Cerebral Aneurysms: Learning from the Past and Looking toward the Future

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INTRODUCTION

Although we cannot fully review the broad topic of "cerebral aneurysms" in this brief chapter, we will attempt to highlight issues that we consider of particular current relevancy, emphasizing the recent literature. The chapter will be heavily influenced by the biases of the senior author (RCH), although we will try to point out where those biases are based on experience and personal opinion rather than on the available published literature. Recent developments in our knowledge of the etiology, growth, and rupture of intracranial aneurysms as well as new information on the natural history of ruptured and unruptured aneurysms will be reviewed. Treatment options, with emphasis on the decision-making process, a topic of particular interest to the senior author, will be discussed. The most common pitfalls and complications encountered with microsurgery of cerebral aneurysms and the surgical results of the senior author are discussed. Finally, we speculate, based on current evolving knowledge and technology, about the changing paradigms in the diagnosis and treatment of intracranial aneurysms.

INCIDENCE

Data regarding the incidence of aneurysms in the population is varied, but several large autopsy studies indicate that at least 2 to 5% of the population may harbor cerebral aneurysms.[10,35,59] A recent review of 3684 angiograms by Winn et al. however, revealed a prevalence of unruptured aneurysms of only 0.65%.[104] The incidence of cerebral aneurysms is highest during the fourth, fifth, and sixth decades of life, and they are relatively rare in the first three decades. Children and young adults who do harbor aneurysms, however, are more likely to harbor internal carotid artery (ICA) aneurysms of giant size.[1] Intracranial aneurysms are far more common in women, with at least a 2:1 ratio.[89] Of the 277 patients surgically treated by the senior author in the past 5 years, 79% have been female. Aneurysm location also shows gender predilection, with women more likely to have an ICA or middle cerebral artery (MCA) aneurysm, and men an anterior communicating artery (AComA) aneurysm.[89] Multiple aneurysms can be found in 15 to 31% of cases, 16.2% in the senior author's most recent series.

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ETIOLOGY

Pathophysiology

The origin of cerebral aneurysms is still incompletely understood, but it is clear that multiple factors play into aneurysmal development. The normal cerebral artery is made of three layers: the adventitia, a prominent muscularis media, and an endothelial intima, with an intervening internal elastic lamina. Aneurysms are devoid of the muscularis media, which usually ceases proximal to the neck.[89] Forbus described congenitally absent areas of muscularis media that occurred at arterial bifurcations and proposed that these played a significant part in the creation of aneurysms.[20] Glynn, however, found that these muscularis defects were equally present in arteries both with and without aneurysms and, as a result of intraluminal pressure studies, concluded that defects of the muscularis *and* internal elastic lamina were necessary for aneurysmal formation.[23] This occurs with focal degeneration of the internal elastic intima by atherosclerotic changes and is supported by a demonstration of the rapid development of saccular aneurysms after destruction of the elastic layer by application of elastase and pulsatile flow.[63] Hemodynamic stress from axial stream impingement and turbulence caused by interruption of lamellar flow results in mechanical weakening of the vessel wall. Pressure generated at the apex of the arterial bifurcation ranges from two to three times the peak luminal pressure in the proximal parent artery, leading to aneurysmal development.[21] The contribution of atheromatous degeneration and hemodynamics to the formation of aneurysms explains why cigarette smoking and hypertension are two of the greatest risk factors for intracranial aneurysms.

Genetic Implications

Despite our current knowledge of the pathogenesis of cerebral aneurysms, there remain multiple unknown caus-

ative variables. Ultrastructural connective tissue alterations are currently under investigation because we have long known that the incidence of aneurysms is higher in people with diseases such as Marfan's, autosomal dominant polycystic kidney, neurofibromatosis type I, and Ehlers-Danlos Type IV. Abnormalities of collagen morphology have been linked to spontaneous carotid dissection and noted in a subset of patients with aneurysms.[25] In addition, three genome-wide linkage studies have identified genetic loci for intracranial aneurysms that include genes coding for structural proteins of the extracellular matrix, such as elastin and collagen type 1A2.[84]

Familial aggregation of intracranial aneurysms was first described in 1954 by Chambers et al.,[8] with the subsequent reporting of hundreds of families. With the advent of chromosomal studies, there has been a renewed interest in the genetics of familial aneurysms. Excluding families with other heritable disorders, familial aneurysms account for 7 to 20% of patients with cerebral aneurysms.[87,103] The occurrence of aneurysms between monozygotic twins is an astounding 87.5%.[70] The prevalence of aneurysms is significantly elevated in family members of those with asymptomatic aneurysms as well as ruptured aneurysms.[51] Familial intracranial aneurysms tend to rupture at a younger age than sporadic aneurysms.[88] In comparison to the general population, first-degree relatives of those suffering from subarachnoid hemorrhage (SAH) are at a fourfold risk themselves. Among families with two or more affected generations, children suffer SAH at a significantly younger age than their parents, similar to the increasing expression of unstable trinucleotide repeats found in other inheritable diseases.[88] Although the benefits have not been quantified, we suggest magnetic resonance angiography (MRA) screening for adult members of families with two or more immediate blood relatives (parents, siblings, and children) afflicted with cerebral aneurysms. Schievink et al. estimates the yield of this practice at approximately 10%.[88]

The inheritance pattern of familial intracranial aneurysms is not known. Transmission is consistent with autosomal dominance,[103] but it is clear that cerebral aneurysms are referable not to a single chromosomal abnormality, but to a conjunction of aberrations. Molecular genetic linkage through chromosomal study seems promising, and several chromosomal regions of interest have been identified (17cen[105] and 2p13[83]) and even replicated (7q11,[18] 19q13 and Xp22[74,105]). These chromosomal studies provide promising scaffolds on which to build for the future.

GROWTH AND RUPTURE

The growth and rupture of an aneurysm must be explained mechanically and pathologically on the basis of wall structure, hemodynamics, and extramural factors.[89] Hemodynamic shear stress and mural tension are key factors in the growth of aneurysms. Both axial stream impingement on the apex of arterial bifurcations and constant pulsatile flow into these areas enlarges a weakened arterial wall, leading to aneurysm growth and rupture.[21] Turbulence, caused by flow exceeding a critical velocity, adds to this effect. Ferguson postulated that structural fatigue results when vibrations produced by turbulence occur at the resonant frequency of the wall.[19] Extramural factors, such as intracranial pressure, are essential to controlling the rupture of aneurysms, as shown by Nornes[71] in his study of intracranial pressure after decompressive measures (spinal tap, mannitol, and ventriculostomy showed initial sharp decrease in pressure followed by a pressure spike as the aneurysm ruptured). Sarner and Crawford[85] found that rupture occurred at the fundus in 64%, the lateral sac in 10%, and the neck in only 2% (undetermined in 24%) of cases, intuitively following the direction of flow and hemodynamic stress. We can now prove this theory with three-dimensional (3D) computational flow dynamics to show the effects of arterial geometry on aneurysmal growth. Maximal growth occurs at the largest impact zone from the direction of flow, leading to growth of the dome in bifurcation aneurysms and the distal edge of the ostium in laterally based saccular aneurysms.[7,93,110]

An increase in size on follow-up studies may warrant treatment of an unruptured aneurysm.[43,108] In a study related to the Finnish report discussed above, 111 unruptured aneurysms were followed for almost 19 years.[43] Subsequent rupture of these aneurysms was associated significantly with lesion growth during the follow-up period. The mean diameter of fatal ruptured aneurysms was 13.2 mm, compared with 10 mm for nonfatal ruptured lesions.[45]

Risk factors for aneurysm formation and growth include the female gender and cigarette smoking,[44] as well as collagen vascular disease. Risk factors for aneurysm rupture are cigarette smoking and aneurysmal size.[43] Hypertension, age, and family history are other significant risk factors.

As technology continues to improve, our ability to study and predict the behavior of aneurysms broadens. Baoshun et al. are using 3D computer-assisted tomographic angiography (CTA) to assess the geometrical shape of unruptured saccular aneurysms, with the thought that shape, not just size, influences aneurysmal rupture.[3] Irregularity of the surface of the dome of the aneurysm seems to be most important, reinforcing the clinical practice of considering "blisters," "daughter sacs," and other irregularities observed at angiography, particularly now with 3D digital subtraction angiography (DSA) and CTA, as important risk factors when considering treatment of unruptured aneurysms. The potential pitfall of those studies is that the surface irregularities observed more commonly on ruptured aneurysms may be a result of rupture rather than a preexisting morphological feature predisposing to rupture. Steinman et al. described radiological simulation of flow dynamics with evaluation of

lumen geometry and flow pulsatility to predict aneurysmal growth,[93] a promising addition to our diagnostic armamentarium that may help with the decision regarding which ruptured aneurysms are more dangerous and, therefore, more deserving of treatment.

NATURAL HISTORY

Unruptured Aneurysms

Our understanding of the natural history of unruptured aneurysms has grown significantly in recent years and continues to evolve. Studies preceding the International Study of Unruptured Intracranial Aneurysms (ISUIA)[36,37] estimated the annual risk of rupture to be 1 to 6.25%, but data was sparse.[27,39,55,56] In addition, very small unruptured lesions were considered to have significant potential for rupture because of potential increases in aneurysm size.[109] As a result, many argued for surgical treatment of essentially all detected unruptured aneurysms in young and relatively healthy patients.

The ISUIA is a dual arm study, published in 1998 and 2003, that has received much attention from both the neurosurgical community and the lay press.[36,37] The results of this study challenged previous notions regarding the natural history of unruptured aneurysms and the correlation of aneurysm size to rupture risk. Because the results of ISUIA have affected contemporary neurosurgical practice, this study deserves special mention.

The ISUIA[37] is the largest study to date evaluating the natural history of unruptured aneurysms. The retrospective part of the study identified 1449 patients with 1937 unruptured aneurysms, divided into two groups: patients with no history of SAH (group 1) and those with previous SAH referable to a second aneurysm (group 2). In group 1, 32 patients among the 1449 had a documented SAH, resulting in an annual risk of rupture for a previously unruptured aneurysm less than 10 mm of 0.05% per year, far less than previous observations. The rate of rupture for aneurysms larger than 10 mm was 1% per year, size being the best predictor of rupture. Also, aneurysms located in the posterior circulation had a relatively higher risk of rupture. In patients from Group 2, the annual risk of subsequent SAH (after treatment of the aneurysm thought to have been responsible for the initial SAH) from an aneurysm smaller than 10 mm was 0.5% per year, 10 times higher than that in Group 1. Unlike Group 1, aneurysm size did not predict risk of future rupture in Group 2. Aneurysm location at the basilar bifurcation was the only factor associated with a higher risk of future SAH.

The more recent prospective limb of the ISUIA,[36] revealed an overall incidence of aneurysm rupture of 0.8% per year, which is much closer to our previous estimate. The statistical demarcation for low rupture risk in terms of size was 7 mm in the new study compared with 10 mm in the first ISUIA. There was also a shorter mean follow-up of 4 years in the recent ISUIA versus 8 years in the first. Notably, anterior circulation aneurysms (excluding posterior communicating artery [PComA] aneurysms) less than 7 mm had a 0% annual risk of rupture in group 1 and a 0.3% risk of rupture in group 2, which increased to 0.5% in both groups for aneurysms measuring 7 to 12 mm. Posterior circulation aneurysms, including PComA aneurysms, had a comparatively higher annual risk of rupture for aneurysms less than 7 mm of 0.5% and 0.7% in groups 1 and 2, respectively. The discrepancies in rupture rates between the two limbs of the ISUIA reinforce the idea that the rupture rate may have been underestimated in the first ISUIA.

