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Interventional treatment of arterio-venous malformations

Arterio-venous malformations (AVM) are one of various congenital vascular malformations that result from a failure of orderly resorption of the primitive blood vessels in weeks 4–6 of gestation involving the vessels of both arterial and venous origins and resulting in high-flow direct AV communications between different sized vessels (AVMs), vein malformations, and lymphatic malformations. An AVM is defined as a high flow, low resistance communication between the arterial and venous systems without an intervening normal capillary system. Thus, the pivotal function of the capillary system—which is to maintain the delicate balance between the high-pressure (arterial) and low-pressure (venous) system—is not present, thereby, causing arterialized venous pressure increases secondary to the AV shunts, and the tissue normal venous drainage slows and becomes delayed having difficulty competing with the higher pressure arterialized veins. This leads to tissue edema due to venous hypertension [1].

Peripheral AVMs are the least common type representing 5–20% of all congenital vascular malformations. Most of the current data regarding incidence and prevalence of AVMs are based on cerebrospinal AVMs. They are rare and usually go undetected until clinical symptoms appear in patients aged 20–40 years [2].

AVMs are the most complex type of congenital vascular malformations with significant hemodynamic alterations to both the arterial and venous systems, which if they are large and centrally positioned in the body (e.g., shoulder, chest, abdomen, pelvis, buttock) can induce high flow cardiac output failure (right heart volume overload), arterial insufficiency (steal) in the affected tissue, and chronic venous hypertension.

Classification

Two classifications were proposed to improve AVM management: the Schobinger clinical classification and an arteriographic classification. The Schobinger classification (**Tab. 1**) was designed to assess AVM lesions in different clinical stages and clinical conditions more accurately based on the patient's clinical status and to select the best suited time for management as a practical guideline.

The arteriographic classification of AVMs was proposed to better define potential treatment strategies. Several classification systems have been published. A cerebrovascular congenital AVM classification system published in 1993 [3] and a peripheral vascular AVM classification system [4, 5] published in 2006 are strikingly similar regarding the angioarchitecture of high-flow AVMs.

The arteriographic classification was further adjusted by angioarchitecture additions not described in these two classification systems by W. Yakes, adding the Yakes type I [direct artery-to-vein connections, e.g., as is seen in pulmonary arteriovenous fistulas (AVFs)] and Yakes type IV AVMs (characterized by a network of innumerable AVFs without a defined nidus infiltrating entire tissue with capillary beds interspersed among the innumerable AVFs) and a further modification of the previous two classification systems with Yakes types II, IIIa, and IIIb (**Tab. 2**, **Fig. 1**).

Management

AVMs remain the most challenging malformation among various congenital vascular malformations due to their potential impact on the cardiovascular system in

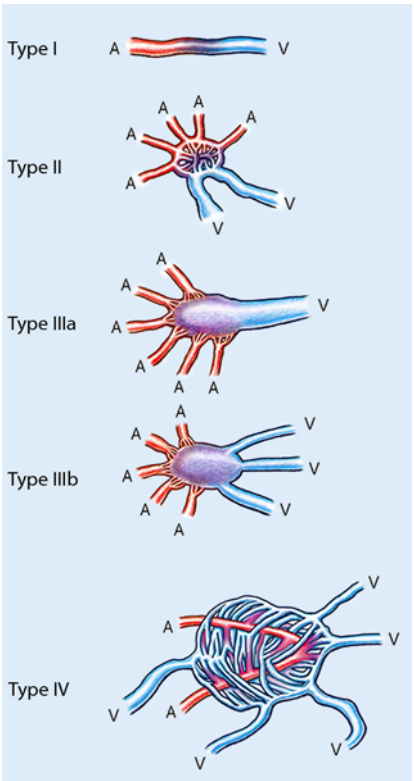

Fig. 1 ▲ Angioarchitecture of high-flow arteriovenous malformations. *A* artery, *V* vene

Tab. 1	Schobinger clinical classification of AVMs symptomatology
Stage I	Quiescence: may or may not have a vascular skin stain, warmth of the affected tissues, and AV shunts can be detected by Doppler US scanning. The AVM is present but causes no clinical symptoms
Stage II	Expansion of the AVM lesion: stage I plus enlargement, pulsations, palpable thrill, audible bruit and enlarged arterialized tortuous/tense veins
Stage III	Destructive tissue changes: stage II plus dystrophic skin changes, skin ulcerations that can be nonhealing, bleeding from the ulcerated areas in the skin or mucosal surfaces, overt tissue necrosis, and lytic lesions of bone may occur
Stage IV	Decompensation: stage III plus congestive cardiac failure with increased cardiac output, abnormally lowered PVR, and venous hypertension secondary tissue and skin changes

AV arteriovenous, *AVM* arteriovenous malformation, *PVR* peripheral vascular resistance, *US* ultrasound.

