

# Controversies in the Neurosurgical Management of Cerebellar Hemorrhage and Infarction

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## Abstract and Introduction

### Abstract

Evidence-based guidelines for the management of hemorrhagic and ischemic cerebellar stroke are sparse, and most available data come from Class III studies. As a result, opinions and practices regarding the nature and role of neurosurgical intervention vary widely. A comprehensive literature review was conducted to adjudicate several contentious issues, such as the difference in the management of cerebellar hemorrhage versus infarction, criteria for imaging to exclude an underlying structural lesion, the value of MRI for patient selection, the role of external ventricular drainage, the indications for operative management, the timing of surgical intervention, and various options of surgical technique, among others. Treatment algorithms proposed in several different studies are compared and contrasted. This analysis is concluded by a summary of the recommendations from the American Stroke Association, which advises that patients with cerebellar hemorrhage who experience neurological deterioration or who have brainstem compression and/or hydrocephalus due to ventricular obstruction should undergo surgical evacuation of the hemorrhage as soon as possible, and that initial treatment of such patients with ventricular drainage alone rather than surgical removal of the hemorrhage is not recommended.

### Introduction

The management of cerebellar hemorrhage has endured controversy ever since Sir Charles Ballance reported the first successful surgical evacuation in 1906.<sup>[2]</sup> Institutional and individual variations abound in the management of cerebellar hemorrhage. To a degree, this debate is due to the relatively flimsy quality of the medical literature in support of different practices. For instance, in advocating against external ventricular drainage alone in the treatment of patients with cerebellar hemorrhage who are deteriorating neurologically or who have brainstem compression and/or hydrocephalus from ventricular obstruction, the American Stroke Association relies on evidence rated as Level C, the weakest category in effect at the time (consensus opinion of experts, case studies, or standard of care).<sup>[24]</sup> Similarly, their prior recommendation that angiography is not required for older, hypertensive patients with cerebellar hemorrhage in whom CT findings do not suggest a structural lesion is based upon Level V evidence, the weakest category in effect at the time (data from anecdotal case series only).<sup>[4]</sup>

Several randomized trials comparing early surgery with initial conservative management for ICH have been conducted, including the recent Surgical Trial in Intracerebral Hemorrhage (STICH).<sup>[23]</sup> Overall, these studies have largely shown no benefit to surgery, although post hoc subgroup analysis reveals some exceptions. Patients with cerebellar ICH have been excluded from all these randomized trials, because clinical equipoise was not believed to be present.<sup>[24]</sup> As one expert commented about the related condition of cerebellar infarction, "the results of surgery have been so consistently favorable in patients who clearly were progressively deteriorating that it seems fair to say that this is one surgical indication that does not need the scrutiny of a randomized study."<sup>[12]</sup>

As a result of these biases, data principally consist of uncontrolled, single institution retrospective case series (Class III evidence). Collectively, however, these reports suggest that the benefit of surgery is not so straightforward. Donauer et al.<sup>[7]</sup> reviewed 21 papers from 1958 to 1993 and performed a meta-analysis comparing medical versus operative treatment of cerebellar ICH. In the cohort of 357 patients who underwent surgery, the mortality rate was 49%, while that in the 269 patients treated conservatively was 50%. Similarly, Hankey and Hon<sup>[10]</sup> reviewed 8 prior series of surgery for infratentorial hemorrhage comprising a total of 405 patients. One study suggested overall benefit, while 2 studies reported benefit only in certain subgroups (conscious or drowsy but deteriorating patients), and the remaining 5 studies were either inconclusive or showed no benefit of surgery.

In an effort to provide more concreteness to this issue and to elucidate related concepts in the management of cerebellar hemorrhage and infarction, this article reviews relevant studies from the past century. It begins with an overview of the pathogenesis and natural history, which form the foundation and rationale for all treatment. Next, 9 separate areas of controversy are explored in detail. The review concludes with a summary of the recommendations from the American Stroke Association, whose position statements have evolved considerably from their first publication in 1999 to 2010.<sup>[3,4,24]</sup>

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## Pathophysiology and Natural History

Spontaneous cerebellar hematomas represent approximately 10%–15% of all ICH.<sup>[11,18,35]</sup> As with cerebellar infarction, cerebellar hemorrhage occurs most frequently in the 5th through the 8th decades of life and with greater frequency in males than in females.<sup>[11,18]</sup> Between 60% and 90% of all spontaneous cerebellar hemorrhages occur in hypertensive patients. Vascular malformations, coagulopathies (including the use of anticoagulants), neoplasms, aneurysms, cerebral amyloid angiopathy, and trauma account for the remainder.<sup>[11,17]</sup> In younger patients, underlying structural conditions are the prevailing causes.

Cerebellar infarctions, on the other hand, may result from cardiac emboli, traumatic injury to the vertebral arteries, and other causes.<sup>[11,14,26]</sup> The majority of patients also have hypertension.<sup>[13,26]</sup> The infarction most frequently occurs in the vascular distribution of the posterior inferior cerebellar artery, but the anterior inferior cerebellar artery and/or superior cerebellar artery territories can also be involved.<sup>[14,26]</sup> Cerebellar infarctions are approximately two-thirds as common as cerebellar hemorrhage.<sup>[11]</sup>

In hypertensive patients, cerebellar hematoma is believed to result from rupture of microaneurysms, as first proposed by Charcot and Bouchard, and recently confirmed.<sup>[31]</sup> Typically, these hemorrhages begin in the area of the dentate nucleus and then spread throughout the ipsilateral hemisphere.<sup>[15]</sup> They may also extend across the vermis to the contralateral side. Although they commonly spread into the cerebellar peduncles or rupture into the fourth ventricle, only rarely do they directly involve the brainstem.<sup>[11]</sup> Dizziness, headache, nausea, vomiting, loss of balance, and difficulty walking are the most common presenting symptoms of both cerebellar hemorrhage and infarction.<sup>[1,11,14,26,30]</sup>

Clinical deterioration befalls up to 50% of patients with cerebellar ICH.<sup>[25]</sup> In its mild form, deterioration manifests as irritability, confusion, or somnolence, while the more severe form presents as coma, stupor, posturing, and hemodynamic or respiratory instability due to loss of brainstem regulation.<sup>[13]</sup> The peak incidence of deterioration is 3 days after onset, although it may occur within hours or even weeks later. When deterioration occurs, mortality has been reported to be high (25%–100%), regardless of treatment.<sup>[1,7,17,18,30]</sup>

Deterioration can occur unpredictably, even in patients who appear to have reached a clinical plateau. <sup>[11,17]</sup> Ott et al. <sup>[25]</sup> reported that 50% of patients who remained alert and relatively stable for 2 days degenerated into coma over the course of the next several days, and a disconcerting 25% of patients who remained awake for 7 days subsequently deteriorated.

