Acute cerebellar infarction or hemorrhage may initially manifest in a clinically indolent manner only to later deteriorate into a life-threatening neurologic catastrophe. At the other end of the spectrum, some patients with cerebellar stroke may present in a moribund comatose state. In both patient groups, it is often unclear at what point surgical intervention should be considered either to prevent further neurologic deterioration or to try to salvage a meaningful neurologic recovery. In this review, we present clinical cases that illustrate decision points in the management of patients with acute cerebellar stroke, with emphasis on clinical and imaging characteristics. We conclude with an analysis of clinical decision making in the management of patients with space-occupying cerebellar stroke. The management of acute cerebellar infarction or hemorrhage often requires difficult and prompt decisions by treating neurologists, and certain easily identifiable clinical and imaging findings may assist in appropriate patient triage and timely neurosurgical intervention.

Acute cerebellar infarction and hemorrhage require constant vigilance by physicians owing to often unpredictable clinical behavior. Cerebellar stroke may manifest with ataxia, vertigo, dysarthria, nausea, vomiting, and often a prominent headache. Rapid deterioration in these patients is particularly vexing because of the difficulty in mobilizing neurosurgical intervention and the uncertainty regarding the mechanism of deterioration. It is also difficult to determine whether ensuing coma results from potentially reversible causes, such as evolving hydrocephalus, brainstem compression by mass effect, or irreversible brainstem infarction.

Data to aid clinical decision making are limited and largely focus on the final patient prognosis rather than on prediction of neurologic deterioration or the effect of intervention on outcome. A few recent large retrospective and prospective case series have attempted to address these issues, and they are reviewed herein in the context of illustrative cases. In this article, we present actual patient cases that show the clinical spectrum of cerebellar stroke, ranging from clinically stable patients to moribund patients who are comatose at presentation. The effects of clinical signs and imaging results on practical management are reviewed, and conclusions based on a review of the literature for guiding decision making are presented.

PREDICTING NEUROLOGIC DETERIORATION IN INITIALLY STABLE PATIENTS

Clinical Case 1: Cerebellar Infarction

A 47-year-old man with hypertension, hyperlipidemia, and diabetes mellitus developed vertigo, nausea, and vomiting. This continued to a mild extent and then worsened 10 days later, with a brief episode of right hemi-numbness, right ear tinnitus, poor balance, and incoordination of the right hand that persisted. Magnetic resonance imaging showed bilateral cerebellar infarcts, right larger than left (Figure 1). Magnetic resonance angiography showed
a small left vertebral artery and a high-grade stenosis in the right vertebral artery before the origin of the posterior inferior cerebellar artery. The patient was admitted to the hospital and was observed closely but had no clinical change. His symptoms resolved, except for clumsiness of the right hand, which gradually improved with physical therapy. He was given antiplatelet agents, and he achieved better risk factor control and was scheduled for a conventional angiogram with the possibility of endovascular intervention of his stenosis.

This case illustrates the value of serial clinical follow-up, which is best performed in a neurologic intensive care unit. Patients with cerebellar stroke should ideally be admitted to a neurologic intensive care unit for 72 to 96 hours after first being seen to permit continuous neuroscience nursing staff observation and frequent physician reexamination. If the patient remains stable through the first few days, when infarct swelling is expected, further deterioration from cerebellar infarction is unlikely. A similar forecast is not always possible with cerebellar hemorrhage; the literature is replete with cases of neurologic deterioration days and even weeks after the clinical ictus of symptom onset.1-4 However, most patients with a cerebellar hematoma who remain stable during the first 5 hospital days are unlikely to deteriorate, and they may be safely transferred to a ward setting with continued close observation.

