeon/interventionalist

## Equipment

- Biplane angiography is considered essential.
- 6-F guide catheter with heparinized saline flush.
- Microcatheter: DMSO compatible for Onyx usage.
- Microwire.

**Table 2** Spetzler-Martin grade

| A point is allocated for size, location, deep venous drainage | |
|---|---|
| Eloquent cortex is sensorimotor, language, visual cortex, hypothalamus, internal capsule, brainstem, cerebellar peduncles, and deep cerebellar nuclei | |
| A separate SM grade VI is considered inoperable | |
| **Size** | **Point** |
| Small (<3 cm) | 1 |
| Medium 3–6 cm | 2 |
| Large >6 cm | 3 |
| (*Usually at the time of diagnosis, 30% is <3 cm, 60% 3–6 cm, and 10% >6 cm*). | |
| *Location* | |
| Non-eloquent | 0 |
| Eloquent | 1 |
| *Venous drainage* | |
| Only superficial | 0 |
| Deep (any) | 1 |

**Table 3** Incidence of postoperative deficit by AVM SM grade

|  | Minor | Major |
|---|---|---|
| I | 0 | 0 |
| II | 5 | 0 |
| III | 12 | 4 |
| IV | 20 | 7 |
| V | 10 | 12 |

*Cerebral AVM treatment recommendations*: *North American Guidelines*
Surgical excision is the single treatment of choice for Spetzler-Martin grades I and II
STRS is preferred single treatment for those <3 cm diameter if the vascular anatomy is unsuitable for surgery and in anatomically difficult locations
A combined approach with embolization (which may be staged) prior to surgery/STRS may be considered in II–V
Surgery alone is unsuitable for grades IV and V (require multidisciplinary discussion – intervention may carry greater risk than natural history)
Palliative embolization is beneficial when a reduced arterial inflow is required in view of venous outflow obstruction or true steal (controversial)

## Embolic Agents

- Liquid embolic agents – penetrate deeply into the AVM achieving permanent embolization – n-butyl cyanoacrylate (NBCA) and Onyx
- Detachable coils – may be helpful adjunct to slow flow in fistulous components
- Particles generally ineffective – high rate of recanalization

### NBCA/Histoacryl/"Glue"

- NBCA is a liquid monomer that undergoes a rapid exothermic polymerization catalyzed by nucleophiles found in blood and on the vascular endothelium, to form a solid adhesive.
- NBCA provokes an inflammatory response in the vessel wall and surrounding tissue, leading to vessel necrosis and fibrous ingrowth.
- Recanalization is very uncommon after an adequate embolization.
- The rate of polymerization is adjusted by diluting NBCA with lipiodol, which is also radiopaque, and allows visualization during injection.
- Higher concentrations of lipiodol will reduce the rate of polymerization and increase the viscosity of the embolic material. Dilute NBCA penetrates deeper into the nidus but with greater risk of escape into the venous system.
- Tantalum powder may be added to provide greater radiopacity and is essential where very high concentrations of glue (>90 %) are required.

*Positive*: NBCA may be preferred to Onyx in some fistulous AV shunts, perforating arteries, leptomeningeal collaterals, en passant feeders, and when the catheter position is away from the nidus.

*Negative*: NBCA is generally considered less predictable than Onyx, even in experienced hands.

### Onyx

- Onyx is ethylene vinyl copolymer (EVOH) dissolved in dimethyl sulfoxide (DMSO) and made radiopaque with tantalum powder.
- A nonadhesive, cohesive liquid – on contact with blood, the DMSO solvent rapidly diffuses away causing precipitation and solidification of the polymer, a permanent spongy material (complete within 5 min).
- Solidification is slower than with NBCA, allowing prolonged controlled injections.
- Precipitation progresses from the outer surface inward, forming a skin with a liquid center that continues to flow (like lava) as the solidification continues.
- Rate of precipitation of copolymer is proportional to concentration of EVOH.

- Onyx 18 (6 % EVOH) is less viscous (viscosity 18 cP), will generally flow further from the catheter tip, and is often used for embolization of the plexiform nidus.
- Onyx 34 (8 % EVOH) is more viscous (viscosity 30 cP) and is generally used for higher flow fistulas.

### Positive

- Better nidal penetration from a single pedicle.
- Multidirectional nidus penetration with retrograde filling of other feeders.
- Control angiography possible mid-injection to assess nidus and draining veins.
- More pliable/less inflammatory than NBCA – easier surgical manipulation.

### Negative

- DMSO toxic effects include vasospasm, angionecrosis, arterial thrombosis, and vascular rupture. Toxicity directly related to the volume infused and endothelial contact time. To avoid angiotoxicity: *Use SLOW DMSO infusion rate not exceeding 0.25 ml/90 s*.
- Limited to DMSO-compatible microcatheters/syringes, etc.
- Tantalum may cause sparking with bipolar cautery during surgery.
- Artifact on CT but not MRI (hypointense on T1- and T2-weighted sequences).

## Pre-procedure Medications

The author uses dexamethasone 12 mg intravenously at the beginning of each procedure to limit any inflammatory response that might be induced by the embolic agent.

