Cardiovasc Intervent Radiol (1996) 19:323–328

CardioVascular and Interventional Radiology

Treatment of High-Flow Vascular Malformations by Venous Embolization Aided by Flow Occlusion Techniques

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Abstract

Purpose: Transvenous embolization techniques may be helpful as alternatives to the arterial route when treating high-flow vascular malformations. We present our experience using these techniques in four patients.

Methods: In one patient the venous portion of the arteriovenous malformation (AVM) was punctured directly; in the other three patients it was catheterized via a retrograde venous approach. Flow occlusion techniques were utilized in all patients during embolization, which was performed with absolute alcohol or *N*-butyl-2-cyanoacrylate.

Results: Excellent clinical and angiographic results were obtained, with obliteration of arteriovenous shunting in all patients. There were no complications.

Conclusion: The embolization of certain AVMs using a venous approach is a safe and effective treatment.

Key words: Arteriovenous malformations—Arteries—Therapeutic blockade

Vascular malformations may be classified into arterial, venous, lymphatic, or mixed lesions depending upon the type of vessel which is predominantly involved. Arterial lesions, or arteriovenous malformations (AVMs) as they are more usually known, exhibit arterial hypertrophy, rapid arteriovenous shunting, and secondary venous dilatation. While they may be asymptomatic, patients will commonly complain of pain (which may be severe), local hyperhidrosis, ulceration, and bleeding; when massive they may cause high-output cardiac failure.

Small, superficial AVMs can be cured by surgical excision. Unfortunately, most AVMs are large and diffuse in nature, involving important normal adjacent structures, and are therefore exceptionally difficult or impossible to excise. Arterial embolization will provide the mainstay of treatment in such cases but, while excellent palliation may be afforded using this technique, a cure is unlikely. New technology in the form of coaxial catheters, and the use of liquid embolic agents such as absolute alcohol [1], has allowed the embolization of lesions which were previously considered untreatable, with good clinical and radiological improvement. There are still some lesions, however, which are either extremely hazardous or impossible to treat via an arterial approach because of one or more of the following reasons: there may be multiple small feeding arteries supplying the lesion; important normal arterial branches may arise in very close proximity to a malformation; extreme arterial tortuosity may preclude successful catheterization; previous therapy (embolization or surgery) may have occluded arterial access to the central portion of the malformation. In such circumstances direct puncture or transvenous embolization techniques may be helpful [2]. While a retrograde venous approach has been used in the treatment of certain intracranial vascular malformations [3, 4], it is a rare, and perhaps underused, technique in peripheral high-flow vascular lesions.

We describe the treatment by embolization of high-flow vascular malformations in four patients. All the procedures were performed by occluding the venous side of the malformation with liquid agents aided by flow-occlusion techniques.

Materials and Methods

Four patients (1 male, 3 female), aged 12–31 years (mean 23 years) with high-flow vascular malformations were treated by embolization of the venous side of their AVMs. The anatomical location of these AVMs was the dorsum of the hand, the forearm, the nose/forehead,

