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Coronary Artery Revascularization: Surgical Approach – Minimally Invasive Management

Valavanur A. Subramanian, Nilesh U. Patel

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"To be seventy years young is sometimes far more cheerful and hopeful than to be forty years old."

(Oliver Wendell Holmes)

INTRODUCTION

Challenges brought on by an aging population, an increase in the number of patients with comorbidities requiring primary and reoperative coronary artery bypass surgery, an emphasis on clinical outcomes, the competitive status of coronary stenting, and cost containment in cardiac surgery have motivated considerable changes and refinements in the surgical treatment of coronary artery disease. The most recent changes aim towards less invasiveness and avoidance

of the traumatic and inflammation-producing aspects of conventional coronary artery surgery while maintaining the same safety and efficacy of proven surgical procedures.

The invasive aspects of conventional coronary artery bypass grafting (CABG) are:

- Full midline sternotomy
- Aortic cannulation and manipulation
- Use of cardiopulmonary bypass

In good risk patients, the deleterious effects of the above-mentioned invasive manipulations are minimized because of the physiological ability of these patients to cope with this surgical insult. As preoperative comorbid conditions increase, the risk of a surgical procedure significantly increases with the increasing degree of surgical invasiveness. A full midline sternotomy usually heals without any adverse consequences in the majority of patients (< 2% infection rate), but becomes increasingly problematic, particularly in patients with irradiated chest walls from cancer treatment, diabetes mellitus, renal failure, chronic obstructive pulmonary disease (COPD), immunocompromised diseases, older patients with decalcified, brittle sternums, and reoperations. Cardiopulmonary bypass (CPB) surgery, with its well-documented systemic inflammatory response, also becomes significantly more risk-laden for patients with renal, pulmonary, hepatic, hematological, and neurological dysfunction. Of all of these, neurological injury has recently been very carefully examined. The incidence may be as high as 6% for gross neurologic injury and 57% for mild cognitive changes [1]. The cause of postoperative neurological dysfunction is not fully understood, but is believed to be a result of both embolic phenomena and hypoperfusion during CPB [2]. The ideal perfusion pressure therefore represents a careful balance of risks. Reduced perfusion pressure and blood flow may decrease cerebral embolic load but increase the risk of hypoperfusion, whereas elevated perfusion pressures and flow may decrease the likelihood of hypoperfusion but increase cerebral embolic load. Cannulation, cross-clamp-

ing, and aortic manipulation are also other significant risk factors for neurological injury and those with severely calcified ascending aorta, ascending luminal aortic atheroma, prior history of neurological disease, and peripheral vascular disease are at a particularly high risk for permanent neurological injury. These reflections, along with the growing recognition and encouraging results of off-pump coronary artery surgery, have encouraged surgeons to embrace less invasive modalities from which high-risk patients such as the elderly can most benefit.

HISTORICAL HIGHLIGHTS

The first quantum leap in the field of surgical treatment of coronary artery disease was made by Vineberg [3] when he performed the first blind internal mammary artery implantation into the ischemic myocardium. In 1954 Murray and associates [4] reported experimental coronary anastomoses using saphenous vein and internal mammary artery. During the same period, Longmire [5] presented a series of beating-heart coronary endarterectomies. In 1962, Sabiston was the first one to describe saphenous vein to right coronary anastomosis. In 1966, Kolessov [6] reported coronary revascularization of the left internal mammary artery to the left anterior descending coronary artery on a beating heart, but later abandoned this technique as the use of cardiopulmonary bypass and cardioplegic arrest became the routine. Bennetti and Buffolo [7, 8] repopularized beating-heart coronary revascularization after reporting a large series between 1978 and 1988. Subramanian [9] was the first to report a multicenter minimally invasive direct coronary artery bypass grafting (MIDCAB) experience, bringing the procedure into the limelight among cardiologists and cardiac surgeons.

DEVELOPMENT OF MIDCAB

Left anterior descending (LAD) coronary artery grafting using the left internal mammary artery (LIMA) through a left anterior small thoracotomy without cardiopulmonary bypass started a new minimally invasive era in 1994 after early encouraging multicenter results of this procedure by Benetti [10], Buffolo [11], Subramanian [8], Pfister [12], and Calafiore [13]. Before April 1996, which we call the prestabilizer era, during which liberal use of β -blockers and calcium channel blockers, and intermittent transient cardiac standstill accomplished with bolus infusion of adenosine helped to facilitate coronary anastomoses. But pharmacological stabilizers did not completely eliminate vertical and transverse movements of the target site, thus predisposing to the formation of multiple intimal tears and thrombus with unpredictably low patency rates [14]. At this point many concerns were raised about the accuracy of coronary anastomoses in minimally invasive bypass surgery due to the higher early target lesion reintervention rate (TLR).

After April 1996, the innovative concept of mechanical target vessel stabilization, i.e., the cardiothoracic system stabilizer (CardioThoracic Systems, Cupertino, CA) and octopus suction pods (Medtronic, Minneapolis, MN), not only improved anastomotic patency rates, making them comparable to conventional bypass surgery, but it also made this approach widely accepted and helped to reduce the learning curve for beginners. In addition, use of a rib cage

retractor system along with a stabilizer allowed for easier harvesting of the mammary artery up to its origin.

One of the major predicaments of minimally invasive coronary surgery with target vessel revascularization was complete vs incomplete revascularization. Although the postoperative follow-up is still short, the remarkably high midterm cardiac-related event-free survival rate indicates that the most important left internal mammary to left anterior descending coronary artery anastomoses is of substantial benefit to most patients with associated comorbid conditions, even if they have multivessel disease [15]. Thus left internal mammary to left anterior descending coronary artery MIDCAB has evolved as a standardized procedure with a procedural success rate of about 98%.

On the other hand, angioplasty has been an accepted treatment modality for coronary artery disease, but the major disadvantage of angioplasty has been restenosis, requiring repeat interventions with resultant loss of initial cost savings. Compared with the right and circumflex coronary arteries, the left anterior descending artery has been more adversely affected by restenosis. This led to the new concept of hybrid optimum revascularization, in which integrated coronary revascularization has been done by combining minimally invasive bypass to the left anterior descending artery and angioplasty or stenting of the other critical lesion in patients who are at high risk for sternotomy or cardiopulmonary bypass [16]. The triumph of the left internal mammary to left anterior descending coronary artery MIDCAB then opened up avenues for other minimal access incisions and also off-pump coronary artery bypass using the sternotomy approach (OPCAB). The indications for this minimally invasive approach have further expanded to revascularization of the lateral and inferior walls through lateral thoracotomy [17] and subxiphoid [18] incisions. To further expand the indications of minimally invasive coronary artery bypass grafting from one-vessel to two-vessel disease, combinations of the incisions were used, mostly in reoperations to avoid the risk of resternotomy. Still, the major challenge with the minimally invasive technique was treating the multivessel and distal vessel disease frequently seen in patients with extensive stent restenosis (full metal jacket syndrome) and reoperative surgery.

To overcome the above technical limitations, a new minimal access incision has been developed at our institute in which all surfaces of the heart are approached through flexible body compartments, i.e., the abdomen and diaphragm. This transabdominal approach has been quite effective for distal and multivessel disease [19]. The major advantages of this approach are a single incision for multivessel grafting, bilateral mammary availability even in diabetics, as bony parts are not violated and multiple arterial conduits are available. After a variety of minimal access incisions became successful, attention was diverted to further minimizing the trauma of access and the pain due to stretching the ribs while harvesting the mammary arteries. With the increasing expertise of thoracoscopic procedures in thoracic surgery, these techniques are now more liberally used for conduit harvesting so that the access incision can be minimized further.

At about the same time, a new method called the port access technique (Heartport Inc, Redwood City, CA), using the endovascular technique for cardiopulmonary bypass, endoluminal aortic occlusion, and cardioplegic arrest has evolved as an alternative to minimally invasive coronary artery bypass grafting. The major advantage is multivessel revascularization on an arrested heart with a

bloodless operative field. Its application remains limited to patients with multiple comorbidities because of the need for longer cardiopulmonary bypass with its associated side effects and a higher incidence of neuropsychiatric complications [20].

With the aim of moving from minimally invasive to microinvasive techniques, the ultimate goal is to perform totally endoscopic CABG without cardiopulmonary bypass. But the greatest technical limitations in achieving this goal are poor visualization and limited range of motion through thoracic ports. Computer- and robot-assisted manipulators and anastomotic devices have been developed to minimize these difficulties. In computer-assisted or robotic cardiac surgery, motion sensors are manipulated by the surgeon on a computer console. Precise, measured and tremor-free hand and wrist movements are translated directly to the robotic instruments to reproduce accurate surgical movements remotely inside the chest [21, 22]. The preliminary experience with robot-assisted endoscopic CABG on the arrested heart is promising and makes the vision of a totally endoscopic ambulatory CABG with endoscopic stabilization on a beating heart a reality in the near future.

INDICATIONS FOR MIDCAB

Patient selection criteria for MIDCAB have been expanding since its inception. However, overenthusiastic use of minimally invasive surgery is sometimes inappropriate. Patient selection should be emphasized because long-term benefits of surgery depend on it. Candidates for this procedure are patients with:

1. Prior CABG with failed saphenous vein grafts [LAD, RCA (right coronary artery), OM (obtuse marginal coronary artery)]
2. Multiple-vessel disease where CPB has presumed high morbidity: cancer, renal failure, diffuse cerebrovascular and peripheral vasculopathy, aortic atherosclerosis, old age, respiratory insufficiency, chronic obstructive pulmonary disease
3. Restenosis after PTCA and stents
4. Unsuitable LAD, RCA, and OM for percutaneous transluminal coronary angioplasty (PTCA) because of severe complex stenosis or chronic total occlusion

Evolving indications for MIDCAB are:

1. MIDCAB with single-vessel grafting as a part of a combined strategy with PTCA in patients with triple-vessel coronary artery disease
2. MIDCAB with LIMA-LAD grafting as an adjunct to major noncardiac surgical procedures, e.g., abdominal aortic aneurysm repair
3. "Culprit" lesions (Is complete revascularization necessary?)
4. Ischemic cardiomyopathy with anterior wall ischemia

The ideal anatomic conditions for LAD-MIDCAB today are:

1. LAD greater than 2 mm
2. Tubular heart on the chest roentgenogram
3. Thin chest wall with wide intercostal spaces
4. Reoperative coronary bypass with deteriorated saphenous vein graft
5. Totally occluded LAD with good collaterals to distal LAD
6. Noncalcified LAD
7. Left ventricular dysfunction

Although there are no absolute contraindications for MIDCAB, relative contraindications are:

1. Intramyocardial LAD
2. Diffusely diseased calcified (< 1.5 mm) coronary artery
3. Severe pulmonary hypertension with large left ventricle

SURGICAL TECHNIQUE

Anesthesia and Intraoperative Monitoring

Standard cardiac anesthetic techniques are used for induction and maintenance of anesthesia. Premedication with clonidine, 3 mg orally prior to surgery, has greatly enhanced this operation by providing a relatively quiet heart during the coronary anastomosis. Clonidine inhibits central release of adrenergic amines and decreases the anesthetic requirements by about 30%, thus keeping the heart rate low with a decrease in blood pressure during coronary anastomosis. Recently, routine use of single-lung ventilation has helped to facilitate the direct-vision internal mammary artery harvesting with reduction in harvesting time to approximately 15–20 min. However, caution is to be exercised when using single-lung ventilation in patients with severe chronic respiratory insufficiency because of the apparent increase in pulmonary complications when the lung is collapsed for longer periods of time. Currently, we keep the lung down with single-lung ventilation only during the last 5 min of the dissection for exposure of the left internal mammary artery near the top part of the thorax beyond the second intercostal space. Anesthetic techniques are fine-tuned to allow rapid awakening and extubation of the patient, routinely in the operating room. Recently we have begun to use epidural anesthesia and narcotics to facilitate extubation and pain control. This is especially helpful in midline sternotomy CABG operations without cardiopulmonary bypass. In patients with extreme chronic obstructive pulmonary disease when single-lung ventilation is not tolerated in the operating room, we change our choice of incision to a midline sternotomy or partial sternotomy for the completion of the procedure. During the period of local coronary occlusion, the depth of anesthesia is increased to decrease the contractility of the left ventricle, lower the blood pressure, lower the heart rate with the use of pharmacological agents, i.e., esmolol, verapamil, dilteizam, and propranolol. We no longer use adenosine for intermittent cardiac standstill during the coronary anastomosis. In addition, during the period of local coronary occlusion and stabilization, we deliberately volume-load these patients in addition to β -blockade to diminish the movement of the heart surrounding the area of the local immobilization. Routine hemodynamic monitoring, continuous transesophageal echocardiographic monitoring of regional left ventricular wall motion, and S-T segment mapping are done during the entire procedure. Mixed venous oxygen saturation is continuously monitored in all of these patients.

INCISION FOR MIDCAB GRAFTING

Anterior MIDCAB

Grafting of the mid-left anterior descending and second diagonal branches are done through an 8-cm submammary incision placed underneath the nipple over the fourth left

MIDCAB Accesses

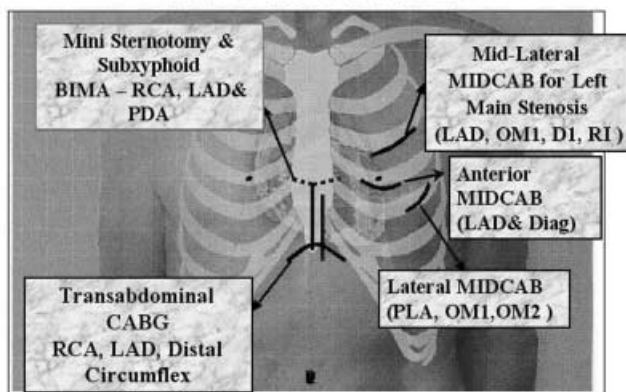


Fig. 1. Various MIDCAB access incisions and the target vessels

intercostal space with two-thirds of the incision medial and one-third of the incision lateral to the nipple (Fig. 1). Pleura is routinely entered and the lung is pushed away by gauze. This incision, in a semianterolateral position using the new retractor systems to offset the fourth and fifth ribs, creates a wide visual tunnel for the exposure of the entire LIMA allowing its mobilization up to its origin under direct vision. Further, this is a mirror image of the technique commonly used in the standard mid-line sternotomy approach for mobilization of the LIMA and this technique is familiar and comfortable for most surgeons. Skeletonization of the LIMA pedicle is not crucial in this approach. The isolation of a long length of LIMA is an essential part of the MIDCAB operation because it:

1. Avoids kinking a short LIMA at the chest wall takedown
2. Allows one to reach the LAD in a large transverse heart or in patients with chronic obstructive pulmonary disease with large residual lung volume
3. Allows tension-free LIMA-LAD anastomosis, enhancing graft patency
4. Makes it unnecessary to extend the LIMA with other arterial conduits
5. Facilitates sequential grafting of LAD and diagonal artery
6. Alleviates the concern of coronary steal from an incompletely ligated intercostal branch of the LIMA

The rare situations where we have excised the costal cartilage are in very old patients with calcified cartilage.

After the IMA is harvested in its entire length, it is clipped underneath the fifth costal cartilage and divided since the LIMA frequently becomes smaller and more muscular beyond this point, becoming more prone to spasm. Prior to the division of the IMA, a bolus of intravenous heparin is given at a dose of 2 mg/kg body weight to keep the activated clotting time twice the baseline control. The IMA is then prepared with intraluminal injections of:

1. Verapamil, 2–3 ml (5 mg in 30 ml of heparinized saline)
2. Papaverine hydrochloride in heparinized saline

Intraluminal injections of these drugs are given with no clamp applied to the IMA pedicle, and hydrostatic dilation of mammary artery is not done.

Following the preparation of the IMA, the distal end is clipped and allowed to autodilate during the preparation of the coronary artery for anastomosis. Routine use of intraluminal verapamil used in all of the multiple arterial graftings

for CABG completely avoids IMA spasm. The arterial grafts are usually very large by the time the coronary artery anastomosis is performed. In a situation where a composite T or Y graft from the LIMA is necessary for grafting to the diagonal, marginal, or ramus intermedius branches, the construction of the composite graft (i.e., radial artery, saphenous vein, inferior epigastric artery) is done prior to the LIMA-LAD anastomosis. Just before the coronary artery anastomosis, the IMA is clamped with a vascular, soft-jaw Fogarty-type bulldog clamp. The IMA is divided at the distal end, spatulated, and suspended from the upper medial edge of the thoracotomy incision with fine 6–0 Prolene sutures in the skin. Further improvements in the type of vascular clamp other than IMA-holding mechanisms will be necessary to reduce trauma to the IMA during anastomosis on a beating heart.

After the LIMA has been prepared, the IMA retractor-spreader is replaced by another spreader (an access platform; CardioThoracic Systems) that carries a coronary artery stabilizer (stabilizer; CardioThoracic Systems) (Fig. 2). The pericardium is incised about one finger breadth lateral to the IMA pedicle parallel to the midline. Here it is important to do a limited target-site pericardiotomy to prevent cardiolumination.

The left ventricle is then inspected. The LAD coronary arteries are located and the pericardium is then suspended by traction sutures.

The sutures placed laterally for the pericardium are usually pulled upward toward the suprasternal notch or the left shoulder. This maneuver cradles the heart and rotates the heart anteriorly to bring it to the surface of the wound, making the coronary anastomosis easier. A small gauze pad can usually be placed underneath the left ventricle to further rotate the heart and bring the LAD to the surface. The combination of the pericardial traction sutures and gauze pad under the left lung keeps the lung away during the entire anastomosis.

After inspection of the LAD, a site for the anastomosis is chosen. Careful fixation of the IMA pedicle to the epicardium and opening the proximal pericardium wide after completing the anastomosis are important so that kinks and twists at the anastomotic site or at the pericardial edge are avoided. Cleaning adventitial bridges around the anastomoses and the IMA graft is crucial, to avoid narrowing and stenosis on postoperative angiography (Fig. 3).

Early in our experience, if the LAD was found to be extensively calcified or intramyocardial in location, we



Fig. 2. Stabilized target vessel (CardioThoracic Systems stabilizer)

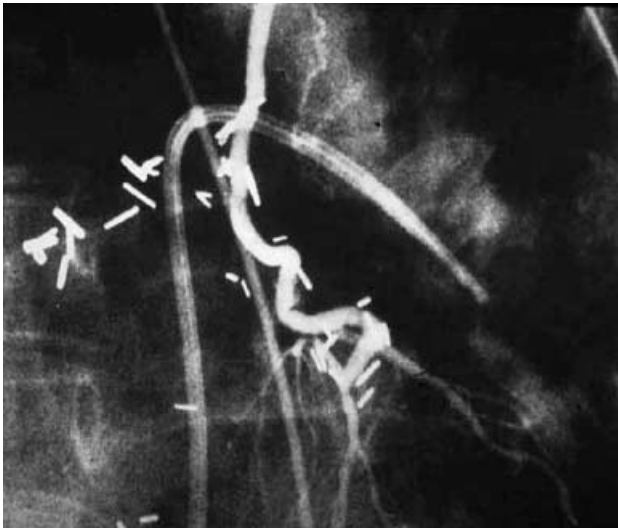


Fig. 3. Postoperative angiogram (36 h) showing left internal mammary artery sequentially anastomosed to left anterior descending and diagonal coronary arteries

abandoned the operation and converted to a standard mid-line sternotomy approach with cardiopulmonary bypass. Recently, for the calcified artery, if it is not less than 1.5 mm, we have used interrupted sutures to complete the anastomosis. With an intramyocardial LAD, we have incised the intramyocardial tunnel to expose the LAD after stabilization with the CTS stabilizer, and the LAD is then presented to the surface by using two lateral epicardial horizontal mattress sutures with Teflon bolsters placed on the sides of the intramyocardial tunnel. In patients with reoperative CABG to the LAD, very little pericardial dissection is needed. The target site of the LAD coronary artery below the old saphenous vein graft is usually immediately under the pericardial incision. In this instance, the pleural cavity frequently is not entered, thus making the operation simpler.

Anterolateral MIDCAB

The anterolateral MIDCAB approach is used to bypass the left anterior descending coronary artery, the diagonal branch, the ramus intermedius, and the first obtuse marginal branch of the circumflex system. An incision is made in the third interspace from the mid-clavicular to the anterior axillary fold. Bilateral IMA grafting to LAD and OM has been made possible by thoracoscopic mobilization of the RIMA with this approach.

Lateral MIDCAB

For grafting the second and third marginal branches and the posterolateral branch of the circumflex system, a 3-in. thoracotomy incision is made below the tip of the scapula extending two-thirds behind and one-third in front of the scapula. Optional excision of the short segment of the fifth or sixth rib may further facilitate the exposure. The chest wall muscles are marsupialized by heavy sutures on the skin edges. A medium size Finochietto retractor is used to spread the interspace. Single-lung ventilation is routinely used. The lung is collapsed. The inferior pulmonary ligament is incised and the lung is packed away superiorly with a lap pad. The



Fig. 4. Postoperative angiogram (36 h) showing radial artery anastomosed to main circumflex artery in the A-V groove. Inflow has been established from the descending thoracic aorta

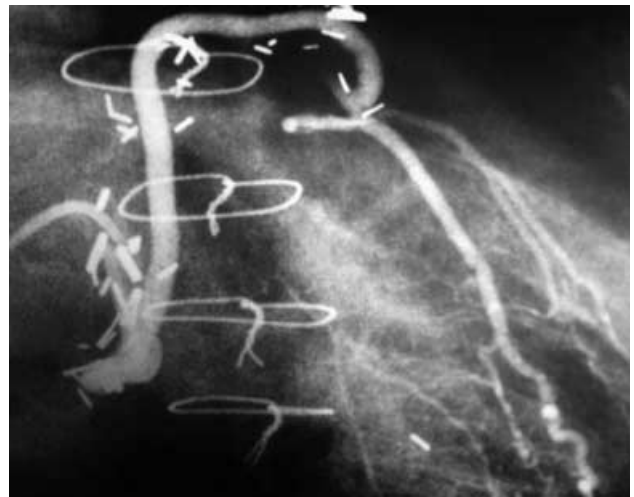


Fig. 5. Postoperative angiogram showing radial artery anastomosed from descending thoracic aorta to obtuse marginal branch in a re-operative patient

pericardium is opened posterior to the phrenic nerve and the target vessels are easily identified. With this approach, even the main circumflex artery is easily approachable by dissecting the fat pad in front of the coronary sinus (Fig. 4). The radial artery or saphenous vein is the preferred conduit for this operation and the inflow is established from the descending thoracic aorta (Fig. 5). Transesophageal evaluation of the descending thoracic aorta is mandatory in patients with a history of severe peripheral vascular disease. In selected cases, when the descending thoracic aorta is atheromatous, alternative inflow can be established from the thoracodorsal artery by dissecting it in front of the latissimus dorsi muscle and bringing it inside the chest from one intercostal space above the incision. Proper lung expansion is also important at the end of the operation to avoid rare lung torsion.

Right Bucket Handle Partial Sternotomy

The right bucket handle partial sternotomy is usually performed through the lower right partial sternotomy for the right internal mammary to mid-RCA anastomoses. Sometimes ECG changes and hemodynamic instability are the major problems, especially in the case of proximal RCA stenosis. With routine use of intracoronary shunts, these problems are less frequently encountered.

SUBXIPHOID APPROACH

Distal RCA and posterior descending coronary artery (PDA) are approached with a 2.5-in. subxiphoid incision. The xiphisternum is excised and bilateral release of costal diaphragmatic attachments helps provide better exposure of the target vessels. The right gastroepiploic artery is a frequently used conduit for this procedure.

Transabdominal Approach

After the development of the subxiphoid approach for inferior wall revascularization, two separate access incisions were used to bypass multiple vessels. The major disadvantages of this approach were the need for two separate incisions, which included violation of some of the bony parts and difficulty with stabilization of inferior wall vessel due to the narrow working angle.

Our preliminary observations, based on a few simple physiological facts, indicate that:

1. The rectus abdominis muscle has the strongest downward pull on the lower sternum.
2. When the rib cage is elevated, the lower ribs project directly forward so that the sternum also moves away from the spine, increasing the anteroposterior diameter of the chest by 20%.

This led us to develop the new transabdominal access approach. A 2–3-in. curvilinear epigastric incision just under the xiphisternum and the lower costal margin is made. Both rectus abdominis muscles with their anterior and posterior sheaths are divided. Neurovascular bundles which run on the lateral margins of the recti are preserved to reduce postoperative pain. The cut edges of the recti are marsupialized by several retractor sutures anchored to the skin. Bilateral costal

attachments of the diaphragm are released with cautery to further facilitate exposure and to increase the working angle. The lower edge of the sternum and the costal arch are lifted with table-mounted IMA retractor hooks. The sternopericardial ligament and fibrous adhesions are divided with cautery to drop down the mediastinum. Because of the bucket handle movement of the lower ribs, the lower sternum is lifted, enabling dissection of the mammary arteries under direct vision up to the 2nd or 3rd space. The thoroscopic technique has been used in some patients to facilitate mammary harvesting. The right gastroepiploic artery is harvested by opening the peritoneum and clipping its branches. Recent usage of the harmonic scalpel has allowed expeditious and bloodless dissection of right gastroepiploic artery. The left anterior descending coronary artery is approached through the inverted T pericardiotomy above the diaphragm with dislocation of the anterior surface of the left ventricle to midline by deep left lateral pericardial stay sutures with retraction of these towards the right side. The mid-right coronary artery can also be approached from above the diaphragm. Taking deep diaphragmatic stay sutures and pulling them right and downwards helps to dislocate the inferior surface of the heart and provides excellent exposure to the mid-right coronary artery. Posterior descending and posterolateral coronary arteries can be approached through a 1.5-in. incision in the central tendon of the diaphragm (Fig. 7). Dividing the left triangular ligament and retracting the left liver lobe downwards and towards the right provides adequate working space (Figs. 5, 6).

LOCAL CORONARY OCCLUSION AND PREPARATION OF THE CORONARY ARTERY FOR ANASTOMOSIS

Two types of suture techniques have been used to achieve local coronary occlusion:

1. Double-looped, large 5–0 Prolene sutures placed proximally and distally to the site of anastomosis, encircling the entire coronary artery, epicardial fat, and accompanying veins, with the coronary artery underneath the looped sutures protected by the use Silastic bolsters
2. Silastic retractor tape (Quest Medical Inc., Allen, Tx) with no double loops

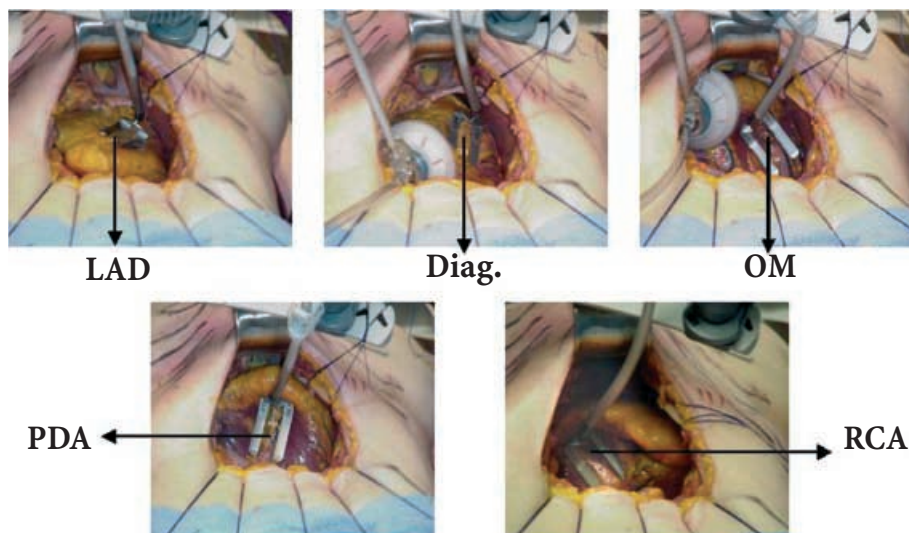


Fig. 6. Exposure of various coronary target sites through single transabdominal incision with the use of a cardiac dislocating device. LAD, left anterior descending coronary artery; Diag, diagonal graft; OM, obtuse marginal coronary artery; PDA, posterior descending coronary artery; RCA, right coronary artery

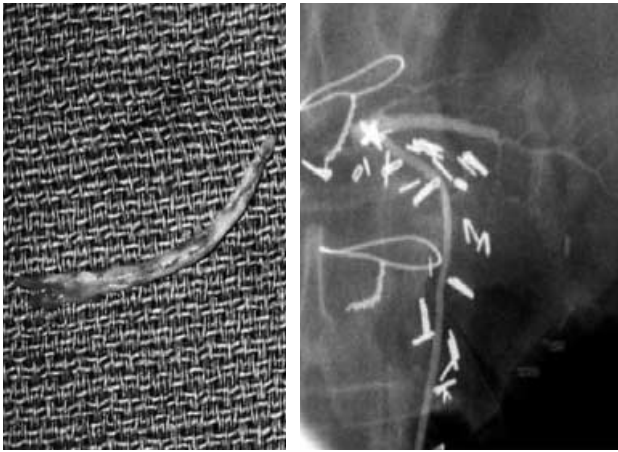


Fig. 7. End-arthrectomized segment from posterior descending coronary artery and postoperative angiogram in the same transabdominal MIDCAB patient

Ischemic preconditioning has been used in more than 90% of the patients. This is accompanied by 5 min of local coronary occlusion, followed by 5 min of reperfusion, and then completed by occlusion for coronary artery anastomosis.

Although the exact role of ischemic preconditioning in MIDCAB is not well established, our clinical experience with this technique has been favorable with local coronary occlusion. It is believed to increase high-energy phosphates in the myocardium so that subsequent ischemia is better tolerated and it also gives clues about whether the patient will tolerate local coronary occlusion. Sustained ventricular tachycardia or ventricular fibrillation was not seen in any of these patients with LIMA-LAD grafting. Unifocal premature ventricular beats were infrequently observed in these patients.

IMMOBILIZATION AND STABILIZATION OF CORONARY ARTERY

Currently there are two types of mechanical stabilization available for immobilization of the coronary artery. The first type of mechanical device achieves immobilization largely by local myocardial compression from direct pressure applied on either side of the grafted artery. A variety of instruments with varying degrees of sophistication are available, including open foot-shaped devices (CTS stabilizer) and rigid circles or rectangles that may be either hand-held or attached to an incisional retractor. To be effective, these devices need to indent the myocardium firmly so as to dissipate the propagated systolic wave across the vessel and immobilize the artery within a bridge of myocardium. The localized area of compression is usually well tolerated hemodynamically and it can often be seen on the intraoperative transesophageal echocardiogram, sometimes being mistaken for an ischemic akinetic area. Using one such mechanical immobilization platform, the CTS stabilizer (Fig. 5), in the last 172 consecutive LIMA-LAD grafts done with the MIDCAB technique, has yielded an early graft patency (studied within 36 h) of 96.2%.

The second type of mechanical device is the Utrecht Octopus (Fig. 6). This device consists of a system of suction cups on two fixed handles that are positioned on either side of the vessel. The Octopus system not only offers immobilization but also enables variable degrees of myo-

cardial traction for possible access to lateral and inferior wall vessels.

Frequent changes in heart size occur during the anastomosis secondary to variations in volume and afterloading, ischemia, and tidal volume changes that may be required for ventilation. These often lead to a shift in the alignment of the device, and methods to improve traction and maintain position are in progress. Using a variety of these immobilization techniques, with experience and practice, will optimize the technically demanding situation of coronary grafting on the beating heart.

Anastomotic Site Control

Despite the many technical improvements with the minimally invasive coronary procedure, isolation and control of the anastomotic site have remained subject to debate. Effective control requires an atraumatic system that provides isolation of an adequate segment of vessel, free of undue bleeding, with provision for dispersing residual blood that may obscure the anastomosis. We routinely use a blower device to blow the blood away during the anastomosis. In addition, the blowing action keeps the incised edges of the coronary artery separated like a spinnaker on the sail boat, greatly simplifying the anastomosis. The correct length of the isolated artery is a balance between an unduly long segment, with the increased potential for excessive and bothersome back-bleeding from septal or diagonal branches, versus an unduly short one. When feasible, the isolated segment should be chosen so as to exclude inflow from any significant-sized visible branches of the artery. With an unduly short segment of isolation, crowding or distortion of the tissues at the anastomotic toe and heel may occur and seriously obscure access to those critical areas. There also needs to be allowance for variable degrees of edema and hematoma formation, secondary to tissue injury, during or after placement of the vessel-controlling system. With the ongoing beating action of the heart, swelling may occur and extend to encroach upon the anastomosis. Of relevance also may be the position of the anastomosis along the course of the vessel. Choosing a more proximal site may well correlate with a greater area of distal vessel ischemia and increase the potential for instability during the grafting. Positioning the anastomosis more distally may offset that risk. Another important concern is suturing the back wall during coronary anastomosis due to movement of the target site. Recent routine use of intracoronary shunts has meant that these problems are less frequently encountered. Intracoronary shunts have been found to provide continuous distal coronary perfusion and bloodless anastomotic field, as well as internal vessel wall stabilization, allowing uniform and equidistant placement of sutures while protecting the posterior wall from suturing.

Anastomotic Technique

After the coronary artery has been immobilized and the ischemic preconditioning is completed, the artery is incised with a knife (Sharp point 15) for 4–5 mm. The anastomosis is performed in a standard fashion using 8-0 or 7-0 Prolene sutures. When doing composite grafts or multiple grafts to the diagonal and the circumflex, the end-to-side of the anastomosis of the circumflex is first completed, followed by the side-to-side anastomosis of the diagonal, and then the final anastomosis between the LAD and LIMA. While the final

anastomosis is completed, the bulldog clamp is placed distal to the origin of the composite graft. In a sequential LAD-diagonal graft, the side-to-side anastomosis of the diagonal is done first, and then the end-to-end anastomosis of the LIMA-LAD is completed. Again, the bulldog clamp is then placed after the diagonal anastomosis to allow perfusion during the LAD anastomosis to the diagonal and the side branches.

After completion of the coronary anastomosis, the looping sutures in the coronary artery are removed, letting the native coronary artery fill the anastomosis into the IMA, thus ensuring good flow through the anastomosis. The stabilizer is then removed, and the bulldog clamp on the IMA pedicle is released. Heparin is not neutralized routinely with protamine.

Pain is a major concern in MIDCAB. Routine intercostal block by local anesthetic with bupivacaine is used for control of incisional pain. More recently cryoablation of the intercostal nerves has provided more extended pain control over a period of 6 weeks. Routine postoperative care is carried out with emphasis on early extubation within the first hour and discontinuation of all invasive lines in the first 6 h. Whenever the coronary artery is diffusely diseased or the quality of the anastomosis is questionable, intraoperative front loading of Clopidogrel (Plavix 150 mg) followed by postoperative continuation of Plavix for at least 3 months helps to improve the graft patency.

Graft patency is tested by immediate postoperative echocardiographic Doppler color flow analysis of the LIMA-LAD anastomosis and routine angiography between 24 and 36 h after surgery. Patients are ambulated within the first 12–24 h and discharged in 36 h. Follow-up stress testing with thallium and treadmill, as well as evaluation of the IMA anastomosis with echo Doppler are performed on patients within a 3–6-month interval.

MINIMALLY INVASIVE CONDUIT HARVESTING

Endoscopic Saphenous Vein Harvesting

Endoscopic saphenous vein harvesting includes harvesting the vein through a 1-in.-wide incision made just above the knee with endoscopic and custom-developed equipment; up to 65 cm of greater quality saphenous vein can be removed. A significant decrease in postoperative leg wound infection [23], decreased patient discomfort, earlier ambulation, and increased patient satisfaction have been demonstrated when compared to the standard open method.

Endoscopic Radial Artery Harvesting

This technique has been developed at our institute. Two 3-cm incisions are made at 5 and 18 cm from the wrist crease. In this method, the radial artery is harvested using the endoscope, harmonic scalpel, and hemoclips. Preliminary clinical experience with this technique has shown the feasibility of harvesting the radial artery endoscopically with early good clinical results. Further clinical evaluation of graft patency, clinical outcome, and electron microscopic and pharmacological analysis is under study at our institution.

