chapter 85  Vertebrobasilar Aneurysms – Midbasilar, Vertebrobasilar Junction and AICA
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INTRODUCTION

Posterior circulation aneurysms account for 5–20% of all aneurysms in both surgical and autopsy series.1-7 Aneurysms located at the vertebrobasilar junction (vertebral confluens), along the midbasilar trunk, and related to the anterior inferior cerebellar artery (AICA) constitute only 20–25% of posterior circulation lesions, thus making them exceedingly rare. Because of their location within the posterior fossa and the fact that few surgeons have experience with them, treatment can be intimidating. The complex nature of such lesions is compounded by the difficulty of exposure, cramped working space, frequent need for brain stem retraction to visualize the aneurysm neck and surrounding vessels, and intimate relationship of the aneurysm to brain stem perforators that may arise from the vertebral and basilar arteries. By adapting known operative exposures, modifying them slightly when the need arises, using careful microsurgical technique, and incorporating recent neuroradiologic interventional advances in selected cases, these aneurysms can be treated successfully.

SURGICAL AIMS

The annual rupture risk of an unruptured aneurysm lies somewhere between 0.5 and 4%.8-18 These numbers vary based upon aneurysm size, location, shape, a patient’s associated medical problems and the population being studied. Once an aneurysm has ruptured the risk of rerupture is approximately 4% in the first 48 h and 1.4% for each of the next 13 d. The 6 month risk is 50% after which the rupture rate falls back to the baseline of 0.5–4% per year. The goal, therefore, of aneurysm therapy is to obliterate the lesion before it ruptures or has a chance to rerupture. This is especially important in view of the fact that each bleeding episode carries a 60–75% incidence of death or disability, especially with posterior circulation lesions.18

Any therapy aimed at treating intracranial aneurysms has as its goals:

- Aneurysm obliteration.
- Preservation of afferent and efferent vessels.
- Minimal disruption of normal surrounding neural tissue so as to preserve neurologic function.

When these goals cannot be met, the surgeon needs to modify therapy so that the aneurysm is treated in a manner that improves the patient’s overall expected outcome in terms of disease natural history, while at the same time neurologic function does not significantly degrade or create a condition created that precludes future recovery.

PREOPERATIVE CONSIDERATIONS

PREOPERATIVE TESTING

The type of preoperative evaluation depends upon a patient’s mode of presentation and the urgency with which treatment needs to be undertaken. Patients with uncomplicated, elective aneurysms undergo primary or repeat angiography if necessary, and computed tomography (CT) and magnetic resonance imaging (MR) scanning. They are medically cleared for surgery and are given a thorough neurologic examination. CT and MRI with or without CT and MR angiography and three-dimensional CT reconstructions can help better localize the lesion relative to surrounding bony and neural structures, and provide vital information concerning the presence of aneurysm calcification and intraneurysmal thrombus.19-22 This is especially true when dealing with giant lesions where the aneurysm mass makes angiographic identification of the aneurysm neck and its relationship with surrounding vessels difficult. If the surgeon feels that localizing the lesion reliably straight forward, no other tests are obtained.

When the aneurysm is more complicated either because of its shape, location, size, neck/fundus anatomy and wall consistency, additional testing may be warranted. If the surgeon is concerned that aneurysm trapping may become necessary, then angiography with basilar or vertebral temporary balloon occlusion may help determine whether or not such treatment is tenable. Angiography can also assist the physician in deciding whether or not dural venous sinus sacrifice is possible, should additional aneurysm exposure be required. For those rare patients who need to be placed on the cardiac bypass unit (see later section for indications), consultation with cardiothoracic surgeons, preoperative echocardiography, and lower extremity arterial and venous Doppler examination is mandatory, to assure that the patient is a good candidate for such intervention, has favorable cardiac function and anatomy, and has the necessary venous and arterial access to complete the procedure.

Some individuals present with neurologic deficits either due to hemorrhage, perianeurysmal perforator vessel
thrombosis, or aneurysm mass effect. Preoperative consultation with an otolaryngologist can help determine the degree, if any, of lower cranial nerve dysfunction. This information is helpful when patients and their families are being counseled as to the urgency of surgery, as well as what to expect postoperatively concerning temporary or permanent tracheostomy and alternate routes of alimentation. Hearing and facial function can also be evaluated in case these cranial nerves are injured during the surgical procedure, leading to the necessity for hearing augmentation and facial reanimation.

INFORMED CONSENT

The risks of surgery aimed at treating midbasilar and confluenes aneurysm include, but are not exclusive to, death, stroke, cranial nerve and brain stem injury, procedure failure, infection, development of hydrocephalus, and recurrence of the original disease process despite what was felt to be a successful operation. While facial weakness, hearing loss and diploria are certainly possible sequelae of such surgery, they are rare. More commonly, patients develop ninth and tenth cranial nerve dysfunction with dysphagia and risk for aspiration. Because of the high likelihood of transient and possibly permanent lower cranial nerve dysfunction, patients and families are counseled about tracheostomy and gastrostomy insertion prior to surgery.

In addition to discussing the risks of surgery, the natural history of the disease process is elaborated upon, and patients are always given the option of choosing not to have treatment or to have alternative treatments. For instance, if endovascular interventional therapies are available for a particular disease process for which surgery also has a role, these are discussed with the patient, and the patient either chooses which therapy to pursue or is informed why one treatment is more suitable than another.

By taking the time to discuss the disease process, procedures, alternatives, and potential outcomes in as calm and clear a fashion as possible, the surgeon can embark upon surgery knowing that every opportunity has been given to the patient and family to participate rationally in the decision-making process and live with the consequences of such a decision.

OPERATIVE TECHNIQUE

SURGICAL ANATOMY

The safe and effective approach and treatment of intracerebral aneurysms is based upon vascular, bony, neural and cisternal anatomy. By thoroughly understanding the relationship each of these have with one another, the surgeon can make appropriate decisions regarding how to best manage such vascular anomalies.