**Clinical Case 2: Cerebellar Hemorrhage**

An 81-year-old woman with a history of hypertension, diabetes mellitus, hyperlipidemia, myocardial infarction, ischemic stroke, and atrial fibrillation treated with warfarin sodium developed nausea and vomiting, followed by vertigo, left-handed clumsiness, and difficulty walking. Head computed tomography (HCT) showed a 1- to 2-cm hematoma in the cerebellum just lateral to the vermis, with a small amount of intraventricular extension (Figure 2).
Her anticoagulation was reversed. On transfer to the University of Iowa Hospitals and Clinics, she was fully alert (Glasgow Coma Scale [GCS] score of 15), and the examination findings were normal except for mild gait, left arm, and left leg ataxia and diminished sensation in a bilateral stocking distribution, with loss of ankle jerks. The next day the patient became progressively drowsy, with a new HCT showing hydrocephalus; ventriculostomy was performed. She returned to full alertness, and during the next few weeks her ataxia improved. She was discharged from the hospital in good condition.

Most data generated on cerebellar stroke have attempted to predict outcome. However, on hospital admission, physicians are most concerned about the potential for a patient’s condition to deteriorate. In a large retrospective study regarding the prediction of neurologic deterioration in cerebellar hemorrhage, 46% of initially alert patients (GCS score of 13-15) experienced deterioration, with a decreasing level of consciousness, evolution of new brainstem signs, or worsened motor response on the GCS. Statistically significant clinical and imaging features that predicted neurologic deterioration included a hospital admission systolic blood pressure greater than 200 mm Hg, pinpoint pupils, abnormal corneal or oculocephalic reflexes, extension of hemorrhage into the cerebellar vermis, a hematoma of 3 cm or greater, visible brainstem distortion on CT, the presence of intraventricular hemorrhage, upward herniation, and acute hydrocephalus. Multivariate analysis demonstrated that hemorrhage in the vermis and acute hydrocephalus independently predicted deterioration. Our patient had a small hemorrhage; however, it involved the vermis, with extension into the fourth ventricle.

The main differential diagnostic possibilities with deteriorating cerebellar stroke are brainstem compression by direct mass effect, evolution of obstructive hydrocephalus from compression of the fourth ventricle, and occlusion of the cerebral aqueduct as a secondary phenomenon accompanying upward herniation of vermian cerebellar tissue through the tentorial notch.

The existing literature regarding the best approach to such deteriorating cerebellar stroke is relatively sparse and inconclusive. When the primary mechanism of neurologic deterioration seems to result from direct brainstem compression, the literature suggests that definitive surgical management requires a suboccipital craniectomy with evacuation of the hematoma or resection of infarcted cerebellar tissue to relieve the directly offending mass effect on the brainstem. In addition, when deterioration seems to result from obstructive hydrocephalus alone and there is no obvious evidence of direct brainstem compression by clinical examination or imaging findings, it remains unclear whether ventriculostomy alone may be adequate for the prevention of further deterioration or whether definitive resection should still be performed to relieve ventricular obstruction and brainstem compression. Some experts have advocated a staged approach, in which patients with deteriorating levels of consciousness, gaze palsy, or a herniation syndrome have emergency HCT to determine whether the mechanism seems to primarily be evolving obstructive hydrocephalus, direct brainstem compression, or upward herniation.

If hydrocephalus seems to be the main mechanism responsible for deterioration, a temporizing ventriculostomy can be considered. If the patient improves, no further surgical therapy may be needed. If the patient does not improve, then a definitive posterior fossa decompressive suboccipital craniectomy should be urgently undertaken. Because craniotomy carries an attendant el-

Figure 2. Head computed tomographic scan showing vermis involvement and minimal hemorrhage into the fourth ventricle, with little mass effect.
When the patient’s clinical course of deterioration is relatively slow for several hours, a strategy of staged procedures with initial ventriculostomy followed by craniectomy if necessary is reasonable. However, with more catastrophic and rapidly evolving neurologic deterioration, for less than an arbitrary 8-hour limit, and when the risk of craniectomy is relatively low, such as in younger individuals, then primary craniectomy should probably be the treatment of choice.