## The Procedure

### Access

- Transfemoral catheterization of the parent vessel (internal carotid, vertebral, or occasionally external carotid artery) with a 6 F guide catheter with continuous heparinized saline flush.
- If the AVM is supplied by >2 vascular territories, e.g., posterior and middle cerebral, the author places guide catheters in both parent vessels (vertebral and ICA) to control the whole arterial territory during embolization.

### Angiography and Assessing the Lesion

A minimum of four-vessel angiography should be repeated at the start of each session of embolization. External carotid artery injections should be considered wherever dural supply is possible – superficial lesion/previous intervention.

### Provocative Testing

- Involves the selective injection of a short-acting anesthetic into the territory in question in the awake patient followed by neurological testing of that region – amobarbital and methohexital are injected into brain arteries and lignocaine to test cranial nerves in external carotid branches.
- Why? Absence of visible normal vessels on superselective angiography does not guarantee embolization without deficit.
- A negative result is not 100 % predictive – technique not in widespread usage.

## Treatment Plan and Technique

- Ultimate goal is usually cure/obliteration – often requiring multimodality approach.
- Any residual early venous filling risks subsequent ICH.
- Partial treatment may increase hemorrhagic risk.
- Invasive treatment in unruptured AVMs may be more risky than natural history (a Randomized Trial of Treatment of Unruptured Brain AVMs (ARUBA) attempts to address this).
- Management requires a careful, experienced multidisciplinary approach.
- Treatment options include conservative management, surgery, stereotactic radiosurgery (STRS), and embolization in any combination.
- No randomized trials have compared treatment types with one another or with natural history.

### Consider

*Patient factors*: presentation, age, comorbidity, occupation, and lifestyle
*AVM factors*: location, size, and angioarchitecture
*Technical factors*: local experience/expertise

## Embolization Strategies

There must be a clear and realistic embolization strategy from the outset.

## Cure

Series report embolization cure rates 15–73 % (mostly around 15–20 %). Cure rates probably better in nidal size <3 cm, supply by 3 or less arterial pedicles, fistulous rather than plexiform, no en passant feeders, superficial venous drainage, cortical location.

## Partial Targeted to Suspected Bleeding Source

Aneurysm, venous ectasia, etc.

## Pre-STRS Adjunct

- *Volume reduction*: (>70 % often achievable) aim <4 cm (Table 4)
- *Target aneurysm*(*s*): remain a risk for ICH until AVM obliterated
- *Target arteriovenous fistulae*: refractory to STRS

## Presurgical Adjunct

Many small AVMs do not require preoperative embolization – exceptions are those grade I and II AVMs with deep arterial feeders that are difficult to access surgically and some grade III AVMs located either deep or involving eloquent cortex. For grade IV and V lesions, staged embolizations often performed at intervals of 3–4 weeks can improve surgical outcome. Advantages of presurgical embolization include targeting of difficult feeders, reducing operative blood loss and operative time, and decreasing nidal volume and blood flow. Staged embolization may reduce risk of normal pressure breakthrough bleeding (NPBB).

## Palliation

To relieve symptoms secondary to shunting – arterial steal/venous hypertension, for example, headache, may be transiently improved by targeted treatment of dural feeders;

**Table 4** Nodal diameter and treatment

| Nidal diameter (cm) | Treatment |
| --- | --- |
| <4 | No evidence embolization + STRS better than STRS alone |
| 4–6 | 90 % can be reduced for STRS |
| >6 | 50 % can be reduced for STRS |

Adapted from Gobin YP, Laurent A, Merienne L, et al. Treatment of brain arteriovenous malformations by embolization and radiosurgery. J Neurosurg. 1996;85:19–28
*STRS* stereotactic radiosurgery

neurological deficits and (medically intractable) seizure activity may improved by partial embolization.

*Caution*: Palliative embolization does not seem to improve the eventual outcome in incurable AVMs and may even worsen the subsequent clinical course. Partial treatment of AVMs may increase the risk of subsequent ICH.

## Embolization Technique: Onyx

- Should only be undertaken by experienced practitioners – proctor supervision is strongly recommended in initial cases for inexperienced users.
- Biplane angiography is essential with patient under general anesthesia.
- Some operators advocate systemic heparinization – but no consensus.