The results of ISUIA have created much controversy, and the study has been criticized for possible selection bias in its retrospective cohort.[17] Because each study patient was selected for observation by a neurosurgeon, the question arises of whether patients thought to have an aneurysm with a high risk of rupture were treated and selected out of the study pool. An additional concern is that the inclusion of cavernous carotid artery aneurysms shifted the results to a lower rupture rate for all aneurysms.

Another issue is the discrepancy between the number of SAH predicted by the ISUIA and the number of SAH actually observed each year. The observed annual incidence of SAH is 30 to 40 per 100,000, which, considering the prevalence of aneurysms, would predict an aneurysm rupture rate of at least 1% per year.[11,42,43] In a Finnish series reported by Juvela et al.,[44] 142 patients with 181 unruptured intracranial aneurysms were followed for an average of 19.7 years. Because aneurysm surgery for unruptured aneurysms was not performed in Finland before 1979, all patients with unruptured aneurysms were observed during the study period, thus, eliminating a possible selection bias. Of the 142 patients, 131 had suffered a previous SAH. The overall annual incidence of hemorrhage was determined to be 1.3%. Further analysis revealed that the annual rupture rate was 2.6% in symptomatic patients, 1.3% in patients with previous SAH, and 1% in patients with incidental aneurysms. Despite having long follow-up in a stable population and no inherent surgical selection bias, the major shortcomings of this study lie in its small sample size and its analysis of patients from the pre-computed tomography (CT) or magnetic resonance imaging era.

The second study challenging the ISUIA results was performed at a single center in Japan[98] but suffers from small numbers. Sixty-two patients without previous SAH and diagnosed by angiography to have an unruptured aneurysm, were followed for at least 6 months. The risk of rupture in incidental aneurysms with no previous SAH was reported to be much higher than previously thought. The cumulative rupture risk for all aneurysms was 7.5% in 5 years and 22.1% in 10 years. The 5- and 10-year cumulative SAH rates for

aneurysms smaller than 10 mm were 4.5% and 19%, respectively. These results were similar to those from other studies but still higher than the ISUIA results. This higher incidence of hemorrhage from unruptured aneurysms was confirmed recently by a review of several published series from Japan.[66] A total of 982 patients with unruptured aneurysms were selected for conservative treatment and were followed for a total of 801 patient years. The annual rate of confirmed SAH was 2.7% and, again, larger aneurysms, symptomatic aneurysms, and posterior circulation aneurysms had a higher rate of hemorrhage. However, 11 of the 40 aneurysms that ruptured were less than 7 mm, indicating that, at least in Japan, there is no aneurysm that is free from risk of rupture. Although carefully performed, this study is a retrospective meta-analysis. Despite this limitation, it does suggest that risk of rupture is higher than suggested by the ISUIA.

Despite the criticisms and challenges to the results of ISUIA, the effects of this influential study cannot be disputed. The exact rupture rates will continue to be debated, but this study has demonstrated that the natural history of unruptured aneurysms is more benign than previously thought, at least in Europe and North America. Guidelines based on the results of the ISUIA have been published recommending observation over treatment for incidental aneurysms smaller than 10 mm in patients without a previous SAH.[5] Exceptions to this are aneurysms approaching 10 mm in diameter and those with daughter sac formations and other unique hemodynamic or morphological features. As newer data appears; however, these recommendations may need to be modified, and many experienced cerebrovascular surgeons, including the senior author, have already adopted (or returned to) a more aggressive therapeutic approach when confronted with a young, healthy patient with an unruptured aneurysm.

Patients with a family history of aneurysmal SAH also deserve special consideration for treatment because the relative risk of unruptured intracranial aneurysms is 4.2 times higher in first-degree relatives of familial intracranial aneurysm families, 6 times higher in siblings, and 1.8 times higher for those with one affected family member.[82,103]

Ruptured Aneurysms

Little has changed in our knowledge of the natural history of ruptured aneurysms during the past several years. The incidence of rupture is approximately 12 in 100,000.[89] Rupture is highest in the middle decades (mean age, 49 years in Finland[103]) and increases with age up to the 8th decade of life.[76] Despite advances in treatment and technology, approximately 50% of these patients will die or become permanently disabled after the initial SAH. Left untreated, there is an approximately 4% risk of rerupture in the first 24 hours and an approximately 20% rerupture rate during the next 2 weeks, which results in another 25 to 35% significant morbidity and mortality.

Dissecting Aneuryms

Of special interest are dissecting aneurysms. There is ongoing controversy regarding the etiology and, therefore, natural history of these aneurysms. Most authors agree that fusiform and dolichoectatic aneurysms can be divided into acute dissecting aneurysms and chronic fusiform aneurysms frequently related to atherosclerosis. There are some authors, however, who maintain that essentially all fusiform aneurysms are caused by dissection and, therefore, hold a higher risk of rupture.[13] Day et al. reviewed 102 cases of spontaneous fusiform MCA aneurysms.[13] Morphological findings in the aneurysms were observed to progress from a small focal dilation or vessel narrowing to a giant serpentine aneurysm. Hemorrhage was the most common presentation in small lesions; the incidence of bleeding progressively diminished in larger lesions. Patients with stenosis or occluded vessels most often presented with ischemic symptoms. Based on the spectrum of clinical, pathological, neuroimaging, and intraoperative findings, Day et al. proposed dissection as the underlying cause of these lesions.[13]

Dissecting aneurysms account for 4.5% of SAH cases at autopsy.[86] One series showed 10.4% of 240 posterior circulation aneurysms were dissecting aneurysms.[106] The age of incidence is similar to that of saccular aneurysms, but there is a male to female predominance of 2:1.[106]

The preponderance of dissecting aneurysms are found in the vertebrobasilar circulation. The Japanese nationwide study reviewed 357 nontraumatic dissecting aneurysms and found that 93% were found in the vertebrobasilar distribution.[107] Fifty-nine percent of these aneurysms presented with SAH and the rest with ischemia or infarction. Lateral medullary syndrome was by far the most common ischemic syndrome.[107] Severe suboccipital headache and nausea was cited by almost all patients. Vertebrobasilar lesions seem to differ in natural history from carotid lesions, rebleeding in 2 to 3 weeks, rather than acutely.[107] However, our own experience is that vertebral dissecting aneurysms frequently rebleed during the first few days.

Only 7% of all dissecting aneurysms are found in the ICA.[86,107] Dissecting aneurysms of the intracranial ICA can result in ischemia, rupture, or both. As opposed to cervical dissections, intracranial dissections do bleed because the plane of dissection is in the muscularis or subadventitial layer, rather than subintimally. Compared with those of the posterior circulation, fewer carotid dissecting aneurysms present with SAH (44% versus 59%), although carotid lesions have a higher tendency to cause thromboembolic events.[107]

The rupture rate of these aneurysms seems to be higher, but only anecdotal data exists to support this observation. The rerupture rate may be as high as 30%,[2,86] although the largest series shows a rebleed rate of only 9%.[107] Three quarters of

patients with intracranial dissecting aneurysms have a favorable outcome.[106] The mortality for carotid aneurysms (49%) was much higher than for vertebrobasilar aneurysms (22%).[107] Patients presenting with SAH tended to achieve better outcomes, probably because they were more amenable to surgical treatment.[107] Treatment of ischemic symptoms is most often conservative with anticoagulation, although this should be used with great caution in dissecting intracranial aneurysms because ischemic symptoms followed by hemorrhage are known to occur. Treatment of dissecting aneurysms presenting with hemorrhage includes sacrifice of parent vessel, trapping, wrapping, and endovascular therapy, and should be aggressive given the high risk of rerupture. Revascularization may be necessary when the posterior inferior cerebellar artery (PICA) origin has to be included in the trapping.

TREATMENT

Unruptured Aneurysms

The management of unruptured intracranial aneurysms has changed significantly in recent years and continues to evolve. In the past, the majority of unruptured aneurysms were discovered in patients presenting with SAH, cranial nerve palsies from aneurysmal mass effect, or, rarely, embolic symptoms. Now, a large number of unruptured aneurysms come to attention as truly incidental, asymptomatic aneurysms. These are often found as a result of increased use of noninvasive neuroimaging for routine evaluation of headache, dizziness, and trauma. Presently, almost one-third of intracranial aneurysms treated at our center fall into this latter category.

Three main factors have affected the management of unruptured intracranial aneurysms. Increased detection of incidental aneurysms, a revised understanding of the natural history of unruptured aneurysms, and the advent of neuroendovascular therapy have created a paradigm shift in the classic aneurysmal treatment patterns. Consequently, the neurosurgeon is now confronted with increasingly complex management decisions regarding the patient harboring an unruptured aneurysm. We will discuss the implications of these factors in the diagnosis and management of truly incidental, asymptomatic aneurysms and review the current practice at our institution.

Advances in noninvasive imaging modalities, such as CTA and MRA have markedly improved our ability to detect unruptured intracranial aneurysms. The rate of detection of aneurysms has increased from 0.3 to 2 in 100,000 person-years between 1965 and 1995.[61] CTA combines rapid injection of an iodinated contrast agent and thin-slice imaging with 3D reconstruction techniques used to improve anatomic detail. Although CTA is effective in the detection of aneurysms, its specificity and sensitivity are dependent on aneurysm size and on institutional experience. The dense bone at the cranial base limits the capabilities of CTA for detection of very small aneurysms. The overall sensitivity for detecting an aneurysm greater than 5 mm is 95 to 100%, with excellent specificity, but, for lesions smaller than 5 mm, the sensitivity ranges from 64 to 83%.[99] The sensitivity of MRA varies from 55 to 100%. This wide range is caused by variation in aneurysm size and image reconstruction techniques. As with CTA, the critical aneurysm size, below which the sensitivity decreases, is 5 mm,[102] although aneurysms as small as 2 mm are now routinely found at our institution. As the technology improves, these modalities may well supplant traditional angiography as the initial diagnostic test of choice.

We are very careful to differentiate unruptured symptomatic aneurysms from those that are truly incidental. With rare exceptions, we recommend treatment of most unruptured symptomatic aneurysms. Unruptured aneurysms become symptomatic most frequently from mass effect on the brain, cranial nerve compression, or as a result of what is probably a "small leak;" uncommonly, they present with ischemic symptoms from arterial branch occlusion or emboli from clot within the aneurysm, most frequently observed in aneurysms of the MCA. The treatment of symptomatic giant intracranial aneurysms seems to be particularly important because of their dismal natural history. After reviewing the literature, Steinberg et al. found that within 5 years of clinical presentation, approximately 80% of patients with symptomatic giant aneurysms will be disabled or dead because of cerebral or brainstem compression, thrombosis of critical arteries, or SAH.[92] The natural history of posterior circulation giant aneurysms is particularly grim, with 80% patients dying within 2 years of onset of symptoms.[16] Relative contraindications to the surgical management of symptomatic, unruptured aneurysms include advanced age, major medical comorbidities, or intracavernous aneurysms. We have learned that cavernous aneurysms, generally occurring in the older age group, have a very benign natural history, even when they present with mild cranial nerve paresis.[54] There are also situations in which the patient's presentation is relatively benign and the risks of treating the aneurysm seem to outweigh the benefits of treatment, particularly in older patients; in these cases, a conservative course of action may be preferable.