Vascular anatomy

The paired vertebral arteries exit the foramen transversarium of the atlas, wrap around the atlanto-occipital joint, penetrate the atlanto-occipital membrane and enter the posterior fossa through the foramen magnum. Approximately 80% of left vertebral arteries are dominant. Each vertebral artery courses anterolateral to the medulla, ventral to the hypoglossal nerve roots and medial to the accessory nerve. At the level of the hypoglossal nerve, the posterior inferior cerebellar artery (PICA), the largest vertebral artery branch, arises. This vessel may, however, originate in 8% of cases extracranially. The PICA originates from the vertebral artery at the anterolateral aspect of the medulla at the level of the inferior olive. At this level it is initially in close proximity to the 12th and then ninth and tenth cranial nerves. Distal to the PICA origin, the vertebral arteries run medially and join in the midline between the two abducens nerves at the level of the pontomedullary sulcus, to form the vertebral confluenes. They then continue on as the basilar artery.

The basilar artery begins at the vertebral confluenes and runs superiorly in the preptontine cistern within a space demarcated by the lateral margins of the clivus and dorsum sellae. This vessel averages 3 cm in length and 1.5–4 mm in width.23 As the vessel courses along the ventral pontine surface it gives rise to paramedian perforators that supply deep medial structures and circumferential perforators that supply the lateral pons. The largest basilar trunk branch is the anterior inferior cerebellar artery (AICA), which generally arises from the lower third of the basilar trunk as a solitary or less commonly duplicated vessel. In rare cases the AICA may arise from the upper basilar trunk (4%), midbasilar trunk (2%), or vertebral artery (1%).24 Its size is often inversely related to that of the ipsilateral PICA and, in 20% of cases, an accessory artery may supplement or replace part of the normal AICA distribution.25 The AICA courses around the pons from the preptontine cistern to the cerebellomedullary cistern below or between the facial and auditory/vestibular nerves. This vessel supplies the seventh and eighth cranial nerves, the inferolateral pons, middle cerebellar peduncle, flocculus and petrosal surface of the cerebellar hemisphere. The AICA divides into a lateral branch that runs around the flocculus and into the horizontal fissure between the superior and inferior semicircular lobules, and a medial branch that courses medially and inferiorly to supply the biventral lobule.23

Regional muscular and bony anatomy

Surgical approaches to midbasilar and vertebral confluenes aneurysms require thorough knowledge of the muscular and bony anatomy of the subocciput and petrous region. In order to develop adequate exposure of the posterior fossa vasculature, lower cranial nerves and brain stem. Adequate mobilization of the numerous muscles encountered in the suboccipital region is a prerequisite part of any attack on aneurysms of the posterior fossa. Suitable muscle reflection allows the surgeon to perform the bony resection necessary for subsequent vessel visualization.
dissection and mobilization. If muscle dissection is not adequate, the entire exposure will be jeopardized.

The most superficial muscles overlying the subocciput in the retroauricular region are the posterior auricular muscle and the occipital belly of the occipitofrontalis muscle. These muscles insert along the occipital bone's nuchal line. More caudally and laterally the sternocleidomastoid muscle inserts on the mastoid process and takes its origin from the sternum and clavicle. Medial and deep to the sternocleidomastoid lie the splenius capitis and semispinalis capitis muscles. The former attaches to the mastoid bone and nuchal line deep and caudal to the sternocleidomastoid, and takes its origin from the cervical spinous processes. The latter attaches to the occipital bone at the nuchal line deep to the splenius capitus and takes its origin from the thoracic transverse processes.

Beneath the suboccipital muscles lie the temporal and occipital bones. The mastoid and petrous portions of the temporal bone lie laterally and the occipital bone lies medially. In the adult, the mastoid generally contains air spaces, the mastoid air cells, which communicate with the middle ear by way of the mastoid antrum. The petrous portion is shaped like a medially directed pyramid. It contains the inner ear and contributes to the boundaries of the middle ear. The apex of the petrous segment is directed forward and medially between the sphenoid and occipital bones. The more lateral and rostral aspect of the petrous bone contains the middle and inner ear, the descending segment of the facial nerve and the sigmoid sinus. An important landmark located near the lateral mastoid is the asterion, which marks the junction of the lambdoid, occipitomastoid and parietomastoid sutures. This confluence of sutures marks the point beneath which the transverse and sigmoid sinuses join.

The lower portion of the skull is formed by the occipital bone, which is arranged around the foramen magnum. This space contains the medulla oblongata, cerebellar tonsils, spinal roots of the accessory nerve, sympathetic plexuses, and the vertebral, anterior and posterior spinal arteries. The squamous portion of the occipital bone extends from the base on to the back of the skull. The external occipital crest, to which the ligamentum nuchae is attached, extends from the foramen magnum to the external occipital protuberance. The nuchal lines extend laterally and delineate areas of muscular insertion. The lateral occipital bone contains the occipital condyles at the sides of the foramen magnum.

**Posterior fossa neural anatomy**

As will be discussed later, the approach to midbasilar and vertebral confluens is a lateral one. Upon opening the dura, the surgeon will encounter, from rostral to caudal, the quadrangular, simple, superior semilunar and inferior semilunar cerebellar lobules. After the simple and superior semilunar lobule has been retracted, the flocculus will come into view. Once the flocculus is retracted laterally, the foramen of Luschka and 4th ventricle choroid plexus will be seen. Displacement of the cerebellum provides a view of the cranial nerves. Rostrally, near the junction of the tentorium and petrous ridge, the surgeon can locate the fourth and fifth cranial nerves. The fourth cranial nerve tends to run along the edge of the tentorium, wandering above and below this dural structure along with the rostral division of the superior cerebellar artery. The fifth cranial nerve exits the pons and travels out towards Meckel's cave. It is usually necessary to divide the superior petrosal vein (Dandy's vein) in order to fully visualize the trigeminal nerve. The sixth cranial nerve exits the pontomedullary sulcus medially. The seventh and eighth cranial nerves are located just below the flocculus, exiting the lateral pontomedullary sulcus at the level of the middle cerebellar peduncle. More caudally are the IX–XI cranial nerves emerging from the medulla just lateral to the tuberculum cinereum. At this same level, the twelfth cranial nerve exits from the anterolateral sulcus medial to the olive.