## Preparing to Inject

- Onyx must be shaken vigorously on the mixer for at least 20 min to fully suspend tantalum. Mixing must continue until just before injection to ensure optimal opacification.
- Select DMSO-compatible microcatheter, including UltraFlow, Marathon, Apollo, Echelon (all ev3-Covidien), and Sonic (Codman).
- Detachable tip microcatheters should be considered. The amount of reflux permitted with the detachable tip catheters (Apollo, ev3; Sonic, Balt) will vary according to the length of detachable tip chosen and relates to the microangiographic anatomy.
- All DMSO-compatible microcatheters usually require microguidewire navigation – e.g., SilverSpeed 10 (0.010 in.) and Mirage (0.008 in.) (both ev3-Covidien) and Traxcess (0.014 in. – good with Marathon) (MicroVention).
- The microcatheter is navigated distally in a supplying arterial pedicle (care to avoid perforation) under road map guidance.
- Microcatheter angiography is vital to assess:
  - Arterial anatomy *proximal* to catheter tip – identifying any branches that might be compromised by reflux of embolic agent.
  - Arterial anatomy *distal* to catheter tip – en passant feeders, distal territory supply, flow dynamics within the pedicle, aneurysms, etc.
  - Segment of *nidus* filling from this pedicle includes aneurysms/venous drainage.
  - *Transit* time from arterial to venous phase.
- Select angiographic projections that elongate the microcatheter and do not overlap with the nidus or draining vein. This allows better visualization of reflux/less risk of occluding proximal branches or catheter retention.
- Flush the microcatheter with saline to clear contrast.
- Check the dead space of the microcatheter from the manufacturer's literature and fill the dead space of the microcatheter and hub with that volume of DMSO drawn up in a compatible syringe (volumes – UltraFlow 0.22 ml; Marathon and Apollo 0.23 ml; Echelon 10 0.034 ml).

### Onyx Injection

- Onyx drawn up in a DMSO-compatible syringe.
- An interface device can be used to reduce initial mixing of the DMSO/Onyx in the hub making emerging Onyx easier to see fluoroscopically.
- Inject Onyx under road map guidance slowly – 0.25 ml over 90 s to replace the DMSO in microcatheter and then at a similar rate.
- Initial aim is to form a dense plug at the catheter tip, promoting subsequent antegrade flow into the nidus.
- If reflux occurs, stop and wait 20–45 s before resuming injection (waits of longer than 2 min may result in Onyx solidification in the microcatheter).
- After each pause, reset the road map to visualize the "fresh" Onyx.
- The stop-start cycle often needs to be repeated several times (occasionally up to 35–40 min).
- Onyx will eventually go forward to penetrate the nidus.
- The amount of reflux permitted with the detachable tip catheters will vary according to the catheter. The length of detachable tip chosen depends upon angiographic anatomy.
- Precipitation of Onyx in one part of the nidus often redirects newly injected Onyx into other arterial territories; therefore, it can reach many arterial territories from one feeder.
- Long injections involving several milliliters of Onyx may take 40–60 min.
- Author recommends occluding no more than approximately 30 % of the volume of nidus per session – excessive embolization risks ICH.
- Perform control angiography at intervals through the guide catheter to assess nidus occlusion, status of draining veins, etc.
- Control blood pressure during the procedure, maintaining a systolic pressure around 100–110 mmHg. (Some premedicate with a beta blocker.)
- Indeed, if the passage of contrast through the nidus is high flow, it might help to reduce arterial blood pressure during the initial injection until flow is slowed.

### *Avoid*

1. Stasis in the AVM – occluding the draining vein until the very end of entire embolization program when arterial inflow is minimal.
2. Excess reflux >2 cm marathon – risks catheter retention (see later). The amount of tolerated reflux will depend upon location of proximal branches, caliber and tortuosity feeders, and density of refluxed Onyx.
3. Forceful injection of Onyx – excess resistance may herald catheter rupture.

### Removing the Microcatheter

- At the end of the injection, gently aspirate the microcatheter to disengage the tip from the Onyx cast and apply gentle traction on the microcatheter – aim for 2–3 cm of catheter stretch, hold for a few seconds, release, and repeat until the catheter is retrieved. Occasionally, this may take some minutes and in some cases may be impossible.

- Do not retrieve at all costs; leaving the catheter is simple and almost always the safest option – symptomatic complications are very rare – catheter endothelialized within a few weeks.

### Managing a Retained Microcatheter

1. Cut retained microcatheter hub from its shaft (sterile scissors better than blade especially in braided microcatheters).
2. Open guide catheter hemostatic valve widely (accept back bleeding) so that the microcatheter moves freely within it.
3. Gently withdraw guide catheter over retained catheter while screening the microcatheter tip (to ensure the guide is not transmitting traction to the microcatheter tip).
4. Once guide catheter is fully removed, pull gently on microcatheter to straighten its course in the cerebral arteries and apply gentle traction.
5. Cut microcatheter shaft as close to hub of groin sheath as possible.
6. Push back end of microcatheter though sheathed into femoral artery using sheath introducer/vascular dilator (screen to confirm it is in artery and not retained in sheath).
7. Mention the retained microcatheter in procedural report (appearances on subsequent imaging can be alarming!).
8. Aspirin 75–250 mg/day for 3 months where possible.

### Modification of Technique for NBCA/Histoacryl/"Glue"

- Mix NBCA with lipiodol to the appropriate concentration on a separate table to avoid contact with blood or other ionic agent that will cause premature polymerization.
- Use of color-coded syringes is recommended.
- At NBCA concentrations >90 %, it is recommended that tantalum powder is used to improve opacification of the NBCA/lipiodol mixture.
- Dilution should be chosen according to flow dynamics and angioarchitecture.
- NBCA can be injected through any microcatheter.
- Purge the microcatheter with 5 % dextrose before injecting NBCA.
- Inject under long angiographic digital subtraction angiography run.
- Injections usually take between 1 and 3 s, but occasionally longer injections are appropriate.
- When NBCA cast is achieved, gently aspirate microcatheter and briskly remove from the patient.
- Perform immediate guide catheter angiography to evaluate the nidus, draining veins, and complications.

