Cardiovasc Intervent Radiol (1996) 19:65–71

CardioVascular and Interventional Radiology

How I Do It

Arteriovenous Malformation Management

Wayne F. Yakes,[1] **Plinio Rossi,**[2] **Henk Odink**[3]

[1]Interventional Radiology and Interventional Neuroradiology, Radiology Imaging Associates, P.C., Colorado Neurological Institute, Swedish Medical Center, 501 E. Hampden Avenue, Englewood, CO 80110, USA
[2]Department of Radiology, Universita Degli Studi Di Roma, ''La Sapienza'', 00161 Rome, Italy
[3]Department of Radiology, De Wever Hospital, Henri Dunanstrat 5, 6419 PC Heerlen, The Netherlands

Abstract
Arteriovenous malformations (AVM) are rare vascular lesions that can present with a myriad of clinical presentations. In our institutions, initial workup consists of a clinical exam, color Doppler imaging, and magnetic resonance imaging. After the initial noninvasive workup, arteriography, at times closed system venography, and ethanol endovascular repair of the AVM is performed under general anesthesia. Depending on the size of the lesion, additional Swan-Ganz line and arterial line monitoring are performed. Patients are usually observed overnight and uneventfully discharged the following day if no complication occurs. Patients are followed at periodic intervals despite cure of their lesion. Long-term follow-up is essential in AVM management.

Key words: Arteriovenous malformation—Embolization—Arterial therapeutic blockade—Ethyl alcohol—Endovascular management

Arteriovenous malformations (AVMs) constitute some of the most difficult diagnostic and therapeutic dilemmas in the practice of medicine. The clinical picture can range from an asymptomatic birthmark to life-threatening congestive heart failure. Attributing any of these varied symptoms to a vascular malformation can be challenging to the most experienced clinician. Compounding the problem is the relative rarity of these lesions. If a physician encounters one patient with this condition every few years, it is difficult to develop sufficient experience for diagnosis and optimal treatment. Typically, these patients seek help from a number of physicians, only to experience disappointing outcomes, complications, and recurrence or deterioration of their presenting symptoms.

We present our approach to the diagnosis and its treatment with ethanol endovascular therapy.

Correspondence to: W.F. Yakes, M.D.

Initial Evaluation

A thorough clinical exam and history can usually establish the diagnosis of pediatric hemangioma or vascular malformation. Hemangiomas are usually not present at birth and have a bright scarlet color that gradually deepens. Vascular malformations have a persistent color depending on the dominant arterial, capillary, venous, or lymphatic component. Evaluating for skeletal abnormalities, abnormal veins, arterial abnormalities, pulsatility or nonpulsatility of a lesion, dependent swelling or flattening upon elevation, and disparity of limb size, along with neurologic evaluation and a good history can frequently enable diagnosis and even categorization of a vascular malformation. The Nicoladoni-Branham test of in-flow arterial occlusion, if positive, will result in a reflex bradycardia if the AVM is of such a high flow that it is causing cardiac consequences.

Color Doppler imaging (CDI) is an essential tool in the diagnostic workup of AVMs. Accurate measurements of flow volumes (a calculated physiologic parameter) and resistive indexes can be helpful in the initial evaluation and also are important noninvasive parameters for follow-up after therapy. Documentation of decreased arterial flow volumes and normalization of the resistive indexes are specific and may obviate the need for repetitive follow-up arteriography [1].

Magnetic resonance (MR) imaging has replaced computed tomography (CT) in the evaluation of vascular malformations. It has proven to be a mainstay in the initial diagnostic evaluation, as well as in assessing