The treatment of incidental aneurysms remains controversial. There is no doubt that the results of ISUIA[36,37] have influenced our practice (although, as stated earlier, we must keep in mind more recent articles that challenge the ISUIA results). Whereas in years past, we would recommend elective treatment of essentially any unruptured aneurysm larger than 3 or 4 mm, provided the patient was relatively healthy, we now take size, location, and patient age into consideration. Other risk factors for rupture, such as smoking, hypertension, family history, previous SAH, and irregularities of the aneurysm sac must also be considered. A history of a "sentinel headache" or

development of a cranial nerve palsy weighs heavily toward surgical treatment. These factors must be balanced against the surgical risk, whether by open microsurgical clipping or endovascular coiling, before a decision regarding the recommendation of elective treatment can be made. Clearly, there can be no definitive protocol or set of rules into which we can fit each patient; nor do we have a definite cut-off size below which we would not recommend treatment. Although we are unlikely to recommend treatment of an aneurysm that is less than 3 mm, we will treat such an aneurysm, usually with bipolar coagulation, if found incidentally at surgery for another aneurysm. We are also unlikely to recommend treatment for an aneurysm less than 5 mm, unless the patient is young, has a family history of aneurysms or previously ruptured aneurysm, and we think that the risk of treatment is minimal. We will generally recommend treatment of an aneurysm larger than 5 mm, provided the risk of treatment seems to be low; however, we may stretch the age limit and the tolerance for comorbidities in a patient with an aneurysm at the origin of the PComA because we know from the ISUIA study that those aneurysms have a higher risk of rupture than others in the anterior circulation and the risk of surgical treatment at this location is very low in our hands.

We do not have age cutoffs regarding offering treatment, but we do consider life expectancy in estimating the benefit of treatment. In general, benefit seems to be outweighed by the risk of treatment if the remaining life expectancy of the patient falls below 20 years.[64] In this context, "young" and "old" are arbitrary terms, once again reinforcing the individualization of treatment.

In terms of open surgical treatment versus endovascular treatment of unruptured aneurysms, the senior author recommends open surgical treatment for the majority (at least 80 to 85%) of patients, given the very good experience in terms of surgical morbidity and the lack of long-term results of permanency of endovascular treatment. A meta-analysis of 28 articles between 1966 and 1992, containing data on 733 patients with unruptured intracranial aneurysms, found that surgical morbidity and mortality rates were 4.1% and 1%, respectively,[50] and these figures are generally concordant with our experience. However, we are aware of the much higher morbidity found in the ISUIA study[36] and admit that we have not performed formal long-term psychometric studies in our patients.

Solomon et al. demonstrated that size, and not location, of the aneurysm is the main variable predicting the incidence of complications during surgery for unruptured intracranial aneurysms.[90] In properly selected patients, unruptured aneurysms less than 10 mm in size can be surgically cured, with a risk of major morbidity of approximately 1%. Surgical treatment of intermediate-sized aneurysms, between 10 and 25 mm in diameter, carries an approximately 5% risk of major morbidity. Conversely, aneurysms greater than 25 mm in diameter carry a 20% risk of significant surgical morbidity or poor outcome after surgery. In short, in unruptured aneurysms less than 25 mm in diameter, essentially 95% of patients can expect a satisfactory outcome, with cure of the aneurysm when operated on by an experienced aneurysm surgeon.[90] A meta-analysis by Raaymaker et al.,[79] including 2568 unruptured aneurysms, supports this with an overall morbidity of 10.9% and a mortality of 2.6% (the preponderance of poor outcomes from the treatment of large and giant aneurysms). The treatment of a subset of small anterior circulation lesions carried 1.9% and 0.8% morbidity and mortality. Our experience is similar except that, in our hands, clipping of basilar tip aneurysms has been accompanied by substantial morbidity, as will be discussed.

Although surgical clipping for unruptured aneurysms remains our standard of care, endovascular treatment may be preferable for unruptured aneurysms in certain locations, such as the basilar tip. In the hands of the senior author, surgical clipping of basilar tip aneurysms (including ruptured and giant) carries an overall 23.8% morbidity and 4.1% mortality, comparable with other reports in the literature. Coiling of basilar tip aneurysms, however, has a procedure-related morbidity and mortality of 4.1% and 1.4%, respectively, and relative durability of complete coiling in 71%.[97] Therefore, if treatment of a basilar tip aneurysm is indicated, endovascular coiling is most frequently our choice, although we remain open-minded in expectation of long-term results of the durability of coiling. Another location where we have had considerable surgical difficulty is with superior hypophyseal aneurysms, particularly those of larger size. As the endovascular results have been good at this location, we choose this latter approach most frequently for these aneurysms.

Regardless of treatment modality, given the benign natural history of small, unruptured aneurysms, we are constantly aware that if treatment is to be performed, it must be performed with the expectation of minimal morbidity. For this reason, when we offer open surgical treatment to a patient with an unruptured aneurysm, we do so with the expectation that the risk will be very small. If, however, at any point before definitive treatment (clipping in the case of open surgery) we find that the risk would be higher than estimated, because of, for example, calcification at the base of the aneurysm or other morphological predictors of higher risk, we do not hesitate to "back off" and either treat the patient conservatively or by the endovascular route (*Fig. 16.1*). Needless to say, the same applies if the first choice of treatment is endovascular, in that, if that procedure does not go as smoothly as predicted, we recommend to our endovascular colleagues to "back off" and consider surgical or conservative treatment.

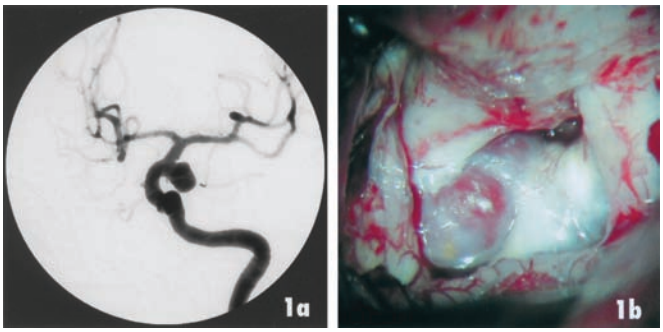

FIGURE 16.1. *A,* anteroposterior (AP) angiogram of a healthy 63-year-old woman, showing an unruptured left posterior communicating artery aneurysm. *B,* surgical view of the aneurysm showing a broad, heavily calcified neck that precluded safe clip placement. The intraoperative decision was made to "back off" and the patient was later sent for endovascular treatment.

Ruptured Aneurysms

Timing of Surgery

Little has changed since the landmark International Cooperative Study on the Timing of Aneurysm Surgery.[49] Before this, surgery on any patient with a decreased level of consciousness was usually deferred until after 2 weeks because outcomes were so poor with early surgery.[89] Vasospasm and rebleeding are the leading causes of morbidity and mortality after the initial hemorrhage. Part one of the Cooperative study showed that predictors for mortality included decreased level of consciousness, older age, thickness of clot on CT scan, medical comorbidities, and basilar aneurysms.[48] Sixty-eight centers contributed 3521 patients to assess the best timing of surgical clipping after SAH. No difference was found between early surgical outcomes (<3 days after SAH) and late outcomes (11 to 14 days), whereas the outcome was clearly worse in those undergoing surgery in the intermediate period.[49] Mortality was 10 to 12% in alert patients and 21 to 25% in drowsy patients undergoing surgery before day 11 compared with 3 to 5% in alert patients and 7 to 10% in drowsy patients on or following day 11.[49] Given the risk of rebleeding, early surgery on good Hunt and Hess grade patients has become the standard. With the advent of endovascular treatment, early coiling of poor Hunt and Hess grade patients is desirable, rather than waiting until the late period for surgical treatment.

Because medicine cannot be performed by algorithms, exceptions to early treatment exist. Patients who present late (after Day 3), have vasospasm on early angiogram, and have aneurysms not amenable to coiling may need to be deferred for surgical treatment until after 12 to 14 days, when the risk of vasospasm is resolved. Similarly, in our opinion, surgical treatment of patients with poor-grade aneurysms (Hunt and Hess Grades 4 and 5) with aneurysms unfavorable for coiling should be deferred until after the period of maximal vasospasm, although, at many centers, all patients, regardless of grade, are either coiled or clipped early.

Management: Surgery Versus Endovascular

Surgical clipping as the "gold standard" for all ruptured aneurysms has been called into question by the results of the International Subarachnoid Aneurysm Trial (ISAT).[38] Nevertheless, until more prospective and long-term data is available regarding the durability and long-term effectiveness of coiling, open surgery should still be considered for many patients with SAH because it remains safe and exceedingly effective. When considering surgical morbidity, operative complications must be separated from complications of the disease itself, such as vasospasm, hydrocephalus, and medical illness. The Cooperative study reports surgical complications in 5% of cases.[49] A more recent look at this issue by McLaughlin and Bojanowski[60] shows a slightly higher complication rate, with 6.3% of patients suffering cerebral edema and hemorrhagic contusions as a result of surgery, and 2.3% with cranial nerve palsies, but 75.9% of these patients had good functional recovery. Surgical mortality was only 0.7%. Our experience is similar with a serious morbidity of approximately 5% from surgical complications in a recent series of mostly anterior circulation aneurysms.

Since its approval by the Food and Drug Administration in 1991, there has been a steady increase in coil embolization of intracranial aneurysms. Although there are serious risks associated with coil embolization, it has proven to be a relatively safe and effective modality (at least in early follow-up) for treating patients with both ruptured and unruptured aneurysms. Concerns regarding endovascular treatment focus on its durability and the potential for aneurysm recanalization from coil compaction. The rapid pace of development in coil and microcatheter technology further complicates adequate assessment of the current literature. When coil embolization is considered for treatment, the risks associated with angiography as well as the risk of the embolization must be considered, given that follow-up angiography is necessary on a regular basis for those treated with coiling. The combined risk of permanent and transient neurological complications for patients with aneurysms or arteriovenous malformations without SAH was 0.3% from angiography.[9] A large meta-analysis showed the thromboembolic rate during aneurysm coiling with Guglielmi detachable coils (GDC) to be 8.2%.[78]

According to the majority of recently published endovascular series, complete or near-complete aneurysm occlusion is achieved in 70 to 90% of patients.[28,36,41,67] Because small aneurysm neck remnants, recanalization, and coil compaction are not uncommon after endovascular therapy, angiography is routinely performed within 6 months of completing the coiling. These examinations reveal some degree of aneurysm recanalization in 50% of all coiled aneurysms and

up to 90% of large and giant aneurysms.[26,67] Recanalization carries a risk of aneurysm rupture, which may necessitate secondary endovascular or surgical treatment.[58] Up to 15 to 20% of patients may require more than one endovascular treatment session.[53] As coil technology improves, aneurysms can be packed more tightly, with progressively lower risk of recanalization. At our institution, follow-up angiography is performed at 6 and 18 months and no ruptured aneurysm during the past 2.5 years has required recoiling.

Murayama et al. reported the University of California, Los Angeles experience of all patients undergoing coil embolization between 1990 and 2002.[67] Because of advances in technology and technique, the group was split into two cohorts: early (1990–1995) and late (1996–2002). Treatment was extended to 818 patients harboring 916 aneurysms, 58% of which were ruptured. They compared the results of embolization depending on the aneurysm and neck size. Overall, complete occlusion was performed in 55%. When broken down, coiling was more successful in small aneurysm with a small neck (75%) and least successful in giant aneurysms (26%). Overall morbidity and mortality was 6% and 3.4%, respectively. Only 53% of patients had follow-up angiography, but the recanalization rate rose precipitously with increased aneurysm and neck size (5.1% overall for small size/small neck and 59.1% overall for giant). Rupture after coiling was rare but not insignificant and improved markedly from the early to the late group. The early group had nine delayed ruptures (4.1% incidence) versus the late group with three delayed ruptures (0.5% incidence).

