Cerebellar hemorrhage arising postoperatively as a complication of supratentorial surgery: a retrospective study

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Object. Postoperative cerebellar hemorrhage as a complication of supratentorial surgery is an increasingly recognized clinical entity. So far, it has remained unclear whether this complication constitutes an intraoperative or postoperative event. The observation of such cases prompted the authors to analyze retrospectively their series of supratentorial craniotomies. The aim of this study was to determine the incidence of cerebellar hemorrhage and its temporal relationship to supratentorial surgery.

Methods. The authors reviewed discharge notes and reports on postoperative computerized tomography (CT) scans for 1650 patients who had undergone supratentorial craniotomy between January 1998 and February 2001.

The retrospective study led to the identification of 10 patients who had sustained cerebellar hemorrhage as a complication of supratentorial surgery. Because it was routine to perform CT scanning following craniotomy, an early CT scan obtained within the 1st postoperative hour (mean 24 minutes after wound closure) was available in eight of the 10 patients.

In seven of these patients no hemorrhage was found immediately after surgery, and in only one patient was there the suspicion of cerebellar hemorrhage. In the whole series of 10 patients, cerebellar hemorrhage was detected during the later postoperative course, after a mean interval of 7 hours and 35 minutes (range 1 hour and 49 minutes–144 hours) following surgery. The incidence of cerebellar hemorrhage was 0.6% of all patients who underwent supratentorial surgery. Among patients suffering from epilepsy the incidence was 4.6%, and in those patients who underwent temporal lobe resection it was 12.9%.

Conclusions. The authors have demonstrated that cerebellar hemorrhage as a complication of supratentorial surgery arises not as an intraoperative event, but as a postoperative event. Resective nontumorous temporal lobe procedures place patients at particular risk for this complication. Evidence suggests that the complication might be precipitated by postoperative suction drainage.

KEY WORDS • cerebellar hemorrhage • temporal lobectomy • supratentorial craniotomy • epilepsy surgery • temporal lobectomy • complication

Cerebellar hemorrhage is an increasingly recognized complication of supratentorial surgery.1–3,7,8 The clinical symptoms secondary to cerebellar hemorrhage are often mild and transient; however, in some cases untoward and even fatal outcomes have been described.1,3,4

Several causative factors have been discussed. These include intraoperative factors such as venous obstruction,4 intraoperative positioning of the patient,5 administration of mannitol,6 and intraoperative drainage of CSF.8 Apparently, the frontotemporal or pterional approach predisposes the brain to cerebellar hemorrhage.1,4 Most operations are performed for lesions involving the sylvian, suprasellar, or temporal area.5 In addition, postoperative causes of cerebellar hemorrhage have been discussed. In particular, it has been suggested that extensive postoperative CSF loss due to suction drainage precipitates hemorrhage.8,9 Other factors of a general medical nature predisposing the brain to hemorrhage, such as coagulopathy and arterial hypertension, have also been discussed.2,3,5

It has been postulated that hemorrhage likely develops at or soon after surgery;1,8 however, it remains unsolved whether cerebellar hemorrhage associated with supratentorial surgery constitutes an intraoperative or a postoperative event. In all previous cases, postoperative neuroradiological imaging was only performed when the hemorrhage had already occurred.1,3,6,8,9 Generally, CT scanning was scheduled when symptoms secondary to cerebellar hemorrhage had emerged.

Our retrospective study of patients who underwent supratentorial craniotomy revealed 10 patients with postoperative cerebellar hemorrhage. We analyzed early postoperative and follow-up CT scans obtained in these patients to determine the point of time when the complication of cerebellar hemorrhage occurred in relation to the supratentorial procedure. The study was enabled by the routine practice at our department of performing control CT scanning immediately after craniotomy procedures.