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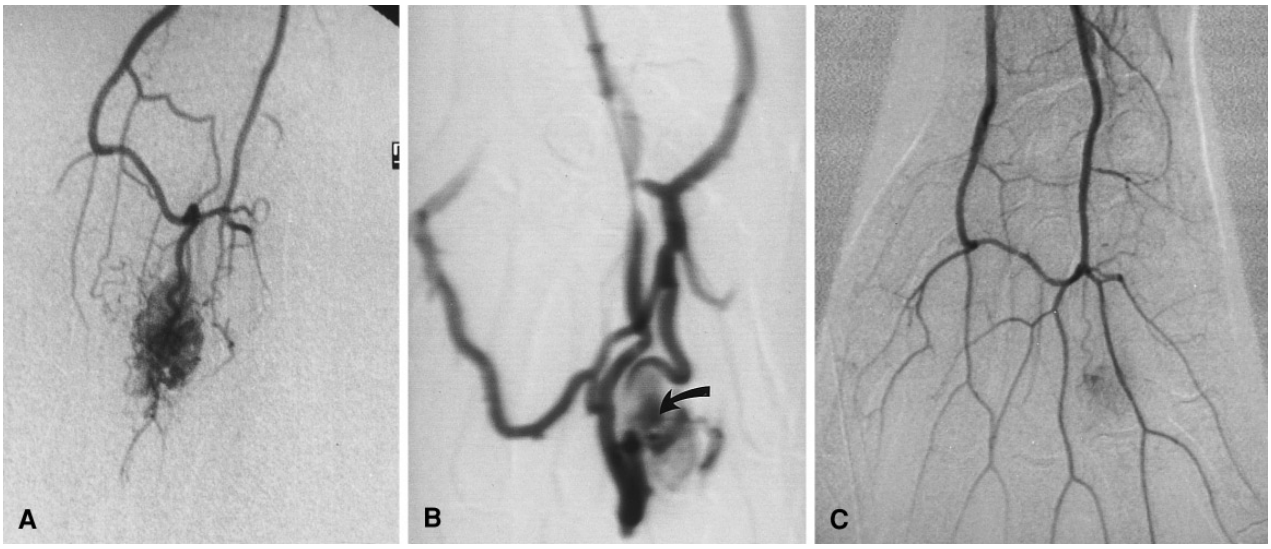

Fig. 1. Arteriovenous malformation (AVM) over dorsum of the left hand. **A** Early arterial phase shows multiple feeding vessels to large venous component arising from palmar arch and digital arteries. **B** A draining vein has been punctured over the wrist and venography performed with a tourniquet inflated to above arterial pressure. Venous component of the malformation is retrogradely opacified. Embolization was performed with 2.5 ml absolute alcohol via a 3 Fr coaxial catheter introduced close to the central venous component (arrow). **C** Arteriogram performed 6 months post-embolization. Arterial phase demonstrates vascular stain at site of AVM. There is no arteriovenous shunting and preservation of all normal arterial branches. The venous phase showed no abnormality.