The causes of deterioration are protean and include increased mass effect from surrounding edema or expansion of the hematoma from repeat bleeding. Either mechanism can cause direct brainstem compression, which leads to upwards herniation through the tentorial incisura or downward tonsillar herniation through the foramen magnum. Obstructive hydrocephalus, caused by intraventricular extension of the hemorrhage or by compression of the fourth ventricle, is another mechanism of clinical decline.

In cerebellar infarction, brain swelling results from both cytotoxic and vasogenic edema. Initially, brain ischemia disrupts cell membrane integrity, which causes the accumulation of intracellular fluid. Later, vasogenic edema results from the diffusion of protein-bound fluid across a damaged blood-brain barrier. <sup>[5,19]</sup> With progressive mass effect caused by the infarct and surrounding edema, brainstem compression and/or fourth ventricle compression can result. The range of time that can elapse between symptom onset and further neurological deterioration is typically 1–7 days, with a median and mode of 3 days. <sup>[5,13,14]</sup> However, the likelihood of deterioration has been reported to be lower in cerebellar infarct (7%–32%) than in hemorrhage. <sup>[1,14]</sup>

Whereas hydrocephalus and brainstem compression can both cause decreased level of consciousness, the latter is alleged to have associated focal neurological signs. <sup>[11,13,29]</sup> Early compression of the dorsal pons results in ipsilateral sixth nerve paresis of voluntary lateral gaze that can be overcome with caloric stimulation. Later, as the compression progresses, conjugate gaze paresis that is unresponsive to caloric stimulation occurs from pressure upon the horizontal gaze center. At this stage, ipsilateral peripheral-type facial paresis is usually present due to concomitant compression of the facial colliculus. Babinski signs, Horner syndrome, and hemiparesis are all late signs of brainstem compression. <sup>[11,13,29]</sup> Recognition of these findings, along with neuroimaging, can help distinguish between altered level of consciousness due to hydrocephalus versus that due to direct brainstem compression. Appropriate therapy (for example, ventricular drainage versus surgical decompression) can then be targeted to the underlying mechanism.

The tenets of medical management of cerebellar hemorrhage are similar to those of supratentorial ICH. <sup>[24]</sup> Patients are generally monitored in a critical care setting, with frequent neurological assessment. Those with severe coagulation factor deficiency or thrombocytopenia should receive transfusion of appropriate blood products to correct the disorder. Patients whose hemorrhage is caused by oral anticoagulation therapy should receive intravenous vitamin K as well as therapy to replace the vitamin K–dependent factors. Prothrombin complex concentrates have not been proven to improve outcome compared with fresh-frozen plasma, but may have fewer complications. <sup>[24]</sup> Recombinant factor VIIa is not routinely recommended as the sole agent for reversal of oral anticoagulation therapy. <sup>[24]</sup> All patients should undergo intermittent pneumatic compression for prevention of venous thromboembolism in addition to elastic stockings. After documentation of cessation of bleeding, low-dose subcutaneous heparin formulations may be considered as well. <sup>[24]</sup> Glucose should be monitored closely, and normoglycemia is recommended. The management of blood pressure remains disputed, without clear guidelines or target parameters, but in patients presenting with systolic blood pressure of 150 to 220 mm Hg, acute lowering to 140 mm Hg is probably safe. <sup>[24]</sup>

Therapeutic cooling has not been adequately studied in cerebellar ICH, although most practitioners favor avoidance of hyperthermia.

### **Hemorrhagic versus Ischemic Cerebellar Stroke**

Since the first reports of decompressive surgery performed by Fairburn and Oliver<sup>[8]</sup> and by Lindgren,<sup>[20]</sup> both in 1956, the potential value of suboccipital craniectomy and resection of necrotic tissue in cerebellar infarction has been recognized. However, cerebellar hemorrhage and infarction are distinct entities, which calls into question whether the same management principles should apply to each.

Mathew et al.<sup>[21]</sup> compared the neurosurgical management of 48 patients with cerebellar ICH to that of 71 patients with cerebellar infarction. They found that patients with hematoma were more likely to be in a coma and more likely to have brainstem compression upon presentation than those with infarction. This explains why 75% of their patients with ICH required surgery, while it was necessary in only 24% for infarction.

In both cerebellar hemorrhage and infarction, perilesional edema can aggravate the space-occupying effect within the confines of the posterior fossa. However, a condition unique to ICH is the toxic effects of blood products and associated inflammation, which might provide impetus for its removal regardless of the mechanical compression of adjacent tissue. Furthermore, cerebellar hemorrhage may extend into the ventricle system, thus providing an additional mechanism of hydrocephalus besides fourth ventricle effacement. Conversely, cerebellar ICH only rarely extends directly into the brainstem.<sup>[17]</sup>

By comparison, cerebellar infarction does not lead to intraventricular hemorrhage and is thus less likely to cause hydrocephalus than cerebellar hematoma. In the patient series of Auer et al.,<sup>[1]</sup> occlusive hydrocephalus developed in 75% of patients with cerebellar hemorrhage but only 23% of those with cerebellar infarction. Patients with cerebellar ICH also had a higher incidence of hydrocephalus than those with cerebellar infarct in the series by Mathew et al.<sup>[21]</sup> However, cerebellar infarction is more likely to directly involve the brainstem than cerebellar ICH due to shared vascular territory; this occurred in 2 of 40 patients in the series of Auer et al.<sup>[1]</sup>

Emerging data suggest that in some circumstances, the area of restricted diffusion apparent on MRI, once believed to represent permanent damage, may be reversible.<sup>[16]</sup> Therefore, it is conceivable that resection of this presumed necrotic tissue in cerebellar infarction may actually compromise recovery.