Henkes et al. performed a single institution, nonrandomized, retrospective review of all aneurysm patients treated with endovascular therapy at a single institution between 1992 and 2003.[28] Of the 1579 patients with 2150 intracranial aneurysms who were enrolled, 1811 (84.2%) aneurysms were treated with coil occlusion, 152 (7.1%) with surgical obliteration, and 187 (8.7%) went untreated. There was a slightly higher preponderance of ruptured aneurysms (55%) compared with unruptured aneurysms. Anterior circulation aneurysms comprised 67.4% and posterior circulation aneurysms made up 32.6%. Overall, rates were good with complete occlusion in 65.8% and near-complete occlusion in 20.7%. Only 2.9% failed coiling. The peri-procedural complication rate was 17.4% with a permanent morbidity and mortality rate of 6.8%. As expected, there were lower occlusion rates in proximal ICA aneurysms, basilar aneurysms, and in larger and wide-necked aneurysms. Higher occlusion rates resulted with the use of 3D and fibered coils but higher complication rates were found when more complex techniques, such as aneurysmal neck remodeling and stent deployment, were needed.

There is limited data directly comparing surgical clipping to endovascular coiling of unruptured aneurysms, especially regarding long-term results. According to the prospective but nonrandomized arm of the ISUIA, the combined morbidity and mortality for surgery at 1 year was 12.2% compared with 9.5% for coiling.[36] These results must be considered in the context of significant underrepresentation of endovascular treatment and selection bias.

Johnston et al.[40] compared the outcome between coil embolization and surgical clipping in 216 patients who were retrospectively judged in a blinded fashion to have been eligible for either surgical or endovascular treatment. Of these patients, 118 were treated by surgery and 98 by coil embolization. In this study, surgery was found to be associated with greater rates of early and persistent disability and more procedure-related complications.

The ISAT was a multicenter, randomized study comparing the safety and efficacy of endovascular coiling to surgical clipping in patients with ruptured aneurysms.[38] Of the 9278 patients considered for the trial, 2143 patients were randomized for coiling or clipping. The other 7135 patients were excluded because their aneurysms were considered to be treated optimally by only one modality. Surgery was determined to be more appropriate for the majority of these excluded aneurysms, except for basilar apex aneurysms, most of which were coiled. Interim analysis of the randomized group revealed a relative risk reduction of 22.6% and an absolute risk reduction of 6.9% of dependency or death at 1 year with endovascular treatment when compared with surgical treatment. During the relatively short follow-up, 2.6% of patients in the endovascular group suffered bleeding after treatment compared with 0.9% of patients in the surgical group.[38] We look forward to the long-term follow-up from this study.

The ISAT study clearly showed that for small, ruptured, anterior circulation aneurysms that are suitable for both surgery and coiling, endovascular therapy has a significant benefit in outcome at 1 year.[38] However, these results cannot be generalized to all aneurysms, because MCA aneurysms, large/giant aneurysms, and posterior circulation aneurysms were, to varying degrees, underrepresented. Importantly, these results cannot be applied to unruptured aneurysms. Long-term rebleeding rates for both surgery and endovascular therapy must be determined by following these patients for many years, as the study investigators are planning to do. Preliminary results from this study suggest that although re-hemorrhage rates after the first year of treatment may be very low for both endovascular and surgical therapy, the risk of rebleeding is clearly higher after coiling and, if this trend were to continue, the early advantage of coiling may be overcome. Qureshi et al. have addressed the issue of long-term follow-up through the use of a computer-generated intention-to-treat model in which reasonable assumptions regarding each therapy's risk and efficacy were taken from the literature.[77] This model predicted that studies such as

ISAT would need follow-up of up to 10 years to accurately determine which therapy proves superior over time.

PERSONAL PRACTICE PATTERNS

The number of incidental aneurysms referred to our institution has been rising steadily in recent years, most likely because of the increased number of noninvasive imaging studies performed. The combined effect of increased incidence of unruptured aneurysms and increased endovascular therapy for ruptured aneurysms has resulted in a shift in our surgical practice. Proportionately, we are operating on more unruptured aneurysms and fewer ruptured aneurysms. In rough numbers, of the approximately 200 patients with aneurysms we are currently treating each year, approximately 50 to 60% now present with SAH. Thirty-five percent of the patients at our institution with ruptured aneurysms are treated surgically and the other 65% endovascularly. In the senior author's practice, current indications for surgery on ruptured aneurysms include a good Hunt and Hess grade (1, 2, and some "good" grade 3), patient age (we favor endovascular treatment in elderly patients), early or late presentation ($<$3 days or $>$10 days) and aneurysms not favorable for coiling (i.e., MCA aneurysms and some of the larger, broad-based aneurysms). Because we are a referral center, many of our patients come on a delayed basis and, because of our location, we see an increasingly aged population.

Of the patients presenting to our institution with unruptured aneurysms, 75 to 85% are treated surgically. The exceptions are basilar apex aneurysms, most of which are treated with coiling and superior hypophyseal aneurysms, most of which present surgical difficulties that are not encountered with endovascular therapy, as previously discussed. Durability of treatment is most germane to the discussion of unruptured aneurysms, especially in young patients with a long life expectancy. Safety and early efficacy have been demonstrated with coiling, however, durability of treatment is an additional factor to consider in the management of any asymptomatic lesion. Our long experience with microsurgical clipping clearly indicates that once an aneurysm is clipped, it is extremely unlikely, although not impossible, for it to bleed in the future. However, we must admit that long-term angiographic follow-up generally has not been as rigorous in surgically treated patients as we currently expect of patients treated endovascularly.

In our practice, long-term angiographic follow-up has not been recommended for the vast majority of patients whose aneurysms seem to have been clipped completely by intraoperative angiography, postoperative angiography, or by direct microsurgical observation. Generally, we recommend long-term angiographic follow-up in young patients with a psychological profile of minimal anxiety, patients with a family history of aneurysms, and in patients with known "dog ear" residua. We could certainly be criticized for this policy because, even in the most experienced hands, there is a very small but real risk of approximately 1.5% of aneurysm regrowth after complete clipping during a 4-year period.[12] Furthermore, the incidence of spontaneous de novo aneurysm development falls between 1% and 1.8%.[12,62] For those patients left with "dog-ear" residua, approximately 25% can show enlargement over several years.[12]

Our practice of avoiding follow-up angiography after an apparently complete clipping balances the very small risk of aneurysmal regrowth or de novo aneurysm formation with the adverse psychological effects of telling the patient that follow-up angiography is indicated because their aneurysm may "grow back" or another aneurysm may develop.

PITFALLS, COMPLICATIONS, AND THEIR PREVENTION

In this section, we discuss, with relative informality, the most common problems encountered in the senior author's experience with open surgical treatment of intracranial aneurysms. These "pitfalls" account for the great majority of the surgical morbidity tabulated in *Tables 16.1* through *16.3*. We discuss each aneurysmal site separately because these pitfalls are quite specific to each different location.

Intracavernous

The main source of morbidity in the treatment of intracavernous aneurysms has been overly aggressive treatment. In our early experience, we considered these aneurysms, particularly when they were large, relatively dangerous and treated them aggressively, including several cases of direct intracavernous approach after the enthusiasm generated

TABLE 16.1. Surgical results (RC Heros) from 1981 to 1998 (giant aneurysms in parentheses)

Location	Good	Poor	Dead	Total	Serious morbidity	Mortality
Carotid	495 (92)	23 (12)	6 (3)	524 (107)	4.4% (11.2%)	1.1% (2.8%)
Anterior cerebral	297 (6)	16 (5)	4 (0)	317 (11)	5.0% (45.4%)	1.3% (0%)
Middle cerebral	277 (41)	17 (9)	4 (2)	298 (52)	5.7% (17.3%)	1.3% (3.8%)
Posterior circulation	104 (16)	27 (8)	5 (4)	136 (28)	19.9% (28.6%)	3.7% (14.3%)

TABLE 16.2. Surgical results (RC Heros) from 1999 to 2004 (giant aneurysms in parentheses)

Location	Good	Poor	Dead	Total	Serious morbidity	Mortality
Carotid	115 (4)	0 (0)	2 (0)	117 (4)	0% (0%)	1.7% (0%)
Anterior cerebral	71 (0)	5 (0)	1 (0)	77 (1)	6.5% (0%)	1.3% (0%)
Middle cerebral	77 (0)	3 (0)	3 (0)	83 (1)	3.6% (0%)	3.6% (0%)
Posterior circulation	15 (0)	0 (0)	0 (0)	15 (0)	0% (0%)	0% (0%)

TABLE 16.3. Overall Surgical Morbidity and Mortality (RC Heros) from 1981 to 2004 (giant aneurysms in parentheses)

Location	Total	Serious morbidity	Mortality
Carotid	641 (111)	3.6% (10.8%)	1.2% (2.7%)
Anterior cerebral	394 (12)	5.3% (41.7%)	1.3% (0%)
Middle cerebral	381 (53)	5.2% (17.0%)	1.8% (3.8%)
Posterior circulation	151 (28)	17.9% (28.6%)	3.3% (32.1%)
Overall	1567 (204)	5.8% (16.7%)	1.6% (6.9%)

by Dolenc.[14] These attempts were quickly abandoned because of unacceptable cranial nerve morbidity. In our early enthusiasm for the extracranial-to-intracranial bypass, we treated a number of these patients with a bypass followed by ligation of the ICA at its origin. We encountered significant problems with thromboembolic events despite a patent bypass graft, and attributed these events to the formation of thrombus in the long, blind segment of the ICA, which then propagated intracranially and embolized distally. Our cases, as well as others informally collected from colleagues, were reported.[29]

We have now learned that the natural history of intracavernous aneurysms is relatively benign.[54] For this reason, in addition to the morbidity of treatment for these aneurysms, we follow a more conservative policy of observation on asymptomatic or minimally symptomatic patients unless their aneurysm is already of very large or giant size. Patients with very large or giant aneurysms or patients who were significantly symptomatic were then preferentially treated by common carotid occlusion with excellent results.[94] Although not discarding common carotid occlusion as a very safe and effective treatment for these patients, we have moved to a policy of assessing these patients with a balloon test occlusion and then proceeding to endovascular occlusion of the aneurysm and frequently the ICA at the neck of the aneurysm without a bypass if they tolerated the balloon test occlusion. In the case of a failed balloon test occlusion, we only perform a bypass graft with aneurysm occlusion (surgical or endovascular trapping) if the patient is young with debilitating symptoms.

PARACLINOID ANEURYSMS

The surgical difficulties with paraclinoid aneurysms are twofold; those related to the optic nerve and those related to compromise of the ICA. Early on, we had significant morbidity, including blindness, related to the ipsilateral optic nerve, particularly in patients who presented with visual problems from optic nerve compression by the aneurysm.[32] We thought that these early problems were mostly related to insufficient drilling with complete uncovering of the optic nerve. Now, we completely unroof the optic nerve and free it both laterally (including, of course, removal of the optic strut) and medially (at the expense of sometimes entering the sphenoid or ethmoid air cells). This allows the optic nerve to be mobilized freely to expose and clip the neck of the aneurysm. We may have also injured the optic nerves through the use of bipolar cautery for dural opening. Now we cut the dura without the use of any cautery to expose the anterior clinoid and unroof the optic canal. Additionally, we used to use the diamond drill around the optic nerve with insufficient irrigation. Nowadays, we favor the cutting drill for most of the removal of the anterior clinoid and use the diamond drill only sparsely, with profuse irrigation to avoid excessive heat production and damage of the optic nerve.