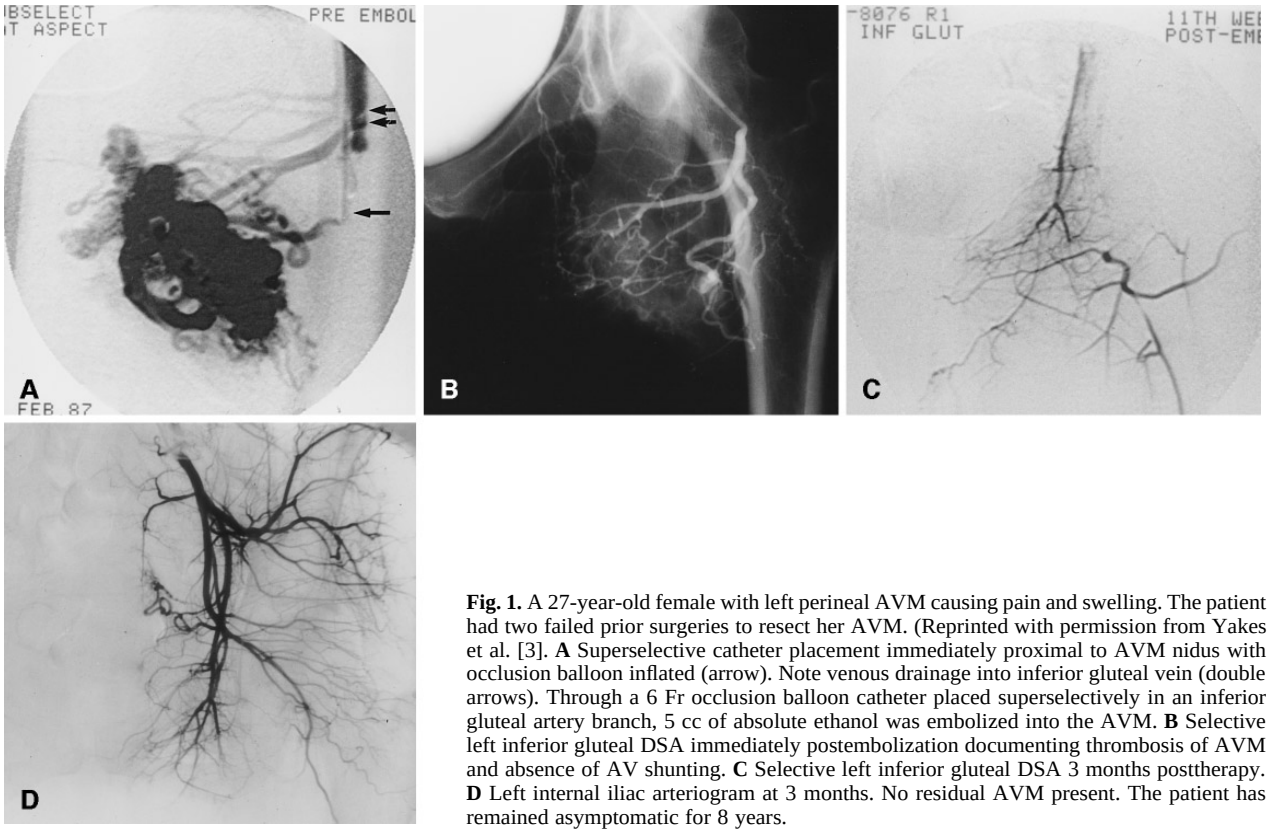

Fig. 1. A 27-year-old female with left perineal AVM causing pain and swelling. The patient had two failed prior surgeries to resect her AVM. (Reprinted with permission from Yakes et al. [3]. **A** Superselective catheter placement immediately proximal to AVM nidus with occlusion balloon inflated (arrow). Note venous drainage into inferior gluteal vein (double arrows). Through a 6 Fr occlusion balloon catheter placed superselectively in an inferior gluteal artery branch, 5 cc of absolute ethanol was embolized into the AVM. **B** Selective left inferior gluteal DSA immediately postembolization documenting thrombosis of AVM and absence of AV shunting. **C** Selective left inferior gluteal DSA 3 months posttherapy. **D** Left internal iliac arteriogram at 3 months. No residual AVM present. The patient has remained asymptomatic for 8 years.

the efficacy of endovascular therapy. MR can accurately distinguish between high-flow and low-flow malformations also, and the relationship to adjacent anatomic structures, such as muscles, nerves, and organs, is easily determined. High-flow malformations typically demonstrate signal void on most sequences. On gradient-echo sequences, increased signal within the vascular structures is present. At follow-up, MR can accurately determine residual areas of AVM as well as those areas that have been treated [2].

After the diagnosis has been established, the next major decision is to determine whether therapy is warranted. The interventional radiologist should plan and direct the patient's care with surgical specialists who are familiar with AVM management and the problems it presents. It is extremely important that appropriate surgical, medical, pediatric, and anesthesiology specialists be involved for optimal patient care.