**Clinical Case 3:**
**Cerebellar Infarction**

A 48-year-old aerobics instructor, on traversing a step to the right while teaching a class, developed abrupt vertigo and continued walking to the right until she reached the wall, which she leaned on for support. Her symptoms subsided, and she finished the class. That night she vomited twice. For the next 2 days she had a severe headache, mild vertigo, and a tendency to veer to the right while walking. She was seen for the continued headache. Her neurologic examination findings were normal, except for falling to the right with tandem gait. After CT, the patient underwent magnetic resonance imaging, which showed an infarct of the medial left lobe of the cerebellum (Figure 3). Magnetic resonance angiography showed occlusion of the left vertebral artery, with otherwise normal cerebrovasculature. Her headache improved, and she was discharged from the hospital.

This case demonstrates the potential discrepancy between the size of a cerebellar infarct on imaging and the severity of the clinical course. The extent and location of the infarct made the possibility of hydrocephalus from obstruction of the fourth ventricle concerning enough to warrant hospital admission for close observation in addition to evaluation for the etiology of her stroke. As it turned out, had she tolerated her headache for 1 more day, it is likely that no one would have ever known about the stroke. Many cases of mild cerebellar infarction probably never reach medical attention.7

**Clinical Case 4:**
**Subdural Hemorrhage in the Posterior Fossa**

A 57-year-old woman with mechanical aortic and mitral valves was admitted to the hospital for ischemic colitis. She underwent hemicolecction, before which warfarin sodium therapy was changed to heparin therapy. After a week of uneventful recovery she became progressively drowsy in a 24-hour period to the point of obtundation. Head CT showed bilateral subdural hematomas in the posterior fossa, with mass effect obliterating the fourth ventricle and basal cisterns, and hydrocephalus (Figure 4). Anticoagulant therapy was stopped. Posterior fossa craniectomy with evacuation of the hematoma and ventriculostomy were performed (Figure 5). The patient returned to full alertness and did well neurologically, although she developed multiple medical complications that kept her in the intensive care unit.

This is an unusual case of a vascular event causing acute mass effect in the posterior fossa. It demonstrates many of the same clinical and imaging features as cerebellar stroke. The small volume of the posterior fossa allows...
even less room for a subdural hematoma than the supratentorial compartment. The ominous features of this case were progressive deterioration in the level of alertness, obliteration of the fourth ventricle and basal cisterns, and acute hydrocephalus. Surgical evacuation and ventricular drainage prevented a likely fatal event in this patient.

**CLINICALLY MORIBUND OR COMATOSE PATIENTS AT INITIAL EXAMINATION**

The following cases demonstrate the opposite end of the clinical spectrum, when patients with a cerebellar stroke are in a comatose state on initial examination. The overriding issue in such patients is determining neurologic salvageability and avoiding futile surgical intervention.

**Clinical Case 5: Cerebellar Hemorrhage**

A 56-year-old man began to feel unwell at a fair and was taken to a local emergency department. During the course of several hours he became comatose and was intubated. A noncontrast HCT showed a 5-cm hematoma in the right cerebellar hemisphere, with extension into the vermis and the fourth ventricle, acute hydrocephalus with blood present in all 4 ventricles, and effacement of the basal cisterns (Figure 6). He was given mannitol and was transferred to the University of Iowa Hospitals and Clinics. On initial examination, the patient was comatose (GCS score of 4) and intubated; his pupils were 3 mm and nonreactive, and corneal reflexes were absent. He had spontaneous cough. There was no motor response except decerebrate posturing of the right arm. A right frontal ventriculostomy and a suboccipital craniotomy with posterior fossa clot evacuation were performed. After surgery, there was lability of blood pressure and heart rate and increasing pupillary size without reaction. A new HCT showed a large fourth ventricle full of blood and upward transtentorial herniation (Figure 7). He was taken back to the operating room for reevacuation and decompression. After the second operation there was no cough, gag, or brainstem reflexes or motor responses. The family decided to withdraw life support, and the patient died.