Some of our early problems were caused by attempts at clip placement without appropriate decompression of the aneurysm, which we now know facilitates the clipping of previously unclippable aneurysms.[4] We have also had some serious ischemic problems related to kinking and even occlusion of the ICA before the routine use of intraoperative

angiography (*Fig. 16.2*). Another problem is related to the use of clips that are too long, inserted with the tips aimed medially in a blind fashion, which, in at least one patient, resulted in diabetes insipidus from damage to the pituitary stalk.

A tragic mortality occurred because of an unrecognized paraclinoid aneurysm that pointed superolaterally beneath the anterior clinoid instead of pointing in the usual direction (superomedially, in the case of ophthalmic aneurysms, and posteromedially, in the case of superior hypophyseal aneurysms). This small aneurysm had an unusual shape, like a mushroom, with a broad and flattened dome. This shape was the consequence of the aneurysm abutting the inferior surface of the anterior clinoid, which limited its superior growth. The aneurysm had clearly eroded the anterior clinoid process which had produced a "punched-out" defect that we did not recognize in the available preoperative CT scan (*Fig. 16.3*). After drilling the anterior clinoid, massive hemorrhage ensued. Because we were unprepared, without cervical carotid exposure, we attempted to stop the bleeding by forcibly packing with Surgicel, resulting in accidental occlusion of the carotid artery and fatal stroke. We labeled this type of projection of a paraclinoid aneurysm "sub-clinoid" to emphasize the potential dangers inherent to this type of projection.[52]

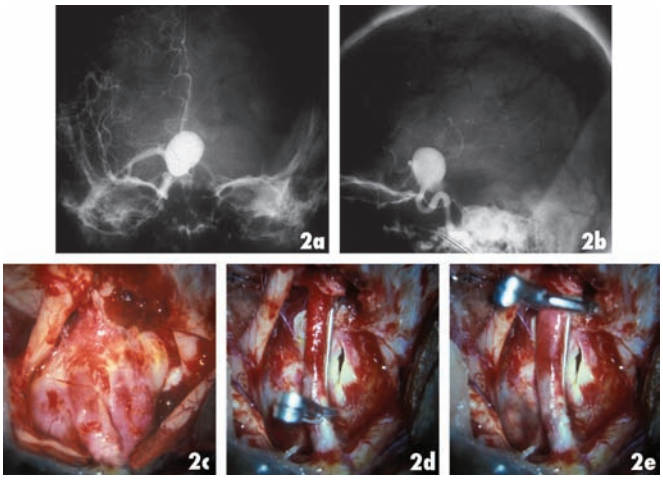

FIGURE 16.2. *A* and *B*, AP and lateral angiogram showing a posterior carotid wall aneurysm. *C* and *D*, at surgery, after drilling the anterior clinoid and temporary clipping of the internal carotid, aneurysmorrhaphy and clipping was performed on the heavily calcified neck. The carotid seemed to be slightly narrowed but the surgeon thought that, under the microscope, there was good flow. No intraoperative angiogram was done. *E*, postoperatively, the patient was found to be hemiparetic. She was taken back to the operating room, where, in retrospect, there was luminal narrowing of the carotid. The clip was replaced, with complete recovery of the patient's neurological deficits.

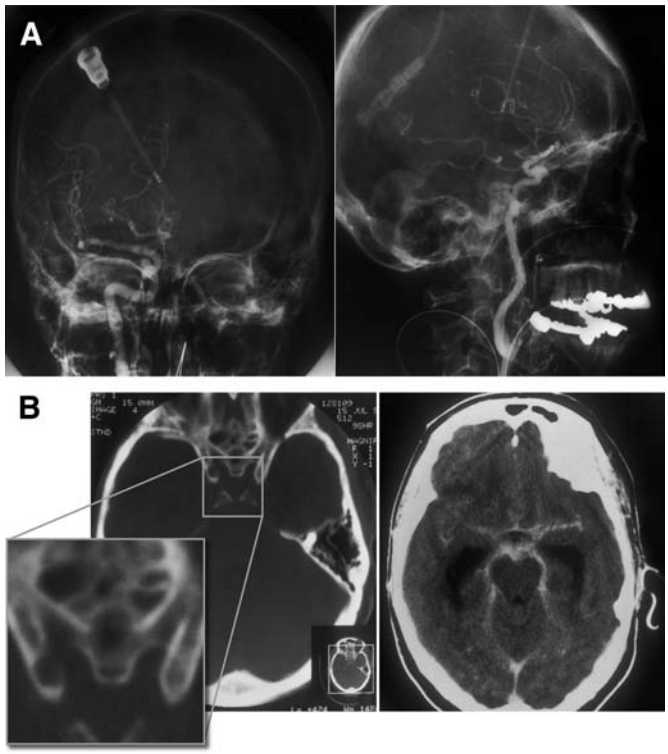

FIGURE 16.3. *A*, AP and lateral angiogram showing a ruptured "subclinoid" aneurysm pointing laterally with unusual flattening of the dome. The aneurysm was ruptured during drilling of the anterior clinoid. *B*, CT showing SAH and the eroded anterior clinoid process with "punched out" defect. This was not recognized preoperatively.

Supraclinoid Aneurysms

With PComA aneurysms, our most serious, but preventable, problem is premature rupture of the aneurysm from temporal lobe retraction. Along with other reports, we have emphasized that when the aneurysm points laterally and the third nerve is not involved, it is most likely that the aneurysm projects over the edge of the tentorium and will be stuck to the temporal lobe. We had several cases of premature rupture early in our experience before we learned that this problem can be avoided by positioning the patient with only slight head turn and using only minimal subfrontal retraction without any manipulation of the temporal lobe. When necessary, the sylvian fissure can also be opened completely in the same manner, avoiding retraction of the temporal lobe. The more the head is turned, the more the temporal lobe will be covering the carotid artery and the aneurysm, and the temptation will exist for the less-experienced operator to retract the temporal lobe to visualize the aneurysmal anatomy better. We also emphasize constantly to our younger colleagues that the carotid artery should never be retracted away from the dome of the aneurysm because the dome is likely to be stuck to some fixed structure. This medial retraction of the carotid

artery, away from the aneurysm, is particularly tempting when the surgeon is trying to visualize the origin of the PComA below and medial to the aneurysm. Sometimes all the surgeon sees is a small "shoulder" at the origin of the PComA and, frequently, this is all that is necessary. If better visualization of the PComA becomes desirable, the surgeon can change the angle of the microscope to a more medial to lateral direction and, by lifting the ICA *toward* the aneurysm (never away from the dome), the PComA can be visualized very well from the opticocarotid corridor. We always use this corridor after clipping to confirm the integrity of the PComA and associated perforators after the clip is applied. A problem that we have encountered on a couple of occasions with PComA and anterior choroidal artery (AChA) aneurysms is oculomotor damage from inappropriate application of the clip without previous clear identification of the oculomotor nerve. This is particularly a problem if the aneurysm is relatively large and because of the recent rupture, the surgeon is reluctant to do much dissection before the application of the clip. The problem can also be related to inserting an excessively long clip blindly in a medial-to-lateral direction in fear of occluding the PComA or AChA and their perforators.

With AChA aneurysms, the surgeon must be particularly thorough in identifying the anatomy around the aneurysm and looking for a "second" choroidal artery. As we know well, the AChA is frequently duplicated and at times, triplicated. We have had cases of inadvertent occlusion of a "second" choroidal artery from complacency in applying the clip before thorough identification of the distal aspect of the neck after identifying what we thought was a single AChA proximal to the aneurysm. AChA tend to project more laterally and, of course, superiorly to PComA aneurysms, and the problem of adhesion to the temporal lobe and potential rupture with temporal retraction is even more important with AChA aneurysms. Although we frequently clip a simple PComA aneurysm without thorough opening of the sylvian fissure, we think that, for AChA aneurysms, it is always advantageous to completely open the sylvian fissure, taking particular care not to produce any temporal lobe retraction to avoid aneurysmal rupture. Another problem that we have encountered, and often not recognized at the time of surgery, is occlusion of perforating branches from the AChA, while clipping an AChA aneurysm without proper dissection of the superior aspect of the neck.

With carotid bifurcation aneurysms, we have had problems from kinking, and in one case, occlusion of the anterior cerebral artery (ACA) from application of the clip in a perpendicular direction to the long axis of the neck. It is, of course, a well-known principle of aneurysm surgery that the clip must be applied whenever possible parallel to the larger longitudinal axis of the neck. To do this in ICA bifurcation aneurysms, the sylvian fissure must be opened completely so that the surgeon can apply the clip in the direction of the MCA, which is usually the long axis of the neck of the aneurysm. At the ICA bifurcation, the surgeon must be particularly careful with dissection of the perforators that come from the origin of the A1 and the M1. The perforators in the "opposite" side of the neck coming from the origin of the A1 are particularly difficult to visualize with large aneurysms and, for that reason, we avoid small bony exposures and prefer to have a relatively wide craniotomy at the base so that we can change the angle of the microscope to a more mediolateral direction to be able to visualize better the neck between the A1 and the aneurysm. Perforators coursing posterior to the bifurcation are also a problem and need to be visualized and separated from the aneurysm by looking behind the aneurysm, usually from the direction of the middle cerebral artery. We recall a case in which the recurrent artery of Huebner was inadvertently included in the clip. This artery courses posterior to the bifurcation in a direction parallel to the ACA and MCA, and it is easy to include it in the clip, particularly if the surgeon uses an excessively long clip. One of our most spectacular intraoperative ruptures occurred when we applied an excessively long clip in an anterior-to-posterior direction and tore the origin of the basal vein of Rosenthal with the clip. Because we did not understand where the bleeding was coming from, we made the mistake of opening the clip widely to remove it and, because the tips of the clip were in the vein of Rosenthal, this wide opening of the clip made the situation much worse. Although the surgeon's first reaction when bleeding is encountered on application of the clip is to open the clip and remove it, this is usually not the best course of action and it is best to complete closure of the clip and inspect the situation or, if the suspicion is that one blade of the clip is into the aneurysm, to move the clip out without any further opening of the blades.

Blister-like Aneurysms

Blister-like aneurysms deserve special mention and have received considerable recent attention, particularly in papers from Japan.[69,72,110] These aneurysms generally occur in the supraclinoid portion of the ICA, although they can occur also in the paraclinoid portion, in relation to the distal ring. The distinguishing characteristic of these aneurysms is that they occur at nonbranching sites of the artery. In addition, they have a tendency to occur on the anterior or lateral surface of the ICA and tend to have a "blister-like" appearance or broad-based irregular shape. At times, the entire supraclinoid portion of the ICA looks irregular and abnormal. Although this aneurysmal subtype has been referred to as blister-like, anterior wall, and dorsal wall, perhaps the best designation may be ICA trunk aneurysms, to best express the diversity of this entity.