CONCLUSION

Minimally invasive coronary bypass surgery techniques are still under intense clinical investigation. Various studies have shown cost savings, especially with the MIDCAB technique, because of the shorter length of hospital stay (2 days), the decreased need for a heart-lung machine and the personnel to run them, elimination of the need for ventilators in postrecovery, and fewer blood tests and blood transfusions. The stroke rate with these procedures is essentially zero and the risk of heart attack is halved without the heart-lung machine. Chance of infection is cut down to about one-fourth of what it is with the cardiopulmonary bypass procedure. Although these benefits of minimally invasive coronary surgery are becoming apparent, more studies are needed to validate the claims that it is at least as safe and effective as open-heart procedures. Currently there is rapid development in enabling technology, i.e., robotic assistance and facilitated coronary anastomotic devices (staples, surgical glue). This will further progress towards truly noninvasive coronary surgery with ports. Hybrid MIDCAB in combination with PTCA and stenting has become increasingly frequent. The new paradigm for coronary artery bypass may consist of LIMA-LAD MIDCAB plus preventative cardiology by risk factor modification or stenting the circumflex or RCA combined with preventative cardiology in addition to the MIDCAB procedure.

Looking beyond the year 2002, minimally invasive coronary bypass surgery will be obviously more frequently used as established scientific validation, safety, and efficacy become available in the future.

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Coronary Artery Disease and Endothelial Function

Volker Schächinger, Andreas M. Zeiher

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PATHOPHYSIOLOGY OF ENDOTHELIAL DYSFUNCTION

The endothelium is not only a single-cell layered mechanical barrier between the blood and vessel wall, but regulates various important functions of the vasculature such as vasomotion and, therefore, blood flow regulation, hemostasis, and wall proliferation processes. Atherosclerosis is associat-

ed with an impairment of these endothelial functions, favoring myocardial ischemia and progression of the disease [1]. Thus, the endothelium plays a central role in the process of atherosclerotic disease [2, 3].

A very important function of the endothelium is to adapt coronary vasomotor tone to metabolic demand, thereby regulating coronary blood supply. To control vasomotor tone, the endothelium releases a variety of substances such as prostacyclin, hyperpolarizing factor, endothelin and, most importantly, nitric oxide (NO) [4] (Fig. 1). Physiologically, exercise is an important mechanical endothelial stimulus

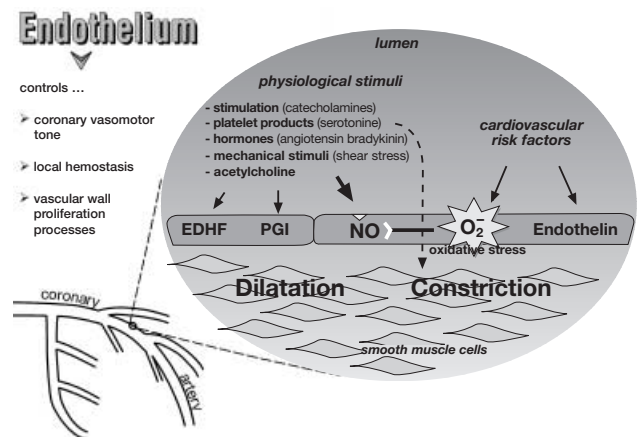


Fig. 1. Various stimuli act on the endothelium, which releases a variety of vasodilating substances such as endothelium-derived hyperpolarizing factor (EDHF), prostacyclin (PGI), and most importantly nitric oxide (NO). In addition, many of these stimuli such as catecholamines, platelet products or hormones have direct vasoconstricting effects on vascular smooth muscle cells. In case of endothelial dysfunction, when nitric oxide is inactivated by reactive oxygen species, paradoxical vasoconstriction might outweigh vasodilation to these stimuli. Pharmacological testing of this endothelial integrity can be performed by acetylcholine. (Modified from [413])

Vascular Diseases of the Central Nervous System: Percutaneous Approach

Peter J. Mitchell, Randall T. Higashida, Christopher F. Dowd, Van V. Halbach

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GENERAL REMARKS

Techniques used in percutaneous neurointervention include embolization (tumors, arteriovenous malformations, dural arteriovenous and caroticoavernous fistulas, and aneurysms), angioplasty (primary or secondary arterial stenoses causing ischemic events), thrombolysis (arterial and venous), drug delivery (chemotherapeutic agents, vasodilators in vasospasm), and vertebroplasty. Most have in common a percutaneous and generally transcatheter approach to achieve a therapeutic goal, which may be adjunctive to surgical or radiation treatment, palliation, or definitive treatment.

Significant advances in equipment that have allowed the routine selective catheterization of distal branches of the carotid and vertebrobasilar arteries include high-resolution

digital subtraction angiography with road-mapping capability and newer catheters and guidewires. The arterial procedures are performed almost exclusively from a femoral arterial puncture using a modified Seldinger technique, with anterior single wall puncture using an open needle followed by a soft atraumatic J-shaped guidewire. Difficult anatomy, particularly tortuous origins of the brachiocephalic arteries, can usually be overcome by using guidewires and catheters of different shapes and materials, but a brachial or axillary approach is occasionally used. Direct puncture of carotid and other arteries and direct puncture of head and neck hemangiomas and arteriovenous malformations can provide diagnostic information and allow intervention. Venous access is usually attained by a common femoral vein puncture and navigation of the catheter over a guidewire through the right atrium to the internal jugular vein, dural venous

sinuses, and cerebral veins. Preliminary positioning of a catheter in a large parent vessel may be all that is required to access the desired anatomical site, but more typically it is used as a guide catheter through which microcatheters are then passed in a coaxial manner.

A pressurized infusion of heparinized saline is used as a constant irrigation between (a) the guide catheter and microcatheter; (b) the microcatheter and microguidewire. This is to prevent the development of thrombus, which can serve as a source of distal embolization (and potential infarction) and also causes increased friction, decreasing the performance of the catheter system being used. Many of the more invasive procedures are performed under full heparinization, monitored by hourly activated clotting time, which allow titration of the (usual) 1000 U/h of heparin. Reversal of heparin with protamine sulfate allows immediate removal of the arterial access sheath, but is not routinely performed. If there is a high risk of thromboembolism, systemic heparin can be continued and the sheath either left in situ with a continuous heparinized flush, or, more commonly, removed and the access site closed using a percutaneous suture system. Antiplatelet agents are continued in all patients and commenced in those considered to be at high risk of platelet aggregation during or after the procedure, most notably angioplasty for atherosclerosis.

Microcatheters may be divided into flow-directed and over-the-wire catheters. The former have the benefit of being relatively atraumatic, and fast in accessing high-flow distal lesions. Many of the newer over-the-wire catheters have various proprietary forms of hydrophilic, low-friction coatings, which result in decreased forces on the vessels while retaining benefits of vessel selection from the guidewire. The advent of flow-directed catheters, compatible with microguidewires, are yet another tool facilitating superselective catheter placement. Catheters for angioplasty and for trial balloon occlusion have increased in number and quality over recent years. The Fastealth catheter (Boston Scientific, Target therapeutics, Fremont, CA) allows catheterization of intracranial arteries of small caliber with a balloon capable of inflation pressures to 6.8 atm, suitable to relatively resistant atherosclerotic or vasculitic stenoses. Coronary angioplasty systems with increased flexibility and lubricity are now available, which also allow access to the intracranial circulation. Premounted stents now exist that can negotiate the tortuous distal carotid and vertebral arteries to enable intracranial stent placement. More compliant, low-pressure balloons are available from a variety of manufacturers and are ideally suited to dilating symptomatic postsubarachnoid hemorrhage vasospasm.

TRANSLUMINAL ANGIOPLASTY AND STENTING FOR INTRACRANIAL CEREBRAL VASCULAR DISEASE

Introduction

Cerebrovascular disease is the third leading cause of major morbidity and mortality in the United States, accounting for over 150,000 annual deaths [1]; the American Heart Association estimates that more than 600,000 new strokes per year occur, at enormous social and medical cost [2–5]. The majority of these strokes are due to cardiogenic thromboembolic disease or to carotid atherosclerosis [6–8], carotid ath-

erosclerosis alone accounting for up to 30% of all strokes. The percutaneous management of extracranial carotid disease will be covered in Chap. 80.

Intracranial arterial stenoses can cause stroke from hypoperfusion or distal embolization. The estimated risk of stroke in this setting varies from 10% to 46% per year, independent of medical therapy [9–14]. Intracranial arterial disease (IAD) is an independent risk factor for subsequent stroke in medically treated patients with symptomatic extracranial internal carotid artery (ICA) stenosis [15]. Numerically less significant a cause of stroke than extracranial disease, IAD presents problems in management. No rigorous large, phase-three, randomized trials are available to assist in the choice of treatment for symptomatic IAD: antiplatelet agents (single or multiple), coumadin, or angioplasty. Extracranial to intracranial bypass surgery has been shown to have worse outcome than nonsurgical treatment, but its place is being reassessed in light of modern anesthetic and surgical techniques.

In 1964, Dotter and Judkins first reported the technique of percutaneous transluminal angioplasty (PTA) for treatment of high-grade atherosclerotic lesions of the peripheral arteries [16]. Since that time, multiple studies have demonstrated that angioplasty is of unequivocal value, with improved quality of life, decreased symptoms, and improved organ perfusion in the coronary, renal, and peripheral vascular territories; and in the coronary circulation improved outcomes have been demonstrated with stenting rather than angioplasty alone [17–21]. In the early 1980s, PTA for symptomatic vascular lesions involving the brachiocephalic arteries were initially reported [22, 23]. The study of PTA for cerebral vessels lagged behind other regions of the body due to the more difficult technical problems of access, particularly for the intracranial territories, and also because of the perceived potential complication of inducing stroke by fragmentation of material at the angioplasty site. As larger series of cases were performed, extracranial angioplasty was shown to be a safe procedure, with major complications in a similar range to those reported for alternative treatments. Intracranial angioplasty and stent-assisted angioplasty carry significantly greater risk of complications than angioplasty at other sites. This is because of the delicate vascular structure with very few media and muscular layers, difficult access, subarachnoid location with fatal outcome from vessel rupture, and the likelihood of major disability from distal embolization. Successful results of cerebral PTA involving patients with atherosclerosis, fibromuscular dysplasia, radiation-induced fibrosis, acute dissection, vasculitis, and post-surgical restenosis from intimal hyperplasia have been described [24–26].

Diagnostic Evaluation

Diagnostic evaluation of all patients is performed in conjunction with a neurovascular neurologist, neuroanesthesiologist, and the neurointerventional surgeon. Patients will generally present with either transient ischemic attacks (TIAs) or stroke from cerebral infarction. The clinical workup of completed stroke is discussed in the section on thrombolytic therapy. Clinical assessment will provide an accurate guide to the underlying anatomical site involved, associated vascular risk factors, possible etiologies, and guide subsequent investigation. General vascular risk factors, coagulation function, and evidence of an inflammatory vasculopathy are all evaluated. In most centers, CT scanning is still performed

first. MRI is more sensitive to ischemic change when diffusion-weighted imaging (DWI) is used, perfusion can be assessed, and detailed MRA of both the intracranial and extracranial arteries is possible. Assessment of cerebral blood flow with single photon emission computer tomography SPECT can provide useful information when sophisticated MRA is unavailable.

If the clinical and imaging findings indicate that large vessels rather than long perforating arterial territories are involved, duplex ultrasound or MRA is performed to assess the extracranial circulation, echocardiography is used to look for patent foramen ovale, valvular or cardiac wall abnormalities, or intraluminal thrombus, and MRA evaluates the intracranial circulation. When no extracranial large vessel, cardiac, or hematological cause for the clinical presentation is found, detailed cerebral catheter angiography is performed. The benefits of digital subtraction angiography have now replaced conventional "cut-film" angiography in all cases, even though the resolution of the latter is still superior on magnified runs in the depiction of distal intracranial vessels. The digital subtraction angiography (DSA) equipment should be capable of 1024×1024 resolution, have advanced road-mapping facilities, and bi-plane is advantageous, although less so than with arteriovenous malformations (AVM) or aneurysm treatment.

Arch aortography is rarely required unless there is evidence to suggest proximal vessel disease (Takayasu's arteritis, dissecting aneurysm) or where catheterization proves difficult. If performed, a 5-F pigtail catheter with multiple side holes is positioned in the ascending aorta, left anterior oblique (LAO) projection, contrast 30–40 ml at 20–25 ml/s, 4 images per second for 5 s then 1/s for 5–6 s.

Selective common carotid catheterization is then performed using 5-F catheters with simple shapes and soft-tip guidewires. The authors' standard guidewire is a 0.035-in. Bentson (Cook), with a very soft and atraumatic tip. If the arch is elongated with tortuous origins of the brachiocephalic vessels, a stiffer wire (e.g., hydrophilic-coated Terumo glide-wire (Medi-Tech) with the same catheters, stiffer larger diameter catheter (e.g., 7-F Berenstein) or a Simmons catheter may be necessary.

The wire is inserted to the tip of the catheter in the ascending aorta, the catheter rotated in an counterclockwise direction until pointing towards the patients head, and then both are slowly withdrawn. This will usually engage the ostia of the arch branches, allowing the guidewire to be advanced to C6–C7, followed by the catheter. The wire is then withdrawn, 2–3 ml of blood aspirated, and heparinized saline injected from a second syringe. Try to avoid passing the wire to the level of the bifurcation to decrease the chance of atheromatous material embolization. Do not advance the catheter to the tip of the wire as this makes local dissection or spasm secondary to the wire more likely. Only proceed if good back flow of blood is obtained; if not, reposition the catheter by withdrawal until good flow is restored (it may be that thrombus occludes the catheter or the end hole is occluded by the vessel wall).

Standard projections are oblique and lateral of the cervical carotid, 5–8 ml at 5–8 ml/s. For the intracranial circulation, the standard views are AP and lateral, 7–10 ml for 6–8 ml/s. In patients with TIA or stroke, there are often multiple sites of arterial disease and selective internal carotid injection is not performed unless the bifurcation is free of significant disease; it is usually reserved for the interventional procedure. Meticulous technique is necessary in

neuroangiography to avoid serious complications; this becomes even more important as more selective catheterization is performed.

The left vertebral artery is the same size or larger than the right in 75% of patients and usually there is a straighter course from the aorta to the left than the right. Hence the first vertebral artery to be studied should be the left (although noninvasive preoperative information from MRA should be noted and may indicate that the right is dominant). As atherosclerotic disease is common at the origin of the vertebral arteries, subclavian injections are first performed to exclude significant disease before passing a wire and catheterizing the artery. The catheter is not usually advanced beyond a tight bend for diagnostic studies; indeed the entire study can be performed from the subclavian artery if significant ostial disease is present. The cervical segment is studied in ipsilateral oblique projection, 5–6 ml at 4–6 ml/s. The intracranial views are the Townes, Waters, and lateral: 6–9 ml at 4–6 ml/s, depending upon vessel size, flow, distal disease. For both the vertebrobasilar and carotid circulations, further targeted oblique views are performed as necessary to assess any specific intracranial lesion. The contralateral vertebral artery is always assessed unless there is a specific contraindication. Special techniques such as cross compression views with either carotid or vertebral artery injection are rarely performed in patients with TIAs or stroke. The preceding describes our standard angiographic evaluation applied to all of the pathologies discussed in the present chapter, with supplementary views or more selective vessel catheterization in certain pathological conditions.

The description of the stenosis includes the precise anatomical site, proximity to branch vessels or tight bends of the parent artery, severity, length, and concentric vs eccentric. The circle of Willis must be characterized, and this will indicate the likely hemodynamic significance of the lesion. When the patient satisfies the inclusion criteria, the final decision on whether intracranial angioplasty is possible and is to proceed is made by the neurointerventional surgeon.

Indications

We currently limit our treatment of intracranial fixed stenotic lesions to patients with stenoses of greater than 70% who have failed maximal medical therapy and are left with no options short of high-risk surgical revascularization procedures. Maximal medical treatment should include at least a trial of two antiplatelet drugs, control of standard vascular risk factors, and anticoagulation should have been considered (although this remains controversial). Focal concentric stenoses are more favorable lesions to treat and eccentric elongated or heavily calcified stenoses are more prone to complications. With the availability of stents, such lesions are still offered treatment if they fulfill the criteria mentioned in this paragraph.

Technique

Following angiographic documentation of a greater than 70% diameter stenosis involving the intracranial cerebral circulation, informed consent is obtained after discussion with the patient and family members. This has often been obtained after previous angiography at the referring institution; occasionally MRA will indicate the likely site and severity of disease, allowing consent followed by diagnostic evaluation and intervention in one stage. Patients are adminis-

tered enteric-coated aspirin (325 mg qd) and clopidogrel (Plavix 75 mg qd) starting 2 days prior to the procedure. Following the procedure, the patient is kept on daily aspirin indefinitely and on clopidogrel for 6 weeks. When the procedure is commenced, the patients are fully anticoagulated. The patient undergoes measurement of a baseline activated clotting time (ACT) and receives an initial weight-based (70 units/kg) intravenous bolus of heparin followed by a postheparin ACT determination to achieve an ACT value equal or greater than 2.5 times the baseline value (> 250 s). The patient then receives either an hourly dose equal to half the initial bolus or is placed on a heparin drip of 15–20 U/kg per hour. The role of glycoprotein IIb/IIIa inhibitors, which have been shown to decrease mortality and morbidity in a number of coronary stent studies [27], remains to be defined in carotid and vertebral angioplasty and stenting. We routinely use these agents in all patients undergoing intracranial angioplasty or stent-assisted angioplasty, in the absence of contraindications.

The patients are maintained well-hydrated during the procedure, although this is tailored to the specific patient and the cardiac status. The procedure consists of placing a

7-F sheath (Avanti, Cordis Endovascular, Miami Lakes, Fla.) in the common femoral artery for the low-profile balloon-mounted coronary stents such as the GFX, S540 (Medtronic, Santa Rosa, Calif.) or the GR-2 (Cook Cardiology, Bloomington, Ind.). A guide catheter (6–7 F) is positioned as far distal as possible – near the skull base in the internal carotid artery or just before the C1–C2 loop of the vertebral artery – to allow a 2.3-F microcatheter (Rapid Transit, Cordis Endovascular, Miami Lakes, FL) over a 0.014-in. microguidewire (Transend 14, Boston Scientific Corporation, Boston, Mass.) to cross the lesion. A 300-cm-long, 0.014-in. exchange microguidewire (Stabilizer, Cordis Endovascular) is passed through the microcatheter well beyond the site of stenosis and the microcatheter is then withdrawn. With the exchange guidewire in place, primary or secondary stent-assisted angioplasty is performed. In the case of primary angioplasty, a low-profile balloon catheter (Titan/Jupiter, Cordis Endovascular, Miami Lakes, Fla.; Bandit NC Balloon or Fastealth Balloon, Boston Scientific Corporation, Boston, Mass.) is used to cross and predilate the lesion. For intracranial vessels, balloon diameter sizes range from 2.0 to 5.0 mm. The size is chosen after careful measurement of the adjacent

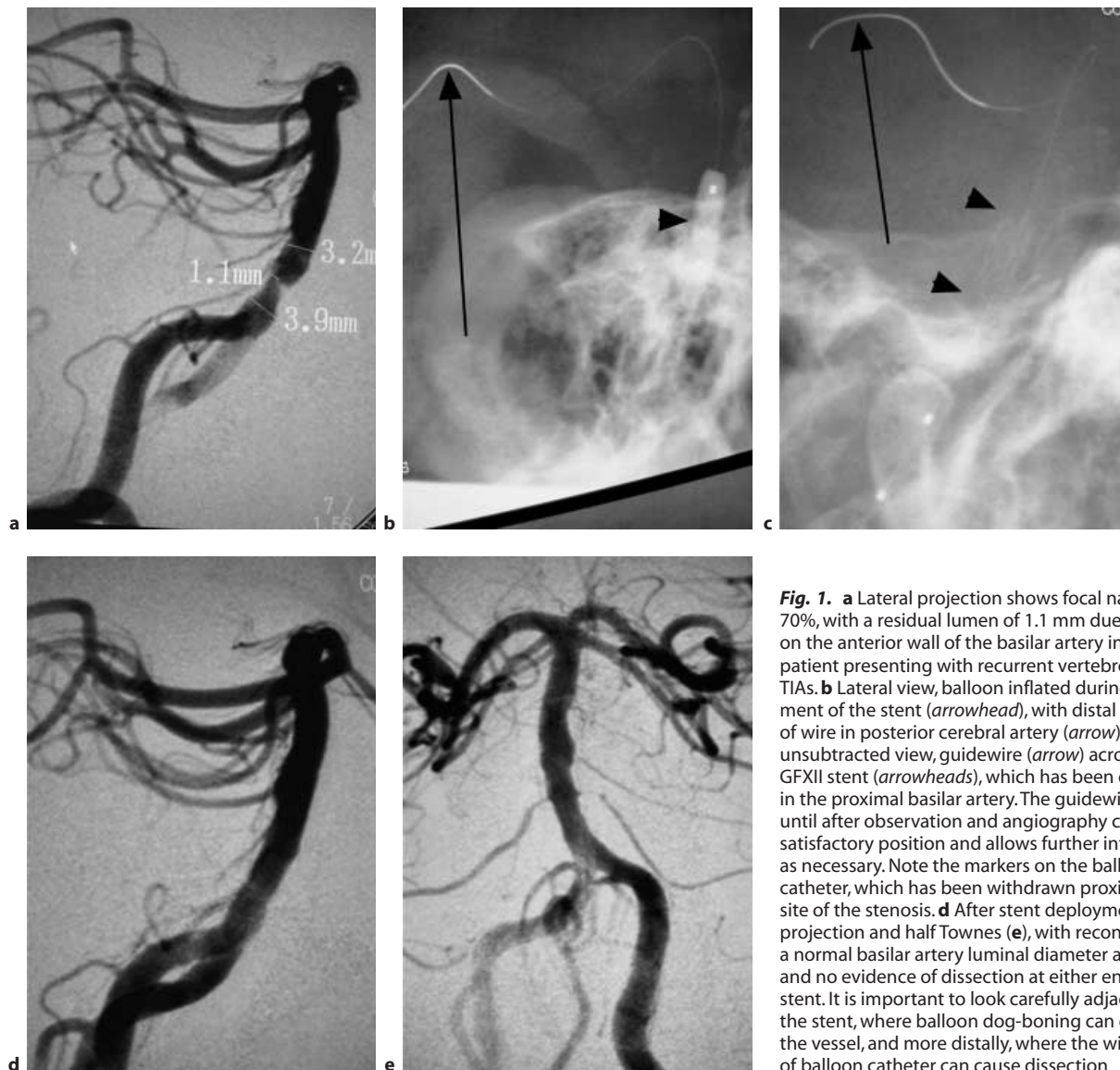


Fig. 1. **a** Lateral projection shows focal narrowing of 70%, with a residual lumen of 1.1 mm due to plaque on the anterior wall of the basilar artery in this patient presenting with recurrent vertebrobasilar TIAs. **b** Lateral view, balloon inflated during deployment of the stent (arrowhead), with distal position of wire in posterior cerebral artery (arrow). **c** Lateral unsubtracted view, guidewire (arrow) across the GFXII stent (arrowheads), which has been deployed in the proximal basilar artery. The guidewire remains until after observation and angiography confirms a satisfactory position and allows further interventions as necessary. Note the markers on the balloon catheter, which has been withdrawn proximal to the site of the stenosis. **d** After stent deployment, lateral projection and half Townes (**e**), with reconstitution of a normal basilar artery luminal diameter and contour, and no evidence of dissection at either end of the stent. It is important to look carefully adjacent to the stent, where balloon dog-boning can damage the vessel, and more distally, where the wire or tip of balloon catheter can cause dissection

normal vessel diameter, ensuring that an area of poststenotic dilatation is not measured and that the balloon diameter is equal to or less than this measurement. For example, if the measurement is 3.4 mm, a 3.0-mm and not a 3.5-mm balloon is chosen. The balloon is inflated to the maximum rated burst pressure for 10–30 s. Some vessels require more than one dilatation to achieve a satisfactory result of less than 30% residual stenosis. Once the stenosis has been decreased, a stent can be deployed, and a high-pressure noncompliant angioplasty balloon is then used to postdilate the stent in order to firmly embed it into the plaque.

Stent-assisted angioplasty is preferred for focal high-grade and eccentric lesions (Fig. 1) to minimize the chance of acute vessel closure or delayed thromboembolic complications related to a dissection flap. Predilatation is uncommonly performed and the balloon and stent diameters are carefully chosen to be the same or 0.1–0.2 mm less than the diameter of the adjacent normal vessel. The stents used are second-generation low-profile balloon-premounted coronary stents (GFX, S540 AVE; GR-2, Cook; MultiLink, Guidant) of 2.5- to 4-mm diameter. Where possible we will primarily stent for basilar and vertebral artery stenoses, only predilating when the morphology and severity suggest difficulty in passing the stent across the lesion. If the stenosis is close to the origin of a major branch and stent placement will necessarily cross that ostium, angioplasty alone is preferred, with stent placement for suboptimal postangioplasty appearance. Most lesions of the internal carotid, middle cerebral, and posterior cerebral arteries are not amenable to stent placement and in these we currently prefer angioplasty alone.

Connors et al. report routine use of ReoPro (Abciximab, Ely-Lilly Australia, Sydney, NSW) [28] with a very low incidence of ischemic stroke since adopting this protocol. We also routinely use ReoPro for all cases of intracranial angioplasty and stent-assisted angioplasty. The site is observed after PTA±S and if no evidence of platelet adhesion or dissection flap, the patient is continued on heparin for 24–48 h and hypotension avoided in the neurointerventional intensive care unit.

Complications

Potential complications associated with angioplasty include vessel occlusion, perforation, dissection, thromboemboli, occlusion of adjacent vessels, stroke, and worsening of the patient's symptoms from temporary occlusion during balloon inflation.

The most important complication is the development of a focal neurological deficit (stroke) after the procedure due to local occlusion (dissection, thrombosis, platelet aggregation) or distal embolization (from ruptured plaque, catheter, or proximal vessel thrombus). Selective angiography should be performed immediately – the development of local intraluminal filling defects may represent adherent platelet aggregation or embolus from a more proximal site. If occurring despite heparin and ReoPro, urokinase is administered locally, 100,000 U over 5–10 min followed by a further 100,000 U if no improvement is noted. Stent placement is performed if anatomically possible or if dissection is suspected as the cause for the occlusion following angioplasty alone. If distal embolization has occurred, heparin is maintained, ReoPro continued, and local urokinase administered if the deficit is significant. However, if a small distal branch occlusion has occurred and there is no or minimal focal neurological deficit, aggressive local chemical or mechanical

measures should be avoided, as there is a high probability of recovery with continued anticoagulation, and support of blood pressure and oxygenation in the neurointensive care unit.

INTIMAL DISSECTION

Intimal dissection is not unexpected following plaque rupture by balloon angioplasty, but commonly heals, often with further reduction in degree of residual stenosis. Dissection should be treated if symptomatic or associated with marked alteration in flow, even if asymptomatic. Stent deployment is preferred, but if at a site where stents are not possible, repeated PTA and treatment of any associated platelet adhesion may be all that is available.

VESSEL RUPTURE

Vessel rupture during angioplasty usually occurs in the context of full anticoagulation, and in recent times in the presence of abciximab, and is usually immediately fatal. Fortunately this is a rare complication.

PERFORATION

Perforation with a catheter or guidewire is recognized by clinical (sudden headache, loss of consciousness, rising blood pressure, and decreasing pulse rate) or angiographic (wire beyond vessel wall, contrast extravasation) features. If the catheter is across the perforation it must not be withdrawn – leave it in place, reverse heparin, and hope that the abciximab infusion has not been started! Platelet infusions could be considered if abciximab has been used, controlling the site mechanically until completed, although we have no experience using this strategy. Depending upon the location, it may be possible to place a microcoil across the perforation site; it is also possible to place the balloon catheter across the site and tamponade the bleeding by prolonged inflation of the balloon. Deflating after heparin reversal may show control of the perforation. If all else fails, vessel sacrifice with detachable balloons may be a life-saving procedure, although will clearly not be possible for all sites.

Most important is the avoidance of such complications, as their successful treatment is difficult or impossible and often associated with significant morbidity. Appropriate training of the neurointerventional surgeon must include neuroanatomy, neurology, neuroradiology, pharmacology, as well as the technical skills necessary to access and intervene in the intracranial circulation if these complications are to be minimized. Stent placement or deliberate underdilatation are two currently available strategies to minimize the occurrence of flow-limiting postangioplasty intimal dissection.

Follow-up Evaluation

MRA is noninvasive and is used to assess the vessel treated at 6 months. There is a tendency to overestimate recurrent stenosis severity, less with newer magnets with higher gradient strengths and lower echo times (TEs). If patients are symptomatic full diagnostic evaluation with DSA is performed. Although repeat DSA is the most specific and sensitive modality to detect recurrent disease, patients with intracranial vascular disease often have severe general vascular disease and are at relatively high risk of complications from cerebral angiography. Routine angiographic follow up is therefore difficult to justify in asymptomatic patients.

TABLE 1. Intracranial angioplasty – selected series

| Group | Year | Patients | Success rate (%) | Stroke rate (%) | Mortality (%) |
|-----------------------------|------|----------|------------------|------------------|---------------|
| Clark [30] | 1995 | 17 | 82 | 12 | 0 |
| McKenzie [31] | 1996 | 11 | 100 | 8 | 0 |
| Terada ^a [36] | 1996 | 12 | 83.3 | 16.6 | 0 |
| Higashida ^a [38] | 1996 | 33 | 100 | 18.2 | 12.1 |
| Takis ^a [33] | 1997 | 10 | 80 | 40 | 0 |
| Mori ^a [32] | 1998 | 42 | 79 | 5 | 0 |
| Terada [36] | 1998 | 3 | 100 | 0 | 0 |
| Marks ^a [34] | 1999 | 23 | 91 | 0 | 4.3 |
| Eckard [35] | 1999 | 8 | 87 | 0 | 0 |
| Connors ^a [28] | 1999 | 70 | 100 | 4.2 ^b | 2.9 |
| Gock [221] | 1999 | 8 | 100 | 12 | 0 |

^a Includes vertebrobasilar sites.

^b Later period ($n = 50$), no occlusive stroke.

Experience to Date

ANGIOPLASTY

Sundt first reported cerebral angioplasty of the intracranial circulation [29]. He described operative exposure of the vertebral artery, insertion of a coronary balloon angioplasty catheter, and dilatation of a basilar artery stenosis. Since that time, numerous case reports and small series of intracranial angioplasty have been published [28, 30–39]. Selected results are summarized in Table 1.

Higashida et al. reported 325 cases in 1996 with a permanent complication rate of 17 strokes (5.2%), including 4 deaths (1.2%). In the 33 cases of intracranial angioplasty, there were 7 (21.2%) cases of transient cerebral ischemia, and 10 (30.3%) strokes, which included 4 deaths. This contrasts markedly with the 292 cases of extracranial angioplasty, with 5.5% transient cerebral ischemia, 2.4% strokes, and no deaths. Intracranial angioplasty appears to be inherently more difficult to perform safely. This is due to the smaller size of the vessels being treated, increased tortuosity of the intracranial vessels, presence of perforating side branches, and more difficult access to this region. Patients treated since the initial series have deliberate undersizing of the balloon, or receive stent-assisted angioplasty to minimize the chance of vessel rupture, clinically significant dissection, and ischemic stroke.

A series of 22 vessels in 17 patients with recurrent neurological symptoms referable to the stenotic vessel despite optimal medical therapy were treated with PTA [30]. PTA was successful in 82% of the vessels, with two strokes during angioplasty for a 30-day morbidity rate of 11.7% per case. The remaining patients were without further events. There was delayed improvement in degree of residual stenosis.

Connors et al. reported a retrospective analysis of balloon angioplasty ($n = 70$) for intracranial atherosclerotic stenosis [28]. They divided their experience into three distinct periods based on the technique used. In the early period, the angioplasty balloon size approximated the vessel size and angioplasty was moderately rapid and brief. No neurological complications occurred. In the middle period, the balloon size approximated the vessel size, but oversizing was permitted. Angioplasty was extremely rapid and brief. Angiographically visible dissection occurred in 75% of patients, necessitating urokinase (Abbott Laboratories, Chicago, Ill.) infusion in 41.7% and producing abrupt occlusion in 8.3%, resulting in death. Good outcome was eventually achieved in 83.3% of patients. In the final period ($n = 50$),

the balloon was always undersized, inflation extremely slow (several minutes), and abciximab routinely administered. Dissection only occurred in 14% patients, necessitating fibrinolysis in 4%, producing no abrupt occlusion or stroke. Overall results from the series were a 4.2% stroke rate (none in the last 50 patients), 2.9% mortality, and 100% technical success (although with moderate residual stenosis). Conclusions reached from analysis of their 9-year experience were to minimize vessel trauma with deliberate undersizing of the balloon, slow inflation, and use of abciximab to decrease the likelihood of platelet activation and aggregation at the angioplasty site. This technique sometimes yields suboptimal angiographic results but achieves the clinical goal safely.

Angioplasty of the cerebral arteries for acute occlusion has been reported in the anterior and posterior circulation, and may allow improved outcomes in cases of suboptimal thrombolysis [40–42].

STENT-ASSISTED ANGIOPLASTY

Intracranial atherosclerotic stenoses of the carotid and vertebral arteries and their branches can now be treated with stent placement concurrently with or following angioplasty. This treatment is reserved for lesions that are symptomatic despite maximal medical treatment and have either had a mechanical complication of angioplasty or are considered to have an increased chance of poor outcome from PTA alone. In the carotid artery, stent placement has been performed or reported in the intracranial petrous, cavernous, and proximal supraclinoid segment. In the posterior circulation, stents have been deployed in the intracranial V4 segment and in the mid segment of the basilar artery [40, 43–45]. Compromise of perforating vessels supplying the brainstem remains a factor which may limit the use of mid-basilar stents, although in our experience symptomatic perforator occlusion is rare.

Conclusion

With improvements in balloon technology, microguidewires, and fluoroscopic equipment, the complication rates may dramatically decrease as with coronary angioplasty techniques. Currently, there are few surgical options available for symptomatic patients with intracranial stenosis who have failed medical therapy; therefore endovascular approaches appear to offer the best opportunity for treatment of hemodynamically relevant intracranial arterial stenoses. Patient selection remains imperfect, as patients with greater than 70% stenosis

are not treated until there is evidence of failed best medical treatment. Unfortunately this may constitute a completed stroke with death or major disability. Identification of patients more likely to fail medical therapy would allow earlier treatment with interventional techniques and improved outcomes for the patient population with symptomatic intracranial arterial stenoses. There will be an increasing role for PTA and PTA in conjunction with stent implantation (PTAS) as the risks of the procedure continue to diminish, the technical results improve, and functional MRI identifies the patients at most risk from their disease. More flexible stents, with excellent radial force, able to prevent neointimal hyperplasia, will be of great assistance in extending the numbers of patients who can benefit from these new techniques.

POSTSUBARACHNOID HEMORRHAGE VASOSPASM

Introduction

Cerebral vasospasm following subarachnoid hemorrhage (SAH) is the single most important cause of death and disability for survivors [46]. It is estimated that in the United States 6,000–10,000 potential patients per year may benefit from revascularization therapy for arterial spasm [47]. Angioplasty of intracranial arteries for the treatment of vasospasm following subarachnoid hemorrhage is a useful technique for treatment of cerebral hypoperfusion that is unresponsive to maximal hypertensive, hypervolemic, and hemodilutional therapy (triple-H therapy) [48–50]; antispasmodic agents delivered via direct transarterial catheter injection can be useful but for a shorter duration [50].

The first reports of balloon angioplasty for cerebral arterial vasospasm were from Zubkov and Nikiforov in 1984 [51]. They reported the successful treatment of 33 cases involving 105 vascular territories, with good clinical outcome in the majority of cases. A number of other investigators have reported upon this technique. Higashida reported on 28 patients involving 99 vascular territories in which 19 patients (66.7%) showed clinical and neurological improvement following angioplasty [49]. Eskridge et al. reported on 50 patients with clinical evidence of vasospasm-induced ischemia; 28 (61%) showed sustained neurological improvement within 72 h of angioplasty [48]. The mechanism of balloon dilatation is very different from the mechanism for atherosclerosis, with no evidence of disruption of the media and intima in animal studies [52].