**Cisternal anatomy**

Up to five cerebrospinal fluid (CSF) compartments need to be opened en route to treating midbasilar, AICA and confluens aneurysms. These are the cerebellopontine, preoptine, premedullary, cerebellomedullary and magna cisterns. The cerebellopontine cistern lies between the anterolateral surface of the pons and cerebellum, and the arachnoid membrane, that covers the posterior surface of the petrous bone. Within it lies the lateral pons, flocculus, V–VIII cranial nerves, the lateral superior cerebellar artery (SCA) and the lateral AICA. The preoptine cistern lies between the arachnoid membrane covering the clivus and the anterior pontine surface. Within it lies the basilar artery and proximal AICA. The premedullary cistern lies between the anterior medullary surface and the clivus. Superiorly it borders the preoptine cistern, and laterally it borders the cerebellomedullary cistern. It contains the ventral and ventrolateral medulla, pyramids, olive, the XII cranial nerve, vertebral confluens, distal vertebral artery and premedullary segment of PICA. The cerebellomedullary cistern lies lateral to the premedullary cistern and caudal to the cerebellopontine cistern. It contains the lateral medulla, tuberculum cinereum, rhomboid lip and biventral lobule of the cerebellum, lateral medullary PICA segment, and IX–XI cranial nerves. The cisterna magna lies dorsal to the vermis and medulla. Its posterior wall is composed of arachnoid along the inner wall of the occipital bone rostral to the foramen magnum. It contains the medulla, PICA, cerebellar tonsils and the caudal cerebellar vermis.

**PATIENT POSITIONING**

Our approach to midbasilar and vertebral confluens aneurysms starts with the patient's head in the lateral position. The bridge of the nose is positioned parallel with the floor and the neck is flexed until two finger breadth
can fit between the chin tip and the chest. Flexion of the neck brings the brain stem into a more parallel orientation, with the surgeon making retraction of the cerebellum and exposure of the ventral and lateral pontine and medullary surfaces easier.

When possible we prefer to have the patient positioned in the lateral decubitus position with the surgical side up. The bottom arm is well padded with foam, slung and placed in the space made by the Mayfield head holder arms, and secured with tape to the table. The patient's shoulders, torso and buttocks are secured to the table with tape so that the table can be rotated during the operation to provide the surgeon with different views of the surgical site. It is important to pad all pressure points with foam, and to be sure that the elbow and popliteal fossa are well protected, so that peripheral nerve neuropraxic injuries do not develop secondary to compression during long operations.

Patient’s with long flexible necks are positioned supine with the neck rotated 90° to the appropriate side. The supine position keeps the patient's shoulders flat with the table and out of the way of the surgeon's right arm in right-sided approaches, and left arm during left-sided approaches. This is especially useful when, in an attempt to look up towards the incisura with the microscope, the surgeon has to position himself caudally. In such situations, the patient's shoulder can get in the way of the surgeon's right or left arm, if the patient is in the full lateral decubitus position. While this makes dissection with that arm difficult, it especially hampers efforts at using that hand to apply clips or place sutures for vessel anastomoses. The supine head turned position requires a supple neck with good range of motion. Rotation can often be facilitated by placing a small shoulder roll beneath the contralateral problem with this position include kinking of the jugular veins, carotid and vertebral arteries, brachial plexus traction, and spinal cord compression in patients with osteophytic or disc disease.

**EXPOSURE**

Numerous approaches have been described to expose the midbasilar and vertebral confluens region.24-30 Two main approaches centered about the subocciput are used most frequently to treat the vast majority of aneurysms located within this region of the posterior fossa.26 The first is retrosigmoid suboccipital and the second is presigmoid suboccipital with or without sigmoid sinus and/or superior petrosal sinus exposure and sacrifice.

**Choosing the side of approach**

We base our approach to vertebral confluens, midbasilar and AICA aneurysms on ease of access to the aneurysm's neck, and the afferent and efferent vessels. For most of these lesions working space is cramped and it is necessary to approach the lesion from the side to which it projects. While this means the surgeon will be facing the aneurysm fundus, it also means he or she will have visibility of and access to the neck. Limitations in operating space, visibility and ability to displace vessels make it difficult to approach these aneurysms from the side contralateral to the aneurysm fundus, because access to the aneurysm's neck from this approach is compromised.

**Muscle exposure**

Muscle exposure and dissection are identical for both retrosigmoid and presigmoid approaches (Fig. 85.1A). A curvilinear incision is made from a point approximately 4 cm above the pinna to a point 7 cm below the mastoid, with the apex of the curve lying approximately two-thirds of the way along the nuchal line. A cutaneous flap is elevated medially to laterally until the rim of the external auditory canal can be palpated through the fascia of the posterior auricular muscle. A sponge roll is placed beneath the flap and fish-hook retractors are used to keep the flap reflected laterally. A linear incision is made with monopolar cautery laterally to medially just beneath the insertions of the sternocleidomastoid, splenius capitis and semispinalis capitis muscles on the nuchal line and mastoid. A second incision is made (leaving a small cuff of tissue to sew back to later) along the lateral border of the sternocleidomastoid muscle to release it from the mastoid. If the muscles are dissected in a subperiosteal fashion with the monopolar cautery, they can be reflected inferiorly and medially, secured with a fish-hook retractor, and subsequently displaced away from the surgical field of view. A vertical incision is now made rostrally from the nuchal line into the occipitofrontalis muscle. The lateral portion of the occipitofrontalis muscle and the posterior auricular muscle are then reflected subperiosteally in a lateral direction, and the medial portion of the occipitofrontalis muscle is reflected medially. This stellated incision involving the muscles of the subocciput provides excellent exposure of the skull from the posterior temporal and inferior parieto-occipital region to the foramen magnum (Fig. 85.1B). The muscles are reflected in directions that clear the operative field, and fascia is adequately preserved so that an anatomic muscle attachment reconstruction is easily achieved at the end of the procedure (Fig. 85.1C).

