Minimally Invasive Neurosurgery for Vascular Lesions

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KEYWORDS

- Keyhole craniotomy • Aneurysm surgery • AVM
- Arteriovenous fistula • Cavernoma

Intracranial vascular lesions are known to affect 2% to 4% of the population, predisposing those affected to a lifetime risk of hemorrhagic stroke, ischemia, focal neurologic deficits, or epileptic seizures. These lesions constitute a heterogeneous group, with different lesion types characterized by distinct biologic mechanisms of pathogenesis and progression. In this article, the minimally invasive management of intracranial aneurysms, arteriovenous malformations (AVMs) including arteriovenous fistulas (AVFs), and cavernous malformations are discussed.

ANEURYSMS

The annual incidence of subarachnoid hemorrhage (SAH) from a ruptured intracranial aneurysm in the United States is approximately 1 case per 10,000 persons, yielding approximately 27,000 new cases of SAH each year.[1,2] Autopsy studies indicate a prevalence of intracranial aneurysms of between 1% and 5% in the adult population,[3] which translates to 10 million to 12 million persons in the United States.[1] SAH is more common in women than in men (2:1),[4] and its incidence increases with age, occurring most commonly between 40 and 60 years of age (mean age $\geq$50 years).[5,6] An estimated 5% to 15% of cases of stroke are related to ruptured intracranial aneurysms.[7]

The most common presentation of intracranial aneurysm is rupture leading to SAH. Given the increased availability of noninvasive imaging techniques, aneurysms are increasingly detected before rupture. An unruptured aneurysm may be asymptomatic and thus be found incidentally, or it may be diagnosed on the basis of symptoms.[8] Unruptured aneurysms may cause symptoms by exerting mass effect, leading to cranial nerve palsies (eg, the rapid onset of a third nerve palsy caused by the enlargement of an aneurysm of the posterior communicating artery[1]) or brainstem compression.[9] Aneurysms presenting with SAH tend to bleed again. Two percent to four percent of aneurysms hemorrhage again within the first 24 hours after the initial episode, and approximately 15% to 20% bleed a second time within the first 2 weeks.[10,11] Aneurysmal SAH has a 30-day mortality rate of 45%; an estimated 30% of survivors will have moderate to severe disabilities.[12] Aneurysm repair performed after an SAH is generally associated with higher mortality and morbidity rates than elective clipping in unruptured aneurysms.[13] Because of the high risk of rebleeding within the first week, increased treatment risks during the vasospasm period between the 4th and 14th day, and limited medical treatment options for vasospasm in patients with unsecured aneurysms, early treatment (surgical or endovascular) within the first 72 hours following SAH is generally recommended.[13–15] The risk for bleeding in nonruptured aneurysms depends on aneurysms' size and configuration, localization, and endogenous factors.[16–18] Presently, surgical or endovascular treatment of unruptured aneurysm is recommended in patients having suffered from an SAH caused by another aneurysm and patients bearing symptomatic and/or larger aneurysms (>12 mm). In young patients with positive endogenous factors and/or irregular aneurysm shape, treatment is recommended also in smaller aneurysms.

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Neurosurg Clin N Am 21 (2010) 673–689
doi:10.1016/j.nec.2010.07.011

Surgical Treatment

The goal of surgical treatment is to completely exclude the aneurysm from the circulation, without impairment of the cerebral perfusion. This treatment may be enabled by elective clipping or wrapping of the aneurysm or trapping of the affected area of the carrying vessel. The latter may need to be combined with a bypass to maintain sufficient cerebral perfusion. Surgical treatment may also be combined with endovascular techniques, such as stenting and coiling, and may be necessary after technically inadequate endovascular treatment.

Minimally invasive aspects of aneurysm surgery include methods for limiting the exposure of brain tissue using keyhole craniotomies for definitive treatment of ruptured and unruptured aneurysms, reduction of number of required procedures and craniotomies in patients with multiple aneurysms, and reduction of hospitalization time and reconvalescence.

Minimally invasive approaches suitable for aneurysm surgery are the eyebrow, pterional, subtemporal, retrosigmoid, and interhemispheric minicraniotomies (see the article by Garrett and colleagues elsewhere in this issue for further exploration of this topic).

Critical steps involved with the surgical approach to an intracranial aneurysm are sufficient visualization of the associated proximal and distal vessels, early control of the feeding vessel, understanding of the individual pathoanatomy and hemodynamics of the specific aneurysm being treated, dissection of the aneurysm neck, manipulation of the aneurysm dome, selection of appropriate instruments and implants for the definitive treatment, and intraoperative control of the result.

Technical Considerations

Important technical requirements for minimally invasive aneurysm surgery are a high-quality operating microscope that is capable of indocyanine green (ICG) angiography, endoscopes with 0° and 30° viewing angle, and micro-Doppler sonography with 1 and 2 mm probes. In more complex aneurysms, where temporary occlusion or even cardiac arrest is anticipated, neurophysiological monitoring (somatosensory evoked potential [SSEP], motor evoked potential [MEP]) should be performed. Customized clip design and clip appliers are also very helpful to gain sufficient overview in limited craniotomies (**Fig. 1**).

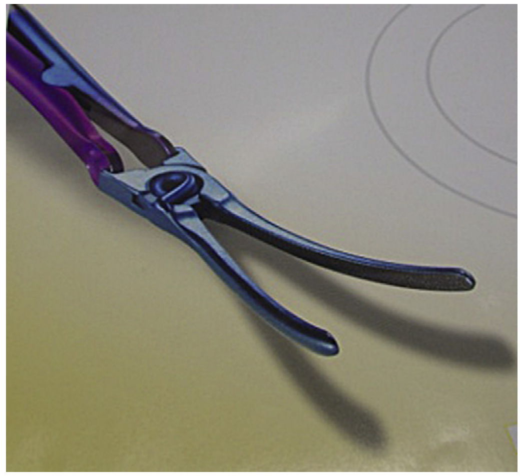

Fig. 1. Special clip applier for limited craniotomies, with fixation of the clip from inside leading to minimal obstruction of the operating field (Peter Lazic, Tuttlingen, Germany; with permission).

Consideration of all described aspects of minimally invasive aneurysm surgery in combination with proper use of intraoperative modern technology enables successful and safe treatment, with limited exposure in almost all patients with unruptured and ruptured aneurysms.

Specific Surgical Considerations and Illustrative Cases

Anterior circulation aneurysms

The vast majority (90%) of all intracranial aneurysms are located in the anterior circulation, specifically at the anterior communicating artery (AComA) in 30%, the internal carotid artery (ICA) including the posterior communicating artery (PComA) in 30%, and the middle cerebral artery (MCA) in 20%.[11]

The specific challenge for AComA aneurysms is for example the need for control of 2 proximal vessels, that is, the ipsilateral and contralateral A1 segments; for MCA aneurysms it is the high risk of subsequent stroke after temporary occlusion; and for some ICA aneurysms it is the difficulty with obtaining proximal control. However, even complex and large aneurysms may be managed safely through minimally invasive approaches. Preferred approaches for aneurysms of the anterior circulation are the supraorbital keyhole craniotomy (AComA, ICA, MCA), pterional minicraniotomy (ICA, PComA), and the interhemispheric minicraniotomy (pericallosal artery). An example of the technique for minimally invasive management of aneurysms of the anterior circulation is demonstrated in a patient with a previously endovascularly coiled AComA aneurysm.

Case 1: A 52-year-old man suffered from an SAH (Hunt & Hess [H&H] grade I). Angiography revealed an AComA aneurysm (**Fig. 2**A), which was treated by coiling (see **Fig 2**B). A surveillance angiogram 18 months later revealed reopening of

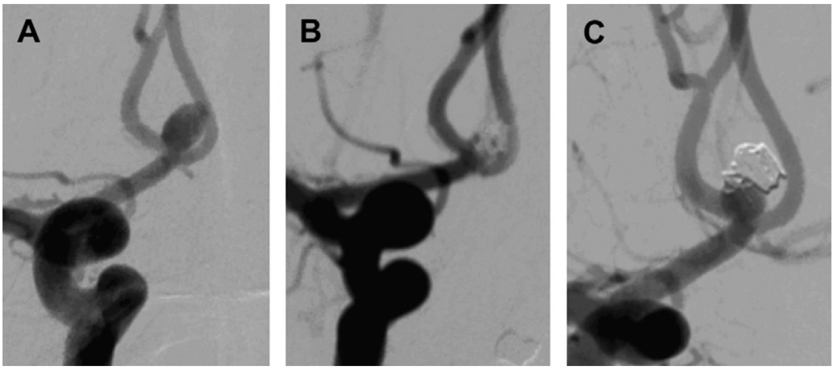

Fig. 2. Cerebral angiography showing the AComA aneurysm (*A*) before and (*B*) after initial complete coiling, (*C*) demonstrating the recanalization in the neck region 18 months later.

