GENERAL SURGICAL PRINCIPLES FOR INTRACRANIAL ANEURYSMS

Preoperative Considerations. When surgical treatment for an intracranial aneurysm is considered, factors such as the condition of the patient, the anatomy of the aneurysm, and the experience of the neurosurgeon must be weighed against the natural history of the aneurysm. With rare exceptions, direct clipping is the gold standard of intracranial aneurysm treatment. The primary surgical goal is to exclude the aneurysm from the circulation completely while preserving normal cerebral circulation and brain tissue.

Timing. Operative timing for ruptured intracranial aneurysms has been debated for nearly 30 years. Diversity in patient populations, variations in specific aneurysm anatomy, and the absence of definitive outcome data account for the continued controversy. In their landmark report, Hunt and Hess (1968) categorized patients based on neurologic status and concluded that good grade patients should be operated on early to minimize rebleeding While delayed surgery should be performed on poorer grade patients to allow for clinical improvement. The International Cooperative Study on the Timing of Aneurysm Surgery analyzed over 3500 patients and confirmed the reduced rate of rebleeding with early surgery (Kassell N.F. et aL, 1990

Disadvantages of early surgery include the need for greater brain retraction and greater difficulty in dissection due to edematous brain and probably higher intraoperative rupture risk. Currently, guidelines published by the American Heart Association recommend early surgery for good grade patients with uncomplicated aneurysms and individual assessment for all other clinical situations (Mayberg M.R. et al., 1994). These guidelines also recommend early specialized intensive care for all patients with subarachnoid hemorrhage. There is no steadfast rule for optimal aneurysm surgery timing, but carefully appraising the aneurysm anatomy, surgical environment, and patient neurologic and medical status will provide the best basis for a decision.

Anesthesia. Although neuroanesthetic concerns have been presented in a previous chapter, several specific considerations are important for successful intracranial aneurysm surgery. Close communication between the neurosurgeon and the neuroanesthesiologist must be maintained to minimize patient risk and to facilitate successful aneurysm obliteration. Prior to the start of the case the neurosurgeon should specify clearly to the neuroanesthesiologist the requirements for an optimal outcome, and communication should continue throughout the operation. Important issues include constant control of blood pressure, optimal cardiopulmonary support, precise electrolyte and fluid balance, neurophysiologic monitoring, pharmacologic cerebral protection, avoidance of hyperthermia, adequate brain relaxation, and accurate intracranial pressure management.

Surgical Instrumentation. For successful operative aneurysm treatment, proper instruments and equipment are essential. Skilled use of the operating microscope is required for modern aneurysm surgery, and the surgical and nursing staff's focus should be the microscope and microsurgical technique. To accomplish this, a particular spatial relationship among the surgeon, assistant, anesthesiologist, scrub nurse, and microscope is needed. For a right-handed surgeon, the surgical assistant and the observer's microscope attachment are positioned to the surgeon's right, the anesthesiologist is at the patient's left shoulder, and the microscope stand is between the anesthesiologist and the surgeon. The scrub nurse should be near the patient's right shoulder, close to the surgeon's dominant hand, to facilitate immediate instrument passage. This arrangement is reversed for the left-handed surgeon. Although numerous microsurgical tools are in the market, only a few selected microinstruments are necessary for intracranial aneurysm surgery. The surgeon should be intimately familiar with them and capable of multiple technical maneuvers with a single instrument. Essentials include microtip bipolar forceps, microscissors, micro dissectors, arachnoid knife, retractors with varying tip shapes and sizes, suction tips with slits on the handle for suction strength regulation, clip applicators, and aneurysm clips. Instruments with varying angles and bayonet shapes to improve the surgeon's visualization in what is often a narrow and deep opening are helpful.

INTRAOPERATIVE CONSIDERATIONS

Intracerebral Hematoma. If an intracerebral hematoma is present, the decision regarding operability based on the neurologic grade becomes less useful. Heiskanen and colleagues (1988) reported in a prospective randomized trial that emergency craniotomy for hematoma evacuation and aneurysm clipping, when compared with delayed or conservative treatment, decreased the mortality rate from 80 to 27%. Wheelock and colleagues (Wheelock B. et al., 1983) reported similar benefits of early operation in patients with aneurysmal intracerebral hematomas. They also found that when the hematoma was emergency evacuated without clipping the aneurysm, patients had a much higher mortality rate (75%). Emergency craniotomy may be indicated as long as some brainstem function remains since virtually all patients rendered comatose by a hemorrhage who harbor a massive clot will die if not decompressed.

Surgical Dissection. The neurosurgeon must be dedicated to achieving elegant dissection techniques. Intraoperative aneurysm rupture and neurovascular injuries due to poor technique can immediately prejudice the patient's chances for a successful outcome. Blunt dissection is ill advised and can lead to large tears near the aneurysm neck that are often difficult to repair. By contrast, bleeding caused by sharp dissection usually results in punctate and more controllable injury (Kopitnik T.A, Samson D.S., 1993). Microscissors or microarach-noid blades should be used in preference to blunt tearing techniques. Normal vessel segment dissection proximal and distal to the aneurysm should be performed early to gain vascular access for control of bleeding if needed. Proximal control should be followed by sharp clot dissection around the aneurysm and neck. Meticulous neck dissection will allow optimal clip placement and reduce clip-induced aneurysm rupture or vessel compromise.

Intraoperative Rupture. Intraoperative aneurysm rupture occurs in 18% to 61% of cases and carries a high morbidity and mortality. Recent reviews in Dallas and Los Angeles found that intraoperative rupture occurred in 19 and 15% of cases, respectively. This complication may occur before dissection, during dissection, and during clip application. Giannotta et al. (1991) reported an 80% mortality rate in five patients whose aneurysm ruptured prior to dissection. During the predissection stage, potential causes of rupture include vibration from power instruments performing the craniotomy, changes in transmural pressure with dural opening, brain retraction, and blood pressure lability. In light of the frequent use of power instruments and the rarity of aneurysmal rupture during the craniotomy, it seems that vibration is an unlikely cause. However, patients who have not reached the anesthetic depth required for complete analgesia during this part of the procedure surely risk increased blood pressure and increased transmural aneurysm pressure. Local scalp anesthetic coupled with adequate general anesthetic depth can potentially prevent rupture at this stage. Additionally, deferring intracranial pressure reduction by hypocarbia, cerebrospinal fluid (CSF) drainage, or diuresis until after dural opening may also prevent sudden changes in transmural aneurysm pressure. Optimal bony removal for exposure and Maximal brain relaxation after dural opening will help reduce required retraction and lessen the intraoperative rupture risk. Procedures for management of intraoperative rupture prior to the stage of dissection of the aneurysm include aborting the procedure prior to dural opening to allow for tamponade or rapid dissection with proximal control of the aneurysm if the dura has already been opened.