Diagnostic Evaluation

The Fisher grade predicts the occurrence and severity of cerebral vasospasm after SAH. Those with significant subarachnoid blood are monitored with transcranial Doppler ultrasound and elevated velocities are managed with aggressive medical therapy. If there is evidence of vasospasm and focal or generalized neurological deficits develop, CT is performed to exclude hydrocephalus, completed infarction, intracerebral hemorrhage, or further SAH. If these are absent full cerebral angiography is performed with the intention to proceed to therapy if vasospasm is confirmed. Routine post-SAH angiography and treatment of asymptomatic vasospasm has been performed, but we believe therapy should be performed where there is a clinical as well as a technical indication.

Indications

The indications for utilizing these techniques include patients who are clinically symptomatic, have failed maximal medical therapy, have angiographic stenosis correlating with their symptoms, and do not have evidence of massive cerebral infarction or cerebral hemorrhage. Acute hemorrhage within 6 h of presentation is a relative contraindication since patients undergoing therapy need to be systemically anticoagulated during the procedure to avoid thromboembolic complications from the balloon dilatation procedure or microcatheter. When an aneurysm remains, which has not been excluded from the circulation, and severe symptomatic vasospasm develops, the risk posed by vasospasm-induced ischemic stroke is probably greater than that from anticoagulation and angioplasty. We would treat the spasm in this situation, but would generally prefer to exclude the aneurysm from the circulation by coils or clipping before treatment.

Procedure

The angioplasty is performed while the patient is anticoagulated, under general anesthesia, and with a specifically designed low-profile compliant balloon mounted on an atraumatic flexible catheter (Interventional Therapeutics Corporation, San Francisco, Calif.). This balloon differs significantly from standard angioplasty balloons. Silicone was chosen because of its unique properties of malleability, isotropic expansion capabilities, and special conformational structure. The material allows for the balloon to conform to the vessel lumen without exerting excessive lateral pressure against the vessel wall, which might result in inadvertent vessel rupture [53]. Standard regimens of heparin, with an ACT of 250–300, are usually safely tolerated in the immediate postoperative period and following endovascular treatment.

The balloon catheter is deployed via a guide catheter positioned in the extracranial internal carotid or vertebral artery. The balloon is gently inflated using the digital road-mapping technique for a short duration to enable intermittent cerebral perfusion, is gently advanced, and the inflation is repeated (Fig. 2). The vessels most commonly treated are the supraclinoid internal carotid artery, the M1 (or first) segment of the middle cerebral artery, and the vertebral and basilar arteries. The A1 (first) segment of the anterior cerebral artery is less commonly treated and can be more difficult to access, particularly after treatment of the M1 segment. While over the wire catheters are more easily positioned in that segment, the chance of vessel rupture or perforation is higher with these less compliant balloons. There is a longer-lasting benefit with mechanical balloon angioplasty than with intraluminal infusion of papaverine [48, 50]. Following the procedure, a postangioplasty or infusion arteriogram should be obtained to evaluate the change in vessel caliber, qualitatively evaluate cerebral blood flow, and determine any vessel damage.

Selective intra-arterial papaverine infusion has been used via a microcatheter in symptomatic spastic vessels with doses of 300 mg in 100 ml of normal saline over 30 min [48, 54, 55]. This appears safe – although there are reports of elevation of intracranial pressure, transient monocular blindness, and significant systemic hypotension – and efficacious in early vasospasm, and has the benefit of dilating vessels beyond the reach of balloon catheters, but is of short-lived ben-

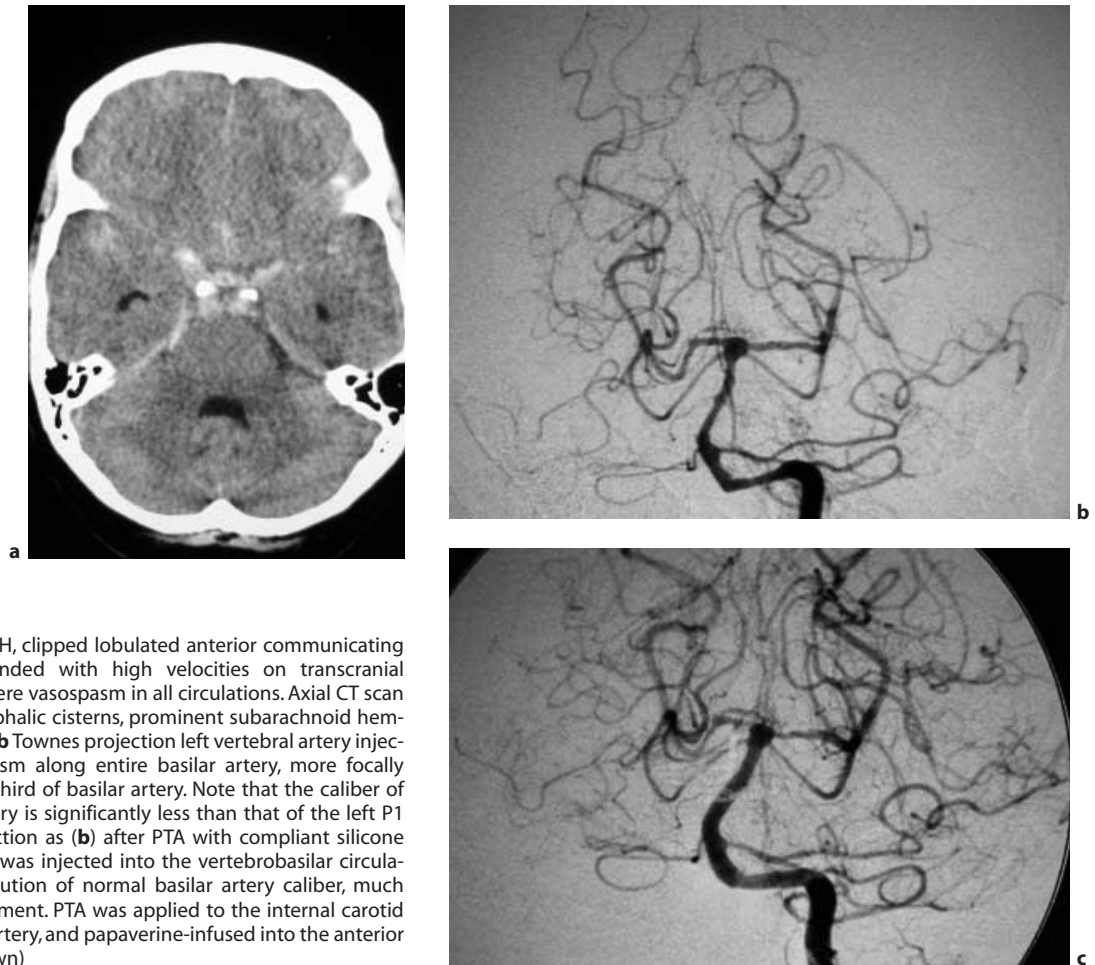


Fig. 2. **a** Day 7 after SAH, clipped lobulated anterior communicating artery aneurysm, obtunded with high velocities on transcranial Doppler ultrasound, severe vasospasm in all circulations. Axial CT scan at level of perimesencephalic cisterns, prominent subarachnoid hemorrhage, Fisher grade III. **b** Townes projection left vertebral artery injection. Moderate vasospasm along entire basilar artery, more focally severe at terminal one-third of basilar artery. Note that the caliber of the terminal basilar artery is significantly less than that of the left P1 segment. **c** Same projection as (**b**) after PTA with compliant silicone balloon. No papaverine was injected into the vertebrobasilar circulation. Note the reconstitution of normal basilar artery caliber, much greater than the P1 segment. PTA was applied to the internal carotid artery, middle cerebral artery, and papaverine-infused into the anterior cerebral artery (not shown)

efit. In our experience, it is less efficacious in late persistent vasospasm, presumably as the pathology at this stage reflects more structural changes within the wall. There are significant numbers of patients with recurrent symptoms, which remain amenable to repeat treatment.

Complications

Complications involve vessel rupture that is usually fatal, which is less common if stiffer angioplasty balloon catheters are avoided. Dissection can be minimized by gentle and slow balloon inflation, using an undersized compliant balloon. Stroke from distal embolization is uncommon and minimized by using full periprocedural anticoagulation with heparin. Recurrent SAH may occur if vasospasm is treated successfully in the presence of an unprotected ruptured aneurysm, and in general, treatment is preferred following aneurysm clipping or coiling.

Conclusion

Vasospasm should be treated by endovascular techniques when severe and symptomatic, preferably after treatment of the cause of subarachnoid hemorrhage (clipping or endovascular coil obliteration of aneurysms). Balloon-dilatation of intracranial vessel segments using a compliant balloon catheter has established its efficacy and relative safety in the treatment of subarachnoid hemorrhage-related intracranial

vasospasm that has failed maximal hypertensive, hypervolemic, and hemodilutional therapy. Intra-arterial papaverine administration can facilitate balloon angioplasty and treat more distal vasospasm beyond the safe reach of the angioplasty catheter. The preferred technique is angioplasty, with local intra-arterial papaverine of use for more distal vessel involvement beyond the safe reach of the balloon catheter.

INTRA-ARTERIAL THROMBOLYTIC THERAPY FOR STROKE

Introduction

Standard medical management of ischemic stroke results in severe neurological deficit or death in many patients. The 30-day and 5-year mortality rates for stroke in the carotid territory are 17% and 40%, respectively [56]. These are higher in the subset with complete occlusion of the proximal middle cerebral artery (MCA) according to Saito et al. who reported up to 78% dead or severely disabled with only 9% having a good outcome [57]. Major vertebrobasilar occlusion with acute severe stroke has a particularly poor prognosis, with up to 80% mortality [58].

The goal of emergency therapy for acute ischemic stroke is preservation of tissue that is not irreversibly damaged. Neuroprotective agents and reperfusion are possible

means of achieving these goals. No large randomized prospective trial has demonstrated significant improvement in outcome from the use of neuroprotective agents. Several multicenter randomized trials evaluating intravenous administration of thrombolytic agents have been conducted [59–64], and only the National Institute of Neurological Disorders and Stroke (NINDS) Tissue Plasminogen Activator Stroke Trial demonstrated an acceptably low rate of intracranial hemorrhage and significant efficacy [59]. There was at least a 30% relative increase in patients with good outcomes in the tissue plasminogen activator (t-PA) group compared with the placebo group, despite symptomatic intracranial hemorrhage being more common in the t-PA group and comparable overall mortality. In addition, t-PA has received Food and Drug Administration approval for intravenous administration within 3 h of onset of stroke symptoms.

Early recanalization correlates with improved outcome in acute stroke patients, especially in patients with good collateral blood flow and no major early signs of infarction on CT [65]. Recanalization rates with intracranial artery (IA) thrombolysis are superior to intravenous (IV) thrombolysis for major cerebrovascular occlusions [66]. The differences in recanalization rates are most apparent with large vessel occlusions [66, 67].

Published series of intra-arterial thrombolysis in the carotid circulation (Table 2) show good outcomes in similar proportions of patients to those in the NINDS intravenous thrombolysis trial and comparable to slightly increased rates of hemorrhage. This was achieved despite significantly worse disability at entry and delayed time to treatment in one series [68].

Randomized multicenter trials of local intracranial intra-arterial thrombolysis include the Prolyse for Acute Cerebral Thromboembolism (PROACT) I and II trials [69, 70]. PROACT I demonstrated the safety of intra-arterial recombinant prourokinase and the PROACT II trial showed a significant increase in the rate of reperfusion, as well as a 60% relative neurological improvement for prourokinase therapy, with no significant increase in morbidity or mortality.

Direct thrombolytic therapy in the vertebrobasilar territory has been associated with a mortality of 31%–68%, with lower figures in more recent reports of case series [71–75] (Table 2). The outcome in all these series is markedly better than natural history. No large prospective randomized trial has been concluded. A wider therapeutic window is recognized in the vertebrobasilar territory, with good outcomes being reported up to 24 h after symptom onset [73].

Diagnostic Evaluation

In most centers, CT scanning is still performed first, as it is readily available, relatively unaffected by a restless patient, easy to monitor the patient during scanning, sensitive to hemorrhage, and sensitive to signs of major cerebral artery territory ischemic infarction within several hours of onset. MRI is more sensitive to postischemic change when diffusion-weighted imaging (DWI) is used, can differentiate infarcted tissue from ischemic tissue, show the carotid and vertebrobasilar arteries (both intracranial and extracranial segments), and has the promise of identifying threatened but viable tissue. Currently, the availability of scanners, the processing time required for perfusion studies, and the lack of trials studying therapy based on the information from these newer functional MR parameters limits the application of these techniques. Within a short period of time, it is highly likely that these limitations will be overcome, allowing identification of patients with stroke in whom the volume of hypoperfused tissue matches that deemed infarcted (in whom reperfusion strategies would carry risk but probably no benefit) and those in whom the infarct volume was much less than the hypoperfused volume (ideal candidates for reperfusion therapies).

Indications

Case selection is crucial to achieve good outcomes in ischemic stroke when using thrombolysis. A CT scan must be obtained before any therapy, to exclude other pathologies, for example, a tumor, and exclude intracranial hemorrhage or a large area of hypodensity. A poor outcome and higher incidence of hemorrhagic complications follows thrombolytic therapy in patients having hypodensity in greater than one-third of the territory of the MCA within 6 h of symptom onset [76]. The presence of coma or tetraparesis for several hours portends a poor prognosis, despite recanalization in patients presenting with vertebrobasilar thrombosis and stroke [74, 77]. Such symptoms do not preclude survival, however, and recovery has been documented after successful recanalization in such patients [77, 78]. Better collateral blood flow is correlated with improved responses to thrombolysis and with longer tolerance of ischemia [79]. CT is very useful in selecting patients with the potential to do well, but our ability to identify these patients should improve further with perfusion CT techniques and with the greater availability of magnetic resonance imaging (MRI) units capable of

TABLE 2. Intra-arterial thrombolysis for acute ischemic stroke

| Author | Patients | Recanalization (%) | Mortality (%) | Hemorrhage (%) |
|--|----------|--------------------|---------------|----------------|
| Barnwell et al. ^a [222] | 13 | 76.9 | 23.1 | 23.1 |
| Del Zoppo et al. ^a [223] | 20 | 90 | 0 | 20 |
| Jahan et al. ^a [68] | 26 | 43.2 | 38 | 38 |
| Jansen et al. ^a [224] | 16 | 12.5 | 56.2 | 56.2 |
| Mori et al. ^a [225] | 22 | 90 | 0 | 20 |
| Theron et al. ^a [226] | 12 | 100 | 16.6 | 25 |
| Hacke et al. ^b [74] | 43 | | 68 | – |
| Mitchell et al. ^b [73] | 16 | 82 | 31 | 0 |
| Becker et al. ^b [75] | 12 | 77 | 75 | 16.6 |
| Higashida et al. ^{a,b} [72] | 27 | 82.2 | 33.3 | 11.1 |
| Bockenheimer et al. ^{a,b} [227] | 18 | | 50 | – |
| Zeumer et al. ^{a,b} [71] | 31 | 93.5 | 0 | 0 |

^a Anterior circulation.

^b Vertebrobasilar circulation.

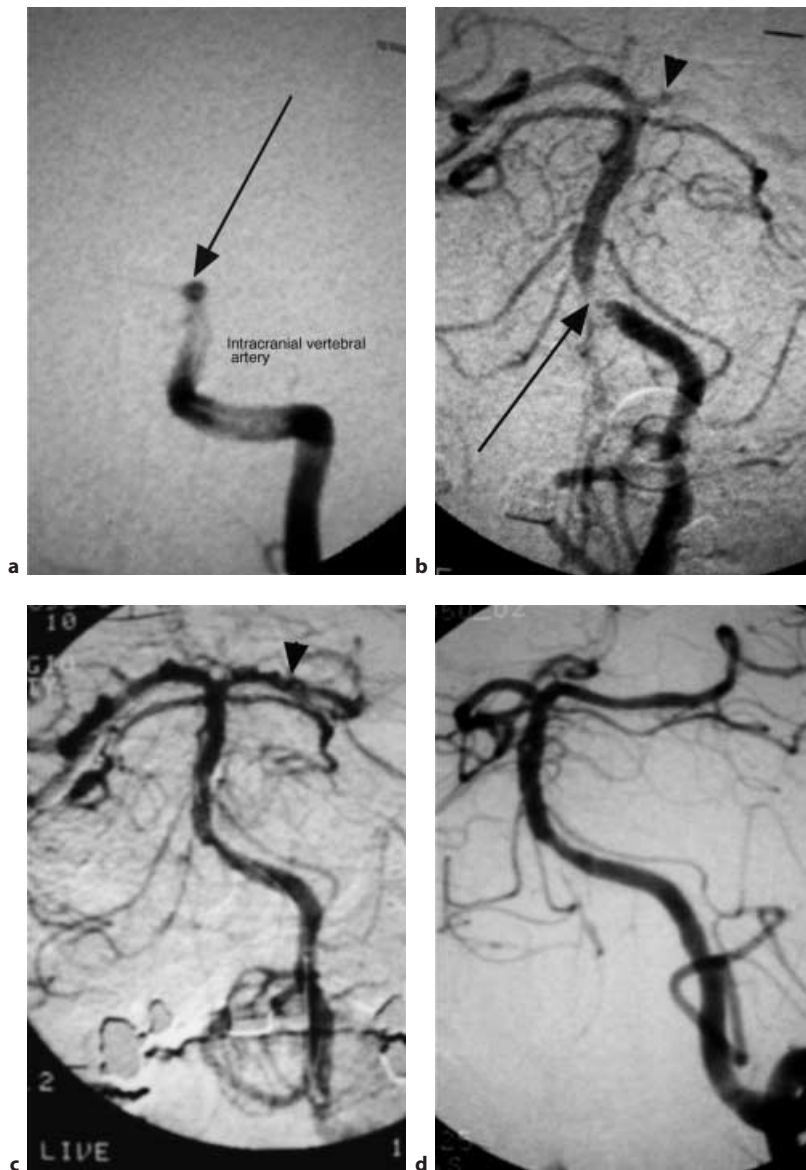


Fig. 3. **a** A 66-year-old man presenting semicomatose, no tumor or hemorrhage on initial CT (not shown), with selective left vertebral artery injection showing complete distal vertebral artery occlusion (arrow). The right vertebral artery was nondominant and carotid injection confirmed inadequate posterior communicating arteries, without significant filling of the distal basilar artery. **b, c** After urokinase, local pulsed infusion shows recanalization of vertebral and basilar arteries and an uncovered stenosis of the distal left vertebral artery extending to the vertebrobasilar junction (arrow). The left posterior cerebral artery does not fill (arrowhead) and no abnormal anatomy had been shown on the carotid study. A catheter (**c**) passes through the basilar artery for administration of more urokinase to thrombus in the posterior cerebral artery (arrowhead), which is now patent but with some remaining irregularity, indicating some persistent clot (treated with more urokinase). **d** Final images after angioplasty of the focal stenosis with reconstitution of a good luminal diameter, no dissection encroaching on the lumen, and good distal flow. The posterior cerebral artery is now free of thrombus. Following this excellent angiographic result, the patient made a good clinical recovery, returning to independence with no major deficit

echo planar imaging (EPI) to differentiate infarcted from ischemic tissue and identify patients in whom reperfusion has already occurred.

Procedure

For intra-arterial thrombolysis, a diagnostic catheter is guided into the high cervical segment of the vascular territory to be treated, followed by the introduction coaxially of a 2.3-F rapid transit microcatheter with a 0.016-in. instinct steerable microguidewire. (Cordis Endovascular Systems, Miami Lakes, FL). Under direct fluoroscopic visualization, the microcatheter is gently navigated through the intracranial circulation until the tip is embedded within the central portion of the thrombus (Fig. 3). Two types of microcatheters are being used for local cerebral thrombolysis, depending upon the extent of clot formation. For the majority of intra-arterial cases, a single end-hole microcatheter is used, while for longer segments of clot formation, multiple side hole infusion microcatheters are utilized. Superselective angiography through the microcatheter is performed at reg-

ular intervals to assess the degree of clot lysis and to adjust the dosage and volume of the thrombolytic agent.

Currently, for intra-arterial thrombolytic treatment, urokinase (Abbott Laboratories, Abbott Park, Ill.) is most frequently used, in the dose range of 25,000–50,000 U over 10-min intervals, at the rate of 250,000–500,000 U/h. Urokinase is available in 250,000-U vials and is mixed with 10 ml of sterile water, yielding a concentration of 25,000 U/ml. After each infusion of 125,000–250,000 U, a superselective angiogram is performed; if there is partial clot dissolution, the catheter is advanced into the remaining thrombus where additional thrombolysis is performed. As the thrombus is dissolved, the catheter is then directed into the more distal branches of the intracranial circulation, so that the majority of the drug enters the occluded vessel and is not washed preferentially into adjacent open blood vessels. Currently, therapy is directed at treating patients within 4–6 h of symptom onset. Treatment should not be denied patients with vertebrobasilar thrombosis even up to 24 h, as long as other factors are favorable. Regimens for the intra-arterial administration of recombinant tissue plasminogen activator (rtPA)

in the cerebral circulation have not been finalized with the manufacturer.

There is no standard systemic heparin regimen established for IA thrombolysis in acute stroke. PROACT I [69] reported a 27% rate of symptomatic brain hemorrhage when a conventional heparin regimen (100 U/kg bolus, 1000 U/h for 4 h) was used with IA r-proUK. Subsequently, a standard low-dose heparin regimen was used (a 2000-U bolus, 500 U/h for 4 h) which reduced the symptomatic brain hemorrhage rate with IA r-proUK to 7% in PROACT I and 10% in PROACT II. Based on the PROACT trials, many neurointerventionalists now use the low-dose heparin regimen during IA thrombolysis.

The potent IIb/IIIa platelet inhibitor abciximab (ReoPro, Eli Lilly & Co, Indianapolis, IN) has been used successfully instead of heparin in patients undergoing acute or elective cerebrovascular interventions [80]. Coronary doses of abciximab appear to be safe in patients with acute ischemic stroke up to 24 h after onset [81]. Abciximab may be most efficacious when the risk of acute reocclusion is great, such as with underlying atherothrombosis. It is typically given as an intravenous bolus of 0.25 mg/kg followed by continuous infusion of 10 µg/min for 12 h.

Conclusion

Intra-arterial stroke thrombolysis is evolving, as yet with no standardization of patient selection, neurointerventional techniques, or adjunctive therapy. Mechanical clot removal, new catheter techniques, and new adjunctive antithrombotic agents should improve the degree, speed, and safety of IA recanalization. Patient selection and the treatment window will be better defined by using new technologies such as perfusion-diffusion MRI. Comprehensive management utilizing all available tools – IV thrombolysis and cytoprotective agents – will be necessary to achieve improved patient outcomes, but only when timely arrival of patients to medical care occurs. The particularly poor prognosis and improved results shown in current series support application in the vertebrobasilar circulation. Patients in whom there is carotid territory stroke and a relative contraindication for systemic thrombolysis, or who fall outside the optimal 3-h time window for systemic therapy, may benefit from direct IA thrombolysis.

INTERVENTIONS FOR INTRACRANIAL VENOUS THROMBOSIS

Introduction

Dural venous sinus occlusion can be asymptomatic, associated with minor symptoms of headache, or with focal neurological deficit and decreasing conscious state. The mortality and morbidity is varied and is difficult to predict. The outcome in the majority of cases of dural sinus thrombosis managed with anticoagulation or supportive measures has been excellent and as a result more aggressive therapy has been reserved for clinical deterioration despite anticoagulation. Of 77 patients aged 18–77 with long-term follow-up [82], 85% had no neurological sequelae, 14% remained neurologically impaired, recurrent seizures occurred in 14% of patients, 12% suffered a second cerebral venous thrombosis (CVT), and noncerebral thrombotic events occurred in 14%. The authors conclude that CVT has an essentially good long-

term prognosis, although with the occurrence of a second CVT or another thrombotic episode in 20% of patients, a minority of cases will need long-term anticoagulation. Other authors have attempted to identify those patients at increased risk of poor outcome by using MRI and direct dural sinus pressure measurement [83]. Acute dural sinus thrombosis leads to distinct stages of parenchymal changes, the severity of which depends on the degree of venous congestion, which, in turn, is closely related to intradural sinus pressure. They identified five distinct stages of brain parenchymal changes (ranging from normal, edema, mass effect, and hemorrhage), each stage correlating with increasing intradural sinus pressure. The pressures measured ranged from 20 to 50 mmHg and brain parenchymal changes were reversible up to stage III if thrombolytic treatment was performed. All stage V patients died.

Excellent results of transcatheter local thrombolysis have been reported with no major complications [83–85]. The selection of patients in whom interventional techniques should be used awaits more precise delineation of the natural history of dural sinus thrombosis. Stent placement within the dural sinuses has been used to preserve patency after recanalization [86].

Diagnostic Evaluation

Diagnosis can be difficult owing to anatomical variation, poor visualization of normal cerebral veins, and the presence of intraluminal filling defects in normal dural sinuses. CT scanning is usually performed at the referring institution, and although often normal, can show sinus thrombosis as increased density or a filling defect on postcontrast scans; parenchymal edema or lobar hematomas should raise the suspicion of venous thrombosis. MRI and MR venography are the preferred imaging modalities and are usually sufficient to make the diagnosis. Review and comparison of interventional studies (angiography and venography) and cross-sectional imaging (MRI, MRA, CT) can be essential to reach a correct diagnosis and facilitate management plans. Full diagnostic cerebral angiography is performed with close attention to the venous phase of the arterial injections. Direct intracranial venography can be easily performed with a standard microcatheter and wire from the transvenous approach through the heart to the internal jugular veins. More often this approach is reserved for the intervention.

Indications

Most patients treated with anticoagulation have a good prognosis. If there is a contraindication to long-term anticoagulation, interventional techniques should be considered. MRI indicating mass effect and hemorrhage, very extensive sinus occlusions with markedly restricted normal venous drainage, evidence of progressive sinus thrombosis, or clinical deterioration are all indications to consider neurointervention.

Procedure

Access is usually from femoral venous catheterization, following diagnostic evaluation with full cerebral angiography and study of the venous phase of the arterial injection. Heparin has usually been commenced prior to the interventional procedure and should be continued even in the presence of small amounts of hemorrhage. Thrombolysis is more

effective in the presence of heparin, as rethrombosis and extension of thrombus into other veins is prevented. If enlarging venous hemorrhages are demonstrated while the patient is on heparin, albeit the lack of definitive trial-based data to guide decisions, most investigators would consider ceasing heparin, particularly if a mechanical device was available and could be used to clear the sinuses. If such a device is not locally available, heparin should in general be continued.

For thrombolysis in the dural sinuses, either bolus or continuous infusions are used through the catheter, at rates between 20,000 and 150,000 U/h of urokinase. If an infusion is used, it can be continued overnight. Angiography is performed intermittently throughout the procedure to document progression of clot lysis and to readjust the catheter placement within the thrombus to insure maximum infusion directly into the occluded segments. The catheter will easily pass through recent thrombus, despite complete occlusion on imaging; if catheterization is difficult there is usually a history of preexisting chronic organized thrombosis of

the dural sinus. The sinuses are accessed from a common femoral venous approach and a 5-F catheter is used to traverse the heart and enter the right internal jugular vein to the skull base. A coaxial system with a microcatheter is then used, the wire passed from sigmoid sinus across the arachnoid granulations of the transverse sinus, into the superior sagittal sinus.

Mechanical devices available include the guidewire, balloon angioplasty catheters, and the rheolytic thrombectomy device (CF105 AngioJet, POSSIS Medical, Inc., Minneapolis, MN), all of which have been used in dural sinus thrombosis. More often, these are used as adjunctive techniques to drug-based thrombolysis, but also as the sole technique when there are contraindications to fibrinolytic drugs. The large volume of thrombus compared to arterial occlusions and the more frequent associated hemorrhage are ideal situations for the use of mechanical clot disruption. The POSSIS device is effective in removing thrombus (Fig. 4) and if continuing improvements allow more reliable and safe access to the more

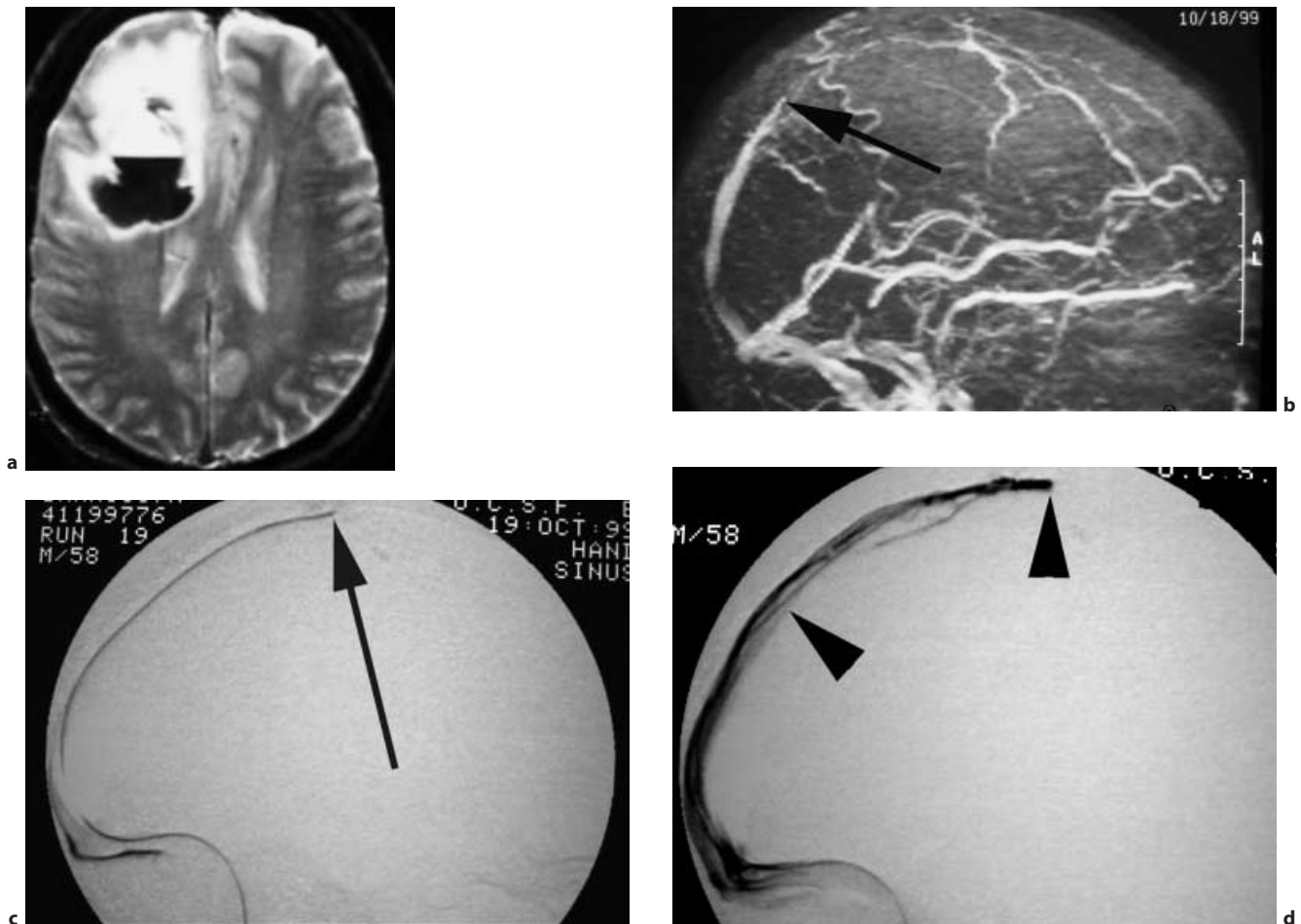


Fig. 4. **a** A 58-year-old man presented with seizures and hemiplegia. Diagnosis of venous hemorrhage secondary to major sinus thrombosis, treated with heparinization. Some hemorrhage progression, anticoagulation ceased, and these further images obtained. The axial T₂-weighted image shows a central fluid/fluid level with surrounding edema, moderate to marked mass effect. Superior sagittal sinus (SSS) hypointense signal intensity. **b** Lateral reconstruction, magnetic resonance venogram, with absent filling of the anterior two thirds of SSS, arrow indicates abrupt cut off of the sinus. **c** Arrow indicates the end of the rheolytic thrombectomy device, which could be advanced through the more proximal occlusion, but only to the junction of the middle and

anterior thirds of the SSS. **d** Final post-SSS venogram, with reconstitution of flow in middle and distal thirds of the SSS (indicated by arrowheads), but unable to recanalize the most proximal (anterior) one third. No thrombolytic drugs were used and heparin had been ceased because of the enlarging venous hemorrhage. Despite a reasonable mechanical result, with re-established flow through the bulk of the SSS, there was no significant clinical improvement. Early treatment will be necessary to maximize the chance of flow restoration, improving outcomes, with the best results likely to follow treating high-risk extensive thrombosis before major cerebral hemorrhage or venous infarction

proximal portions of the dural sinuses it will become the preferred mode of treatment. Displacement of small amounts of thrombus into the venous system and to the lungs has been shown to be safe in the periphery (recanalization of dialysis access grafts, iliofemoral thrombolysis, and mechanical disruption are known to cause embolism which rarely causes symptoms of chest pain or dyspnea).

Results and Conclusions

A significant proportion of patients – many young and otherwise healthy – with dural sinus or cortical venous thromboses suffer major morbidity or die despite therapy with anticoagulation. Patients deemed at higher risk can be identified: extensive occlusion with compromised remaining venous outflow on angiography, MR indicating major parenchymal changes, elevated intrasinus pressure at venography, and failed anticoagulant therapy. Combinations of pharmacological and mechanical thrombolysis are available and safe to use and should be used in these higher-risk patients. If improved outcomes result from this strategy and devices improve, application to larger numbers of patients under a prospective trial may be appropriate.

EMBOLIZATION

General Principles

PATIENT MANAGEMENT

A complete neurological examination must be performed prior to the procedure, as it is imperative to know the patient's baseline neurological examination so that even subtle changes can be detected and evaluated during and after the procedure. High-quality diagnostic cerebral angiography and review of all previous imaging is essential prior to embolotherapy. It is preferable to perform a complete four-vessel diagnostic cerebral arteriogram in a separate session rather than at the time of treatment. This allows additional time to review the films for more detailed planning and better understanding of the procedure. The patient and family must be given a complete explanation of the benefits and risks of the procedure and be given adequate time to ask questions so that they can truly make an informed decision.

As with all interventional procedures, patients should receive standard monitoring, including continuous EKG, heart rate, blood pressure, and pulse oximetry. Many procedures are now routinely performed under general anesthesia, especially in the pediatric population and when patients may not be able to fully cooperate. Disadvantages of general anesthesia are that it precludes direct neurological testing during the procedure, but this is generally outweighed by the ability to perform meticulous procedures with an immobile patient, the ability to perform diagnostic runs with apnea, and to proceed to immediate surgical or endovascular rescue of complications. Hemispheric or brainstem function can be monitored using electroencephalographic analysis and/or evoked potentials (acoustic or somatosensory).

Since most complex neurointerventional procedures require relatively large catheters and long procedure times, systemic anticoagulation is routinely used at most centers. Minor or short duration procedures can probably be performed without anticoagulation and without increased risk. Acute treatment of ruptured intracranial aneurysms are commonly performed under full anticoagulation, as the risks

from thromboembolic episodes would seem to outweigh any risk of adverse outcome from the heparin. Protamine sulfate is always available for immediate heparin reversal, should aneurysm perforation or on-table rupture occur.