### Management of Associated Aneurysms

- If aneurysms are present on a feeding vessel, consider aneurysm occlusion first because embolization of the feeding pedicle will increase the perfusion pressure and may risk aneurysm rupture. Removal of the microcatheter after embolization also risks traction on the aneurysm.

## Post-procedural Care

- *Hydration*: Keep the patient well hydrated – 2L N saline in 24 h.
- *Close monitoring*: Patient may be extubated immediately post-procedure; they optimally should be monitored in Neuro ITU or HDU for 24 h.
- *Blood pressure control*: Keep mildly hypotensive. General rule is to decrease post-procedural mean arterial pressure by 20 % to minimize NPBB (see later). Nicardipine and labetalol are the recommended antihypertensive agents.
- *Steroids*: Consider dexamethasone 4 mg qds for 3 days in larger AVMs, reducing over 1 week to reduce risk of perilesional edema/inflammatory response.
- *Antithrombotics*: Heparin may be considered for sluggish venous outflow which could otherwise lead to venous thrombosis. Some use aspirin and some anticoagulate for 3–6 months post-procedure. (The author does not anticoagulate but keeps the patients well hydrated for 24 h post-procedural with intravenous fluids.)
- After Onyx, the patient may notice a garlic-like taste, which may last for hours, and a characteristic odor, which may last for 1–2 days.

## Complications

- The reported incidence of procedure-related complications (similar using either Onyx or NBCA) varies between 3 and 25 % and almost certainly reflects the complexity/grading of the AVM. Many deficits improve with time.
- Permanent deficit is around 8–10 % (*per patient*) (*probably higher with grade IV and V*).
- Procedure-related mortality is around 2–4 % (*per patient*) (*largely due to ICH*).

### Hemorrhage (ICH)

Reported in 2–18 % using Onyx (similar using NBCA) and usually happens within hours to days post-procedure:

- Clinical picture ranges from asymptomatic to headaches to severe/life-threatening. Emergency craniotomy may be required – prompt surgical backup is essential.
- Many potential causes of post-procedural hemorrhage (Table 5).

### Ischemia

Ischemia usually results from reflux of embolic material into normal arteries (despite heparinized flushes). Rarely due to microcatheter rupture – can result in proximal deposition of embolic material/closure of nontarget arteries.

**Table 5** Potential causes of post-procedural hemorrhage

| Cause | Prevention |
| --- | --- |
| Intranidal aneurysm rupture | Target intranidal aneurysm |
| Subtotal nidal occlusion with venous outflow obstruction/thrombosis | Avoid venous escape of embolic material |
| | Consider continuing to complete obliteration if venous outflow blocked |
| | Hemodilution/consider IV heparin |
| Substantial eradication of the AVM leading to venous stasis/thrombosis | Do not embolize more than 1/3 AVM volume at a time |
| | Hemodilution/consider IV heparin |
| Vessel perforation during navigation | Do not put microguidewire out through the catheter tip near to the nidus |
| Forceful microcatheter angiogram | Gentle microcatheter angiography/avoid injecting at bends |
| Tearing vessel on catheter retrieval | Use detachable tip microcatheter/be prepared to leave microcatheter in situ |
| Normal perfusion pressure breakthrough[a] | Suppress cerebral perfusion pressure by reducing arterial pressure |
| Vascular inflammation/necrosis secondary to embolic material | Steroids may help? |

[a]NPPB tends to occur following excessive embolization in patients with high-flow, large AVMs with multiple large feeders and is considered due to a sudden increase in perfusion pressure in the surrounding normal brain parenchyma which has impaired autoregulation due to chronic hypoperfusion. Avoid by limiting embolization to 30 % of the nidus per session and actively managing periprocedural blood pressure

## Microcatheter Retention

Reported in <3 % of embolizations with either Onyx or NBCA – attempts to retrieve catheter result in unacceptable deformity of cerebral arteries/hemodynamic changes/vasospasm:

- More common with:
  - Long injections
    - 2 cm reflux
  - Tortuous distal arterial loops
- Microcatheter retention may be less likely to occur with the newer detachable tip catheters (Apollo, Sonic).

## Radiation Injury

Prolonged intranidal injections and multiple staged procedures especially those with Onyx are associated with high radiation doses which may cause transient hair loss or erythema. Try to alter the embolization projection for subsequent procedures/modify imaging parameters where possible.

## Follow-Up

Angiographically "cured" lesions require catheter angiography at 3–6 months after final embolization. Where Onyx is the primary embolic agent, the author considers 1-year/5-year catheter angiography as late recurrences have been described.

Imaging follow-up after STRS/surgically treated lesions varies between centers.

