Traumatic Hematomas of the Posterior Cranial Fossa

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Diagnostic improvements such as carotid angiography and ultrasound encephalography plus more effective methods of combatting cerebral edema with steroids, hypothermia, and massive decompression, have probably saved many patients with supratentorial intracranial hematomas. However, traumatic hematomas located within the posterior cranial fossa continue to escape detection for they are not readily demonstrated by the new diagnostic studies and all too often are not considered until found at autopsy. This study contains data on all patients seen within the last 12 years at the Massachusetts General Hospital with clinically significant verified posterior fossa hematomas attributed to trauma.

Table 1 shows the 17 cases of traumatic infratentorial hematoma that have been verified since 1955. The number of cases emphasizes the relatively infrequent occurrence of such lesions when compared with 344 cases of clinically significant traumatic supratentorial hematomas seen in this same period.

Extradural Hematomas

Hematomas confined to the extradural space are probably the most common traumatic space-occupying lesions in the posterior cranial fossa. Coleman and Thomson in 1941 reported the first successful operation for removal of an infratentorial traumatic hematoma. Since then several reports of 1 or 2 such cases have appeared,2,3,10,13,15,17,20,22,26,29 and larger series have been documented by Campbell et al.,4 Fisher et al.,9 Hooper,14 and Petit-Dutaillis et al.25 A total of over 50 cases have been recorded. Over half of these have occurred in children, and in virtually every case an occipital fracture crossing the lateral sinus has been present. Since symptoms are usually delayed for several hours or days it seems likely that the bleeding is of venous origin from the lateral sinus or bone. The sad fact that one-half of these reported patients died without operation emphasizes the difficulty in diagnosing a hematoma of the posterior fossa.

In our series, 6 patients had extradural hematomas in the posterior cranial fossa (Table 1). In each instance the patient had received a blow on the back of the head resulting in fractures extending from the occipital region downward into the foramen magnum. In 4 of these patients the diagnosis was suspected during life. Operation was performed, and each made an uneventful recovery.

Only 1 of these (No. 9) was operated upon within a few hours of injury. His automobile had struck a tree and he was thrown onto a paved highway. On admission to the hospital an hour later he remained stuporous and would obey no commands although all limbs were briskly withdrawn from painful stimuli. The deep tendon reflexes were symmetrical and normal; both plantar responses were up-going. X-rays showed a right occipital fracture extending downward into the foramen magnum. By the time these films were completed, stupor had increased and both eyes deviated to the left. At operation three hours after the accident a large right suboccipital extradural hematoma was removed and a laceration of the transverse sinus repaired. Recovery was complete.

Three patients (Nos. 2, 6, and 10) all sustained occipital trauma in falls and were admitted to the hospital for observation after x-rays had demonstrated fractures extending across the lateral sinus. Two of the patients (Nos. 2 and 10) had been drowsy and confused from the time of injury. In Case 10, these were the only abnormal neurological findings; Case 2 also had a left 6th cranial nerve palsy. Posterior fossa explorations were undertaken and large extradural hematomas, left-sided in Case 10 but bilateral in Case 2, were removed several days after the initial injury. Gratifying recovery ensued in both cases. In patient 2, the venous phase of right carotid arteriogram done be-

