of this balance results in the precipitation of grave symptoms of increased intracranial pressure. In both of the cases reported here, the precipitating factor was trauma.

No definite statements as to the vermis can be made in the 2 cases here reported. Certainly it was smaller than normal in both cases but in neither case was inspection very prolonged.

The surgical treatment of atresia of the foramina of Luschka and Magendie has been attended by poor results as in only 3 previously reported cases have the patients been cured. Certainly operation should be performed as soon as possible in order to prevent cortical atrophy. Operation at an early date in Case 1 would surely have prevented much cortical atrophy and permitted a better operative result. The patient may have to be tided over a crisis by ventricular puncture but surgery should be performed as soon as the patient's condition permits. Excision of the membrane should be as wide as possible but no attempt should be made to remove it in its entirety. Such an attempt avails nothing and immeasurably hazards the operation.

SUMMARY

1. Two cases of surgical cure of atresia of the foramina of Luschka and Magendie are reported.
2. The absence of the typical roentgenologic findings is noted and the possible mechanism of such absence cited.

REFERENCES


BILATERAL EXTRADURAL HEMATOMA

REPORT OF CASE*

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Extradural hemorrhage has been thoroughly reviewed by Roy, Wiesmann, Kennedy and Wortis, Pringle, Munro and Maltby, Gurdjian and Webster and others. One of the earliest cases of middle meningeal hemorrhage recorded in the literature is an example of

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bilateral extradural hematomas. The patient was operated on by Hill in 1750. Since that time, there have been other instances of bilateral extradural hematomas. King in 1943 adequately expressed the frustration experienced by evacuating an extradural hematoma on one side, only to find an extradural hematoma on the contralateral side at necropsy. We feel that the following case is worthy of presentation in order to emphasize the importance of bilateral exploration in cases of acute injury of the head in which the presence of extradural bleeding is suspected.

REPORT OF CASE

A 23-year-old man was admitted in an unconscious state to the U. S. Naval Hospital Bethesda, Maryland on July 7, 1946. His mother said that he had fallen from a motorcycle about 30 hours before his admission and that he had not regained consciousness. He had been treated immediately after the accident in another hospital by means of sedation and oxygen. A note from this hospital stated that the patient had bled from the nose and the left ear and that gross blood had been present in the urine. It also stated that the patient had reacted sluggishly to stimulation in the first few hours after the injury and that he had not regained consciousness. Reluctance to use the right arm and leg had been noticed.

The patient was obviously seriously injured. The axillary temperature was 104°F., the pulse rate 160, the respiratory rate was 24, and the blood pressure was 150 mm. of mercury systolic and 90 mm. diastolic. He groaned continually, he repeatedly threw himself around in a purposeless manner and he rubbed his head with his left hand. His state of consciousness was markedly impaired. There were a few minor lacerations of the left temporoparietal region and numerous lacerations of both arms and legs. The left eye was swollen and ecchymotic. Both pupils reacted to light and were equal in size. Crusted blood was seen in the left external auditory canal. Hemiparesis was present on the right side; all deep tendon reflexes were hyperactive but bilaterally equal. There were no pathologic reflexes. The remainder of the general physical and neurologic examinations did not reveal any abnormality.

Urinalysis revealed numerous erythrocytes. The results of roentgenographic examination of the skull were unsatisfactory owing to the patient's restlessness. There seemed, however, to be fractures of the cranial vault. It was the impression of the neurosurgical staff that the patient presented the picture of increasing intracranial pressure with decompensation. A bilateral exploration with local anesthesia was considered advisable. A blood transfusion was started and oxygen therapy was instituted.

The patient was taken to the operating room and his head prepared in the usual manner. A bilateral subtemporal decompression was performed through linear incisions by one of us (C. S. M.). The operation on the left side was done first because of the presence of hemiparesis on the right side. A fracture was apparent in the left frontotemporal area. The fracture had severed the middle meningeal artery and had produced a moderate-sized hematoma which extended toward the sphenoid ridge but did not go over the frontal or temporal lobes. After the clot had been evacuated and the middle meningeal artery had been coagulated, the dura was opened. Blood was present in the subarachnoid fluid and there was edema of the hemisphere. The patient's condition did not improve appreciably. On performing the right subtemporal decompression, it was found that a fracture had injured the middle meningeal artery. There was active bleeding from this artery and an enormous extradural clot extended over the hemisphere and under the frontal lobe to the sphenoid ridge. The clot had caused marked deformity of the entire hemisphere. The clot was evacuated and the artery coagulated. On opening the dura, it was noticed that the hemisphere did not appear edematous and that there was no blood in the subarachnoid fluid. Both craniotomies were enlarged to allow adequately for the cerebral edema, particularly of the left hemisphere. The dura was opened widely, and the defect covered with animal membrane. It was felt that the patient had a fair chance of survival if the damage to the brain had not been severe. Postoperatively, the patient improved rapidly. Twenty-four hours after operation, he was alert but restless. The remainder of the postoperative course was uneventful, and the patient was dismissed on Aug. 12, 1946.