Pain Control

With the use of intravascular ethanol, pain control is a significant problem. Anesthesiologists can greatly aid in solving this problem and determine whether general anesthesia or intravenous (i.v.) sedation is required for the procedure. This leaves the interventional radiologist free to concentrate on the case at hand. For children, general anesthesia is required.

In patients with large AVMs, as opposed to small lesions, Swan-Ganz and arterial line monitoring are performed. Pulmonary artery pressures are consistently monitored during the injection of absolute ethanol. Decadron (dexamethasone sodium phosphate, USP, Merck & Co., West Point, PA, USA) is given i.v. to all patients prior to the procedure, usually 10 mg for adults and 3–10 mg for children, depending on body weight.

Ethanol Endovascular Therapy

The area of vascular access, whether it be the groin, the arm, or other points of percutaneous catheter access, is prepped and draped sterile, as is the area of the AVM that is to be treated percutaneously. Fluoroscopy and/or CDI techniques are used in those patients requiring percutaneous access. Detailed arteriography is performed to determine the angioarchitecture of the AVM; the major compartments and endovascular access to those compartments are delineated. If the patients have had prior therapy [surgical ligations, partial resections, intraarterial coil placement, tissue adhesives (NBCA/IBCA) embolization], direct puncture techniques may be required. Superselective placement of

the catheter tip or the needle tip is a requirement; only then can ethanol be injected into the malformation and all normal vascular structures spared (Figs. 1–3).

To achieve superselectivity, coaxial and triaxial systems may be required. In some instances, a long 6 or 7 Fr sheath may be placed into a selective artery. Through this sheath which provides support, a 5 or 6 Fr catheter would then be advanced. We use the Glidecatheter (Meditech Inc., Watertown, MA, USA). Through the 5 or 6 Fr distally placed catheter, a microcatheter is triaxially placed into an even more distal position. Multiple arterial punctures may be required to place catheters for a distal embolization, along with an additional puncture for proximal occlusion-balloon placement to achieve vascular stasis.

At times, even these complex maneuvers may fail, and direct puncture of the malformation or an in-flow vascular pedicle may be required. The area of percutaneous puncture is prepped and draped; a needle, usually 18–20 gauge, is advanced under real-time ultrasound guidance or by contrast injections and fluoroscopic guidance. Once correct placement has been achieved with the direct puncture needle, contrast injections, as well as ethanol injections, can be performed through the needles. Proximal inflow occlusion may be necessary. Occlusion balloon catheters and, for extremities, tourniquets may be required to achieve vascular stasis. At the end of the embolization procedure, if hemorrhage occurs after removal of the needle, hemostasis can be achieved with a simple injection of Avitene (Alcon Laboratories, Ft. Worth, TX, USA), a topical hemostatic agent, which can be injected (mixed with contrast) as the needle is retracted.

For extremities, external pneumatic blood pressure cuffs, as well as hand-tied tourniquets, may be useful and necessary for vascular stasis within an AVM. In the chest, abdomen, pelvis, and head and neck area, intravascular occlusion balloons may be necessary to achieve some element of flow arrest. Arteriograms must be performed in both the nonocclusive and occlusive state to determine exactly the flow characteristics of the AVM so that an appropriate volume and rate of ethanol injection may be determined. The amount of ethanol used is equal to the flow-volume characteristics of the malformation compartment being treated.

After ethanol injection, occlusion is usually maintained for 10–15 min. Then the vascular occlusion is released and arteriograms are performed to determine if therapy is complete or further embolization is required. Frequently, additional compartments of AVM will then fill as others become thrombosed. Meticulous repetition of the previously described technique is then required.

The maximum volume of ethanol used in treating patients with AVMs rarely exceeds 0.5–1.0 ml/kg body weight total dose. Most patients will tolerate these total ethanol volumes very well. Exceeding these doses can lead to ethanol toxicity. Cardiopulmonary collapse is a rare but dreaded sequela, and pulmonary artery Swan-Ganz line and arterial line monitoring are essential to minimize the possibility of this event occurring. Once pulmonary artery pressures begin to rise, it is best to wait and not inject any more ethanol until the pulmonary pressures begin to normalize. If pulmonary artery pressures become pathologically high, the infusion of nitroglycerin, adenosine, or prostaglandin E_1 through the Swan-Ganz line can lower the intrapulmonary pressures; we favor nitroglycerin. We have determined that increased pressures related to ethanol injection reaching the pulmonary artery capillary bed may cause, in some patients, precapillary spasm which is a transient phenomenon. However, the infusion of nitroglycerin through the Swan-Ganz line is helpful in reducing the elevated pulmonary artery pressures.