The principles of patient care immediately following interventional therapy are similar to the postoperative surgical management for the particular disorder being treated. Following embolization, patients will often have worsening of their original symptoms and may complain of moderate to severe headaches. Transient worsening of symptoms is not uncommon in patients who present with focal deficits and is usually due to local edema, swelling, and acute inflammation from thrombosis. In the majority of cases, with conservative treatment consisting of analgesics, steroids or nonsteroidal anti-inflammatories, these symptoms will improve and usually they disappear. Headache is also a common complaint following embolization, especially with arteriovenous malformation (AVM) therapy. The etiology of the pain is not well understood; however, it is probably also due to the acute thrombosis and associated edema with local inflammatory response. Both headaches and worsening of the patient's symptoms may continue for several days to weeks following treatment. Imaging is essential to exclude hemorrhage or infarction in this setting.

Embolic Materials

Some of these agents are currently under clinical investigation in the United States and require institutional review board approval prior to use.

Embolic agents are conveniently divided into particulate and liquid, each of which has advantages and disadvantages. The liquid agents are generally more permanent, and will penetrate more distally – as such they are more technically demanding and are associated with higher risk of ischemic complications than the particulate agents.

GELFOAM

When Gelfoam (Upjohn, Kalamazoo, Mich.) is cut into sheets of sizes compatible with 5-F catheters, it is a useful agent for temporary occlusion of larger vessels, for example, as part of the treatment of intractable epistaxis, but has a limited role in intracranial embolizations.

POLYVINYL ALCOHOL PARTICLES

Polyvinyl alcohol (PVA) is a permanent agent commercially available in a range of sizes ranging from 50 to 1000 μm . This allows selection of a size appropriate for the arterial territory and clinical situation. The smaller particles allow greater penetration of the vascular bed being embolized, but have a greater risk of causing ischemia (cranial nerve deficits, skin necrosis). They are used for preoperative embolization of arteriovenous malformations (AVMs), dural arteriovenous fistulas (DAVFs), and tumors.

SILK SURGICAL SUTURES

Silk surgical sutures are prepared by cutting them into lengths of between 5 and 10 mm, which are placed in the end of a 1-ml syringe and injected. They cause an intense inflammatory reaction and are radiolucent. They are useful in situations of persistent flow despite the use of microcoils, when they can be injected into the coil basket, rapidly promoting thrombosis. Their use in arteriovenous malformations should be limited, due to the inflammatory response and increased pressure within the malformation during injection [87].

DETACHABLE BALLOONS

Detachable balloons are available in two basic varieties: latex and silicone. Detachable silicone balloons (DSB) were approved by the FDA for carotid occlusion and carotid cavernous fistula obliteration in the summer of 1998. Both types are available with self-sealing valves and can be detached from microcatheters [88]. Latex is impermeable; however, in vivo it may start to deflate within 2–3 weeks following placement. Silicone balloons are semipermeable and therefore must be inflated with solutions, metrizamide or visipaque, which are isotonic to blood [89]. Silicone balloons tend to deflate at a much slower rate and have been shown to be inflated beyond 3 years [90]. If permanent inflation is needed, the balloons can be filled with 2-hydroxyethyl methacrylate (HEMA) [91]. Some types of latex balloon are incompatible with HEMA, with fissuring and rupturing reported. The balloons have to be attached with sufficient force to avoid premature detachment, but they need to be detached with sufficient ease so that the balloon is left in the correct position without trauma to the parent vessel. They are used for carotidocavernous fistula ablation, direct carotico- and vertebrojugular fistulas, and parent-vessel occlusion in management of giant aneurysms or major skull base surgery. They are now infrequently used for elective direct management of aneurysms with vessel preservation because of the development of detachable coils (see “Coils”).

COILS

Stainless steel coils are very thrombogenic, are available in a range of outer coil diameters, and can be deployed through inexpensive 5-F catheters. As such they are ideal for peripheral embolization, and for neurointervention involving larger arteries or veins and sinuses. The need to deploy them through a large catheter, and the marked artifact generated during magnetic resonance imaging limits their applications in the nervous system vasculature.

Platinum microcoils are available in a variety of sizes and shapes. They produce a relatively permanent occlusion and can be easily placed either by injection or by conventional pushing with a special introducing guidewire to the desired location. They may have attached fibers for increased thrombogenicity (e.g., Dacron) and come in a range of sizes and shapes. The more complex the shape, the longer the coil; this and the presence of fibers all significantly increase the friction and hence the difficulty in deploying the coil. Coils are utilized in the treatment of large fistulous connections in AVMs, or as part of dural fistula management.

The development of detachable coils, in particular the Guglielmi detachable coil (Target Therapeutics, Fremont, Calif.), has largely overcome the problem of retrieving coils that have migrated from their intended site or are of inappropriate size. The Guglielmi detachable coil (GDC) is a soft platinum coil, attached to a wire that extends the length of the catheter. After satisfactory positioning, the coil is detached by an electric current passed through the wire. Mechanically detachable coils exist, but the GDC is better, and has been subject to more rigorous and widespread evaluation than the mechanically detached coils currently available. Both have been used for intracranial aneurysm obliteration, and also in carotidocavernous fistula and pseudoaneurysm treatment, either alone or with the use of stents.

LIQUID AGENTS

Liquid agents in current use include the cyanoacrylates and absolute alcohol. The polymerizing agent most commonly

used is *N*-butyl-2-cyanoacrylate (NBCA) (Histoacryl, Braun, Melsungen, Germany), a tissue adhesive which polymerizes on contact with ionic solutions. The time to polymerize can be altered using Lipiodol or glacial acetic acid, which facilitates deposition at the intended site. Other variables affecting deposition are flow rate, angioarchitecture, and the catheter position achieved. This agent sets rapidly (ideal for high-flow lesions) and is effectively permanent [92]. Because of its ability to penetrate the vascular bed, it has a higher risk of embolizing unwanted territories and potentially causing infarction, and the delivery catheter can be glued in place. It is of particular use in high-flow cerebral AVMs, DAVFs, and spinal dural arteriovenous fistulas.

Alcohol (96% ethanol) is used by direct injection into venous malformations of the head and neck. The alcohol causes immediate intimal damage, red blood cell clumping, and thrombosis. It may be injected via superselective arterial catheterization for management of DAVFs and parenchymal AVMs, but great care is necessary to avoid extensive skin necrosis or neurological deficit. Ethylene vinyl alcohol (Embolux, Micro Therapeutics Inc., Irvine, Calif.) is a liquid agent, which forms a nonadherent cast of the vessel or nidus [93]. It has been approved in Europe as an alternative to cyanoacrylates. Depth of penetration depends on the viscosity of the Embolux and the flow characteristics of the circulation.

There is currently no ideal embolic material for use in treating cerebral AVMs or aneurysms. Continued work is necessary and virtually all available agents could be improved by further development.

Tumors

INTRODUCTION

By decreasing the vascularity of head and neck tumors, surgical excision is facilitated (better visibility, shorter operative times) and the decreased blood loss can potentially avoid blood transfusion.

Diagnostic Evaluation. The tumor workup is with CT or more usually contrast-enhanced MRI, targeted to the site and nature of the tumor. These images and the likely pathological diagnosis will determine the likelihood of proceeding with neurointervention as a preoperative adjunct. For example, gliomas almost never receive any significant blood supply from the external carotid artery and embolization is inappropriate. Meningiomas, on the other hand, are usually supplied by the meningeal vessels alone and we would perform diagnostic cerebral angiography in most. The specific site will allow prediction of the supply and likelihood of embolization – frontal or parietal lesions often receive supply from the meningeal branches of the ophthalmic artery or cavernous internal carotid artery, in which case embolization is associated with increased risk of neurological complications and is not undertaken.

Full cerebral angiography is performed, with the addition of detailed selective external carotid injections. Proximal external carotid artery (ECA) injection is first performed, 5–8 ml at 4–6 ml/s, avoiding distal placement of wire or catheter to prevent spasm. More selective injections are performed as part of the embolization procedure, looking for anatomical variants and dangerous anastomoses (Fig. 5).

Indications. Tumors in which preoperative embolization is commonly used include meningioma, glomus tumors, and juvenile nasopharyngeal angiofibroma. Particles are the pre-

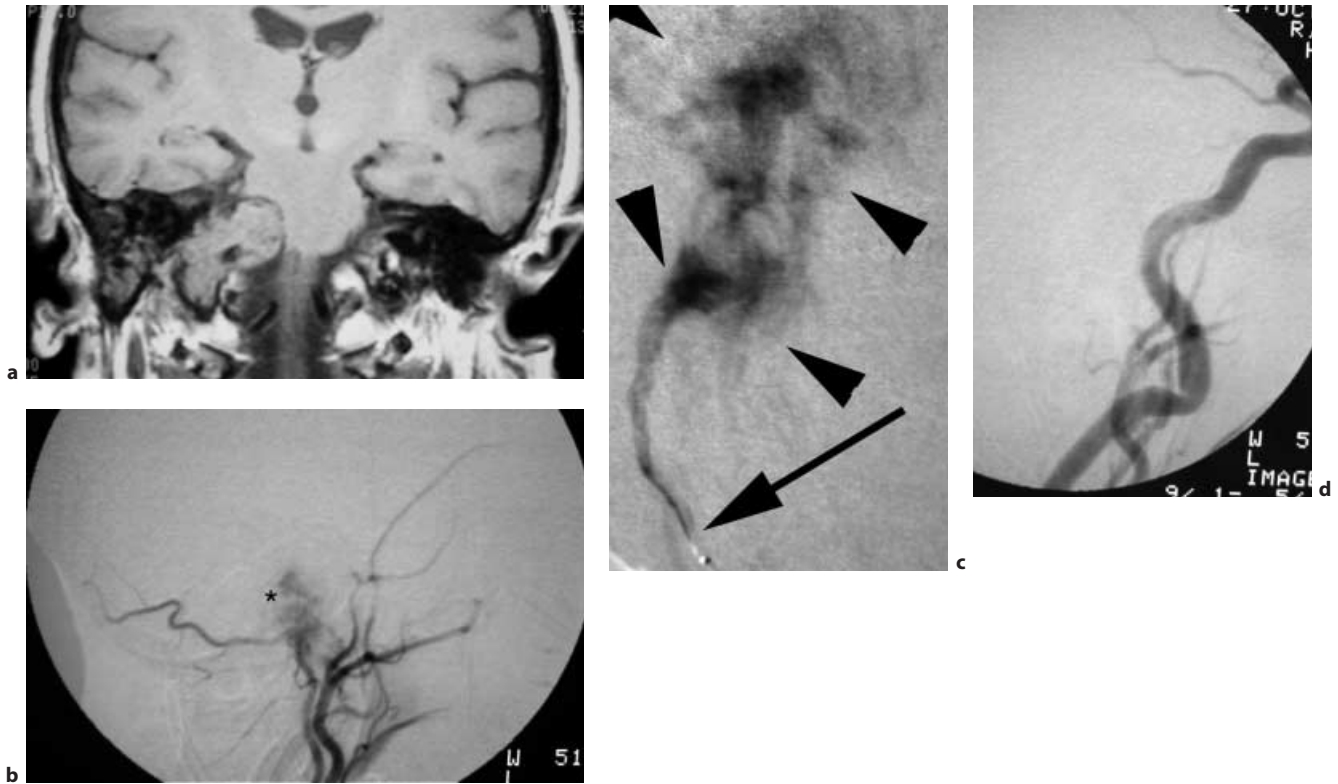


Fig. 5. **a** A 78-year-old woman presenting with pulsatile tinnitus. Coronal short TE and TR image, without intravenous contrast, showing right jugular foramen mass lesion indenting the pons, extending through the foramen into the extracranial space along the line of the jugular vein. Vivid enhancement followed intravenous contrast, and the lesion was heterogeneous on T₂-weighted images (not shown). **b** Lateral projection, common carotid injection, with tumor blush at jugular foramen and into carotid space. Selective injections confirmed supply

from transmastoid branches of the occipital artery (not shown) and **c** the ascending pharyngeal artery. The microcatheter tip (*arrow*) is placed selectively; a further angiogram was performed showing more clearly the extent of the tumor (*arrowheads*) prior to embolization with 300–500 μ m PVA. **d** Postembolization lateral view confirms good angiographic devascularization prior to surgical resection, which occurred with no morbidity and minimal blood loss

ferred agent in most cases, allowing good devascularization at the level of the tumor vascular bed. Glyceryl trinitrate (GTN) paste can assist in avoiding spasm. Selective catheter placement in the appropriate vessels – typically the middle meningeal, occipital, ascending pharyngeal arteries – is then performed with the microcatheter. Once the arteries supplying the tumor are identified, the catheter is placed distal to any dangerous anastomoses or anatomical variants and embolization with PVA particles is performed. These are usually between 150 and 600 μ m; when in a safe position beyond any potential anastomoses with the internal carotid or vertebral circulations and extensive devascularization is desired, smaller particles in a more dilute suspension are used. It is important to stop embolization well before stasis, to minimize the risk of reflux of the embolic agent and inadvertent nontarget vessel occlusion and distal infarction. If bilateral arterial embolization with small particles is performed, the distal muscular and cutaneous branches must be protected by either superselective embolization of the branch to the tumor alone, or if not possible, by occluding the distal vessel with large particles, coils, or gelfoam before embolizing with free flow-directed particles more proximally.

Complications are rare when appropriate particulate agents are used at appropriate anatomical sites, having excluded variant anatomy (e.g., ophthalmic artery arising from middle meningeal artery). They include stroke, cranial nerve deficit, and soft-tissue necrosis.

Epistaxis

INTRODUCTION

Intractable epistaxis is uncommon and when encountered (particularly if recurrent), an underlying abnormality should be suspected. Severe hypertension is most frequently encountered, but disorders of coagulation and hereditary hemorrhagic telangiectases (HHT, Osler-Weber-Rendu disease) can also present in this manner. Embolization has been applied to epistaxis for the past 30 years, and is effective in both anterior and posterior epistaxis. Merland et al. reported a 94% success rate in 54 patients treated with embolization [94]. In general, success rates of 95%–100% are achievable, with few complications (although Elahi et al. reported 96% success and 6% “major neurological complications” [95]).

DIAGNOSTIC EVALUATION

Clinical evaluation to identify anterior versus posterior and unilateral vs bilateral bleeding is extremely useful, but not always possible in the situation of severe epistaxis. Complete cerebral angiography is followed by selective injection of the distal internal maxillary artery and facial artery. Any contribution from the internal carotid artery via the ophthalmic artery is not amenable to safe embolization in most cases. It is not common to identify a bleeding point; sometimes removal of nasal packs is followed by further bleeding which can then be identified and the site embolized.

PROCEDURE

In the absence of a specific site, the sphenopalatine artery and superior labial branch of the facial artery should be embolized bilaterally. In confirmed posterior epistaxis on one side only, it may be reasonable to embolize both sphenopalatine or internal maxillary arteries with 150–300 μ m PVA, without treating the facial artery branches. It is imperative that all clinicians involved in the management of epistaxis are aware of the very high success rates in controlling bleeding using these interventional procedures. Ligation of the external carotid artery not only is less efficacious and more invasive than embolization, it may preclude or make it very difficult when (as so commonly occurs) there is recurrent bleeding. This is particularly the case in hereditary hemorrhagic telangiectasia, in which recurrent bleeding should be anticipated after all therapeutic strategies. Specialized skin grafting to the nasal mucosa or laser ablation offer more long-lasting effects in HHT, but embolization retains a role for the acute uncontrollable episode.

Dural Arteriovenous Fistulas

INTRODUCTION

The terms dural arteriovenous fistulas (DAVF) and dural arteriovenous malformation (DAVM) are often used interchangeably. As the pathology is a myriad of small fistulas, DAVF is an appropriate term and serves to clearly separate these from parenchymal arteriovenous malformations of the brain. Most are considered to be acquired, are related to major dural venous sinuses, and can both follow and precede dural sinus thrombosis. They are also reported to follow trauma, infection, recent surgery, and vascular disease. Symptoms are related to the specific site of the abnormality and can range from minor inconvenience to disabling neurological deficit. The symptoms and angiographic features will determine the need and the mode of treatment. Several large comprehensive reviews of the literature [96–98] have emphasized the importance of the pattern of venous drainage in predicting intracranial hemorrhage and several classifications have been proposed based on venous anatomy. Cognard et al. [99] classified dural AVFs into five types:

- I: Located in the main dural sinus, with antegrade flow
- II: Located in the main dural sinus, with poor antegrade flow
 - IIa: With reflux into the sinus
 - IIb: With reflux into cortical veins alone
 - IIa + b: Both of the above
- III: Direct cortical venous drainage without venous ectasia
- IV: Direct cortical venous drainage with venous ectasia
- V: Spinal venous drainage

Type I dural AVFs had a benign course; type IIa had intracranial hypertension in 20% of cases and IIb had hemorrhage in 10% of cases. Hemorrhage was present in 40% of cases of type III dural AVFs and 65% of type IV. Type V produced progressive myelopathy in 50% of cases. The implications for treatment are that types I and IIa may not need treatment or subtotal treatment may be adequate and should therefore not be treated with sinus occlusion. In types IIb and above, treatment is indicated and can include sinus occlusion to achieve complete cure.

Cavernous Dural Arteriovenous Fistulas

Carotid cavernous sinus fistulas are spontaneous or acquired communications between the carotid artery and the cavernous sinus, classified into direct and indirect, depending upon the pattern of arterial supply. Cavernous DAVFs refer to indirect arterial supply. These consist of an abnormal communication between dural branches of the internal or external carotid arteries and the cavernous sinus, functioning as arteriovenous fistulas (the connections are nearly always multiple). They therefore cause a marked increase in the pressure within the cavernous sinus and increased flow through the superior ophthalmic vein and/or the inferior petrosal sinus. The direction of venous flow determines the clinical presentation. Ocular symptoms include proptosis, chemosis, glaucoma, venous hemorrhages and pallor of the retina, and papilledema. Central symptoms are related to the recruitment of cerebral venous drainage and reflect elevated venous pressures and flow, and include cerebral dysfunction due to elevated intracranial pressure, venous hemorrhages, infarction, and bruit [100, 101].

Barrow has classified them into four groups [102]:

1. Direct communication between intracavernous ICA and cavernous sinus
2. Supply from the meningeal branches of the ICA alone
3. Supply from meningeal branches of the ECA alone
4. Mixed supply from the ICA and ECA meningeal branches

RADIOGRAPHIC EVALUATION

The diagnosis is clinical, although MRI, MRA, or CT scanning may have been performed, depending on the clinical presentation. These can demonstrate an enlarged superior ophthalmic vein (SOV), cavernous sinus, and proptosis suggesting the diagnosis. All cases require detailed angiographic assessment because MRI and MRA are inadequate to allow definitive management decisions as they cannot reliably detect high-risk features such as cerebral venous drainage and cannot exclude the diagnosis.

INDICATIONS FOR TREATMENT

Management aims to prevent visual loss, ophthalmoplegia, cerebral venous infarction, and hemorrhage, and to achieve complete closure of the abnormal connection between the arteries and veins. All patients with cerebral venous drainage and those with persistent ocular symptoms or decreased visual acuity should be treated. These lesions can spontaneously thrombose or occlude following angiography or caroticojugular compression. The hemodynamic features of each case will determine spontaneous and postangiographic occlusion rates, but figures of 20%–50% have been reported [103].

Caroticojugular compression consists of instructing the patient to compress the carotid artery and jugular vein just below the angle of the mandible with the contralateral hand for 1 min two to three times every hour when awake, which can continue for several weeks. It is important to observe the patient to recognize a transient ischemic event, a contraindication to the procedure.

PROCEDURE

For transarterial embolization alone, success up to 78% has been reported, although in most series considerably less than this was achieved. Supersselective embolization of individual feeders and embolization with 150- to 250- μ m PVA is the first choice, with low probability of cranial nerve palsy. Permanent agents and alcohol should be avoided in all but

the most refractory high-risk fistulas. The highest likelihood of success comes with combined transarterial and transvenous embolization [100].

We perform transarterial superselective embolization with polyvinyl alcohol (PVA) particles and in cases with a significant ICA supply and persistent flow, we proceed to direct transvenous coil embolization of the cavernous sinus via the inferior petrosal sinus. The typical supply is from branches of the distal internal maxillary artery, which are selected using a microcatheter coaxial system and embolized with either 150- to 300- or 300- to 600- μ m PVA particles, depending upon anticipated anastomoses. The risks of embolizing the branches from the internal carotid artery are higher and in general should be avoided in favor of combining treatment with transvenous embolization or allowing time for delayed thrombosis after the initial embolization.

The cavernous sinus is easily catheterized from a femoral vein approach by either the ipsilateral or contralateral inferior petrosal sinus in the majority of patients, allowing direct deposition of microcoils (typically fibered platinum of 2–4 mm in diameter and 10–60 mm in length). Note that even in the absence of significant opacification during angiography, the inferior petrosal sinus (IPS) can usually be found by probing with the combination of the guide catheter and microguidewire or microcatheter, which have been appropriately shaped. The anatomy of drainage of the inferior petrosal sinus varies from a classic large single venous sinus to a plexus of small channels and can join the internal jugular vein above, at, or below the skull bases. Of these patterns, there is clearly a subset in which a transvenous approach via the IPS to the cavernous sinus is precluded. This group of patients and those with persistent but slowed flow after combined embolization, usually from the multi-compartmented nature of the cavernous sinus, are instructed in the technique of caroticojugular compression. Some of these will completely occlude during follow-up; in the remaining symptomatic group, a surgical approach to the superior ophthalmic vein allows further fibered platinum coils to be placed via a microcatheter into the anterior patent part of the sinus [104, 105]. In five cases requiring this approach [105], there was universal success in accessing the residual sinus and in obliteration of the fistulas. Although it has been suggested that the primary approach could be via such a surgical approach to the SOV [106], this is unnecessary in most patients and should be reserved for failed percutaneous embolization. Postoperative management does not usually involve corticosteroids, heparin, or antiplatelet agents.

A few patients remain in whom, despite complete obliteration of the fistulas, there is paradoxical (usually transient) worsening of ocular symptoms [107], probably due to poor venous collaterals after occlusion of the cavernous sinus and the adjacent portion of the superior ophthalmic vein. This is usually self-limited.

POSTPROCEDURAL FOLLOW-UP

Close angiographic and clinical follow-up is essential, but one must be aware that the bruit can alter in pitch or volume, or even disappear in the presence of persistent and potentially high-risk fistulas. Complete obliteration and resolution of all clinical signs is the goal and in this group of patients, clinical follow-up is acceptable. Angiography should be performed in all patients not falling in the above category: within 4–6 weeks if associated with any persistent clinical features, 6 months if clinically silent, earlier if any changes to clinical state.

Transverse and Sigmoid Sinus Dural Fistulas

Transverse and sigmoid sinus fistulas typically present with pulse synchronous tinnitus and may have an audible bruit. They are the second most common sites after the cavernous sinus. Classification provides a basis for estimating prognosis and the means for treatment.

INDICATIONS AND OPTIONS FOR TREATMENT

Management includes a conservative approach of no active intervention in low-risk malformations (I and IIa) in which the patient is not distressed by the tinnitus. In symptomatic patients, carotid-jugular compression and transarterial embolization, either alone or combined with transvenous occlusion of the sinus, should be considered. The latter combination has the highest success rate (70%–90%), but should only be performed when a careful analysis of the entire intracranial venous drainage indicates an intact contralateral pathway, preferably with evidence of long-standing venous hypertension having already redirected flow to the contralateral side. Evaluation is performed by studying the venous phase of the cerebral angiograms, with carotid and vertebral venous drainage assessed. Before sacrificing a transverse and sigmoid sinus, there should be no antero-grade flow through the sinus, a contralateral transverse/sigmoid sinus of good caliber, and evidence of retrograde flow into cerebral veins away from the involved sinus. In complex high-risk fistulas, in which complete cure is mandatory but venous outflow anatomy precludes safe venous sinus sacrifice, a surgical or combined interventional and surgical procedure should be performed.

PROCEDURE

The technique of transarterial embolization has been described for cavernous DAVF, but transvenous occlusion of the larger and higher flow major dural sinuses is often more problematic. If a soft 5-F catheter can negotiate the sigmoid sinus, Gianturco coils – very thrombogenic – can be deposited as an anchoring basket, which can be subsequently filled with coils of progressively smaller diameter. More typically, a coaxial approach with a microcatheter is used, often deploying several coil types (GDC, standard platinum fibered and nonfibered microcoils) to achieve a dense coil basket. Gradual progressive packing of the coil basket with fibered, complex, and smaller coils is necessary to maximize the prospect of complete occlusion. Alternatively, liquid agents have also been used with comparable results [108]. Persistent flow, or involvement of a segment of sinus that cannot be sacrificed (e.g., torcular herophili) may be occluded by using adjunctive transarterial embolization with agents capable of permanent occlusion such as 96% alcohol or NBCA (Fig. 6). With both agents, tissue damage is possible and careful distal positioning of the catheter, provocative testing, avoidance of known branches with cranial nerve supply, and preventing reflux are crucial to minimizing the risk of the procedure. It may be more appropriate to use a combined endovascular and surgical approach in some patients.

Superior sagittal and inferior petrosal sinuses are less common sites, with similar concepts and management to the fistulas described in this section.

Anterior Cranial Fossa Dural Fistulas

These are uncommon sites of fistulas, but warrant comment as they tend to present in a uniform manner with frontal

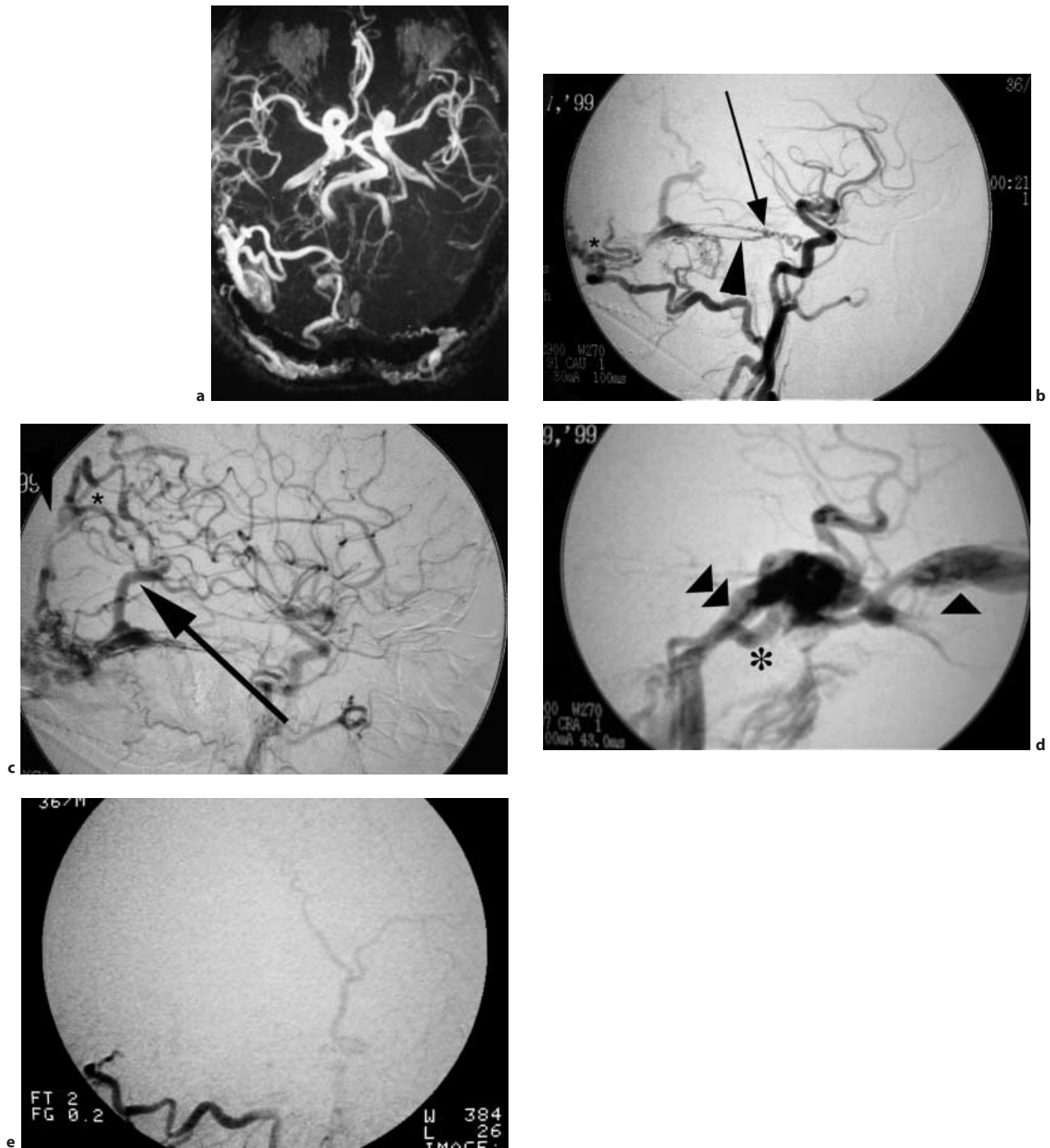


Fig. 6. **a** A 55-year-old man presenting with headache and seizure. No history of parenchymal hemorrhage. MRI congested veins over right hemisphere, occluded right transverse sinus. **b** Digital subtraction angiography (DSA) in the lateral view shows external carotid artery (ECA) supply from occipital artery (*asterisk*), middle meningeal artery (*arrowhead*) and meningohypophyseal trunk (*arrow*) from ICA. **c, d** Lateral view in the early and late venous phase from carotid injection, with retrograde opacification of cerebral veins overlying the right occipital and posterior parietal lobes (*arrow* and *asterisk*). This degree of parenchymal venous opacification constitutes a high risk of cerebral hemorrhage or infarction. The right transverse sinus is occluded and a large vein of Labbe demonstrates retrograde filling. Complete obliteration

of the DAVF with transarterial embolization alone is not possible because of the numerous small distal branches supplying the area; involvement of the torcular precludes venous occlusion as a safe option. The goal of therapy was to completely and permanently obliterate the superior sagittal sinus component (not shown, achieved with alcohol), treat as much of the right transverse sinus component with permanent agents (alcohol and polyvinyl alcohol used), and perform more limited devascularization of the torcular component as a preoperative measure. **d** Lateral view ECA injection postparticulate and distal alcohol injection, complete obliteration on the right. **e** Lateral view ICA injection with final images after skeletonization of the torcular, confirming no evidence of any arteriovenous shunting

lobe hemorrhages and an underlying high flow abnormality, usually consisting of a few well-defined fistulas from the meningeal branches of the ophthalmic artery (from the anterior ethmoidal) [96, 98, 109]. The importance lies in recognizing the pattern of hemorrhage and in stimulating the definitive diagnosis with angiography to allow subsequent management. Of 33 cases reported in the literature [100], there was a strong male predominance (87%), unlike dural fistulas at other anatomical locations. Surgical ligation can be safely performed after delineation of the site angiographically, at the same time as evacuation of the parenchymal hematoma. Endovascular embolization via the ophthalmic artery is possible, but to achieve complete permanent obliteration may be associated with higher risk than surgery, which is usually very effective and straightforward for experienced neurovascular neurosurgeons.

ARTERIOVENOUS FISTULAE AND TRAUMATIC VASCULAR LESIONS

Direct Carotidocavernous Fistula

INTRODUCTION

These are distinct from cavernous DAVFs and hence are considered separately. They can arise from rupture of an intracavernous carotid aneurysm, traumatic carotid rupture within the cavernous sinus or spontaneous rupture secondary to weakness in the carotid wall. The incidence of direct carotidocavernous fistulas has decreased since the more widespread use of seat belts. The pathophysiology is of a high-flow arteriovenous shunt from the internal carotid artery to the cavernous sinus. These fistulas are invariably symptomatic, they almost never spontaneously occlude, and usually require treatment, unlike cavernous DAVFs, which have a significant spontaneous closure rate after angiography and may have minor symptoms not requiring treatment.

DIAGNOSTIC EVALUATION

MR or CT has often been performed and the diagnosis is made by observing an enlarged superior ophthalmic vein and cavernous sinus. CT will show any associated fractures through the sphenoid sinus or adjacent skull base. Parenchymal edema or hemorrhage may be due to closed head injury, but should also prompt urgent angiography to rule out retrograde filling of cerebral veins. Full preoperative diagnostic cerebral angiography is necessary, in multiple projections, to define the site and size of the defect in the internal carotid artery and the venous drainage pattern. Injection of the vertebral artery while compressing the ipsilateral carotid, or injecting the ipsilateral carotid artery while simultaneously compressing it, are techniques that can help characterize the anatomy. If the anatomy is such that carotid occlusion is likely or possible, elective trial carotid occlusion should be performed.

PROCEDURE

Optimal management utilizes flow-directed detachable balloon embolization from a transarterial approach [110–112]. A transfemoral coaxial technique is utilized, exchanging the diagnostic catheter for a guide catheter capable of transmitting the microcatheter and attached the uninflated detachable balloon. The guide should be positioned as far distal in the internal carotid artery as can safely be obtained, a road mapping procedure is then performed, and in the lateral

projection the balloon is navigated to the site of the fistula. Balloon navigation can be achieved with a minimal number of partial inflations of the balloon. The rate of flow is generally sufficient to readily carry the partially inflated balloon into the cavernous sinus; if not, repositioning with varying degrees of inflation generally allows an appropriate position to be obtained. The balloon is positioned in the cavernous sinus via the defect in the arterial wall, test inflations are performed using contrast medium to determine the volume necessary to occlude the fistula without compromise of the internal carotid artery, and final permanent inflation is carried out with a polymerizing agent (e.g., HEMA). The use of a permanent agent is preferred over contrast, as deflation of balloons has been observed, requiring a repeat procedure (Fig. 7).

Note that when testing for the volume of liquid necessary to inflate the balloon to occlude the fistula, there is often only 0.1 ml difference between occluding the carotid artery and incomplete obliteration of the fistula. Large fistulas will need larger balloons and the use of multiple balloons may be necessary to achieve complete occlusion.

Alternative strategies are used when the usual arterial approach is not possible.

In cases previously treated by surgical internal carotid ligation, now with recurrent symptoms, direct carotid puncture can allow access and successful treatment [113]. Transvenous embolization is an alternative when the arterial route (direct puncture or transfemoral) fails. Halbach et al. reported treating 14 of 165 (8.5%) cases of direct carotid cavernous fistula via the transvenous route, 12 through the inferior petrosal sinus and one through the superior ophthalmic vein [114]. Reasons for the transvenous approach included an occluded carotid artery caused by trauma (3), failed transarterial balloon attempts (9), and a prior trapping procedure. Of the patients treated, 11 were completely cured, one showed angiographic and clinical improvement, one failed, and one death occurred due to venous diversion and pontine hemorrhage. Alternative embolic agents are more commonly used when the transvenous approach is used, but fibered and detachable coils have been used from the transarterial approach.

COMPLICATIONS

Current endovascular techniques achieve complete obliteration of the fistula in 90% of cases. Parent vessel occlusion is rare. The majority of the remaining cases are cured after repeated transarterial or transvenous approaches, with an overall success rate over 95% [112]. Stroke from parent vessel occlusion or premature balloon detachment and migration into cerebral arteries is rare (estimates are less than 1%). Cranial nerve palsy from pressure effect of the balloon on the structures of the cavernous sinus are usually transient, sixth nerve palsy being the most often encountered. Orbital congestion can paradoxically worsen, as described in "Cavernous Dural Arteriovenous Fistulas" for cavernous dural fistulas, but usually the preoperative state is so severe that there is almost always immediate and dramatic improvement.