Clinical Material and Methods

The observation of cases in which infratentorial hemorrhage complicated supratentorial craniotomy at our department prompted us to undertake a systematic analysis of our...
Cerebellar hemorrhage as a complication of supratentorial surgery

Preoperative and intraoperative clinical characteristics of 10 patients with cerebellar hemorrhage as a complication of supratentorial surgery*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs)</th>
<th>Sex (F/M)</th>
<th>Epilepsy Surgery</th>
<th>Anticonvulsant (daily dose in mg)</th>
<th>Craniotomy Site</th>
<th>Operation</th>
<th>Pathological Finding</th>
<th>Intraop Mannitol</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>57, F</td>
<td>yes</td>
<td>none</td>
<td>lamotrigine (400)</td>
<td>rt temporal</td>
<td>TL &amp; AH</td>
<td>cortical dysplasia &amp; MTS</td>
<td>no</td>
</tr>
<tr>
<td>2</td>
<td>44, M</td>
<td>yes</td>
<td>none</td>
<td>phenytoin (400)</td>
<td>rt temporal</td>
<td>TL &amp; AH</td>
<td>cortical dysplasia &amp; MTS</td>
<td>no</td>
</tr>
<tr>
<td>3</td>
<td>28, M</td>
<td>yes</td>
<td>none</td>
<td>lamotrigine (400)</td>
<td>rt temporal</td>
<td>TL &amp; AH</td>
<td>ganglioglioma &amp; temporomesial astrocytosis</td>
<td>no</td>
</tr>
<tr>
<td>4</td>
<td>21, M</td>
<td>yes</td>
<td>none</td>
<td>carbamazepine (1600)</td>
<td>rt temporal</td>
<td>TL &amp; AH</td>
<td>cortical dysplasia &amp; MTS</td>
<td>no</td>
</tr>
<tr>
<td>5</td>
<td>44, M</td>
<td>yes</td>
<td>none</td>
<td>carbamazepine (1650)</td>
<td>lt pterional</td>
<td>selective AH</td>
<td>MTS</td>
<td>yes</td>
</tr>
<tr>
<td>6</td>
<td>20, M</td>
<td>yes</td>
<td>none</td>
<td>carbamazepine (1600)†</td>
<td>lt pterional</td>
<td>selective AH</td>
<td>MTS</td>
<td>yes</td>
</tr>
<tr>
<td>7</td>
<td>33, M</td>
<td>yes</td>
<td>oxcarbazepine (1500)</td>
<td>lt pterional</td>
<td>selective AH</td>
<td>MTS</td>
<td>yes</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>33, M</td>
<td>yes</td>
<td>none</td>
<td>carbamazepine (900)</td>
<td>lt frontal</td>
<td>resection of intraventricular meningioma (3rd recurrence)</td>
<td>no</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>54, M</td>
<td>no</td>
<td>none</td>
<td>none</td>
<td>lt occipital</td>
<td>lesionectomy &amp; MST</td>
<td>astrocytoma Grade II</td>
<td>no</td>
</tr>
<tr>
<td>10</td>
<td>47, M</td>
<td>no</td>
<td>none</td>
<td>none</td>
<td>rt pterional</td>
<td>ACoA aneurysm clipping</td>
<td>aneurysm only</td>
<td>no</td>
</tr>
</tbody>
</table>

* MST = multiple subpial transections; MTS = mesiotemporal sclerosis; TL = temporal lobectomy.
† Patient’s medical regimen had previously included valproic acid (1500 mg daily), which was discontinued 16 days before surgery.

Results

Cerebellar hemorrhage was observed in three (3.9%) of 76 patients who had undergone selective AH and in four (12.9%) of 31 patients who had undergone temporal lobectomy including AH. The incidence of cerebellar hemorrhage was only 0.6% for the whole series of 1650 patients who had undergone supratentorial craniotomy. If supratentorial procedures other than those performed for epilepsy were considered, the rate of cerebellar hemorrhage was as low as 0.14%.

Operative Procedures

Eight patients were treated for medically refractory epilepsy (Table 1). In four cases, a temporal lobectomy including AH was performed via a temporolateral craniotomy. Three patients underwent selective AH, which was performed via a pterional craniotomy and a transsylvian approach. In one patient, a left frontal low-grade (Grade II) astrocytoma that caused the epilepsy was treated via a frontal craniotomy. Two patients underwent surgery for reasons other than epilepsy (Cases 9 and 10; Table 1).

Intraoperative Management

In three patients, 125 ml of mannitol (20% solution) were administered before the dura mater was opened so that brain relaxation could be achieved (Table 1).