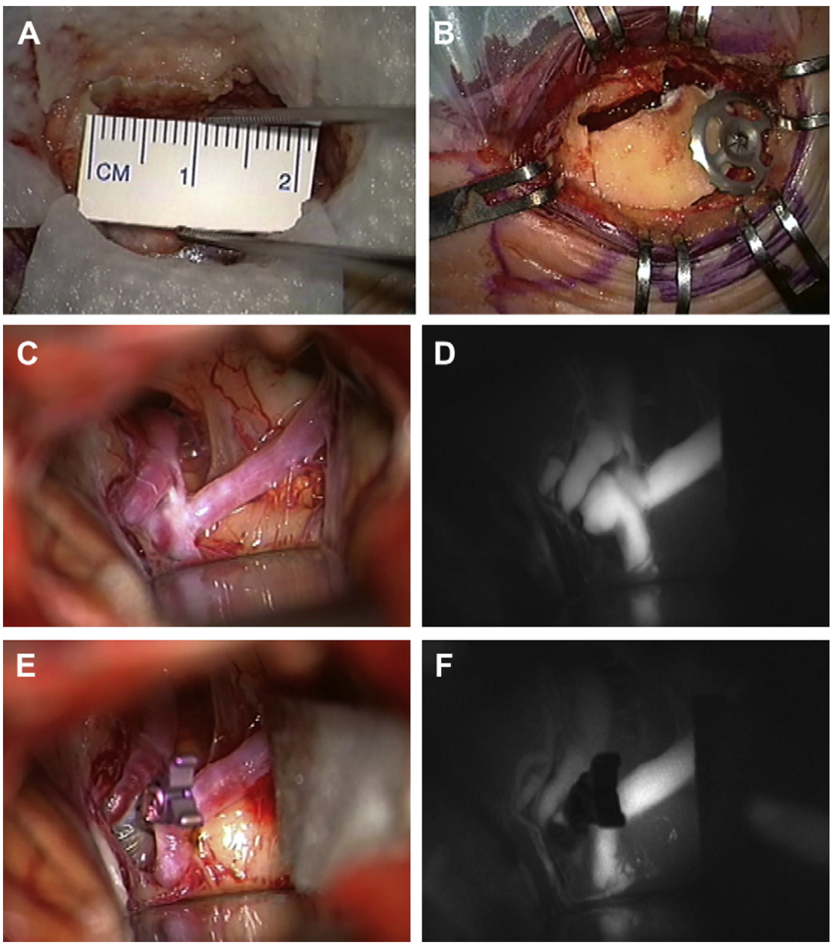

Fig. 3. Intraoperative image of (*A*) the supraorbital keyhole craniotomy before dural opening and (*B*) after reimplantation of the bone flap. Microscopic view of the AComA complex with the aneurysm neck and good overview of both A1 and A2 segments (*C*) before and (*E*) after clipping with the corresponding ICG angiographic images (*D*, *F*).

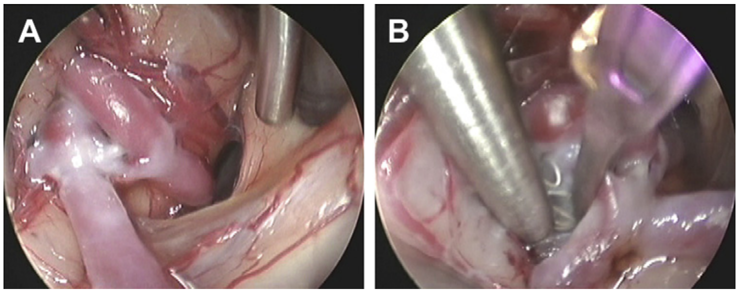

Fig. 4. Different perspective of endoscopic view compared with the microscopic image of the AComA complex demonstrating the aneurysm neck and both A1 and A2 segments (*A*) before and (*B*) after clipping.

the neck (see **Fig. 2**C). Definite surgical treatment was performed through a supraorbital keyhole craniotomy on the right side (**Fig. 3**A and B) using intraoperative ICG angiography (see **Fig. 3**C–F) and endoscope-assisted technique (**Fig. 4**). A postoperative angiogram demonstrated complete aneurysm occlusion (**Fig. 5**).

Posterior circulation aneurysms

Aneurysms of the posterior circulation (basilar artery [BA], posterior cerebral artery [PCA], superior cerebellar artery [SCA], vertebral artery [VA], posterior inferior cerebellar artery [PICA]) represent only about 10% of intracranial aneurysms.[11] Most of these aneurysms are presently managed using endovascular techniques because of the high rate of morbidity associated with transcranial surgical approaches. However, unfavorable anatomy of the aneurysm or alterations in the proximal vessel may still mandate surgical management. Preferred minimally invasive approaches to the posterior circulation are the supraorbital keyhole craniotomy (BA, PCA, SCA), the subtemporal minicraniotomy (BA, PCA, SCA), and the retrosigmoid minicraniotomy (PICA, VA). Minimally invasive management of aneurysms of the posterior circulation is demonstrated in a patient with a pretreated formerly ruptured PICA aneurysm.

Case 2: A 58-year-old woman suffered from an SAH from a ruptured PICA aneurysm. This hemorrhage was initially treated by endovascular coiling. A routine angiogram demonstrated significant recanalization of the neck (**Fig. 6**). Surgical clipping via a retrosigmoid minicraniotomy was performed. Endoscope-assisted technique was used to enhance understanding of the aneurysm pathoanatomy before and after clipping (**Fig. 7**). Despite this complex clinical situation, complete occlusion of the aneurysm was achieved through a limited approach (**Fig. 8**).

Multiple aneurysms

More than 1 aneurysm is found in 10% to 30% of all patients with aneurysm.[1] Aneurysm distribution

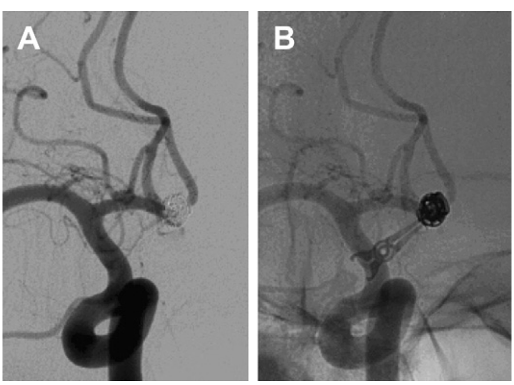

Fig. 5. Postoperative cerebral angiography showing the complete occlusion of the AComA aneurysm (*A*) with and (*B*) without subtraction.

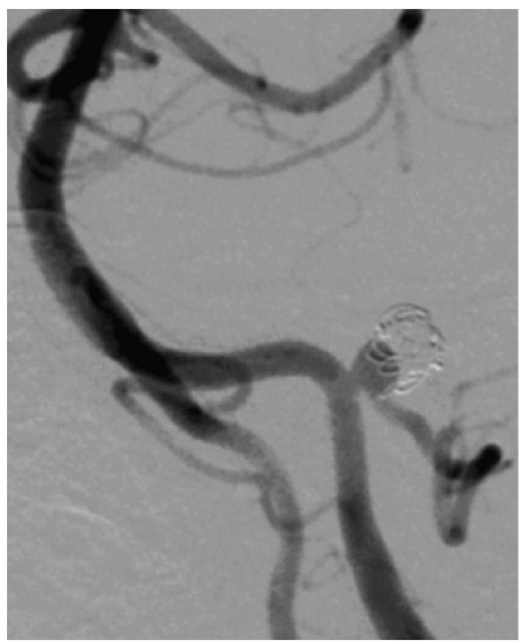

Fig. 6. Cerebral angiography of an asymptomatic patient with a history of SAH and endovascular coiling of a left-sided PICA aneurysm showing recanalization.