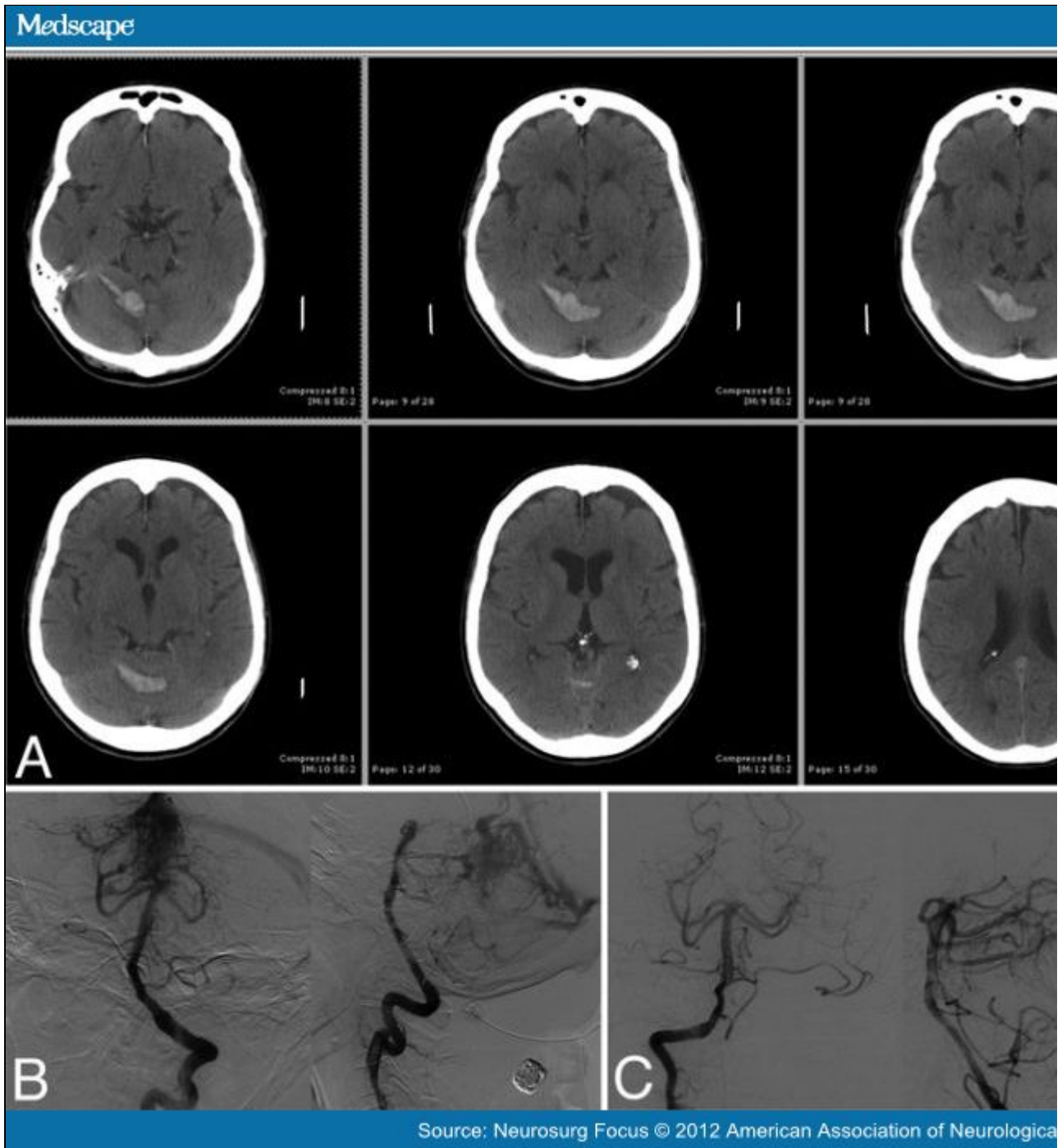
In light of these considerations, a policy that limits the extent of resection of apparent necrotic tissue to the minimum needed to achieve adequate decompression appears reasonable, although the data in support of this practice are not robust.

### **Criteria for CT Angiogram or Catheter Angiogram**

Although most cases of spontaneous cerebellar hemorrhage are the result of hypertension, some are caused by underlying lesions. In Kobayashi et al.'s series of 110 patients,<sup>[18]</sup> for example, 5 hemorrhages resulted from a cerebellar AVM, 2 resulted from a cerebellar tumor, and the remaining 103 were believed to be caused by hypertension on the basis of prior history and/or negative angiographic studies.

Even in the presence of preexisting hypertension, however, as many as 36% of all ICH cases are associated with secondary causes.<sup>[35]</sup> The indications, nature, and diagnostic

accuracy of imaging for an underlying structural lesion in spontaneous cerebellar hemorrhage remains controversial. The presence of subarachnoid blood, calcification, prominent vascular structures, or edema out of proportion to the size and age of the hemorrhage might suggest the presence of an underlying lesion. Similarly, a hemorrhage that has an unusual (geographic or noncircular) shape or is located in an unusual location, such as an epicenter remote from the dentate nucleus, might prompt further study (Fig. 1). However, features of CT in isolation had a sensitivity of only 77% and specificity of only 84% in 1 study.<sup>[9]</sup> Clinical features such as age and history of preexisting hypertension also affect the decision to pursue advanced imaging.