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TABLE I

Traumatic infratentorial hematoma: review of 17 cases

<table>
<thead>
<tr>
<th>No.</th>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Skull Fracture</th>
<th>Site of Posterior Fossa Hematoma</th>
<th>Inval Between Trauma &amp; Operation</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>W.A.</td>
<td>44</td>
<td>M</td>
<td>Occipital and suboccipital</td>
<td>Subdural</td>
<td>2 days</td>
<td>Continued coma; died third postoperative day</td>
</tr>
<tr>
<td>2</td>
<td>J.B.</td>
<td>47</td>
<td>M</td>
<td>Occipital and suboccipital</td>
<td>Extradural</td>
<td>5 days</td>
<td>Recovery</td>
</tr>
<tr>
<td>3</td>
<td>L.B.</td>
<td>74</td>
<td>F</td>
<td>Occipital and suboccipital</td>
<td>Cerebellar</td>
<td></td>
<td>No operation; died day after injury</td>
</tr>
<tr>
<td>4</td>
<td>J.D.</td>
<td>18</td>
<td>M</td>
<td>Occipital and suboccipital</td>
<td>Extradural</td>
<td></td>
<td>No operation; died 12 hours after injury</td>
</tr>
<tr>
<td>5</td>
<td>A.F.</td>
<td>22</td>
<td>F</td>
<td>—</td>
<td>Subdural</td>
<td>2–3 hrs.</td>
<td>Continued coma; died 2 hours after operation</td>
</tr>
<tr>
<td>6</td>
<td>D.F.</td>
<td>10</td>
<td>F</td>
<td>Occipital and suboccipital</td>
<td>Extradural</td>
<td>16 days</td>
<td>Recovery</td>
</tr>
<tr>
<td>7</td>
<td>P.H.</td>
<td>15</td>
<td>M</td>
<td>—</td>
<td>Cerebellar</td>
<td>3 mos.</td>
<td>Recovery</td>
</tr>
<tr>
<td>8</td>
<td>R.H.</td>
<td>19</td>
<td>M</td>
<td>Postoperative suboccipital craniectomy</td>
<td>Subdural</td>
<td>1 mo.</td>
<td>Recovery</td>
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<tr>
<td>9</td>
<td>R.M.</td>
<td>27</td>
<td>M</td>
<td>Occipital and suboccipital</td>
<td>Extradural</td>
<td>3 hrs.</td>
<td>Recovery</td>
</tr>
<tr>
<td>10</td>
<td>C.O.</td>
<td>9</td>
<td>M</td>
<td>Occipital and suboccipital</td>
<td>Extradural</td>
<td>4 days</td>
<td>Recovery</td>
</tr>
<tr>
<td>11</td>
<td>L.P.</td>
<td>33</td>
<td>F</td>
<td>Occipital and suboccipital</td>
<td>Subdural</td>
<td>7 days</td>
<td>Recovery</td>
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<tr>
<td>12</td>
<td>M.S.</td>
<td>5</td>
<td>M</td>
<td>—</td>
<td>Cerebellar</td>
<td>3 days</td>
<td>Recovery</td>
</tr>
<tr>
<td>13</td>
<td>H.S.</td>
<td>56</td>
<td>M</td>
<td>Occipital and suboccipital</td>
<td>Subdural</td>
<td>5 days</td>
<td>Recovery</td>
</tr>
<tr>
<td>14</td>
<td>E.S.</td>
<td>40</td>
<td>F</td>
<td>Occipital and suboccipital</td>
<td>Extradural</td>
<td></td>
<td>No operation; died second day after injury</td>
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<tr>
<td>15</td>
<td>J.S.</td>
<td>58</td>
<td>F</td>
<td>—</td>
<td>Cerebellar</td>
<td>10 hrs.</td>
<td>Continued coma; died eighth postoperative day</td>
</tr>
<tr>
<td>16</td>
<td>R.W.</td>
<td>47</td>
<td>M</td>
<td>—</td>
<td>Cerebellar</td>
<td></td>
<td>No operation; died 15 hours after injury</td>
</tr>
<tr>
<td>17</td>
<td>P.W.</td>
<td>17</td>
<td>M</td>
<td>—</td>
<td>Cerebellar</td>
<td></td>
<td>No operation; died 3 days after injury</td>
</tr>
</tbody>
</table>

fore the posterior fossa operation demonstrated separation of the torcular Herophili from the inner table of the skull (Fig. 1). Although this separation was small it suggested the presence of an extradural clot and at operation a hematoma measuring from 3 to 10 mm. in thickness was removed from this region on both sides.