While exposure of the extracranial vertebral artery is generally not necessary when vertebral confluens and midbasilar aneurysms are involved, the surgeon may on occasion desire preliminary proximal control of the ipsilateral vertebral artery prior to dural opening. Such control is easily achieved by exposing the muscle as described above, simply by extending the dissection inferiorly. After the sternocleidomastoid, splenius capitis and semispinalis capitis muscles have been reflected caudally and laterally, the obliquus capitis superior and obliquus capitis inferior muscles will come into view. The former originates on the transverse process of the atlas and inserts on the occipital bone between the superior and inferior nuchal lines. The
latter originates from the apex of the spine of the axis and inserts on the transverse process of the atlas. The vertebral artery can be palpated between these two muscles in what is called the vertebral triangle. If the oblique muscles are removed from the C1 transverse process and reflected medially, the vertebral venous plexus and underlying vertebral artery can be exposed as it exits the C1 foramen transversarium to run along the top of the C1 ring before penetrating the posterior fossa dura. A useful landmark for identifying the artery is palpation of a sharp edge along the C1 ring. This edge represents the margin of a trough in which the vertebral artery rests as it runs horizontally along the atlas. The ring can be exposed by simply rubbing it with a periosteal until the bone becomes evident. Venous bleeding from the vertebral plexus can be controlled using bipolar cautery and packing.

**Bone exposure**

The extent of the bony resection hinges on the surgical approach, be it retrosigmoid, presigmoid/petrosigmoid or retrosigmoid/petrosigmoid/transpetrosal. Each of these approaches has at its core a retrosigmoid exposure with extended mastoid, and temporo/parieto/occipital drilling to achieve the presigmoid and transpetrosal exposures; this allows easy transition from one approach to the other (Fig. 85.2).

While exposure of the retrosigmoid region can be performed via a craniectomy or craniotomy, we prefer a craniectomy with the use of a drill to thin the bone, rapidly followed by a variety of rongeurs to remove the thin inner layer of cortical bone in order to expose the posterior fossa dura and transverse and sigmoid dural sinus edges. The retrosigmoid exposure begins with identification of the asterion which marks the confluence of the lambdoidal, occipitomastoid and parietomastoid sutures. Beneath this point lies the junction between the transverse and sigmoid sinuses. Once the sinus junction has been localized, the surgeon can rapidly uncover the dura and sinuses with minimal risk of damage to the large venous channels. The small amount of inevitable sinus bleeding can be controlled readily with topical hemostatic agents and cotton pledges. It is important to expose the edges of the transverse and sigmoid sinuses fully, so that when the dura is opened the lateral aspect of the cerebellar hemisphere is exposed, thus reducing the need for cerebellar retraction, and improving the view of the cerebellopontine angle and brainstem. Bony exposure is carried caudally along the edge of the sigmoid sinus until the jugular bulb is identified and partially uncovered. This requires aggressive drilling of the medial mastoid and the occipital bone, located posterior and lateral to the occipital condyle. The foramen magnum is ultimately opened and its lip removed. Such caudal and lateral exposure allows the surgeon to see and get access to the vertebral artery as it enters the dura through the dural ring (Fig. 85.2).

If additional exposure is required, the surgeon should
remove bone lateral to the sigmoid sinus (presigmoid). If the surgeon elects to approach the lesion from a retrosigmoid trajectory, the additional bone removal lateral to the sigmoid sinus allows the sinus to be tilted upward and laterally once the dura is opened. Even if the sinus is not sacrificed, this tilting maneuver can often provide precious additional millimeters of viewing and working space. If a more ventral approach is desired, the dura is opened lateral to the sigmoid sinus, thus providing a presigmoid trajectory. In cases where sinus sacrifice is physiologically possible, the sinus can be ligated and reflected laterally, thus permitting the surgeon to work through both the pre-and postsigmoid spaces. This latter exposure reduces the need for cerebellar retraction to an absolute minimum, and again provides the surgeon with a more ventrolateral view of the brain stem, vertebrobasilar system and any associated aneurysm.

At times the surgeon may need to expose the vertebrobasilar system from the intradural penetration point of the vertebral arteries to the basilar apex. The transpetrosal/presigmoid approach is an excellent means for doing so. The bony opening is that described above, although a portion of the temporo/parieto/occipital bone above the transverse sinus must also be removed. Working rostral and lateral to the transverse sinus and transverse/sigmoid junction, the surgeon exposes the sinodural angle. This represents the point where the tentorium joins the transverse/sigmoid sinus along the petrous bone, and marks the confluence between the transverse/sigmoid sinus and the superior petrosal sinus. By opening the temporal dura above the sinodural angle, opening the presigmoid dura and then connecting the two openings after ligating the superior petrosal sinus with hemoclip or suture ligatures, the surgeon can expose the lateral edge of the tentorium cerebelli. With this exposure the posterior temporal lobe is gently elevated; special attention should be made to preservation of the temporal lobe veins joining the transverse and sigmoid sinuses. One of these vessels may be the vein of Labbe, loss of which can lead to venous infarction of the posterior temporal lobe and inferior parietal lobule. Sequential cauterization and incision of the tentorium laterally to medially eventually leads to complete division of the tentorium through the incisura. Once this has been
completed, the surgeon has a lateral view of the cerebral peduncle. Once presigmoid dural opening from tentorium to foramen magnum has been made, the vertebrobasilar system can be seen from basilar apex to intradural origin of the vertebral arteries. It also gives the surgeon complete access to the ventrolateral brain stem and the III–XII cranial nerves.