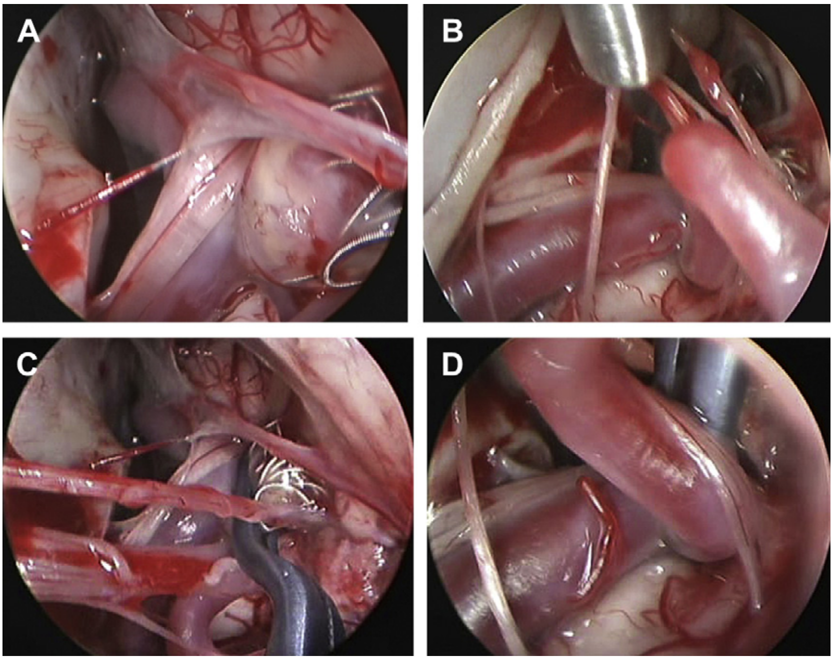

Fig. 7. Intraoperative endoscopic image of the PICA aneurysm in the left cerebellopontine angle, (*A*) demonstrating the proximal and lateral aspects of the neck, (*B*) close-up of the PICA, (*C*) overview after clipping, and (*D*) close-up showing the uncompromised PICA.

in these patients follows the same patterns as seen in patients with single aneurysms. Basically, all potential combinations of multiple aneurysms can be seen. The minimally invasive surgical strategy for addressing multiple aneurysms is to occlude as many aneurysms as possible through a single craniotomy, which means that sometimes not all aneurysms can be addressed via the optimal approach.[19] A thorough analysis of possible approaches to each aneurysm and possible additional risks associated with applying this strategy is mandatory to struggle for the best treatment of any individual patient. Preferred approaches for multiple aneurysms are the supraorbital keyhole craniotomy and the pterional minicraniotomy. Minimally invasive management of multiple aneurysms is demonstrated in a patient with incidental bilateral MCA aneurysms and a patient with a ruptured BA aneurysm and an unruptured right ICA aneurysm.

Case 3: A 58-year-old woman was referred with an H&H grade IV SAH and atypical right frontal intracerebral hemorrhage (ICH). Angiography revealed multiple aneurysms of both MCA and the

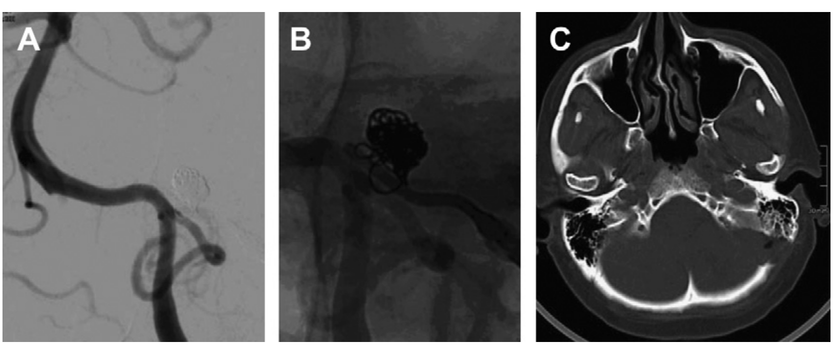

Fig. 8. Cerebral angiography following clipping, (*A*) with and (*B*) without subtraction showing perfect reconstruction of the PICA, clip location, and the coils left inside; (*C*) postoperative computed tomography showing the retromastoid minicraniectomy.

right ICA. Based on the location of the ICH, the right ICA aneurysm was thought to be the bleeding source. Coil occlusion of the ICA aneurysm was performed. Surveillance angiography 6 months later demonstrated complete coil occlusion of the ICA aneurysm, but 4 additional aneurysms were located at the right M1, right ICA bifurcation, left M1, and left M2 (**Fig. 9**). The patient had recovered well from her hemorrhage and therefore was offered surgical occlusion of all further aneurysms via a unilateral supraorbital keyhole craniotomy. Endoscope-assisted technique was used for the ipsilateral (**Fig. 10**) and the contralateral aneurysms (**Fig. 11**). Complete occlusion of all aneurysms was achieved without complications as demonstrated by postoperative angiography (**Fig. 12**).

Case 4: A 68-year-old woman suffered from acute headache and dizziness. A computed tomography of the head showed a basal and right sylvian SAH. Angiography revealed an aneurysm of the BA tip, thought to be the bleeding source, and an incidental medially directed aneurysm of the right proximal ICA (**Fig. 13**). Endovascular treatment was felt to be a poor option because of severe arteriosclerotic changes of the VAs and BA, and a rather broad neck of the BA aneurysm. Occlusion of both aneurysms was achieved using a left supraorbital keyhole craniotomy. Endoscope-assisted technique was used to enhance the understanding the pathoanatomy of the basilar tip aneurysm before and after clipping (**Fig. 14**). The contralateral location of the craniotomy to the right ICA aneurysm was chosen because of the medial orientation of the aneurysm, which could be visualized well from the opposite side using the endoscope (**Fig. 15**). Postoperative angiography demonstrated complete occlusion of both aneurysms (**Fig. 16**).

SUMMARY

Today, ruptured and unruptured aneurysms of all locations may be surgically treated successfully using minimally invasive techniques. In choosing the correct keyhole approach, it becomes possible to dramatically reduce the size of the craniotomy with less brain exposure and retraction, thereby minimizing clipping-related morbidity.[19] Minimally invasive surgical management of aneurysms provides the possibility of enabling the higher long-term occlusion rates possible with surgical management, with a more favorable morbidity profile. By diminishing the risk profile of open surgery, coupled with the benefits of less frequent and less invasive follow-up investigations, the microsurgical management of aneurysms may once again become the treatment of choice for patients with ruptured and unruptured aneurysms. However, minimally invasive aneurysm surgery requires thorough planning, a strong commitment of the surgical team to improving their technique with these methods, the availability of modern technical equipment, and a comprehensive knowledge of neurovascular pathoanatomy and pathophysiology. Therefore, minimally invasive aneurysm surgery should be performed preferably in high-volume and specialized neurovascular centers, which continuously monitor and assess their results with the goal of continuously improving their treatment protocols.

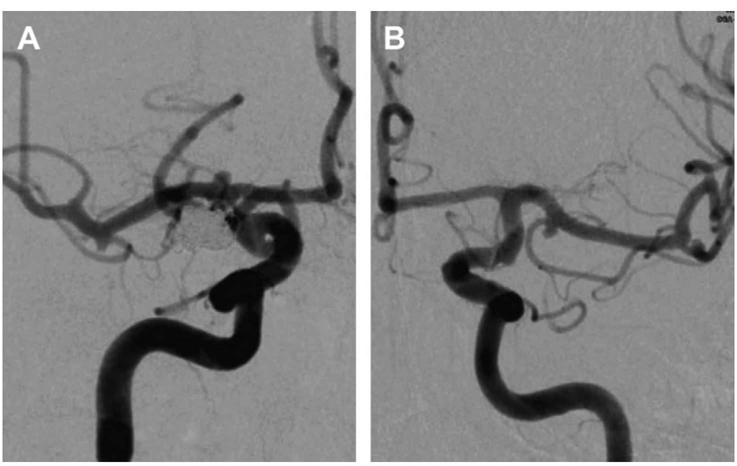

Fig. 9. Cerebral angiography of (*A*) the coil-occluded right ICA aneurysm and 2 further small aneurysms of the right M1 and right ICA bifurcation, (*B*) left ICA with further 2 small aneurysms of the M1 and M2 segments.