Patient 6 had an unusual clinical course. She fell from a horse, striking her head on the ground. There was no loss of consciousness and shortly thereafter an occipital scalp laceration was sutured. She felt perfectly well until 5 hours later when headache, drowsiness, and vomiting gradually developed. On admission to the hospital after another 3 hours she was very drowsy and speech was slurred; she was, however, fully oriented and remembered clearly her accident earlier in the day. The left pupil was 1 mm. larger than the right but both reacted briskly to light and accommodation. There were extensor plantar responses on both sides. X-rays showed a left occipital skull fracture which extended from the region of the lambdoid suture downward to disappear behind the petrous pyramid. For several days her condition remained virtually unchanged, then headache lessened a little and she became more alert. By the 12th day after her fall, bilateral papilledema was noted although otherwise she seemed improved. Over the next few days the papilledema increased and a left abducens palsy developed. Finally, incoordination of the left limbs and horizontal nystagmus on left lateral gaze appeared. At suboccipital craniectomy on the 16th day after injury an enormous largely liquefied left
extradural hematoma was removed. No point of active bleeding could be detected. Recovery was complete.

Patients 4 and 14 died of posterior fossa extradural hematomas that were not diagnosed during life. The former, an 18-year-old man, was in an automobile accident after drinking heavily. On admission to the hospital shortly thereafter, he was drowsy but when aroused would shout and fight examiners vigorously. On one occasion he tried to run from the Emergency Ward. A large subgaleal hematoma was present in the occipital region and x-rays demonstrated an underlying fracture extending just to the left of the midline downward into the foramen magnum. Left corneal sensation seemed less than the right and there was slight weakness at the left corner of the mouth. Both plantar responses were upgoing but deep tendon reflexes, strength and tone in the extremities seemed within normal limits. His condition remained the same for 9 hours, then the left pupil dilated but otherwise vital signs and state of consciousness remained unchanged. Twenty-five minutes later the right pupil dilated and pulse and respirations ceased. Autopsy showed an egg-sized extradural hematoma overlying the left cerebellar hemisphere and a large pontine hemorrhage (Fig. 2). Supratentorially there were only a few scattered contusions on the orbital surfaces of both frontal lobes.

Patient 14 died abruptly from a similar lesion. She sustained occipital and suboccipital fractures when she fell from an automobile skidding on ice. At first she had severe headache and was slightly confused but neurological examination was otherwise normal. Lumbar puncture 10 hours after injury showed an initial pressure of 256 mm. and cerebrospinal fluid contained 3,000 RBC's per cu. mm. The confusion cleared and her condition seemed quite satisfactory. At 11:00 p.m. on the second hospital day her condition was unchanged. Forty minutes

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Fig. 1A. Patient J. B., Case 2. Lateral view of venous phase of right carotid angiogram. The slight separation of the torcular Herophili from inner table of the skull suggests the presence of a posteriorly situated extradural hematoma. At operation the hematoma was found to be much thicker laterally.

Fig. 1B. Plain skull x-ray, Case 2, showing inverted V-shaped fracture extending downward into the suboccipital region on both sides.

Fig. 2. Large pontine hemorrhage found at autopsy in Patient J. D., Case 4. Twelve hours after injury this patient rapidly deteriorated and expired. At postmortem examination there was a large extradural hematoma compressing the left cerebellar hemisphere.
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later she was found dead. Post-mortem examination again showed an extradural hematoma confined to the posterior cranial fossa.

These two tragic deaths could probably have been prevented had suboccipital hematomas been suspected and removed early in the clinical course. Patients with fractures over the posterior fossa must be observed constantly, for, as these two cases illustrate so vividly, deterioration can occur with great rapidity and only a prompt operation will save them.

Subdural Hematomas

Infratentorial subdural hematomas probably occur less frequently than do extradural hematomas. Their true incidence is unknown but they occurred in 1 of 62 cases of subdural hematoma reported by Munro and in 2 of 389 reviewed by McKissock et al. Ciernoniewicz has recently added 3 cases. Single cases of acute subdural hematomas in the posterior cranial fossa were reported by Coblenz (birth injury) and Webster et al. (penetrating cerebellar wound). Fisher et al. reported 4 cases caused by head injury. Chronic subdural hematomas below the tentorium are truly rare. Achslogh reported an interesting case in which there had been no head injury, but symptoms of a posterior fossa mass lesion for more than a year. One other case of posterior fossa chronic subdural hematoma probably resulted from trauma at the time of mastoidectomy rather than other injury.