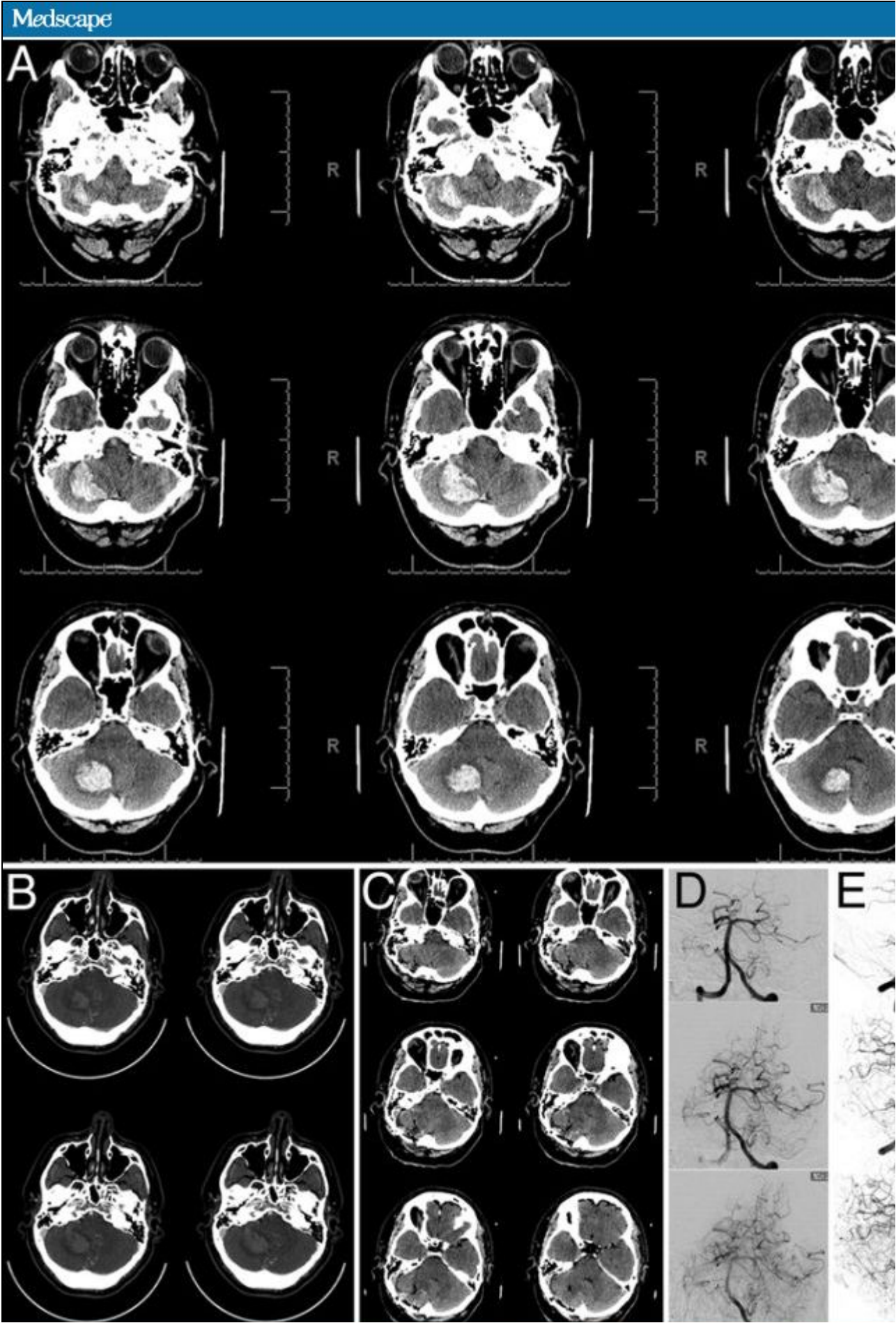
**Figure 1.**

Images obtained in a 77-year-old woman with a medical history significant for hypertension, heart murmur, and Crohn disease requiring large doses of aspirin. She awoke with headache, dizziness, and incoordination of the right arm. **A:** Unenhanced axial CT scans of her brain reveal acute hemorrhage of the right cerebellum extending to the tentorial surface and across the vermis to the left hemisphere. **B:** Although she had multiple risk factors for spontaneous intracerebral hemorrhage (such as hypertension, possible cardiac embolism related to the cause of her heart murmur, aspirin use, and possible age-related amyloid angiopathy), the unusual location of the hemorrhage and questionable prominence of vessels near the vein of Galen prompted further imaging. This frontal view of a left vertebral catheter angiogram reveals an AVM of the cerebellar vermis supplied primarily by branches of the bilateral superior cerebellar arteries. High risk features include feeding artery aneurysms bilaterally and venous outflow restriction of the draining vein coursing to the straight sinus. **C:** The patient underwent a series of staged liquid adhesive embolization sessions to protect against rehemorrhage. This frontal view of left vertebral catheter angiography after embolization reveals elimination of the feeding artery aneurysms and significantly reduced flow through the nidus.

Halpin et al. <sup>[9]</sup> performed a prospective evaluation of catheter cerebral angiography in the workup of 102 patients with spontaneous cerebral hematoma. Both supratentorial and cerebellar hemorrhages were included in the analysis. Overall, an aneurysm or AVM was the cause of the hemorrhage in 12.8% of hypertensive patients and in 18.2% of those with posterior fossa hemorrhage. The authors prospectively stratified the patients into 2 groups: those suspected to have a high likelihood of an underlying structural lesion based on CT features (Group 1) and those without such findings (Group 2). Catheter angiography was positive for AVM or aneurysm in 84% of the high-suspicion cohort and 24% in the low-suspicion one.

In another study, Zhu et al. <sup>[35]</sup> reviewed 206 consecutive patients with spontaneous ICH to determine the diagnostic yield of cerebral angiography. Both supra- and infratentorial hemorrhages were included. Patients in whom severe coagulopathy accounted for the ICH, those with bleeding into tumor, or those with predominant subarachnoid hemorrhage were excluded. Overall, the angiographic yield was significantly higher in patients less than the median age of 45 years and those without prior hypertension. In 15 patients with posterior fossa hemorrhage, 5 (33%) were found to have an underlying AVM. All 5 were normotensive, and the oldest patient in this group was 39. Another 6 patients had preexisting hypertension, the youngest of whom was 48. None of those 6 patients had positive angiography. The authors amalgamated hemorrhages in the putamen, thalamus, or posterior fossa into a single group for analysis. In this collective, the angiographic yield in patients with younger age and without preexisting hypertension was 48%, while in hypertensive patients the yield was 0%. They concluded that diagnostic angiography should not be routinely performed in patients with cerebellar hemorrhage over 45 years old with preexisting hypertension.

Although CT angiography and catheter angiography are potentially useful in the workup of spontaneous cerebellar ICH, neither is completely reliable. In some cases, compression of adjacent vessels by the hematoma can give the false appearance of a vascular malformation, thus reducing the specificity of these tests (Fig. 2). Conversely, the mass effect can conceal an underlying lesion, thus reducing the sensitivity of vascular studies performed acutely. In the series of Halpin et al., <sup>[9]</sup> for example, follow-up angiography at 3 months showed an AVM in 1 of 7 patients in the high-suspicion group, even though the original study results were normal. Thus, when clinically warranted, vascular studies should be repeated in a delayed fashion even if the initial workup is negative.



## Figure 2.

Images obtained in a 43-year-old man with no prior medical history, who awoke with headache, nausea, and dizziness, and whose blood pressure was 109/54 mm Hg. **A:** Axial CT scans without contrast demonstrate a large hemorrhage of the right cerebellar hemisphere. **B:** Because of the patient's young age and lack of hypertension, a high suspicion for an underlying structural lesion led to the performance of a CT angiogram, which demonstrates an abundantly prominent collection of vessels suggesting a possible AVM. In retrospect, however, this finding was found to merely represent compression of normal vessels by the mass effect of the bleed. **C:** Axial CT scan after surgical evacuation of the hemorrhage reveals no further mass effect. **D:** Frontal view of a vertebral artery catheter angiogram confirms the absence of an underlying AVM. **E:** Lateral view of a vertebral artery catheter angiogram confirms the absence of an underlying AVM.

### Role of External Ventricular Drainage

The indications for ventricular drainage in cerebellar hemorrhage and infarction remain contested. Many propose that hydrocephalus resulting from fourth ventricle obstruction should be treated with surgical decompression rather than CSF diversion.<sup>[5,17,33]</sup> Among this group, some advocate for pre- or intraoperative placement of a ventricular catheter, in case the decompression fails to achieve reconstitution of CSF pathways, while others renounce ventricular drainage altogether. One argument in favor of this approach is that decompressive surgery can shorten the duration of CSF diversion and reduce the need for a permanent shunt.<sup>[5]</sup> In many cases, the catheter can be removed within 72 hours after surgery.<sup>[5]</sup> In the series of Mathew et al.,<sup>[21]</sup> no patient required external ventricular drainage or delayed shunt placement after initial treatment with craniectomy.

Conversely, others argue that the effects of hydrocephalus can be falsely interpreted as resulting from brainstem compression. They advocate liberal use of CT imaging to distinguish between the two and institution of ventricular drainage if hydrocephalus is present and there is any clinical sign of elevated intracranial pressure.<sup>[7,17,18,26,29]</sup> In this algorithm, decompression of the brainstem follows only if the patient does not improve with relief of the hydrocephalus.

The threshold of drainage is another debated issue, due to the risk of upward herniation caused by altering the pressure gradient across the tentorium. This phenomenon is purported to have occurred in 2 of 30 patients in the series of van Loon et al.,<sup>[30]</sup> although others believe that concern for this event is overstated.<sup>[4,26,29]</sup> Conventional neurosurgical training advises drainage at no less than 15–20 mm Hg above the level of the third ventricle.<sup>[7]</sup>

These debates began in the pre-CT era, when attribution of the underlying cause of clinical deterioration (hydrocephalus vs brainstem compression) was left to clinical examination alone. In 1960, McKissock et al.<sup>[22]</sup> reported 34 cases of cerebellar hemorrhage. Nine patients were treated with ventricular drainage, and all of them died. In some of these patients, sudden decline after ventricular deterioration was hypothesized to be due to upward herniation, although this was not confirmed. Conversely, of the 14 patients treated with craniectomy and hematoma resection, 9 survived.