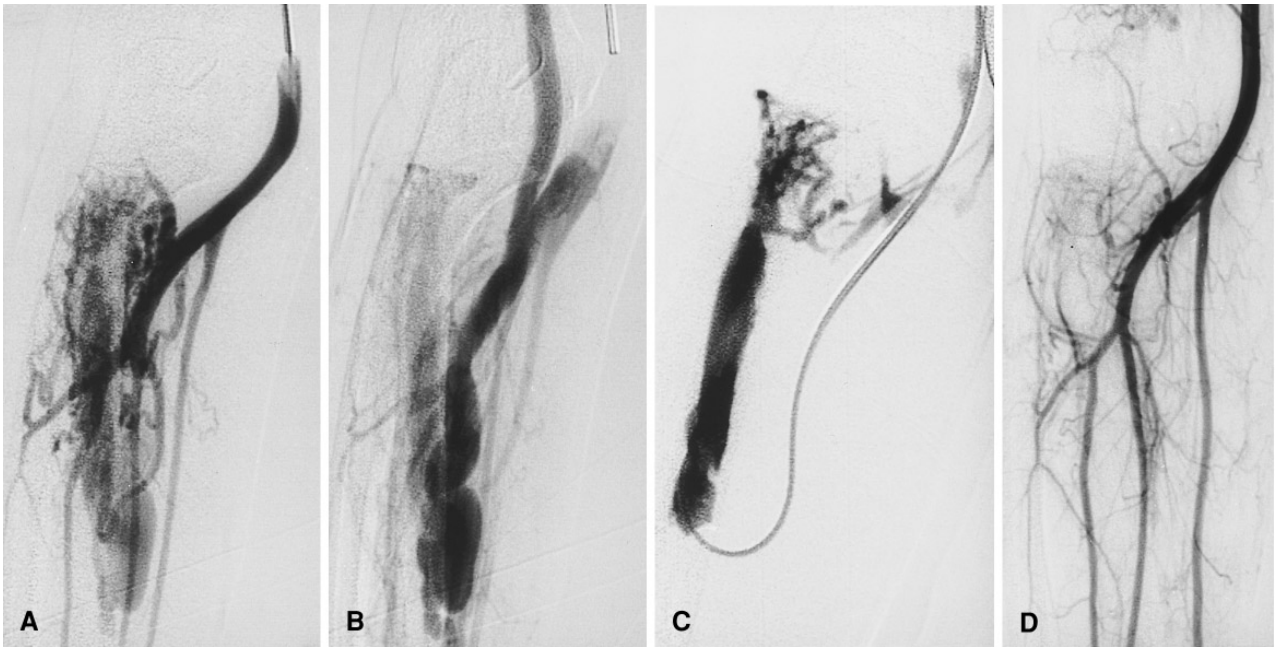

Fig. 2. Radial head AVM. **A** Multiple feeding vessels are seen supplying the large venous component of the AVM in the head and proximal shaft of radius. **B** Large draining vein is opacified later in the same run. **C** A catheter has been introduced via the draining vein into the venous component of the malformation within the radial shaft. Venogram was performed with a tourniquet inflated around the forearm to above arterial pressure. Embolization was performed with a total of 8 ml absolute alcohol in four divided doses. **D** Arteriogram performed 6 months post-embolization. Arterial phase shows no residual filling of the malformation. The venous phase was normal.

