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Posterior Fossa Hemorrhage after Supratentorial Surgery

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PURPOSE: To evaluate the imaging findings, possible pathogenesis, and frequency of hemorrhage near the tentorial surface of the cerebellum after supratentorial surgery. **METHODS:** Over a 5-year period, 13 patients with posterior fossa hemorrhage after supratentorial surgery were identified with postoperative CT studies, which were obtained in all patients after craniotomy. Four of the 13 patients also had postoperative MR examinations. Preoperative and postoperative radiologic studies and medical records were reviewed. **RESULTS:** All hemorrhages were located along the superior aspect of the cerebellum, transversely oriented along the cerebellar folia and fissures. None of the patients had coagulopathy or radiologic evidence of posterior fossa tumor or vascular malformation. None of the hemorrhages had an appearance typical of hypertensive hemorrhage. Ten patients (77%) had lumbar drains placed before surgery and all had epidural drains placed at the conclusion of surgery. One patient had surgery for a meningioma, and 12 had surgery for aneurysms. The frequency of hemorrhage after aneurysmal surgery was 3.5%. No neurologic deficits developed referable to the cerebellum. **CONCLUSION:** Hemorrhage should be viewed as a potentially benign sequela of surgery rather than as an indication of hypertension, aneurysm, or previously unrecognized vascular malformation or neoplasm.

Index terms: Cerebral hemorrhage; Brain, surgery; Iatrogenic disease or disorder

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Intracranial hemorrhage at a location remote from the surgical site is a known sequela of intracranial surgery. This includes supratentorial intracerebral hemorrhage complicating posterior fossa surgery (1–7), hemorrhage occurring in the hemisphere contralateral to the surgical site (5, 7–9), posterior fossa hemorrhage complicating supratentorial surgery (7, 10–17), cerebellar hemorrhage complicating cervical spine surgery (18), and subdural (19) and epidural (20) hematomas remote from sites of intracranial surgery. Our purpose was to determine the characteristic imaging findings in a series of 13 patients with hemorrhage near the

tentorial surface of the cerebellum after supratentorial surgery and to ascertain the possible pathogenesis of these hemorrhages.

Materials and Methods

Routinely, all patients undergoing craniotomy at our institution have computed tomography (CT) of the head within 24 hours of surgery. By means of a computerized search of our radiologic database, we identified 13 patients who had postoperative posterior fossa hemorrhage after supratentorial surgery performed between January 1, 1991, and January 1, 1996. Twelve patients had surgery for saccular aneurysms, 11 in the anterior circulation and one at the basilar tip, and one patient had surgery for a sphenoidal ridge meningioma. Six patients were men and seven were women; ages ranged from 40 to 72 years (mean, 59 years).

Aneurysmal surgery was performed in 394 patients at our institution between January 1, 1991, and January 1, 1996. Seventy-seven patients had aneurysms of the posterior circulation and 317 had aneurysms of the anterior circulation. All aneurysms of the anterior circulation were treated via a supratentorial pterional approach. The majority of posterior circulation aneurysms were at the basilar tip and were also managed from a supratentorial approach, but the exact number of these procedures is not known. The number of supratentorial operations per-

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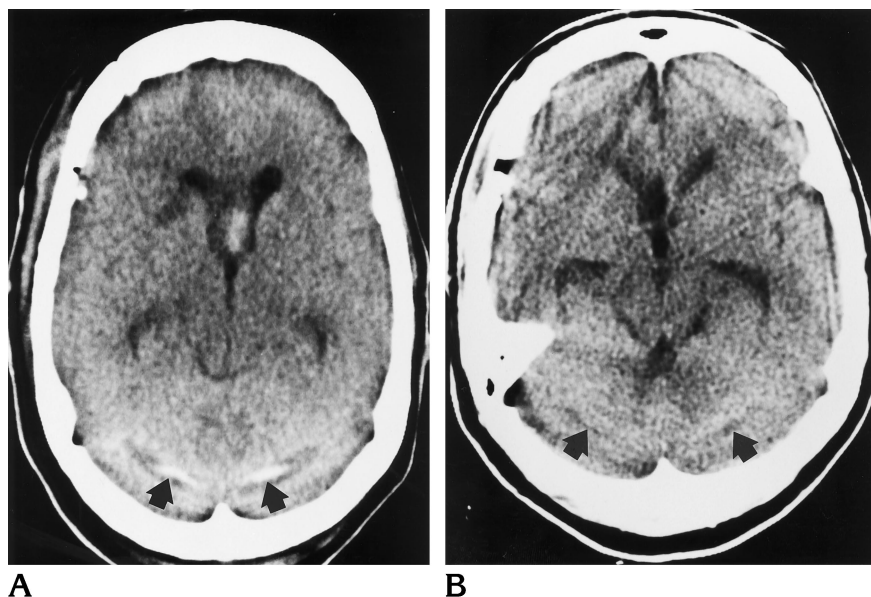
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Fig 1. A 40-year-old woman with ruptured anterior communicating artery aneurysm.