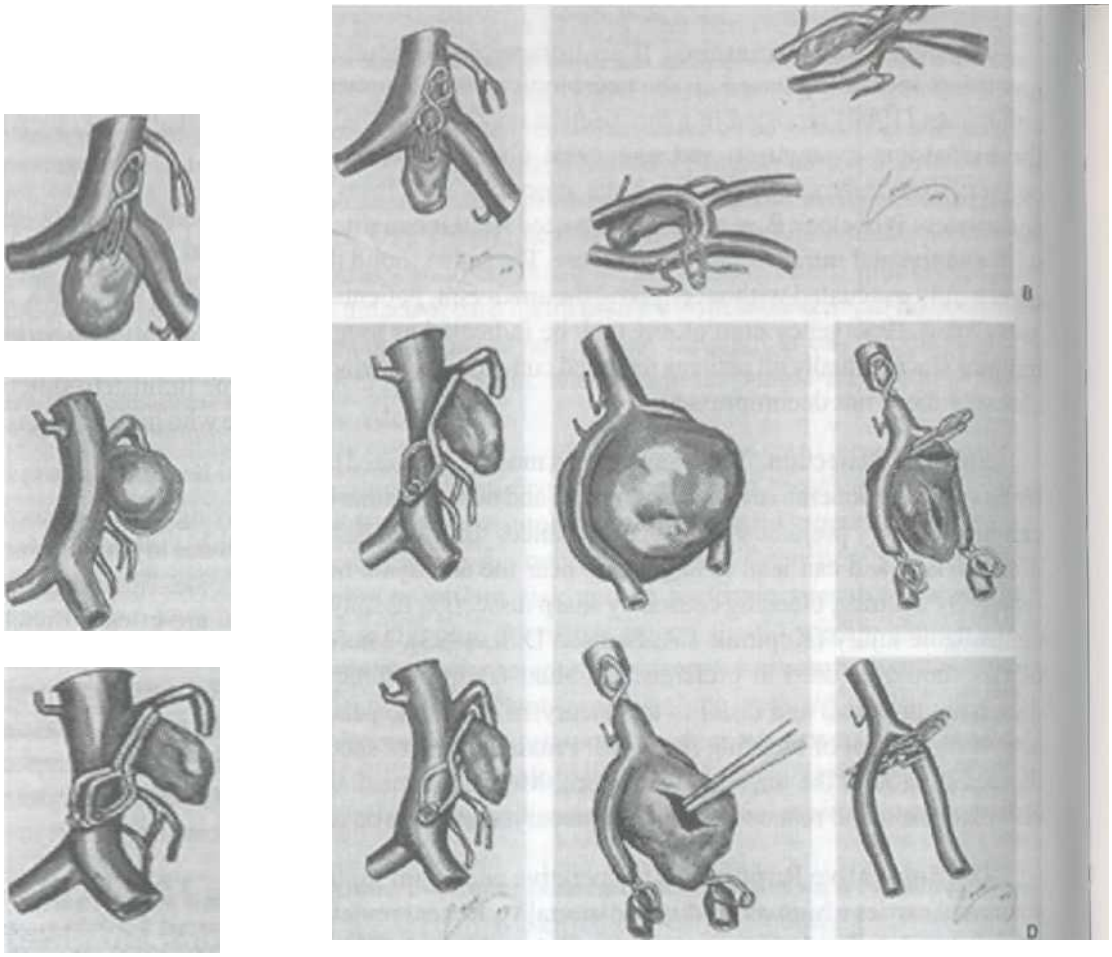


Fig. 1. Clip application techniques. (A) Conventional clip placement followed by a fenestrated tandem clip prevents continued filling due to lumen irregularities. (B) Parallel clip placement avoids parent artery stenosis produced by a perpendicular clip. (C) Sequential clip placement. The initial clip produces parent artery stenosis, so a second distal clip is placed and the first removed. (D) Aneurysm trapping and decompression prior to clip placement. This broad, atheromatous neck necessitates incision with successive clip placement to span the entire neck.

Intraoperative rupture occurs most frequently during microdissection in the subarachnoid space prior to clipping the aneurysm. As mentioned above, sharp dissection is the preferred technique in our opinion since blunt dissection can lead to tears that are more difficult to manage. When a blunt tear has occurred, it is best not to clip the aneurysm definitively until the anatomy is clearly defined. This can be accomplished by placing two suction devices into the field to clear the blood, enabling the surgeon to identify the bleeding site. If the anatomy is still not clearly defined, as is often the case, temporary clips should be placed on afferent and efferent vessels as far from the aneurysm as possible and sharp dissection continued until exposure is satisfactory. Microsutures may be necessary to repair tears that extend beyond the aneurysm neck into the parent vessel. Bleeding caused by sharp dissection often responds to gentle tamponade with small pieces of cotton or judicious bipolar cautery. In both circumstances, definitive clip placement should only be done when the neurosurgeon is satisfied with the anatomic exposure of the aneurysm. During clip application, ruptures are usually due to inadequate exposure or to poor clip application technique. Attempts at clip placement prior to complete exposure may lead to tears similar to those resulting from blunt dissection. These tears are difficult to manage because the tears tend to be large, proximally lo cated on the aneurysm, and associated with excessive bleeding. Bleeding from this cause often occurs prior to complete closure of the clip as the blade has entered the sac. Prompt removal of the clip at the first hint of bleeding will minimize the tear. Management in this situation is the same as for tears due to blunt dissection. Bleeding due to poor clip placement technique is usually of lesser magnitude. These injuries are often due to incomplete neck obliteration by a short clip, insufficient closing pressure, or differential neck thickness. These problems

can be remedied with various techniques and clip types such as longer clips, stronger clips, booster clips, or tandem clips **(Fig. 1**).

Temporary arterial occlusion and cerebral protective agents including anesthetics that produce an electroen-cephalographic (EEG) burst-suppression pattern are now employed in the event of an intraoperative rupture. For cerebral protection, barbiturates have been used to reduce cerebral metabolism and electrical activity to a pattern of burst suppression. Disadvantages of barbiturates include hypotension and a long half-life of action. **Etomidate** is a useful alternative cerebral protective agent that reduced cerebral metabolism with minimal toxicity and rapid reversibility (Batjer H.H. et al., 1988). Etomidate produces EEG changes similar to those produced by barbiturates yet does not induce significant cardiovascular depression. In addition, patients regain consciousness within a few minutes after etomidate withdrawal, which allows for earlier extubation and neurologic assessment. The use of temporary arterial occlusion and etomidate has generally replaced the use of induced hypotension to prevent aneurysmal rupture during dissection and to control bleeding from a rupture.