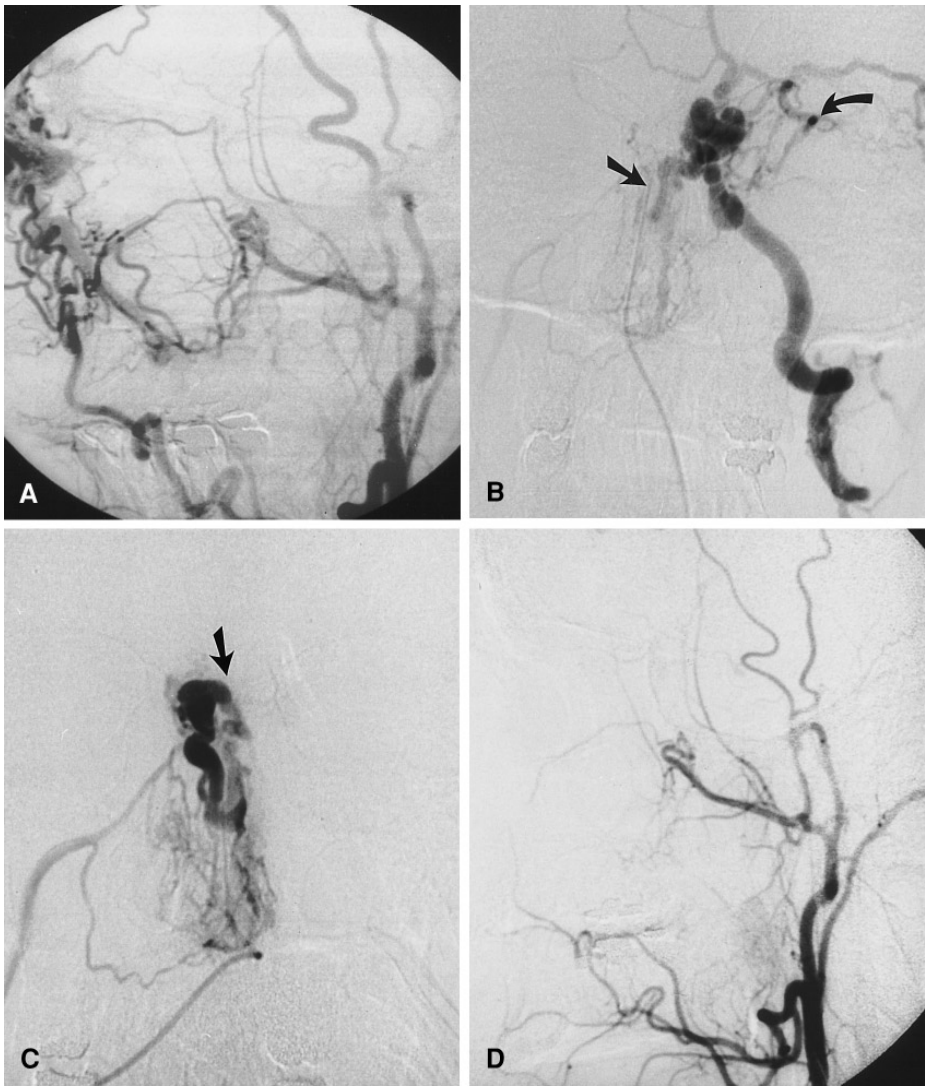

Fig. 3. AVM involving bridge of the nose. **A** Left external carotid arteriogram (lateral view). AVM is supplied by branches of facial, maxillary, and superficial temporal arteries. Corresponding vessels on the contralateral external carotid arteriogram also supplied the AVM, as did both ophthalmic arteries. **B** Needle (arrow) within the venous portion of AVM over the right side of nasal bridge (frontal view). Rapid venous drainage is seen into the enlarged left facial vein and also into the left ophthalmic vein (curved arrow). **C** Venogram in the same projection as **B**. Digital compression of left angular vein performed by the patient (arrow), with most of the contrast persisting within the venous portion of the malformation. Note some venous drainage via the right facial vein. Embolization performed with 1 ml of tissue adhesive (Histoacryl). **D** Left external carotid arteriogram performed 6 months post-embolization. Normal appearances. AVM is not opacified.

and the shoulder. Three of these patients presented with pain and two had undergone previous unsuccessful treatment, either embolization or surgery. Arterial embolization had been performed on five occasions over the previous 7 years in the patient with a shoulder AVM. During the first embolization procedure a combination of polyvinyl alcohol, absorbable gelatin sponge, and steel coils had been utilized. At subsequent treatment sessions polyvinyl alcohol alone was used. Embolization had resulted in relatively proximal occlusion of the major feeding arteries but arteriovenous shunting persisted through numerous small collateral vessels.

Selective arteriography was performed in all four patients (Figs. 1–4). The malformations all exhibited multiple feeding vessels, rapid arteriovenous shunting, and dilated veins. All four AVMs could be classified as being of an arteriolovenous type (see Discussion) with more than three arteries feeding directly into a central dilated venous component, and in view of this angio-architecture a venous approach was attempted. The procedure was performed under general anesthesia in two patients.

In the hand and forearm AVMs, the central venous component was catheterized via a hypertrophied draining vein close to the malformation and embolization was performed with absolute alcohol (2.5 ml in the hand AVM, 8 ml in the forearm AVM) in divided small doses (Figs. 1, 2). Each aliquot of absolute alcohol (between 0.5 and 2 ml) was injected with an arm tourniquet inflated to above systemic arterial pressure and was preceded by a test dose of a similar volume of contrast medium. The tourniquet was kept inflated for 1–3 min after each aliquot.

In the nasal/forehead AVM, a large draining vein was punctured on the bridge of the nose (Fig. 3). The left angular vein was then compressed digitally by the patient to prevent the flow of any of the embolic agent into the left ophthalmic vein, and the efficacy of this compression was checked by preliminary contrast medium studies. Two separate injections of 0.5 ml N-butyl-2-cyanoacrylate (Histoacryl, Davis and Geck, Hampshire, England) mixed with 0.5 ml Lipiodol Ultrafluid (to delay polymerization) were injected to achieve complete occlusion of the lesion.

The large central venous component of the shoulder AVM was catheterized via a transfemoral venous approach using a conventional 7 Fr Headhunter I catheter and a 3 Fr coaxial catheter (Target Therapeutics, Freemont, CA, USA). Balloon occlusion of the proximal subclavian artery was performed during embolization of the central venous component, with an 11.5 mm diameter occlusion balloon (Boston Scientific, Watertown, MA, USA) inserted via a transfemoral arterial approach. Embolization was performed with a total of 1.5 ml of Histoacryl (three separate ''shots'' of 0.5 ml Histoacryl mixed with 0.5 ml of Lipiodol Ultrafluid).