The incidence of blister aneurysms is low, ranging from 0.9 to 6.5% of all ICA aneurysms.[72,110] There is a female dominance and high association with hypertension, arterio-

sclerosis, and dissection. Several authors think that these are essentially dissecting aneurysms.[69]

We have operated on several of these relatively small aneurysms and have encountered more problems than expected. One case bled massively at surgery when the aneurysm was approached and carotid sacrifice became necessary (which, fortunately, was well tolerated). In another case, a tightly applied encircling clip caused narrowing and kinking of the carotid artery. We elected to leave this alone, and despite several postoperative transient ischemic attacks, the patient eventually did well. Another case treated by an encircling clip initially showed perfect angiographic results but, after some time, regrowth of the aneurysm distal to the clip occurred. This required treatment with carotid sacrifice after a successful balloon test occlusion. The best way to prevent these problems is preoperative recognition. When we recognize these aneurysms, we perform a balloon test occlusion with a hypotensive challenge, to ensure that the patient would tolerate carotid occlusion if such became necessary at surgery. For these aneurysms, we always expose the cervical ICA, even though it may seem that the surgeon would have a space for intracranial temporary proximal occlusion. The reason for this is that, frequently, the abnormality in the wall of the artery extends more proximally and involves the arterial wall more extensively than the angiogram would suggest. We make sure that we always have the Sundt graft encircling clips of various sizes and lengths available for these operations because, frequently, they are the only solution to these aneurysms. At surgery, we are extremely careful in "peeling away" the clot and fibrinous tissue frequently encountered over the aneurysmal dome, because this can lead to rupture. Some degree of "peeling off" of this clot is necessary, however, to be able to apply the encircling clip without constricting the lumen of the carotid artery. We are particularly careful to make sure that the encircling clip involves *all* of the abnormal segment of the artery and, frequently, this necessitates occlusion of the origin of the PComA (which is usually well tolerated if there is communication with the posterior cerebral artery and a good P1 segment). The AChA origin, of course, must be preserved. This may require several adjustments of the clip or the use of two different clips encircling the artery above and below the origin of the AChA. Finally, intraoperative angiography is essential in these cases to assess carotid patency. We are hopeful and optimistic regarding the fact that with improvements in intracranial stents, these aneurysms will be treated mostly endovascularly in the future.

Anterior Cerebral Territory Aneurysms

AComA aneurysms that point anteriorly and inferiorly are usually much "easier" because they point away from the important anatomy (the A2s and the perforators). However, one potential pitfall with this aneurysmal projection is early rupture from frontal retraction if the aneurysm dome is stuck to either the planum sphenoidale or the ipsilateral optic nerve. Frequently, this situation can be suspected preoperatively from an abnormal "flattening" of the dome of the aneurysm against the basal structures. This is an important clue because, now, with excellent subtracted angiographic films, the surgeon frequently does not see the bony anatomy to visualize the relation of the dome of the aneurysm to the anterior cranial base. We had a memorable rupture of an aneurysm under these circumstances and reported that case because it resulted in a very large contralateral subdural hematoma that we could not recognize at surgery. Although we were able to clip the aneurysm successfully by rapidly developing a transcerebral trajectory to the aneurysm with suction, we did not recognize the contralateral subdural hematoma causing massive herniation of the ipsilateral side of the brain through the dural opening. We rapidly closed the wound and obtained an immediate CT scan, revealing the hematoma, which was then successfully removed.[24] This problem can be avoided by being particularly careful not to retract the frontal lobe in AComA aneurysms that project anteroinferiorally. With wide opening of the sylvian fissure, the surgeon can approach these aneurysms from a more-temporal direction and, then, as the aneurysm anatomy is approached, the surgeon should work through the gyrus rectus to identify clearly the anatomy at the neck of the aneurysm without any retraction in the frontal lobe.

Needless to say, we have had considerable morbidity from perforator injury with difficult AComA aneurysms, particularly those that project backwards or, as they frequently do, those with a bilobed configuration with one lobe projecting superiorly between the A2s and one lobe projecting posteriorly, among the perforators. The resulting problems with memory, personality changes, and mutism are well known, and, although frequently these problems improve, we recall a few patients who remained permanently incapacitated. We can only advise that the entire aneurysmal complex must be well dissected so that the surgeon can visualize the perforators and ensure that they are free from the blades of the clip (before and after clip application). This complete dissection of the aneurysmal complex could have prevented one of our important complications, which was the occlusion of a "third" A2 that took origin directly from the aneurysmal complex. We were satisfied after visualizing an A2 on each side of the aneurysm and applied the clip, failing to achieve a complete dissection of the complex after clip application. A third A2, present but unrecognized in the preoperative angiogram, was clearly absent on the postoperative angiogram performed after the development of a frontal lobe infarction. Some of our other problems, including the late recognition of an unclipped portion of a bilobed aneurysm after a rebleed, could have been prevented with more liberal or routine use of intraoperative angiography.

With superiorly projecting AComA aneurysms, one or both A2s can be densely adherent to the neck of the aneurysm, and we have had a few cases of serious intraoperative rupture during neck dissection. With a relatively wide exposure of the aneurysmal complex through the gyrus rectus, we now frequently stop shy of complete dissection of the neck and apply a fenestrated clip longitudinally in a direction parallel to the AComA artery. This leaves the ipsilateral A2 in the fenestration of the clip and allows exact clip placement across the neck, even if the exposure was incompletely developed between the A1 and the aneurysm. It is important that the clip is long enough to occlude the neck completely, but short enough to avoid contralateral A2 compromise. Incidentally, with recently ruptured AComA aneurysms, we frequently place a temporary clip on the opposite A1 (usually the smaller one because we generally approach these aneurysms from the side of the dominant A1) during the early stages of aneurysmal dissection. With this maneuver, a second temporary clip can be rapidly applied to the ipsilateral dominant A1, which is always exposed early on through the sylvian fissure, in case of an inadvertent rupture.

With pericallosal aneurysms, we recall cases in which a small craniotomy flap was too superior and the neck of the aneurysm was under the genu of the corpus callosum, making it difficult to achieve proximal control. This can be avoided by carefully studying the preoperative angiographic anatomy and using a more anterior inferior bone flap for more proximal aneurysms. The other problem that we have had with pericallosal aneurysms is rupture from an interhemispheric exposure with retraction on the side to which the aneurysm dome was stuck. This can be avoided if the surgeon, on recognizing that the freshly ruptured aneurysm is stuck to the brain, proceeds to expose the aneurysmal anatomy through a small subpial dissection through one cingulate gyrus. The aneurysmal neck can then be exposed subpially without undue retraction.

Middle Cerebral Aneurysms

The main problem that we have encountered with proximally located MCA aneurysms (M1 trunk) is perforator occlusion. These aneurysms are almost invariably associated with the origin of one of the important perforators from the M1, which must be identified and protected. Even when a large perforator proximal to the aneurysm is identified, the surgeon is still at risk of occluding other perforators that may be stuck to the posterior aspect of the aneurysm. There is no alternative to complete dissection of the neck of the aneurysm in these cases. With aneurysms at the bifurcation, most of the problems have occurred with large and giant aneurysms. The smaller aneurysms are not usually a problem because the surgeon has the luxury of a wide exposure through the sylvian fissure, allowing for complete mobilization and application of the clip from a variety of different angles. We have comfortably achieved this same wide exposure by working through the superior temporal gyrus in cases of fresh rupture when the sylvian fissure may be difficult to open because of edematous brain and/or congested sylvian veins.[33,73] Occasionally, even with smaller aneurysms, one of the M2 divisions comes off the base of the aneurysm, which precludes complete clipping and preservation of that division. In these cases, we limit ourselves to a partial clipping of the neck with wrapping of the remaining neck. This can be accomplished satisfactorily in the middle cerebral region because of its peripheral location and the ability of the surgeon to completely dissect the complex. This is the one location in which we have felt relatively secure of being able to completely wrap the aneurysmal complex using muslin gauze held tightly around the aneurysm, with clips applied under pressure to achieve some compression of the aneurysm by the gauze. A division of the MCA can initially appear to arise from the base of the aneurysm but, frequently, with inspection under high magnification, the surgeon realizes that the division is stuck to the aneurysm and an attempt to completely separate it is mandatory. Sharp dissection is imperative in these cases, as learned from a case in which we were using a blunt instrument for dissection and produced a large tear, resulting in necessary sacrifice of that division with fatal infarction. By using only sharp dissection, any small tear that may occur is controllable.

As previously indicated, most of our problems with MCA aneurysms have occurred in cases of very large and giant aneurysms in which the neck usually incorporates one or both M2 divisions and calcium or atheroma may be present at the base. In this location, because of the ease of aneurysm exposure, the surgeon frequently has the luxury of being able to perform an aneurysmorrhaphy on large, partially thrombosed aneurysms. One major problem that we have encountered was related to an excessively vigorous aneurysmorrhaphy, removing atheroma from the base only to end up with a major tear between the origin of the two divisions. That patient suffered infarction despite an emergency bypass. It is best to be cautious in removing atheroma and calcium at the base of the aneurysm and, sometimes, it is preferable to leave some neck that can be closed as a small sac either by suturing or clipping. Excessive dissection of the atheroma at the neck can also lead to occlusion of one of the divisions, probably through a mechanism of dissection when flow is reestablished, which has occurred in at least one case that we recall. Of course, the surgeon always has the choice of not attempting aneurysmorrhaphy and performing a distal bypass with proximal occlusion. This has been a satisfactory solution for many difficult aneurysms. Superficial temporal artery bypass should be used with caution, however, because, early in our experience, such a "low-flow" bypass resulted in postoperative infarction, despite a patent graft. Since then, we, as most other surgeons, have used a "high-flow" bypass with a sa-

phenous vein graft or, if feasible, with a direct anastomosis (for example, a more proximal large anterior temporal branch to one of the distal divisions that had to be sacrificed).

Basilar and Posterior Cerebral Aneurysms

Paradoxically, we will be brief in this section even though, as alluded to before, we have had more problems with basilar aneurysms than with aneurysms in any other location. The reason for brevity is that we have few answers to the many problems we have encountered in this area, usually caused by perforator damage. With basilar tip aneurysms, we had preferentially used the subtemporal approach for many years, following Drake's teaching.[15] However, we always had difficulty with the opposite side of the neck in larger aneurysms. We recall two particularly striking patients who awoke with bilateral third nerve palsies and were, of course, unable to see because of bilateral complete ptosis. In these cases, we presumed that the ipsilateral third nerve palsy occurred from manipulation of the third nerve on that side and the contralateral palsy was central, from occlusion of an early large perforator coming off the distal side of the neck from the contralateral P1. The latter assumption was made because these patients had not only bilateral third nerve palsies, but also pseudobulbar palsy and significant ataxia. To have better visualization of the contralateral side of the neck, we gradually switched to a combined pterional/anterior temporal craniotomy, giving us a more anterolateral approach (*Fig. 16.4*).[31] We have been happier with the greater flexibility of this approach, but still have not found the solution for the problem of the perforators behind the aneurysm. For this reason, we now use endovascular occlusion for most basilar tip aneurysms.