Temporary Arterial Occlusion. Under certain circumstances, temporary arterial occlusion can provide the cerebrovascular surgeon with a variety of benefits. These benefits range from minimizing the risk of intraoperative hemorrhage risk during dissection and facilitating optimal aneurysm clipping to facilitating the repair of complex giant aneurysm. Temporary arterial occlusion permits definitive aneurysm dissection and permanent clip placement with minimal risk of cerebral ischemia. Optimal temporary occlusion requires that consideration be given to the patient's blood volume and blood pressure, the use of agents for cerebral protection, the safety and effectiveness of the temporary clips, the location of the temporary clips, and the duration of occlusion. In general, patients undergoing temporary arterial occlusion can best tolerate the occlusion when their intravascular volume and blood pressure are in the «normal» range, that is 120 to 130 mm Hg or slightly higher for hypertensive patients. To minimize the intimal damage caused by a clip placed on a vessel, clips designed for temporary arterial occlusion should be used. These clips have a closing pressure that is approximately one-half that of permanent clips. The proper location for placement of temporary clips is specific for each case, but the principle to keep in mind is minimal cerebral ischemia with maximal aneurysm exposure. If intra-cranial temporary arterial occlusion is not possible, extracranial occlusion via the cervical carotid artery should be considered. Alternatively, endovascular control with a nondetachable balloon can be performed.

In general, patients with optimal intravascular volume, blood pressure, blood rheology, and cerebral protective drugs that have produced EEG burst suppression can tolerate temporary occlusion of any major intracranial vessel. A recent report found that patients with Hunt-Hess grades I or II typically tolerate temporary occlusion for 19 minutes, whereas higher grade patients tolerate 15 minutes with a 95% confidence level (Batjer H.H., Samson D.S., 1994). This report also found that patients with occlusion greater than 31 minutes all suffered cerebral infarctions. Other findings included a lower tolerance of temporary occlusion in older patients. Induced mild hypothermia (32°C) may represent a safe and effective adjunctive maneuver to extend ischemic tolerance. With appropriate precautions, temporary arterial occlusion is a well-tolerated maneuver that enhances the surgeon's ability to deal with even the most difficult intracranial aneurysms.

Clip Application, Aneurysm clip application may appear to be a mechanically straightforward task, but many intricacies are associated with it. In general, aneurysm neck obliteration is most precise if the clip is placed parallel to the long axis of the parent vessel. This principle obviously does not apply to all situations but should serve as a guiding principle to minimize parent vessel stenosis and aneurysm shearing forces **(Fig. 1**). Other considerations important in clip placement include preserving perforating vessels and adjacent neural structures.

When an aneurysm remains patent after clipping, contributing factors may include the following:

1.The aneurysm clip does not span the entire length of the neck.

2.Intrinsic clip weakness at the most proximal aspect of the blade prohibits proper closure.

3.An atheroma exists in the aneurysm neck.

These problems can be remedied by repositioning the clip or adding a second tandem clip **(Fig.1**). For broad-based, expansive aneurysms such as those commonly encountered at the middle cerebral artery (MCA) bifurcation, temporary clipping and subsequent deflation greatly help in placing the permanent clip. If clip length is insufficient for a broad-based aneurysm, smaller clips can be placed sequentially followed by incision along the outer edge of the previous clip to provide access for each successive clip

(Fig. 1). When a clip will not close the neck of an atheromatous aneurysm, two methods can be used, both of which require temporary arterial occlusion. The first, described by Drake, is forming a new neck by crushing the atheromatous wall with a hemostat and placing a clip over the crushed path. The second technique involves opening the aneurysm of the wall followed by endarterectomy and thrombectomy with subsequent clip placement. In giant aneurysms with broad necks, the closing forces of clips are often insufficient to maintain neck closure after temporary clip removal. Two methods have been described to overcome this problem. The first is stacked or tandem clip placement one on top of another until the aneurysm does not refill (Fig. 24). Each clip should span the entire aneurysm neck. As many as four tandem clips have been placed on one aneurysm. A second method, also described by Drake, is placing a «piggy back» clip to reinforce the original clip. More recently, Sundt (1984) developed secondary clips for this specific purpose and called them «booster clips.»

Wrapping, Trapping, and Ligation. When aneurysm obliteration by direct clipping is not possible, other options include wrapping or coating, trapping, proximal ligation, and endovascular occlusion. The recent Cooperative Aneurysm Randomized Treatment Study reported that carotid ligation did not lead to a significant improvement in mortality or rebleeding in the acute period but did reduce rebleeding rates at 3 years and mortality at 5 years. Proximal ligation has been reported to be very effective for giant cavernous carotid aneurysms, and Drake and others have reported success with this technique in basilar artery aneurysms (Steinberg G.K., Drake C.G., Peerless S.J., 1993). Complications such as thromboembolism and distal ischemia must be taken into account when contemplating this therapy. Aneurysm trapping involves interrupting the proximal and distal arterial flow to the aneurysm. The major complication in aneurysm trapping is ischemic injury in the territory of the parent vessel or associated perforators. Trial occlusion with balloons and revascularization procedures have made parent vessel sacrifice much safer in recent years.

SURGICAL MANAGEMENT OF SPECIFIC ANEURYSMS PTERIONAL APPROACH

Most anterior circulation and many distal basilar artery aneurysms are best approached by this route or one of its modifications. The pterional approach allows wide basal exposure with minimal brain retraction by taking advantage of naturally occurring planes and spaces. Initial discomfort with an oblique and inverted orientation is quickly overcome by using this approach for a wide variety of subfrontal and parasellar lesions.

Patient Positioning. Careful attention to patient positioning helps utilize gravity to minimize retraction and to facilitate venous drainage. For the pterional approach, the patient is placed supine with the head of the bed elevated at 10 to 15 degrees and the ipsilateral shoulder slightly ele vated. As with all aneurysm operations, absolute head stability is essential. A three-point skull fixation device such as the Mayfield-Kees apparatus should be used. The three-point fixation device can best be placed with the two-pin arm anterior and contralateral to the planned exposure and the one-pin arm posterior and ipsilateral just above the mastoid. A right-sided approach is preferred for most midline lesions because operating on the patient's nondominant (usually right) hemisphere may be associated with lower morbidity. After the Mayfield-Kees three-point skull fixation device is placed, the head is positioned by three maneuvers—rotation, flexion, and tilt. After these maneuvers, and after the patient is in a satisfactory position, the head holder is secured to the operating table.