A broad access to the ventricular system was created during the surgical procedures in nine of the 10 cases. Wide opening of the temporal horn is mandatory for temporal lobe surgery (four cases) and for selective AH (three cases). In the patient who underwent surgery for an aneurysm (Case 10), intraoperative CSF drainage from the ventricles was achieved by opening the lamina terminalis. In the patient who harbored a ventricular meningioma (Case 9), a wide occipital defect that communicated with the ventricular system remained from previous operations. In only one patient (Case 8) was the ventricular system not opened. The sylvian fissure was widely opened in the three patients in whom selective AH was performed (Cases 5–7) and in the patient undergoing surgery for an ACoA aneurysm (Case 10).
TABLE 2
Postoperative clinical characteristics of 10 patients with cerebellar hemorrhage as a complication of supratentorial surgery*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>1st Postop CT Scan W/O Hemorrhage</th>
<th>Postop CT Scan W/ Hemorrhage</th>
<th>Location of Hemorrhage</th>
<th>Postop Symptoms</th>
<th>Symptoms at Discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>24 mins</td>
<td>7 hrs</td>
<td>upper folia (ipsilat), upper vermis</td>
<td>disorientation, cerebellar signs</td>
<td>cerebellar dysmetria</td>
</tr>
<tr>
<td>2</td>
<td>21 mins†</td>
<td>8 hrs</td>
<td>upper folia (bilat), upper vermis</td>
<td>disorientation</td>
<td>none</td>
</tr>
<tr>
<td>3</td>
<td>20 mins</td>
<td>27 hrs</td>
<td>upper folia (bilat), upper vermis</td>
<td>drowsiness, headache</td>
<td>none</td>
</tr>
<tr>
<td>4</td>
<td>NA</td>
<td>19 hrs</td>
<td>upper folia &amp; cerebellar hemisphere</td>
<td>headache</td>
<td>none</td>
</tr>
<tr>
<td>5</td>
<td>20 mins</td>
<td>7 hrs</td>
<td>upper cerebellum (contralat), upper vermis</td>
<td>lethargy, drowsiness, vomiting</td>
<td>lethargy</td>
</tr>
<tr>
<td>6</td>
<td>17 mins</td>
<td>28 hrs</td>
<td>upper folia (contralat more than ipsilat)</td>
<td>drowsiness, headache, vomiting</td>
<td>none</td>
</tr>
<tr>
<td>7</td>
<td>59 mins</td>
<td>48 hrs</td>
<td>upper folia (contralat more than ipsilat)</td>
<td>seizures</td>
<td>none</td>
</tr>
<tr>
<td>8</td>
<td>NA</td>
<td>1 hr &amp; 49 mins</td>
<td>upper folia (bilat), subdural (infratentorial)</td>
<td>asymptomatic</td>
<td>none</td>
</tr>
<tr>
<td>9</td>
<td>24 mins</td>
<td>2 hrs &amp; 42 mins</td>
<td>upper cerebellum (bilat), subarachnoid</td>
<td>coma</td>
<td>squint deviation</td>
</tr>
<tr>
<td>10</td>
<td>10 mins</td>
<td>144 hrs</td>
<td>upper folia (bilat)</td>
<td>asymptomatic</td>
<td>none</td>
</tr>
</tbody>
</table>

* NA = not applicable.
† In this case findings raised suspicion of hemorrhage.

Suction Drainage

In all cases, a subgaleal suction draining tube was inserted. In cases in which a pterional or temporal craniotomy was performed, the draining tube ended in the epidural space basal to the bone flap, where additional osteoclastic bone removal was performed for basal exposure. The drainage tube was routinely removed on the 1st postoperative day. In one patient (Case 10), an additional drainage tube, which released CSF without suction, was inserted into the sylvian fissure.

Neuroradiological Findings

Eight of our 10 patients underwent routine CT scanning within the 1st postoperative hour (Table 2). These CT scans were obtained within a mean of 24 minutes after wound closure (range 10–59 minutes). The early postoperative CT scans obtained in seven patients provided no evidence of cerebellar hemorrhage. In only one patient (Case 2) did our retrospective analysis of the early CT scan, which in this case was obtained 21 minutes after surgery, elicit the suspicion of a right-sided tiny hemorrhage in the cerebellar folia. Overt cerebellar hemorrhage was demonstrated on later follow-up CT scans in all patients (Table 2 and Fig. 1).