After the procedure and recovery from anesthesia, patients are sent to the general hospital ward; it is unusual for them to require intensive care. Postoperative management consists of i.v. Decadron, i.v. fluids, and i.v. Inapsine (Droperidol injection, Janssen Pharmaceuticals, Inc., Titusville, NJ, USA) as needed to control nausea. Oral or i.m. Toradol (ketorolac tromethamine, Syntex Laboratories, Palo Alto, CA, USA) by body weight is helpful for controlling pain and swelling in adult patients. Pain is unusual, however, oral and i.v. pain medications may be given additionally, if required. Patients with GI sensitivity to steriods can also be placed on Zantac (ranitidine hydrochloride, Glaxo Inc., Research Triangle Park, NC, USA) to protect against gastric or duodenal ulcer development. Patients are usually observed overnight. The following morning, patients with AVMs of the extremities undergo CDI to evaluate the presence or absence of deep vein thrombosis of the normal deep veins. This rare complication needs to be evaluated in order to institute appropriate treatment immediately. Discharge medications usually include a tapering dose of steroids over 7 days, Zantac management to prevent ulcer development, and pain medications, if required. All patients are usually seen 7–10 days postdischarge or sooner if any problems develop.

Patients usually exhibit focal swelling in the area of the AVM, which in most patients will resolve within 2 weeks. In patients with lower-extremity and foot AVMs, swelling may last longer due to the fact that the leg and foot are not only dependent organs but are weight-bearing structures as well. Usually after 4 weeks all swelling is resolved and the patient is ready for follow-up therapy as required.

Follow-Up

After serial therapy, MR and CDI can be used to document the efficacy of the therapy. CDI spectral analysis