Rotation. The head is rotated around the long axis of the body toward the contralateral side. The degree of rotation is determined by the anatomic target site. In general, the further anterior and distal the lesion, the greater the degree of rotation. Thus, patients with internal carotid or distal basilar artery aneurysms are rotated about 20 degrees from vertical, whereas patients with MCA aneurysms are rotated about 45 degrees. A greater degree of rotation is employed for an anterior communicating artery (ACoA) aneurysm to simplify medial gyrus rectus and interhemispheric fissure exposure. Gravity pulls the temporal lobe posteriorly and medially with increasing rotation, thus encroaching upon exposure of the internal carotid artery.

SUBARACHNOID HEMORRHAGE

Subarachnoid hemorrhage (SAH) is an event with potentially devastating consequences. It accounts for as many as 10% of all strokes and 25% of all cerebrovascular deaths. Each year, approximately 30,000 Americans will sustain nontraumatic SAH, 25% of whom will die and 50% of whom will experience significant morbidity (Ingall T.J., Wiebers D.O., 1993). Recent studies have shown that approximately half of those who die of SAH do so before coming to medical attention (Broderick J.R, Brott T.G., Duldner J.E., Tomsick T, Leach A., 1994). Even after patients are hospitalized, morbidity and mortality rates remain high due to re-bleeding, vasospasm, and other sequelae. Early surgery and improved critical care interventions, both diagnostic and therapeutic, have favorably altered the prognosis in the last 10-20 years for patients surviving the initial event (Le Roux P.D., Elliot J.P., Downey L. et al., 1995).

Worldwide the incidence of SAH varies dramatically, with the highest rates being reported in Japan (25 cases per 100,000 persons). In the United States, this rate has remained largely unchanged over time at 6 to 16 per 100,000. A more precise estimate of incidence is difficult to ascertain because some patients who sustain SAH may not seek medical attention or may be misdiagnosed (Mayer P. et al., 1996).

Risk of SAH increases with age (mean age 50 years) (Mayberg MR., Batjer H.H-, Dacey R. et al., 1994). Although women have a slightly higher cumulative incidence of SAH overall, the incidence is higher in males in the first decade of life (ratio of males to females 4:1). The incidence is equal in the fifth decade, and by the eighth decade the incidence is 10 times greater in women than in men (Weir B., 1994). African-Americans appear to have higher rates of SAH than do white Americans.

Smoking has been shown to increase the risk for SAH (Longstreth W. T., Nelson L. M., Koepsell T.D,, 1992). It is not clear whether tobacco use is a short or long-term risk factor, although one study has indicated that among smokers risk of SAH was greatest at 3 hours after smoking. Former smokers appear to be at less risk than light or moderate smokers and the longer the time after cessation of smoking, the less the risk of SAH.

Moderate to excessive alcohol ingestion has been shown to be a risk factor for hemorrhagic stroke. The data for risk of intracerebral hemorrhage appear to be more compelling than those for risk of SAH. Alcohol intake appears to be predominantly a short-term risk factor for hemorrhagic stroke in that reduced intake is associated with reduced incidence.

Hypertension is a leading risk factor for intracerebral hemorrhage, although not necessarily for SAH. Although blood pressure control has improved substantially in the United States in the last decade, there is no evidence that the overall incidence of SAH has changed measureably in this time (Mayberg M.R., Batjer H.H., Dacey R. et al., 1994).

Oral contraceptive use has been linked to SAH in numerous studies (Thorogood M., Mann J., Murphy M., Vesscy M., 1992) showing that women using such contraceptives may run a risk of SAH two to six times that of the general population. Unfortunately, most of the studies did not control for other possible risk factors, such as smoking, hypertension, and alcohol intake. Because of these confounding variables, the association between oral contraceptive use and SAH has been called into question (Mayberg M.R., Batjer H.H., Dacey R.etal., 1994).

Cocaine and other illicit drugs have been associated with increased risk for stroke, both ischemic and hemorrhagic (Brown E., Prager J., Lee H.Y., Ramsey R.G., 1992). In cases of SAH associated with cocaine abuse, approximately 50% of patients are shown to have underlying structural abnormalities [e.g., aneu-rysm, arteriovenous malformation (AVM)] on radiographic evaluation.

Some investigators have asserted that **there is a definite and predictable seasonal fluctuation in incidence of SAH** (Chyatte D., Chen T.L., Bronstein K., Brass L.M., 1994). Chyatte et al. (1994) reported **a peak incidence of SAH in men in late Fall and a corresponding peak for women in late Spring.** Although most investigators have attributed this association to **direct effects of the climate (e.g., barometric pressure), stressful, season-related activities have also been implicated** (Mayer P.L., Awad I., 1994). Still other investigators have failed to demonstrate any clear seasonal trend in SAH incidence.

CLINICAL PRESENTATION

The onset of SAH is usually heralded by a sudden severe headache. This headache]s often unremitting and typically is associated with nausea, meningismus, and photophobia. hi more severe cases, the person's level of consciousness is impaired. This impairment can range from mild somnolence to deep

coma. Sudden death is uncommon but not rare (Broderick J.P., Brott T.G., Duldner J.E., Tomsick T, Leach A., 1994). Often, SAH occurs during an activity involving heavy straining or exertion, such as exercise, intercourse, or a bowel movement.

Patients can also present with focal neurologic deficits, most commonly hemiparesis, cranial nerve palsy, or visual field defect. A focal neurologic deficit can indicate the location of pathology. This clinical suspicion is useful information during subsequent diagnostic evaluation.

In such cases cerebral angiography is performed, with particular attention paid to these locations.

Seizures have also been reported in every large series dealing with SAH. Although percentages vary, approximately 15% of patients with aneurysmal SAH exhibit seizure activity.

The clinical grading scale proposed by Hunt and Hess has been shown to be a valid method of assessing patients who sustain SAH **(Table 5).** The patient's Hunt and Hess grade on admission has been shown to correlate significantly with both short- and long-term clinical outcome.

Many patients have premonitory symptoms before SAH becomes overt, usually atypical headaches or neck stiffness that have been attributed to small hemorrhages. They are termed warning headaches or sentinel leaks. As many as 70% of patients experience these symptoms days or weeks before a more significant bleed occurs (Mayer P.L., Awad I., 1994). About half of these patients seek medical attention for these premonitory symptoms, and many are misdiagnosed. A higher clinical suspicion of SAH among healthcare providers would probably lead to earlier and more accurate diagnosis in this group of patients. Appropriate treatment might then be given in a more timely fashion, and outcome might be improved.