In six of 10 patients, cerebellar hemorrhage was documented by CT scans within the 1st postoperative day (Fig. 1). The mean time interval after wound closure was 7 hours and 35 minutes (range 1 hour and 49 minutes–19 hours). In the remaining four patients, hemorrhage was exhibited at 27 hours, 28 hours, 2 days, and 6 days postoperatively, respectively.

Additional follow-up CT scans were available in seven patients (Fig. 1). Slight progression of hemorrhage was found in one patient. In six patients, no further increase in cerebellar hemorrhage was observed.

In five patients, additional MR imaging was performed throughout the postoperative course. The MR images demonstrated hemorrhage within the cerebellar parenchyma. In two patients MR angiograms were available, but no abnormalities were detected.

Clinical Findings

Primary symptoms when awaking from anesthesia in the ICU were observed in four patients and two patients later experienced secondary symptoms (Cases 4 and 6; Table 2). One patient (Case 7) became symptomatic with epileptic seizures, and the cerebellar hemorrhage detected on CT scans in that case was considered a chance finding. One patient (Case 3) suffered from a progressive postoperative headache and became drowsy secondarily. Computerized tomography scans demonstrated rebleeding in the surgical field and additional cerebellar hemorrhage. This patient fully recovered from his symptoms after evacuation of the supratentorial rebleeding, and the cerebellar hemorrhage was considered an incidental finding. In the remaining two cases, cerebellar hemorrhage was incidentally found on routine follow-up CT scans.

At the time of discharge, three patients displayed residual symptoms due to cerebellar hemorrhage. One had cerebellar dysmetria, one was still slightly lethargic, and the third patient had a residual squint deviation (Table 2). The remaining seven patients were free of symptoms.

Blood Pressure and Coagulation Parameters

Only one patient (Case 10) suffered from mild arterial hypertension preoperatively, but did not receive regular antihypertensive medication. The other patients had no history of arterial hypertension. In all cases, blood pressure remained within normal limits throughout the operative procedure, as documented in the charts prepared by the anesthetists. One patient (Case 5) experienced a period of slight hypertension during which his maximum systolic blood pressure was 175 mm Hg in the evening following the operation. In another patient (Case 10), a single elevated blood pressure value (systolic pressure 180 mm Hg) was measured when the patient arrived at the ICU. Postoperative blood pressure was otherwise normal in all patients.

Preoperative and postoperative prothrombin times, partial thromboplastin times, and platelet counts were normal in all cases. One patient with epilepsy (Case 6) had previ-
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Case 1

**History.** This 57-year-old woman suffered from epilepsy that had proved to be resistant to all common anticonvulsant drugs. At the time of surgery her medical regimen consisted of lamotrigine 400 mg daily.

**Examination.** Magnetic resonance images demonstrated a marked right hippocampal sclerosis and a basal and lateral dysplasia of the right temporal lobe. Video–electroencephalographic monitoring performed using scalp and sphenoidal electrodes demonstrated seizure onset in the right temporal lobe. She was otherwise healthy and had no history of arterial hypertension.

**Operation.** Surgery was performed while the patient lay supine. Her head was rotated 70° with her right shoulder elevated to avoid venous congestion due to compression of the jugular veins. A standard right-sided temporal craniotomy was performed. The temporal lobe was resected over a length of 4.5 cm along the sylvian fissure. Amygdalohippocampectomy was performed. During wound closure, a subgaleal suction drain tube was placed and this was attached to a suction drainage system while the patient was still in the operating room.

**Postoperative Course.** A routine postoperative CT scan was obtained 24 minutes after surgery and demonstrated no abnormalities (Fig. 2 upper left and right). In particular, no evidence of cerebellar hemorrhage was found. The patient awoke in the ICU. When she was extubated 3 hours after surgery, she was found to be confused. On neurological examination, cerebellar signs with cerebellar dysmetria and ataxia were detected. Computerized tomography scanning was therefore repeated 7 hours after surgery. The CT scan revealed hemorrhage within the upper vermis and right cerebellar folia underneath the tentorium (Fig. 2 center left and right). An additional control CT scan was obtained 27 hours postoperatively and it demonstrated no further increase in the blood collection within the upper cerebellum (Fig. 2 lower left and right). The suction draining tube was removed on the 1st postoperative day. Two days after surgery, MR images revealed that the hemorrhage was located within the folia and not subdurally (Fig. 3). During the patient’s later postoperative course, her symptoms gradually diminished. A control CT scan obtained 2 weeks after sur-

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Illustrative Cases

**Case 1**

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gery revealed that the hemorrhage was partially resorbed. The patient’s blood pressure was normal throughout her hospital stay. At the time of discharge, her neurological state was normal, apart from a mild dysmetria.