**Intradural aneurysm exposure and clipping**

Cerebellar relaxation is achieved by opening the arachnoid encasing the cisterna magna sharply, and gently allowing CSF to escape the subarachnoid space. Once the cerebellum is slack, proximal control of the aneurysm becomes a priority. For vertebral confluens, midbasilar and AICA aneurysms, proximal control is most readily and safely achieved by identifying the ipsilateral and contralateral vertebral arteries. Elevation of the ipsilateral cerebellar tonsil generally provides a view of the eleventh and twelfth cranial nerves. The vertebral artery can usually be located within the premedullary cistern just medial to the eleventh cranial nerve, where the origin of PICA is found where the twelfth cranial nerve crosses the vertebral artery. Once the ipsilateral vertebral artery is isolated, more medial dissection across the ventral medulla provides access to the contralateral vessel. When the two vertebral arteries have been identified and isolated, temporary clips of appropriate lengths are selected and placed aside for quick insertion, should proximal control of blood flow be required.

If the vertebral arteries are followed through the premedullary cistern and into the preoptic cistern, the vertebral confluentes can be identified. It is located just medial to the sixth cranial nerve and the distal portion of AICA. For vertebral confluentes aneurysms, the surgeon will want to gain exposure and control of the basilar artery prior to approaching the confluentes aneurysm itself. This is achieved by sharply opening the arachnoid that covers and invests the V–X cranial nerves. This provides access to the cerebellar alar and flocculus which, once elevated and retracted laterally, allow the surgeon to see the brain stem and midline vascular structures better. Sectioning of the arachnoid also eliminates the risk that the forces of cerebellar retraction will be transmitted to the seventh and eighth cranial nerves. Such forces can damage these sensitive nerves leading to postoperative dysfunction, despite a lack of noticeable direct mechanical trauma. Working medial to VI–VIII cranial nerves and opening the remainder of the preoptic cistern, the surgeon can then identify the proximal basilar artery. Dissection about its circumference ensures that a temporary clip can be placed across its lumen, should distal vascular control become necessary. For AICA and midbasilar aneurysms, control of the more distal basilar artery may be preferable. This can be achieved by identifying the basilar artery medial to the fifth cranial nerve. By angling the microscope in a caudal to rostral direction and looking towards the incisura, the surgeon will see the upper basilar trunk, basilar quadridification and third cranial nerve. This is easier if a presigmoid transpetrosal and transtentorial approach is used, although such an approach is not always required.

Once proximal and distal control are established, the surgeon can approach the aneurysm using the same basic rules that apply to all aneurysm clippings. The proximal and distal vessels are followed until the proximal and distal aneurysm neck is identified. Sharp dissection with microscissor and arachnoidal knife is the preferred means of dissecting the aneurysm neck, as it limits the chance that a broad hole will be placed in the aneurysm by tugging on surrounding arachnoid, adherent vessels or clot. Even when thick subarachnoid hemorrhage is present, the surgeon can safely identify the aneurysm neck by first identifying afferent and efferent vessels and then hugging the surface of these vessels with scissor and suction until the neck is visualized. Once the neck is found, final dissection can be made safer and easier by the placement of temporary clips on the afferent vessel or vessels. This will reduce the turgor within the aneurysm, making manipulation of the neck and fundus safer, as perforating and efferent arteries are dissected free and the neck is completely identified. At times complete trapping of the aneurysm is necessary: clips are placed on all afferent and efferent vessels. This is especially valid if the aneurysm ruptures prior to complete neck dissection, or if the aneurysm is so large that it is necessary to puncture and deflate it prior to placing the aneurysm clip or clips.\(^{30–34}\) Partially thrombosed aneurysms or lesions with significant atheroma will often need to be completely trapped so that they can have thrombus and atheroma removed by curettes and/or the ultrasonic aspirator. Often such removal is a prerequisite for successful clipping.

**USE OF CARDIAC BYPASS**

In those cases where we feel a particular aneurysm will require more than 20 min of local circulatory arrest and associated cerebral ischemia, we consider performing the operation with hypothermic cardiac standstill.\(^ {33,35}\) The use of extracorporeal circulation is beneficial in such cases because it the surgeon can then cool the patient to 15–18°C. With body temperature reduced, metabolic activity is slowed, and release of posts ischemic excitatory neurotransmitters is reduced. As a result of these mechanisms, the body and brain's energy needs are temporarily attenuated. Patients may then have their surgery completed under low flow states in which perfusion persists, but at low pressures or at no flow states, during which the pump is stopped and flow through the cerebral circulation is arrested. It appears that the safe duration for flow interruption is 45–50 min, after which the pump is restarted and the patient is rewarmed.
Employment of extracorporeal circulation is not risk free. Complications include thrombophlebitis and vessel dissection secondary to femoral cannulations, difficulty with hemostasis, cardiac instability and fluid shifting. For this and other reasons we use the bypass pump for those rare cases that will require prolonged periods of flow arrest. An example would be a giant, partially thrombosed and calcified midbasilar aneurysm which requires aneurysmorrhaphy and lumen reconstruction.

CLOSURE
The dura is closed in a 'watertight' fashion, and if any large gaps exist they are patched using small pieces of muscle. The mastoid bone and air cells are completely covered with bone wax to avoid postoperative CSF leaks. If the bone is not well waxed, CSF will migrate through the air cells, enter the middle ear and gain access to the oropharynx via the eustachian tube. Such leaks will present as rhinorrhea or oropharyngeal drip. The sub-occipital bony defect is repaired with a methylmethacrylate cranioplasty that is secured with two or three screwed miniplates. The cranioplasty serves a cosmetic purpose, while also reducing the incidence of postoperative headaches by lessening the likelihood of muscle/dural adhesions. Miniplates eliminate the risk of postoperative cranioplasty migration. Once the bony defect is repaired, the muscle is reapproximated. A suction drain is placed in the subgaleal space and brought out through a separate stab incision. The subcutaneous tissue is closed by galeal interrupted sutures, and the skin is closed with suture or staples. Sterile dressings are applied and the patient is transferred to the intensive care unit for postoperative care.