FOLLOW-UP

Angiography is recommended in 3–6 months to assess for residual fistula, which can be asymptomatic, or the development of a false aneurysm. If the patient is asymptomatic and the false aneurysm small, treatment is not performed, but in all other cases of false aneurysm, further treatment is necessary. Although usually with a wide neck, treatment with

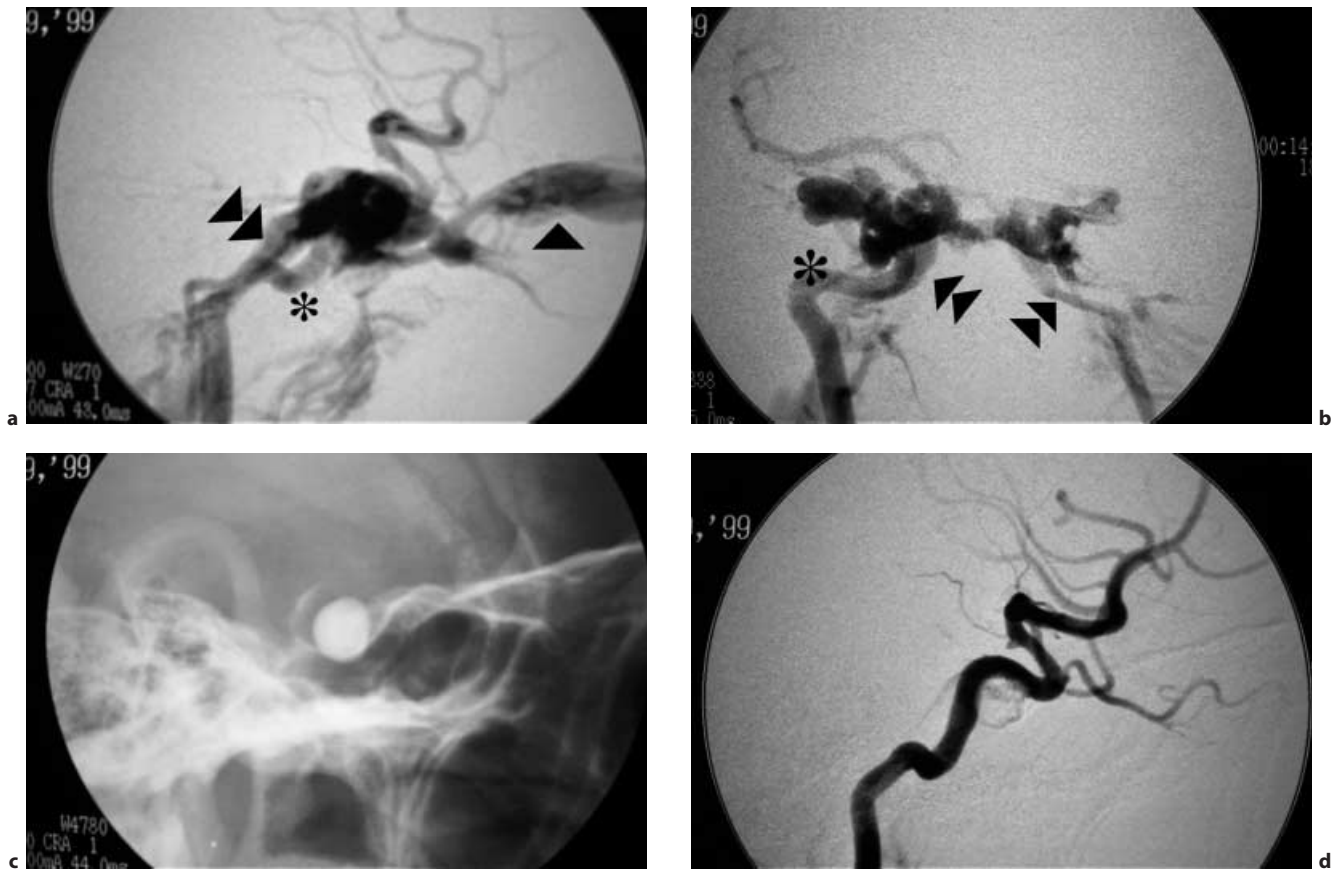


Fig. 7. **a, b** A 45-year-old woman presenting with diplopia, headache, and found to have mild proptosis, conjunctival edema, and a bruit over the orbit. Lateral and frontal projections of internal carotid artery injection (asterisk), with early opacification of an enlarged cavernous sinus draining anteriorly by a markedly enlarged superior ophthalmic vein (SOV) (arrowhead), inferiorly by inferior petrosal sinus (IPS) (double arrowhead) and clival and pharyngeal plexus. Diagnosis of high-flow direct carotidocavernous fistula (CCF), cross filling to opposite cavernous sinus. **c** Lateral projection, unsubtracted to show detachable balloon (DSB with HEMA) placed through defect in cavernous carotid wall, lying within the cavernous sinus. **d** Final postdetachment lateral projection carotid injection, no opacification of IPS or SOV, minimal contrast at base of balloon, and no compromise of the ICA lumen. Complete and immediate clinical cure

further balloons or detachable coils is preferred initially, aiming at vessel preservation.

Carotid and Vertebral Jugular Fistula

Carotid and vertebral jugular fistulas are usually posttraumatic, but spontaneous and presumed congenital fistulas are recognized. Local symptoms occur if subcutaneous, tinnitus is common, neurological symptoms are uncommon, and a bruit is audible on examination. Typically high flow and often a very short fistula connecting the artery and vein can be demonstrated by angiography. If long standing, a cirroid aneurysm can develop, with radiating draining veins of markedly increased caliber. The risk of neurological deficit is usually low. Bleeding can be massive, but when superficial can be controlled by local pressure; local tissue necrosis can follow from gross mass effect and decreased tissue perfusion. Treatment is not always required. Emergency treatment

is rare, and will usually be in the context of massive hemorrhage that is not well controlled by local pressure.

When elective treatment is indicated, the goal is obliteration of the fistula with preservation of the parent vessel. Optimal benefits are achieved using detachable balloons; occasionally coils may be necessary, but care must be taken to avoid distal migration of whichever agent is used. If coils migrate, they can usually be retrieved from the heart or proximal pulmonary arteries; migration into distal pulmonary arteries often remains asymptomatic. If the length of fistula precludes the use of coils or balloons, covered stents can be considered, usually from the arterial but occasionally also from the venous side. When all other options fail, obliteration can be achieved with sacrifice of the parent artery. In these cases detachable balloons are preferred and it is important to occlude the site of the fistula or to trap the fistula to avoid retrograde filling or collateral filling from local anastomoses.

Successful treatment of vertebral jugular fistulas is achieved in more than 90% of cases [115, 116], with less optimal results in cirroid aneurysms in which combined catheter intervention and surgical excision may be necessary [117].

ARTERIOVENOUS MALFORMATIONS

Introduction

Arteriovenous malformations are the commonest symptomatic vascular malformation of the central nervous system (80%–85%) and consist of a central nidus of abnormal vascular channels connecting enlarged feeding arteries and

TABLE 3. Grading of intracranial arteriovenous malformations. (From [121], with permission)

| Feature | Points |
|--------------------------------|--------|
| Size of AVM | |
| Small (< 3 cm) | 1 |
| Medium (3–6 cm) | 2 |
| Large (> 6 cm) | 3 |
| Eloquence of surrounding brain | |
| Noneloquent | 0 |
| Eloquent | 1 |
| Pattern of venous drainage | |
| Superficial only | 0 |
| Deep | 1 |

draining veins, without intervening normal brain tissue. Subclassification into arteriovenous, arteriovenous, and arteriovenous types has relevance to endovascular management planning [118]. They present clinically with hemorrhage, ischemia, elevated intracranial pressure, or they represent incidental findings. The angioarchitecture must be meticulously analyzed to define the risk to the patient from the malformation, the range of treatment modalities applicable, and the risk of any intervention [119, 120]. High-risk features include aneurysms of the circle of Willis, intranidal aneurysms, a dominant large feeding artery with a short course before entering the nidus, venous pouches or aneurysms, venous outflow restriction, and probably size (certainly the size increases the risk of surgical intervention) [121–123]. Spetzler and Martin proposed a grading scale, summarized in Table 3, with widespread acceptance as a good predictor of surgical risk.

Surgical excision is the definitive treatment for these lesions and the gold standard by which other techniques must be measured. Stereotactic radiosurgery is possible for lesions less than 3 cm in diameter, but the hemorrhage rate remains that of untreated lesions for 2 years. Therefore it has been suggested that radiosurgery should not be routinely used for small symptomatic AVMs [124]. Deep lesions not primarily amenable to surgical excision or radiosurgery may become treatable after partial embolization with volume reduction [125]. Residual AVM following radiation can also be treated with embolization [126]. The use of permanent agents is mandatory if no other treatment is possible, either 96% alcohol or NBCA.

Diagnostic Procedure

Noninvasive investigations include computed tomography and magnetic resonance imaging. When clinically presenting with acute headache, seizure, or focal neurological deficit, computed tomography has usually been the initial investigation. A parenchymal hematoma must be suspected of indicating an underlying AVM if in a lobar rather than typical hypertensive site (basal ganglia, thalamus, cerebellum), with enlarged vessels, or local calcification. If no hematoma is present, calcification, enlarged vessels, and serpentine enhancement can provide an indication of the diagnosis. Magnetic resonance imaging is more important than magnetic resonance angiography (MRA) and is performed in all patients with the diagnosis of AVM. High field strength units (1.5 Tesla), capable of echo planar imaging, very short echo times (TEs), and high-quality MRA are necessary. Standard imaging of the brain is followed by coronal

three-dimensional T1-weighted sequences, 2–3 mm thick, to allow identification of gyral anatomy and the relationship of the nidus to the brain parenchyma. An important distinction is between sulcal and gyral malformations; the MRI combined with subsequent angiography will allow this classification. It is important to stress that noninvasive investigations cannot exclude an AVM.

Invasive investigations involve selective and superselective DSA, performed on equipment capable of high resolution, rapid frame rates, and road mapping. Standard assessment of extracranial vertebral and common carotid arteries are performed. If there is no high-grade stenosis of the vessels, selective injection of the internal carotid arteries is performed with at least half Townes, lateral and per-orbital oblique views. The latter are particularly important in assessing the circle of Willis for aneurysms, as well as providing views of the arterial feeders, nidus, and venous outflow. Further oblique views may be necessary to show intranidal or venous aneurysms and venous stenoses. Rapid frame rates give information on flow characteristics and may reveal angioarchitecture otherwise obscured by rapid filling of the nidus and draining veins. Selective injections of the external carotid arteries will show any meningeal supply, which could cause surgical difficulty or endovascular therapy failure if unrecognized. The vertebral arteries are studied with Townes, AP, and lateral projections. Both vertebral arteries should be studied unless there is reflux to the opposite posterior inferior cerebellar artery and a hypoplastic contralateral vertebral artery. Superselective angiography refers to placing a microcatheter sequentially into all arterial pedicles and has a much higher chance of demonstrating intranidal and local aneurysms; it can show the compartmentalization of some AVMs. Our practice is to perform superselective angiography as a prelude to embolization, rather than as a separate purely diagnostic procedure. This approach avoids nontherapeutic distal microcatheter placement with the attendant small risk of vessel damage.

Findings

Access vessels are assessed for degree of tortuosity and stenotic disease which may preclude transfemoral endovascular treatment. All arterial feeders must be identified, the angioarchitecture must be identified as either en passant or end-artery supply, and the presence and likely functional consequence of occlusion of branches supplying normal parenchyma must be assessed. The nidus must be measured, compartments identified, and intranidal aneurysms shown. The venous outflow can be too deep, involving a higher risk of hemorrhage with surgical treatment or superficial veins, and may have focal stenoses. At the end of the diagnostic procedures, the AVM should be fully described, allowing an assessment of the risk of future hemorrhage and of the various treatment modalities.

Indications for Percutaneous Therapy

The decision as to which AVMs should be managed with preoperative embolization is always reached in consultation with neurosurgical colleagues planning surgery. In general, most arteriovenous malformations referred to a tertiary referral unit (large, high-flow, or anatomically complex) benefit from preoperative embolization; the occasional exceptions are anatomically accessible malformations of Spetzler grade 1 or 2, in which it is possible to proceed directly to surgery with-

out exposing the patient to additional morbidity. Our policy is to use embolization as a preoperative adjunct. High-flow arteriovenous malformations are associated with loss of autoregulation in the adjacent cerebral vasculature. Rapid occlusion of the feeders to the nidus, either by surgical or endovascular techniques, can result in abnormal increased flow to adjacent normal brain and cause cerebral edema and neurological deterioration, described as normal perfusion pressure breakthrough. By targeting high-flow dominant feeders, a staged hemodynamic alteration is possible, which may minimize this phenomenon of normal perfusion pressure breakthrough [119]. We do not commonly use embolization as the definitive treatment, although embolization alone can result in permanent obliteration in 7%–11% of cases [127]. These are also the lesions most easily managed with surgery and they should probably be managed by a single technique (surgery or embolization) to minimize complications.

Interventional Procedure

The goal of the procedure is clearly defined prior to treatment and it is most important to identify and target arterial feeders which the surgeon indicates as inaccessible or of higher risk. These may be difficult for anatomical reasons or the position of draining veins or nidus may obscure arterial pedicles that would otherwise be easily accessed. Depending upon the size and number of feeders, more than one session may be required. The ideal time between embolization and surgery has not been determined, but it would appear that it is best to perform surgery 1–3 weeks after the final embolization of a large AVM, to avoid problems from collaterals, nonsprouting angiogenesis, and early hemodynamic instability. Obliteration of the nidus must be the final result of whatever treatment strategy is undertaken, otherwise recanalization or sprouting angiogenesis can occur, with increased difficulty of subsequent treatment.

A guide catheter (5.8–6 F, hydrophilic if the anatomy is difficult) is placed as far distal in the appropriate parent vessel as practicable. This facilitates the subsequent placement of a microcatheter to a superselective site, at or in the malformation. The agent to be used and the local anatomy will determine the catheter chosen. If very distal with few feeders and high flow, then a flow-directed catheter is fast and atraumatic. An over-the-wire catheter allows more control of the vessels selected and a wider range of embolic material can be used. Newer flow-directed hydrophilic catheters are compatible with guidewires and are preferred in most cases. Note that where there is increasing tortuosity of vessels, there can be an increase in forces applied to the delicate cerebral vasculature, which is painful; hence the procedure is preferably performed under general anesthesia. The benefit of this approach outweighs the loss of routine provocative testing that can be performed when the patient is awake.

The role of superselective injections of amylobarbitone in percutaneous AVM therapy, with or without concomitant EEG monitoring, deserves mention. The principle is that if vessels supply the normally functioning cerebral cortex, their presence will be disclosed by a neurological deficit after superselective injection of amylobarbitone and embolization will be avoided at that particular site of the microcirculation, avoiding a potential permanent neurological deficit [128, 129]. Others who do not routinely perform provocative testing argue that good superselective angiography

is more important in deciding whether to embolize [123], that in the presence of a high-flow lesion, the agent injected is likely to bypass the normal lower-flow vessels (sump effect) giving a false sense of security. As the embolization proceeds, the hemodynamic status may or may not change and embolization of hitherto silent vessels and cortical territories can still occur. We do not routinely perform provocative testing, using it on specific cases to provide adjunctive information where the anatomical features offer a higher risk of eloquent brain being supplied by the vessel to the AVM.

The agents used depend upon the individual case; the most frequently used is PVA, as most cases are preoperative. The aim is to use particles that will penetrate the nidus rather than occlude the feeding artery more proximally. Starting out with smaller (150–250 μm), progressing to larger particles, allows maximal penetration of the nidus. If no change in flow is seen after one to three syringes of particles, large fistulas must be suspected and larger particles chosen (Fig. 8). Liquid coils (Boston Scientific, Target Therapeutics), and platinum microcoils (standard and detachable) allow occlusion of larger fistulas or in noneloquent sites of the parent artery when intranidal positioning is not possible or safe.

When planning definitive treatment with embolization alone or combined with radiosurgery, permanent agents are used. The cyanoacrylates allow good penetration of the nidus, the polymerizing time can be adjusted to allow appropriate deposition in even high-flow lesions, can occlude the parent vessel (and hence lead to a further decrease in perfusion pressure of the nidus), and behave in a predictable manner. The disadvantages of NBCA are a relatively long learning curve, risk of gluing the catheter in situ, venous penetration, pulmonary embolization, and embolization of adjacent normal cerebral arterial branches, potentially causing strokes. Improved outcome follows increasing experience [130]. NBCA is effectively permanent [92] even if polymerization time is adjusted using nonpermanent oily contrast media rather than glacial acetic acid. Rapid sequence angiographic runs allow assessment of polymerization time to achieve deposition within the nidus, while minimizing venous penetration or proximal arterial occlusion. Using intranidal injection and flow control, more dilute glue mixtures are typically used (1 : 2 or 1 : 3 NBCA : lipiodol). Over a longer period of time. Ethibloc (not FDA-approved, but it has been used for this application in Europe and Australia) is another agent that has been used for intermediate flow lesions in which permanent obliteration or partial embolization is the goal, with a more complete gradual filling of the nidus allowed by the slower time to solidify without risk of gluing the catheter to the vessel.

Alcohol has been used for intracranial AVM obliteration, can achieve permanent obliteration, but can cause tissue necrosis when nontarget embolization occurs [131–133]. Opacification of the alcohol with metrizamide preserves the effectiveness of the agent, while using iodinated contrast agents for opacification results in decreased endothelial damage, and consequently lower efficacy. Unfortunately, metrizamide is no longer readily available, hence monitoring of intra-arterial injections becomes problematic. Injections of unopacified alcohol should not be performed if there is any possibility of occlusion and reflux occurring during the injection.

All agents (approved for use in the United States) have a role to play and must be chosen based upon the individual clinical, radiological, and angiographic features. For a pre-

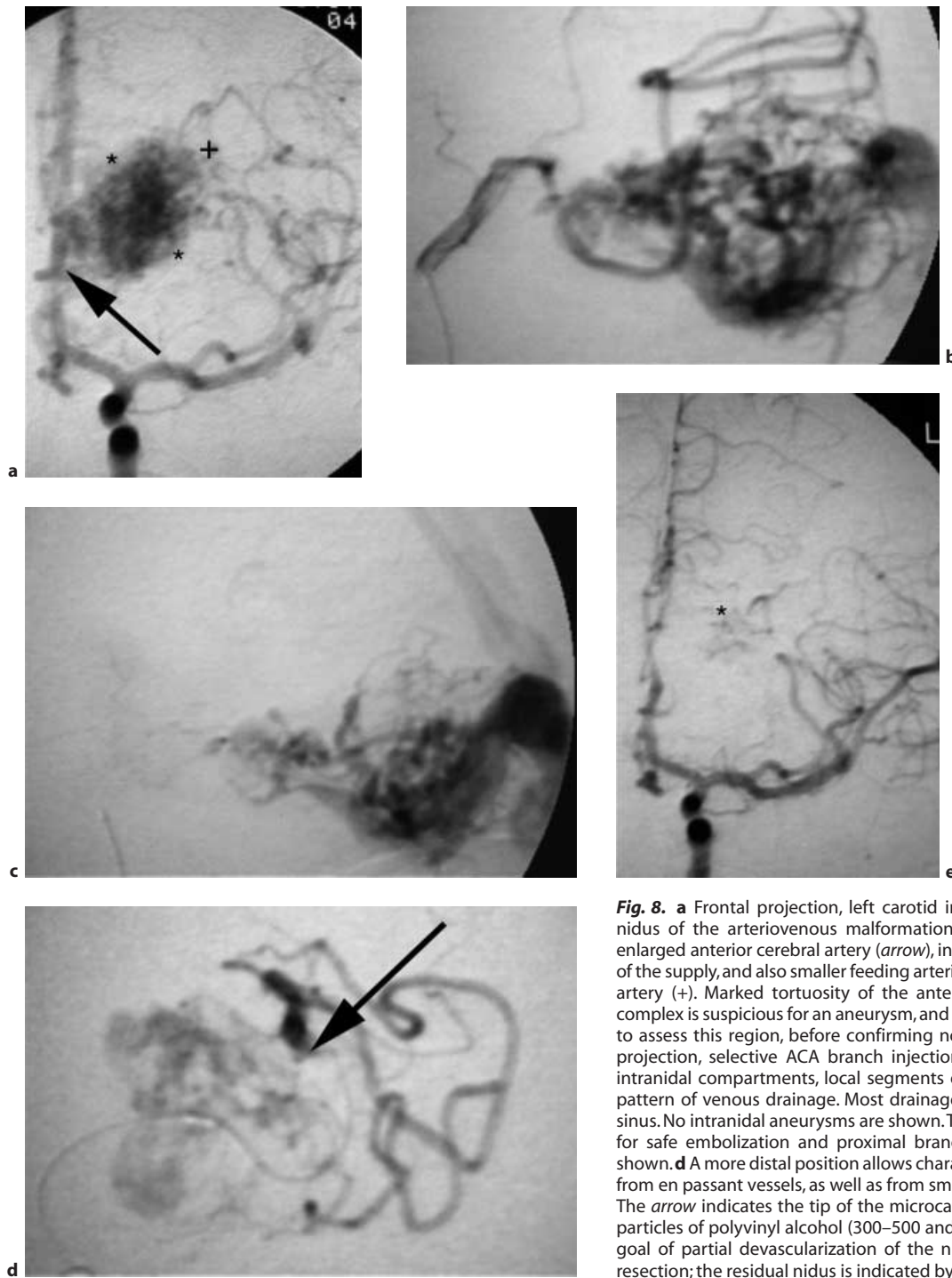


Fig. 8. **a** Frontal projection, left carotid injection, demonstrates the nidus of the arteriovenous malformations (AVM) (asterisk), a much enlarged anterior cerebral artery (arrow), indicating the source of most of the supply, and also smaller feeding arteries from the middle cerebral artery (+). Marked tortuosity of the anterior communicating artery complex is suspicious for an aneurysm, and further views were required to assess this region, before confirming normal anatomy. **b, c** Lateral projection, selective ACA branch injection, allowing analysis of the intranidal compartments, local segments of artery and vein, and the pattern of venous drainage. Most drainage is to the superior sagittal sinus. No intranidal aneurysms are shown. This position is too proximal for safe embolization and proximal branches to normal cortex are shown. **d** A more distal position allows characterization of the supply as from en passant vessels, as well as from small terminal distal branches. The arrow indicates the tip of the microcatheter. **e** Embolization with particles of polyvinyl alcohol (300–500 and 500–700 μ m) achieves the goal of partial devascularization of the nidus prior to microsurgical resection; the residual nidus is indicated by an asterisk

operative case, we typically perform embolization with PVA after selective placement of a catheter as close as practicable to the nidus, beyond normal branches. If there are high-flow fistulas, these are controlled with either standard platinum or “liquid” coils, followed by PVA to close the remaining nidus. If coils cannot be delivered, and in all cases in which the embolization is adjunctive to radiosurgery or as sole treatment, permanent agents are used. Alcohol is preferred where safe catheter placement can allow slow embolization of all of the nidus or compartment; NBCA is useful when there are higher flow fistulas to control.

Results

For immediate preoperative embolization, using coils or other agents and only occluding the feeding artery while sparing the nidus or using nonpermanent agents may be sufficient. The risks of combined embolization and surgery are probably lower than those of either alone. In a few cases surgery or embolization alone carries an unequivocally lower risk and combined therapy is not justifiable [134].

There is good indication of the usefulness of preoperative embolization from multiple sources [119, 135]. Additional

evidence is also available, indicating that endovascular preoperative embolization is cost effective [136] and makes lesions previously considered to be at unacceptable risk of neurological deficit amenable to surgery or radiosurgery following superselective embolization. This reflects advances in microsurgical and neurointerventional techniques. Combined radiosurgery and embolization can achieve good outcomes, even in larger AVMs [137], although as size increases so do complications of rehemorrhage or radionecrosis. A quantitative measure of benefit is difficult to obtain in AVMs, with multiple features contributing to surgical and endovascular difficulty, only partially incorporated in grading systems such as the Spetzler system. Although markers such as blood loss, duration of surgery, and neurological outcome have been assessed, these attempt to compare selected patients without randomization and over a time when management techniques have been rapidly changing. All patients benefit from being considered for adjunctive preoperative embolization; in most it is feasible and appropriate and provides a safer surgical environment with an overall decrease in the morbidity and mortality of managing complex cerebral AVMs.

Complications

Procedural complications have been shown to relate to the experience of the operator and have also decreased as newer catheters and agents have become available. Published figures from experienced operators, using modern equipment and materials report 1.6% mortality, 1.4% severe deficits, 5.6% mild deficits, and 10.9% transient deficits [119]. The complications from combined surgical and endovascular treatment are probably less than those of each if used alone in larger malformations, but in smaller superficial lesions, this may not be the case [134, 138]. Each case requires an assessment of these factors to decide on single, combined, or staged management.

Procedural complications include distal arterial embolization and stroke (embolic agent or thromboembolism related to catheter system), pulmonary embolization (pulmonary hypertension), arterial dissection, perforation, catheter glued to vessel, hemorrhage (venous penetration and occlusion, perforation), and normal perfusion pressure breakthrough. Experience leading to optimal selection and use of the embolic agents and the delivery system will decrease these complications. Embolization with thrombus can be managed with judicious use of local thrombolytics and heparinization; abciximab is not usually used in the presence of an untreated or partially treated AVM, especially in the setting of recent SAH. Embolization with embolic agents cannot usually be remedied and general supportive measures and management of cerebral infarction should be instituted (oxygen, antiplatelet agents, neuroprotective drugs). If perforation occurs within the nidus or adjacent feeding vessel, the catheter or wire should *not* be removed. Halbach detailed management of fifteen vascular perforations occurring during 1200 endovascular procedures [139]. Treatment of these perforations included immediate reversal of anticoagulation, placement of coils across the perforation site, temporary inflation of a balloon across the perforation, and the institution of medical therapy such as anticonvulsant administration. Recognition of the perforation by the catheter tip is also critical since the catheter should be left in place for placement of a coil across the perforation site as the tip is withdrawn into the arterial lumen. Other options

include taking the patient with the catheter tip still across the perforation site immediately to the operating room for direct visualization of the perforation site with definitive repair or excision of the involved artery and AVM.

Normal perfusion pressure breakthrough can cause deterioration either immediately or soon after surgery or embolization [140, 141]. Staged embolization can avoid or minimize this cause of posttreatment neurological deterioration [140].

Postprocedural complications occur in the peri-interventional period. Probably the most disturbing problem relates to delayed thrombosis of the draining veins of the AVM. It may be difficult to diagnose the underlying cause of neurological deterioration secondary to thrombosis of veins, i.e., mass effect vs venous overload with resultant acute venous hypertension. Wilson and Hieshima suspect that an early neurological deterioration (within the first 24 h after embolization or postsurgical resection of the larger high-flow AVMs), is probably the result of differing degrees of venous outflow restriction, leading to venous overload and associated cerebral edema [142]. Late deterioration is probably secondary to ongoing delayed thrombosis of critically draining veins, and may be minimized by optimal volume expansion avoiding hemoconcentration, and pre-embolization administration of corticosteroids (e.g., 8 mg of Decadron, continued with 4-mg doses every 6 h with a steroid taper over a period of 1 week). This approach applies primarily to very large and complicated AVMs in which such mass effect or propagating venous thrombosis may be anticipated.

Conclusion

A team comprising specialists from multiple disciplines (including neurointerventionists, neurosurgeons, neurovascular neurologists and intensivists, and radiotherapists) should discuss all cerebral parenchymal AVMs in which treatment is indicated. Treatment modalities available are often combined and must be tailored to the individual patient. Embolization is uncommonly the definitive sole modality, but is of benefit in most complex AVMs undergoing radiosurgery or microsurgical resection. Simple, superficial, and small AVMs may not benefit from combining embolization with other modalities.

HEAD AND NECK VASCULAR MALFORMATIONS

Classification schemes vary enormously in peripheral vascular malformations: descriptive, embryological, pathophysiological, and angiographic flow patterns have all formed the basis for separating these lesions. Important to management is the ability to predict natural history, as treatment is often difficult and associated with some risk. This needs to be communicated with the patient before embarking on prolonged, multiple-stage therapies, inappropriate for what may constitute a mild cosmetic problem, but necessary where progression to a crippling, life-threatening deformity is likely. Clear treatment goals are necessary to guide the choice of direct injection of sclerosant, transarterial embolization, and combined surgical and endovascular treatment. Discussion with a vascular malformation team is to be recommended in these difficult cases.

Diagnostic Evaluation

Careful clinical evaluation is essential, including palpation (thrill, compressibility, rate of refilling), auscultation (bruit, change with local arterial compression, transmission), and assessment of local tissues for ischemic or hemorrhagic changes. All cases should have selective cerebral DSA as described, with selective vertebral and carotid/external carotid injections and views of the target site. In addition, injection of the subclavian artery and selective injections of the major branches of the subclavian artery is necessary. Direct puncture with a 19- to 21-gauge needle can allow injection of standard nonionic contrast, showing the compartmentalization and the venous drainage. When large or anatomically complex, and in all low and no-flow lesions, MRI and MRA will give valuable information on the anatomical extent of the malformation. Dynamic contrast injection should improve the delineation of these lesions.

Procedure

Practical separation into high-flow, low-flow, and no-flow lesions facilitates a discussion of percutaneous therapy. Most require general anesthetic, as alcohol is particularly painful with direct injection and can cause marked edema. Venous malformations (low- or no-flow lesions) are angiographically occult, compressible, and contain vascular spaces coalescing to draining veins. If the draining veins are easily compressible, the sclerosant can be injected after compressing them for the duration of the injection. If drainage is to deep areas, there may be edema in the pharynx or larynx if sclerosants are injected, and hence these agents must be used with caution or other techniques tried. Alcohol (96%) is our preferred agent for these lesions. Cure is expected in a majority of cases – more than one treatment session may be necessary to achieve this.

Arteriovenous malformations with intermediate or high flow are managed primarily with transarterial embolization: PVA if preoperative, alcohol (96%) if definitive. Direct puncture may be tried if portions of a nidus remain that cannot be reached by the transarterial route or where the transarterial branches are off critical vessels. NBCA or alcohol are possible. It is possible to combine this with surgical ligation of high-risk anatomical pathways from external to intracranial cerebral arteries (e.g., ligation of the ethmoidal arteries in nasal vascular malformations). If penetration of the nidus with alcohol can be achieved, there is a high expectation of marked devascularization. Be aware that complete cure is elusive in the often complex and extensive cases seen and that a preoperative embolization strategy may be more appropriate than multiple embolization attempts aiming at cure.

Complications

Ischemic complications are possible, both to the nidus and overlying skin (target tissue ischemia) or to adjacent tissues (non-target ischemic complications). Ischemia to the region embolized can result in tissue loss which can be predicted, explained, and planned as part of overall treatment, i.e., combined with surgical reconstructive techniques which otherwise may not have been possible, or as sole technique associated with high recurrence rates. Failure of complete embolization can result in accelerated growth of the residual malformation, which has been recognized after either embolization or surgery.

In some cases, this can convert a relatively stable malformation with minimal clinical deficit into a lesion with marked local aggressive behavior.

Non-target embolization can occur with intranidal injection, but is more common with transarterial embolization. Clearly dependent upon the site of the malformation and the vascular territory being injected, the complications can include cranial nerve palsy, ischemic stroke, orbital infarction including retinal artery ischemia and ocular loss, and extensive scalp and/or periosteal necrosis.

VEIN OF GALEN MALFORMATIONS

Introduction

Vein of Galen aneurysmal malformations (VGAMs) are rare congenital abnormalities that can cause severe morbidity and mortality, particularly in neonates but also in infants and older children [143–150]. Neonatal presentation is usually with high-output cardiac failure, most often fatal despite optimal medical management [145, 146, 151]. Surgery offers little improvement with fatal outcomes in 80%–100% of cases [145, 146, 150, 152]. Markedly improved results have been seen with endovascular management over recent years in infants and children, but mortality and morbidity remain high in the neonatal group, with mortality ranging from 23% to 75% and morbidity from 21% to 88% in larger series [148, 150, 153–155]. Presentation in older children and adults is with hydrocephalus, failure to meet normal milestones of development, and less commonly signs of cardiac failure.

Several proposed classification systems have been used in describing malformations of the vein of Galen [118, 156–158]. Lasjaunias and colleagues have importantly separated vein of Galen malformations from vein of Galen aneurysmal dilatations; the latter have a parenchymal arteriovenous malformation that drains via the vein of Galen and more commonly present with intracranial hemorrhage [156]. Postulated causes of cerebral damage are reduced cerebral perfusion secondary to venous hypertension, abnormalities of cerebrospinal fluid reabsorption leading to hydrocephalus and elevated intracranial pressure, and brain herniations [148, 159, 160]. Antenatal diagnosis has been associated with improved outcome of survivors [161].

Cardiac manifestations are variable, ranging from mild cardiomegaly to gross multisystem organ failure. Failure is rarely present at birth and usually progresses over the first few days of life. Associated cardiac anomalies are reported and should be excluded by cardiac ultrasound.

Diagnostic Evaluation

DIAGNOSIS AND CLASSIFICATION

Antenatal ultrasound is increasingly diagnosing these rare malformations, especially with the more widespread availability to perform color Doppler imaging. When a neonate presents with undiagnosed cardiac failure, VGAM must be thought of in diagnosis. Auscultation of the head will reveal a bruit and bedside transcranial ultrasound will identify the malformation, give some indication of flow velocities, the size of the outflow veins, and any associated hydrocephalus. If interventional embolization treatment is necessary in the neonatal period, MRI/MRA facilitates safe immediate intervention, as it allows a minimum number of diagnostic angiographic runs in this situation where total contrast

volume may be limited to 10–12 ml [162]. The number, size, site, and tortuosity of arterial feeders can be shown, as can the anatomy of the malformation itself and the draining veins and dural sinuses. Typically, the malformation is 1–4 cm in diameter with a venous outflow of 0.5–2 cm. MRI should include a long echo time and long repetition time imaging in the coronal and axial planes, a short echo time and a short repetition time in the sagittal and axial planes, and three-dimensional time-of-flight MRA through intracranial volume, available on a workstation for review in multiple projections. Contrast administration is not necessary.

In older children, MRI and MRA should be performed instead of ultrasound; CT may be more readily available for follow-up.

ASSESSMENT OF PARENCHYMAL BRAIN DAMAGE AND CEREBROSPINAL FLUID

Assessment of parenchymal brain damage and cerebrospinal fluid is best achieved by MRI as described in the preceding section, although more limited information is available from CT or transcranial ultrasound.

In neonates in whom transvenous embolization is the necessary treatment, angiography is not performed. Transvenous catheterization is followed by injection into the VGAM to outline the size and any stenoses of the venous outflow. In all other cases, selective cerebral angiography is performed with standard views, as described in “Diagnostic Evaluation” under the section “Transluminal Angioplasty and Stenting for Intracranial Cerebral Vascular Disease.” Often the entire procedure is performed using the microcatheter through a sheath without a guide catheter; this is adequate for diagnostic information. In general, the left vertebral artery is first injected in the frontal projection, as the largest feeding arteries usually arise from the vertebrobasilar circulation (posterolateral or posteromedial choroidal arteries).

Indications for Treatment

We believe that patients with a mild increase in CSF spaces and high-output cardiac failure that cannot be controlled with pharmacological means are candidates for treatment. When treatment is indicated, the timing of the first embolization procedure depends on the clinical presentation and response to medical management. If well controlled medically, treatment should be postponed until approximately 6 months of age. Poor control of cardiac failure is an indication to treat. Shunt placement should be avoided where possible, as reports have suggested worse outcome in patients with shunts [163], supported by hemodynamic theories predicting worsened cerebral perfusion in the presence of a shunt and patent VGAM.

Severe parenchymal damage and hydrocephalus (including extensive calcification on CT) are commonly associated with a very poor clinical state and are contraindications to surgical or endovascular treatment. Gross multiorgan systemic failure also constitutes a contraindication to treat.

Procedure

NEONATE

Neurointerventionists, neurosurgeons, neonatal intensivists, and anesthesiologists should all be involved in a team effort to plan intervention once medical management has proved ineffective. As much information as possible should be obtained from reviewing noninvasive imaging. When there is a limited number of arterial feeders, a primary transarterial

approach is performed, whereas a large number of transdiencephalic feeding arteries in an unstable patient is best approached from a transvenous approach.

A percutaneous transfemoral transarterial approach can use a microcatheter directly through the sheath, or through a 4-F diagnostic catheter used as a guiding catheter. The larger vessels demonstrated on prior imaging are targeted, the microcatheter positioned either within the VGAM (from which site coils can be deposited within the malformation, across the fistula into the terminal feeding artery) or as close as possible to the fistula. Complete closure of the fistula is the ultimate goal, using NBCA, coils, or PVA particles, alone or in combination. It is possible to deploy coils on the venous side from the arterial approach. As many pedicles as possible are embolized, within the contrast volume limitations of treating a neonate. The sheath may be left in place to facilitate a second stage as typically multiple stages are required in the majority of neonatal treatments.