In more recent series, however, the potential value of ventricular drainage has been affirmed. In 2003, Raco et al.<sup>[26]</sup> reviewed 44 patients with cerebellar infarction. Of 17

patients who deteriorated clinically and required intervention, 13 patients with hydrocephalus underwent treatment with ventricular drainage, while the remaining 4 without hydrocephalus underwent craniectomy. Of the 13 initially managed using CSF diversion, 5 required subsequent craniectomy, while 8 were able to be treated with ventricular drainage alone. Similarly, in the series of Hornig et al.,<sup>[13]</sup> of 10 patients with cerebellar infarction and clinical deterioration initially treated with ventriculostomy, only 4 required secondary craniectomy because of continued decline. Outcome concerning mortality and functional status was not different when results of external ventricular drainage and suboccipital craniectomy were compared in this study.<sup>[13]</sup> In the series of van Loon et al.,<sup>[30]</sup> secondary craniectomy was necessary in only 6 (20%) of 30 patients with cerebellar ICH, while in other series the percentage of patients requiring craniectomy because of deterioration or failure to improve after ventricular drainage ranges from 25% to 80%.<sup>[1,13,16,30]</sup> Mathew et al.<sup>[21]</sup> found that more than half of their patients with cerebellar ICH who were initially treated with ventricular drainage subsequently required craniectomy, in contrast to only 2 (18%) of 11 patients with cerebellar infarction.

In summary, while some patients with cerebellar ICH and stroke who deteriorate neurologically and develop hydrocephalus have been successfully managed with external ventricular drainage alone, others still require surgery. As discussed below, the American Stroke Association favors operative resection of the ICH in this scenario.

### Indications for Craniotomy

In the management of cerebellar ICH and infarction, the indications for operative intervention remain the supreme controversy. Some authors invoke a size threshold, typically 3 or 4 cm, above which they recommend surgical evacuation of the hemorrhage regardless of clinical status. Others use the criteria of radiographic evidence of brainstem compression or cisternal effacement, which accounts for surrounding edema in addition to the size of the ICH or infarct in determining overall mass effect. For instance, Taneda et al.<sup>[28]</sup> reported 75 cases of spontaneous cerebellar ICH and classified the appearance of the quadrigeminal cistern into 3 groups: Grade I (normal), Grade II (compressed), and Grade III (absent). Good outcomes were reported in 88%, 69%, and 0% of Grade I, II, and III cases, respectively. However, they noted that the size of the hematoma was unrelated to the degree of cisternal compression, pointing out the influence of edema or hydrocephalus on overall mass effect. The predictive value of quadrigeminal cistern compression was confirmed in the series of van Loon et al.,<sup>[30]</sup> and patients with totally obliterated cisterns had poor outcomes regardless of treatment.

Still others discount these radiographic features and emphasize the neurological examination, including level of consciousness and brainstem reflexes, in determining criteria for surgery. Kobayashi et al.<sup>[18]</sup> performed a retrospective review of 52 patients with hypertensive cerebellar ICH. On the basis of this analysis, they proposed new criteria for intervention that were prospectively applied to the next 49 patients for validation and confirmation. Patients with GCS scores of 14 or 15 and with hematoma sizes less than 4 cm in maximal diameter were treated conservatively, while patients with GCS scores of 13 or less at admission or with a hematoma measuring greater than 4 cm underwent surgical evacuation. For patients with flaccid tetraplegia and absent brainstem reflexes, intensive therapy was not rendered.

Kirollos et al.<sup>[17]</sup> developed a different protocol, based on compression of the fourth ventricle as a measure of mass effect, which they applied prospectively in the management of 50 consecutive patients with cerebellar ICH. The appearance of the fourth ventricle was divided into 3 groups: Grade I (normal size and configuration), Grade

II (partially compressed and shifted), and Grade III (completely obliterated). The ICH was evacuated for all patients with Grade III compression and for patients with Grade II compression when the GCS score deteriorated in the absence of untreated hydrocephalus. Patients with Grade I or II compression were initially treated using only ventricular drainage if they developed hydrocephalus and clinical deterioration. Stable Grade I and II patients were managed conservatively. Acute deterioration to comatose state occurred in 6 (43%) of the 14 patients with Grade III compression who were conscious at presentation; none of them experienced good outcomes. However, 15 (60%) of 25 patients with hematomas greater than 3 cm and Grade I or II compression did not require clot evacuation.

In summary, clinical considerations should complement radiographic appearance in the management algorithm, and decisions for surgical intervention should rarely be made on the basis of imaging findings alone.

### Timing of Surgical Intervention

Evidence consistently shows that postoperative outcomes generally correlate with preoperative status.<sup>[6,17]</sup> For instance, in the series of Ott et al.,<sup>[25]</sup> the mortality rate was 17% for patients who were conscious at the time of surgery and 75% for those who were unconscious. Similarly, in the series of Donauer et al.,<sup>[17]</sup> patients presenting with GCS scores less than 6 had a 60% mortality rate and Karnofsky Outcome Index total of 26, while those with GCS scores greater than 10 had only a 20% mortality rate and a Karnofsky Outcome Index total of 66. In the series of Kobayashi et al.,<sup>[18]</sup> of the 5 patients with GCS scores of 4, 3 died and 2 remained vegetative despite surgery. In the series of van Loon et al.,<sup>[30]</sup> patients with total obliteration of the quadrigeminal cistern had a poor outcome irrespective of treatment.

Furthermore, many patients who experience clinical deterioration improve significantly after surgery. Some have even been restored to functional capacity. On this basis, it is natural to question whether patients who remain dependent after surgery would have fared better if intervention had been performed earlier in their course.<sup>[12,19]</sup>

For these reasons, many recommend surgery prior to clinical deterioration.<sup>[12]</sup> Yoshida et al.<sup>[34]</sup> emphasize the importance of surgical therapy even for alert patients if the hematoma is larger than 3 cm to avoid delayed deterioration. Similarly, in the series of Kirolos et al.,<sup>[17]</sup> no patient with Grade III compression of the fourth ventricle who was conscious at the outset but then developed a GCS score less than 8 experienced a good outcome. These authors thus advocate aggressive early surgical evacuation of the hematoma for all Grade III patients, regardless of initial examination results, before deterioration occurs.