Tab. 2	Yakes classification of high-flow AVM angioarchitecture [6]
Type I	A direct artery/arteriole to vein/venule connection *Direct AV connections can be treated with fibered coils, wires, Amplatzer plugs. Mechanical device occlusion can be curative*
Type II	Multiple arteries/arterioles connecting through an intervening "nidus" without any intervening capillary beds draining into multiple out-flow veins. *Ethanol embolization superselectively delivered by transcatheter and direct puncture techniques is curative*
Type IIIa	Multiple inflow arterioles shunting into an aneurysmal vein that has a single vein outflow. (AV fistulae which infiltrate the wall of a draining aneurysmal vein with single outflow vein.) *Ethanol and/or coils can be curative*
Type IIIb	Multiple inflow arterioles shunting into an aneurysmal vein with multiple outflow veins. (AV fistulae which infiltrate the wall of the aneurysmal vein with multiple outflow veins). *More challenging to be treated with coils as multiple veins must be occluded*
Type IV	Multiple arteries/arterioles which form innumerable microfistulae that diffusely infiltrate the affected tissue. Interspersed within these innumerable AVFs are capillary beds that maintain the viability of the affected tissue. The innumerable AVF drain into multiple veins. Venous hypertensive changes can occur as the capillary bed out-flow veins have greatly restricted venous access competing with the arterialized AVM draining veins that have arterialized venous pressures (Schobinger II and III changes). *A 50–50% mixture of ethanol and non-ionic contrast can be curative in this lesion type*

the affected tissue and the subsequent hemodynamic consequences related to arterial steal and venous hypertensive changes. Clinical manifestations associated with AVM are dependent on the anatomical location and may produce cardiac failure, local venous hypertension with abnormal tissue changes, and arterial steal. In addition, local effects of AVMs may include ulcerations that may be nonhealing and chronic, bleeding and infections in those ulcerated tissues, necrosis of the affected tissues, and gangrene if the arterial vascular steal is significant.

Hormonal factors known to trigger progression of clinical symptoms in vascular malformations that have been previously quiescent include the onset of puberty/menarche, pregnancy, birth control pills, direct trauma, surgery with partial resection of the AVM lesion, proximal ligation of prominent feeding arteries, "skeletonization" surgical exclusion procedures, etc. All these produce neovascular stimulation enlarging the AVM and worsening its symptoms in the Schobinger classification.

An ill-planned and improper treatment strategy (incomplete resection, surgical ligation/skeletonization and/or proximal coil embolization of the feeding arteries) can stimulate an AVM lesion to transform from a dormant state to a proliferative state, resulting in AVM growth with advancement of the Schobinger classification to higher stages of symptomotology.

The main goal of treatment should be to elimination the nidus by vascular occlusion procedures or complete surgical extirpation of the AVM. Partial resection will result in lesion recurrence and enlargement with increased symptoms by neovascular stimulation.

Overview of endovascular occlusive procedures

Endovascular therapy with various embolization and sclerotherapy modalities is reported in the literature by many vascular malformation centers as the preferred therapeutic option for the majority of AVM lesions, rather than surgical extirpation [7–10]. Precise delivery of the embolic agent to ablate the AVM nidus is required for successful endovascular therapy, to minimize complications, and to achieve a long-term cure.