If the patient is very unstable or arterial access not possible, a transvenous approach is indicated. This is preferable and usually possible from the femoral vein; transthoracic venous embolization is an alternative if the transvenous approach fails, but is associated with greater morbidity. Transthoracic intervention can be performed by ultrasound-guided percutaneous puncture [162], but more typically access is obtained by neurosurgical techniques. The intracranial veins are fragile, and a microcatheter is preferred for coil embolization, often stabilized with a guide catheter. Large platinum coils are first deployed, with deposition of more coils of complex shape and decreasing size to achieve dense packing within the VGAM. The goal is to partially occlude venous flow, stopping when there is a sustained increase in blood pressure and lessening of cardiac failure. Further staged treatment may be required if cardiac failure worsens and subsequent embolizations may be either arterial or repeat transvenous.

INFANT AND LATER PRESENTATIONS

Patients of the age of one and older are, compared to neonates, hemodynamically more stable, can tolerate increased volumes of contrast, and femoral access is easier. Transarterial approaches are used in most cases, using coaxial techniques, and NBCA is the preferred agent for permanent occlusion. One to three procedures are typically required, fewer in mural (more common at this age) than choroidal VGAMs. The goal of therapy is complete obliteration of abnormal shunting, although it may be reasonable to accept less than complete anatomical obliteration where numerous small perforating arteries contribute persistent flow, if development is normal and there is no imaging evidence of cerebral ischemia or CSF flow abnormality.

Conclusion

Choroidal type VGAMs classically present in the first few days of life, usually with cardiac failure. MRI and MRA are important in selecting patients and planning and timing therapy. Treatment should be deferred until infancy if cardiac failure can be controlled and is then optimally achieved with transarterial embolization with NBCA alone or with coils. If control of cardiac failure is not possible with medical therapy a variety of embolization techniques and agents can be used to allow survival; with appropriate selection of patients, good clinical outcomes can be achieved. Results from different series cannot be easily compared because of the manner in which the results are presented, the selection of

patients (referral patterns, excluding patients with higher risk features from treatment), length of follow-up, and the means of assessing neurological outcome. We would not deny treatment to neonates with difficult-to-control cardiac failure, as long as there was no evidence of gross cerebral parenchymal damage or severe multiorgan failure.

VGAMs presenting in later infancy and childhood are more likely to be of the mural type, should be treated by a transarterial approach in most cases, with much better prognosis for these patients than those presenting as neonates.

SPINAL VASCULAR LESIONS

Introduction

Vascular tumors of the spinal cord or vertebral column, spinal dural arteriovenous fistulas, and other vascular malformations of the cord comprise most indications for interventional neuroradiological techniques in the spine. The preoperative embolization of highly vascular primary or secondary tumors of the vertebral column facilitates excision and minimizes blood loss, indicates whether the artery of Adamkiewicz arises locally, and in the absence of such a local major radiculomedullary branch is performed in a straightforward manner using a coaxial system and PVA. Spinal hemangioblastomas are hypervascular tumors, in whom an enlarged artery of supply is usually found and often suitable to preoperative embolization.

Vascular malformations of the spine comprise spinal cord vascular malformations (juvenile and glomus types), perimedullary fistulas, cavernous malformations, spinal dural arteriovenous fistulas, and metameric arteriovenous malformations [164, 165]. The pathophysiology and clinical presentations have been presented in Chap. 75.

Spinal Dural Arteriovenous Fistula

The commonest symptomatic vascular malformations of the spine are spinal dural arteriovenous fistulas (SDAVFs), usually presenting with a progressive myelopathy in elderly male patients.

DIAGNOSTIC EVALUATION

MRI is the preferred modality for first investigation and is positive in most cases of SDAVF presenting with significant symptoms. Intramedullary increased signal intensity on T2-weighted images is present in most cases; enlarged vessels over the cord are seen in fewer patients, but when present allow a specific diagnosis. They are usually more prominent over the dorsum of the cord and when they are low thoracic or conus. The site of the fistula cannot usually be determined. If contrast is administered, intramedullary enhancement can be seen in significant numbers of patients, and is usually of mild degree. Myelography can make the diagnosis on the basis of coned supine AP views showing enlarged vessels, but is invasive and should no longer be performed for this indication unless there is a contraindication to MRI such as a pacemaker or a ferromagnetic aneurysm clip.

Angiography is usually performed after a positive MRI, as the yield of angiography is very low in the presence of a normal MRI. In some instances of high clinical suspicion, angiography is necessary to exclude the diagnosis even when other tests are normal. Full spinal angiography is re-

quired for their diagnosis, consisting of injections of all lumbar, intercostal, internal iliac, subclavian, vertebral, costocervical, and thyrocervical arteries using hand injections of contrast and a 5- or 5.5-F curved spinal catheter.

Angiography reveals fistulas in relation to the dural sleeve of an exiting thoracic or lumbar nerve. Supply is usually unilateral, although supply from adjacent levels to the fistulas is seen. The tortuous vessels over the dorsum of the cord are the perimedullary veins, carrying arterialized blood away from the fistula. Identification of a major radiculomedullary artery arising from the same artery as the fistula should constitute a contraindication to endovascular treatment.

INDICATIONS FOR TREATMENT

Patients presenting with myelopathy should be treated as soon as possible for a maximum chance of functional recovery. Both surgical treatment (ligation and ablation) and endovascular treatment can complete cure. Surgery is equally effective when the shunt is excised and the optimum management should take into account the availability of skilled surgeons or neurointerventionists and the general medical fitness of the patient. We prefer endovascular treatment, particularly in the many elderly patients with other comorbidities. Even when diagnosis has been delayed, treatment should be performed to decrease the chance of further deterioration of neurological function, although the chance of recovery may be limited.

PROCEDURE

Cure can be achieved by embolization of the fistula with a permanent agent. A hydrophilic microcatheter is positioned at the site of the fistula, the radiculomedullary supply to the cord having been previously identified. NBCA is mixed with lipiodol, depending on flow rates and catheter position, and injected to occlude the fistula alone. The site of the fistula must be occluded to avoid the development of collateral flow and persistent venous hypertension and potential clinical deterioration. This management is definitive and permanent, is associated with few complications, and avoids surgical and anesthetic-related morbidity, an important factor in these elderly patients. The duration of anticoagulation is controversial: anticoagulation may prevent delayed progressive thrombosis of the perimedullary veins, which can cause worsening of a neurological deficit and should probably be continued for 24–48 h.

Follow-up DSA is recommended at 3–6 months to confirm complete cure.

Spinal Cord Arteriovenous Malformations

Spinal cord arteriovenous malformations (SCAVMs) are more common in younger patients, tend to present with SAH or hematomyelia, and do not have the male preponderance seen with SDAVFs. Integration of clinical features, detailed MR imaging, and total spinal angiography allow estimating the risk from natural history and endovascular and surgical treatment. Once symptomatic, there is a high likelihood of neurological decline or death over subsequent years; hence most cases should be considered for treatment.

DIAGNOSTIC EVALUATION

MRI will show an intramedullary or surface nidus of hypointense vascular spaces, enlarged overlying vessels, and evidence of any current or remote hemorrhage. Hemosiderosis of the cord or posterior fossa structures can be seen if there

has been repeated subarachnoid hemorrhage and is shown as hypointense staining of the surface and is maximal on gradient echo or echo planar heavily T2-weighted images. The mass effect is usually minimal, there is no enhancement.

INDICATION FOR PERCUTANEOUS THERAPY

If surgically accessible, preoperative embolization can facilitate the operation and PVA is appropriate with a short interval between embolization and surgery. If inoperable or surgery presents high risks, partial embolization with PVA may allow palliation of symptoms from the mass effect of a varix or obliteration of an aneurysm may protect from subsequent hemorrhage.

PROCEDURE

If the major radiculomedullary artery arises remote from the lesion and a superselective injection does not reflux to the anterior spinal axis, permanent obliteration with NBCA can be the goal of therapy. More typically, we perform preoperative embolization. Using a guide catheter, a hydrophilic microcatheter is placed in or as close as possible to the nidus. Provocative testing is with cardiac lidocaine, which can be combined with electrophysiological testing. As in all vascular malformation embolization, to achieve cure, the NBCA must penetrate the nidus, hence the site of injection, rate, volume, flow, and delay of polymerization all interact.

EMBOLIZATION OF INTRACRANIAL ANEURYSMS

Introduction

The frequency of detection of intracranial aneurysms ranges from 0.2% to 9.9%, with a mean of 5%. Angiographic series detect aneurysms in 0.65%–1% of the population, and are likely to be larger and more significant than smaller aneurysms detected in selected postmortem series. The majority of intracranial aneurysms are berry or saccular aneurysms located in the anterior circulation (85%), arise at branch points of the circle of Willis, and are multiple in 20%–30% of cases. Subarachnoid hemorrhage (SAH) is a major clinical complication, with an annual incidence of 1/10,000, higher than many other neurological disorders, including brain tumors and multiple sclerosis. The ratio of females to males is approximately 2 : 1 affected, with the difference being most pronounced in the postmenopausal age group. Of patients suffering aneurysmal subarachnoid hemorrhage, 50%–60% will die, 12% before reaching hospital. Survivors have a 35%–50% likelihood of long-term disability, and half of patients assessed as having a good outcome are unable to return to previous employment and suffer from neuropsychological and cognitive impairment. The outcome is primarily determined by the extent of the initial hemorrhage, the grade at presentation, and complications, including repeat hemorrhage, cerebral vasospasm, and hydrocephalus [166–169]. A detailed discussion of epidemiology, pathogenesis, familial syndromes, presentation, and diagnosis is found in Chap. 70.

Endovascular Management – Evolving Technique

Neurointerventional radiologists have been involved in the management of aneurysms for many years. There remain surgically difficult aneurysms in which endovascular parent

artery vessel occlusion, using detachable balloons, can be safe, convenient, and effective, with an acceptable morbidity and mortality rate compared to surgical clamping, ligation, and trapping [88, 170–176]. Packing the lumen of the aneurysm with balloons, standard coils, and even liquid embolic agents can achieve obliteration, but permanent obliteration rates were low and embolic complications high [88, 173, 177–184]. All of these techniques were reserved for high-risk or inoperable aneurysms. Guglielmi detachable coils (GDC) [185–188] are the current agent of choice for endovascular occlusion of aneurysms. They are soft platinum coils placed through a microcatheter into the aneurysm lumen, which remain attached to a wire until satisfactory positioning is obtained. This allows repositioning of the coil if prolapse occurs into the parent vessel or removal and replacement with new coil if of incorrect size. A DC current causes electrolysis of the junction of stainless steel wire and platinum coil, the wire is withdrawn, and the coil remains within the aneurysm. Further coils are placed sequentially through the catheter until dense packing is achieved. There is no craniotomy, no manipulation of the brain to access the aneurysm, and multiple aneurysms can be treated from the same approach.

In the initial FDA-sponsored trial, all patients were deemed to be inoperable, had failed surgery, or were of unusually high risk. Acceptable outcomes were achieved, prompting application of the technique to a wider population [189]. The technique can be applied in high-grade post-SAH patients with little increased morbidity associated with the procedure and vasospasm can be treated with angioplasty or papaverine injection at the same procedure [169]. A prospective trial comparing surgical and endovascular management of ruptured aneurysms is in progress, initiated in the United Kingdom, with results expected in the next few years.

Major challenges in the more widespread application of GDC to aneurysm treatment are incomplete packing and aneurysm regrowth, aneurysm perforation, and thromboembolic complications. Aneurysm perforation is now less likely to produce clinical sequelae, and is probably no more common than intraoperative rupture. More aggressive anticoagulation strategies have minimized thromboembolic complications. Incomplete packing and aneurysm regrowth are the most important potential limitations of the technique, if recurrent hemorrhage rates are similar to those from aneurysm remnants after surgical clipping. The best results are in small aneurysms with small necks [189, 190]. Careful case selection based on aneurysm geometry and new technological developments (intracranial stents, the balloon remodeling technique, softer and smaller coils, and two-dimensional coils) are increasing the number of aneurysms potentially treatable by GDC. Estimates of the number of aneurysms that will be treated by GDC vary widely, from none to 90%, some centers currently treating 80% of aneurysms with coils [190, 191].

Which Aneurysms to Treat by Endovascular Techniques

Key issues that remain unresolved are whether to treat unruptured aneurysms and which aneurysms should be treated by endovascular techniques. Treatment of ruptured aneurysms is associated with much greater morbidity and mortality than that associated with treating unruptured aneurysms. Once an aneurysm is identified, elective surgical

or endovascular treatment before rupture is associated with much less risk to the patient than treatment after subarachnoid hemorrhage. There is therefore an argument to treat all identified aneurysms and to screen high risk populations to identify and then treat aneurysms to decrease the morbidity and mortality of subarachnoid hemorrhage in the population. Opposing views refer to a lower rate of rupture of aneurysms than previously accepted, higher than suspected risk of open surgical procedures, combining to produce more morbidity from treatment than from the natural history of the aneurysm [169]. Lower spontaneous rupture rates have been identified in anterior circulation aneurysms (other than the posterior communicating artery) of less than 10 mm in diameter in asymptomatic patients. Our approach is to treat all aneurysms in patients in whom there is a past history of subarachnoid hemorrhage, all posterior circulation and posterior communicating artery aneurysms, and most aneurysms of the anterior circulation. Exceptions are asymptomatic patients with aneurysms of the anterior circulation that are extradural and less than 5 mm in diameter.

Assuming treatment of the aneurysm is necessary, what factors should decide whether to use endovascular or surgical techniques? Morbidity and mortality associated with surgical treatment of unruptured intracranial aneurysms varies with operator and institutional experience, site, and size of aneurysm [169]. The morbidity and mortality associated with the surgical treatment of unruptured aneurysms remains significant, with reported mortality of 0%–8% and morbidity of 0%–30% [192–194]. If cognitive effects are also assessed, rates of postsurgical combined morbidity and mortality in unruptured aneurysms of up to 16% were reported in one prospective study [195]. Johnston et al. reported a decreased morbidity and mortality of endovascular versus surgical management of unruptured intracranial aneurysms [196]. Endovascular management of unruptured aneurysms will allow overall improved outcome if subsequent aneurysm rupture is as low as seen postsurgical clipping.

The obliteration of the ruptured aneurysm is an important component of management of the patient with SAH. Microvascular surgical clipping of the aneurysm is the gold standard against which other techniques must be measured. In particular, the long-term clinical efficacy of clipping is well documented, with low rates of subsequent SAH or aneurysm regrowth. Late angiographic follow-up of surgically treated aneurysms was presented by David et al. [197], demonstrating only two recurrent aneurysms in 135 cases with no residual aneurysm after clipping. Of 12 cases with known residual aneurysm rests, most appeared stable, but a subset with broad necks grew, with higher risks of SAH.

Surgery is associated with much higher morbidity and mortality in patients with ruptured rather than unruptured aneurysms. The outcome after SAH may be more determined by the initial hemorrhage and subsequent hydrocephalus or vasospasm than the technique used to manage the aneurysm. However, the endovascular technique can be applied in high-grade post-SAH patients with little increased morbidity associated with the procedure and vasospasm can be treated with angioplasty or papaverine injection at the same procedure. While there may be improved outcomes with endovascular management of ruptured aneurysms, this may be less than seen in unruptured aneurysms due to the direct effects of SAH.

Aneurysm morphology will influence outcomes of endovascular techniques using currently available devices. Not all aneurysms are geometrically suited to GDC treatment and the

best results are in small aneurysms with small necks, absolute neck size of less than 4 mm, aneurysms with dome-to-neck ratios of more than 2, and side wall aneurysms rather than termination aneurysms [189, 198]. The number of aneurysms potentially treatable by the coil techniques is increasing with new technological developments such as intracranial stents (to provide a scaffold with which to contain the coil), the balloon remodeling technique, softer, smaller coils, and three-dimensional coils [199–201]. Giant aneurysms remain difficult to successfully treat while preserving the parent vessel, with current endovascular or microsurgical techniques.

Surgery and endovascular repair can be complementary, and it is possible to create a neck with a clip and to complete obliteration with coils, to clip an aneurysm in which coiling fails, and to coil an aneurysm in which surgery has failed [202–204].

There will remain patients with aneurysms that cannot be obliterated using current surgical or endovascular techniques of vessel preservation. These may be treated by parent vessel occlusion, a useful technique that, with appropriate selection, can allow palliation of symptoms due to the mass effect and decrease the chance of SAH [174, 181, 182, 204–206]. Surgical or endovascular occlusion can be performed, with the use of extracranial-to-intracranial bypass if trial occlusion or anatomy of the circle of Willis suggests a high likelihood of cerebral hypoperfusion and stroke [204–206]. Morbidity is lower with an intact circle of Willis, posterior communicating artery diameters of more than 1 mm, no clinical or perfusion deficit during trial balloon occlusion, endovascular vs surgical occlusion. This latter may be due to balloon occlusion close to the site of the aneurysm, with less thrombus burden in the ICA stump to permit distal embolism.

Until definitive studies of long-term efficacy are completed, a multidisciplinary case by case approach is recommended, making a decision based on aneurysm size, location, geometry, patient clinical grade, presence or absence of vasospasm, vascular access, and patient preference. If geometrically favorable, Guglielmi detachable coil treatment is the preferred modality for posterior circulation aneurysms, partially extradural aneurysms, paraclinoid aneurysms, and for ruptured aneurysms regardless of site in critical patients.

Equipment and Technique

All procedures should be performed using digital subtraction angiography capable of high resolution, road mapping, and preferably biplane image acquisition.

The GDC System is comprised of a low-voltage power source, connecting electrodes, and the coils themselves; it is under continuous development. Coil types currently used in treating intracranial aneurysms include the GDC-18 and GDC-10 standard coils; GDC-10 Soft, GDC-18 Soft; GDC-18 2-D and GDC-10 2D; GDC-10 Stretch Resistant; and GDC-3D (Fig. 9).

Perioperative Medication

ANTICOAGULATION

Anticoagulation should be used in all cases of aneurysm treatment, in ruptured and unruptured aneurysms. The timing, degree, method, monitoring, and duration of anticoagulation differ among different endovascular therapy units. No randomized trial has resolved these issues. We commence anticoagulation with heparin bolus of 5000 U, tailored to the

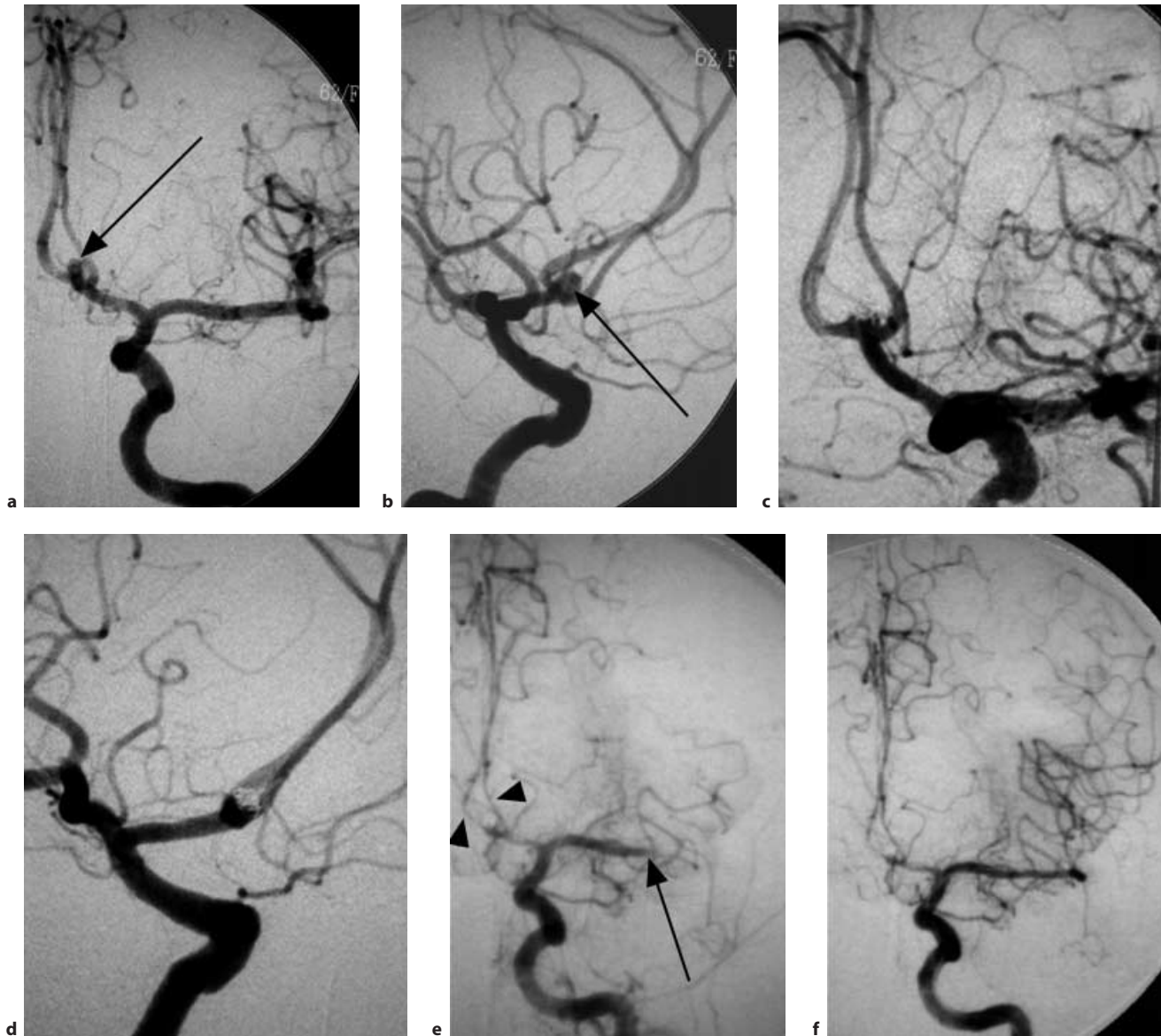


Fig. 9. **a, b** Woman, 76 years old, presenting day 1 with acute SAH, Hunt and Hess Grade 3. AP and oblique views of left ICA injection show an anterior communicating artery aneurysm (arrows), with a well-defined neck, acceptable dome-to-neck ratio, and filling from dominant left A1 segment. **c, d** Corresponding views after deployment of two GDC-10 coils (one standard, one soft stretch-resistant) with minimal filling at base of aneurysm, no filling of interstices, and normal caliber of the A1 and A2 segments. Note good caliber of the distal MCA and ACA vessels and compare to later figures. **e** Day 3 after GDC treatment of ruptured ACOM aneurysm, the patient developed left MCA clinical deficit with aphasia and hemiplegia, and severe vasospasm

on transcranial Doppler. DSA shows vasospasm A1 and A2 (arrowheads) and abrupt occlusion of the distal MCA (arrow) secondary to either embolus or more probably thrombus-complicating vasospasm. **f** After 300,000 U of urokinase and 300 mg papaverine, stable obliteration of ACOM aneurysm, preservation of both anterior cerebral arteries, and recanalization of the MCA with resolution of the proximal vasospasm. Note some persistent vasospasm in distal ACA and MCA beyond M1 and A1, respectively, but markedly improved compared to pretreatment DSA. Clinical improvement occurred, but was incomplete, leaving an inferior division MCA territory stroke on delayed imaging

patients body habitus. The bolus is administered at the commencement of catheterization in unruptured aneurysms and after microcatheter placement in ruptured aneurysms. Anticoagulation is monitored by hourly ACT and usually maintained by regular intravenous bolus injections rather than continuous infusion. In most instances, the heparin is not reversed, clotting being allowed to return gradually to normal. Heparin is continued for 1–2 days only in cases of thromboembolic complication requiring treatment, or in cases assessed as at unusually high risk of embolization such as giant aneurysms with a very broad neck.

ANESTHESIA

All procedures are performed with the patients under general anesthesia. This is essential to ensure a still patient throughout the procedure and to facilitate immediate management of any complication, with either endovascular or surgical rescue.

CATHETERIZATION

Catheterization of the aneurysm is facilitated in nearly all cases by having the appropriate shape steamed into the terminal portion of the microcatheter, appropriate to the

specific geometry and measurements of the aneurysm to be treated. Do not oversteer the catheter, as the lumen can be compromised and the length of the catheter can be altered. Magnified road-mapping images, biplane if possible, are used to guide placement of the catheter and subsequent coils. During placement within the sac, tension within the system must be avoided; otherwise the catheter and wire, or the catheter alone, can “jump” forward and potentially perforate the aneurysm.

COIL SELECTION

Coil selection depends primarily upon the geometry and size of the aneurysm and whether there has been recent SAH. GDC-18 coils are more rigid, form an excellent basket, bridging the aneurysm neck, and are ideal as first coils in larger aneurysms. GDC-18 Soft can be used subsequently to obtain more dense central packing or GDC-10 coils can be placed. GDC-10 standard coils are used in small and medium-sized aneurysms and can be combined with GDC-10 Soft coils for final packing. The latter are more prone to unraveling and rupture and must be used with caution. 2D coils and 3D coils are used as initial coils when wide-necked aneurysms or aneurysms encroaching on adjacent vessels are present. Initial coil selection should be equal to the largest diameter of the aneurysm, or slightly less in acutely ruptured aneurysms, and of a sufficient length to form a good basket crossing the neck and provide stability for later denser packing with smaller softer coils. As a general rule, the diameter of the coils placed should never increase, nor should stiffer standard or 3D coils follow softer coils. As dense a packing as possible should be the goal of therapy; subsequent packing of neck remnants is usually harder than at the initial treatment. In poor-grade patients with SAH, it is sometimes expedient to densely pack the body and fundus, providing protection from immediate rebleeding with minimal morbidity and allowing elective surgical or repeat packing when the patient improves to a better grade. Follow-up angiography is scheduled to be performed at 6 months, 1 year, and 2 years after treatment.

Unfavorable Geometry – Alternative Techniques

Remember that microsurgical clipping remains the gold standard and some aneurysms are better-suited to surgery than heroic efforts at endovascular treatment. In particular, careful selection of the middle cerebral artery, anterior communicating artery, and giant aneurysms is essential if long-term occlusion rates are to be optimal. A variety of techniques are currently available to assist in the goal of complete exclusion of the aneurysm lumen from the circulation. The balloon remodeling technique [200] has had a significant impact on aneurysm treatment, allowing those with wider necks to be treated with increasing density of packing and with little or no increase in morbidity [199, 200]. A balloon catheter is first placed across the neck of the aneurysm, the aneurysm is then catheterized and the coils deployed while the balloon is inflated, causing the coils to be deployed within the aneurysm against the inflated balloon (Fig. 10). Inflation is timed and restricted to 2–4 min and is not initiated until the coil is at the end of the microcatheter. A road map is then taken and the balloon deflated, observing carefully for any movement of the coil mass. If stable with no prolapse into the parent vessel, the coil is detached and the cycle repeated until dense packing is achieved.

Newer coils assist in treating larger aneurysms with unfavorable neck size and the GDC-3D and GDC-2D have already been mentioned. The combination of intravascular stents and GDC has allowed false aneurysms and berry aneurysms to be treated [201, 207]. Parent vessel occlusion can be combined with extracranial to intracranial bypass grafting if the patient fails trial occlusion testing (Fig. 11).

Complications

THROMBOEMBOLISM

Thromboembolism remains the most common major complication of GDC treatment, from thrombus formation around the catheter system used (despite heparinized pressure infusions), local atherosclerosis, or within the aneurysm. The process of electrocoagulation involves inducing thrombus within the aneurysm and coil mass; packing further coils into the lumen can potentially displace thrombus or aggregated platelets. Heparinization remains the mainstay of prevention, together with the usual care of pressurized flushing solutions and catheter placement. Injections within the aneurysm (aneurysmogram) should be avoided in most cases. The use of abciximab is a potent means of preventing platelet aggregation, but is long-lasting and to be avoided in acute SAH. Once recognized management should depend upon the site and likely significance of the occlusion if a major vessel heparinization should continue and local infusion of urokinase be administered (Fig. 9). This has been safely performed even in the presence of a ruptured aneurysm, as long as low doses distal to the aneurysm can be administered directly into the clot. It remains a hazardous procedure and the risks of hemorrhage must be weighed against the risk of the deficit likely to result from the vessel occlusion.

ANEURYSM RUPTURE

Rupture of the aneurysm is most likely to occur with small aneurysms presenting with acute SAH. Avoidance is best, but it will be encountered as more and smaller aneurysms are encountered. When recognized by hemodynamic changes reflecting elevated intracranial pressure, heparinization must be reversed with protamine, the coil deployed (never remove a coil if it is suspected of lying partially outside the aneurysm), and further coils deployed as rapidly as possible to densely pack the aneurysm. Emergency extra-ventricular drain placement in the angiography laboratory can be a life-saving procedure. Other complications to be discussed with the patient and family include contrast-related issues of renal toxicity and anaphylaxis and local puncture site problems (hematoma, false aneurysm, vessel occlusion).

Residual aneurysm rests after surgical clipping have been demonstrated to pose a significant risk of subsequent growth and rupture [208, 209], although there may be a subset that do not pose as great a risk [197]. While incomplete GDC obliteration of a recently ruptured aneurysm has been demonstrated to markedly reduce the chance of rehemorrhage in the short term, significant rehemorrhage rates have been reported on later follow-up. These are approximately 1%–5% in smaller aneurysm rests, but up to 33% has been reported in giant ruptured aneurysms with large unpacked areas [189, 198, 199, 210–217]. It would seem reasonable to pursue complete obliteration as the goal of therapy, to retreat residual rests if large, but very small dog-ear remnants may not pose the same risk of hemorrhage and should be

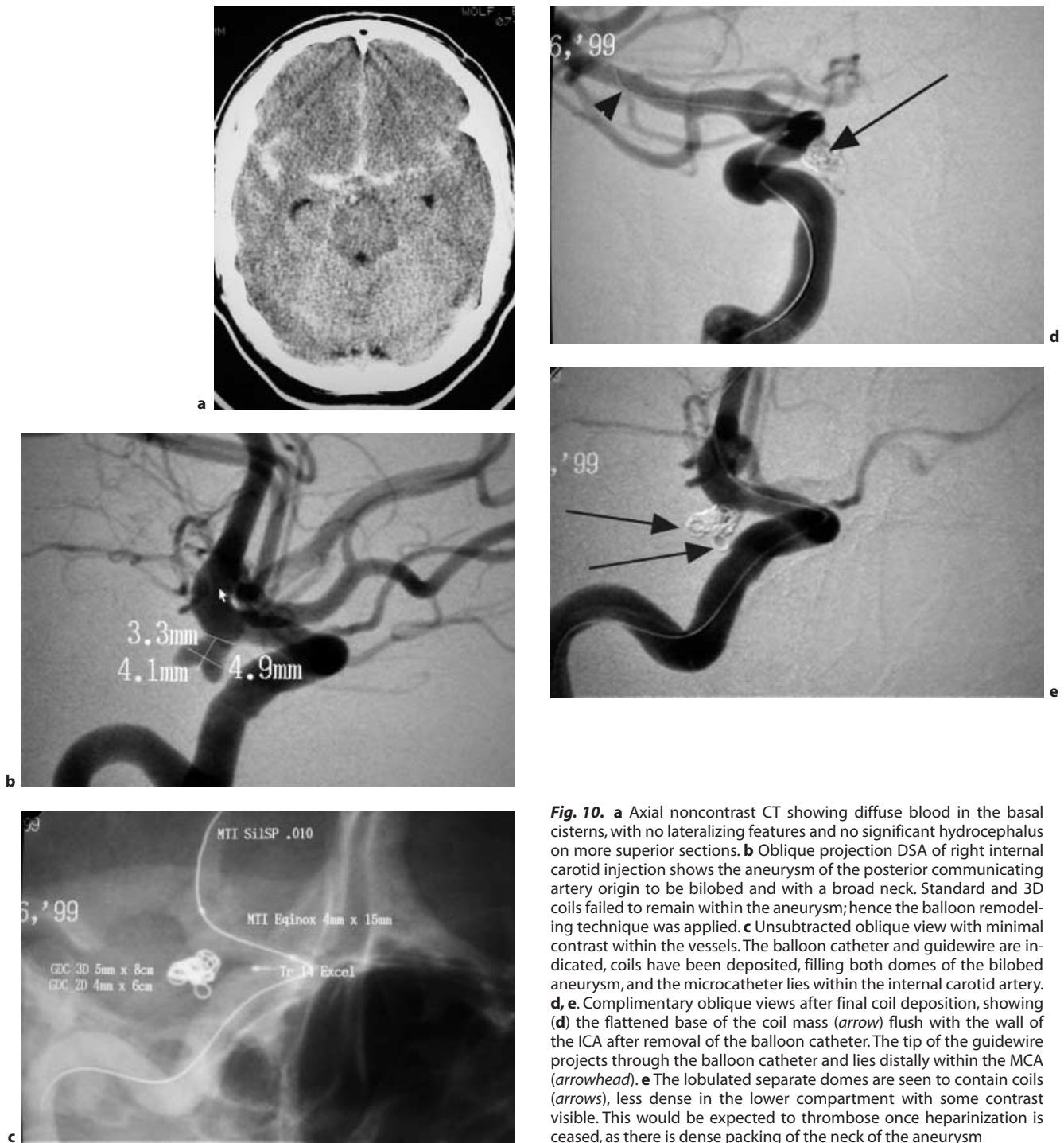


Fig. 10. **a** Axial noncontrast CT showing diffuse blood in the basal cisterns, with no lateralizing features and no significant hydrocephalus on more superior sections. **b** Oblique projection DSA of right internal carotid injection shows the aneurysm of the posterior communicating artery origin to be bilobed and with a broad neck. Standard and 3D coils failed to remain within the aneurysm; hence the balloon remodeling technique was applied. **c** Unsubtracted oblique view with minimal contrast within the vessels. The balloon catheter and guidewire are indicated, coils have been deposited, filling both domes of the bilobed aneurysm, and the microcatheter lies within the internal carotid artery. **d, e.** Complementary oblique views after final coil deposition, showing **(d)** the flattened base of the coil mass (arrow) flush with the wall of the ICA after removal of the balloon catheter. The tip of the guidewire projects through the balloon catheter and lies distally within the MCA (arrowhead). **e** The lobulated separate domes are seen to contain coils (arrows), less dense in the lower compartment with some contrast visible. This would be expected to thrombose once heparinization is ceased, as there is dense packing of the neck of the aneurysm

followed. If they remain stable, treatment can probably be deferred indefinitely.