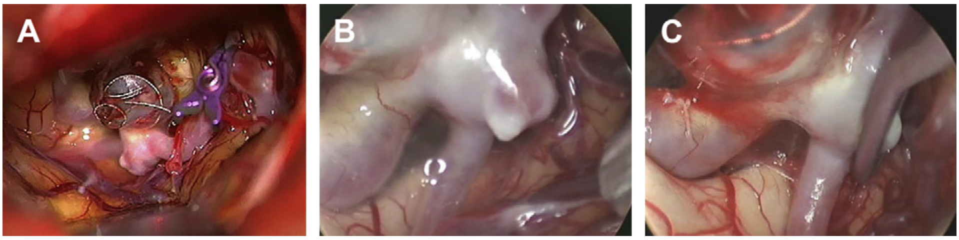

Fig. 10. (*A–C*) Intraoperative microscopic image showing the right coil-occluded ICA aneurysm, the clipped right M1 aneurysm, and the additional aneurysm of the ICA bifurcation; endoscopic view of the aneurysm of the right ICA bifurcation (*A*) before and (*C*) after clipping with perfect view of the large perforator.

ARTERIOVENOUS MALFORMATIONS

Cerebral AVMs are congenital vascular malformations consisting of a network of feeding arteries and draining veins within the brain parenchyma, in which loss of normal vascular organization at the subarteriolar level without an intervening capillary bed results in abnormal arteriovenous shunting. The direct arteriovenous connection leads to fibromuscular thickening and incompetent elastica interna particularly in veins, with an increased risk of rupture.[20] Malformations with only 1 feeding vessel that lack an intervening nidus

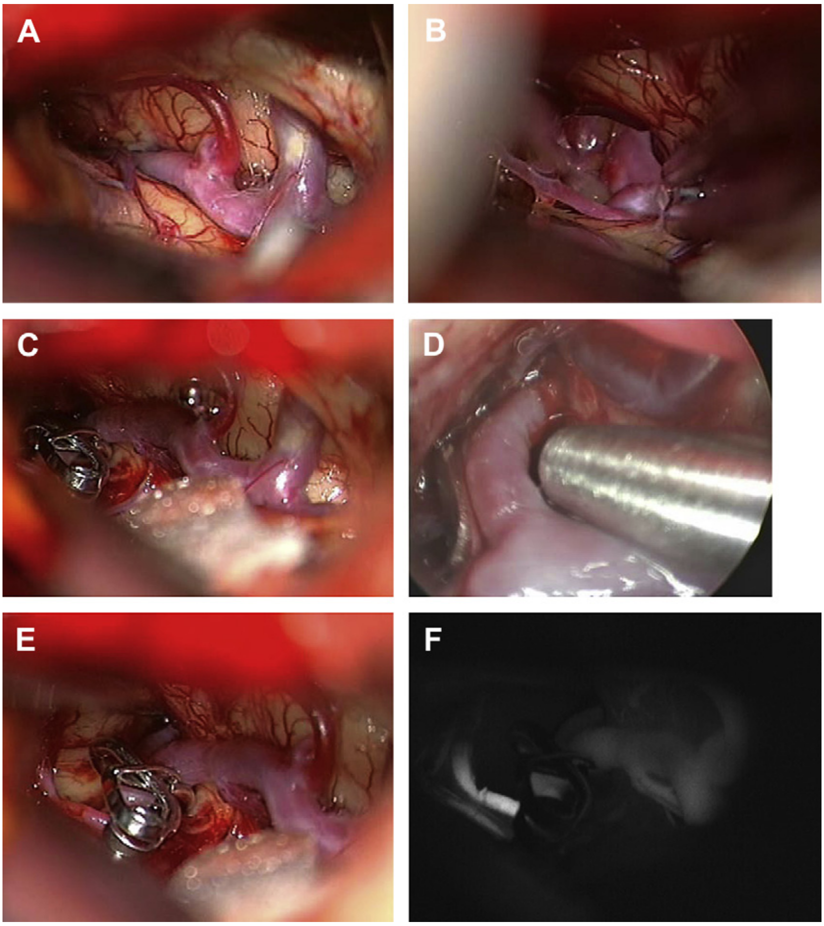

Fig. 11. Intraoperative view of (*A*) the contralateral M1 and (*B*) M2 aneurysm, (*C*) showing both aneurysms clipped with the non-involved M2 segment running through the clip fenestration, (*D*) endoscopic clip control showing the involved M2 without compression, (*E*) microscopic image and (*F*) ICG angiography of the clipped contralateral M2 aneurysm.

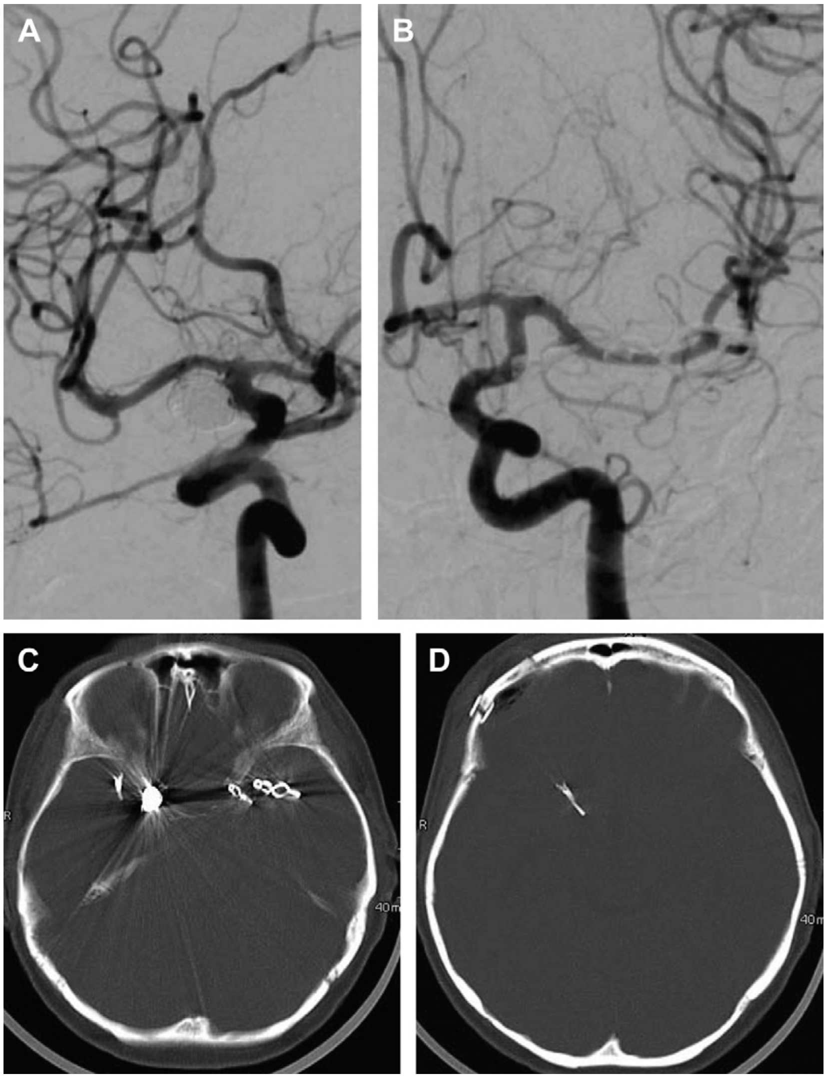

Fig. 12. Cerebral angiography following clipping of (*A*) all ipsilateral right-sided and (*B*) all contralateral left-sided aneurysms; CT image showing (*C*) all clips inserted from the right side and (*D*) the right-sided supraorbital keyhole craniotomy.

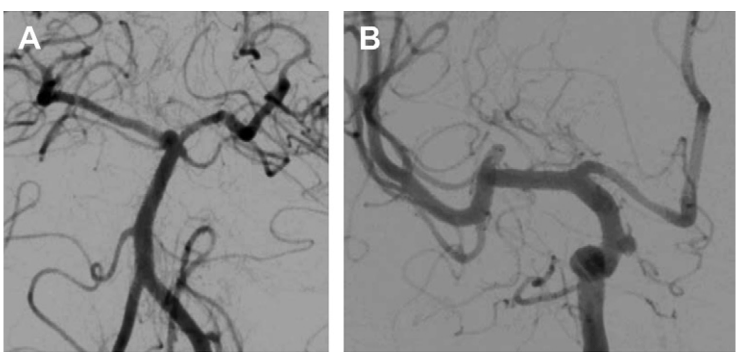

Fig. 13. Cerebral angiography of a patient presenting with an SAH from (*A*) a BA tip aneurysm and (*B*) an additional incidental right ICA aneurysm.