Early in our experience, a particularly impressive fatal complication occurred when we made the mistake of using a high-flow saphenous vein bypass graft to the P1 for a giant basilar tip aneurysms, with the intention to occlude the basilar artery the next day (in the angiographic suite by tightening a tourniquet applied at surgery as recommended by Drake[16]). The addition of distal bypass, however, was not included in Drake's description and the problem we encountered was increased outflow resistance from the aneurysm with a distal high-flow bypass with continued proximal inflow from the basilar artery. The aneurysm ruptured catastrophically that night and, at autopsy, we found that the bypass was open with a large tear in the aneurysm dome. The lesson learned was to proceed with proximal occlusion immediately after performance of a high-flow bypass. Another fatal complication was related to incomplete clipping of the base of a basilar tip aneurysm. In this case, a perforator arose from the base of the aneurysm and a small opening was left at the base of the aneurysm with a fenestrated clip to allow the perforator free passage. Because approximately 95% of the neck was occluded, we hypothesized that the aneurysm would clot completely, however, after waking up perfectly well, the patient suffered a massive rupture a few hours later. Clearly, we changed the flow dynamics inside the aneurysm, which led to a massive rupture in a patient who, when the neck was completely open, suffered only a mild SAH. Ever since, we have worried about statements in the literature recommending partial clipping of the neck to convert a large neck into a small one to allow for endovascular coiling.

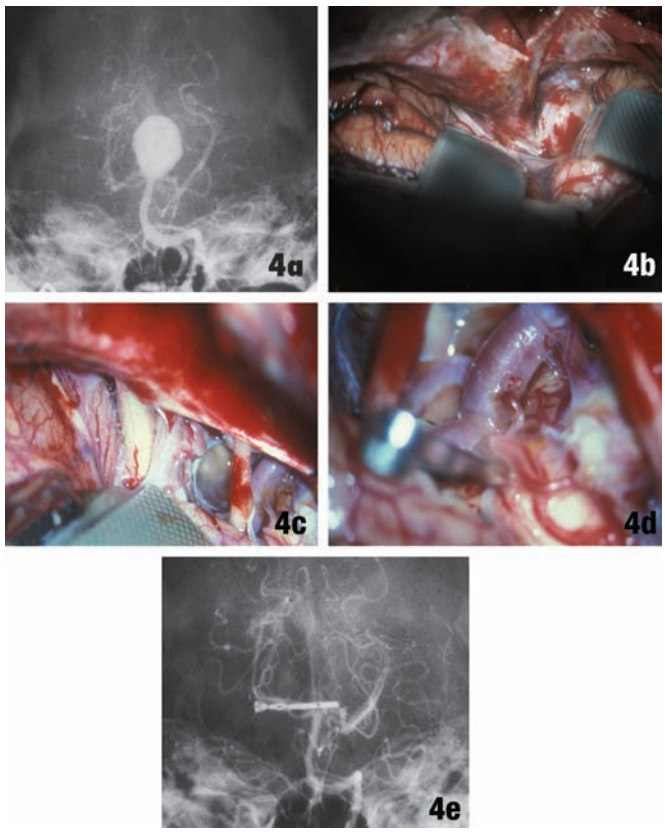

FIGURE 16.4. *A*, AP angiogram showing a large, high basilar tip aneurysm. Surgical treatment was performed through a combined pterional/anterior temporal craniotomy in a delayed fashion 14 days after rupture. *B*, intradural view of the combined pterinal/anterior temporal craniotomy approach before opening the sylvian fissure. Note the absence of subarachnoid blood and relaxed brain 2 weeks after rupture. *C*, magnified surgical view after opening of the sylvian fissure with the tentorium retracted. Note the surgical corridor is centered on the oculomotor nerve. *D*, a fenestrated clip has been placed around the posterior cerebral artery, with complete obliteration of the basilar aneurysm neck. *E*, postoperative angiogram showing satisfactory clipping of the aneurysm.

With basilar trunk aneurysms, we have had damage to cranial nerves VI, VII, and VIII in addition to the usual problems encountered by all surgeons with larger aneurysms of basilar origin. We recall a case of massive rupture from inadequate visualization and inappropriate mobilization of a large aneurysm that was stuck to the clivus. Although initially

we recommended the far lateral suboccipital approach for those aneurysms,[30] we now use this approach only when the aneurysm is very low and prefer the presigmoid transpetrosal approach for higher aneurysms, which gives better exposure with a more direct angle of vision.

We have treated posterior cerebral artery aneurysms with good results, following Drake's recommendations for proximal occlusion or trapping of these aneurysms. However, we remember a case in which there was complete occlusion of a large P2 branch after "reconstructing" a large fusiform aneurysm with fenestrated clips. Flow appeared excellent at the time of surgery, but, on the postoperative angiogram, the artery was completely occluded, probably because of kinking from the clips. The patient was clinically asymptomatic because of the good fortune of having excellent collateral circulation.

As we all know, aneurysms at the origin of the superior cerebellar artery are considerably easier because perforators are usually not a problem in this location. Here, the difficulty is only related to the depth of dissection and, of course, to the proximity of the third nerve, which is almost always related to the dome of these aneurysms. We have no clues to offer for prevention of at least a temporary third nerve palsy when the nerve must be manipulated. The incidence of third nerve palsy in our hands decreased substantially when we changed from a subtemporal to the anterolateral combined approach.

Vertebral and PICA

Vertebral aneurysms at the origin of the PICA are generally more comparable in difficulty to anterior circulation aneurysms than they are to the deeper basilar trunk and basilar bifurcation aneurysms. They are relatively peripheral and, particularly, by using a far lateral suboccipital exposure,[30] essentially no brain retraction is required and the surgeon has immediate proximal access to the vertebral artery as it enters the dura. Therefore, the results with these aneurysms have generally been better than those with other posterior circulation aneurysms, and they tend to "improve" the appearance of the surgical results for posterior circulation aneurysms. However, there are frequent cases of difficulty with dissection of the PICA from the base of the aneurysm. As expected, the PICA is always proximal to the aneurysm and may be quite stuck to it. We know that we have had cases of PICA occlusion and infarction, although fortunately, these have often (but not always) been asymptomatic with sparing of the brainstem, probably because of good collateral circulation. This problem can frequently be avoided by using a fenestrated clip, leaving the origin of the PICA in the fenestration, rather than insisting on complete dissection of the neck, which can lead to rupture at the base or kinking of the origin of the PICA when the clip is applied between the PICA and aneurysm neck. We have also had our share of problems with lower cranial nerves, which are very frequently in the way of the dissection for these aneurysms; however, most patients have recovered well from these temporary difficulties.

Although dissecting aneurysms of the intracranial portion of the vertebral artery are less common, they are being recognized with increased frequency and have led to significant problems. It is clear that these aneurysms have a tremendous tendency to rebleed, because the dissection usually occurs within the muscularis or subadventitially, as opposed to the cervical dissections, which are generally subintimal and do not bleed. These intracranial vertebral dissections are extremely friable and we had a case of massive rupture as we approached one, without having been able to obtain proper distal control. Despite a proximal clip, the aneurysm kept bleeding and we had to proceed with a rapid dissection, which led to major lower cranial nerve damage to obtain distal control of bleeding from retrograde flow from the opposite vertebral artery. With simple proximal occlusion, we recall one case of rebleeding several days after surgery, which required reoperation and trapping, and another case with continuing growth of the aneurysm from retrograde flow from the opposite vertebral artery. That is why now, we would recommend trapping of these aneurysms rather than just proximal occlusion, even if the aneurysmal segment includes the origin of the PICA. This can be handled by either a bypass to the PICA, reimplantation of the origin of the PICA, or a PICA to PICA anastomosis. Hopefully, as better stents become available, these aneurysms will be handled endovascularly in the future, avoiding the significant problems encountered with open surgery. Incidentally, although we had a very satisfactory case of "reconstruction" of one of these dissecting fusiform aneurysms that seemed to involve only one side of the wall by using a series of fenestrated clips to preserve the lumen, we think that this was an exceptional case and we would not recommend attempts at reconstructing these dissected arteries because the dissection frequently involves the entire wall (*Fig. 16.5*).

Distal PICA aneurysms are more frequently straightforward even when they are very large or giant. We recall one case in which the aneurysm was largely thrombosed and was referred to us as a posterior fossa "tumor." Excision and end-to-end anastomosis of the PICA produced excellent results.[57] In terms of complications, we remember a massive rupture of a distal PICA aneurysm that occurred in an attempt to approach an aneurysm at the choroidal point by separating the tonsils of the cerebellum. Frequently, these more distal aneurysms in or near the midline are stuck to the tonsils and since our case of massive rupture, which fortunately was controlled with good result, we prefer to approach these aneurysms by subpial dissection through the tonsil rather than by spreading the tonsils and risking rupture.

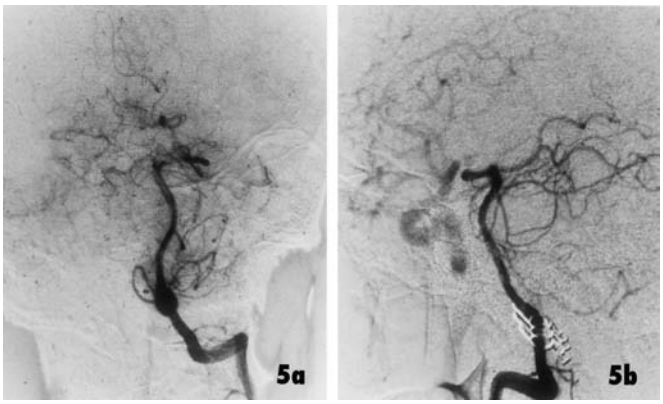

FIGURE 16.5. *A,* AP angiogram showing a ruptured dissecting vertebral aneurysm. *B,* far lateral exposure was performed, allowing for "reconstruction" of the aneurysm with three clips. The follow-up angiogram shows good flow without residual aneurysm.

RESULTS

Although we present our personal (RC Heros) results for completeness, we admit that the very imprecise, retrospective manner in which we have assessed these results makes them worthy of only a short discussion. Within these numbers, both ruptured and unruptured aneurysms are included, despite the expectation of differing results between the treatment of these two different groups of patients. During the early years of the senior author's practice, most ruptured aneurysms were operated on a delayed fashion (usually between days 10 and 15 after SAH), which made those results similar to those expected for unruptured aneurysms. These results concern only surgical morbidity and mortality and do not take into account morbidity from the initial hemorrhage or from vasospasm. Additionally, these are early results (at the time of discharge from the hospital to either home or a rehabilitation facility) and we know that many of these patients improve with time. Furthermore, these early results, in addition to the fact that they were assessed by the surgeon or his team with no independent examination, suffer from a lack of assessment of important psychological and sociological factors, such as long-term psychosocial malfunction, return to work status, etc. This is why we have used the term "good" to denote a patient who has no significant focal deficits and is independent at the time of discharge home rather than using the term "excellent," which would imply a patient who has completely returned to premorbid condition. In rough terms, what can be gleaned from our results is fairly consistent with other large series. Surgical morbidity and mortality is much higher with aneurysms of the basilar artery, excluding the superior cerebellar origin, than those with aneurysms in other locations and, as expected, morbidity and mortality with giant aneurysms is much higher than with smaller aneurysms.

The results of the senior author's aneurysm series have been collected in two parts. The first series spans the years of 1981 to 1998 (*Table 16.1*). The author treated 1272 aneurysms, 136 of which were in the posterior circulation. One hundred ninety-eight of these were giant-sized aneurysms. Most of the patients had ruptured aneurysms, but approximately one-third, during our early experience, were operated on in a delayed fashion (11 to 15 days after rupture). Of the anterior circulation aneurysms treated, 93.9% had a good outcome. Serious morbidity was 4.9% and mortality was 1.2%. Surgical morbidity for posterior circulation aneurysms was higher at 19%, with a mortality of 3.6%.