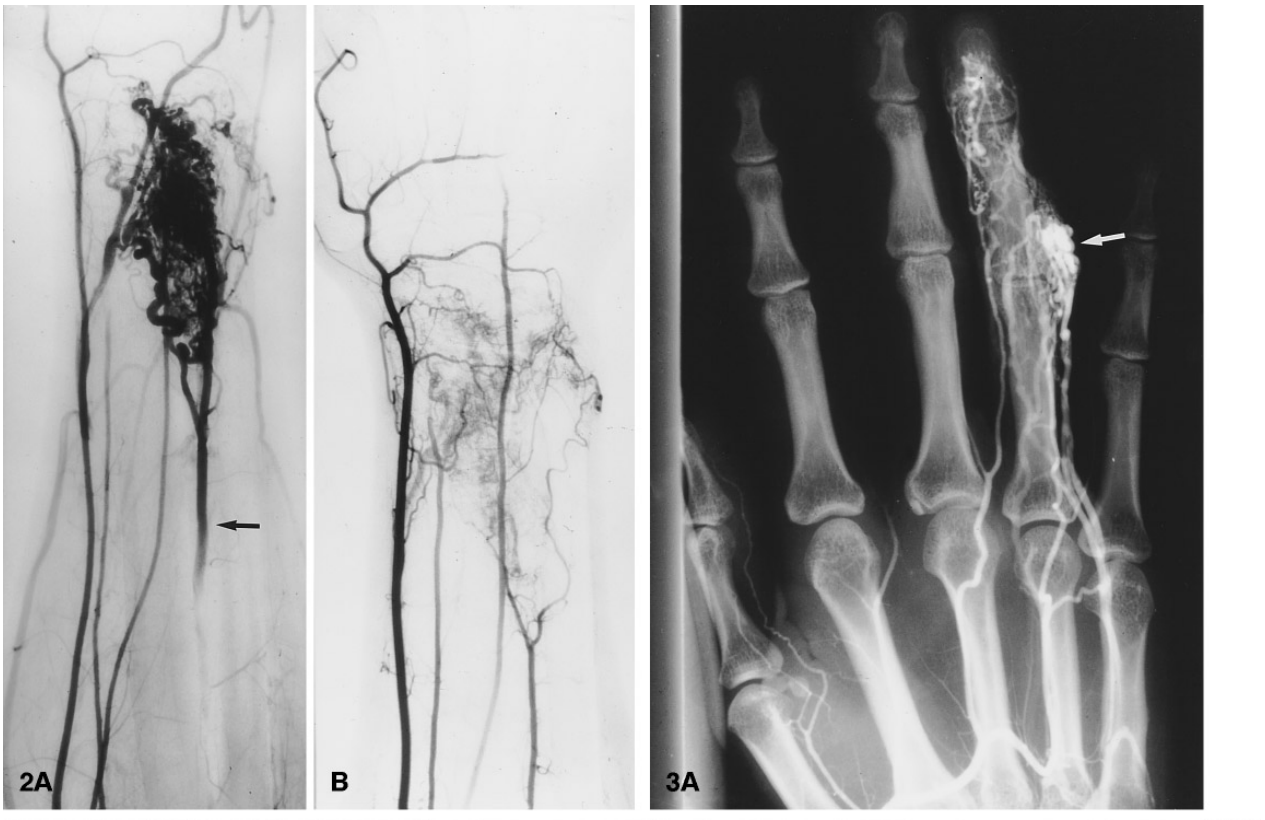

Fig. 2. A 26-year-old male with dorsal wrist AVM causing extreme pain and swelling and nonfunction of the right hand. (Reprinted with permission from Yakes et al. [22].) **A** Brachial artery DSA demonstrating AVM overlying the dorsum of the distal forearm wrist and hand. Note normal branches of the radial, ulnar, and median artery. Note retained primitive arterial branch (arrow) arising from the brachial artery supplying the AVM. **B** Follow-up brachial DSA at 4 months showing persistent occlusion of AVM. Note resultant decrease in size of embryonal arterial remnant because of lack of AV shunting. The patient has remained asymptomatic for 7 years.

Fig. 3. A 25-year-old female with painful AVM on the ulnar side aspect of the interphalangeal joint of the fourth digit. (Reprinted with permission from Yakes et al. [3].) **A** Hand arteriogram demonstrating AVM (arrow). **B** Direct puncture into AVM nidus is demonstrated. Note filling of AVM nidus with reflux into the radial side digital artery (arrow). Note venous outflow on ulnar aspect of digit (arrows). **C** AP right hand DSA preembolization. Again note AVM nidus (arrow). Note collateral flow towards AVM at the distal finger tuft (triple arrows). **D** Right hand DSA after direct puncture ethanol embolization. Note normal filling of all digital branches, thrombosis of AVM, absence of venous drainage, and continuation of the now-normal ulnar side digital branch no longer supplying AVM (arrow).

gives accurate information in treated and untreated high-flow AVMs. AVMs demonstrate high velocity and low resistance waveforms. As the malformation serially becomes ablated, the waveform will normalize and the resistive indexes and the flow volumes that can be calculated will become normalized as well. MR is also an important investigative follow-up modality [1, 2].

One of the most important aspects of AVM management is determining whether a patient's symptoms have been alleviated. In those patients who have no symptoms other than an increased cardiac output, Swan-Ganz line placement and calculation of cardiac output, cardiac index, and systemic vascular resistance will be important parameters to follow. Current noninvasive technologies used by cardiologists to evaluate these parameters are not as accurate as placement of a Swan-Ganz line and direct measurement. Our patients are evaluated with noninvasive and arteriographic studies annually. After several years of a persistent AVM closure, noninvasive imaging modalities will usually be sufficient for longer-term follow-up.

Complications

Various complications can occur with any interventional procedure. In patients with AVMs, complication rates greater than 10% must be expected. In our initial series [3], we reported a total complication rate of 30% (10% were major, 20% were minor). With more experience, our complication rate has dropped to around 10%. Complication rates are related to the tissues that are being embolized. Nontarget embolization with ethanol will lead to tissue necrosis, as capillary beds of normal arteries will be totally destroyed. Therefore, it is essential that superselective catheter positioning be achieved before ethanol can be used. Direct-puncture techniques may need to be performed in the event that the catheter cannot reach the desired position to treat only the malformation and not embolize normal tissues.

Vascular spasm, edematous tissues, and venous thrombosis can lead to complications as well. Localized skin blisters may occur, usually a minor annoyance that heals uneventfully. Injury to adjacent muscles, organs, or other tissues is possible. In the pelvis, the colon is the most sensitive organ and great care must be taken against nontarget embolization to avoid localized infarction. In only one patient in our series did this occur; the patient had to have a diverting colostomy to put the injured segment at rest. We believe this injury may have been related to vascular spasm as the AVM was intimately involved with the superior hemorrhoidal artery.