In our series, 4 patients (Nos. 1, 5, 11 and 13) had acute subdural hematomas in the posterior cranial fossa. In 1 patient (No. 8) the lesion was subacute. We have not seen the chronic form in the posterior fossa. All 4 patients were operated upon but patients 1 and 5 did not survive.

Patient No. 1 might have fared better had operation been done sooner. This 44-year-old man, a long-standing alcoholic, lived alone and accurate history was not available. A physician outside the hospital saw him earlier on the day of admission, found him to be drowsy and tremulous, and administered Librium, 100 mg. intravenously and 50 mg. by intramuscular injection simultaneously. On arrival at the Emergency Ward several hours later the patient was drowsy and would not obey commands or speak although he withdrew all limbs vigorously from painful stimuli. Lumbar puncture showed an initial pressure of 130 mm. and cerebrospinal fluid contained 200,000 RBC’s and 500 WBC’s (50 per cent neutrophils) per cu. mm. The initial impression was Librium intoxication.

On the next day he became less responsive and on 3 or 4 occasions respirations ceased for 30 to 45 seconds but always resumed spontaneously. That evening skull x-rays were made which showed a right occipital fracture extending into the foramen magnum. Bilateral suboccipital craniectomy the same evening revealed a small extradural hematoma measuring 3 mm. in thickness on the right side under the fracture site. This was thought insignificant. On opening the dura the surgeon found that a large hematoma filled the vallecula and compressed the right cerebellar hemisphere laterally. Both cerebellar hemispheres were contused and swollen. The patient did not improve. The day after operation he was deeply comatose and the right pupil began to enlarge. A right carotid arteriogram showed only a slight shift of the anterior cerebral artery, compatible with traumatic edema inasmuch as the middle cerebral branches were in normal position. Despite institution of moderate hypothermia (92 to 95°F.) and steroids, the patient did not improve and died the next day. Autopsy showed multiple small brain stem hemorrhages and contusions of the right frontal lobe.

Patient 5, the only case of infratentorial subdural hematoma in whom skull fracture was not present, was moribund immediately following an automobile accident. On arrival at the Emergency Ward respirations were periodic and shallow, systolic blood pressure was 30 mm. Hg., and a pulse was unobtainable. She was unresponsive to painful stimuli, the pupils were small but fixed, and the muscle tone was increased in all limbs. After bilateral temporal and parietal burr holes showed only contused edematous brain, exploration of the posterior fossa revealed a layer of clotted blood, 3 to 8 mm. in thickness, over both cerebellar hemispheres. This was removed, but she died an hour later.

Patients 11 and 13 were more fortunate. Both were alert with normal neurological examinations when first seen but were admitted
for observation after x-rays showed skull fractures. Patient 11 had fallen from a horse, was unconscious for a few minutes, and then awoke. Patient 13 had had idiopathic epilepsy for many years and sustained occipital trauma when he fell backward during a seizure. The fracture in this case is shown in Fig. 3. Both patients had increasing headache and somnolence, and posterior fossa explorations were performed 5 and 7 days respectively after injury. In each case, massive amounts of liquefied dark subdural blood were removed. Both patients had uneventful recoveries.

Patient 8 is unusual in that the hematoma developed following a fall 4 weeks after removal of a cerebellar astrocytoma. He had recovered well from the posterior fossa exploration. Ten days after returning home he fell backwards while shaving one morning, struck the operative site on the edge of the bathtub, and was unconscious for a brief period. In the days that followed he noted severe headache, neck stiffness, diminished vision and swelling at the operative site. Papilledema recurred and he had horizontal nystagmus on lateral gaze to both sides. On readmission to the hospital, lumbar puncture showed an opening pressure of 170 mm.; cerebrospinal fluid was the color of port wine yet it contained only 11,000 RBC's and 1,100 WBC's (92 per cent neutrophils) per cu. mm. Bacterial cultures were negative. Pneumoencephalogram outlined an oval plum-sized mass outside the cerebellum (Fig. 4) displacing it and the 4th ventricle anteriorly. At re-exploration 2 months after the first operation and 1 month after the fall, a hematoma was exposed and removed. Recovery was uneventful.