TABLE 5. Commonly used clinical grading scales for subarachnoid hemorrhage

Grade	**Hunt** - Hess	Cooperative Aneurysm Study	World Federation of Neurological Surgeons
I	Mild headache	Asymptomatic	Asymptomatic (GCS - 15)
II	Severe headache, meningismus, cranial nerve deficit	Headache, aware/alert, mildly ill	Headache/meningismus, no focal signs; GCS-15
III	Altered LOC± focal sings	Moderately ill, or alert with focal signs	Headache /meningismus, no focal signs; GCS 13-14
IV	Stupor + hemiparesis	Severely ill (stupor) or drowsy with focal signs	Headache /meningismus and GCS 9 - 12 or focal signs and GCS 13 -14
V		Not available	Headache /meningismus + focal signs (GCS □ 9)

LOC - level of consciousness; GCS - Glasgow Coma Scale

DIAGNOSIS

Early treatment for aneurysmal SAH in good-grade **(I-il)** patients is so successful and the consequences of a misdiagnosis so potentially devastating that clinicians who evaluate such patients should promptly pursue the appropriate workup. Impaired level of consciousness and/or focal neurologic deficit should not be considered «required signs» for pursuing a diagnosis of SAH.

With or without accompanying neurological signs, a sudden unusually severe headache should signal the need for computed tomography (CT) scan to rule out SAH. With no excessive patient movement aunng the scan and CT equipment of good quality, subarachnoid blood can be detected on a noncontrast head CT in as many as 95% of cases (van Gijn J., Bromberg J.E., Lindsay K.W., Hasan D., Vermeulen M., 1994). The site of the aneurysmal rupture can frequently be inferred from the location of the hemorrhage or hematoma on CT **(Fig.** 1).

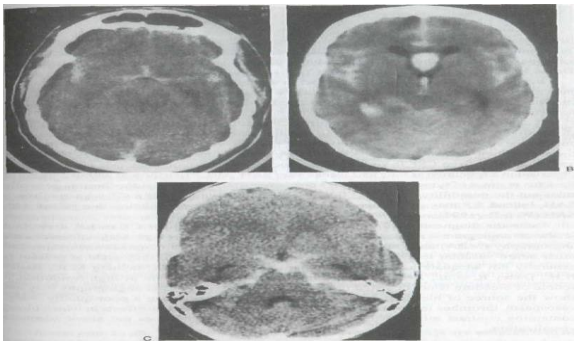


Fig. 1. Axial, noncontrast brain computed tomography scans demonstrating different varieties of subarachnoid hemorrhage (SAH) (A) Diffuse. (B) Anterior interhemispheric with associated hematoma. (C) Perimesencephalic.

The amount of subarachnoid blood apparent on CT scan has prognostic value in predicting the risk of subsequent vasospasm. Patients with scant amounts of subarachnoid blood on CT are much less likely to develop clinically significant vasospasm than are those with thick basal clots (Fisher CM., Kistler J.P. Davis J.M., 1980).

In patients with negative CT scans but in whom SAH is strongly suspected, a lumbar puncture (LP) is indi cated. A CT scan may be negative if the amount of SAH is small. It may be mistakenly read as negative if the bleed is subacute and isodense with surrounding structures or if the scan is of poor quality.

It is important to distinguish a positive LP result from a traumatic tap. Although this distinction is generally made by noting a diminution of red blood cell (RBC) counts in successive specimen tubes, centrifugation of the cerebrospinal fluid (CSF) and checking the supernatant for xanthochromia is the more sensitive method. The rate of clearance of xanthochromia from CSF is variable. Xanthochromia and lymphocytosis can usually be observed in a specimen as long as 2 weeks after SAH. An LP (performed within 2 weeks of the ictus or onset of symptoms) that does not demonstrate RBC or xanthochromia generally rules out the possibility of SAH. LP is rarely, if ever, indicated if a CT scan confirms a SAH. Indeed, LP may cause increased risk of rebleeding or herniation in a person with SAH (Weir B., 1994).

Once the diagnosis of SAH has been established by either CT or LP, a cerebral (catheter) angiogram is needed to ascertain the source of hemorrhage. Magnetic resonance angiography (MRA) and CT angiography have been used to diagnose aneurysms and other neuro-vascular lesions **(Fig.** 22), but the anatomic detail they yield at present is generally not adequate to supplant conventional angiography (Mayberg M.R., Batjer H.H., Dacey R. et al., 1994). Even with a four-vessel angiogram of high resolution, a source of bleeding is not detected in 15% to 20% of cases. Initial angiography may not show the source of bleeding for a variety of reasons, including a poor-quality study, vasospasm, thrombus in the aneurysm preventing filling, and flow effects in which blood containing contrast mixes with blood without contrast and does not allow adequate visualization.

Fig. 22. Anterior communicating artery aneurysm visualized in the same patient. (A) Magnetic resonance angiography (MRA).

If the initial angiogram is negative, repeat angiography performed 1 to 2 weeks after the initial study (to allow resolution of acute medical issues, cerebral edema, or vasospasm) will demonstrate a lesion in an additional 1% to 2% of cases. Whether this relatively small yield is worth the additional risk and cost of repeat angiography is debatable. This decision is usually made on the basis of several factors specific to the particular case, such as the quality of the original study, the patient's age and general medical condition, and the pattern of hemorrhage observed on the initial CT scan. Mounting evidence shows that a patient who sustains nontraumatic perimesencephalic SAH and who has a negative angiogram (of good quality) need not undergo repeat angiography. This subset of patients does not appear to experience vasospasm or early rebleed (Rinkel G.J., van Gijn J., Wijdicks E.F., 1993). Their overall long-term outcome is generally excellent, and there are no documented cases of delayed hemorrhage. Although it is not known with certainty, the source of perimesencephalic SAH is believed to be a ruptured vein. Before one attributes a patient's SAH to this source, the existence of structural pathology, particularly posterior circulation aneurysms, should be excluded. Patients with patterns of hemorrhage that are more extensive than the perimesencephalic type, in whom results of cerebral angiography are negative, appear to have a less benign course. For such patients, repeat angiography is strongly recommended.

Spinal angiography is considered in cases in which results of cerebral angiography are negative and there is clinical and/or radiographic (CT or MRI) suspicion of spinal pathology. MRI is performed in patients with angiogram-negative SAH to rule out the presence of structural lesions that might be angiographically occult, particularly cavernous malformation.

ETIOLOGY

Aneurysm. In all, 75% to 85% of cases of nontraumatic SAH are estimated to be attributable to ruptured aneurysms. Most such aneurysms are saccular or berry aneurysms rather than infectious, traumatic, dissecting, fusiform, or tumor-related aneurysms. The pathogenesis of saccular aneurysms is not fully understood, although there appear to be both congenital and acquired components.