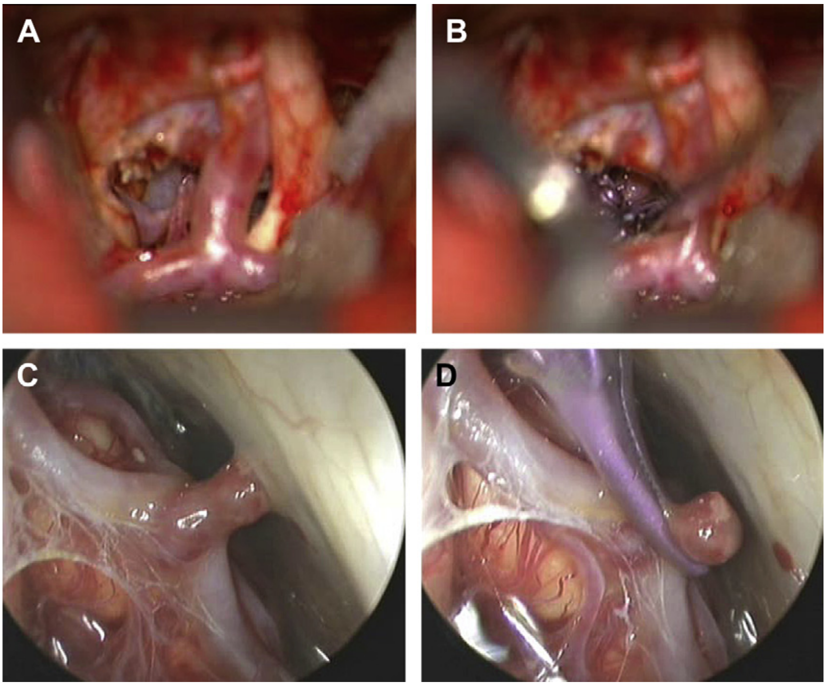

Fig. 14. Intraoperative microscopic view via a left supraorbital keyhole craniotomy of the posterior cerebral artery and the basilar artery tip aneurysm (*A*) before and (*B*) during clipping. Endoscopic view of the same aneurysm (*C*) before and (*D*) after clipping.

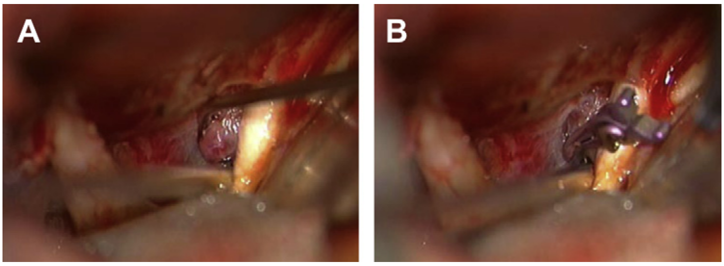

Fig. 15. Intraoperative microscopic view of the additional incidental right ICA aneurysm (*A*) before and (*B*) after clipping.

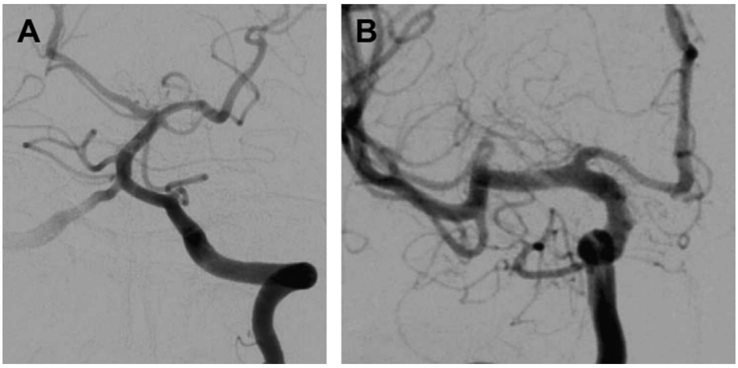

Fig. 16. Cerebral angiography after endoscope-assisted clip occlusion of (*A*) the BA tip aneurysm and (*B*) the incidental right ICA aneurysm via a left supraorbital keyhole craniotomy.

are called AVFs. If the location of the fistula is located within the dura, they are called dural AVFs (DAVFs). DAVFs are thought to be acquired.

The prevalence of arteriovenous malformation is estimated at approximately 0.1% of the general population, but reported rates range from 0.001% to 0.52%.[20] AVMs may become symptomatic by rupture, leading to ICH and/or SAH but can also cause symptoms without rupturing by causing seizures, symptoms due to mass effect, or ischemic steal. AVMs are most commonly located supratentorially (75%) and equally distributed within the cerebral lobes. AVMs occur less frequently in the insula and basal ganglia.

The overall risk of hemorrhage of arteriovenous malformations is estimated to range from 2% to 4% per year, and each hemorrhage is associated with a 5% to 10% chance of death and a 30% to 50% chance of permanent, disabling neurologic deficits.[20] In most patients, the first hemorrhage typically occurs between 20 and 40 years of age.[20,21] The risk of bleeding is increased by the presence of aneurysms (feeding artery, intranidal, or venous), drainage into the deep venous sinuses, deep location (ie, basal ganglia, internal capsule, thalamus, or corpus callosum), a single draining vein, venous stenosis, and previous hemorrhage.[20,22–24] After first hemorrhage, the annual risk of a subsequent hemorrhage has been reported to range from 4.5% to 34.4% during the first year, with a return to the baseline risk after the first year.[20,23]

Immediate surgical treatment is indicated only in space occupying ICH. Preoperative embolization may be helpful also in these cases. Contemporary treatment strategies include in most instances an initial endovascular embolization, often in a multistep procedure, followed by surgical resection or radiosurgery.

Surgical Treatment

The goal of surgical treatment is to completely occlude or resect the nidus or fistula without impairment of the normal cerebral perfusion. Partial occlusion or resection of the AVM does not lead to a relevant reduction of the bleeding risk. The risk of treatment in AVMs can be estimated by the Spetzler-Martin grading system. Larger size (<3, 3–6, >6 cm), eloquent location, and deep venous drainage were found to be associated with a higher treatment risk.[25]

Minimally invasive aspects of AVM surgery involve limiting the exposure for complete occlusion or resection of the AVM or AVF and highly selective occlusion or resection of ill-defined vessels, thereby leading to reduction of hospitalization time and reconvalescence.

Basically, all previously mentioned minimally invasive approaches are suitable for AVM and AVF surgery, that is, the supraorbital keyhole, pterional, retrosigmoid, and interhemispheric minicraniotomies (see the article by Garrett and colleagues elsewhere in this issue for further exploration of this topic). But frequently an individually tailored craniotomy has to be placed over the AVM on the convexity of the skull.

Critical aspects for successful surgical treatment are sufficient visualization of the feeding and draining vessels, early control of the feeding vessel, intraoperative understanding of the individual haemodynamics, proper occlusion of the ill-defined vessels, and intraoperative control of the result. Applying this concept in combination with modern technology, even complex AVMs can be successfully and safely treated with limited exposure.

Technical Considerations

Necessary requirements for minimally invasive AVM surgery is a high-quality operating microscope equipped with ICG angiography. Alternatively, high-quality sonography or micro-Doppler sonography may be used intraoperatively to assess the individual haemodynamics. Endoscopes may also be helpful to enhance pathoanatomical orientation, in particular in DAVFs. In all AVMs with relevant relation to the motor or sensory cortex neurophysiological monitoring (SSEP, MEP) should be performed. Occlusion of the AVM is performed by bipolar coagulation or application of clips. Specific nonstick bipolar coagulation forceps are advisable.

Specific Surgical Considerations and Illustrative Cases

The surgical strategy is to secure early control of all feeding vessels while carefully sparing the draining veins. At the end of the resection, the veins should be occluded or resected if not involved in important drainage of healthy brain tissue to avoid recruitment of new arterial vessels and thus leading to recurrence. Furthermore, the complete nidus should be removed, even the embolized parts. Minimally invasive management of AVMs is demonstrated in a patient with a left-sided parieto-occipital AVM and a patient with an ethmoidal DAVF.