Arteriography was not carried out preoperatively in any of these cases of suboccipital subdural hematomas. One might suspect that such lesions would be outlined on air study and by displacement of small cerebellar vessels on the lateral view of vertebral arteriograms. We felt the delay caused by these procedures constituted a greater risk than that of diagnostic suboccipital burr holes.

Traumatic Intracerebellar Hematomas

Reports of hematomas within the cerebellum following trauma are also rare. Early accounts by Mackenzie,\textsuperscript{13} Echols\textsuperscript{8} and Hagib\textsuperscript{12} all followed such mild cranial trauma that Schneider\textsuperscript{24} thought that they all might have been spontaneous hemorrhages. He himself reported 4 cases of intracerebellar hematoma due to occipital trauma. All had skull fractures extending into the posterior cranial fossa and 3 of the 4 also had major contra-coup hematomas above the tento-

![Fig. 3. X-ray of skull in Patient H. S., Case 13, showing left occipital fracture extending inferiorly into foramen magnum.](image)

![Fig. 4. Patient R. H., Case 8. Pneumoencephalo-gram showing post-traumatic subdural hematoma that occurred following occipital trauma. Normal posterior fossa structures are displaced anteriorly. A cerebellar astrocytoma had previously been removed.](image)
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rium. Schneider stressed, therefore, that in cases of occipital trauma, every effort should be made to rule out major collections of blood above the tentorium as well as in the posterior fossa. Fisher et al. noted 2 fatal cases of intracerebellar hemorrhage that occurred in a series of 98 patients with fractures of the occipital bone. Gurdjian, reporting a recent series of 38 traumatic hematomas within the brain, mentioned that 2 of these were intracerebellar.

Traumatic intracerebellar hematomas occurred in 6 patients (Nos. 3, 7, 12, 15, 16, and 17) in our series. Proper diagnosis and evacuation of the clot saved the lives of patients 7 and 12.

Case 7 had the longest interval between injury and operation for hematoma of any patient in this series. Three months prior to admission, this patient, a 15-year-old boy, was struck on the occiput by a baseball bat. At first he was stunned but did not lose consciousness. Within a day or two, recurring severe frontal headaches began. Mental deterioration with marked decline in the quality of his school work ensued. Finally, intractable headache, vomiting and ataxia of the left limbs led to hospitalization. Papilledema and nystagmus were not present, and x-rays showed no fracture. A ventriculogram showed displacement of the aqueduct and 4th ventricle to the right, and at operation a very large liquefied hematoma was evacuated from the center of the left cerebellar hemisphere. Recovery was complete.

Patient 12 struck the back of his head when he fell from his tricycle onto the sidewalk. He, too, was not unconscious and continued playing. However, several hours later, headache, lethargy and vomiting began. On admission to the hospital 2 days later, he was moderately lethargic, but otherwise the neurological examination was normal. Skull x-rays showed no fracture. Lumbar puncture showed blood-tinged xanthochromic fluid under a pressure of 230 mm. By the next day, papilledema was present, and there was nystagmus on left lateral gaze. In addition, tone now seemed increased in all limbs. Bilateral supratentorial burr holes showed no abnormality, and fortunately the surgeon proceeded with suboccipital burr holes. On finding a severely confused and swollen left cerebellar hemisphere, the bony opening was enlarged and a clotted hematoma within the left cerebellum was removed. The patient recovered completely.

Case 15 is the only instance of possible contra-coup cerebellar hematoma in this series. She had fallen, striking her forehead. Regrettably, operation was undertaken only after the patient was moribund and spontaneous respirations had ceased and the patient died.

Patients 3, 16 and 17 were not correctly diagnosed during life. The first of these, an elderly lady comatose after a head injury, had carotid arteriograms which showed no evidence of a supratentorial hematoma. Despite a large occipital scalp ecchymosis and posterior fossa skull fracture, no effort was made to investigate this region. She died the day after admission. A left cerebellar hematoma was found at autopsy.