Case 9  

History. This 56-year-old man had previously undergone surgeries for a left ventricular meningioma 25, 22, and 12 years previously. At the time of the study he presented with increasing headache and blurred vision.

Examination. Magnetic resonance images revealed a recurrence of tumor in the atrium of the left lateral ventricle (Fig. 4) and two additional tiny meningioma nodules. On examination, he displayed no neurological deficits, apart from a previously known homonymous hemianopsia.

First Operation. Surgery was performed while the patient lay prone. Following a left occipital craniotomy, the meningioma was extirpated through a preexisting occipital brain defect that communicated with the lateral ventricle. Surgery was performed without any intraoperative complications. The bone defect was closed by a large cranioplasty and a subgaleal suction draining tube was inserted.

First Postoperative Course. A routine postoperative CT scan was obtained 24 minutes following wound closure. The CT scan demonstrated a minor subarachnoid blood collection in the perimesencephalic cisterns, but there was no evidence of cerebellar hemorrhage (Fig. 5 left). During his stay in the ICU, the patient remained intubated because of a reduced level of consciousness. Control CT scanning was arranged 2 hours and 42 minutes postsurgery when he suffered a generalized epileptic seizure. The control CT scan revealed hemorrhage within the upper cerebellum, a swelling of the cerebellum, and upward herniation with midbrain compression.

Second Operation. The patient was transferred to the operating room and a suboccipital decompressive craniotomy was performed. No blood was encountered on the cerebellar surface. A large duraplasty was inserted.

Second Postoperative Course. After decompressive surgery, the patient’s clinical condition markedly improved. He was extubated on the 4th day following decompression and displayed an awake and responsive state. He was discharged from the hospital 2 weeks after surgery. At the time of discharge, neurological examination revealed a squint deviation and his previous hemianopsia, but no other neurological deficits.

Discussion

The results of our retrospective study demonstrate that cerebellar hemorrhage as a complication of supratentorial surgery occurs as a postoperative rather than an intraoperative event. The onset of hemorrhage takes place within a relatively brief postsurgical period. Except for one case (Case 9), no further increase in hemorrhage was observed in those patients in whom additional follow-up control CT scans were available. This finding suggests that an end point of hemorrhage had already been reached by the time the hemorrhage was detected by CT scanning.

The underlying mechanisms of cerebellar hemorrhage have been extensively discussed. König, et al., have suggested that removal of a supratentorial space-occupying mass may induce a reduction in intracranial pressure with a critical increase in the transmural pressure of veins or venules, which might account for the hemorrhage. These authors were the first to mention that the reduction in the intracranial pressure with consecutive hemorrhage was perhaps enforced by the postoperative subgaleal suction drainage, which was used in all of their three cases.

Yoshida and associates inserted an epidural draining
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tube with a suction device into each of their three patients and additional cisternal drainage tubes without suction in two of them. A large amount of fluid mixed with CSF was drained postoperatively. These authors found a temporal relationship between postoperative CSF loss and neurological deterioration due to cerebellar hemorrhage.

In our opinion, postoperative suction drainage is a predominant causative factor for cerebellar hemorrhage. Our subgaleal suction system exerts a negative pressure of 60 to 120 mm Hg. Even if the closure of the dura mater is apparently watertight, the high negative pressure will remove CSF from the intradural compartment between stitches made in the dura. Furthermore, the negative pressure exerted at the dura itself will cause a suction effect that is likely to be transmitted to the intradural space. As mentioned, suction drainage has also been used in previously reported cases, although not in all cases.

There is evidence to suggest that the pathophysiological mechanism is venous in origin. First, hematomas are almost uniformly located in the upper vermis and in the upper cerebellar folia facing the tentorium, where the draining veins of the cerebellar hemispheres are located. Second, hemorrhage is often bilateral. An arterial origin would instead cause unilateral bleeding. Angiography has been performed following postoperative cerebellar hemorrhage and revealed no abnormalities.

