SUMMARY

Two cases of laminar fractures of the cervical vertebrae, occurring near the intervertebral foramina and complicated by root compression were reported. The close resemblance of the clinical picture to that of a ruptured cervical disk was emphasized. Diagnosis, differential diagnosis and treatment were outlined.

REFERENCES

TWO CASES OF ACUTE SUBDURAL HYGROMA SIMULATING MASSIVE INTRACRANIAL HEMORRHAGE

EVERETT H. DICKINSON, M.D., AND BERNARD H. PASTOR, M.D.
Neurosurgical Service, Philadelphia General Hospital, Philadelphia, Pennsylvania

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The occurrence of subdural collections of fluid was mentioned by Cushing in 1908. In his recent review, Wycis recorded a total of 99 cases of subdural hygroma, including 7 of his own. Naffziger discussed subdural collections of fluid, and observed that they could occur in the first few hours after injury as well as in the later days. Most of the cases in the literature have been of the more chronic variety. Cohen recorded an interesting case in which operation was performed 6 weeks after the initial injury, and during the succeeding 4 months, the subdural space was emptied 9 times, yielding a total volume of 2,300 cc. of fluid. Such recurrences appear to be rare. Scott recorded 3 cases in which prolonged stupor, varying from 23 to 28 days, was the outstanding symptom, and McConnell reported 6 cases with post-traumatic amnesia of 6 to 66 days' duration.

Of the acute variety which appears to occur less frequently, Walsh and Shelden each reported a similar case in which rapid onset of symptoms following head injury led to a pre-operative diagnosis of epidural hemorrhage. A total of 5 such acute cases were reported by Lanigan and Haynes. In all of these, clear fluid was found in the subdural space at operation. In a more extensive report, McConnell recorded 7 cases in which the patient was deteriorating rapidly, usually within 48 hours of injury, in which subdural collections of fluid were found, as well as 92 cases in which the course was more chronic. All of his cases, he felt, were clinically indistinguishable from massive intracranial hemorrhage as it is commonly observed. Da Costa and Adson had a considerable series of 19 cases, and they pointed out that symptoms may come on immediately, or may be delayed a period varying from hours to weeks. Three of Wycis' 7 cases were of the acute variety.

We have recently observed, within a short period of time, 2 cases in which, following a head injury, there was more or less acute onset of symptoms suggesting massive intracranial hemorrhage. In both cases, exploration revealed large collections of xanthochromic fluid in the subdural space.

CASE REPORTS

Case 1. H. C., a 66-year-old colored male, was admitted to the service of Dr. S. Gilpin at the Philadelphia General Hospital, at 7:15 p.m. on June 11, 1946. Earlier in the afternoon, the patient had been thrown from a wagon, knocked unconscious, and found lying on the highway by the police. When seen in the ward, he had regained consciousness, but was some-
what confused and disoriented. Bloody spinal fluid was leaking from his right ear. Pupils were equal and reacted to light, and examination of the fundi revealed only vascular changes. The only abnormal neurological findings were a positive Babinski sign on the right, and marked deviation of the tongue to the right. General physical examination was negative except for an apical systolic murmur. His temperature was 101°F., pulse 70, respiration 20, and blood pressure 140/80.

An hour after admission, during a period of about 20 minutes, the patient became rapidly more confused and lapsed into unconsciousness. He then had 3 generalized convulsions at approximately 5-minute intervals. The right pupil became dilated, and reacted sluggishly to light. A left hemiparesis developed. The patient’s respiration became deep, slow, stertorous, and later of the Cheyne-Stokes variety. When operation was decided on at 8:30 P.M. he was deeply comatose. A diagnosis of epidural hemorrhage was made.

Exploratory trephining was done on the right over the middle meningeal artery. No epidural bleeding was found, but the dura, although normal in color, was tense and did not show the usual pulsation. Upon incising the dura, there was a gush of slightly blood-tinged straw-colored fluid under considerable pressure. An estimated 200 cc. of fluid were evacuated, and the intact arachnoid could then be seen lying deep to the dura and pulsating. At this time, the patient awoke and began to struggle and protest loudly. It was necessary to use intravenous sodium pentothal anesthesia to complete the operation. The patient left the operating room conscious and in good condition. Postoperatively his temperature was 100°F., pulse 80/min., respirations 20/min., and blood pressure 130/70. At 12:00 midnight he was fully conscious.

The following day (June 12) his condition was excellent, he was fully conscious, and fairly well oriented. By June 14, he was out of bed in a wheel chair. On June 18, there was still slight nuchal rigidity, but this was attributed to concomitant subarachnoid hemorrhage. Spinal fluid culture was negative. On June 21, the spinal fluid was still bloody. It was xanthochromic on June 23, and the neck was no longer stiff. On July 7, crystal-clear spinal fluid was obtained and studied. There were no abnormal findings. At no time was there any regression in the mental picture, and the patient was discharged asymptomatic on July 4, 1946. When seen in the follow-up clinic a month later, the tongue still deviated markedly to the right.

Comment. This case represents one in which rapidly progressive signs of increasing intracranial pressure led to a diagnosis of epidural hemorrhage. The dramatic recovery immediately after evacuation of a large subdural effusion leaves little room for doubt that it was producing the symptoms.

Case 2. H. R., a 67-year-old colored male, was admitted to the service of Dr. W. McConnell at 11:00 p.m. on July 25, 1946. He had fallen from a 9-foot-high scaffolding at 2:00 p.m., was unconscious for a few minutes, and subsequently behaved in a confused manner all afternoon, and up to the time of admission. When first seen, he was confused, imperfectly oriented, and insistent that he wanted to go home at once. His pulse was 100/min., respirations 20/min., blood pressure 170/100, and temperature normal. There was a soft, tender swelling in the right parietal region, and a scalp abrasion on the occiput. Pupils were normal and reacted to light and accommodation. No papilloedema was noted. General physical examination was essentially negative except for the hypertension, an apical systolic murmur, and a loud "tambour" aortic second sound. Neurological examination at this time was negative. A diagnosis of cerebral concussion and hypertensive cardiovascular disease was made.