Patient 16 was knocked to the ground by a falling truck tail gate. At first he was alert but within 2 hours drifted into coma. Bilateral supratentorial burr holes showed no abnormality, and the cerebral hemispheres were not swollen. He died 12 hours later. At autopsy a massive right cerebellar hematoma was found and only minor lacerations of the frontal lobes. This case occurred in 1955 when our interest and experience with posterior fossa hematomas was scant. It is unlikely that such a lesion today would escape diagnosis and treatment.

Patient 17 had a similar course. He was briefly unconscious after falling backward on ice but thereafter remained alert for a half hour. He then became lethargic and finally unresponsive to painful stimuli. X-rays showed no fracture. Lumbar puncture showed pink cerebrospinal fluid under pressure greater than 300 mm. During this maneuver spontaneous respirations ceased. Bilateral frontal and temporal burr holes showed no hematoma, and a respirator was used. Papilledema was noted the following day. He remained flaccid and without spontaneous respirations until death 3 days after injury. Autopsy showed large bilateral intracerebellar hematomas (Fig. 5). This case illustrates once more the great danger of lumbar puncture in the presence of rapidly expanding lesions in the posterior cranial fossa.

Discussion

Despite increasing cognizance of traumatic tentorial hematomas over the past 12 years,
were caused by injury directly to the occipital region. That case, patient 15, may have
developed the intracerebellar hematoma from contra-coup injury when she fell striking her forehead.

There were posterior fossa skull fractures in all 6 cases of extradural hematoma, in 3 of the 4 cases of subdural hematoma, but in only 1 of 6 cases of intracerebellar hematoma. This last fact perhaps deserves emphasis, for all the cases of traumatic intracerebellar hematoma reported by Schneider and Fisher et al. had overlying skull fractures. Moreover, no patient in our series had a significant supratentorial hematoma.

The common finding in all these 17 patients was diminished consciousness. Those able to speak all had severe headache. When diagnosis was delayed either because of our unfamiliarity with the lesion or because the hematoma was increasing in size very slowly, papilledema and 6th cranial nerve palsies were frequently present. Cerebellar ataxia developed only when the lesion had been present for some time as in Cases 6, 7 and 12.

In patients whose clinical course resembles that outlined above and in whom no evidence of supratentorial hematoma can be found, posterior fossa hematomas should be considered. This is particularly true if there has been occipital trauma and if an associated posterior cranial fossa fracture is present. The absence of a fracture, however, certainly does not rule out an infratentorial hematoma. Patients who remain drowsy after occipital trauma must be carefully observed, for rapid deterioration and death can develop rapidly. In the absence of a simple diagnostic test, the use of exploratory suboccipital burr holes followed by craniectomy when necessary is the only reliable method of handling this problem.

**Summary**

Infratentorial hematomas caused by trauma are much less frequent than those located above the tentorium and consequently have received little attention. These lesions, whether extradural, subdural or intracerebellar, are usually caused by occipital trauma. Acute forms are difficult to diagnose during life because of the paucity of clinical findings other than alterations in the state of consciousness. In some of our cases a long

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**Fig. 5:** Post-mortem specimen showing bilateral traumatic intracerebellar hematomas not suspected during life in Patient R. W., Case 16.

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one-third of the cases in our series were not diagnosed during life, and the results were fatal. Two other patients were allowed to deteriorate to a moribund state before sub-occipital explorations revealed hematomas. Another seriously injured patient continued a downhill course and died shortly after removal of the subdural hematoma. The remaining 8 patients, operated upon before irreversible neurological damage had occurred, all made virtually complete recoveries.

Ages of patients in this series ranged from 5 to 74 years suggesting that no particular age group is more prone to develop this complication of trauma. With one exception, all the posterior fossa hematomas in this series...
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lucid or stable interval was followed by rapid deterioration and death. Subacute or chronic hematomas gave rise to signs and symptoms of posterior fossa mass lesions. Suboccipital skull fractures were usually present in cases of extradural and subdural hematoma but usually absent when the hematoma was within the cerebellum.

References