A, Noncontrast CT scan the day after surgery shows hemorrhage (arrows) spread along a cerebellar fissure.

C, Follow-up CT scan 19 days after surgery shows low-attenuation material within a cerebellar fissure (arrows) in which high-attenuation blood was seen previously.



formed for other reasons is also not known, but certainly exceeds the number of operations performed for aneurysms.

All patients were examined before surgery for coagulopathy with prothrombin time, partial thromboplastin time, and platelet count. All patients with cerebral aneurysms had preoperative cerebral angiography and CT of the head. Preoperative imaging in the patient with a meningioma consisted of magnetic resonance (MR) imaging of the brain. All patients underwent a pterional craniotomy and were operated on in the supine position. All patients had their head turned 45° to 70° away from the side of the craniotomy. Blood pressure was monitored before, during, and after surgery. None of the patients had a jugular venous catheter placed.

A lumbar drain was placed in 10 patients before surgery and opened to allow intraoperative brain collapse. One of the three patients who did not have a lumbar drain placed had a ventriculostomy catheter placed for hydrocephalus. The other two patients without lumbar drains were operated on for middle cerebral artery aneurysms, which generally are treated without a lumbar drain at our institution. An epidural drain was placed in all patients at the end of surgery.

All patients had postoperative CT within 24 hours of surgery. CT was performed with 5-mm axial sections through the posterior fossa and 10-mm sections through the remainder of the brain. Nine patients had follow-up CT scans 5 days to 12 weeks after surgery. Four patients had postoperative MR imaging on a 1.5-T unit with conventional spin-echo pulse sequences; that is, 5-mm-thick sagittal and axial T1-weighted images (400–500/15–20/2 [repetition time/echo time/excitations]) and 5-mm-thick axial dual-echo images (2500/30,90/1). One patient had MR imaging at 46 and 115 days after surgery, and the others had a single MR examination 10 days, 20 days, and 23 days, respectively, after surgery. All follow-up MR and

CT studies were obtained because of neurologic changes not related to the cerebellum. Two patients had no imaging follow-up after their initial postoperative CT study, because they had no new or unexpected neurologic changes.

Results

All patients had postoperative hemorrhage at the superior aspect of the cerebellum on CT scans. In each case, the hemorrhage spread transversely along the cerebellar folia and fissures, indicating that the blood was located mainly in the subarachnoid space (Figs 1–4). In all cases, there was low attenuation in the cerebellum immediately adjacent to the hemorrhage, which presumably represented local edema (Figs 1–4). In seven cases the hemorrhage was strictly curvilinear (Figs 1–3), whereas in six cases the hemorrhage was somewhat irregularly curvilinear (Fig 4). Five of the hemorrhages crossed the midline, and eight were confined to one cerebellar hemisphere. Of the eight unilateral hemorrhages, six were ipsilateral to the side of surgery and two were contralateral. There was no evidence of direct extension of subarachnoid blood from the supratentorial compartment into the posterior fossa in any of the cases.

