CONCLUSIONS

1. When local tissue deficiency precluded a successful autoplastic repair in a patient with a simple lumbar meningocoele, the imbrication of lumbar fascia over tantalum gauze effected a solid repair.

2. The closure of a nasal meningocoele is reported using tantalum gauze to cover the cranial dehiscence, and to support the overlying dura.

REFERENCES


EXTRADURAL CEREBELLAR HEMATOMAS

REPORT OF TWO CASES*

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Extradural hematoma is a lesion almost universally resulting in fatality when not attacked surgically, yet recovery should be the rule in properly managed cases. The symptom complex of such lesions occurring above the tentorium is generally well recognized and treated. On the other hand, only 7 cases were found in the literature in which extradural hematoma occurring below the tentorium had been recognized and cured to date. The successive occurrence of 2 such cases without localizing cerebellar signs prompted this report. Both cases terminated fatally. It is hoped this experience may be of value in raising the "suspicion index" in cases where seemingly minor trauma to the occiput has been sustained.

In 1901, Wharton reviewed 70 cases of intracranial venous sinus wounds, finding 8 instances of bleeding in the posterior fossa. Anderson, however, in reporting a successfully treated case, noted that no examples of this lesion had appeared at the Los Angeles County Hospital during the preceding 14 years, where an average of 2000 head injuries are seen each year. Gurdjian and Webster, Kessel, Turnbull, Coleman and Thomson, Grant and Austin, Anderson, and Bacon have each reported 1 case of extradural cerebellar hematoma successfully evacuated by operation.

Head trauma, particularly a blow to the occiput, usually initiates the lesion. The probable mechanism of injury is a laceration of some portion of the lateral sinus, with subsequent escape of blood downward into the potential epidural space of the cerebellar fossa. A fracture line will most generally be found crossing the lacerated sinus. It would appear that extradural cerebellar clots may be formed acutely or may accumulate more slowly.

Headache, stiff neck, vomiting, and irritability are generally found in those patients not unconscious from the blow. In acute cases the early onset of coma may preclude neurologic evidence which would otherwise correctly direct one's attention to the posterior fossa. Such individuals may exhibit hypotonia, positive

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Babinski signs and little else before spontaneous respirations cease. Should bleeding progress more slowly, the drowsy, though conscious patient may exhibit papilledema, nystagmus or ataxia. It should be emphasized that the symptomatology of these clots is notoriously unreliable. Additional neurologic findings, such as hemiparesis, unequal deep reflexes and unequal pupils may erroneously lead the observer to other intracranial localization. Such was the situation in the first instance of such a clot seen by the author.

**CASE REPORTS**

*Case 1.* J.L., a 20-year-old white female, was injured when she was thrown from a horse on Mar. 29, 1950, and probably kicked in the back of the head. When carried to a nearby ranchhouse she was unconscious, but soon could respond to commands. She complained of severe headache, and pain in the neck. The family physician was called, and found the patient semiconscious, with a stiff neck, equal pupils, and vital signs that were within normal limits. Codeine was left to control the pain.

She failed to improve and was taken to the local hospital some 12 hours later. A lumbar puncture was done, the fluid being reported grossly clear, and the pressure "normal." Portable lateral x-rays of the skull revealed no fracture. The patient continued to moan with pain and thrash about in bed. Forty hours after injury she was transferred to a hospital in this city, because her local physician felt she did not seem to be doing well.

*Examination.* The patient was comatose, lying restlessly in bed and moving all extremities equally. Painful stimuli elicited appropriate defense reactions. No abrasions nor lacerations of the scalp were noted, though a Battle's sign was present on the left. Attempted flexion of the head demonstrated boardlike resistance of the neck. There was no bleeding from the cranial orifices. The pupils were equal and reacted to light. The eyegrounds were normal. The deep tendon reflexes were equal, but there was a positive Babinski on the left. BP 116/78, pulse 86, respirations 22, temperature 99.6°.

*Course.* Feeling that the patient was suffering from a left basal skull fracture with cerebral contusion, and subarachnoid bleeding, a conservative course was decided upon. Sedation was withheld in order that changes in the state of consciousness and vital signs could be followed more accurately. Restlessness continued for 2 hours. At this time, and for no apparent reason, the patient sat upright in bed, became cyanotic and fell backward with markedly irregular pulse and respirations. Within 5 minutes she was dead.

*Autopsy.* There was a 7 cm. linear fracture curving from the left lateral sinus to the left jugular foramen (Fig. 1). Associated with the fracture site was an extradural collection of liquid blood and clot estimated at between 30 cc. and 40 cc. in amount. The adjacent left lobe of the cerebellum was moderately compressed and there was herniation of the cerebellar tonsils, more marked on the right side. A contrecoup small linear fracture of the right frontal region with minimal subdural hemorrhage was present. There were contusions and superficial areas of laceration of the tip of the right frontal and parietotemporal lobes with small areas of subarachnoid hemorrhage. Multiple sections through the entire brain showed only moderate edema of the brain stem and slight softening of the cerebellum. There was no intraventricular bleeding.

This patient probably could have survived, had the proper diagnosis been sus-