At the author's center, early postoperative angiography is only performed if there are surgical concerns. Otherwise, a single angiogram is obtained at 3 months.

Following STRS, MRI/A is performed 2 years post-STRS. If the AVM appears obliterated, cerebral angiography is performed. If obliterated, stop. If not, the process is repeated at 3 and if necessary, 4 years. If not obliterated beyond 3 years, other treatment options are considered, according to the Sheffield protocol. In our experience, if the AVM has not substantially changed by 2 years, it is unlikely to obliterate by 4 years. Angiographic cure is considered obliteration of early venous drainage (some abnormal vessels usually persist).

## Alternative Treatments

### Stereotactic Radio Surgery (STRS) (Gamma Knife)

STRS generates vascular injury and induces subsequent thrombosis.

- Useful for small AVMs (<10 ml volume, <3 cm diameter) especially in critical areas of the brain where the surgical risk is high.
- May improve seizure control – 2/3 may be seizure-free.
- Major disadvantage of STRS is the persistent risk of ICH until the lesion obliterates (up to 4 years).
- Adverse effects – radiation necrosis, intracranial arterial stenoses, and cranial nerve injury – more likely with increasing dose/deep AVMs.
- Permanent treatment-related deficits reported in around 6–12 %.
- Transient deficits occur in an additional 6 %.
- Mortality rates are 4–9 %.
- The estimated cure at 2 years 80–88 % for AVMs <10 ml.
- Cure rate for larger AVMs considerably lower.
- Post-STRS, absence of residual angiographic AVM nidus or AV shunting may not equate with definite permanent AVM obliteration – suspect tiny residuum if enhancement is demonstrated on gadolinium-enhanced MRI.

### Surgical Resection

This is usually an elective procedure, even following ICH. The surgical risks in expert hands are outlined in the Spetzler-Martin grading system (Tables 1 and 2) and reported in a review on AVMs (Baskaya et al. 2006). Angiographic cure rate is quoted as 94–100 %.