Motor or sensory nerve injuries may occur as well. We have found that most injuries have been related to the swelling involved with resultant nerve compression, rather than nontarget embolization of the vasa nervorum. Aggressive Decadron therapy is essential to minimize the effects of this swelling and to allow the nerve to recover more quickly. It is unusual for nerve injuries to become permanent.

Involvement of the appropriate clinical specialist in the management of complications is essential to minimize the morbidity of that complication. Those patients who present with tissue necrosis, whether related to arterial steal phenomenon or venous hypertension, must be counseled that treatment of the AVM may not reverse their necrotic process. In those patients who develop ischemic complications, particularly of the digits, this process can be halted, but may come too late to save the digit. What is important, therefore, is to treat the malformation to prevent further tissue loss.

Bleeding is an uncommon complication of peripheral AVMs, unlike those of the brain and spinal cord, whose propensity to bleed is their main presenting symptom. In the periphery, AVMs will cause bleeding only if they involve the alimentary canal or if they cause superficial tissue ulceration. In this situation, the malformation requires primary treatment. Only then will the tissues become normal, heal, and discontinue the hemorrhagic process. Attempts at skin grafting without treating the underlying malformation are usually doomed to fail.

Comments

AVMs are congenital vascular lesions typified by hypertrophied inflow arteries and shunting through a primitive vascular nidus into tortuous dilated outflow veins. No intervening capillary bed is present. Symptoms are usually referable to the location of the AVM. The larger and more centrally located AVMs have a greater likelihood for cardiac overload. Other presenting symptoms include pain, progressive nerve deterioration or palsy, disfiguring mass, tissue ulceration, hemorrhage, impairment of limb function, and limiting claudication.

Vascular anomalies were first treated by surgeons. The early rationale of proximal arterial ligation proved totally futile as neovascular recruitment reconstituted arterial inflow to the AVM nidus. Microfistulous connections became macrofistulous feeders. Complete extirpation of an AVM nidus proved difficult and extremely hazardous, ending in many suboptimal partial resections. Partial resection could produce initial clinical improvement, but over time the patient's symptoms recurred or worsened [3–8]. Because of the significant blood loss that frequently accompanies surgery, the skills of interventional radiologists were eventually employed to embolize these lesions preoperatively, hoping for a more complete resection. However, AVM surgery still proved extremely difficult and total removal was rarely possible. As catheter delivery systems and embolic agents improved, embolization has since emerged as a primary mode of therapy in the management of AVMs. In many cases, vascular malformations are in anatomically and surgically difficult or inaccessible areas; this has led to increased reliance on interventional radiology to manage these lesions.

According to D. Emerick Szilagyi [4], former editor of the *Journal of Vascular Surgery* (USA), ''. . . with few exceptions, their (AVMs) cure by surgical means is impossible. We intuitively thought that the only answer of a surgeon to the problem of disfiguring, often noisome, and occasionally disabling blemishes and masses, prone to cause bleeding, pain, or other unpleasantness, was to attack them with vigor and with the determination of eradicating them. The results of this attempt at radical treatment were disappointing.'' Indeed, of 82 patients seen in this patient series, only 18 were deemed operable; of these 18 who were operated on, 10 improved, 2 remained unchanged, and 6 were worse at follow-up.

Many endovascular occlusive agents (embolic agents) are currently in use to treat AVMs. Agents include autologous clot, Gelfoam, polyvinyl alcohol particles (PVA), various metallic coils with or without fibers, tissue adhesives (IBCA/NBCA), detachable balloons, Ethibloc, Sotradecol, and ethyl alcohol [9–18]. It is well known that Gelfoam, PVA, coils, or detachable balloons rarely cure peripheral AVMs. Ethibloc has primarily been used for venous malformation management and is uncommon for high-flow AVMs. Tissue adhesives (IBCA/NBCA) were initially thought to be permanent occluding agents, however their use is difficult and it is now well documented that recanalizations do occur [12, 18]. As polymerization occurs, the cyanoacrylates generate heat which may contribute to some level of histotoxicity in the adjacent area and angionecrosis. Once solidified intravascularly, the cyanoacrylates incite a mild inflammatory response. In the head and neck area, undesirable cosmetic results may occur from black tantalum powder used to opacify cyanoacrylates. Furthermore, hard acrylate masses in muscular structures can cause muscular dysfunction and tissue erosions. Miscalculations with the polymerization time can lead to disaster, with too distal or too proximal a polymerization and solidification.