Aneurysms develop at vascular bifurcations at or around the circle of Willis. Studies in which models were used have demonstrated that maximal wall stress exists at the apex of a vascular bifurcation in line with the direction of flow. As aneurysms enlarge, risk of hemorrhage appears to increase. This is in accord with the law of LaPlace, which states that as the radius of a sphere increases, less force is necessary to enlarge it (Mayberg M.R., Batjer H.H., Dacey R. et al., 1994). Prospective trials have shown that aneurysms with diameters less than 3 mm are at substantially less risk of rupture than those with diameters greater than 10 mm. Nonetheless, an aneurysm of any size can rupture. For unruptured aneurysms, the annual risk of hemorrhage is between 1% and 2% per year (Juvela S, Porras M, Heiskanen O., 1993). Ruptured aneurysms have a higher rebleed rate particularly soon after the initial event. In the Cooperative Aneurysm Study, the rate of rerupture was 4% in the first 24 hours and 1 % to 2% per day for the next 4 weeks. After the first month, the annualized rebleed rate is approximately 3% to 4%. Approximately 20% of patients with aneurysmal SAH will harbor multiple aneurysms. The history of the event, the physical findings on examination, and the distribution of blood apparent on CT scan provide information that allows the clinician to decide which aneurysm bled. If this information is inconclusive, the size and configuration of the aneurysms on angiography can indicate the likely site of hemorrhage. Typically, the larger and/ or more irregular or loculated aneurysm is more likely the one that bled (Kopitnik T.A., Samson D.S., 1993). **Cerebral aneurysms have been associated with various systemic diseases,** including Marfan's syndrome, Ehlers-Danlos syndrome, pseudoxanthoma elasticum, fibromuscular dysplasia, sickle cell disease, polycystic kidney disease, and coarctation of the aorta .

A small number of patients with aneurysms have a proven familial occurrence of aneurysmal SAH. As compared with nonfamilial («sporadic») cases, the patients are usually younger and more often female (Leblanc R., 1996). For families with two affected first-degree relatives, screening of the remaining first degree family members has been recommended. MRA is an adequate initial screening tool. Although MRA is not as sensitive as catheter angiography, especially when one is searching for aneurysms less than 4 mm in diameter, neither is it as risky or costly. If MRA screening shows pathology or a suspicion of pathology, catheter angiography should be performed to allow more definitive evaluation. **Approximately 90% of intracranial aneurysms are found in the anterior circulation** (Rodman K.D., Furlan A.J., Awadl.A., 1993). Common sites include the anterior cerebral/anterior communicating artery (ACA) junction, the internal carotid/PCA bifurcation, and the middle cerebral artery bifurcation

(or trimrcation). Common sites for posterior circulation aneurysms include the vertebral/basilar artery junction, the vertebral/ posterior inferior cerebellar artery junction, and the basilar summit.

Aneurysms of diameter greater than 25 mm are termed «giant.» These comprise approximately 5% to 7% of all intracranial aneurysms. Treatment of patients who sustain aneurysmal SAH is predicated on the understanding that rebleeding is the most significant preventable cause of death and disability for those who survive the initial ictus (Broderick J.P., Brott T.G., Duldner J.E., Tomsick T, Leach A., 1994). Therefore, a premium is placed on expedient transfer of the patient to a facility where diagnosis can be confirmed and stabilization and management can be undertaken in timely fashion.

Arteriovenous Malformations. Approximately 5% TO 9% OF PATIENTS WHO experience spontaneous SAN will be shown to harbor an AVM (Kopitnik T.A., Samson D.S., 1993). AVM hemorrhages are generally intraparenchymal but SAH occurs in about one third of AVM hemorrhages. AVM hemorrhage is usually the result of rupture of arterialized venous channels. Often, intranidal or arterial feeder aneurysms are evident on angiography. As compared with that in aneurysmal SAH, the **risk of rebleeding owing to an AVM is low** (<l% in the first 2 weeks versus approximately 20% for aneurysms in the same time period). In addition, vasospasm is rare in this group of patients. For these reasons, the initial treatment for patients with AVM-related SAH differs from that for patients with aneurysmal SAH. After the acute hemorrhage is resolved, a patient with an AVM usually has time to weigh the available treatment options. Early surgical intervention is undertaken only when a life-threatening hematoma with possible brain herniation exists. Early intervention after AVM-associated hemorrhage is still indicated in the settling of mass effect from hematoma or rupture of arterial aneurysm associated with AVM. This latter scenario is managed as other cases of aneurysmal SAH.

A decision regarding the advisability, timing, and method or methods of treatment is made on an individual basis. The patient's age, general health, family situation, as well as the size, location, drainage pattern, and specific angioarchitectural features of the AVM, are some of the factors of the decision-making process. AVMs are often treated with more than one procedure and more than one method; and embolization, microneurosurgery, and radiosurgery may be used in their treatment. In patients with both an aneurysm and an AVM demonstrated angiographically, the source of hemorrhage is more likely to be the aneurysm.

Bleeding Disorders. In itself anticoagulation is an uncommon cause of spontaneous SAH, in contrast to subdural or intracerebral hemorrhage. In a series of 116 patients receiving anticoagulant treatment with intracranial extracerebral hemorrhages only seven had SAH. Two of the seven had had associated trauma and two had aneurysms. Therefore, in only three cases was no other underlying cause for the SAH discovered (other than the anticoagulation).

Severe coagulopathy that is unrelated to anticoagulant treatment can lead to intracranial hemorrhage, but rarely is it confined to the subarachnoid space. Typically, these hemorrhages are in a cortical or subcortical location. The subset of patients with such hemorrhages tend to have lobar hematomas of substantial volume. Patients with spontaneous intracerebral hemorrhages as a result of underlying bleeding disorders often have evidence of systemic bleeding as well.

Vascular Disease. Vertebral artery dissection can lead to SAH if the diseased portion of the blood vessel extends into the intradural space. In one postmortem series of patients who died of SAH, vertebral artery dissection was discovered in 5 of 110 cases (Sasaki O., Ogawa H., Koike T., Koizumi T., Tanaka R.A., 1991). Vertebral artery dissection should be considered in any patient with a history of unusual neck movement or manipulation before onset of symptoms and/or findings of lower cranial nerve palsies.

Carotid artery dissection is believed to be a much less common cause of SAH than vertebral artery dissection, probably because the site of most carotid dissections is in the neck far from the portion of vessel that enters the subarachnoid space. In the vertebral artery, the dissections is often adjacent to the craniocervical junction and may extend into the subarachnoid space. Carotid artery dissections associated with traumatic basal skull fractures may involve more distal segments of the vessel. These cases are more likely to be associated with SAH than are other carotid artery dissections.