Case 5: A 19-year-old boy suffered from a left-sided atypical ICH. Angiography revealed a left occipital grade III (Spetzler-Martin) AVM (**Fig. 17**). Partial embolization was achieved leaving still a multicompartmental AVM with significant arteriovenous shunt. At that time, the patient had visual

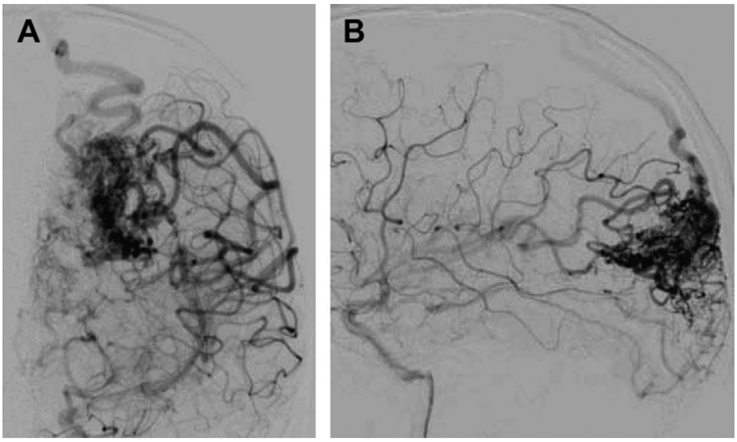

Fig. 17. Cerebral angiography of a left occipital AVM in (*A*) anteroposterior and (*B*) lateral views.

field deficits at the lower right quadrant. Complete surgical resection was performed through a tailored paramedian parieto-occipital craniotomy in prone position with the aid of neuronavigation and intraoperative ICG angiography (**Fig. 18**).

Case 6: In a 69-year-old man who suffered from an SAH 15 years ago, angiography at that time did not show a bleeding source. A ventriculoperitoneal shunt had to be inserted because of posthemorrhagic hydrocephalus. Now, a follow-up magnetic resonance imaging (MRI) revealed a significant stenosis of the right MCA. Angiography showed complete occlusion of the right MCA but sufficient perfusion of the right hemisphere. In addition, it revealed a left-sided ethmoidal DAVF at the basal and frontal aspects of the falx (**Fig. 19**A). An endovascular treatment trial was unsuccessful. Complete surgical occlusion was achieved using an endoscope-assisted technique (see **Fig. 19**B; **Fig. 20**A) through a left-sided supraorbital keyhole craniotomy using intraoperative ICG angiography (see **Fig. 20**C–F).

SUMMARY

Contemporary treatment of AVMs involves a team approach among neuroradiologists, neurosurgeons, and radiation oncologists. Initial embolization reduces surgical morbidity, blood loss, and operating time. Generally, complete destruction by resection or irradiation is required to cure the disease. AVMs frequently involve larger areas of the brain surface. Therefore, limited craniotomies are often not applicable. However, when possible, minimally invasive approaches to the treatment of AVMs consist of a tailored craniotomy, strict intraoperative strategic workflow, and advanced intraoperative visualization and resection control using ICG angiography and/or technologically advanced sonography. As with all minimally invasive neurosurgical procedures, AVM surgery requires thorough planning, an organized team dedicated to neuroendoscopy, availability of modern technical equipment, and an extraordinary knowledge of neurovascular pathoanatomy and pathophysiology. Treatment of AVMs should be performed in experienced centers that are capable of performing all available treatment modalities (ie, endovascular, surgical, and radiation therapies).

CAVERNOUS MALFORMATIONS (CAVERNOMAS)

Cavernous malformations (cavernomas) are benign vascular lesions consisting of cavernous-like enlarged veins with dysplastic walls located within the brain or rarely in the spinal cord lacking intervening neural parenchyma, large feeding arteries, or large draining veins.[26,27] Cavernomas affect 0.4% to 0.5% of the population[27] and become symptomatic either by bleeding (20%) and thereby leading to an (often small) ICH with headaches and/or focal neurologic deficits, or frequently by initiating epileptic seizures (60%).[27,28] Most of all cavernomas are located supratentorially, but 10% to 23% are found in the brainstem, the cerebellum, and the myelon. Within the brainstem, there is a predilection for formation in the pons.[27] Two-thirds of the spinal cavernomas are located in the thoracic and one-third in the cervical myelon. In 15% of all cases, cavernomas are associated with a so-called developmental venous anomaly. The bleeding rate is variable and the definition of symptomatic hemorrhage is controversial; however, the overall

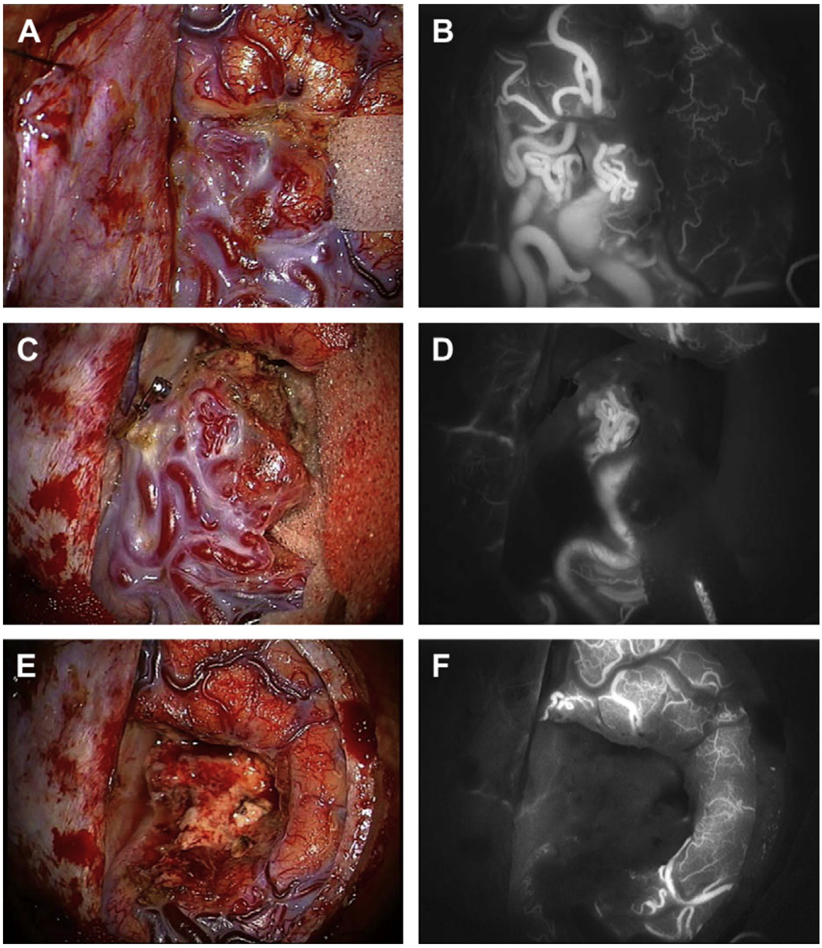

Fig. 18. Intraoperative images of microsurgical resection of a left parieto-occipital AVM with the patient in a prone position, (*A*) before, (*C*) after occlusion of the medial compartment of the AVM, and (*E*) after complete resection with the corresponding images of the intraoperative ICG angiography (*B*, *D*, *F*). The medial vein seems to be still arterialized as seen under the microscope (*C*), but ICG angiography (*D*) clearly shows no arteriovenous shunt anymore in that compartment of the AVM.

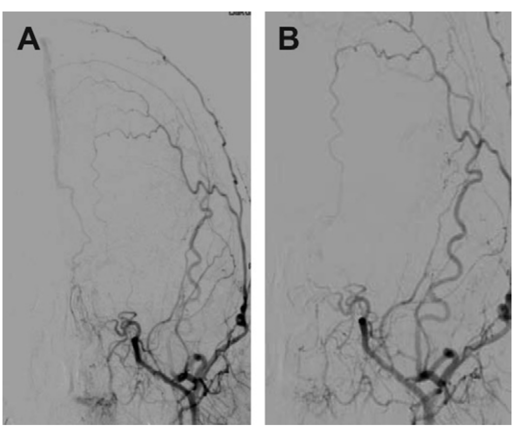

Fig. 19. Cerebral angiography of the left external carotid artery showing (*A*) a left fronto-mediobasal (ethmoidal) DAVF, (*B*) postopertively in coronal view.

rate is estimated between 0.6% and 3.1% per year.[27,28] Cavernomas situated in the brainstem or the cerebellum tend to bleed more easily and cause more damage than those found in the cerebrum.

Surgical Treatment

The goal of surgical treatment is to completely resect the cavernoma. In patients with seizures and supratentorially located lesions, removal of the surrounding yellowish gliosis harboring epileptogenic ferritin deposits should be addressed if not located in a highly eloquent region. The only effective treatment for cavernomas is complete surgical resection. Radiotherapy including radiosurgery has not conclusively been demonstrated to alter the natural history of these lesions.