Liquid sclerosing agents include Sotradecol and ethanol. Sotradecol, available in a 1% or 3% aqueous solution, has a soapy texture and contains 2% benzyl alcohol. Toxicities and complications with larger injections have not been documented. Ethanol is a sclerosing agent whose metabolism and excretion in humans is well known. We have treated many Sotradecol failures and were successful in the treatment of AVMs with the use of ethanol. In our opinion, ethanol is the more effective and superior liquid sclerosing agent [3, 19–26].

Ethanol induces thrombosis by denaturing blood proteins, dehydrating vascular endothelial cells and precipitating their protoplasm, denuding the vascular wall totally of endothelial cells, and segmentally fracturing the vascular wall to the level of the internal elastic lamina. Any one of these events, and especially the combination of all these factors, causes an acute thrombosis. Again, extreme caution and superselective placement are required when using ethanol as an endovascular occlusive agent. In the treatment of AVMs, ethanol has demonstrated its curative potential, as opposed to palliation which is commonly seen with all other embolic agents. One of the factors that may lead to the permanence demonstrated by ethanol on long-term follow-up is the fact that the endothelial cell of the vascular wall is totally obliterated. It has not been unequivocally proven but we have much indirect evidence that endothelial cells mediate vascular recanalization by activating a cellular response to remove thrombus and embolic debris. Endothelial cells then line the new channels in the recanalization process. With regard to angiogenesis factors and neovascular recruitment/stimulation, it is also felt that endothelial cells mediate this response by the release of angiogenesis factors. Again, these concepts are theoretical at this point.

Since ethanol completely destroys the endothelial cell, the phenomenon of recanalization and neovascular recruitment are noticeably absent. The permanence encountered with ethanol is unusual with other agents.

Because cure is possible by endovascular procedures, the role of surgery for AVMs has diminished. If cure is not possible by embolization, then either repeated transcatheter procedures and/or surgery may still have a role. With current fluoroscopic systems, vessels smaller than 1 mm can be imaged, allowing planning to spare normal structures and embolize the AVM nidus superselectively.

Vascular malformations are best treated where these patients can be seen on a regular basis. The interventional radiologist who occasionally evaluates a patient every year or so will never gain enough experience to manage these challenging lesions effectively. All too frequently, the patient ultimately pays for the interventional radiologist's initial enthusiasm, inexperience, folly, and lack of necessary clinical backup. To optimally manage these patients, a dedicated team should be in place. Interventional radiology and the various surgical and medical specialties function together, much like the tumor board team of specialists. When patients are seen and treated regularly, then experience can be gained, rational decisions can be made, complications can be appropriately managed, and patient care is optimized. It cannot be emphasized enough that, as a group, vascular malformations pose one of the most difficult challenges in the practice of medicine. A cavalier approach to their management will always lead to significant complications and dismal patient outcomes.