The second set of results were collected from 1999 through the end of 2004 and are reported separately to better reflect our current experience (*Table 16.2*). These results are obviously much better than the earlier experience, not because of increased surgical finesse, but rather the increased number of unruptured aneurysms treated and the senior author's policies to treat patients with poor-grade and difficult aneurysms, such as those at the basilar top, endovascularly. The senior author treated 277 patients with a total of 293 aneurysms clipped. Female patients comprised 79% of those patients treated. The mean age at onset was 51 years, with a range from 19 to 84 years old. Anterior circulation aneurysms (80.5%) comprised the majority of aneurysms treated (MCA, 83; AComA, 67; carotid, 39; ophthalmic, 22; AChA, 13; pericallosal, 10; and superior hypophyseal, 2). In addition, 41 PComA, 8 PICA, 3 basilar, 2 vertebral, and 2 superior cerebellar artery aneurysms were treated. One hundred thirty-two patients presented with SAH (48%), 24 with cranial nerve palsies (8.6%) and 123 were essentially asymptomatic (44%). Forty-five patients (16%) were found to have multiple aneurysms. Small aneurysms were most prevalent in this series, with 58.7% of aneurysms less than 7 mm, 23.1%, 7 to 10 mm; 15.6%, 11 to 24 mm; and only 6 giant aneurysms (25 mm). The vast majority of patients (218 patients) underwent complete clipping of their aneurysms. Eleven patients had wrapping of their aneurysms with muslin gauze because of calcification of the neck or other difficult morphology, and 24 patients underwent clipping with wrapping of a small residual neck. Carotid ligation or trapping with or without bypass was performed in 14 patients with giant or large cavernous or paraclinoid aneurysms. Surgical treatment was aborted in 13 cases, 9 of which were sent for endovascular treatment, because of heavy calcification at the neck of the aneurysms and an increased risk versus benefit ratio. Surgical complications were minimal (8.7%) and included seven intraoperative aneurysm ruptures, most of which could be controlled without sequelae, nine cranial nerve palsies, two carotid pseudoaneurysms, four wound infections and three cerebrospinal fluid leaks. Vasospasm occurred in 42 patients, with infarcts in 12. Rebleeding occurred in five patients after arrival to our institution. Delayed clipping occurred in 26 patients, usually

as a result of presentation after 3 days or significant medical comorbidities, such as myocardial infarction. Outcome in the vast majority of patients was good, with discharge to home in 86% and discharge to rehabilitation in 9.8%. Six patients were discharged to a nursing home and there were five deaths, although none directly related to surgical intervention. As discussed earlier, the main difference between the series of patients from the last 5 years and the earlier series is the increased number of unruptured aneurysms and the decreased percentage of basilar and giant aneurysms, reflecting an increase in detection and referral of patients with incidental aneurysms and the senior author's bias toward endovascular therapy for patients with poor-grade SAH and those with difficult aneurysms.

These improved results are a good reflection of the increased satisfaction that the availability of excellent endovascular surgery has meant to a busy aneurysm practice, given that the early results of endovascular treatment of patients with poor-grade and with surgically difficult aneurysms, although not discussed here, seem to be considerably better than that of microsurgical treatment. Of course, the caveat is whether these good early endovascular results hold at long term follow-up. For now, we think they will, at least in terms of preventing rebleeding, and that with improved technology we will find better and safer ways to "re-treat" endovascularly those aneurysms that recanalize during follow-up.

Overall, open microsurgical treatment of aneurysms in the hands of the senior author has carried a 5.8% rate of morbidity and 1.6% rate of mortality, as indicated in *Table 16.3*.

TECHNOLOGICAL ADVANCES

As our technology has improved, so has our ability to treat certain aneurysms with better exposure and decreased risk. Most aneurysms do not require cranial base approaches, but, under certain circumstances, they can certainly prove helpful. The exposure of paraclinoid aneurysms can be improved with an anterior clinoidectomy and complete unroofing of the optic canal and exposure of the clinoidal segment of the ICA. Orbital osteotomy can improve visualization of AComA aneurysms, but the senior author has only rarely found this necessary. Good exposure of complex and giant basilar apex aneurysms can be obtained through an orbitozygomatic craniotomy or subtemporal craniotomy plus anterior petrosectomy in cases of low basilar bifurcation. We have found that both midbasilar and lower basilar artery aneurysms can be approached through a presigmoid-transpetrosal approach, although we prefer to approach lower basilar aneurysms that are located at the lower fourth of the clivus and PICA aneurysms with a suboccipital far-lateral approach.[30] Large and giant aneurysms of the ICA and most basilar top aneurysms are approached through the simpler combined pterional/anterior temporal approach described by the senior author.[31] These approaches are, of course, used in selected cases only. When cranial base approaches are required, the increased risk and operative time must always be weighed against the risks and efficacy of endovascular therapy.

Another recent innovation has been endoscopic-assisted aneurysm surgery. The term "endoscope-assisted microsurgery" was coined by VanLindert and Perneczsky in 1998,[100] although the use of the endoscope to aid in neurosurgical procedures dates back to the early 1900s. Major improvements in the optics of present-day endoscopes and in image-guidance techniques have encouraged many surgeons to examine the usefulness of endoscopy during aneurysm surgery.

Kalavakonda and Sekhar studied 55 patients with 79 aneurysms treated with endoscope-assisted aneurysm clipping.[46] The endoscope was used in addition to a standard microsurgical approach to ascertain anatomic detail before and after clipping. It was particularly helpful for areas that are excluded from direct line of sight, including the back wall of the aneurysm, and was used to check the clip position, completeness of aneurysm clipping, inadvertent compromise of the parent vessel or major branch by the clip, sparing of perforators, and pressure on surrounding vital structures. The subjective feeling of the authors was that more complete regional anatomic detail was obtained in 26 of 79 aneurysms, and unique anatomic information that could not be obtained by the microscope was provided by the endoscope in 15 of 79 cases. The endoscope identified two cases in which the clip was across the PComA artery, four cases in which there was a residual aneurysm neck, and one case in which the clip placed pressure on the optic nerve, resulting in repositioning of the clip in these cases. Rupture of an aneurysm was directly related to the use of the endoscope in one case without sequelae. Similarly, Taniguchi et al. studied 48 patients with 54 aneurysms treated with rigid endoscopic assistance.[96] More complete regional anatomic detail was obtained in 44 of 54 aneurysms and unique anatomic information was provided by the endoscope in 9 of 54 cases. The endoscope identified two cases of incomplete neck obliteration, two cases of involvement of perforating vessels by the clip, and one case of significant cranial nerve compression by the clip for which clip reapplication was performed. There were no endoscope-related complications. This technique has intuitive appeal, but, thus far, we have not incorporated it into our practice. We suspect that there is a steep learning curve that comes with experience in endoscopic use and should, therefore, be used only by those experienced in microsurgical aneurysm clipping as well as in endoscopic use.

Minimally invasive (keyhole) approaches are another technological innovation. This movement originated in 1978 with Brock and Dietz's description of a small frontolateral approach to anterior circulation aneurysms.[6] This concept has

recently grown in popularity, fueled by improved image-guidance and superior endoscopic optics and by the efforts of several surgeons, including Perneczky[100] and Fukushima.[22]

The supraorbital keyhole approach for supratentorial aneurysms was described by Van Lindert et al.[100] This approach was used on 139 patients with 197 aneurysms and included an eyebrow incision with limited temporalis muscle mobilization. One hundred eighty-five aneurysms were clipped (12 wrapped) with endoscopic assistance in 25 cases. There were no approach specific complications and cosmetic results were excellent. There were four patients (3%) with premature rupture—all during dissection around the dome of the aneurysm or during clip application. The authors think that this is lower than would be expected in conventional craniotomies for aneurysms.

Steiger et al. described the transorbital keyhole approach to AComA aneurysms in 33 patients.[91] All 33 aneurysms were successfully clipped with transient diplopia in one patient and a frontalis palsy in two patients. Long-term cosmetic outcome was subjectively better than with a standard pterional craniotomy.

Although early studies have described results that are comparable to those reported in the literature with conventional craniotomies, and cosmetic results may be improved, we have not integrated these approaches into our practice. The advantages of a keyhole approach are decreased brain exposure, minimal manipulation of temporalis muscle, and reduced operative time. The main disadvantages of the keyhole approach are the narrow angle of vision afforded during aneurysm exposure and clipping, as well as poorer illumination of the surgical field and decreased access to proximal control if early premature rupture occurs. The surgeon is committed to viewing the aneurysmal anatomy from one direction without having the freedom to "look behind" the aneurysm by changing the direction of the microscope along a wide margin at the base of a wide exposure. Although some of these disadvantages can be reduced by the adjunctive use of endoscopy, the reported advantages of these "minimally invasive" approaches, in our opinion, do not outweigh the apparent disadvantages.

Of all technological advances, endovascular treatment has improved most rapidly since its introduction in 1991. Since the approval of Gugleilmi detachable coils, countless new coils have come onto the market with technical advances, such as increased resistance to stretch and coil compaction, improved 3D technology, and bioreactivity. Use of balloon assistance and intracranial stents now allows for complete coiling of wide neck aneurysms that were previously impossible to treat endovascularly. Three-dimensional angiography is becoming the gold standard for decision making regarding optimal aneurysm treatment.

The latest breakthrough in endovascular treatment has just become available to us in the United States. In selected patients with aneurysms that are unsuitable for coil treatment or in whom previous treatment has failed to occlude the aneurysm, Onyx treatment offers an endovascular alternative. Onyx is a liquid ethylene vinyl alcohol polymer suspended in dimethyl sulfoxide (Micro Therapeutics, Inc., Irvine, CA) that adheres to itself and solidifies slowly from the outside in, allowing conformation to the aneurysm sac with subsequent occlusion. Cerebral Aneurysm Multicenter European Onyx (CAMEO), a prospective observational study conducted in 20 European centers enrolled 119 patients with 123 aneurysms for Onyx treatment.[65] Seventy-nine aneurysms were large or giant. Twelve-month follow-up angiography findings were available for 71 aneurysms. The angiographic follow-up showed complete occlusion in 56 (79%) aneurysms, subtotal occlusion in 9 (13%), and incomplete occlusion in 6 (8%). Of seven deaths, two were procedure related. Delayed occlusion of the parent vessel occurred in nine patients, with permanent neurological deficit in two. Aneurysm occlusion rates were equal to or superior to the current reported rates with coil occlusion. Morbidity was comparable to that of published prospective data on endovascular results for this subgroup of patients, but this requires further study and cannot be extrapolated to small- or moderate-sized aneurysms. The most important limitation of this technique is the relatively poor control of migration of the liquid embolic agent into the parent artery. The use of a microballoon across the neck of the aneurysm, a microstent deployed across the neck of the aneurysm, or the deposit of GDCs into the aneurysm allowed faster and more complete filling of the aneurysm with Onyx.[68]

CONCLUSIONS

Our understanding of the etiology, anatomy, natural history, and treatment of aneurysms continues to evolve. New technology and innovation have given us more tools in our armamentarium but have certainly complicated our choices of management. It is imperative that, as we continue to develop our knowledge of cerebral aneurysms and their management, we remain focused on the optimal treatment for the individual patient, doing not only what *can* be done, but what *should* be done. This last decade has seen extraordinary changes in the treatment of ruptured and unruptured aneurysms alike, and we fully expect that theses paradigms will continue to shift as new randomized studies and long-term follow-up data on endovascular treatment becomes available. The field of neurosurgery must not only stay abreast of these changes, but actively direct these changes by expanding our field into the interventional arena. We look forward to the challenge.

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