Yoshida and associates postulated that the cerebellum is displaced due to CSF loss, with stretching of the cerebellar veins, which may subsequently tear; however, tearing of the cerebellar veins would cause subdural rather than intracerebellar hemorrhage. In our opinion, it appears likely that a negative pressure is built up in the supratentorial, intradural compartment due to withdrawal of CSF and suction exerted at the exposed dura mater. On the other hand, the posterior fossa is protected by the tentorium and a normal pressure is maintained on the cerebellar surface. The crucial mechanism might be a transtentorial pressure gradient, that is, a gradient between the supratentorial and infratentorial venous systems. The tentorium carries the venous channels into which the superior cerebellar veins drain. On the other hand, the tentorial veins drain into the supratentorial deep venous system and the transverse sinus. We favor the theory that the negative supratentorial pressure reduces the pressure within tentorial and supratentorial veins, whereas the cerebellar venous pressure remains unchanged. Hence, by a suction effect blood is withdrawn from the infratentorial veins draining the upper cerebellum and vermis. The small venous channels and the capillary bed of the cerebellum, which are dependent on this blood flow, are traumatized by this suction mechanism and intracerebellar hemorrhage is precipitated. It appears unlikely that obstruction of the cerebellar veins is responsible for hemorrhage because deliberate obliteration of the supracerebellar veins during infratentorial supracerebellar surgery does not produce cerebellar hemorrhage.

Our cases support the findings by Toczek, et al., that temporal lobe resection predisposes the brain to cerebellar hemorrhage. We also found cerebellar hemorrhage after selective AH, but the incidence of hemorrhage was only 3.9% following selective AH as opposed to 12.9% following temporal lobectomy. In both procedures, the temporal horn is widely opened. Nevertheless, the cavity of resection underlying the dural opening is more extensive in temporal lobectomy and the tentorium is widely exposed. Furthermore, none of the 32 patients in whom temporal lesionectomy alone was performed sustained cerebellar hemorrhage. Apparently, the expansion of the CSF spaces, the size of the resection cavity, and/or the exposure of the tentorium are crucial for the development of postoperative cerebellar hemorrhage in temporal lobe surgery.

Cerebellar hemorrhage has been described in surgeries performed to treat aneurysms of the anterior circulation and suprasellar lesions during which a wide opening has been created in the sylvian and perisellar cisterns. The mechanism precipitating cerebellar hemorrhage might be similar to what occurs in those cases in which the procedure was temporal lobe surgery. With opening of the cisterns, a negative pressure caused by suction drainage might be transmitted to the deep venous system or the tentorial surface. Severe cerebellar hemorrhage has been reported by Yoshida and associates who used an additional cisternal drainage system.

Because hemorrhage occurs postoperatively, some other intraoperative mechanisms that have been considered in the literature appear less important in the pathophysiology of cerebellar hemorrhage. Intraoperative rotation or extension of the head with relative obstruction of the jugular veins has been considered, but can hardly explain postoperative hemorrhage. Similarly, intraoperative displacement of the cerebellum with kinking and obstruction of draining veins is unlikely to cause postoperative hemorrhage.

Arterial hypertension, which is the major risk factor for spontaneous cerebellar hemorrhage, can be ruled out as an important contributing factor to this complication. Grossly disturbed coagulation due to postoperative heparin administration has only been observed by König, et al. The administration of the anticonvulsive drug valproic acid can interfere with coagulation and has previously been used in some patients afflicted with cerebellar hemorrhage. Nevertheless, none of our eight patients with epilepsy was receiving valproic acid at the time of surgery. A clear association between cerebellar hemorrhage and coagulopathy can be excluded.
Conclusions

We conclude that cerebellar hemorrhage as a complication of supratentorial surgery occurs as a postoperative event. In our opinion, postoperative suction drainage with ensuing loss of CSF is the most likely cause of this complication. Extensive postoperative CSF drainage may be hazardous, especially during resective nontumorous temporal lobe surgery for epilepsy. We now insert a draining tube without applying any suction during these procedures and during surgeries in which a large resection cavity is created, a broad access to the cisterns or ventricles is provided or the tentorium is exposed. Additionally, we avoid intraoperative dehydration with mannitol whenever possible to avoid brain dehydration and additional increases in the CSF spaces during the early postoperative period.

References


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