Results – Selected Literature Review

Several more recent publications present results from large series of patients with GDC treatment of aneurysms [189, 198, 199, 210–216]. Morbidity and mortality are highly dependent upon the proportion of patients with poor-grade SAH, giant aneurysms, and posterior rather than anterior circulation sites, and hence average reports have little mean-

ing. A meta-analysis indicated the rates of procedural morbidity and mortality to be 3.7% and 1%, respectively [218]. The morbidity and mortality have decreased with time and the proportion of aneurysms with complete obliteration has increased, especially when more rigid criteria for treatment are applied [198]. Thromboembolic events occur more frequently with acutely ruptured aneurysms, especially aneurysms with a wide base. Recurrence and incomplete obliteration are more common with giant or large aneurysms and aneurysms with a wide base. Vasospasm rates do not vary significantly from those found in surgical series when cor-

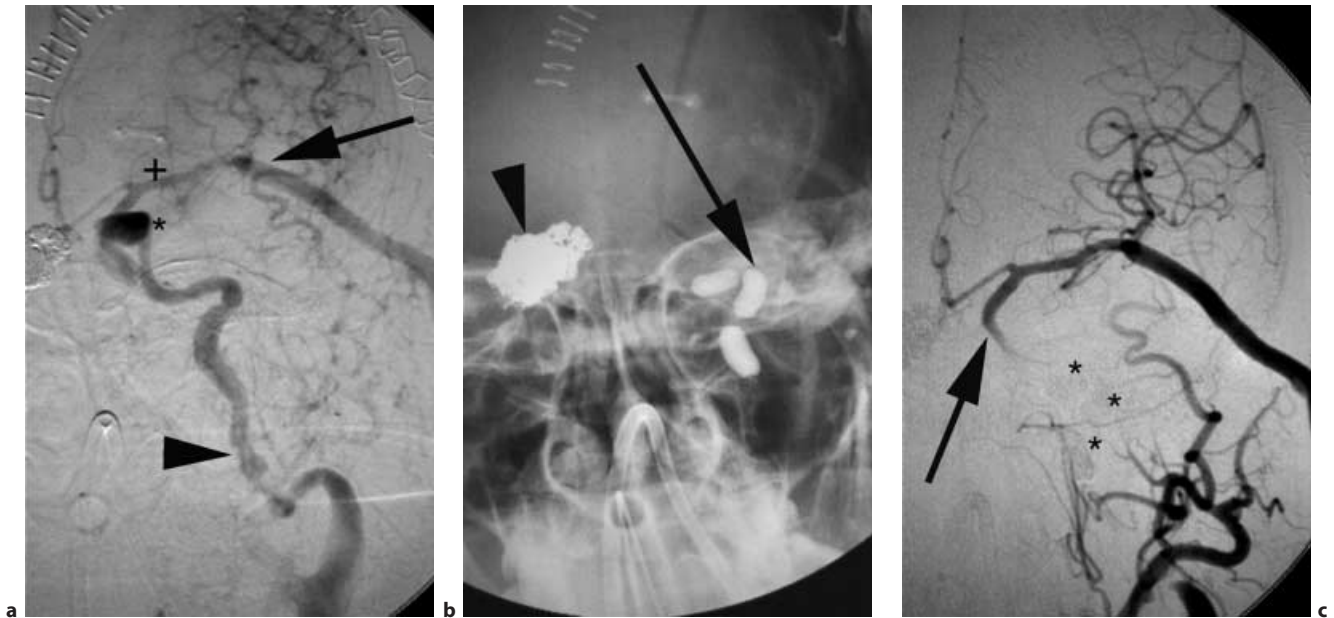


Fig. 11. **a** Patient with large predominantly intracavernous carotid aneurysm. Trial carotid occlusion suggested high risk of ischemia after carotid sacrifice, hence planned surgical bypass and endovascular occlusion. An arrow indicates the ECA-to-MCA bypass graft with good opacification of the intracranial vessels on frontal projection. Fibromuscular disease of the mid cervical ICA (arrowhead); asterisk, ipsilateral cavernous carotid aneurysm. The M1 segment of MCA is shown by a plus sign (+). **b** Three detached balloons in the distal cervical and petrous carotid artery (arrow). Coil mass from previous treatment of right cavernous DAVF (arrowhead). **c** After DSB deployment, complete occlusion of the ICA; ECA/MCA graft fills M1 and A1 segments and refluxes down terminal ICA (arrow). Balloons indicated by (asterisks). No significant filling of aneurysm on delayed images, even in immediate postocclusion films and with full anticoagulation with heparin

rected for Fisher grouping. Morbidity and mortality rates as well as Glasgow outcome scores are at least as good as what would be expected from surgery during both the acute and nonacute setting [212]. Details of several larger series are presented to illustrate these trends and the results in specific situations.

From December 1990 to July 1995 a prospective clinical study evaluated the safety of the Guglielmi detachable coil (GDC) system for the treatment of aneurysms [189]. There were 403 patients who presented with acute subarachnoid hemorrhage from a ruptured intracranial aneurysm. Of the total group of patients, 70% were female and 30% were male, with a mean age of 58; 57% of the aneurysms were located in the posterior circulation. Eighty-two patients were classified as Hunt and Hess Grade I (20.3%), 105 Grade II (26.1%), 121 Grade III (30%), 69 Grade IV (17.1%), and 26 Grade V (6.5%). All patients were excluded from surgical treatment either because of anticipated surgical difficulty (69.2%), attempted and failed surgery (12.7%), the patient's poor neurological (12.2%) or medical (4.7%) status, and/or refusal of surgery (1.2%). Complete aneurysm occlusion was observed in 70.8% of small aneurysms with a small neck, 35% of large aneurysms, and 50% of giant aneurysms. There was an 8.9% immediate morbidity rate related to the GDC technique. Seven deaths were related to technical complications (1.74%) and 18 (4.47%) to the severity of the primary hemorrhage. This series demonstrated lower mortality and morbidity

than would have been expected from surgical management or the natural history of a similar cohort.

The results of a cohort of 150 patients with either ruptured (83 patients) or unruptured (67 patients) basilar tip aneurysms treated with GDC in the early part of the United States multicenter GDC clinical trial were reviewed [213]. Conservative mortality rates included up to 23% for the ruptured aneurysm group and up to 12% for the unruptured aneurysm group; the rebleeding rate for treated ruptured aneurysms was up to 3.3% and the bleeding rate for unruptured aneurysms up to 4.1%. Permanent deficits due to stroke in patients with ruptured or unruptured aneurysms occurred in up to 5% and 9%, respectively. Periprocedural mortality was 2.7% (four patients with ruptured aneurysms). In selected patients, GDC treatment offers lower incidences of morbidity and mortality compared with conservative medical management. The role of this procedure in unruptured basilar tip aneurysms is unclear, with less supportive results. More long-term follow-up evaluation is necessary and results are expected to improve.

A retrospective analysis of 329 patients with 339 cerebral aneurysms treated at a single institution from May 1994 to June 1997 was reported [198]. When selection for GDC treatment was based on nonsurgical candidates or patients at higher risk from surgery, there were high morbidity and mortality rates, with 56% of the treated aneurysms occluded at 6 months. When aneurysm geometry, as determined by pretherapeutic angiography, was the basis for selection for GDC treatment (119 patients with 123 aneurysms), there was no mortality directly related to the coiling procedure and permanent morbidity was limited to 1.0%. The morphological results were strongly correlated with the geometry of the aneurysms, with a complete occlusion rate of 72% among the acutely ruptured aneurysms and 80% among the nonacute aneurysms, when patients were selected for treatment based on the geometry of the aneurysms and the dome-to-neck ratio was at least 2. Aneurysms that were considered to be favorable for coiling included those that had a dome-to-neck ratio of at least 2 and an absolute neck diameter less than 5 mm. The percentage of complete aneurysm occlusion is related to the density of coil packing, which is strongly de-

pendent on the geometry of the aneurysm. Optimal results are obtained when the dome-to-neck ratio is at least 2.

Boccardi et al. reported their experience in 100 patients with 118 intracranial aneurysms treated with GDC [210]. The clinical outcome at follow-up (up to 3 years) was 72 patients without neurological deficits, 19 patients improved preexisting neurological deficits, 3 patients worsened for procedural complications, and 6 patients died (1 patient unrelated). Midterm clinical outcome (2–6 years, average 3.5 years) was reported for 94 patients of an initial consecutive series of 100 [215]. The midterm post-GDC embolization hemorrhage rate was 0% for small aneurysms, 4% (one case) for large aneurysms, and 33% (five cases) for giant lesions. Patients with Grade IV or V subarachnoid hemorrhage in this series generally had poor outcomes even if the GDC procedure was successful in occluding the aneurysm. Kuether et al., over a period of 4.5 years, treated 74 patients with intracerebral aneurysms with GDC at a single center [214]. They reported no completely occluded aneurysm hemorrhaged after GDC treatment (follow-up period, 1.9 years). Of near complete occlusions, 2.6% hemorrhaged after embolization, at a rate of 1.4% per year. Casasco et al. reported 295 aneurysms in 217 patients treated by detachable coils (GDC), 50% presenting with SAH [211]. Complete occlusion was obtained in 78%, subtotal in 19%, and partial in 3%. There was only a 3.05% permanent neurological deficit. The overall results were 80% good results, 5.8% moderate disability, 2% severe disability, and 11% deaths.

Cognard et al. [190] reported on 208 patients with 236 intracranial berry aneurysms treated by endovascular coil embolization. The technique-related morbidity rate was 4% and the mortality rate was 2%. Rebleeding occurred in one patient after incomplete occlusion.

There is evidence that aneurysms causing symptoms through mass effect can also be successfully treated with GDC. In a series of 19 patients presenting with cranial nerve dysfunction due to mass effect treated with GDC [216], the response was classified as complete resolution of symptoms in six patients (32%), improvement in eight patients (42%), no significant change in four patients (21%), and symptom worsening in one patient (5%). Patients with smaller aneurysms and those with shorter pretreatment duration of symptoms were more likely to experience an improvement in their symptoms following GDC treatment. The presence of aneurysmal wall calcification (six patients) or intraluminal thrombus (12 patients) showed no correlation with the response of mass effect symptoms.

Conclusion

Treatment of unruptured aneurysms with GDC has a low complication rate of around 3% with minimal mortality; all recent reports have indicated improving outcomes due to a combination of technological advances and increasing experience. In selected cases, up to 70% of aneurysms can be completely obliterated, but 20% of aneurysms can regrow a neck remnant, usually due to compaction of the coils [191]. The neck remnants pose a risk of delayed SAH and if growing or large should be treated with further GDC, or less commonly with surgical clipping. The procedural morbidity and mortality in ruptured aneurysms depends upon the grade, but seems to be markedly less than for surgery. Estimates of the number of aneurysms that will be treated by GDC vary widely, from none to 90%. There are centers where 80% of aneurysms are currently treated with coils. The best strategy, until definitive studies of long-term efficacy are completed,

is to have a multidisciplinary approach discussing each case and making a decision based on aneurysm size, location, geometry, patient clinical grade, presence or absence of vasospasm, vascular access, and patient preference.

TRAINING REQUIREMENTS AND RECOMMENDATIONS

Proposed Training Program

The unique clinical and invasive nature of this subspecialty requires special training and skills [219, 220]. Training and experience in the following are necessary:

1. Signs and symptoms of disorders amenable to diagnosis and treatment by interventional neuroradiology (INR) techniques
2. Neurological examinations to evaluate patients with neurological disorders
3. Pathophysiology and natural history of these disorders
4. Indications and contraindications to interventional neuroradiology (INR) procedures
5. Clinical and technical aspects of interventional neuroradiology (INR) procedures
6. Medical and surgical alternatives
7. Preoperative and postoperative management of neurovascular patients
8. Neurointensive care management
9. Fundamentals of imaging, radiation physics, and radiation biology
10. Interpretation of radiographic studies pertinent to the practice and integration of information available from imaging studies that is pertinent to the neurovascular practice

Recommended program paths for INR training are as follows:

1. Radiology residency pathway
 - Normal training and 1-year diagnostic neuroradiology fellowship
 - 2 years INR fellowship
2. Neurosurgery residency pathway
 - Normal training
 - 2 years INR fellowship

The first 12 months of the INR training program will prepare the trainees with experience in which they learn knowledge-based and procedural skills from an interventional neuroradiology specialist in the following areas:

1. The use of needles, catheters, guidewires, and angiographic devices and materials
2. The basic radiological sciences, including radiation physics, radiation protection, and the pharmacology of radiographic contrast materials
3. Angiography and basic image interpretation evaluation relevant to the specialty of interventional neuroradiology (INR)
4. The proper use of and indications for laboratory tests and methods that are adjunctive to INR procedures such as physiological monitoring, noninvasive neurovascular testing, and noninvasive neurovascular imaging
5. The evaluation of patients with neurological disease
6. The basic and clinical neurosciences, including neuroanatomy, neurobiology, and the pathophysiology and natural history of neurological disorders, especially cerebrovascular and neoplastic conditions

7. The clinical aspects of patient assessment, treatment planning, and patient management related to INR therapy, including the fundamentals of invasive monitoring and neurointensive care management
8. The clinical indications, risks, and limitations of INR procedures
9. The use and administration of analgesics, antibiotics, anticoagulation agents, neuroanesthetic agents, and other drugs commonly used in INR procedures

This hands-on experience must include catheter-based diagnostic angiography. Each trainee must have been involved in at least 100 catheter-based diagnostic cerebral angiograms before undertaking more complex cases involving therapeutic and interventional procedures.

The second of 12 continuous months must be spent in the clinical INR training program, during which the trainee has the opportunity to carry out all of the following procedures under close supervision:

1. Perform clinical preprocedure evaluations of patients
2. Interpret and evaluate preliminary diagnostic studies
3. Consult with clinicians on other services
4. Perform diagnostic and therapeutic INR procedures
5. Generate procedural reports
6. Participate in short-term and long-term postprocedural follow-up care, including neurointensive care

Patient Population

The institution's patient population must have a diversity of illnesses from which broad experience in INR therapy can be obtained. The case material should encompass a range of neurological diseases, including neurovascular diseases. An adequate variety and number of INR procedures must be available for each trainee. Each program must perform a minimum of at least 100 therapeutic INR procedures per trainee per year. These procedures include the treatment of aneurysms, brain arteriovenous malformations, arteriovenous fistulas of the brain, tumors of the central nervous system, occlusive vascular diseases, revascularization, traumatic injury, and maxillofacial vascular malformation and tumors. In addition, the program must provide adequate training and experience in invasive functional testing. Each trainee must maintain a personal case log, which the program director must certify at the completion of training.

Equipment and Facilities

Modern imaging and procedure rooms and equipment must be available and must permit the performance of all INR procedures. Rooms in which INR procedures are performed should be equipped with physiological monitoring and resuscitative equipment. The following state-of-the-art equipment should be available: MRI scanner, CT scanner, high resolution digital subtraction angiography equipment, with live subtraction and road-mapping capabilities, simultaneous biplane fluoroscopy and filming (highly desirable), and ultrasound equipment.

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Gastrointestinal Vascular Diseases: Endoscopic Treatment

Todd H. Baron, Piet C. de Groen

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INTRODUCTION

In this chapter we will describe the equipment used in the management of gastrointestinal hemorrhages, the endoscopic devices that can be manipulated through endoscope channels, the conditions that determine the type of equipment and procedures used, and the various therapeutic procedures performed in the gastrointestinal tract by the experienced therapeutic endoscopist. For each procedure we will list the indications, contraindications, technique used, and results. Where appropriate, we will discuss post-procedure management and procedure-related complications. The purpose of this chapter is to provide an overview, not a detailed description of each technique. General knowledge of endoscopic procedures and equipment (upper or gastroscopes, lower or colonoscopes, and side-viewing endoscopes) is assumed.

THERAPEUTIC ENDOSCOPES

Standard diagnostic endoscopes have a suction channel diameter of 2.8 mm. During emergency endoscopies, where large amounts of fluid, fresh blood, and blood clots may need to be removed in order to obtain and maintain adequate vision, large-channel therapeutic endoscopes with at least a single 3.7-mm-diameter channel, with or without a second channel, are used (Fig. 1). The large suction channels on these endoscopes improve both the diagnostic and therapeutic endoscopic procedures for patients with severe bleeding. Several companies manufacture video endoscopes with

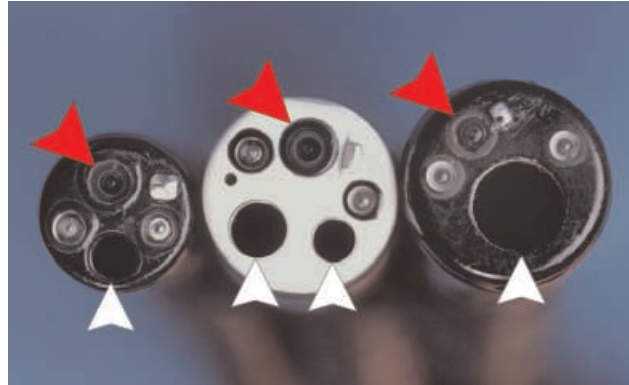


Fig. 1. Comparison of upper gastrointestinal endoscopes. The white arrows denote the suction/accessory channels. The red arrows denote the optical channel. Diagnostic endoscope (left) with a 2.7-mm working channel. Double-channel therapeutic endoscope (middle) with 2.7-mm and 3.2-mm working channels. Prototype therapeutic endoscope (right) with single 6-mm working channel

dual 3.7-mm and 2.8-mm-diameter suction channels (Fig. 1). Additionally, a prototype of an endoscope with a single 6-mm-diameter channel and separate suction canisters is currently being evaluated (Fig. 1). The dual channel endoscopes allow a therapeutic hemostatic device to be passed through one channel while fluid or blood is suctioned through the other. Single-channel diagnostic video endoscopes are sufficient for the diagnosis of actively bleeding lesions but may not be adequate for treatment purposes. As a hemostatic device already occupies the suction channel, adequate suction is not possible and blood or clots may obscure an actively bleeding lesion and its precise bleeding point.

ENDOSCOPIC DEVICES AND PROCEDURES

Monopolar and Bipolar (Multipolar) Coagulation

Coagulation devices are passed through the endoscope channel and allow coaptation of the underlying vessel by both pressure and current applied to the lesion. As resistance is greatest at the point of application, heat is generated locally within the tissue, which results in coagulation. The first types of electrocoagulation probes used were monopolar. As the name implies, monopolar probes consist of a single pole, and current flows from the probe to a ground-plate attached to the patient's trunk or extremity. The tissue damage produced by monopolar electrocoagulation may be severe and difficult to predict based upon power settings. Application of pressure needs to be *en face*, which may be difficult depending on the location of the lesion. Additionally, monopolar probes frequently adhere to the tissue during coagulation. Pulling the probe away from the lesion after coagulation can cause rebleeding at the site [1].

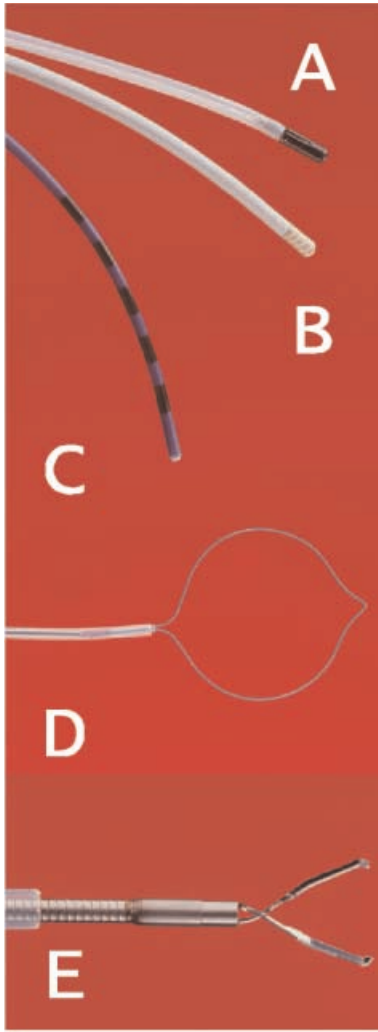


Fig. 2. Assortment of endoscopic accessories for achieving endoscopic hemostasis: *A* heater probe, *B* large 10-Fr bipolar coagulation probe, *C* probe for the argon beam plasma coagulator, *D* detachable endoloop. After closure around the vessel, the loop is released from the delivery catheter. *E* Endoclip device: After closure around the vessel, the clip is released from the delivery catheter

Monopolar devices have been succeeded by bipolar electrocoagulation devices (Fig. 2, *B*), as the latter are safer and have an improved efficacy. Bipolar electrocoagulation currently is a widely used technique for nonvariceal hemostasis. The advantages of bipolar equipment are a high efficacy, a good safety profile, portability for transportation to bedside treatment, and the ability to combine irrigation, tamponade, and coagulation [1, 2]. Unlike monopolar coagulation, bipolar coagulation probes are equally effective when coagulation is applied with the side of the probe (tangentially) and when applied *en face* [3].

Heater Probe Coagulation

The heater probe, as its name implies, is used to apply heat directly from the tip of the probe to lesions (Fig. 2, *A*). The mechanism of tissue coagulation and the advantages of its use for emergency hemostasis are essentially the same as for bipolar electrocoagulation: excellent efficacy, good safety profile, portability, and ability to combine irrigation, tamponade, and coagulation. Because of this, endoscopic use of heater probe and bipolar electrocoagulation has largely replaced the use of lasers (discussed below) for thermal endoscopic treatment of nonvariceal hemorrhage [4].

Nd:YAG Laser Coagulation

Laser therapy coagulates by vaporization. Several different instruments have light guides with metal tips designed for endoscopic use. Many continuous-wave, high-power lasers are water-cooled units requiring specialized electrical and water connections. Additionally, the laser units are large and therefore not portable. Currently, lasers such as Nd:YAG (neodymium-doped yttrium-aluminum-garnet) and argon are rarely used for hemostasis because of the simplicity, lower cost, and portability of other techniques. However, high-power Nd:YAG lasers achieve adequate hemostasis of bleeding ulcers [1, 5]. Since the depth of coagulation is greater compared with other thermal methods there is a greater risk of perforation when used in thinner areas of the gastrointestinal tract, such as the small bowel and right colon [6].

Infrared Coagulator

The infrared coagulator is a hand-held device with a high-intensity infrared light bulb connected to a probe with a flat tip. The heat generated by the infrared light coagulates vessels and tethers the mucosa to subcutaneous tissues. The device is used primarily for the treatment of hemorrhoids.

Argon Beam Plasma Coagulation

Argon beam plasma coagulation is a new method of noncontact electrocoagulation in which monopolar current is transmitted to the tissue through ionized argon gas. As described for monopolar probes, current flows from the probe via the ionized argon gas to a ground-plate attached to the patient's trunk or extremity. Grounding of the electrical charge in tissue generates thermal energy, leading to coagulation and desiccation. The depth of coagulation is 2–3 mm, which theoretically reduces the risk of perforation in thinner-walled gastrointestinal structures. Endoscopic probes are available in a variety of sizes and shapes to optimize the shape and direction of the argon gas beam (Fig. 2, *C*). Advantages of argon beam plasma coagulation include coagulation without the need for tissue contact and relative portability as compared with laser. The disadvantage of this procedure is the need to constantly pump argon gas during treatment, which may result in overdistention and perforation of the intestines.

Hemoclips

Although endoscopic clipping devices were proposed 20 years ago, they have only recently become available for use. The hemoclip application device is used for treatment of nonvariceal hemorrhage in both the upper and the lower gastrointestinal tract. Clips are composed of stainless steel and are applied directly to the bleeding vessel and then deployed (i.e., closed) (Fig. 2, *E*). The advantages of the hemoclip device over other endoscopic hemostatic devices are the ability to treat larger vessels and the relative lack of induction of tissue injury or perforation. However, loading the clip onto the application device can be cumbersome, and the device can be difficult to rotate inside the endoscope in order to position it optimally before application.

Loops

The endoloop is a detachable nylon loop (snare) that can be tightened around a vascular lesion (Fig. 2, *D*). It has been used

predominantly to prevent bleeding by ensnaring the pedicle of large vascular polyps prior to snare resection. However, endoloops have also been used in the treatment of esophagogastric varices.

Band Ligation

Band ligation was originally developed for the treatment of hemorrhoids. Once a method of endoscopic deployment of bands was developed, band ligation rapidly became widely used for the treatment of esophageal varices. Currently, it is used also to treat acute upper or lower tract gastrointestinal bleeding attributable to nonvariceal causes. The endoscopic band ligation device consists of a cylindrical chamber that is attached to the tip of the endoscope. One or more rubber bands are preloaded over the chamber. A bolus of tissue (varix, mucosal lesion) is suctioned into the chamber and a rubber band is deployed around the tissue. The resulting strangulation of the tissue with vessel results in thrombosis within the vessel. Multi-band ligators allow deployment of up to ten individual bands without having to “reload” the device.

Injection Therapy

Endoscopic injection therapy is performed by passing an injection needle (23–26 gauge, 3–5 mm long, attached to a catheter) through the instrument channel of the endoscope into the intestinal epithelium. Once the needle is placed, a variety of agents can be injected. Injection therapy is one of the most widely used endoscopic methods because of its ease of application, accessibility, and cost. Multiple substances have been utilized for injection therapy; they are chosen based on the indication (variceal versus nonvariceal bleeding). Injection agents may be broadly grouped as follows:

- a. **Sclerosants:** These agents are used primarily for variceal therapy but have been employed for nonvariceal upper gastrointestinal bleeding as well. Because they cause local tissue destruction, these agents have generally not been used outside of the esophagus and stomach. Examples of sclerosants include sodium morrhuate (5%), sodium tetradecyl sulfate (1%–1.5%), ethanolamine oleate (5%), and polidocanol (1%).
- b. **Desiccants:** These agents cause thrombosis by fixation of tissue. Absolute alcohol is an example of a desiccant.
- c. **Saline:** Saline (0.3%, physiologic [0.9%] or hypertonic) causes a local tamponade effect. Saline has virtually no side effects. It may be used to provide initial control of active nonvariceal bleeding, but at present it is rarely used as a single agent.
- d. **Dilute epinephrine:** Epinephrine at a ratio of 1 : 10,000 diluted in physiologic saline is used as a vasoconstrictive agent for treatment of nonvariceal upper and lower gastrointestinal bleeding.
- e. **Polymers/glue:** Derivatives of cyanoacrylate have been used with success. The most commonly used polymer is *N*-butyl-2-cyanoacrylate. Vascular occlusion by way of polymerization occurs when the agent comes into contact with blood. These agents have been used primarily for the control of gastric variceal bleeding, which is difficult to control with other endoscopic methods.
- f. **Fibrin glue:** This relatively new therapy combines a fibrinogen and thrombin solution through a double-lumen catheter. After the components enter the tissue or vessel, a stable clot is formed.

Endoscopic Sewing and Stapling Machines

Endoscopic sewing and stapling machines are in the early stages of development. The theoretical advantage of these devices is permanent, complete hemostasis with the ability to treat large vessels. At present, their use is limited to experimental animal studies.

CHOICE OF ENDOSCOPIC DEVICE OR PROCEDURE

Location

ESOPHAGUS

Most massive esophageal bleeding is caused by varices. Upper endoscopy using large-caliber endoscopes is preferred, although the band-ligation device will not fit on very large, prototype endoscopes. Endoscopic options most widely employed are injection therapy using sclerosants or band ligation.

Stomach. Gastric ulcers, varices, and Dieulafoy's lesions are the most common sources of bleeding. Large-channel devices are preferred for suctioning and clearing. Nearly all therapeutic devices and procedures are used in the treatment of gastric vascular lesions.

Duodenum. Duodenal ulcers and angiomata are most commonly seen as causing bleeding from duodenal lesions, and the endoscope equipment and devices are the same as for gastric lesions. Duodenal varices are rarely seen.

Small Bowel. Small bowel ulcers beyond the duodenal bulb are uncommon causes of gastrointestinal bleeding. Vascular ectasias may be found throughout the small bowel, but endoscopic treatment – using long endoscopes – is difficult and frequently not possible. The endoscopic options most commonly used for treatment of small intestinal bleeding are injection therapy, bipolar electrocoagulation, and argon beam plasma coagulation. Power settings are reduced to prevent transmural injury of the bowel wall.

Colon. Diverticula, post-polypectomy scars, and vascular ectasias are common causes of colonic bleeding. Rarely, Dieulafoy's lesions may be encountered. Injection therapy, bipolar electrocoagulation, and argon beam plasma coagulation are most commonly used to treat these lesions. Distal lesions, in particular hemorrhoids, are amenable to band ligation.

Rate of Bleeding

In the nonbleeding patient with stigmata of hemorrhage, endoscopic therapy can be easily performed. Massive gastrointestinal bleeding requires stabilization of hemodynamics. If bleeding is from an upper gastrointestinal source, protection of the airway by liberal use of endotracheal intubation is mandatory. A tagged red blood scan or arteriography may be needed to guide the endoscopic intervention of continued overt bleeding by an unidentified source. Tagged red blood scans are more sensitive and hence allow detection of bleeding at a lower rate than arteriography. A bleeding rate of about 0.5–1 ml blood/min is required to visualize a bleeding site with arteriography.

Experience and Preference of Operator

The experience and preferences of the endoscopist will influence the choice of endoscopic devices used in the treatment of vascular lesions. Some endoscopists will use sclerotherapy for acute bleeding esophageal varices, while others prefer band ligation. Additionally, the type of sclerosant and location of injection for esophageal variceal sclerotherapy varies widely based upon operator preference and experience. According to a recent survey, 65% of endoscopists preferred treating nonvariceal bleeding with injection therapy followed by thermal therapy [4].

THERAPY

Esophagus

ESOPHAGEAL VARICES

Indications. A major indication for variceal treatment is massive gastrointestinal bleeding. As many as one third of patients presenting with variceal hemorrhage may have active bleeding during the initial diagnostic endoscopy. This group of patients requires extensive resuscitation with blood products. However, these resuscitative measures generally do not stop the acute bleeding. Endoscopy using therapeutic equipment offers the benefit of providing immediate hemostasis during the initial diagnostic procedure. A second indication for treatment is an episode of upper gastrointestinal hemorrhage and the presence of nonbleeding esophageal varices at endoscopy. Endoscopic treatment of the varices is indicated if no other lesions are identified to explain the bleeding. Follow-up variceal therapy for obliteration is recommended after the initial bleeding episode is controlled. Prophylactic endoscopy to prevent an episode of variceal bleeding is controversial but has recently been reported to be more effective than medical therapy [7].

Contraindications. Endoscopic treatment of acute hemorrhage is contraindicated for patients who cannot be stabilized hemodynamically, or for those who have a free gastrointestinal perforation.

Technique. Band ligation and sclerotherapy are commonly used to treat esophageal varices. Actively bleeding varices are always treated first. Beginning with the esophagogastric junction, each column of varices is treated with at least one band (Fig. 3) or 0.5–2 ml of sclerosing agent. The process is continued proximally, treating only the distal 5 cm of the lower esophagus. At the initial session, usually a minimum of six and not more than 12 bands are placed. Once bleeding is controlled, follow-up band ligation is performed every week or two until all distal esophageal varices have been obliterated.

Although sclerotherapy has been used worldwide since the early 1980s, there is no uniformly accepted technique to which all endoscopists adhere. On the contrary, there is considerable variation in the choice of sclerosants, injection sites,

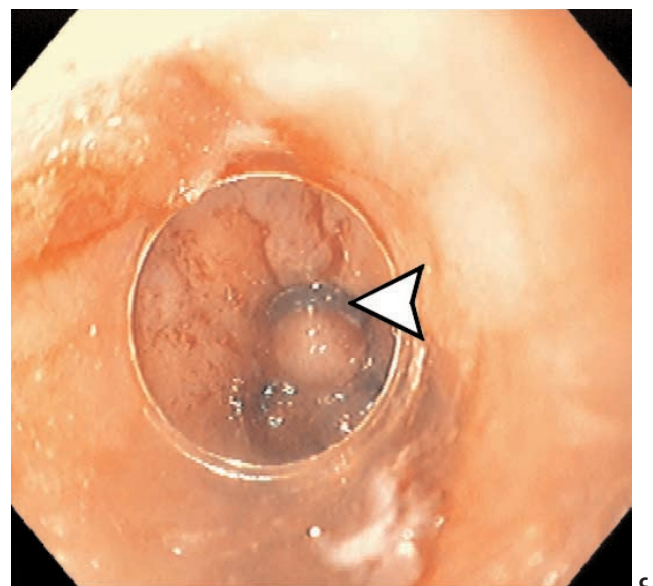
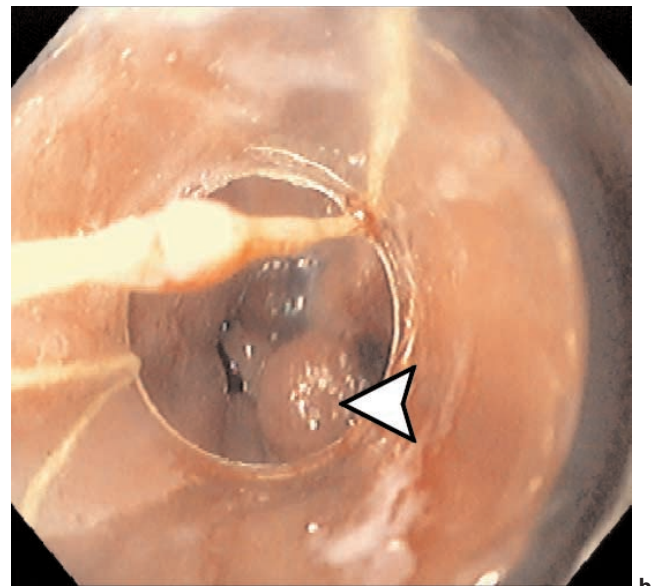
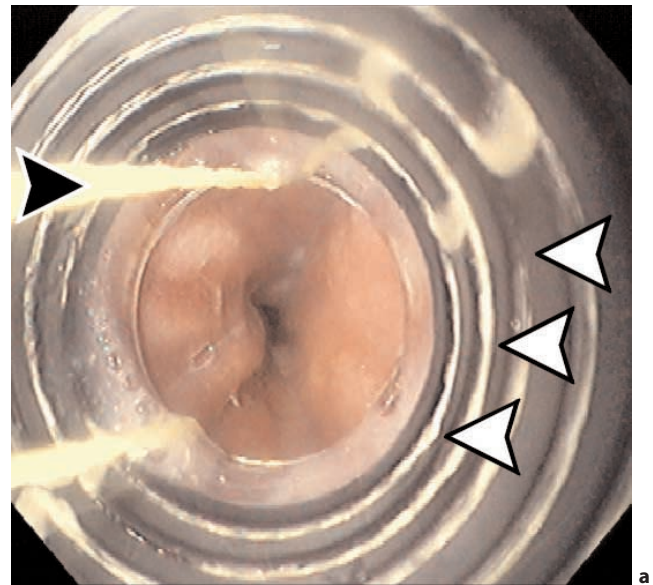


Fig. 3a–c. Endoscopic band ligation of esophageal varices. **a** Endoscopic view with the band ligation device loaded on the endoscope tip. The pull string (black arrow) for releasing bands (white arrows) is controlled by the endoscopist at the instrument head after the varix is suctioned into the device. **b** One of several banded varices is seen (arrow). Note one remaining band on the ligation adaptor. **c** Full deployment of bands. There is no remaining trip wire. Again, note banded varix (arrow)

and treatment schedules. In the United States, sodium morrhuate and sodium tetradecyl sulfate have been most widely used. Other commonly used sclerosing agents are ethanolamine aleate and polidocanol. Sclerosing agents are injected either directly into the varix or alongside the varix in a paravariceal fashion. As mentioned, a volume of 0.5–2 ml is administered per injection, beginning at the esophagogastric junction and progressing proximally for maximally 5 cm. In general, not more than 20–30 ml of sclerosing agent is injected per therapeutic session.

Post-procedure Management. Following completion of sclerotherapy or band ligation, patients are observed for several hours prior to resuming oral intake of liquids or food. Outpatients undergoing variceal therapy are not hospitalized unless procedure-related complications occur. Temporary dysphagia and odynophagia immediately after the procedure are common and may require analgesics and temporal use of a liquid diet.

Complications. Complications occurring during either sclerotherapy or band ligation include near-uniform development of local esophageal ulceration, side effects of sedation, aspiration, perforation, induction of bleeding, chest pain, and odynophagia. A severe local inflammatory response with subsequent development of mediastinitis and esophageal stricture sometimes occurs after sclerotherapy. In addition, rare systemic complications such as ARDS, bacteremia, and pericarditis may also occur after sclerotherapy.

Results

Control of Initial Bleeding. A meta-analysis of ten prospective trials using sclerotherapy for control of active bleeding within 24 h of admission showed that initial hemostasis was obtained in approximately 90% of patients. This result was significantly better than those achieved with medical therapy or balloon tamponade. Trends were also seen for improvements in rebleeding, acute mortality, and deaths directly related to bleeding in patients undergoing sclerotherapy [8]. However, long-term survival did not change. In eight randomized trials comparing sclerotherapy with band ligation, both techniques controlled initial bleeding in 86%–89% of patients [9–16]. Both techniques also appeared to reduce the rebleeding rate following initial hemostasis without significant reductions in short-term mortality. A meta-analysis of eight controlled trials comparing long-term sclerotherapy with noninvasive, medical therapy showed a significant reduction of recurrent bleeding in patients undergoing sclerotherapy (30%–50% versus 70%–80%) [17]. A meta-analysis of seven prospective randomized studies comparing band ligation with sclerotherapy for long-term efficacy demonstrated a statistically significant reduction of rebleeding in the band ligation group, whether from varices or from treatment-induced ulcers [18]. This same meta-analysis showed a reduction in mortality using band ligation.