ANESTHETIC CONSIDERATIONS
The principal anesthetic management goals for aneurysm surgery are prevention of intraoperative rupture or rebleeding, and protection against cerebral ischemia. Other goals include brain relaxation, continued management of the patient’s ongoing medical problems, and rapid recovery from anesthesia for a timely postoperative neurologic evaluation.

PREOPERATIVE EVALUATION
A thorough preoperative evaluation must be made with particular attention focused on the patient’s neurologic, cardiovascular and pulmonary status. Some common disease processes associated with ruptured aneurysms are hypertension, arrhythmias, myocardial ischemia, aspiration pneumonia, pulmonary edema, electrolyte imbalances, and diabetes mellitus.

INTRAOPERATIVE MANAGEMENT
Premedication
The choice of premedication is guided by the patient's preoperative neurologic status and anxiety level. In patients with unruptured aneurysms and those with Hunt and Hess grades I or II, premedication with a benzodiazepine anxiolytic (e.g. midazolam 1–2 mg i.v.) is reasonable. Patients with Hunt and Hess grades III or higher are not further sedated for fear of depressing respiration and altering consciousness further. When in doubt, premedication is withheld. Following sedation blood pressure (BP), respiratory rate and oxygenation are carefully monitored. Prophylaxis against pulmonary aspiration (ranitidine 150 mg p.o. or 50 mg i.v.; metoclopramide 10 mg p.o. or i.v.) are usually given.

Monitors
Routine intraoperative monitoring for aneurysm clipping includes non-invasive BP device, five-lead EKG, pulse oximeter, esophageal stethoscope, temperature probe, Foley catheter, capnograph, peripheral nerve stimulator, arterial catheter and central venous catheter. A precordial Doppler and a multiport central venous catheter are used for detection and possible treatment of venous air embolism, should the patient be placed in the sitting position. A pulmonary artery catheter is warranted in patients with congestive heart failure or decreased left ventricular function, and in those who are being treated for vasospasm.

Induction
The primary anesthetic goal at induction is minimization of the risk of intraoperative aneurysm rupture. Sudden increases in systemic arterial BP and sudden decreases in intracranial pressure (ICP) must be avoided. The incidence of aneurysm rupture during induction and tracheal intubation has been reported as 1–2% with mortality approaching 75%. Prolonged periods of hypotension are also avoided, as they may lower cerebral perfusion in patients with increased ICP.

Induction proceeds with gradual increase in anesthetic depth. After monitors are placed, the awake patient is denitrogenated with 100% oxygen by facemask. (Denitrogenation is omitted if the patient is already intubated and ventilated.) Induction is accomplished with propofol (1–2 mg/kg i.v.). Etomidate (0.2–0.5 mg/kg i.v.) may be administered should the patient have any cardiac risk or be hemodynamically unstable. A small dose of sufentanil (10 μg i.v.) is given initially, followed by a low dose infusion of alfentanil (0.25–0.5 μg/kg/min) or remifentanil (0.125 μg/kg/min) to blunt the hemodynamic response to laryngoscopy. Muscle paralysis is provided by one of many non-depolarizing muscle relaxants with stable cardiovascular profile such as rocuronium (0.8 mg/kg i.v.), vecuronium (0.1 mg/kg i.v.), pipercuronium (0.07 mg/kg i.v.) or cisatracurium (0.2 mg/kg i.v.). The patient is then hyperventilated with oxygen, nitrous oxide (N₂O) and isoﬂurane (1 MAC or less), until intubation. Boluses of short-acting opioids such as alfentanil and remifentanil are used to temporarily increase the anesthetic depth, in order to prevent the hypertensive
response during laryngoscopy and intubation. Alternatively, short acting beta-adrenergic antagonists, such as esmolol (10 mg increments i.v.), or vasodilators, such as nitroprusside or nitroglycerin (50 μg increments i.v.), are also effective in blunting the sympathetic response. Longer acting agents or high concentration inhalational anesthetics are avoided, because they can lead to prolonged periods of hypotension after the transient sympathetic response has subsided.

Maintenance
A balanced anesthetic technique using oxygen, N₂O, isoflurane, opioid and a non-depolarizing muscle relaxant is used. Hemodynamic goals are to avoid wide swings in BP and to control ICP at periods of intense stimulation, which include head pinning, skin incision, craniotomy, dural incision and skin closure. Short-acting opioids will allow for rapid deepening of anesthesia. Local anesthetic infiltration before head pinning and skin incision can reduce the hemodynamic response.

Other goals are directed at providing optimal surgical conditions and protection against brain ischemia. A relaxed brain is needed for maximal exposure of the surgical site and to minimize brain-retraction pressure.¹ Diuresis, hyperventilation and placing the patient in the head-up position (if appropriate) are employed. Diuresis is established with mannitol (0.5–1 mg/kg i.v.) given approximately 30 min prior to dural incision. A rapid infusion of mannitol may cause a transient but significant reduction in systemic vascular resistance and BP, and can produce acute volume overload in patients with impaired cardiac function. Anticipation of potential complications and immediate treatment of hemodynamic changes are warranted in these patients. Furosemide, which does not lead to transient increase in intravascular volume, can be substituted in these patients. Significant fluid and electrolyte abnormalities can occur; therefore, volume status and electrolyte values are closely monitored and treated appropriately. Hyperventilation is aimed at maintaining the Paco₂ at 25–30 mmHg.