The diagnosis of vascular dissection in SAH is usually made at the time of angiography. The demonstration of an intimal flap, luminal narrowing, a double lumen, a tapered (or truncated) occlusion, or a pseudoaneurysm can confirm the diagnosis. Nonetheless unremarkable angiograms are possible. In one series of 14 patients with veitebral artery dissection, the initial angiogram was normal in two and showed only nonspecific arterial narrowing in two others. MRI imaging (MRI) often demonstrates mural thrombus or a false lumen, thereby establishing a diagnosis that may be unclear based on the angiogram. Rebleeding from arterial dissection is common and the mortality rate approximates 50%. Other vascular

pathologies that rarely present as SAH include amyloid angiopathy, hypertensive vasculopathy, Moyamoya disease, advanced atherosclerosis, fibromuscular dysplasia, and vasculitis.

Infection. Patients with infectious endocarditis can present with SAH after rupture of a mycotic aneurysm. In general, mycotic or infectious aneurysms are smaller and more distally located on the cerebral vasculature than saccular or berry aneurysms. They often present as intracerebral hematomas, although basal SAH (indistinguishable from that of a saccular aneurysm) can also occur.

Mycotic aneurysms are focal thin outpouchings of the vessel wall with inflammatory infiltrate. Fibrous or inflammatory tissue has largely replaced normal wall components. Because wall integrity is so severely compromised, clipping these aneurysms may be treacherous. Trapping and excision of the diseased portion of vessel, with or without a bypass procedure, is frequently necessary.

Although surgical or endovascular treatment of these lesions is generally recommended because of the risk of rebleeding, there are reports of spontaneous angio-graphic resolution of mycotic aneurysms after systemic antibiotic administration.

Fungal infections such as aspergillosis can also present with SAH secondary to mycotic aneurysm rupture. Aspergillosis usually involves the proximal part of the carotid or vertebrobasilar arteries, and SAH associated with this infection is often fatal.

Neoplasia. Rarely, brain tumors will present with SAH. More commonly, hemorrhage associated with neoplasms is intra-tumoral or intraparenchymal. Hemorrhages occur principally in malignant tumors such as glioblastoma multiforme or metastasis (e.g., renal, bronchogenic, melanoma) but may also occur with benign tumors. SAH from a cervical meningioma has also been reported.

Trauma. Traumatic SAH is found in as many as 25% of cases involving severe closed head injury (SCHI). In these cases, SAH is rarely seen in isolation; it is commonly associated with cerebral contusion and/or diffuse brain injury (Demircivi F., Ozkan N., Buyukkcccci S. Yurt I., Miniksar F., Tektas S., 1993). Among patients with milder head injuries, traumatic SAH is unusual, occurring in less than 5% of cases (Fogelholm R, Hernesniemi J, Vapalahti M., 1993)- **Fatal traumatic SAH** has been reported extensively, particularly in the forensic and medicolegal literature. The most common etiology is vertebral artery stretch injury or laceration at the level of CI to C2, with intracranial dissection. These cases generally involve one or more blows to the head, with extension or twisting of the neck. Alcohol intoxication is detected in a substantial percentage of these cases. The history of injury may distinguish whether the observed SAH was a result of the traumatic incident or if spontaneous SAH led to the subsequent accident.

TREATMENT

Microsurgery. Clipping of a ruptured intracerebral aneurysm remains the definitive method for lesional obliteration and prevention of rebleeding. Furthermore, once an aneurysm is clipped, subsequent vasospasm can be treated aggressively and more effectively. The timing of aneurysm surgery is still debated to some degree, though there is general consensus that good-grade patients (Hunt-Hess grades I to III) should have early (within the first 48 to 72 hours) surgery. This approach is predicated on the understanding that recurrent hemorrhage is the major cause of death in patients who survive their initial bleed (Broderick J.P., Brott T.G., Duldner J.E., Tomsick T., Leach A., 1994). With modern neurosurgical techniques, early surgery in these patients is not as formidable an undertaking as it once was. Experienced surgeons in some neurovascular centers have advocated early surgery in poor-grade patients if they are hemodynamically stable and intracranial pressure (1CP) is manageable (Crowell R.M., Ogilvy C.S., Gress D.R., Kistler J.R, 1996).

For patients who are of extremely poor neurologic grade and/or otherwise medically unstable, surgery is delayed until they have stabilized and are believed capable of making a meaningful recovery. Surgery may also be delayed if a patient experienced SAH several days earlier and is neurologically unstable from symptomatic vasospasm. Whether surgery should be delayed solely because of angiographic, but asymptomatic, spasm is not clear. If surgery is undertaken in such circumstances, careful attention must be paid to avoiding hypotension, dehydration, and hypocapnea.

Another option for some patients who are too unstable to undergo early surgery is endovascular therapy (described below). Whether a better overall outcome is achieved in such patients by early endovascular intervention or by medical management followed by surgery once the patient has stabilized is not clear. Immediate surgical intervention is indicated for patients who present with

significant intracerebral clot and mass effect. In such case, there is rarely time for formal angiography. The location of the aneurysm can often be inferred from the location of the clot on CT scan. Occasionally, a contrast CT scan will identify the aneurysm.

Surgery also provides an opportunity to irrigate the basal cisterns to remove the blood that causes vasospasm. Some centers are instilling tissue plasminogen activator (tPA) into the basal cisterns to lyse clot and reduce the incidence of vasospasm (Mizoi K, Yoshimoto T, Takahashi A, Fujiwara S, Koshu K, Sugawara T., 1993). Preliminary reports are promising, but the risk of rehemorrhage may be increased.

A postoperative angiogram is usually performed to confirm obliteration of the aneurysm. Studies have shown that even an aneurysmal remnant of 1 to 2 mm can grow in time (Whisnant J.P., Sacco S.E., O'Fallon W.M., Fode N.C., Sundt T.J., 1993). Small remnants should be followed angiographically and considered for treatment if they grow. Larger remnants should be treated with reoperation or endovascular coiling.