**Clinical Case 6: Cerebellar Hemorrhage**

A 66-year-old man experienced the worst headache of his life and became unresponsive within hours. He was intubated and then defibrillated and resuscitated after ventricular tachycardia. An HCT showed a 4-cm hematoma in the left cerebellar hemisphere, with compression of the fourth ventricle, blood in the third and lateral ventricles, perihematoma hypodensity, a suggestion of upward herniation, and obliteration of the basal cisterns (Figure 8). His GCS score was 3, and his blood pressure was 67/50 mm Hg. He was administered vasopressors and was transferred to the University of Iowa Hospitals and Clinics. On arrival, the patient was comatose and intubated, with pupils mid-position and fixed and bilateral vitreous hemorrhages. There were no corneal, gag, oculocephalic, or motor responses. There were no spontaneous respirations. His family decided to withdraw life support, and the patient died.

**CASE DISCUSSION**

These cases are included in our discussion of space-occupying cerebellar stroke to point out that not all patients with deterioration have a reversible mechanism. Taneda et al described 3 patient groups with acute cerebellar stroke causing coma that vary by their clinical presentation, course, and neuroradiologic appearance:

1. Alert at onset, with paroxysmal cerebellar symptoms and signs and subsequent stabilization for 10 to 36 hours, and then rapid and progressive deterioration of consciousness in 2 to 18 hours. This group was usually found to have a syndrome of progressive direct brainstem compression by the expanding hematoma or edema.
2. Sudden cerebellar symptoms with rapid deterioration of consciousness, representing hemorrhage into a cerebellar infarct. These patients present with a cerebellar hematoma, with a subsequent diagnosis of ischemic infarction as the pathogenic mechanism made only at the time of surgery or autopsy.
3. Sudden unresponsiveness and coma within hours of symptom onset, without substantial further deterioration. This was found to almost universally result from extensive concurrent brainstem infarction at the time of cerebellar hemorrhage or infarction, as was likely seen in our case 6 described previously. Amarenco also found that in patients with a hemiplegia or a tetraplegia, often a massive comorbid paramedian pontine infarction rather
Figure 6. A noncontrast head computed tomographic scan showing a large hematoma with vermis involvement, blood in all of the ventricles, hydrocephalus, and loss of the prepontine and quadrigeminal cisterns.

Figure 7. Despite hematoma evacuation, another computed tomographic scan of the patient in Figure 6 shows the persistence of basal cistern effacement, hydrocephalus, and upward herniation.

Figure 8. A head computed tomographic scan showing a large hematoma that obliterates the fourth ventricle, with loss of the basal cisterns, hydrocephalus, and upward herniation.
than brainstem compression was responsible for their deterioration.

Several retrospective studies\textsuperscript{3,10-14} have analyzed clinical and imaging features that are predictive of poor outcome in cerebellar hemorrhage. Clinical features on hospital admission in patients with cerebellar hemorrhage previously correlated with a poor outcome have included systolic blood pressure greater than 200 mm Hg (presumed to be part of the Cushing response),\textsuperscript{4} gaze paresis,\textsuperscript{9,13,30} and a decreased level of consciousness,\textsuperscript{1,12,13-18} which are all indications of compression at the pontine level. The CT scan features indicative of a poor prognosis are a midline location,\textsuperscript{13,15,19-21} an obliterated fourth ventricle and basal cisterns,\textsuperscript{1,13,19,22} upward herniation,\textsuperscript{8,23} intraventricular hemorrhage,\textsuperscript{24} and hydrocephalus.\textsuperscript{2,4,18,23} A widely accepted neurosurgical decision making. One retrospective study\textsuperscript{25} of patient outcomes in cerebellar hemorrhage identified clinical and imaging features predictive of poor outcome. Predictors of disability included a hospital admission systolic blood pressure greater than 200 mm Hg, a hematoma size greater than 3 cm in diameter, visible brainstem distortion, and acute hydrocephalus on hospital admission CT. Significant predictors of death were abnormal corneal and oculocephalic reflexes, a GCS score less than 8, motor response less than localization to pain, acute hydrocephalus, and intraventricular hemorrhage on CT. An absent corneal reflex on hospital admission was found to be highly predictive of poor outcome. The best outcomes were in patients younger than 70 years with normal corneal reflexes. After adjustment for confounding factors, coma on hospital admission (GCS score <8) and intraventricular hemorrhage were independently predictive of death. Although retrospective data are subject to bias, the clinical and imaging features identified may help curtail the generation of unrealistic hope and may affect discussions regarding prognosis and intervention with family members.