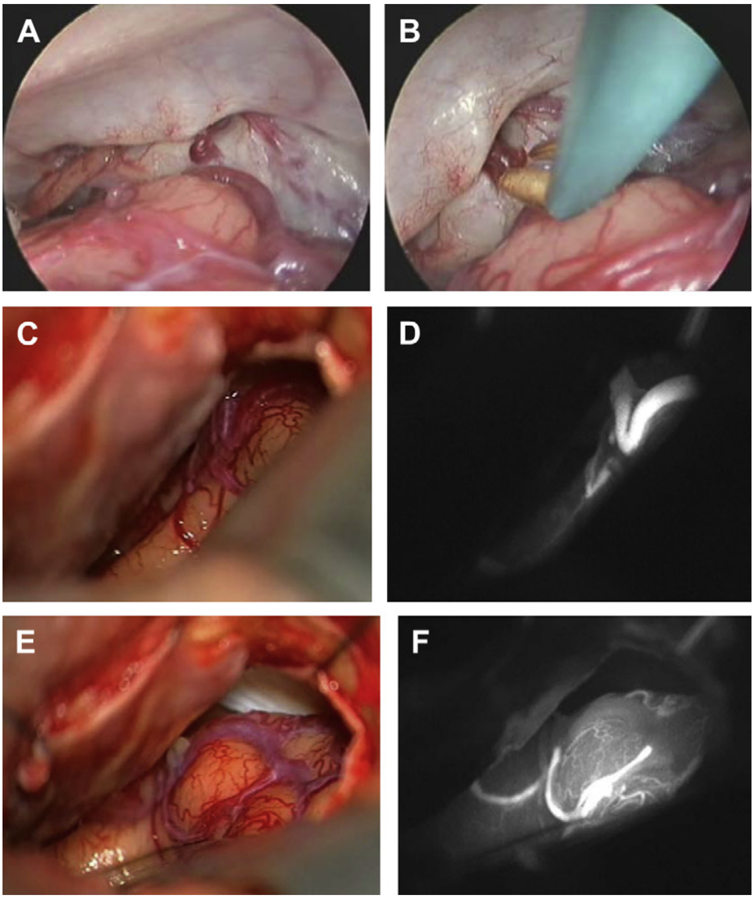

Fig. 20. Endoscopic view of a left fronto-medio-basal DAVF approached via a left supraorbital keyhole craniotomy (*A*) before and (*B*) during treatment. Microscopic and ICG angiographic images (*C*, *D*) before and (*E*, *F*) after coagulation and transsection of the DAVF.

Minimally invasive aspects of cavernoma surgery involve reduction of approach-related morbidity in deep-seated and brainstem lesions, lesionectomy-related morbidity, and hospitalization and back-to-work time. Minimally invasive approaches suitable for brainstem cavernomas are the eyebrow, pterional, subtemporal, retrosigmoid, and suboccipital minicraniotomies (see the article by Garrett and colleagues elsewhere in this issue for further exploration of this topic).

Critical aspects for successful surgical treatment are selection of the optimal approach, improved intraoperative visualization by using an endoscope, recognition of fiber tracks and anatomic landmarks using neuronavigation, electrophysiological monitoring of fiber tracks and cranial nerve nuclei, selection of microsurgical instruments suitable for complete removal of the lesion, and intraoperative control of the result, namely obtaining adequate hemostasis following lesionectomy. Applying this concept in combination with advanced technology, most deep-seated and even brainstem cavernomas can be successfully and safely removed.

Technical Considerations

The basic requirements for minimally invasive cavernoma surgery are a high-quality operating microscope and a neuronavigation system. Alternatively, high-quality sonography may be used intraoperatively to visualize a deep-seated cavernoma. Endoscopes may also be helpful to enhance pathoanatomic orientation, in particular to screen for remnants in the resection cavity. Detailed neurophysiological monitoring (concentric needle electromyography/stimulation, SSEP, MEP, auditory evoked potential) is mandatory in all brainstem cavernomas and other highly eloquent locations.

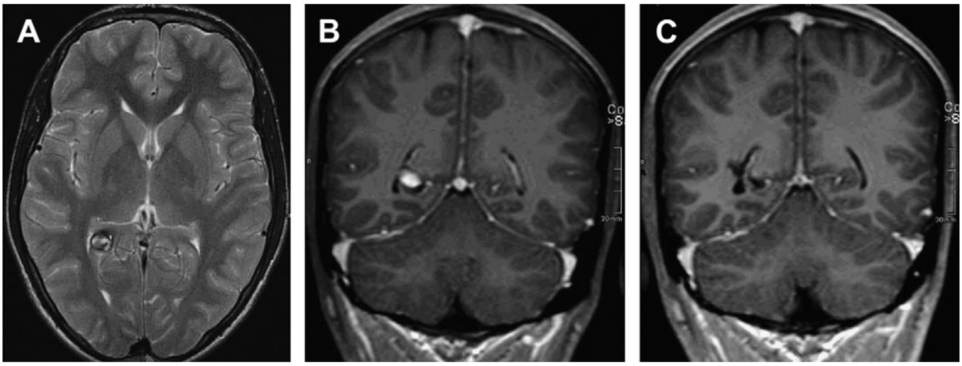

Fig. 21. (*A*) T2-weighted axial and (*B*) T1-weighted coronal gadolinium-enhanced MRI of a right temporo-occipital deep-seated cavernoma (*B*) before and (*C*) after complete removal.

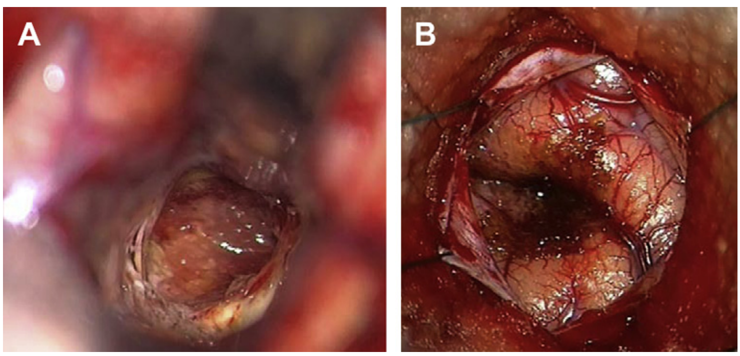

Fig. 22. Microscopic view (*A*) through the right lateral ventricle into the resection cavity after complete removal of the cavernoma and (*B*) of the transsulcal, transcortical approach at the end of the surgery.

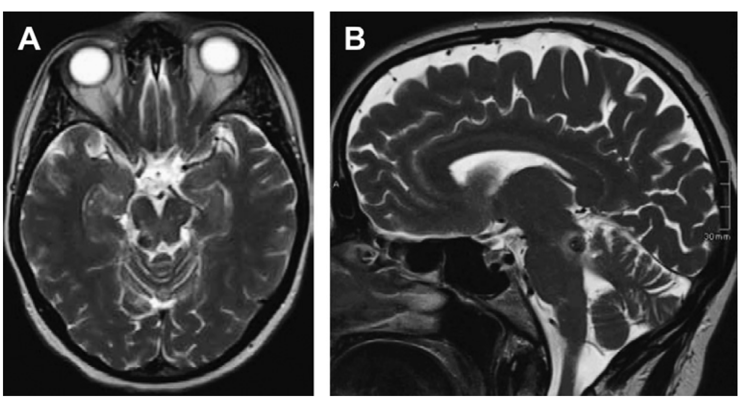

Fig. 23. T2-weighted (*A*) axial and (*B*) sagittal MRI of a patient with a right dorsolateral cavernoma of the midbrain.

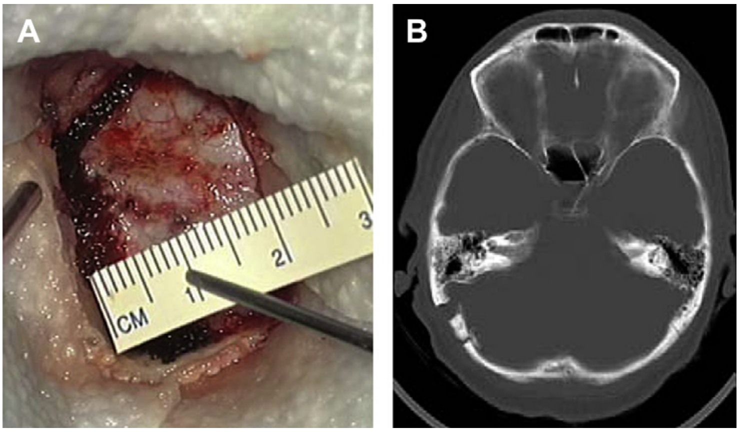

Fig. 24. Right-sided osteoplastic retrosigmoid minicraniotomy (*A*) before opening of the dura and (*B*) in the computed tomographic scan postoperatively, the bone flap fixed with CranioFix (B.Braun/Aesculap, Tuttlingen, Germany; with permission).