In 12 patients, the hemorrhage was present on the initial postoperative CT scan. In one patient, the hemorrhage was not present on the initial CT scan obtained on the day after surgery, but was first identified on a CT scan obtained 3 days after surgery (Fig 2). A frontal,

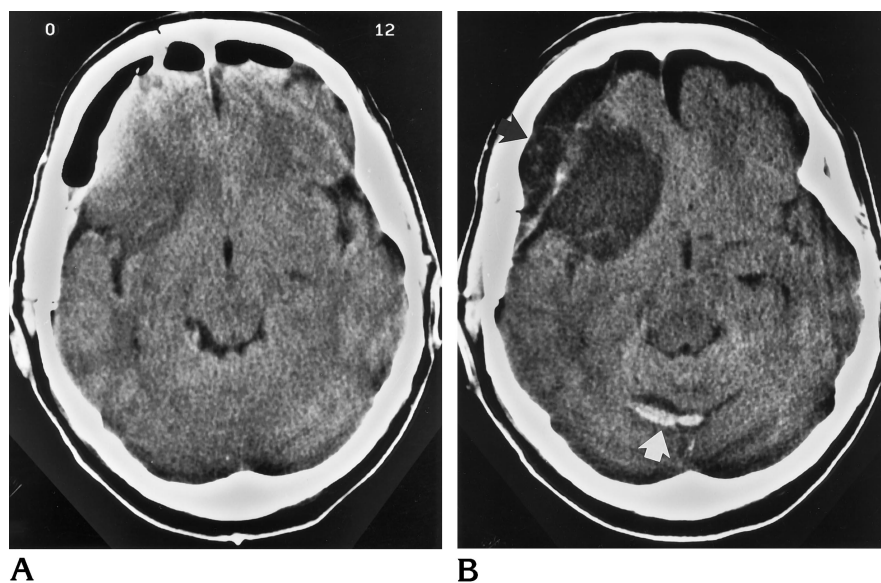


Fig 2. A 62-year-old woman who underwent clipping of right middle cerebral artery aneurysm.

A, No posterior fossa hemorrhage is present on the initial CT scan the day after surgery. A small amount of subarachnoid blood is seen in the quadrigeminal cistern on the right.

B, Posterior fossa hemorrhage (white arrow) is identified on CT scan 3 days after surgery. A right frontal low-attenuation extraaxial fluid collection (black arrow) developed at the site of surgery between the time the CT scans were obtained on the first and third postoperative days. A right middle cerebral artery distribution infarction has also developed. Subarachnoid blood in the quadrigeminal cistern on the right is unchanged.

low-attenuation, extraaxial fluid collection had also developed at the site of surgery between the first and third postoperative days, and 100 mL of serosanguinous fluid was drained from this collection. No other patient had focal mass effect or herniation related to the hemorrhage. Two patients had diffuse edema without herniation after surgery, and one had postoperative hydrocephalus. One patient had an additional 2-cm focus of hemorrhage, which was located in the frontal lobe contralateral to the surgical site.

There was no evidence of rebleeding at the site of posterior fossa hemorrhage on follow-up CT or MR studies. The follow-up CT examinations obtained 3 to 12 weeks after surgery showed low-attenuation material within the cerebellar fissure in which high-attenuation blood had been seen previously (Fig 1). Follow-up MR examinations in three patients obtained at 10, 20, and 23 days after hemorrhage, respectively, showed subacute blood isolated within a cerebellar fissure (Fig 3). The one patient in whom long-term (46 days and 115 days) follow-up MR studies were obtained had widening of the cerebellar fissure into which bleeding had occurred and hemosiderin staining on the cerebellar surface, but there was no definite cerebellar parenchymal extension (Fig 4).

A review of the preoperative imaging findings revealed no evidence of posterior fossa tumor or vascular malformation in any of the patients. Among the 12 patients who had preoperative angiography, the jugular veins and transverse

and sigmoid sinuses were widely patent bilaterally and fairly symmetric in 10 cases, significantly smaller on the left in one case, and not seen well in one case. In the patient with meningioma, the transverse and sigmoid sinuses were patent bilaterally and fairly symmetric on MR images. This patient and seven of the patients with aneurysms had no evidence of preoperative intracranial hemorrhage. Five of the patients with aneurysms had subarachnoid hemorrhage (two with extensive diffuse subarachnoid hemorrhage; two with interhemispheric subarachnoid blood, intraventricular blood, and frontal lobe blood from rupture of an aneurysm of the anterior communicating artery; and one with subarachnoid hemorrhage limited to the basal cisterns). Nine patients had mild cerebellar and cerebral atrophy, and the remainder had normal brain volume.