**SUGGESTIONS FOR CLINICAL DECISION MAKING IN PATIENTS WITH CEREBELLAR STROKE**

These cases illustrate the range of clinical presentations and courses in patients with cerebellar stroke. There are many retrospective studies and a few prospective observational trials that are helpful in guiding clinical decision making. In the absence of data from a randomized controlled trial, a reasonable framework for patient care has been suggested by Kirollos et al.,\textsuperscript{24} who treated 50 consecutive patients using a protocol based on the appearance of the fourth ventricle and the GCS score. If the fourth ventricle was completely effaced, the patient underwent surgical evacuation and ventricular drainage. If the fourth ventricle appeared normal, the patient was treated conservatively, unless the GCS score deteriorated, in which case the patient underwent ventricular drainage. If the fourth ventricle was compressed (but not completely effaced), the patient was treated conservatively if fully alert, shunted if the GCS score deteriorated and hydrocephalus was present, or evacuated if there was no hydrocephalus (or if the GCS score did not improve with shunting). Mortality at 3 months was 40% (a sizeable proportion from medical issues). Of the survivors, 80% had a good outcome (independent in activities of daily living, with a Glasgow Outcome Scale score of 4 or 5). None of the patients with a completely effaced fourth ventricle and coma (GCS score <8) survived with a good outcome. In this analysis by Kirollos et al.,\textsuperscript{24} outcome correlated best with fourth ventricular grade (P < .002) and preoperative GCS score (P < .003). Their data suggested that patients did better if they underwent surgery before deterioration.

**CONCLUSIONS**

The clinical and imaging features of patients with cerebellar stroke can be helpful in clinical decision making. No single clinical or imaging finding can be used in isolation when deciding whether to use aggressive surgical management or to withhold such therapy; the overall clinical gestalt of the patient with cerebellar stroke remains paramount.

During the patient’s first week in the hospital, observation in a neurologic intensive care unit may facilitate the timely recognition of neurologic deterioration and permit immediate repeated imaging to guide an appropriate and tailored surgical approach. Ventriculostomy may be an adequate temporizing measure in the patient with deterioration predominantly resulting from obstructive hydrocephalus, whereas definitive treatment of progressive brainstem compression most often requires craniectomy for decompression of the posterior fossa.

Patients with declining levels of consciousness, new brainstem signs (particularly loss of corneal reflexes), evolving hydrocephalus, and midline cerebellar stroke are at increased risk for deterioration and poor outcome. Whether prophylactic ventriculostomy or decompression prevents neurologic deterioration is not clear, and this issue should be addressed in a randomized controlled trial; perhaps diffusion-perfusion magnetic resonance imaging could contribute to a better understanding of the importance of infarct volume or ischemic penumbra on the risk of subsequent deterioration and poor outcome.

**Accepted for Publication:** April 20, 2004.

**Correspondence:** Erik K. St Louis, MD, Department of Neurology, University of Iowa Hospitals and Clinics, 200 Hawkins Dr, Iowa City, IA 52242 (erik-stlouis@uiowa.edu).

**Author Contributions:** Study concept and design: Jensen and St Louis. Acquisition of data: Jensen and St Louis. Analysis and interpretation of data: Jensen and St Louis. Drafting of the manuscript: Jensen and St Louis. Critical revision of the manuscript for important intellectual content: Jensen and St Louis. Administrative, technical, and material support: Jensen. Study supervision: St Louis.