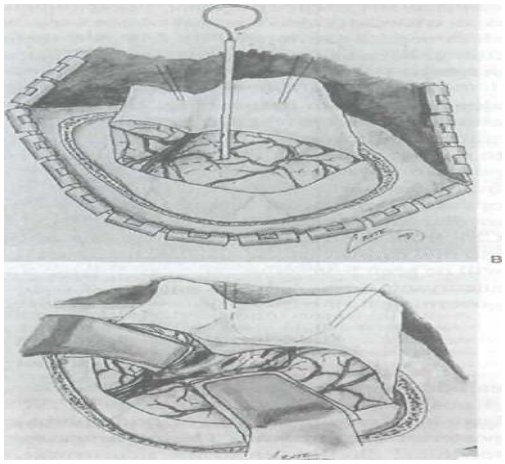
Endovascular Therapy. Endovascular placement of Guglielmi detachable coils (GDCs) has been shown to be a safe treatment option in the hands *of* an experienced
endovascular therapist for select patients. Currently, approved indications for this treatment are limited to patients deemed to be of poor surgical risk or aneurysms with anticipated surgical difficulty. GDCs appear to be most effective in non giant aneurysms with narrow necks. Currently, the long-term success of this technique cannot be assessed because aneurysmal recanalization (and hemorrhage) has occurred, even after apparently successful GDC obliteration of the lesion is demonstrated on the initial angiogram. Endovascular balloon occlusion of a parent vessel or trapping is considered in cases in which direct surgical clipping is not possible or carries an unacceptably high morbidity, but is rarely performed in the acute period.

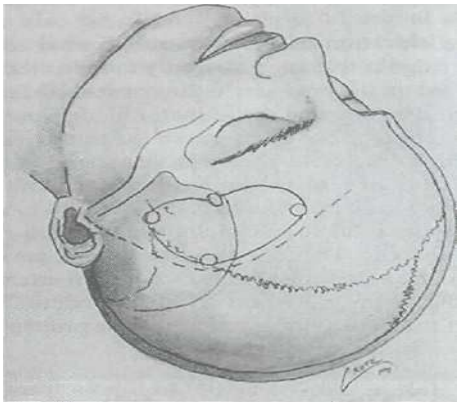
Tilt is accomplished by tilting the vertex of the skull so the maxillary eminence rises superior to the brow. Tilt allows the frontal lobe to fall away from the anterior cranial fossa floor, exposing the basal vascular structures with minimal retraction. Care must be taken to preserve rotation and flexion during this maneuver.

Surgical Incision. The optimal surgical incision provides adequate exposure, acceptable cosmesis, and tissue preservation. For a pterional craniotomy, the surgical incision begins about 1 cm anterior to the tragus and is curved anteriorly and vertically to the midline, as shown in Fig. 2. Keeping the incision behind the hairline provides the best cosmetic result. The frontal branch of the facial nerve and at least one branch of the superficial temporal artery are preserved. The incision is taken through skin, galea, temporalis fascia, and muscle to bone, allowing reflection of these structures in a single layer. A thick, folded sponge is placed under the reflected flap to protect the neurovascular bundle and the eye. This flap is then held in place by fishhook retractors. A very promising innovation to the single layer flap involves scalp reflection in the subgaleal plane with direct facial nerve branch elevation from the fat pad within the temporalis fascia. The temporalis muscle can then be incised and reflected anteriorly or posteriorly (for basilar aneurysms), leaving a cuff of fascia and muscle for subsequent closure.

Craniotomy. The pterional craniotomy flap may vary somewhat depending on the surgical target, but the standard flap is delineated with three or four burr holes (Fig. 2). The first burr hole is placed low on the temporal squama, the second superior and slightly anterior to the first below the superior temporal line, the third on the «anatomic keyhole,» and the fourth on the forehead anterior and medial to the second hole. This optional fourth burr hole is useful in the elderly patient with adherent dura. A power-driven craniotome is used to connect these holes, and the craniotomy flap is removed. A generous subtemporal craniectomy is then performed with the aid of rongeurs, and the dura is carefully freed from its sphenoid wing attachment. The lateral third of the sphenoid ridge is then drilled using a cutting burr or resected with rongeurs as far medially as the lateral aspect of the superior orbital fissure. This sphenoid ridge removal described by Yasargil (Yasargil M.G., Fox J.L., Ray M.W., 1975) provides excellent exposure of the circle of Willis. The inner table of the frontal bone is then drilled anteriorly from the anatomic keyhole to the most anterior margin of the bony incision near the midpupillary line. This maneuver gives excellent exposure to the parasellar region with minimal frontal lobe retraction and is particularly helpful in ACoA aneurysm exposure.

Fig. 2. The pterional approach to a left carotid - ophthalmic aneurysm. (A) head position, planned scalp incision, burr hole, placement, and craniotomy. (B) craniotomy, dural incision and the reflection of dural flap. A ventricular catheter is placed at Paine's point. (C) Initial brain retraction of the posterior frontal lobe along the sylvian fissure.





Dural Incision. The dura is opened in a semilunar fashion beginning posteriorly and proceeding with a gentle curve anteriorly and inferiorly to the craniectomy corner. This dural flap is then reflected over the sphenoid ridge and tacked up with sutures to maximize cortical exposure **(Fig.** 2). The remaining intact dura is covered by a large, moist gelatin sponge or Telfa pad and may be tacked to the bony ridges with dural sutures. Once the parasylvian region is satisfactorily exposed, the surgical microscope is brought into the field and the remaining procedure is performed with microsurgical technique.

Brain Retraction and Initial Exposure. To promote maximal brain relaxation, some surgeons who prefer the use of spinal fluid drainage will have the anesthesiologist open the drain at this point. An alternative is to place a ventricular catheter for CSF drainage intraoperatively. A method frequently used by the authors (Paine J.T. et al., 1988) is ventricular catheter placement perpendicular to the cortex on the vertex of an isosceles right triangle (Paine's point) whose base overlies the sylvian fissure and whose remaining sides are 2.5 cm long **(Fig.** 2). A catheter placed to 5-cm depth will reliably enter the frontal horn of the lateral ventricle. This ventricular catheter may be removed following surgery.

Initial brain retraction involves progressive elevation of the posterior frontal lobe along the sylvian fissure **(Fig.** 2). The sphenoid ridge is followed medially to the anterior clinoid process to reach the carotid cistern. To avoid undue pressure to the cortical surface, smooth, uniform orbital cortex elevation is achieved by keeping the retractor blade parallel to the bony floor of the frontal fossa. When the retractor has been advanced to the most posterior aspect of the frontoorbital surface, a thick arachnoid sheath is visualized at the frontotem-poral junction. Using either microscissors or a micro-knife, this arachnoid is opened sharply from the prechiasmatic region across the carotid cistern and laterally to the medial aspect of the sylvian fissure. Additional brain relaxation is achieved by releasing CSF from the basal cisterns with this arachnoid opening. The olfactory tract, optic nerve, internal carotid artery, and carotid bifurcation into the anterior and middle cerebral arteries will be seen following generous sylvian dissection. To this point, nearly all procedures for aneurysms through the pterional approach are performed in a similar fashion. The principles governing further dissection are influenced by the specific aneurysm site.