All patients had normal prothrombin time, partial thromboplastin time, and platelet counts at the time of surgery. Two patients were taking a nonsteroidal antiinflammatory drug before surgery, which has been shown to be a risk factor for intracranial hemorrhage complicating neurosurgical procedures (21). Six patients had a perioperative episode of blood pressure elevation to >180 mm Hg systolic, while the systolic blood pressure in the other seven patients remained below this level. None of the patients had a documented postoperative hypotensive episode.

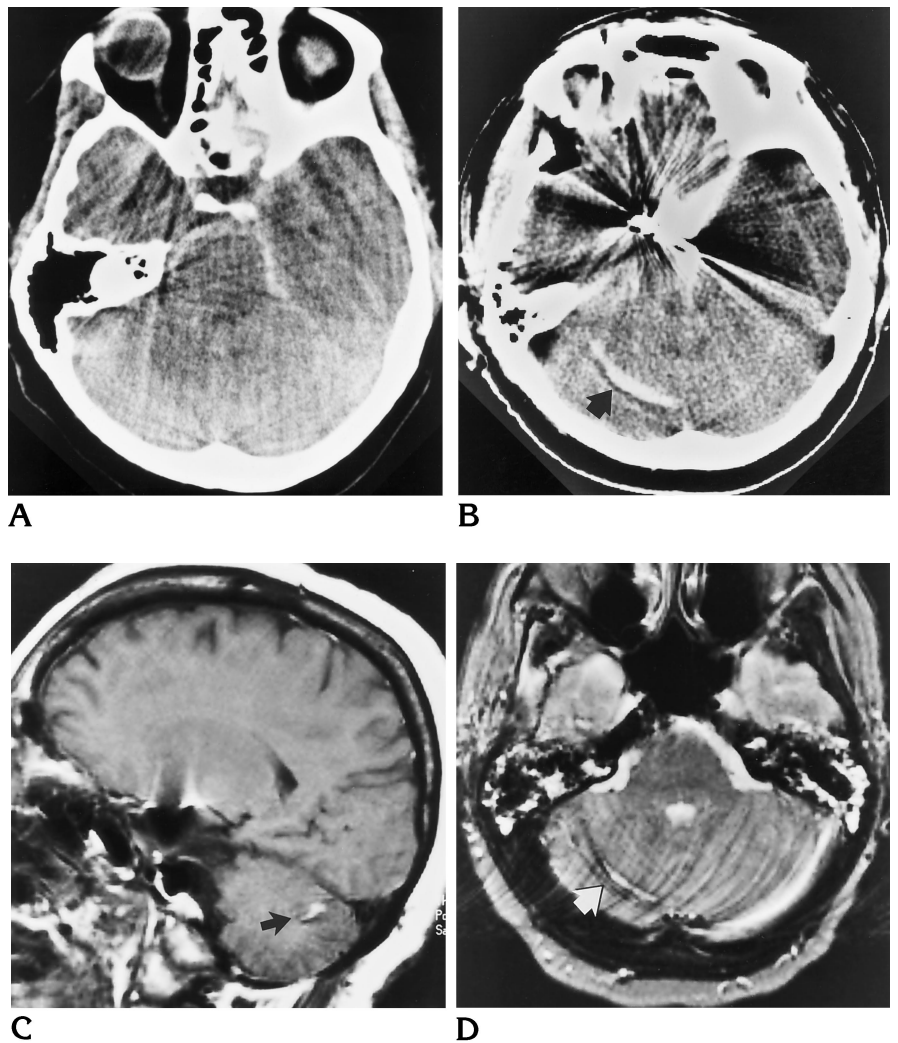
None of the patients had cerebellar signs or symptoms after surgery, and the presence of

Fig 3. A 63-year-old woman with ruptured basilar tip aneurysm.

A, Preoperative CT scan shows subarachnoid blood surrounding pons but no hemorrhage involving the cerebellum.

B, Postoperative axial CT scan shows curvilinear hemorrhage at superior aspect of right cerebellar hemisphere (*arrow*).

C and D, Sagittal T1-weighted (400/15/2) (C) and axial T2-weighted (2500/30,90/1) (D) images obtained 19 days after hemorrhage show subacute hemorrhage isolated within a cerebellar fissure (*arrows*).



hemorrhage did not change treatment of any of the patients. One patient died of other complications 1 week after surgery, but a request for autopsy was denied.