Conversely, Dammann et al.<sup>[6]</sup> reviewed their series of 57 patients who underwent surgical evacuation of spontaneous cerebellar ICH. The initial neurological condition proved to be highly predictive of outcome. Based on the excellent results in patients with good initial clinical condition who underwent surgery due to secondary deterioration, this group advises against the preventive evacuation of cerebellar hemorrhage. Similarly, in the German Austrian Cerebellar Infarction Study, 84 patients with massive cerebellar infarction were prospectively observed after assignment to 1 of 3 groups: craniotomy and evacuation, ventriculostomy, or medical therapy alone. Treatment was left to the discretion of the provider in this unrandomized trial. In a logistic regression model, there was found to be no benefit to performing surgery in patients who had not yet deteriorated to coma.<sup>[14]</sup>

Despite the focus on early evacuation of cerebellar ICH, even delayed removal might have benefit. Auer et al.<sup>[11]</sup> reported 2 patients with cerebellar hemorrhage and focal signs of a posterior fossa lesion. Because they had no impairment of consciousness, they were initially managed conservatively. Because their symptoms had not shown a tendency to improve, however, they underwent evacuation of the hematoma performed on Days 23 and 54, respectively. Both patients then recovered fully without neurological deficit.

### **What Constitutes Futility?**

As stated, evidence consistently shows that postoperative outcomes generally correlate with preoperative status. However, there are numerous anecdotal accounts of good postoperative outcome among comatose patients.<sup>[11,12]</sup> In some cases, even patients with fixed and dilated pupils or other absent brainstem reflexes have recovered.<sup>[32]</sup> In the series of Hornig et al.,<sup>[13]</sup> 38% of comatose patients achieved a good recovery (nondisabled at hospital discharge) after decompressive surgery. In the German-Austrian Cerebellar Infarction Study, half of all patients who deteriorated into coma and were treated with ventricular drainage or decompressive craniotomy experienced a meaningful recovery (modified Rankin score of 2 or less).<sup>[14]</sup> Similarly, Kobayashi et al.<sup>[18]</sup> reported 2 patients with ruptured cerebellar AVMs who had flaccid tetraplegia and apnea at admission. After emergency surgery, both recovered to enjoy "a useful life."<sup>[18]</sup>

Furthermore, pathoanatomical studies reveal surprisingly few structural changes due to brainstem compression in patients with fatal space-occupying cerebellar infarcts.<sup>[27]</sup> Similarly, there is no intrinsic damage to supratentorial telencephalic structures in cerebellar ICH and infarct, which suggests the possibility of full intellectual and cognitive recovery in some cases.<sup>[7,12]</sup> In light of these considerations, it is reasonable to question whether any patient's condition is "too poor" to forego surgical intervention and what constitutes futile treatment. From a practical standpoint, surgery might be considered, even if the situation appears "hopeless."

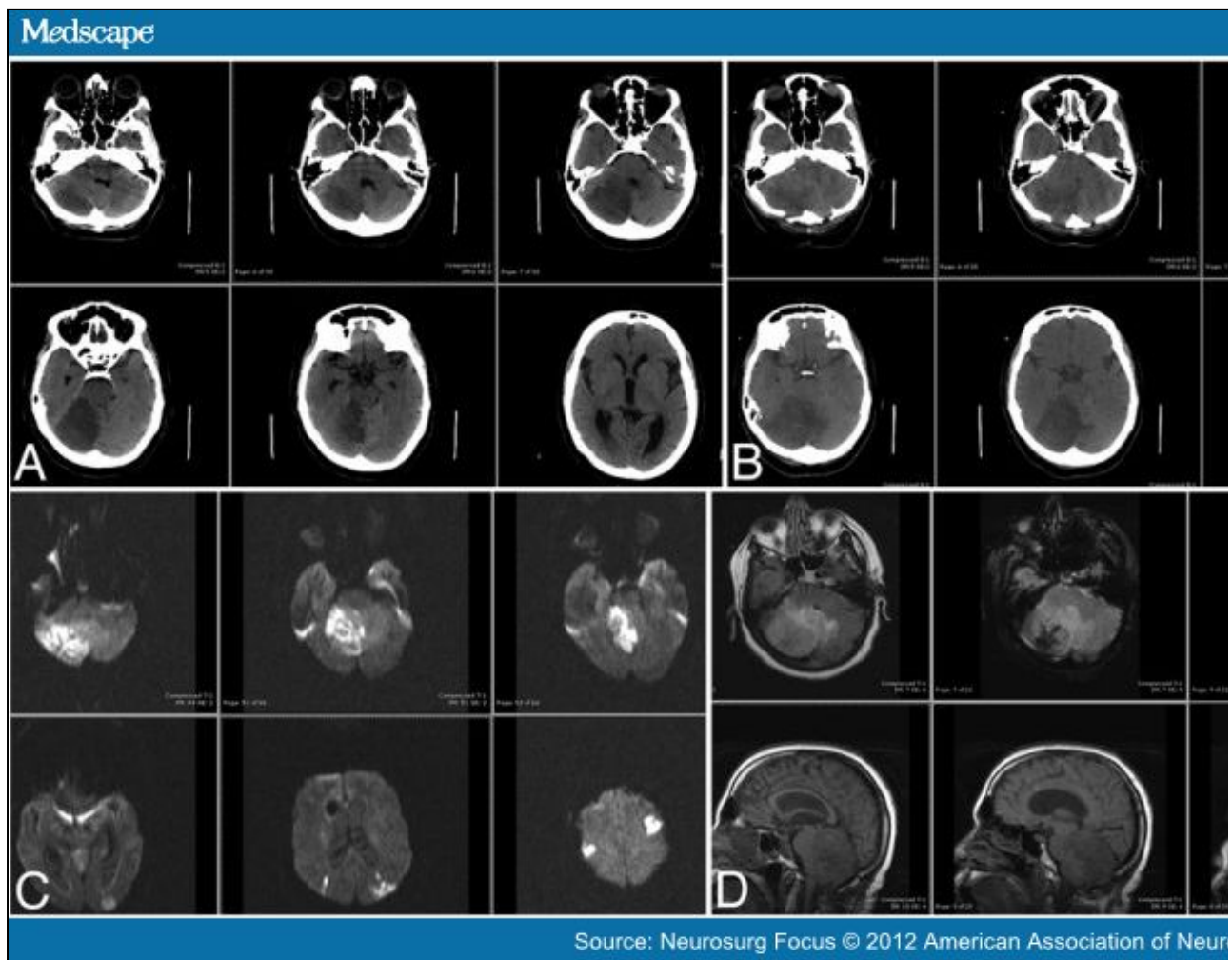
### **Value of Preoperative MRI**

Yanaka et al.<sup>[33]</sup> studied the prognostic value of postoperative MRI in 31 patients, all with GCS scores of 8 or less, who underwent surgical evacuation of cerebellar ICH. The patients were divided into 2 groups based on outcome. Good recovery or only moderate disability was achieved in 8 patients, while the remaining 23 died or became severely disabled/vegetative. There were no significant differences between the 2 groups in preoperative CT findings such as hematoma size, presence of hydrocephalus, fourth ventricular compression, or obliteration of the perimesencephalic cistern. However, the incidence of high signal intensity in the pons and midbrain on T2-weighted MRI, indicating brainstem damage, was significantly higher in the poor outcome group. These intriguing results raise the question of whether preoperative MRI can be used as a predictive tool to screen patients for brainstem injury, thus improving patient selection for aggressive therapy. However, no study has yet addressed this issue, possibly because of logistical impediments to performing MRI scans acutely in critically ill patients. The absence of brainstem injury, confirmed by preoperative MRI, might provide impetus for surgical intervention in patients who otherwise might have been considered "hopeless."