Complications. For the eight prospective randomized studies comparing sclerotherapy with band ligation, the overall complication rate was 24% for sclerotherapy and 5% for band ligation.

MALLORY-WEISS TEAR

Indications. Treatment is indicated only during a major upper gastrointestinal hemorrhage, or with continued active bleeding.

Contraindications. Hemodynamic instability, inability to obtain informed consent, an uncooperative patient, and severe coagulopathy are contraindications for treatment.

Technique. Injection therapy, coagulation therapy, band ligation and clipping of a visible vessel within a Mallory-Weiss tear are effective in treating these tears.

Complications. The complications of endoscopic therapy are sedation complications, pulmonary aspiration, perforation, and bleeding from treatment-induced ulceration.

Results. There are few data from prospective or randomized trials regarding endoscopic hemostasis of Mallory-Weiss lesions. It appears that the group of patients most likely to benefit has active ongoing arterial bleeding. Two studies demonstrated that active arterial bleeding from Mallory-Weiss tears can be treated endoscopically. Significant benefits over standard medical therapy include lower rates of continued bleeding and emergency surgery and fewer transfusions [19, 20].

Stomach

GASTRIC VARICES

Indications and Contraindications. The indications and contraindications for gastric varices are the same as for esophageal varices.

Technique. Injection of standard sclerosants or cyanoacrylate glue is performed using an injection needle. Injections are performed at the cardia and largest trunks. With standard sclerosants, the volume used per injection is higher than that for esophageal varices [21]. Cyanoacrylate is injected in aliquots of 0.5–1 ml. Recently, endoscopic band ligation and detachable snares (endoloops) have also been utilized [22, 23].

Complications. The complications of treating gastric varices are sedation-related symptoms, pulmonary aspiration, and induction of bleeding. Delayed bleeding as a result of injection ulcers may be life threatening.

Results. The control of gastric variceal bleeding using endoscopic methods is better if the varices are contiguous with esophageal varices rather than isolated gastric varices. Case series suggest that cyanoacrylate is effective in achieving acute hemostasis and ultimate variceal obliteration in patients with bleeding gastric varices [24, 25]. Injection of thrombin may hold promise for the future [26]. Prospective randomized studies of these endoscopic techniques have not yet been published. A preliminary report suggests that band ligation and detachable snares are also effective in treating gastric varices [22].

GASTRIC AND DUODENAL ULCERS

Since the management of peptic ulcers involving the stomach and duodenum is similar, the endoscopic treatment data will be presented together, with the exception of posterior, bleeding duodenal ulcers. Therapy of these ulcers will be discussed separately below.

Indications. Upper gastrointestinal bleeding should prompt upper endoscopy if the procedure is likely to lead to a change in management of the patient. The timing of the

procedure will depend on severity of bleeding. Certain clinical features are known to be associated with an increased risk of poor outcome related to bleeding. Patients with the following features should undergo emergent endoscopy to determine if the lesion is treatable: (a) development of an upper gastrointestinal hemorrhage in a patient already hospitalized for another severe medical or surgical problem; (b) bleeding in patients with severe co-morbid medical or surgical illnesses; (c) vomiting of red blood or passage of red blood via a nasogastric suction tube; (d) persistent hematochezia; (e) persistent hypotension or shock; (f) transfusion requirements of 6 or

more units of blood within 24 h; and (g) rebleeding in the hospital [4].

Contraindications. Contraindications to emergency upper endoscopy include suspected gastrointestinal perforation and an uncooperative patient. Relative contraindications to endoscopic hemostasis of nonvariceal lesions are irreversible, severe coagulopathy, especially low platelet counts, and abnormal platelet function.

Technique. Ulcer appearance at the time of endoscopy allows the endoscopist to determine the treatment regime

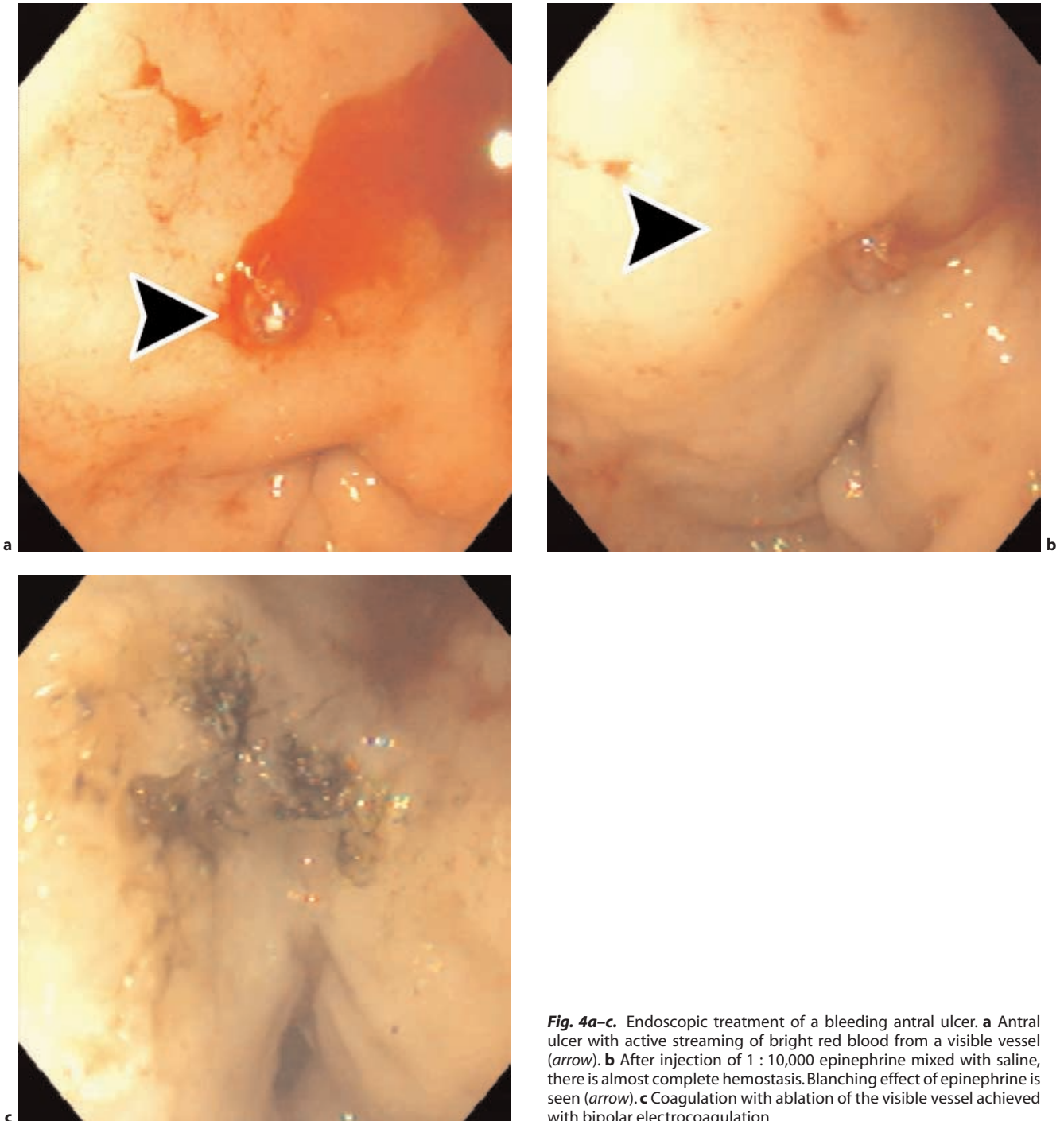


Fig. 4a–c. Endoscopic treatment of a bleeding antral ulcer. **a** Antral ulcer with active streaming of bright red blood from a visible vessel (arrow). **b** After injection of 1 : 10,000 epinephrine mixed with saline, there is almost complete hemostasis. Blanching effect of epinephrine is seen (arrow). **c** Coagulation with ablation of the visible vessel achieved with bipolar electrocoagulation

to be used. Clean-based ulcers without any other findings are generally not treated endoscopically, because the risk of rebleeding is estimated to be less than 3%. Actively bleeding lesions and ulcers with a nonbleeding visible vessel have high rates of continued bleeding or rebleeding and therefore warrant endoscopic therapy [27]. Recent evidence suggests that a densely adherent clot should be removed using a “cold” (i.e., without use of cautery) snare. This so-called guillotine technique permits inspection of the underlying lesion and treatment as needed [27]. Most endoscopists will treat actively bleeding lesions with an initial injection of dilute epinephrine followed by a thermal technique such as bipolar electrocoagulation (Fig. 4) [4].

Injection Technique. Epinephrine, commonly diluted in saline, is injected in four quadrants around the lesion and then centrally using 1- to 3-ml aliquots for up to a total of 20 ml.

Bipolar Electrocautery. Probes with large diameters appear to have a greater efficacy than smaller probes. Large probes, however, require a therapeutic endoscope with a channel of at least 3.7 mm. The tip of the probe is firmly pressed against the bleeding point or visible vessel for 5–10 s until the bleeding stops or the visible vessel is flattened.

Heater Probe. The technique used is similar to that of bipolar electrocautery.

Post-procedure Management. Continued medical care with hemodynamic monitoring is indicated for patients with a moderate to high risk of rebleeding. Early refeeding does not adversely affect the outcome for patients with endoscopic features, suggesting a low-risk of rebleeding. Administration of high-dose proton-pump inhibitors may reduce the risk of rebleeding, and aspirin or nonsteroidal anti-inflammatory drugs should be withheld. Treatment of *Helicobacter pylori* infection, when present, should be initiated.

Complications. The complications of endoscopic therapy for bleeding gastric and duodenal ulcers are perforation, adverse effects of sedation, aspiration, and induction of bleeding in a nonbleeding lesion.

Results. A prospective, randomized trial in patients with ulcer-related gastrointestinal bleeding compared the efficacy of medical therapy, bipolar cauterization, and heater probe application. Inclusion criteria included actively (spurting) bleeding ulcers or ulcers with visible vessels. Control of bleeding was seen in 14% of medically treated patients, in 93% of patients treated with bipolar cauterization, and in 95% of patients after heater probe treatment [28, 29]. Significant reductions in the frequency of recurrent bleeding, overall complications, blood transfusion requirements, and emergency surgery were seen in the group treated with the heater probe. A second study compared injection of dilute epinephrine without coagulation with medical therapy only for oozing and spurting ulcers. Patients treated with dilute epinephrine injection demonstrated a significant reduction in the need for surgery and in blood transfusion requirements [30]. A recent randomized study compared heater probe treatment with argon beam plasma coagulation in 41 patients with major stigmata of ulcer hemorrhage. No significant differences were found in the rates of initial hemostasis, recurrent bleeding, 30-day mortality, and the need for emergency surgery [31].

DIEULAFOY LESIONS

Indications and Contraindications. The indications for and contraindications to treatment of Dieulafoy lesions are the same as those for gastric ulcers.

Technique. The endoscopic techniques for injection, bipolar cauterization, and heater probe are the same as those for bleeding gastric ulcers, with the exception that injection therapy of epinephrine alone is not considered adequate. Additionally, mechanical methods such as endoscopic band ligation and application of hemoclips are ideal for Dieulafoy lesions, because of the presence of a visible vessel without an ulcer crater.

Results. Randomized trials have not been performed to determine the optimal treatment option regimen for Dieulafoy lesions, but permanent hemostasis can be achieved endoscopically in over 90% of patients using sclerosant injection therapy, coagulation therapy, hemoclips, endoloops, or band ligation [32].

GASTRIC ANTRAL VASCULAR ECTASIA (GAVE), OR WATERMELON STOMACH

Indications. Indications for treatment of GAVE, or watermelon stomach, are clinical upper gastrointestinal bleeding and refractory iron-deficiency anemia.

Contraindications. Contraindications to endoscopic therapy are severe coagulopathy and suspected bowel perforation.

Technique. The goal is to coagulate all visible lesions using the heater probe, bipolar coagulation, or laser (Fig. 5). A relatively high amount of energy is applied. Recently, we have used argon beam plasma coagulation with an argon flow rate of approximately 1 l/min and energy of between 65 and 90 watts. Sweeping motions are made while slowly withdrawing the gastroscope from the prepyloric area towards the gastric body.

Post-procedure Management. Complete avoidance of all aspirin and nonsteroidal anti-inflammatory drugs is recommended. Iron supplementation is prescribed to correct iron-deficiency anemia.

Complications. The complications of endoscopic management of watermelon stomach are perforation and bleeding.

Results. The optimal treatment modality is unknown. Excellent results have been obtained using endoscopic photo-coagulation with Nd:YAG laser, heater probe therapy, and bipolar electrocauterization, as well as argon plasma coagulation. However, small numbers of patients have been included in these studies [33]. In general, multiple endoscopic treatment sessions are required due to the extensive nature of the disease. The end points of therapy are near complete obliteration of endoscopic disease and clinical response in the form of cessation of bleeding or resolution of anemia.

PORTAL HYPERTENSIVE GASTROPATHY

There are no endoscopic treatment options for portal hypertensive gastropathy due to the extensive and diffuse nature of the condition.

CAMERON-HIGGINS LESIONS

Indications. The main indication is severe clinical upper gastrointestinal bleeding.

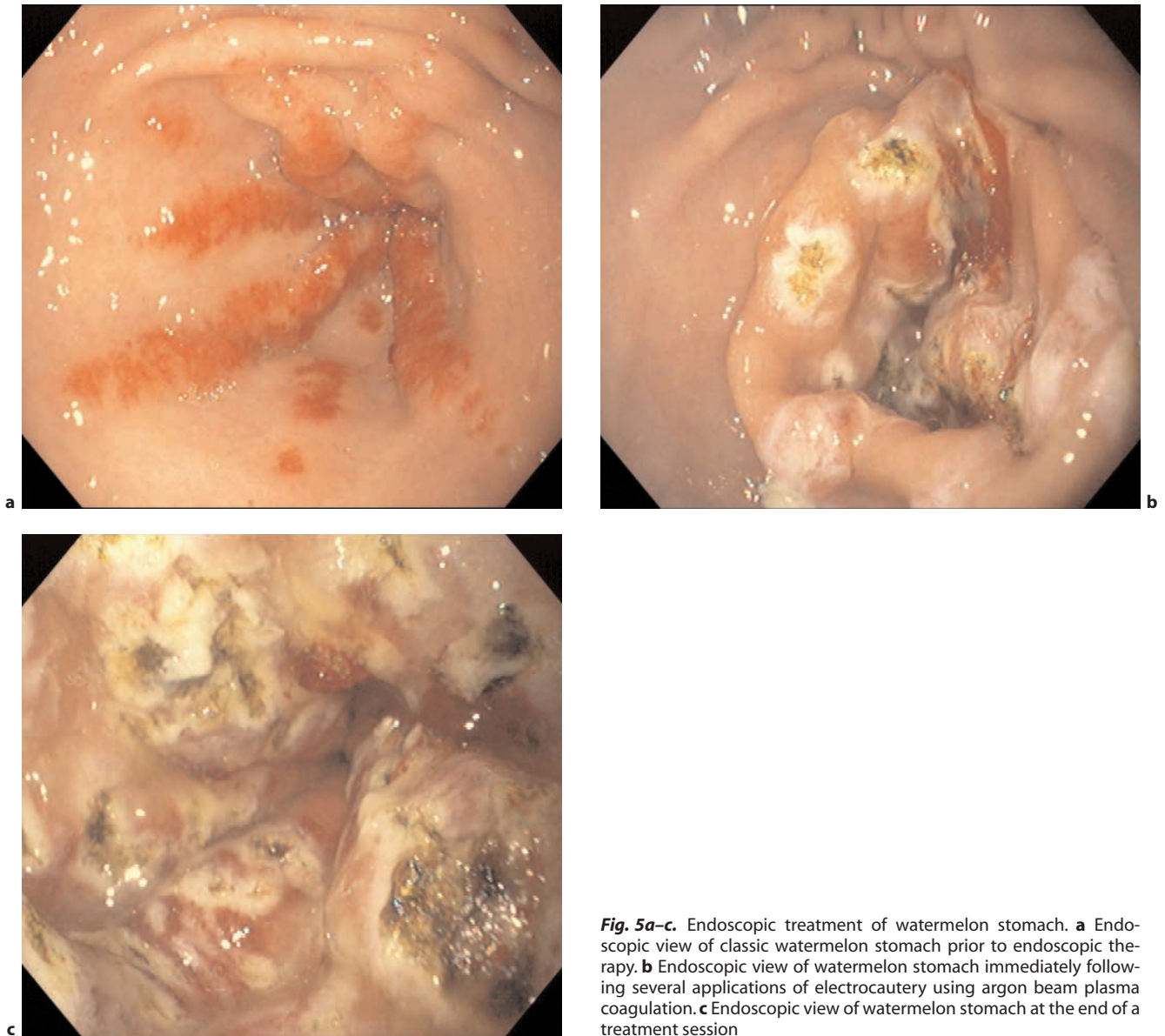


Fig. 5a–c. Endoscopic treatment of watermelon stomach. **a** Endoscopic view of classic watermelon stomach prior to endoscopic therapy. **b** Endoscopic view of watermelon stomach immediately following several applications of electrocautery using argon beam plasma coagulation. **c** Endoscopic view of watermelon stomach at the end of a treatment session

Contraindications. Contraindications to endoscopic therapy are severe coagulopathy and suspected bowel perforation.

Technique. The endoscopic technique for treating Cameron-Higgins lesions is identical to the technique used for bleeding gastric ulcers.

Post-procedure Management. Proton-pump inhibitors and iron supplementation are administered following endoscopic treatment.

Complications. Gastric perforation is the only real complication of endoscopic therapy for these lesions.

Results. There are no reports describing results of endoscopic treatment of Cameron-Higgins lesions.

ARTERIOVENOUS MALFORMATIONS (AVM) OR ANGIOMATA Arteriovenous malformations (AVM) or angiomata may involve the entire upper gastrointestinal tract, including the

stomach. Indications, contraindications, techniques, post-procedure management, complications, and results are independent of the location of the lesion. Therefore, this section applies to stomach and duodenal as well as small bowel AVM.

Indications. The indications for treating AVM are clinical evidence of gastrointestinal hemorrhage and refractory iron-deficiency anemia.

Contraindications. Contraindications include severe uncorrectable coagulopathy, hemodynamic instability, and suspected perforation.

Technique. Thermal methods such as heater probe, bipolar electrocoagulation, argon plasma coagulation, and laser are all effective for ablating angiomata. However, a significantly lower amount of energy is required for coagulation of AVM than for coagulation of bleeding ulcers. Thus, lower amounts of energy are applied per lesion. In addition, excessive dis-

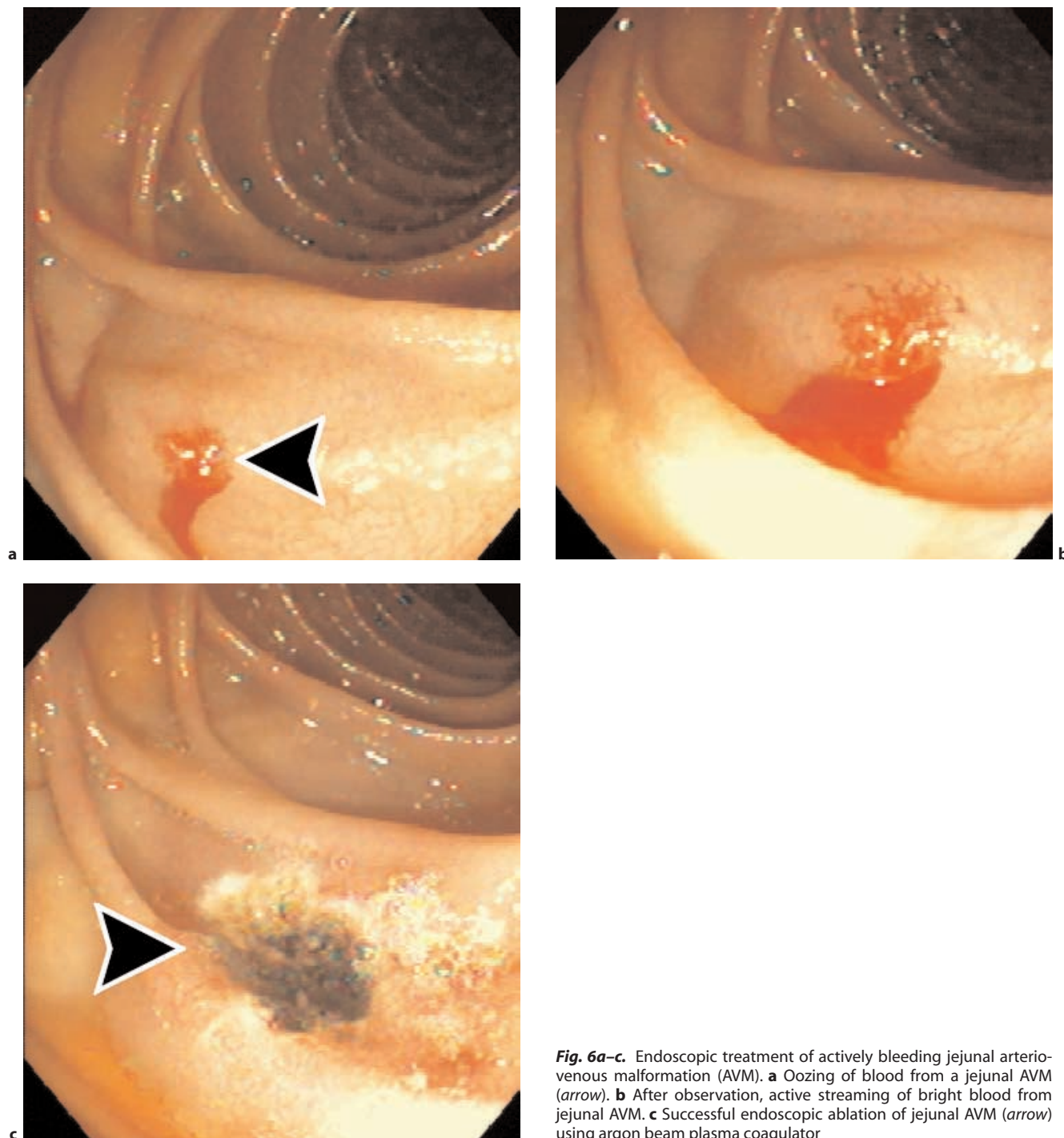


Fig. 6a–c. Endoscopic treatment of actively bleeding jejunal arteriovenous malformation (AVM). **a** Oozing of blood from a jejunal AVM (arrow). **b** After observation, active streaming of bright blood from jejunal AVM. **c** Successful endoscopic ablation of jejunal AVM (arrow) using argon beam plasma coagulator

tention of the gut should be avoided, particularly when lesions within the small bowel are treated. Coagulation of the entire malformation, from the center to the periphery of the lesion, is the goal of treatment (Fig. 6).

Post-procedure Management. Aspirin and nonsteroidal anti-inflammatory drugs are withheld, and iron supplementation is prescribed as needed.

Complications. Complications of endoscopic therapy are perforation, bleeding (immediate or delayed), ulceration, and pulmonary aspiration.

Results. There are no reported randomized controlled trials of endoscopic hemostasis for bleeding AVM. The largest reported prospective study of endoscopic treatment for bleeding upper gastrointestinal AVM used argon laser, bipolar coagulation, or heater probe treatment [4]. No major complications were reported in a study that compared medical with endoscopic treatment over 2.5 years. Results demonstrated that patients with documented upper gastrointestinal bleeding from AVM had good palliation with endoscopic treatment. Significant increases in hematocrit and decreases in transfusion requirements and hospitalization for gastrointestinal bleeding were also reported in these pa-

tients. The study allowed comparison only between heater probe and bipolar coagulation: Both were safe and equally effective. In another study, long-term results of Nd:YAG laser treatment for AVM showed significant reductions in bleeding episodes and blood transfusion requirements [34].

Duodenum

UNCOMPLICATED ULCERS

The regular, uncomplicated duodenal ulcer is treated in a fashion similar to that for gastric ulcers (see above).

POSTERIOR WALL DUODENAL BULB ULCERS

These ulcers are frequently more difficult to treat endoscopically because of their inaccessibility and source of bleeding, which stems from the large gastroduodenal artery. The tangential location of the lesion to the operating channel makes effective treatment difficult or at times impossible.

Indications, Contraindications, Technique. See Stomach, section Gastric and Duodenal Ulcers.

Post-procedure Management. If bleeding cannot be controlled by endoscopic measures, arteriography with embolization is an effective nonsurgical alternative [35]. High-dose proton-pump inhibitors may reduce the risk of rebleeding. Treatment of *H. pylori* infection, if present, is indicated. In patients who have had recurrent life-threatening bleeding, who require long-term anticoagulation or nonsteroidal use, or who would not tolerate recurrent bleeding because of severe underlying medical conditions, life-long administration of H_2 receptor antagonists or proton-pump inhibitors may be used to reduce the risk of recurrent bleeding.

Results. A study performed to evaluate clinical or endoscopic predictors of endoscopic treatment failure for bleeding peptic ulcers showed that failure was significantly related to the ulcer location (specifically the posterior or superior wall of the duodenal bulb) [36]. A second study demonstrated that ulcer size greater than 2 cm and the presence of shock were significant predictors of endoscopic treatment failure [37].

AORTODUODENAL FISTULA

Indications. The prime indication is massive, life-threatening upper gastrointestinal bleeding.

Contraindications. See Stomach, above.

Technique. There is no effective endoscopic therapy for aortoduodenal fistulae. Endoscopy is utilized primarily as a diagnostic adjunct to other modalities such as abdominal CT and angiography. It is also used to rule out other sources of bleeding [38].

ARTERIOVENOUS MALFORMATIONS OR ANGIOMATA

The indications, contraindications, techniques and results are described above.

Small Bowel

Treatment of small bowel lesions are limited by the length of the endoscope. Using colonoscopes or dedicated small bowel enteroscopes, lesions as far as the mid-jejunum can be identified and treated.

ULCERS

Indications. The indications for endoscopic treatment of small intestinal ulceration are gastrointestinal bleeding and anemia not attributable to another source.

Contraindications. Contraindications to endoscopic treatment are suspected perforation and hemodynamic instability.

Technique. The principles and types of therapy for treating bleeding small intestinal ulcers are similar to those for ulcers in the stomach and duodenum. Epinephrine injection is recommended for initial hemostasis. Since the small bowel wall is thin, thermal injury could result in perforation, and coagulation must thus be used with caution. Argon beam plasma coagulation, with its limited depth of injury and ability to treat lesions tangentially, appears to be ideally suited for the treatment of small bowel ulcers.

Post-procedure Management. Nonsteroidal anti-inflammatory drugs should be avoided.

Complications. The main complications are bowel perforation and induction of bleeding.

Results. There are no reports comparing the results of various endoscopic techniques for the treatment of small bowel ulcers.

ARTERIOVENOUS MALFORMATIONS OR ANGIOMATA

The indications, contraindications, techniques, and results are described above.

ISCHEMIA

Ischemia of the small bowel may be recognized endoscopically.

Indications and Contraindications. Clinical gastrointestinal bleeding is the indication for endoscopic treatment. The contraindication is hemodynamic instability.

Technique. Few if any descriptions of endoscopic treatment of bleeding from small bowel ischemia have been reported. Injection therapy or clipping of a visible vessel may be effective for initial hemostasis.

Post-procedure Management. Post-treatment management consists in correction of the underlying causes, if possible.

Complications. Complications of endoscopic therapy in this setting include perforation and bleeding.

Results. There are no data regarding the outcome of endoscopic therapy in this setting. The overall outcome likely depends on the underlying ischemic insult.

VASCULITIS

Any lesions due to vasculitis that require endoscopic treatment are approached as described above for ischemic lesions of the small bowel.

Colon

Treatment of colonic lesions is more difficult than treatment of upper gastrointestinal lesions because of the inability to

clear the colon of stool and blood during a bleeding episode. Rapid lavage with nonabsorbable solutions may allow good endoscopic visualization to identify and treat these lesions.

DIVERTICULA

Indications. Diverticular bleeding is treated endoscopically when lower gastrointestinal bleeding has not ceased with supportive care and transfusion of blood products.

Contraindications. Contraindications to endoscopic therapy are suspected perforation, hemodynamic instability, and severe uncorrectable coagulopathy.

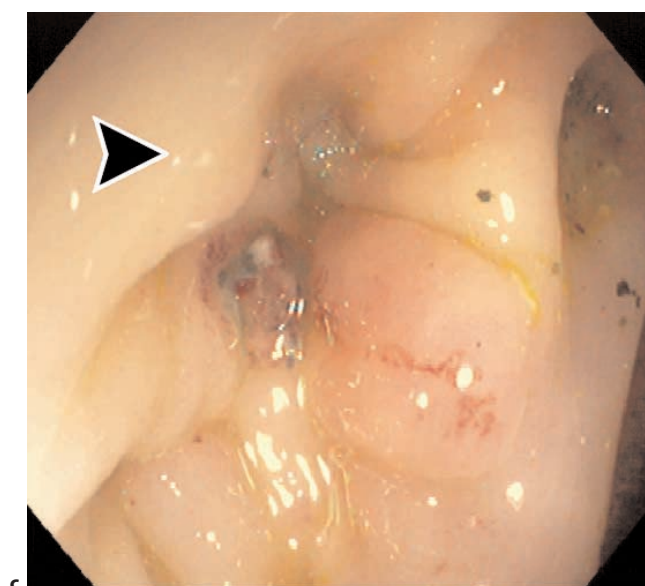
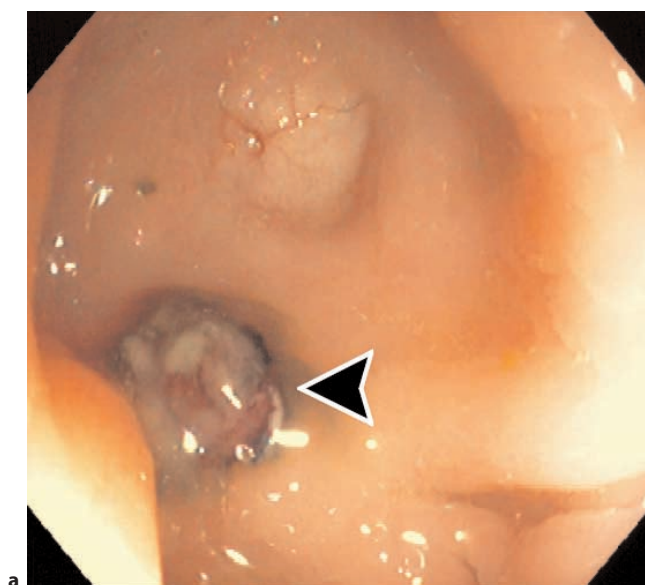
Technique. Urgent colonoscopy is performed after the large bowel has been cleared with a nonabsorbable lavage solution. All diverticula, if present, are carefully inspected for signs of recent hemorrhage. Identification of an actively bleeding diverticulum or visible vessel prompts endoscopic therapy. Injection therapy and bipolar coagulation as used for treatment of gastric ulcer bleeding has recently been described (Fig. 7) [39]. However, due

to the thin wall of the colon, less energy is used during coagulation in the colon than in the upper gastrointestinal tract.

Complications. Complications of endoscopic therapy within the colon are perforation and treatment-induced bleeding and ulceration.

Post-procedure Management. The patient is transfused with blood products as needed and monitored for signs of re-bleeding. Clear liquids may be initiated early following the procedure.

Fig. 7a-d. Endoscopic treatment of bleeding colonic diverticulum. **a** Endoscopic view of sigmoid colonic diverticulum containing densely adherent clot/visible vessel (arrow). **b** Injection catheter seen during injection of 1 : 10,000 epinephrine/saline. **c** Endoscopic appearance following epinephrine injection. Note blanching effect adjacent to visible vessel (arrow). **d** Complete ablation of visible vessel (arrow) following electrocoagulation with the argon beam plasma coagulator



Results. Only one published study has critically evaluated endoscopic therapy in patients with definite signs of bleeding diverticula (visible vessel, active bleeding, or adherent clot). In this study, all patients received conservative management with colonoscopy within 6–12 h after hospitalization or the diagnosis of hematochezia. The first 73 patients received surgical treatment (hemicolectomy) if diverticular bleeding persisted. The subsequent 48 patients received endoscopic therapy (epinephrine injection, bipolar coagulation, or both) if required [39]. In the first group (no endoscopic therapy), nearly one third of patients who were treated eventually required surgery. In the second group (endoscopic therapy), surgery was prevented in all cases.

ARTERIOVENOUS MALFORMATIONS OR ANGIOMATA

Indications. The indications for treatment of colonic AVMs are lower gastrointestinal bleeding or refractory iron-deficiency anemia.

Contraindications. The contraindications are similar to those for treatment of colonic diverticular bleeding; see above.

Technique. Ablative thermal therapies are used, as described for treatment of AVM in the upper gastrointestinal tract; see above.

Post-procedure Management. Management includes blood transfusion and iron supplementation therapy as needed and avoidance of aspirin and nonsteroidal anti-inflammatory drugs.

Complications. The main complications are bowel perforation and gastrointestinal bleeding.

Results. Limited studies suggest that endoscopic ablation of colonic AVM is effective for both acute and chronic lower gastrointestinal bleeding with a low complication rate [40, 41].

RADIATION PROCTITIS

Indications. The indications for treatment of radiation proctitis are recurrent, severe rectal bleeding or chronic rectal bleeding leading to refractory iron-deficiency anemia.

Contraindications. The contraindications to therapy are the same as to the treatment of colonic AVM.

Technique. Multiple thermal modalities effectively treat radiation proctitis. Laser therapy, bipolar therapy, and most recently argon beam plasma coagulation are used in a manner similar to that for watermelon stomach [42, 43]. All visible lesions are coagulated using a proximal-to-distal approach. Treatment is performed to produce a white coagulum while avoiding cavitation or charring of tissue.

Post-procedure Management. Iron supplementation therapy is administered when indicated, and nonsteroidal anti-inflammatory drugs are avoided. The administration of analgesics for post-procedure discomfort may be necessary.

Complications. Deep ulcers following treatment may occur and cause significant bleeding. Because the thickened rectum is the primary target of treatment, perforation is uncommon but may occur. Development of rectovaginal fistulae in women has been reported following treatment of radiation proctitis. Transmural necrosis and fibrosis with perforation

or stricture formation may occur more commonly after Nd:YAG laser treatment.

Results. Up to 90% of patients treated endoscopically will respond with a decrease in frequency and severity of rectal bleeding and improvement in anemia. Multiple sessions are usually required to achieve clinical success.

INTERNAL HEMORRHOIDS

Indications and Contraindication. The indications for internal hemorrhoid treatment are problematic rectal bleeding, with or without anemia, prolapsing tissue, fullness after defecation, and pain. A contraindication is suspected bowel perforation.

Technique. The treatment choices for internal hemorrhoids include infrared coagulation, radiofrequency coagulation, electrocoagulation, rubber band ligation, sclerotherapy, cryosurgery, regular surgery, and laser surgery [44]. The most commonly employed method currently is band ligation. The endoscopic technique of band ligation of internal hemorrhoids is identical to the technique used for treatment of esophageal varices. A flexible upper endoscope or sigmoidoscope is used to retroflex within the rectum. At least one band is placed on each column of hemorrhoids beginning just above the dentate line [45].

Post-procedure Management. Independent of the treatment modality used, the goal of postoperative management is to keep patients' stools soft by providing a high-bulk diet and ample amounts of fluids. Discomfort, if present, is usually treated satisfactorily with nonsteroidal anti-inflammatory drugs. Sitz baths may alleviate symptoms as well.

Complications. Injection therapy has been associated with perirectal infections and impotence. Ulcer formation may occur with all treatment modalities.

Results. A meta-analysis of 18 randomized, controlled trials assessed two or more treatment modalities for symptomatic hemorrhoids. Overall, band ligation was effective in up to 95% of patients. In addition, band ligation was judged to be superior to other modalities for grades 1–3 hemorrhoids in terms of efficacy and complication frequency [46].

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