A combination of approaches utilizing any of the three routes of delivery of embolic agents (transarterial, transvenous, direct puncture) may be required to ablate the AVM. Multisession endovascular therapy is preferred and every effort should be exercised to minimize the risks of embolo-sclerotherapy during each session. The most appropriate embolic agents for primary control of AVM include transcatheter embolization, direct puncture embolization, and retrograde vein embolization approaches primarily using ethanol in Yakes types II, IIIa, and IIIb AVMs. Coils and other mechanically occlusive devices can be curative in Yakes type I AVFs. Coils can also lessen complications when densely packed in the aneurysmal veins of Yakes types IIIa and IIIb AVMs. In Yakes type IV AVMs, a 50–50% mixture of ethanol and contrast as an embolic agent is curative in these infiltrative lesions and can spare the capillary beds from occlusion. The use of nBCA (n-butyl-2-cyanoacrylate) and/or Onyx are completely inadequate to cure or provide long-term control for AVMs as a primary form of therapy. In the literature, it is reported that these polymerizing embolic agents are palliative at best and recurrences are common [11–15].

Embolic agents

Many embolic agents have been used in the treatment of AVMs. Particulate agents, which include polyvinyl alcohol (PVA) particles, microspheres, gelfoam, and collagen powders, have been used to embolize AVMs. Their use, alone or in combination with other agents, is well documented in the literature and is palliative with high rates of recurrences of the AVM [16–21].

Unfortunately, these agents do not possess properties that are well suited for treating AVMs. The particles are often either too large and occlude the vessels proximal to the nidus, or too small traveling through the AV shunt causing nontarget embolization. Since they are not well suited to treat AVMs, their primary purpose is to alter the hemodynamics of the lesion to improve the possibility for surgical resection, which is difficult to achieve.

Fibered and non-fibered coils

Coils are designed to mechanically occlude larger vessels and are not able to penetrate into the AVM nidus, as they are an endovascular occluding device sized to the diameter of the artery/vein that is being embolized. When used as a transarterial embolic occlusive device, the result is proximal arterial ligation that is not curative and will cause neovascular stimulation of the AVM, thus, stimulating growth of new arteries/arterioles to the AVM and worsening of the situation [22].

A major disadvantage of coil embolization therapy is that its mechanism of action is limited to the occlusion and subsequent thrombosis of the artery or vein in which it is placed. Permanent damage to the blood vessel endothelium does not occur, thus, allowing for subsequent regeneration or recovery of the endothelium. This can result in recanalization with recurrence of the lesion.

However, coils can be extremely effective and curative in AVM treatment when placed in the aneurysmal outflow veins and is a definitive treatment. This is particularly effective where there is a single AV connection (Yakes type I) and multiple arteries connect to a single draining vein (Yakes types IIIa/IIIb AVM lesions). In these types of AVM, occlusion of the venous outflow with coils can be very effective and dramatically reduces the risk of tissue injury and complications associated with transarterial ethanol embolization.

In AVMs that have an aneurysmal dilatation of the draining vein, with single outflow vein (Yakes IIIa) and multiple outflow veins arising from it (Yakes type IIIb), coil embolization can be used in combination with ethanol to achieve a definitive treatment. Once flow has been slowed in the outflow veins by the placement of the coils, the injection of absolute ethanol can then reflux into the many vein fistulae in the wall of this aneurysmal vein to allow permanent occlusion of the AVM. This "retrograde vein occlusion" technique in curative treatment of high-flow vascular malformations was first described by Yakes et al. in 1990 [23] and later confirmed by Jackson et al. [24] and Cho et al. [25].

Absolute ethanol

The curative potential of absolute ethanol as an AVM embolic/sclerosant agent lies in the fact that it destroys the endothelial cells on the vascular wall by precipitating its protoplasm (endothelial cells lining arteries, veins, capillaries, lymphatics) and causes fractures of the vascular wall to the level of the internal elastic lamina. Because the endothelial cells are destroyed, the phenomena of neovascular stimulation with new vascular inflow due to se-

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Abstract

Background. Due to their hemodynamic effects and tendency to progress, the majority of congenital arterio-venous malformations (AVM) require treatment. AVM are classified according to clinical severity (Schobinger classification, stages I–V) and angiographic appearance (types I–IV).

Treatment. Endovascular embolization is the treatment of choice. However, because complications such as necrosis and neuropathy occur in up to 15% of cases, treatment is challenging and is dependent on the angiographic appearance. Critical for treatment success is elimination of the so-called nidus, which is the location of the short circuit connection between the artery and the often extended aneurysm-like drainage vein. Embolization of only the feeding artery without the actual nidus or the incomplete elimination of drainage veins should be avoided. Absolute 96% alcohol in addition to coiling of the nidus has been established as the most effective technique.