**Key Points**

- Management of cerebral AVMs requires a strategy agreed upon by an experienced neurovascular MDT.
- Treatment of unruptured AVMs is controversial and risk of treatment may outweigh natural history particularly for Spetzler-Martin grades IV and V.
- Indications for treatment include primarily ICH, but also progressive neurological deficit, intractable seizures, and other disabling symptoms.
- Annual bleeding risks vary from 0.9 to 34.4 % and depend upon presentation, location, and venous drainage.
- Embolization alone cures a small number of AVMs (usually <3 cm, small number of feeding pedicles, etc.) and is more often performed as a preoperative adjunct or to reduce the size to facilitate STRS. Palliative embolization is controversial.
- Liquid embolics are preferred. Onyx is more predictable and controllable than NBCA.
- In general, embolize less than 1/3 of the volume at each session to avoid NPPB and venous stasis/thrombosis.
- Complications as a result of embolization occur in up to 25 % of procedures resulting in on average 8–10 % permanent deficit per patient (probably higher in SM IV and V) and 2–4 % mortality (Fig. 1).
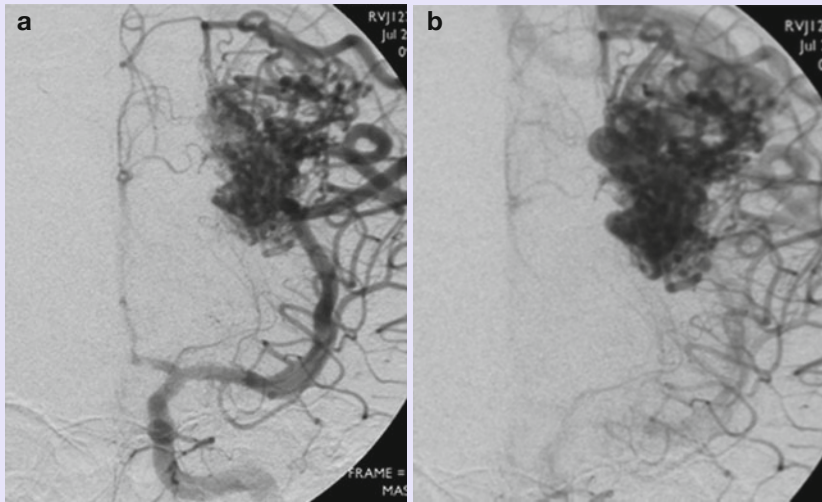
**Fig. 1** These images demonstrate serial embolization of an unruptured left occipital AVM in a 17-year-old boy with seizures who wanted treatment. He had been discussed at a neurovascular MDT, and all risks had been thoroughly explained prior to starting the first embolization. The aim here was to reduce the size of the AVM to facilitate STRS and so achieve complete obliteration. The AVM is supplied by distal branches of the left MCA (**a**–**d**) and temporal branches of the left PCA (**e**, **f**). Enlarged collaterals from the distal PCA to MCA also supplied the AVM (**e**, **n**). Venous drainage is superficial. There were no venous stenoses. Intranidal aneurysms were seen in a medial component supplied by the left temporal branch of the PCA (e, *arrow*). A Marathon microcatheter (ev3-Covidien) was navigated down a distal left MCA feeder and a satisfactory position obtained very close to the nidus, without any normal branches arising in the vicinity (**g**, **h**). The position allowed 2 cm Onyx reflux without compromise of any arterial branches supplying normal brain. The best projection for Onyx injection was frontal (**g**). The path of the microcatheter distally is well demonstrated and is clear of the nidus allowing reflux to be readily visualized. During the first embolization, approximately 4 ml Onyx 18 was injected without complication obliterating a little under one-third of the AVM (**i**). Note the Onyx cast filling the nidus (**j**). It was intended to occlude the intranidal aneurysm component during the second session (*arrow*, **k**), but a satisfactory position from the temporal PCA feeder could not be achieved; there were too many normal vessels supplying normal brain in the vicinity (**l**, **m**). Superselective injections into the distal PCA-MCA collaterals also suggested that this route also was not amenable to safe, adequate embolization (**n**). A satisfactory position for the second embolization was therefore achieved by selective catheterization of another MCA feeder (**o**, **p**). Five milliliter Onyx 18 was injected again using the frontal projection as the optimal projection for assessing nidal embolization, Onyx reflux, etc. It is interesting to note that during this embolization, Onyx passed from the MCA component of the AVM into the temporal PCA component obliterating this component and the intranidal aneurysms (**q**). At the end of the second embolization, there was residual supply from PCA-MCA collaterals (**q**) and distal MCA feeders (**r**). Progressive AVM thrombosis was demonstrated in the interval between the second and third embolizations. Compare (**r**) with (**s**) and (**q**) with (**t**). After the third embolization, the patient was referred for STRS as the AVM had now been significantly reduced in size and the demonstrated intranidal aneurysms had been obliterated
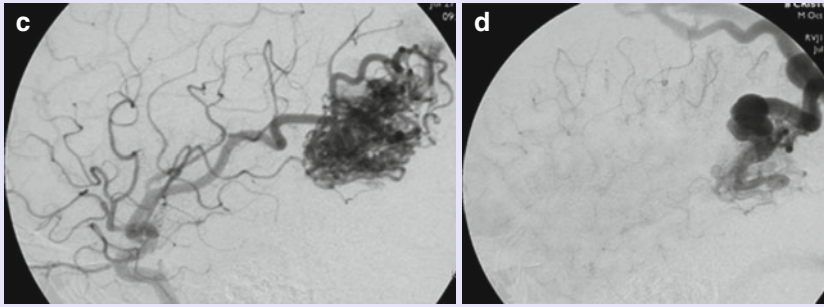
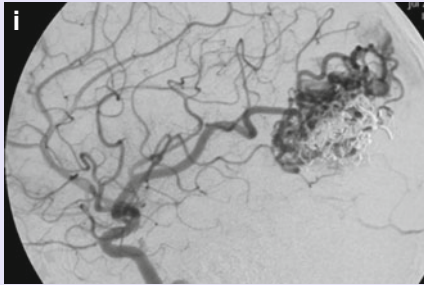Left internal carotid angiogram: frontal (a,b) and lateral (c,d) projections
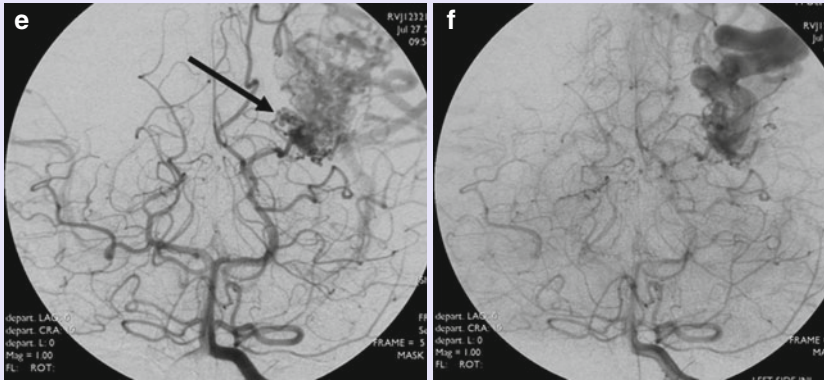
Left vertebral angiogram: frontal projections
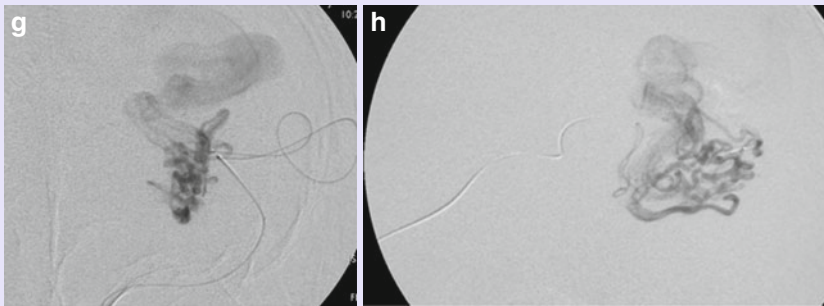
first embolisation