Since we were unable to determine the total number of supratentorial surgeries at our institution during the study period, we cannot calculate the overall frequency of posterior fossa hemorrhage occurring after this procedure. We do know that 317 operations for anterior circulation aneurysms were performed from a pterional approach, and that 11 patients with posterior fossa hemorrhage had anterior circulation aneurysms, yielding a frequency of hemorrhage of 3.5% in this subset of patients.

Discussion

The horizontal, curvilinear configuration of the hemorrhages on CT scans indicates that

they probably extend superficially in the subarachnoid space of a cerebellar fissure. The location of the hemorrhages within a cerebellar fissure was confirmed on MR images in four cases. In a case recently reported by Toczek et al (17), a posterior fossa hemorrhage after supratentorial surgery paralleled the cerebellar folia and fissures, quite similar to our cases, but was located primarily in the cerebellar parenchyma. Since CT does not give the same resolution as MR imaging in the posterior fossa, and we have no pathologic proof, we cannot be sure that some of the hemorrhages were not located mainly in the cerebellar parenchyma. Whether the hemorrhages in our series arose predominantly in the subarachnoid space or superficially in the cerebellar hemisphere with rupture into the subarachnoid space is not entirely clear. Six of the hemorrhages were somewhat irregular rather than purely curvilinear, which

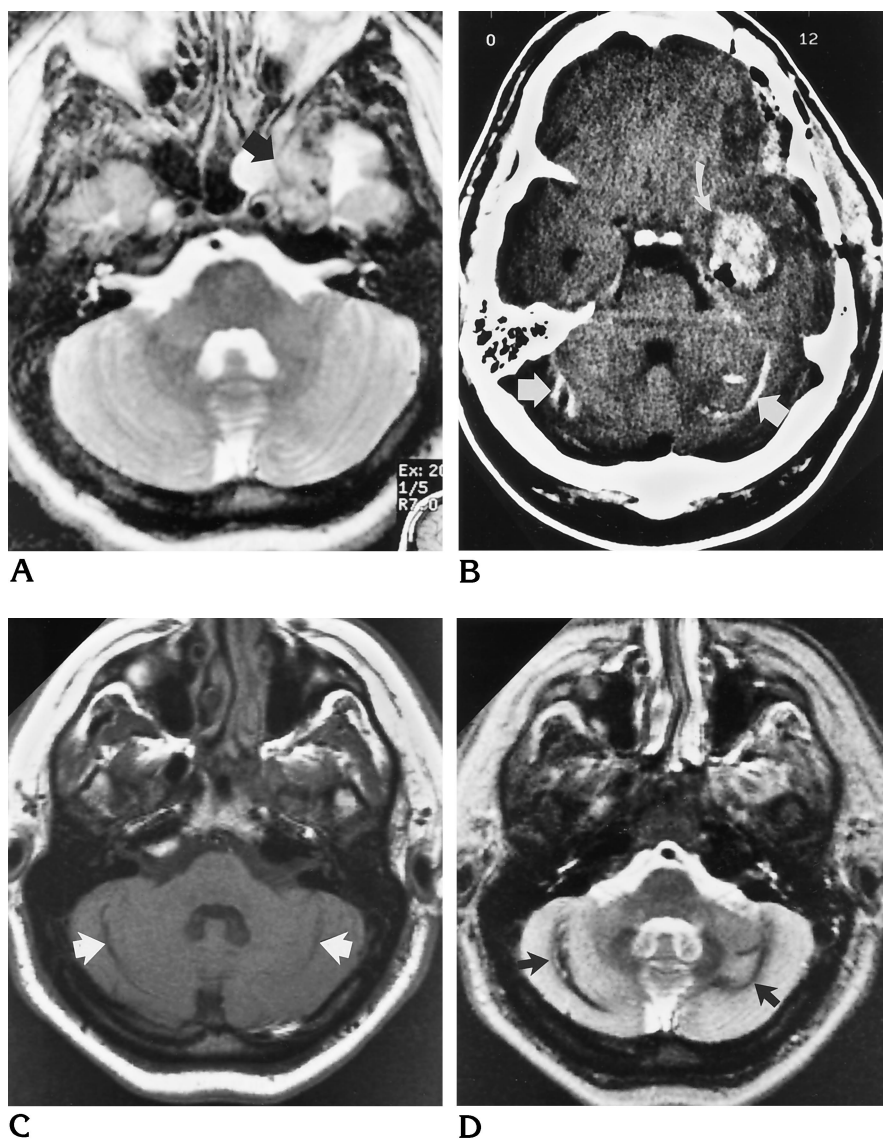


Fig 4. A 47-year-old man with left sphenoid wing meningioma.