### **Technical Aspects of Surgery**

Numerous technical considerations in the operative management of cerebellar hemorrhage and infarction remain in the realm of individual preference. These include the size of the suboccipital bone removed and whether to fixate the bone flap (craniotomy) or

float it or abandon it (craniectomy) at the end of the procedure. Other adjuncts such as the removal of the arch of the first cervical vertebra remain optional. In the German Austrian Cerebellar Infarction Study, for instance, decompressive surgery consisted of a large craniotomy, duraplasty, and resection of the posterior atlas arch if tonsillar herniation was apparent, but resection of necrotic tissue was not mandatory.<sup>[14]</sup> In other series, however, craniectomy with resection of the infarcted tissue was applied, including possible resection of cerebellar tonsils.<sup>[13,25]</sup> One risk of too large a craniectomy is subsequent sagging of the cerebellar hemispheres. Conversely, a bone flap that is too small and then replaced may fail to achieve adequate decompression (Fig. 3). Because the degree of mass effect is different in each patient, intraoperative judgement must be exercised in determining the extent of bone removal necessary to achieve decompression, and no rigid guidelines can be offered about a prespecified size threshold.



**Figure 3.**

Images obtained in a 56-year-old woman with mitral regurgitation, who underwent cardiac surgery and developed decreased consciousness 1 day later. **A:** Axial CT scans of the brain without contrast demonstrate a large infarction of the right cerebellar hemisphere with mass effect and hydrocephalus. **B:** Due to progressive obtundation, the patient underwent suboccipital craniotomy and partial removal of infarcted tissue. The bone flap was replaced and left floating. These postoperative axial CT scans reveal persistent

mass effect. **C:** Postoperative diffusion-weighted axial MR images demonstrate persistent, widespread infarcted tissue and mass effect. **D:** Magnetic resonance imaging performed on postoperative Day 3. Axial FLAIR sequence (*upper left*) shows persistent edema and mass effect. Axial gradient echo sequence (*upper center and right*) shows hemorrhagic transformation. Sagittal T1-weighted images (*lower row*) show ascending transtentorial herniation and tonsillar herniation through the foramen magnum. The patient remained symptomatic for the next 2 weeks, suggesting that the bone decompression and/or removal of infarcted tissue was insufficient.

### Role of Other Interventions

In lieu of suboccipital craniectomy and evacuation of the hemorrhage, several other surgical approaches have been proposed for the management of cerebellar ICH, including stereotactic aspiration, endoscopic bur hole evacuation, and local infusion of a thrombolytic agent such as tissue plasminogen activator.<sup>[1]</sup> Data regarding the safety and efficacy of these procedures are lacking, and they are currently not considered mainstream therapy.

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## American Stroke Association Guidelines

Recognizing that the management of ICH by neurologists and neurosurgeons throughout the world varies greatly, the Stroke Council of the American Heart Association formed a task force to develop practice guidelines and to suggest areas where further research was needed. In 1999, the first guidelines were published, although the authors acknowledged that the strength of their recommendations was limited by the quality of the medical literature, which consists more of anecdotal case series than well-designed clinical trials.<sup>[4]</sup> Since then, the guidelines have undergone updates in 2007 and 2010.<sup>[3,24]</sup> Besides the evolution of the position statements, these updates reclassified the level of certainty of the treatment effect and recategorized the class of evidence from which they are derived.

Regarding the indications for vascular imaging to search for an underlying structural cause of the ICH, the 1999 Council wrote, "Angiography should be considered for all patients without a clear cause of hemorrhage who are surgical candidates, particularly young, normotensive patients who are clinically stable (level of evidence V, grade C recommendation)." <sup>[4]</sup> Additionally, they wrote, "Angiography is not required for older hypertensive patients who have a hemorrhage in the basal ganglia, thalamus, cerebellum, or brain stem and in whom CT findings do not suggest a structural lesion ....(level of evidence V, grade C recommendation)." <sup>4</sup> Under the definitions in effect at that time, these were the weakest possible recommendations and based on the lowest quality data. In 2010, the guidelines state that, "...CT angiography, CT venography, contrast-enhanced CT, contrast-enhanced MRI, magnetic resonance angiography, and magnetic resonance venography can be useful to evaluate for underlying structural lesions, including vascular malformations and tumors when there is clinical or radiological suspicion ( *Class IIa; Level of Evidence: B*)." <sup>[24]</sup>

Regarding the indications for surgical removal of ICH, the 1999 council wrote: <sup>[4]</sup>

Patients with cerebellar hemorrhage > 3 cm who are neurologically deteriorating or who have brain stem compression and hydrocephalus from ventricular obstruction should have surgical removal of the hemorrhage as soon as possible (levels of evidence III through V, grade C recommendation)... Stereotactic aspiration may be associated with better outcomes than standard craniotomy for moderate-sized

cerebellar hemorrhages, but this hypothesis has yet to be tested in a randomized study (no recommendation).

In 2007, there was no change to this recommendation, although the authors revised the categorization of its strength as "Class 1, Level of Evidence B," which is an intermediate grade.<sup>[3]</sup> In 2010, the qualification of the 3-cm-size threshold was abandoned, and a new recommendation concerning ventricular drainage was offered:<sup>[24]</sup>

Patients with cerebellar hemorrhage who are deteriorating neurologically or who have brainstem compression and/or hydrocephalus from ventricular obstruction should undergo surgical removal of the hemorrhage as soon as possible ( *Class 1; Level of Evidence: B*). (Revised from the previous guideline). Initial treatment of these patients with ventricular drainage alone rather than surgical evacuation is not recommended ( *Class III; Level of Evidence: C*). (New recommendation).

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## Conclusions

The management of cerebellar hemorrhagic and ischemic stroke is controversial. Issues such as the difference in the treatment algorithm of cerebellar ICH versus infarction, criteria for imaging to exclude an underlying structural lesion, the value of MRI for patient selection, the role of external ventricular drainage, the indications for operative management, the timing of surgical intervention, and various options of surgical technique remain unresolved. Professional society guidelines for these considerations are sparse and based on relatively poor quality data. Nonetheless, the potential value of neurosurgical intervention remains well established.