References

1. Yakes WF, Stavros AT, Parker SH, Luethke JM, Rak KM, Dreisbach JN, Slater DD, Burke BJ, Chantelois AE (1990) Color

Doppler imaging of peripheral high-flow vascular malformations before and after ethanol embolotherapy. RSNA presentation. Radiology 177 (p):156
2. Rak KM, Yakes WF, Ray RL, Dresibach JM, Parker SH, Luethke JM, Stavros AT, Slater DD, Burke BJ (1992) MR imaging of symptomatic peripheral vascular malformations. AJR 159:107–112
3. Yakes WF, Haas DK, Parker SH, Gibson MD, Hopper KD, Mulligan JS, Pevsner PH, Johns JC Jr, Carter TE (1989) Symptomatic vascular malformations: Ethanol embolotherapy. Radiology 170:1059–1066
4. Szilagyi DE, Smith RF, Elliott JP, Hageman JH (1976) Congenital arteriovenous anomalies of the limbs. Arch Surg 111:423–429
5. Decker DG, Fish CR, Juergens JL (1968) Arteriovenous fistulas of the female pelvis: A diagnostic problem. Obstet Gynecol 31:799–805
6. Flye MW, Jordan BP, Schwartz MZ (1983) Management of congenital arteriovenous malformations. Surgery 94:740–747
7. Tanner NSB, Pickford MA (1993) Preliminary report: Intratumoral ligation as a salvage procedure for the management of life-threatening arteriovenous malformations. Br J Plast Surg 46:694–702
8. Habal MB, Murray JE (1972) The natural history of a benign locally invasive haemangioma of the orbital region. Plast Reconstr Surg 49:209–214
9. Nakno H, Igawa M (1986) Complication after embolization of internal iliac artery by gelatin sponge powder. Hiroshima J Med Sci 35:21–25
10. Swarc TA, Carrasco CH, Wallace S, Richli W (1986) Radiopaque suspension of polyvinyl alcohol foam for embolization. AJR 146:591–592
11. Brothers MF, Kaufmann JCE, Fox AJ, Deveikis JP (1988) N-butyl-2-cyanoacrylate substitute for IBCA in interventional radiology: Histopathologic and polymerization times studies. AJR 10:777–786
12. Widlus DM, Murray RR, White RI Jr, Osterman FA Jr, Schreiber ER, Satre RW, Mitchell SE, Kaufman SL, Williams GM, Weiland AJ (1988) Congenital arteriovenous malformations: Tailored embolotherapy. Radiology 169:511–516
13. Riche MC, Hadjean E, Tran-Ba-Huy P, Merland JJ (1983) The treatment of capillary-venous malformations using a new fibrosing agent. Plast Reconstr Surg 71:607–612
14. Hasimoto Y, Matsuhiro K, Nagaki M, Tanioka H (1988) Therapeutic embolization for vascular lesions of the head and neck. Int J Oral Maxillofac Surg 18:47–49
15. Persky MS, Berenstein A, Cohen NL (1984) Combined treatment of head and neck vascular masses with preoperative embolization. Layrngoscope 94:20–27
16. Strachan J, Hemingway AP, Mansfield AO, Allison DJ (1986) Embolization of an arteriovenous malformation following surgical reconstruction of a previously ligated lingual artery. J Intervent Radiol 1:79–82
17. Anavi Y, Har-El G, Mintz S (1988) The treatment of facial hemangioma by percutaneous injections of sodium tetradecyl sulfate. J Laryngol Otol 102:87–91
18. Rao VRK, Mandalan KR, Gupta AK, Kumar S, Joseph S (1989) Dissolution of isobutyl 2-cyanoacrylate on long-term follow-up. AJNR 10:135–141
19. Yakes WF, Pevsner PH, Reed MD, Donohue HJ, Ghaed M (1986) Serial embolizations of an extremity arteriovenous malformation with alcohol via direct percutaneous puncture. AJR 146:1038–1040
20. Takebayaski S, Hosaka M, Ishizuka E, Hirokawa M, Matsui K (1988) Arteriovenous malformations of the kidneys: Ablation with alcohol. AJR 150:587–590
21. Vinson AM, Rohrer DB, Willcox CW, Sigfred SV, Wheeler JR, Jacobs JS, Ruffin W Jr (1988) Absolute ethanol embolization for peripheral arteriovenous malformation: Report of two cures. South Med J 1:1052–1055
22. Yakes WF, Haas DK, Parker SH, Gibson MD, Hopper KD, Mulligan JS, Pevsner PH, Johns JR Jr, Carter TE (1989) Alcohol embolotherapy of vascular malformations. Semin Intervent Radiol 6:146–161
23. Yakes WF, Luethke JM, Parker SH, Stavros AT, Rak KM, Hopper KD, Dreisbach JN, Griffin DJ, Seibert CE, Carter TE, Guilliland JD (1990) Ethanol embolization of vascular malformations. Radiographics 10:787–796
24. Yakes WF, Luethke JM, Merland JJ, Rak KM, Slater DD, Hollis HW, Parker SH, Hodes JE, Casasco A, Hopper KD, Stavros AT, Carter TE (1990) Ethanol embolization of arteriovenous fistulas: A primary mode of therapy. J Vasc Intervent Radiol 1:89–96
25. Mourao GS, Hodes JE, Gobin YP, Casasco A, Aymard A, Merland JJ (1991) Curative treatment of scalp arteriovenous fistulas by direct puncture and embolization with absolute alcohol. J Neurosurg 75:634–637
26. Yakes WF (1994) Extremity venous malformations: Diagnosis and management. Semin Intervent Radiol 11:332–339