A, Preoperative axial T2-weighted (2500/30,90/1) MR image shows meningioma in middle cranial fossa (*arrow*) but no hemorrhage involving the cerebellum.

B, Postoperative axial CT scan shows curvilinear hemorrhage at superior aspect of both cerebellar hemispheres (*straight arrows*). Hemorrhage is also seen in surgical bed (*curved arrow*).

C, Axial T1-weighted (400/15/2) MR image obtained 3 months after hemorrhage shows widening of the cerebellar fissure into which bleeding had occurred (*arrows*).

D, Axial T2-weighted (2500/30,90/1) MR image shows low signal intensity, consistent with hemosiderin staining on the cerebellar surface, but no definite cerebellar parenchymal extension (*arrows*).

also raises the question of whether there could be some intraparenchymal component. These hemorrhages did not have the typical appearance of spontaneous cerebellar hemorrhage (often presumed to be due to hypertension), which is centered in the cerebellar parenchyma and usually does not involve the subarachnoid space, although it may extend into the fourth ventricle (22, 23).

In our patients, rupture of veins as they course through the subarachnoid space of a cerebellar fissure or where they enter the cerebellar parenchyma owing to a loss of cerebrospinal fluid (CSF) may have been the mechanism of hemorrhage. If such shifts tear veins as they enter the cerebellar parenchyma, some bleeding into the parenchyma could occur pri-

marily, or secondarily, as a result of venous infarction caused by interruption of the vein. However, we found no evidence of venous infarction on any of the follow-up imaging studies, and none of the patients had cerebellar symptoms. CSF loss through the craniotomy and lumbar drain, as well as possible suction drainage of CSF through the epidural drain from a small dural leak after surgery, might have contributed to the development of the hemorrhages. The hemorrhages in our series occurred primarily in the patients with aneurysms, probably because of a greater need to relax the brain by draining large volumes of CSF through a lumbar drain in these patients as opposed to those whose craniotomies were performed for other indications. Three patients did not have

lumbar drains, but one of these three did have CSF drained through a ventriculostomy catheter. The other two patients might have lost large amounts of CSF either during surgery or after through the epidural drains.

In the one patient in whom hemorrhage did not occur immediately after surgery but, rather, 3 days later, along with a new frontal epidural low-attenuation fluid collection at the surgical site, the cause may have been additional post-operative loss of CSF into the epidural space. To our knowledge, this type of posterior fossa hemorrhage has not been reported as a complication of intracranial hypotension resulting from lumbar puncture. Perhaps the much greater volume and rate of CSF loss from surgery as compared with lumbar puncture can explain why such hemorrhages have not been described after lumbar puncture.

We found twenty-three cases of posterior fossa hemorrhage (7, 10–17) complicating supratentorial surgery reported previously. Four cases reported by Toczek et al (17), three cases reported by Yoshida et al (14), one case reported by Waga et al (7), and one case reported by Miyamoto et al (11) are similar to those reported here, with blood spreading horizontally along the cerebellar folia and fissures. Three additional hemorrhages reported previously (13, 15) were not as clearly oriented along the cerebellar fissures. The four patients described by König et al (12) all had posterior fossa hemorrhages larger than those described here, with clear extension deep into the cerebellum, but were all coagulopathic owing to the prophylactic use of heparin for deep venous thrombosis. Whether these patients hemorrhaged from the same mechanism as those in our study but bled more severely because of their coagulopathy or whether their hemorrhages were a direct complication of anticoagulation is unknown. A recent article by Papanastassiou et al (16) reports five cases of hemorrhagic infarction after supratentorial surgery in which the patients suffered acute neurologic compromise. They suggested venous occlusion caused by rapid shifting of the brain consequent to CSF loss as the possible mechanism (16). Cerebellar hemorrhage was reported as a complication of extracranial bypass in two patients (10) but no images or details about the patients were provided. Four cases of pontine hemorrhage (24) after supratentorial surgery were reported in 1960, but these were most likely brain stem

hemorrhages subsequent to transtentorial herniation (1).