superselective angiograms in left MCA feeder:
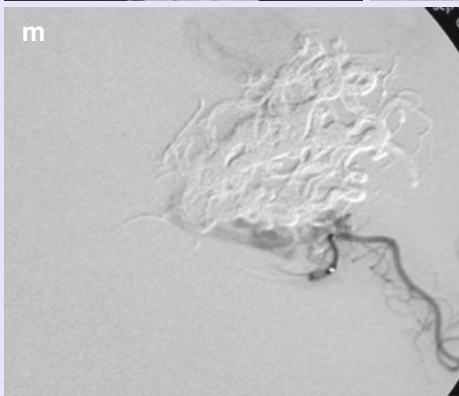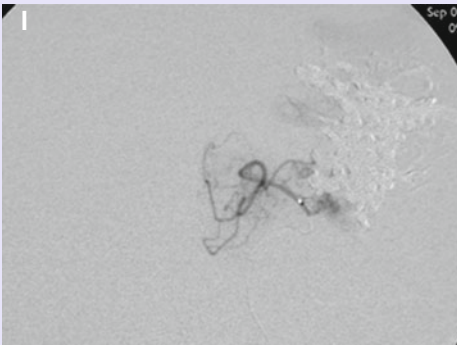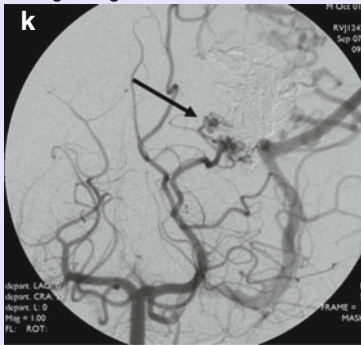frontal projection (g) lateral projection (h)
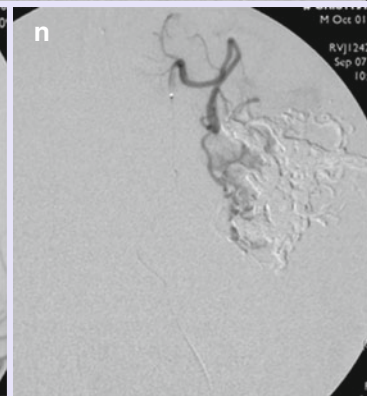
**Fig. 1** (continued)