## References

1. Auer LM, Auer T, Sayama I: Indications for surgical treatment of cerebellar haemorrhage and infarction. *Acta Neurochir (Wien)* 79:74–79, 1986
2. Ballance HA: Case of traumatic haemorrhage into the left lateral lobe of the cerebellum, treated by operation, with recovery. *Surg Gynecol Obstet* 3:223–225, 1906
3. Broderick J, Connolly S, Feldmann E, Hanley D, Kase C, Krieger D, et al: Guidelines for the management of spontaneous intracerebral hemorrhage in adults: 2007 update: a guideline from the American Heart Association/American Stroke Association Stroke Council, High Blood Pressure Research Council, and the Quality of Care and Outcomes in Research Interdisciplinary Working Group. *Circulation* 116:e391–e413, 2007
4. Broderick JP, Adams HP Jr, Barsan W, Feinberg W, Feldmann E, Grotta J, et al: Guidelines for the management of spontaneous intracerebral hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. *Stroke* 30:905–915, 1999
5. Chen HJ, Lee TC, Wei CP: Treatment of cerebellar infarction by decompressive suboccipital craniectomy. *Stroke* 23:957– 961, 1992
6. Dammann P, Asgari S, Bassiouni H, Gasser T, Panagiotopoulos V, Gizewski ER, et al: Spontaneous cerebellar hemorrhage— experience with 57 surgically treated patients and review of the literature. *Neurosurg Rev* 34:77–86, 2011

7. Donauer E, Loew F, Faubert C, Alesch F, Schaan M: Prognostic factors in the treatment of cerebellar haemorrhage. *Acta Neurochir (Wien)* 131:59–66, 1994
8. Fairburn B, Oliver LC: Cerebellar softening; a surgical emergency. *BMJ* 1:1335–1336, 1956
9. Halpin SF, Britton JA, Byrne JV, Clifton A, Hart G, Moore A: Prospective evaluation of cerebral angiography and computed tomography in cerebral haematoma. *J Neurol Neurosurg Psychiatry* 57:1180–1186, 1994
10. Hankey GJ, Hon C: Surgery for primary intracerebral hemorrhage: is it safe and effective? A systematic review of case series and randomized trials. *Stroke* 28:2126–2132, 1997
11. Heros RC: Cerebellar hemorrhage and infarction. *Stroke* 13: 106–109, 1982
12. Heros RC: Surgical treatment of cerebellar infarction. *Stroke* 23:937–938, 1992
13. Hornig CR, Rust DS, Busse O, Jauss M, Laun A: Space-occupying cerebellar infarction. Clinical course and prognosis. *Stroke* 25:372–374, 1994
14. Jauss M, Krieger D, Hornig C, Schramm J, Busse O: Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study. *J Neurol* 246:257–264, 1999
15. Kanno T, Sano H, Shinomiya Y, Katada K, Nagata J, Hoshino M, et al: Role of surgery in hypertensive intracerebral hematoma. A comparative study of 305 nonsurgical and 154 surgical cases. *J Neurosurg* 61:1091–1099, 1984
16. Kidwell CS, Alger JR, Saver JL: Beyond mismatch: evolving paradigms in imaging the ischemic penumbra with multimodal magnetic resonance imaging. *Stroke* 34:2729–2735, 2003
17. Kirollos RW, Tyagi AK, Ross SA, van Hille PT, Marks PV: Management of spontaneous cerebellar hematomas: a prospective treatment protocol. *Neurosurgery* 49:1378–1387, 2001
18. Kobayashi S, Sato A, Kageyama Y, Nakamura H, Watanabe Y, Yamaura A: Treatment of hypertensive cerebellar hemorrhage— surgical or conservative management? *Neurosurgery* 34:246–251, 1994
19. Koh MS, Goh KY, Tung MY, Chan C: Is decompressive craniectomy for acute cerebral infarction of any benefit? *Surg Neurol* 53:225–230, 2000
20. Lindgren SO: Infarctions simulating brain tumours in the posterior fossa. *J Neurosurg* 13:575–581, 1956
21. Mathew P, Teasdale G, Bannan A, Oluoch-Olunya D: Neurosurgical management of cerebellar haematoma and infarct. *J Neurol Neurosurg Psychiatry* 59:287–292, 1995
22. McKissock W, Richardson A, Walsh L: Spontaneous cerebellar haemorrhage: a study of 34 consecutive cases treated surgically. *Brain* 83:1–9, 1960
23. Mendelow AD, Gregson BA, Fernandes HM, Murray GD, Teasdale GM, Hope DT, et

al: Early surgery versus initial conservative treatment in patients with spontaneous supratentorial intracerebral haematomas in the International Surgical Trial in Intracerebral Haemorrhage (STICH): a randomised trial. *Lancet* 365:387–397, 2005

24. Morgenstern LB, Hemphill JC III, Anderson C, Becker K, Broderick JP, Connolly ES Jr, et al: Guidelines for the management of spontaneous intracerebral hemorrhage: a guide-line for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 41:2108– 2129, 2010
25. Ott KH, Kase CS, Ojemann RG, Mohr JP: Cerebellar hemorrhage: diagnosis and treatment. A review of 56 cases. *Arch Neurol* 31:160–167, 1974
26. Raco A, Caroli E, Isidori A, Salvati M: Management of acute cerebellar infarction: one institution's experience. *Neurosurgery* 53:1061–1066, 2003
27. Sybert GW, Alvord EC Jr: Cerebellar infarction. A clinicopathological study. *Arch Neurol* 32:357–363, 1975
28. Taneda M, Hayakawa T, Mogami H: Primary cerebellar hemorrhage. Quadrigeminal cistern obliteration on CT scans as a predictor of outcome. *J Neurosurg* 67:545–552, 1987
29. Tulyapronchote R, Malkoff MD, Selhorst JB, Gomez CR: Treatment of cerebellar infarction by decompression suboccipital craniectomy. *Stroke* 24:478–480, 1993 (Letter)
30. van Loon J, Van Calenbergh F, Goffin J, Plets C: Controversies in the management of spontaneous cerebellar haemorrhage. A consecutive series of 49 cases and review of the literature. *Acta Neurochir (Wien)* 122:187–193, 1993
31. Wakai S, Nagai M: Histological verification of microaneurysms as a cause of cerebral haemorrhage in surgical specimens. *J Neurol Neurosurg Psychiatry* 52:595–599, 1989
32. Yanaka K, Meguro K, Fujita K, Narushima K, Nose T: Immediate surgery reduces mortality in deeply comatose patients with spontaneous cerebellar hemorrhage. *Neurol Med Chir (Tokyo)* 40:295–300, 2000
33. Yanaka K, Meguro K, Fujita K, Narushima K, Nose T: Postoperative brainstem high intensity is correlated with poor outcomes for patients with spontaneous cerebellar hemorrhage. *Neurosurgery* 45:1323–1328, 1999
34. Yoshida S, Sasaki M, Oka H, Gotoh O, Yamada R: [Hypertensive cerebellar hemorrhage with signs of lower brainstem compression—a case report (author's transl).] *No Shinkei Geka* 6:379–383, 1978 (Jpn)
35. Zhu XL, Chan MS, Poon WS: Spontaneous intracranial hemorrhage: which patients need diagnostic cerebral angiography? A prospective study of 206 cases and review of the literature. *Stroke* 28:1406–1409, 1997

### Abbreviations used in this paper

AVM = arteriovenous malformation; GCS = Glasgow Coma Scale; ICH = intracerebral

hemorrhage.

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