We found five cases of intracerebral hemorrhage complicating lumbar puncture have been reported (25), but they all were hemorrhages into a cerebral hemisphere. Supratentorial subdural hematoma has been described after lumbar myelography (26). Primary intracranial hypotension has also been implicated as a potential cause of subdural hematomas along the cerebral convexities (27). Intracranial hypotension stemming from loss of CSF can result in downward displacement of the cerebellum and brain stem (28). This downward displacement relative to the tentorium may put tension on the veins extending from the cerebellar surface to the tentorial and straight sinuses (7, 18). Veins from the vermis and cerebellar hemispheres have been shown to connect with tentorial sinuses in 90% of patients (29). Some of these veins are known to arise from deep within the cerebellar fissures (30).

It has been suggested that the position of the head is related to the pathogenesis of posterior fossa hemorrhages after supratentorial surgery (16, 17). Toczek et al (17) proposed that turning and extending the head could occlude the jugular vein on the side toward which the head is turned, resulting in elevated venous pressure, especially if it is the dominant jugular vein being compressed. This is an interesting theory; yet, without proof that such occlusion occurs or that a unilateral jugular vein occlusion would cause a substantial intracranial venous pressure increase, it remains speculative. We found the jugular veins and transverse and sigmoid sinuses to be widely patent bilaterally in 11 of 12 patients in whom this could be assessed, which suggests that venous outflow through a contralateral jugular vein could occur even if one assumes that complete occlusion of an internal jugular vein related to head positioning is occurring. There were also no internal jugular venous catheters used in our patients, eliminating them as a possible cause of jugular vein occlusion.

Papanastassiou et al (16) reported five patients who had hemorrhage in the cerebellar hemisphere contralateral to the side of pterional craniotomy. These authors theorized that the hemorrhage occurred in the contralateral cerebellar hemisphere because this was the most dependent part of the brain when the head is turned away from the craniotomy, and that with

CSF removal, this part of the cerebellum would move posteriorly to abut the transverse sinus, kinking and obstructing the draining veins from the superior aspect of the cerebellar hemisphere (16). We found no such predilection for the hemorrhage to occur in the contralateral cerebellar hemisphere, and it is likely that the small sample size in their series accounts for their failure to find ipsilateral or bilateral hemorrhages.

The frequency of posterior fossa hemorrhage after anterior circulation aneurysm in our series was 3.5%. This is much higher than the frequency of 0.13% to 0.5% that has been reported by others (12, 15, 16). Toczek et al (17) reported a 5% frequency of posterior fossa hemorrhage after temporal lobectomy. The higher rate in our series and in that of Toczek et al may be due to a more thorough database search to identify patients, to performance of routine CT after all craniotomies (which would identify more asymptomatic hemorrhages), and to differences in surgical technique.

The postoperative posterior fossa hemorrhages described here have a characteristic appearance that is unlike that of cerebellar hemorrhage caused by hypertension. None of the patients suffered adverse sequelae from the hemorrhage or required a change in management. Toczek et al (17) reported four cases with a similar, benign course. This is in contrast to five cases of hemorrhagic cerebellar infarction after supratentorial surgery recently described by Papanastassiou et al (16) in which all patients were profoundly affected and required intervention. Other cases have also been reported in which the patients were significantly affected by the hemorrhage (7, 12, 14). The hemorrhages in these patients were larger than those in our series and in the series of Toczek et al (17), and they appear to have been a more severe form of the same pathologic process, perhaps with more extensive venous disruption resulting in infarction. Presence of hemorrhage alone does not appear to be an indication for follow-up imaging, since none of the hemorrhages progressed. It is important to recognize this type of hemorrhage, when small, as an apparently benign sequela of surgery rather than as a result of arterial hypertension, as previously unrecognized vascular malformation or neoplasm, or as subarachnoid bleeding from a ruptured aneurysm.

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