Conclusion. Unlike other embolic agents, alcohol leads to a definitive destruction of the vessel wall. Recanalization and recurrence are excluded due to adequate elimination of the nidus.

Keywords

AVM classification · Angioarchitecture · Coils · Congenital vascular malformation · Ethanol embolization

Interventionelle Behandlung von arteriovenösen Fehlbildungen

Zusammenfassung

Hintergrund. Angeborene arteriovenöse Malformationen (AVM) stellen aufgrund ihrer hämodynamischen Auswirkungen und Progressionstendenz mehrheitlich eine Therapieindikation dar. AVM werden nach ihrem klinischen Schweregrad (Schobinger-Klassifikation, Stadium I–V) und ihrem angiographischen Erscheinungsbild (Typ I–IV) eingeteilt.

Behandlung. Therapie der Wahl ist die kathetertechnische Embolisation wobei die Behandlung aufgrund der hohen Komplikationsraten mit Nekrosen und Neuropathien in bis zu 15% der Fälle anspruchsvoll ist, und sich die Technik nach dem angiographischen Erscheinungsbild richtet. Entscheidend für den Therapieerfolg ist die Ausschaltung des sog. „Nidus", der den Ort der Kurzschlussverbindung zwischen der Arterie und der oft aneurysmartisch erweiterten Drainagevene darstellt. Zu vermeiden ist die alleinige Embolisation der zuführenden Arterie ohne den eigentlichen Nidus zu erreichen oder die inkomplette Ausschaltung von Drainagevenen. Absoluter 96%-iger Alkohol in Verbindung mit einem Coiling des Nidus ist als die effektivste Technik etabliert.

Schlussfolgerung. Im Gegensatz zu anderen Embolisaten führt Alkohol zu einer definitiven Zerstörung der Gefäßwand, wodurch eine Rekanalisation und ein Auftreten eines Rezidivs bei adäquater Ausschaltung des Nidus ausgeschlossen ist.

Schlüsselwörter

AVM-Klassifikation · Angioarchitektur · Coils · Kongenitale vaskuläre Fehlbildungen · Alkohol

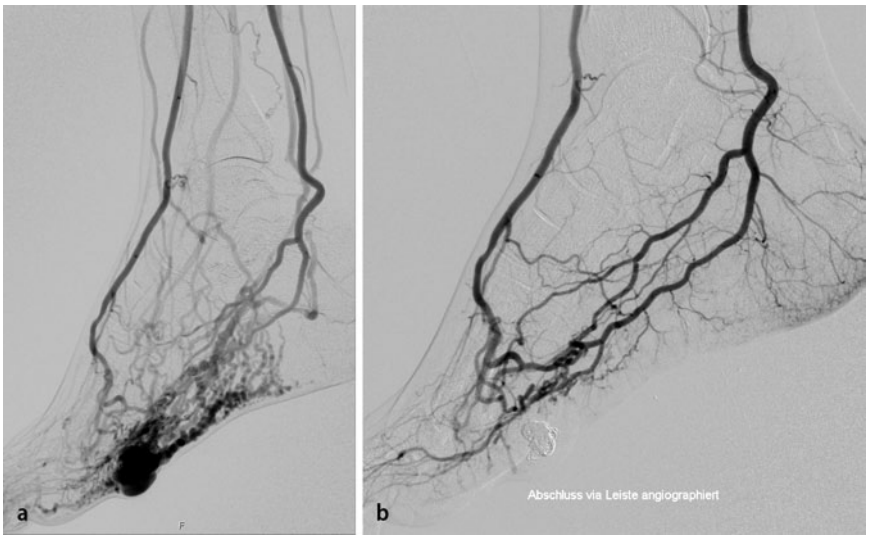


Fig. 2 ▲ Type IIIb arterio-venous malformation **a** before and **b** after combined percutaneous ethanol embolization (21 ml, 96% ethanol) and coiling (12 Nester® coils) of the aneurysmal vein that has a single vein out-flow. Total cure was achieved in a single procedure

cretion of angiogenesis factors, and the recanalization phenomenon due to secretion of chemotactic cellular factor to carry out intravascular debris, is noticeably absent and the potential for a permanent occlusion is now possible [26–34].