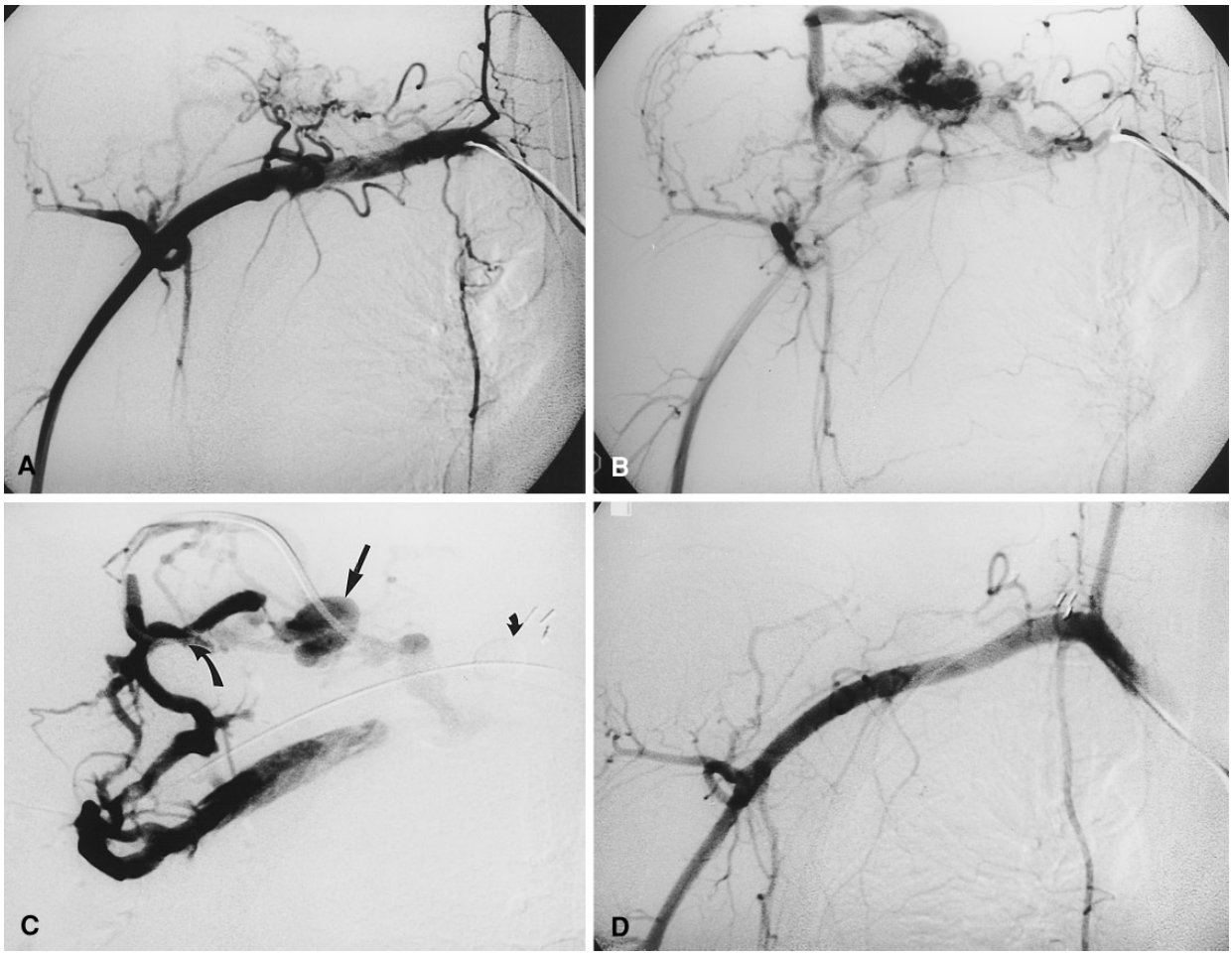

Fig. 4. AVM involving the right shoulder. **A** Right subclavian arteriogram demonstrates numerous small vessels supplying a high-flow vascular malformation around the right shoulder. **B** Large venous component of the malformation is seen on image 2 sec later. **C** Central venous portion of AVM has been catheterized retrogradely. Venogram performed during balloon occlusion of proximal subclavian artery (small curved arrow) shows the tip of the venous catheter (large curved arrow) a few centimeters from the central venous component (straight arrow) which was catheterized and embolized with Histoacryl via a coaxial 3 Fr catheter. **D** Arteriogram 6 months post-embolization. Arterial phase demonstrates normal size of branches previously hypertrophied and no residual arteriovenous shunting.

Follow-up angiography was performed in all patients at 6 months and clinical follow-up of all patients has continued for between 8 months and 3 years.

Results

The procedure was tolerated well by all patients. Pain and soft tissue swelling developed in three individuals immediately following the embolization and was most marked in the patient with the AVM involving the radial head, whose forearm remained tense, swollen and painful for 10 days. Pain without soft tissue swelling was present in the patient with the shoulder AVM.

After resolution of the soft tissue swelling the patients with the hand, forearm, and nasal AVMs described complete disappearance of the symptoms of which they had originally complained and they have remained asymptomatic on follow-up at 3 years, 2 years, and 18 months, respectively. Patient 1 has been left with an approximately 5-mm-diameter hard lump on the dorsum of the hand which is non-pulsatile. Patient 2 has resumed playing sports with no restriction of her activities; the cutaneous birthmark is still present but her forearm veins now appear normal and there is radiographic evidence of re-ossification of the radial head. Patient 3 has a normal contour to his nose and the pulsatile mass which was obvious previously is no longer palpable. The patient with the shoulder AVM continues to complain of some pain in her upper arm 8 months following embolization, but the abnormal pulsation present before embolization has disappeared.

On follow-up angiography performed at 6 months there was no evidence of residual arteriovenous shunt-