Intraoperative fluid administration is guided by the patient’s maintenance requirement, blood loss and urine output. Since patients are on aggressive diuresis, the urine output is not a good indicator of their volume status. The central venous pressure and pulmonary capillary wedge pressure followed as trends, and other hemodynamic parameters such as BP and heart rate, provide better guides of the intravascular volume. Iso-osmolar crystalloid and colloid can be used to replenish fluid loss. Hypo-osmolar solutions are avoided to prevent cerebral edema.³ Glucose-containing solutions are also not used because hyperglycemia may potentiate brain injury following brain ischemia.³⁰,⁴⁰

Temporary occlusion and cerebral protection
Frequently, temporary arterial occlusion is used to facilitate aneurysm clipping. Such steps, however, can create ischemia in territories distal to the temporary clips. Many different methods are empirically used for ‘ischemic brain protection’. Unfortunately, no randomized clinical trials have been done to test them systematically. Pentothal (5–15 mg/kg i.v.),⁴ etomidate (1 mg/kg i.v.),⁴² and propofol (100–300 μg/kg/min i.v.),⁴¹ have all been administered during periods of temporary occlusion. Even isoflurane and halothane have been tried.⁴³⁻⁴⁵ The end-point is indicated by the burst suppression pattern on the EEG monitor. Many animal studies have shown that barbiturates⁴⁶ can reduce the magnitude of neurologic damage, but the protective effects of propofol and etomidate are still questionable.⁴⁵,⁴⁶ With the dose used for burst suppression, etomidate confers the most hemodynamic stability and causes the least hypotension of all three drugs. Pentothal causes the longest period of sedation and sometimes requires the patient to be on overnight ventilation. Propofol causes the least postoperative sedation. Inhalational anesthetics have been shown to offer no protection.⁴⁵,⁴⁸,⁴⁹

Mild to moderate hypothermia has demonstrated protection, but the protective value of hypothermia is not proportional to cerebral metabolic rate (CMR) depression.⁵⁰,⁵¹ The theory of CMR depression and brain protection is therefore questioned, since the effects of hypothermia cannot be completely explained by this hypothesis. An alternative hypothesis proposes that cerebral protection from hypothermia may be conferred by preventing the release of neuroexcitatory transmitters triggered by ischemia.⁵²⁻⁵⁵

At our institution, all the methods discussed above are combined into a ‘formula’ that is empirically employed during temporary occlusion. Cooling the patient to 32 or 33°C by using cooling blankets is instituted immediately after induction. Glucose is carefully monitored and kept between 80 and 120 mg/dl. When the neurosurgeon requests burst suppression prior to placing temporary clips, several additional steps are taken. Mannitol (0.25 g/dl) is given as a free radical scavenger. N₂O is discontinued, the patient is ventilated with 100% O₂, and a hematocrit of at least 30% is maintained for optimal oxygen delivery. The patient is kept normotensive and normovolemic. The Paco₂ is normalized from 25–30 to 35–40 mmHg to decrease cerebral vasoconstriction. Etomidate, propofol or low dose pentothal (according to the dosages above) is given until a burst suppression pattern is noted on the EEG monitor. The choice of agent depends on the patient’s cardiovascular status and physician’s personal preference. When the temporary aneurysm clips have been applied, the patient’s BP is raised 20% above baseline to increase collateral flow. This increase in BP can be accomplished by decreasing the concentration of the inhalational anesthetic agent or by administering a vasopressor. After temporary occlusion is completed, burst suppression is terminated and anesthetic maintenance is resumed as before.
EMERGENCE
The main goal for emergence is to allow for a smooth and rapid wake-up, thus facilitating neurologic assessment. Coughing and straining are avoided. Emergence hypertension can cause bleeding at the surgical site and cerebral edema. BP can be controlled with rapidly titratable agents such as nitroprusside, nitroglycerin, esmolol or labetalol. Patients with poor preoperative grades, those that were given a large amount of medications intraoperatively for burst suppression, those suspected of having brain stem injury and lower cranial nerve damage, or those with cardiovascular instability may require sedation and continued ventilatory support.

INTRAOPERATIVE NEUROPHYSIOLOGICAL MONITORING
We feel that it is important to monitor brain stem and cranial nerve function while the surgeon is operating within the posterior fossa. By continuously assessing eighth cranial nerve integrity with brain stem auditory evoked potentials (BAEP), the surgeon can monitor hearing preservation and brain stem function simultaneously. In addition to recording the five eighth cranial nerve Jewett waves for latency and amplitude, we also monitor electromyographic data from the seventh cranial nerve and its innervated muscles. Such monitoring helps us during both the bone resection portion of the operation (especially when we are working presigmoid) and the intracranial dissection portion. While it is also possible to record IX–XII cranial nerves EMGs by placing electrodes in the stylopharyngeus, cricothyroid, trapezius and tongue muscles respectively, we do not routinely use such data during our procedures.

POSTOPERATIVE CARE
Postoperative management of patients with vertebral confluens, midbasilar and AICA aneurysms depends to some extent upon whether or not the patient has suffered a subarachnoid hemorrhage (SAH). If the patient has had a recent hemorrhage, management needs to focus on reducing the incidence and treatment of seizures, vasospasm and hydrocephalus. Whether or not the patient has bled, postoperative management of posterior fossa aneurysms focuses on the effects of surgery on the cranial nerves. Patients may suffer diplopia from traction on the third, fourth and sixth cranial nerves, facial numbness from trauma to the fifth cranial nerve, facial weakness secondary to seventh cranial nerve damage, hearing loss from traction on the eighth cranial nerve, dysphagia from damage to the ninth and tenth cranial nerves, and tongue weakness from twelfth cranial nerve trauma. The most dangerous of these nerve injuries is damage to the ninth and tenth cranial nerve, which puts the patient at risk for immediate or delayed aspiration. Interestingly, many patients will have symmetric vocal cords during the first few postoperative days, because of cord swelling from prolonged operative intubation. Therefore, accurate evaluation of the ninth and tenth cranial nerve function may not be possible until several days following surgery. As a result, patients should not be fed orally until a full and accurate swallowing evaluation can be carried out. This includes evaluation of the cords by an otolaryngologist, and swallowing studies by a speech pathologist. The latter may include modified barium swallow and cine swallowing studies to assess a patient’s risk for aspiration. If there is any question in our minds that a patient is at risk for aspiration, we tend to favor tracheostomy and surgical feeding tube insertion. Such procedures are easily reversible when the patient’s cranial nerves recover and provide some protection from aspiration in the interim.