Because the endothelial cells are denuded from the vascular wall, its protoplasm precipitated, and the vascular wall is fractured to the level of the internal elastic lamina, platelet aggregation occurs on the denuded vascular wall surface. This thrombotic process progressively occludes the vascular lumen from the vascular wall surface to the central lumen. There will be no more chemotactic cellular factors and angiogenesis factors secreted since the endothelial cells are completely destroyed [35].

However, the risk of cardiopulmonary complications during ethanol sclerotherapy administration is significant; therefore, appropriate measures should be taken which include administration of general anesthesia and close cardiopulmonary monitoring. Use of a pulmonary artery catheter during ethanol sclerotherapy will allow continuous pulmonary artery pressure monitoring. Shin et al. [36] reported that if no more than 0.14 ml ethanol/kg body weight is embolized every 10 min, no cardiopulmonary complications will occur [37, 38].

Pulmonary hypertension is a potentially fatal complication associated with ethanol sclerotherapy and occurs when a significant dose of ethanol is allowed to reach to the lungs. The etiology of pulmonary hypertension is felt to be related to either pulmonary arterial spasm or extensive microthromboembolization. The development of pulmonary hypertension can lead to subsequent cardiopulmonary arrest if not controlled effectively.

A total dose of ethanol used during an embolization procedure should be less than 1 ml/kg, since volumes greater than this can be toxic. Limiting ethanol injections to 0.14 ml ethanol/kg ideal body weight every 10 min will be able to obviate the need of a pulmonary artery catheter when anticipating large injections of ethanol in large lesions [39]. By adhering to these principles, the risk of ethanol flowing to the pulmonary circulation, thus, causing pulmonary vascular spasm with subsequent acute right heart failure, is obviated.

Because absolute ethanol is associated with various complications and morbidity, safe use of ethanol in AVM embolization requires accurate delivery into the nidus by precise placement solely in the AVM nidus vasculature that is nonnutritive and without capillaries. Proximal injection of ethanol into a feeding artery would cause severe tissue necrosis by destroying nutritive capillary beds.

In order to enhance the denaturating or sclerosing effect on the endothelial cells, lowering the flow in the AVM itself is a highly effective technique to allow the injected ethanol to remain in longer contact with the cells. Decreasing the flow through the lesion can be achieved in different ways (◘ **Fig. 2**):

- arterial approach: this approach can make use of occlusion balloons,
- direct puncture injection of the sclerosing material, or
- direct puncture or retrograde transcatheter venous approach. Occlusion of the aneurysmal vein with coils can be curative.

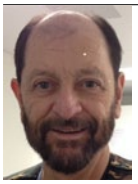
To decrease postembolization swelling in the endovascularly treated area, intravenous dexamethasone is routinely used prior to the procedure as well as nonsteroidal anti-inflammatory agents and steroid therapy for 5 days postprocedure, thus, greatly reducing the risk of developing compartment syndrome or subsequent possible nerve injuries due to edema. Accurate and meticulous embolization techniques, treating *only* the AVM, total avoidance of ethanol embolization of capillary beds, use of fibered coils in the vein approach in Yakes types I, IIIa, IIIb AVMs, will greatly diminish complications from vascular malformation endovascular treatment procedures in these complex vascular lesions [40, 41].

Conclusion

- **Due to their hemodynamic effects and tendency to progress, the majority of congenital arterio-venous malformations (AVM) require treatment.**
- **AVM are classified according to clinical severity (Schobinger classification, stages I–V) and angiographic appearance (types I–IV).**
- **Depending on the type of AVM, combined intraarterial, percutaneous or transvenous embolization is the treatment of choice, but requires specialized interventional expertise.**
- **Important for treatment success is the elimination of the nidus, whereby the interventional treatment of congenital AVM differ from other embolization treatments.**

- **The use of 96% alcohol is the most effective, but also associated with the most complications.**
- **Transvenous or percutaneous coiling is a treatment option in AVM with an aneurysm-like nidus.**

Corresponding address



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Compliance with ethical guidelines

Conflict of interest. W. Yakes and I. Baumgartner state that there are no conflicts of interest.

The accompanying manuscript does not include studies on humans or animals.

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