ing in any of the patients (Figs. 1–4). More importantly all normal arterial branches have been preserved and venous drainage appears normal. There is no clinical or angiographic evidence of venous outflow obstruction.

Discussion

Arteriovenous malformations form a heterogeneous group for which numerous classifications have been proposed. Arguably the best of these is that suggested by Mulliken [5]. He divides vascular birthmarks into congenital malformations, which are described according to the type of vessel which is predominantly involved (i.e. arterial, venous, lymphatic, mixed), and hemangiomas. The latter are acquired tumors of infancy which pass through a period of rapid proliferation prior to undergoing complete or partial involution. These lesions are histologically distinct from vascular malformations. The term hemangioma should be reserved for this tumor of infancy and not be confused with congenital vascular malformations.

Houdart et al. [6] have more recently proposed a classification of intracranial AVMs which is based entirely upon the angiographic appearances of these lesions and which can, in our opinion, be usefully applied to peripheral AVMs. It is important to point out that this classification applies only to high-flow lesions and does not contradict that proposed by Mulliken. Rather, it is complementary to Mulliken's classification in that it can be used to plan the best route for embolization.

Houdart et al. [6] reviewed 99 intracranial AVMs in 98 patients and distinguished three types of fistula within them. These they termed:

1. arteriovenous, in which there are no more than three separate arteries supplying a single initial venous component;
2. arteriolovenous, in which multiple arteries shunt into a single central dilated venous component;
3. arteriolovenulous, in which there are multiple shunts between arterioles and venules. In this type the first identifiable normal venous component is separate from the shunts.

Of these types of fistula, the authors suggested that types 1 and 2 can be treated by an arterial or a venous approach while type 3 can be treated only from the arterial side. The choice of approach in types 1 and 2 will depend upon several factors including the number of arterial feeders, their accessibility to catheterization and the vulnerability of adjacent normal vessels to inadvertent occlusion if an arterial embolization is attempted.