Specific Surgical Considerations and Illustrative Cases

It is critical to approach the cavernoma in the least traumatic manner. Compared with other neurovascular lesions, dealing with the lesion itself is often less difficult than approaching the lesion, which is particularly true in the case of deep-seated and brainstem cavernomas. As a rule of thumb, a cavernoma is best approached from where it reaches any surface of the brain. In some cases, this region may be the ependymal surface of the ventricular system.

The cases below demonstrate minimally invasive management of cavernomas in a patient with a deep-seated cavernoma of the right temporo-occipital paraventricular region and a patient with a symptomatic brainstem cavernoma.

Case 7: A 15-year-old boy presented with severe headaches but otherwise neurologically intact. MRI showed a right temporo-occipital paraventricular lesion, typical for a cavernoma (**Fig. 21**A, B). A transtemporal, transventricular minimally invasive approach (**Fig. 22**) was used for complete removal (see **Fig. 21**C). With the aid of neuronavigation, the optic fibers could be spared. Postoperatively, the patient was completely neurologically intact, in particular with no visual field deficits.

Case 8: A 48-year-old woman suddenly developed numbness and sensory deficits of the right side of her body. MRI showed a cavernoma of the right dorsolateral midbrain (**Fig. 23**). A right-sided retromastoid (**Fig. 24**), supracerebellar keyhole approach was chosen for complete removal (**Fig. 25**) with the support of neuronavigation, endoscopy, and, neurophysiological monitoring.

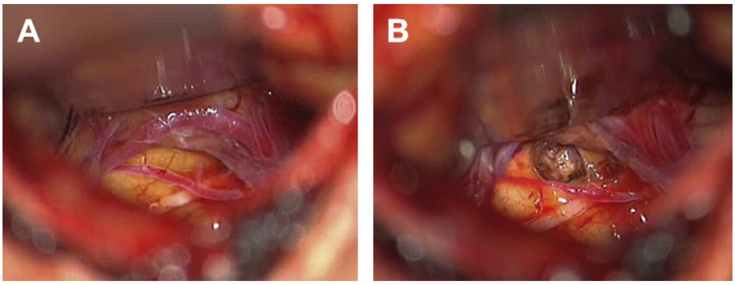

Fig. 25. Intraoperative microscopic view of the dorsolateral midbrain showing (*A*) the yellowish discoloration from the cavernoma bleeding and (*B*) the empty cavity after complete removal.

SUMMARY

Cavernomas are frequently found in highly eloquent and/or deep locations. Complete surgical removal is the only proven treatment for definitive management of these lesions. Surgical morbidity is not only related to the lesionectomy but also to the approach. Minimally invasive concepts may help to reduce both approach-related and lesionectomy-related morbidities. Thorough planning and the willingness to familiarize oneself with these challenging and often unfamiliar approaches is an essential requirement for safe and technically successful minimally invasive cavernoma surgery. Furthermore, technical preconditions such as advanced neuronavigation and multimodal neurophysiological monitoring are essential for clinical success.

REFERENCES

1. Schievink WI. Intracranial aneurysms. N Engl J Med 1997;336:28–40 [erratum in: N Engl J Med 1997; 336:1267].
2. Wijdicks EF, Kallmes DF, Manno EM, et al. Subarachnoid hemorrhage: neurointensive care and aneurysm repair. Mayo Clin Proc 2005;80:550–9.
3. Wiebers DO, Whisnant JP, Huston J III, et al. Unruptured intracranial aneurysms: natural history, clinical outcome, and risks of surgical and endovascular treatment. Lancet 2003;362:103–10.
4. Stapf C, Mohr JP. Aneurysms and subarachnoid hemorrhage epidemiology. In: Le Roux PD, Winn HR, Newell DW, editors. Management of cerebral aneurysms. Philadelphia: Saunders; 2004. p. 183–7.
5. Rinkel GJ, Djibuti M, Algra A, et al. Prevalence and risk of rupture of intracranial aneurysms: a systematic review. Stroke 1998;29:251–6.
6. Bederson JB, Connolly ES, Batjer HH, et al. Guidelines for the management of aneurismal subarachnoid hemorrhage. A statement for the healthcare professionals from a special writing group of the stroke council, American Heart Association. Stroke 2009;40:994–1025.
7. Bederson JB, Awad IA, Wiebers DO, et al. Recommendations for the management of patients with unruptured intracranial aneurysms: a statement for healthcare professionals from the stroke council of the American Heart Association. Stroke 2000;31: 2742–50.
8. Suarez JI, Tarr RW, Selman WR. Aneurysmal subarachnoid hemorrhage. N Engl J Med 2006; 354:387–96.
9. Connolly ES, Solomon RA. Management of unruptured aneurysms. In: Le Roux PD, Winn HR, Newell DW, editors. Management of cerebral aneurysms. Philadelphia: Saunders; 2004. p. 271–85.
10. Greenberg MS. SAH and aneurysms. In: Greenberg MS, editor. Handbook of neurosurgery. 5th edition. New York: Thieme Medical; 2000. p. 754–803.
11. Brisman JL, Song JK, Newell DW. Cerebral aneurysms. N Engl J Med 2006;355:928–39.
12. Johnston SC, Selvin S, Gress DR. The burden, trends, and demographics of mortality from subarachnoid hemorrhage. Neurology 1998;50:1413–8.
13. Raaymakers TWM, Rinkle GJ, Limburg M, et al. Mortality and morbidity of surgery for unruptured intracranial aneurysms: a meta-analysis. Stroke 1998;29:1531–8.
14. Hunt WE, Hess RM. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. J Neurosurg 1968;28(1):14–20.
15. Whitfield PC, Kirkpatrick PJ. Timing of surgery for aneurysmal subarachnoid hemorrhage. Cochrane Database Syst Rev 2001;2:CD001697.
16. International Study of Unruptured Intracranial Aneurysms Investigators (ISUIA). Unruptured intracranial aneurysms – risk of rupture and risk of surgical intervention. N Engl J Med 1998;339:1725–33.
17. Clarke G, Mendelow AD, Mitchell P. Predicting the risk of rupture of intracranial aneurysms based on anatomical location. Acta Neurochir 2005;147: 259–63.
18. International Study of Unruptured Intracranial Aneurysms Investigators (ISUIA). Unruptured intracranial aneurysms: natural history, clinical outcome, and risks of surgical and endovascular treatment. Lancet 2003;362:103–10.
19. Hopf NJ, Stadie A, Reisch R. Surgical management of bilateral middle cerebral artery aneurysms via a unilateral supraorbital key-hole craniotomy. Minim Invasive Neurosurg 2009;52:126–31.
20. Friedlander RM. Arteriovenous malformations of the brain. N Engl J Med 2007;356(26):2704–12.
21. Brown RD Jr, Wiebers DO, Torner JC, et al. Frequency of intracranial hemorrhage as a presenting symptom and subtype analysis: a population-based study of intracranial vascular malformations in Olmsted Country, Minnesota. J Neurosurg 1996; 85:29–32.
22. Redekop G, TerBrugge K, Montanera W, et al. Arterial aneurysms associated with cerebral arteriovenous malformations: classification, incidence, and risk of hemorrhage. J Neurosurg 1998;89:539–46.
23. Stapf C, Mast H, Sciacca RR, et al. Predictors of hemorrhage in patients with untreated brain arteriovenous malformation. Neurology 2006;66:1350–5.
24. Nataf F, Meder JF, Roux FX. Angioarchitecture associated with hemorrhage in cerebral arteriovenous malformations: a prognostic statistical model. Neuroradiology 1997;39:52–8.

25. Spetzler RF, Martin NA. A proposed grading system for arteriovenous malformations 1986. J Neurosurg 2008;65:476–83.
26. Robinson JR, Awad IA, Little JR. Natural history of the cavernous angioma. J Neurosurg 1991;75:709–14.
27. Greenberg MS. Handbook of neurosurgery. 6th edition. New York: Thieme Medical Publishers; 2006.
28. Amin-Hanjani S, Ojemann RG, Ogilvy CS. Surgical management of cavernous malformations of the nervous system. In: Roberts DW, Schmidek HH, editors. Schmidek & sweet's operative neurosurgical techniques. indications, methods, and results. 5th edition. Philadelphia: Saunders Elsevier; 2006. p. 1307–24.