SPECIAL CONSIDERATIONS
INTERVENTIONAL NEURORADIOLOGIC TREATMENTS
In recent years, new transcatheter techniques have evolved for the treatment of intracranial aneurysms. The most successful of these, and the one currently used by the majority of interventional surgeons, is the Guglielmi Detachable Coil (GDC); [Target Therapeutics, Fremont, CA, USA]. The GDC system provides the interventionalist with the ability to insert a coil into an aneurysm or blood vessel, assess its position, and withdraw it, if the result is less than satisfactory. Other coil systems are not detachable but rather pushed or injected into position. Once these coils leave the catheter, they are difficult if not impossible to retrieve.

The mechanism by which GDC coils occlude aneurysms is still debated. We have made observations at surgery on recently coiled aneurysms that lead us to question the theory that the positive charge within the aneurysm during electrolysis induces significant thrombus formation. Coils probably provide immediate protection against rehemorrhage by reducing blood flow within the aneurysm sac, buffering arterial pulsations within the fundus, and sealing the weak portion of the wall or hole. Eventually, organized thrombus does form within the aneurysm and the aneurysm is excluded from the parent vessel by the formation of an endothelialized layer of connective tissue that covers the neck’s ostium.

While the indications for GDC coils is continually expanding as interventionalists become more skillful in their placement, they tend to be most successful in cases of aneurysms with small necks or necks that are smaller in diameter than the maximal aneurysm diameter, and aneurysms without significant intradural thrombus. Nevertheless, decisions concerning their applications are made on a case-by-case basis, and few dogmatic rules exist.

In 1995 Fernando Vinuela (personal communication) reviewed the USA Multicenter GDC Study Group’s results
with 753 aneurysms treated in 715 patients. Complete occlusion of small aneurysms with small necks occurred in 62% of cases, while complete occlusion in small aneurysms with wide necks was 33%. Large aneurysms with small necks and giant aneurysms with thrombus each had a 37% occlusion rate, and giant aneurysms alone had 35% occlusion rates. Technical complications occurred in 11% of cases and included aneurysm perforation (1.5%), parent artery narrowing (0.5%), parent artery occlusion (3.8%), embolization (3.7%) and coil migration (1.1%). Complications that had permanent clinical implications, however, occurred in only 4.4% of the cases. The procedure related mortality rate was 1.12% and the overall mortality rate for the entire study population was 5.2%. The postembolization aneurysm hemorrhage rate was 1.26%. Aneurysm recanalization occurred in 7.7% of small aneurysms, 15% of large aneurysms, 29% of giant aneurysms, and 31% of giant/partially thrombosed lesions. Since 1994, results have improved as newer sized and less traumatic coils were introduced into the market. These advances allow for denser fundus packing and improved obliteration rates with reduction in delayed recanalization.

OTHER TREATMENT OPTIONS
Not all aneurysms of the vertebral confluentis, midbasilar segment and AICA origin are amenable to direct clipping or coiling of the neck, or clip reconstruction of a vascular lumen. These would include some aneurysms with atherosclerotic/calcified necks and fusiform lesions. In such cases, the surgeon should consider the option of parent vessel occlusion (Hunterian ligation), or aneurysm trapping with or without a bypass procedure. The former procedure has as its goal reduction of the pressure head to which the aneurysm is exposed, and possibly thrombosis of the aneurysm sac if one exists. The latter excludes the aneurysm from the circulation leading to thrombosis. Hunterian ligation does not provide the same degree of protection that complete trapping does, because the aneurysm in many cases will continue to fill in a retrograde fashion and thus be at risk for hemorrhage or growth. For those reasons we use proximal occlusion only in the most dire circumstances.

In cases of elective aneurysms that we feel cannot be directly clipped, patients are first evaluated with a temporary balloon occlusion (TBO) test. This involves inflation of a non-detachable silicon balloon in one or both vertebral arteries above the PICA or in the proximal basilar artery, depending upon the question being asked and the location of the aneurysm being treated. Patients are continuously evaluated while the balloon is inflated and, if at any time during a 15 min period of balloon inflation they have a change in their neurological condition, the balloon is deflated. During the balloon inflation the patient has CTC-91c injected intravenously so that on post-balloon deflation, a single photon emission computed tomogram (SPECT) of the brain can be performed to assess qualitatively the symmetry of cerebral blood flow (CBF) to the cerebellum and occipital lobes during vessel occlusion. Other centers use hypotensive challenge, EEG monitoring and xenon CBF analysis, together with TBO testing to assess a patient’s tolerance for vessel sacrifice.

If the above evaluations indicate that a patient is not a candidate for vessel sacrifice without a high risk for subsequent stroke, because they either fail the TBO test on clinical or blood flow criteria, then an arterial bypass procedure must be devised, if Hunterian ligation or trapping is to take place. Depending upon the aneurysm’s location, this may involve a direct end-to-side occipital artery or posterior superficial temporal artery-to-superior cerebellar or posterior cerebral artery (PCA) bypass, or an interposition radial artery or saphenous vein bypass from the external carotid artery to the SCA or PCA.

CONCLUSION
Posterior circulation aneurysms, because of their relative rarity, deep location, intimacy with cranial nerves and small vascular perforating vessels, can be among the most difficult cerebrovascular anomalies to treat. They can be approached in a systematic and controlled fashion, however, if the surgeon is aware of the regional anatomy and various techniques to gain exposure to the area and control of the involved vessels.

REFERENCES