Whichever route is employed, embolization must be performed as near as possible to, or across, the arteriovenous communications themselves. Unduly proximal embolization of feeding arteries is to be deplored since, as is the case with surgical ligation, recurrence is invariable and subsequent access to the lesion for more definitive treatment will be severely compromised.

When a venous approach is attempted for the treatment of a peripheral high-flow lesion, the most commonly used embolic agent is the metallic coil [2–4]. The reason for this is that liquid or particulate embolic materials may be swept away from the venous side before they have managed to achieve occlusion, while an appropriately sized coil will not. The disadvantages of coil embolization on the venous side are: (1) they will expand completely to fill the venous cavity and may in fact stretch it with a potential risk of rupture; (2) as a result of point 1 above, many coils may be required to achieve complete occlusion; (3) complete occlusion of the venous side may be difficult to achieve with coils alone, particularly when the lesion is large, and if there is persistent flow at the end of the procedure then recurrence is almost certain; (4) in peripheral AVMs the venous component may be very superficial and coils would be easily palpable; and (5) coils in a very superficial location may erode through the overlying skin causing ulceration; these coils may eventually be extruded. For all these reasons, liquid embolic agents are preferred by the present authors if venous drainage and/or arterial inflow can be controlled. It is important to point out, however, that Histoacryl will produce a hard mass which itself has the potential to erode through the skin if in a superficial location, although this did not occur in either of the two patients in whom it was used in this series.

In our series all the patients can be described as having arteriolovenous type malformations. The hand AVM has a single dominant feeding artery but multiple smaller feeding vessels are also visible (Fig. 1A). A large central venous structure is present in all patients from which one or more smaller veins emanate. In the hand and forearm AVMs, both arterial inflow and venous outflow could be controlled with tourniquets. In the nasal AVM, venous drainage was occluded by digital compression prior to embolization with tissue adhesive. In the shoulder AVM, arterial inflow was controlled with an occlusion balloon.

The major potential complication of venous embolization when liquid agents are used is that of inadvertent occlusion of the normal venous drainage from surrounding tissues with resultant venous hypertension and deep venous thrombosis. This is best avoided by performing test injections of contrast material to determine the amount of embolic agent that can be administered without causing spillover into normal veins and by injecting small intermittent aliquots of the embolic agent rather than one large bolus. Absolute alcohol may

be mixed with nonionic contrast material without precipitation; the present authors prefer, however, to use it undiluted so as to preserve its maximal sclerosant activity, thus making it even more important that great care is taken not to instill too much during any one injection.

Absolute alcohol may cause a peripheral sensory and/or motor neuropathy if it leaks out into the adjacent soft tissues during embolization. This is a particular hazard if a direct puncture technique is being used as leakage may occur along the needle track. There is less chance of this occurring, however, when a retrograde venous approach is being used, as the puncture site is often at some distance from the point of alcohol instillation and this is perhaps a further advantage of this route being used for embolization.

A further potential complication of this technique when both arterial inflow and venous outflow are being occluded is that of retrograde filling of the arterial feeding vessels and reflux of embolic material into the arterial side of the circulation. This has not been shown to occur in the authors' experience even when a tourniquet has been inflated to above arterial pressure, as was performed during embolization of the hand and forearm AVMs. The possibility of this occurrence, however, underscores the importance of performing test injections of contrast medium prior to the instillation of any liquid embolic agents.

While the results of this technique may be dramatic, it is unfortunate that only a small minority of peripheral AVMs are suitable for this form of therapy. Most peripheral AVMs, in the authors' experience, are of the arteriolovenulous type in which the first identifiable venous structure is at some distance from the arteriovenous shunts. A retrograde venous approach is, therefore, contraindicated as this will only result in occlusion of normal venous outflow without treating the small abnormal arteriovenous communications within the center of the malformation. A worsening of peripheral venous hypertension is likely to result, with a subsequent increased risk of ulceration and hemorrhage. During the 3-year period in which these four patients have been treated, approximately 100 other patients have been embolized using an arterial approach. In practically all these patients a retrograde venous approach was not considered suitable because of the unfavorable angio-architecture of the lesions.

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