Lateral left ICA angiogram at the end of the first embolisation

Frontal projection:
Left vertebral angiogram:
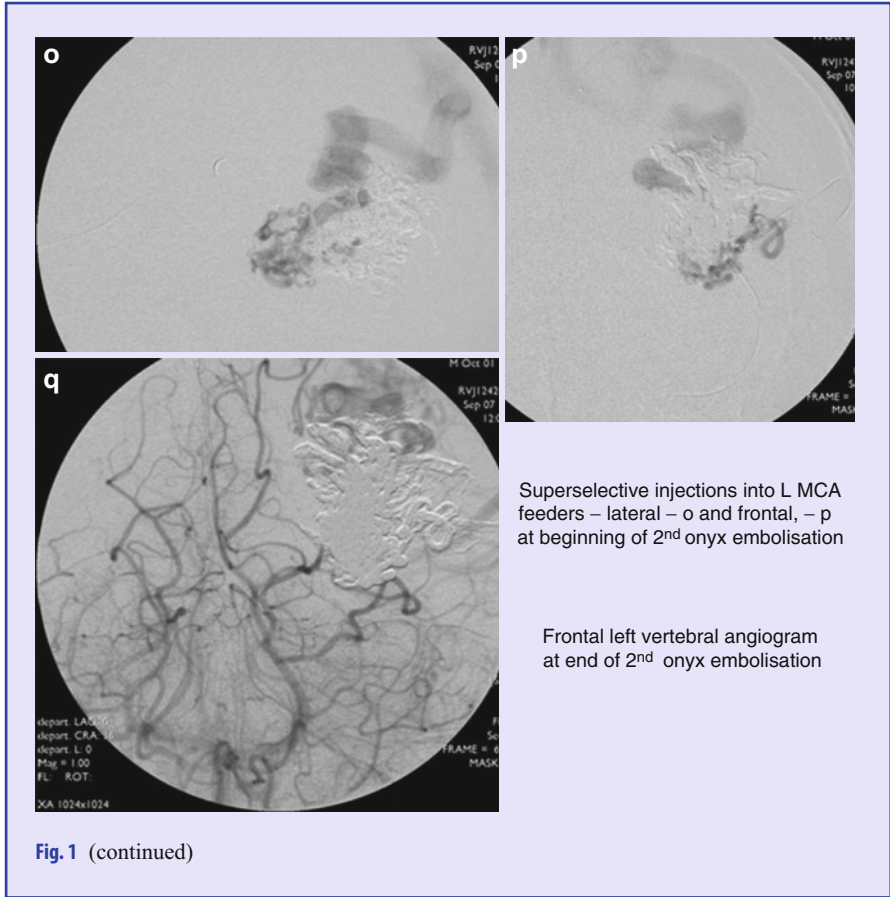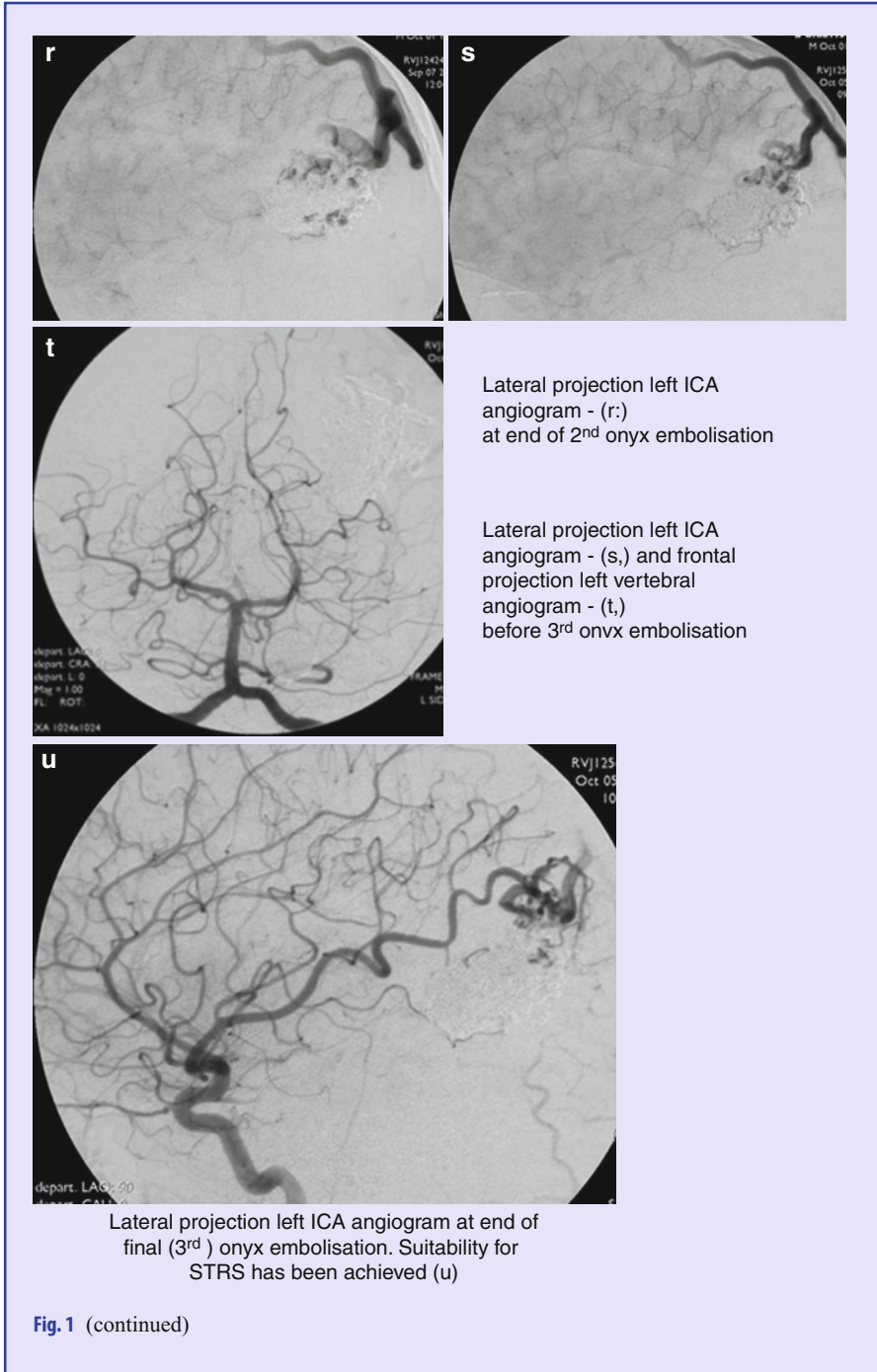beginning of second session



Superselective injections into L PCA
temporal feeders −1, m

Superselective injections into
L PCA − MCA  collateral feeders − n

**Fig. 1** (continued)

Superselective injections into L MCA feeders – lateral – o and frontal, – p at beginning of 2nd onyx embolisation

Frontal left vertebral angiogram at end of 2nd onyx embolisation

**Fig. 1** (continued)

Lateral projection left ICA
angiogram - (r:)
at end of 2nd onyx embolisation

Lateral projection left ICA
angiogram - (s,) and frontal
projection left vertebral
angiogram - (t,)
before 3rd onvx embolisation

Lateral projection left ICA angiogram at end of
final (3rd ) onyx embolisation. Suitability for
STRS has been achieved (u)

**Fig. 1** (continued)

## Suggested Reading

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8. Strake RM, Komotar RJ, Otten M, et al. Adjuvant embolization with N-butyl-cyanoacrylate in the treatment of cerebral arteriovenous malformations: outcomes, complications and predictors of neurological deficit. Stroke. 2009;40:2783–90.
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