The patient was apparently well during the next 48 hours, except for the persistence of mild confusion. He was quiet and alert, and very cooperative. Skull x-rays taken on admission showed no evidence of fracture.

On July 28, at 3:00 a.m., the patient was noted to be having Jacksonian convulsions involving the left arm, leg and face. He appeared to be conscious, but was more confused. The convulsions were almost continuous, but during a momentary cessation of convulsions, there were definite left hemiparesis and left facial weakness. The pulse was 120/min., respiration rapid and shallow with periods of Cheyne-Stokes, and blood pressure 170/100. Lumbar punc-
ture showed grossly bloody fluid under a pressure of 110 mm. of water. At 5:00 A.M. the patient had convulsive movements of the right arm and leg.

Operative intervention was considered but delayed until later in the day (Sunday) when it became apparent that there was no improvement in the patient's condition. With a preoperative diagnosis of acute subdural hematoma, 2 trephine openings were made on the right side. There was no epidural bleeding, and the dura was normal in appearance. When the dura was incised, there was a spurt of straw-colored fluid, and a copious amount, estimated at about 250 cc., escaped through the dural opening. After evacuation of the fluid the intact arachnoid could be seen pulsating deep to the dura. The patient's respiration improved immediately. He was returned to the ward still convulsing, but obviously much improved.

The following morning, July 29, the patient was mentally much clearer. He still had generalized clonic movements, but they were diminished in intensity. His temperature was 102°F. Although given penicillin and sulfadiazine, he continued to run a febrile course with temperature from 101 to 104°F, which fell by lysis until it was normal on August 5. Convulsive movements continued, controlled somewhat by sodium phenobarbital, until August 2, when they ceased spontaneously.

On August 3, the patient was fully conscious, and although somewhat disoriented as to time and place, answered questions well. He had no headache, although he complained of some dizziness. There were no abnormal neurological signs. The pulse, respiratory rate, and temperature were normal, and the blood pressure was 130/80, at which level it remained. By August 7, he was well oriented and alert although he had complete amnesia for his accident. At this time a urinary tract infection developed and the temperature rose to 104°F. This was rapidly brought under control, and after this his temperature remained normal. There was no regression in his mental state or neurological picture during this episode.

He was discharged well to be followed in the out-patient department.

Comment. Although the response was not quite as dramatic in this case as in the preceding one, the improvement in the patient immediately after removal of the subdural collection was quite striking. His rather stormy course we attribute to the concomitant cerebral concussion he must have suffered as evidenced by the grossly bloody spinal fluid.

DISCUSSION

The mechanism by which subdural collections of fluid form acutely is not clear. The most generally accepted explanation is that they result from cerebrospinal fluid pouring into the subdural space through a tear in the arachnoid. According to Da Costa and Adson this tear is usually in the region of the Sylvian fissure or the optic chiasm. Resistance to flow is less through the tear than over the cortex, and fluid continues to accumulate as long as the brain can be displaced or compressed. The fluid is locked in the subdural space by pressure of the edematous brain against the arachnoid opening. The fluid cannot be removed via the spinal subarachnoid route, and in many cases the pressure as measured by lumbar puncture is normal.

This condition is apparently a distinct clinical entity, and although not usually so recognized, it should be classified with the other traumatic expanding intracranial lesions.

SUMMARY

In 2 cases of head injury recently observed, the acute onset of signs of a rapidly progressive cerebral lesion led to a diagnosis of massive intracranial hemorrhage. In both cases at operation, no hemorrhage was found, but a large collection of clear fluid was present in the subdural space which undoubtedly accounted for the symptoms. In both cases there was striking improvement following evacuation of the fluid.

Although most of the cases of subdural hygroma reported are of the more chronic variety, there are reports in the literature of acute subdural hygromas such as those described here. The probable mechanism of their formation is the extravasation of fluid from the subarachnoid space into the potential subdural space through a tear in the arachnoid.
REFERENCES


A SIMPLE AND INEXPENSIVE LAMINOGRAPH
FOR USE IN NEUROSURGERY

WILLIAM BEECHER SCOVILLE, M.D., AND GERHARD DANELIUS, M.D.*

Hartford Hospital, Hartford, Connecticut

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Laminography has great value in the visualization of the 3rd and 4th cerebral ventricular systems and, to a lesser extent, certain sinus, skull and cervical spine areas. Nevertheless, the majority of neurosurgeons have failed to include a laminograph in their diagnostic armamentarium because of the false belief that such machines are both expensive and complicated. In an effort to dispel this illusion, a brief description of an easily constructed, hand-operated laminograph is described and illustrated. Such a model was built in the local machine shop of the x-ray and neurosurgical departments of the McCaw General Army Hospital at a total cost of $22.50. A similar power-driven machine is now manufactured by the Franklin X-ray Company, Philadelphia (Fig. 2). No claim for originality is made for the described model, as similar machines have been variously described and more frequently constructed throughout Great Britain and the United States during the past ten years.1, 2, 3, 4, 5

Construction can be carried out easily by any machinist and consists of a straight steel rod which connects the x-ray tube arm to the movable Bucky diaphragm. This rod passes through a fulcrum or point of axis of rotation which is attached to the x-ray table. Both the rod and fulcrum are held in place by thumb screws and are quickly removable. The height above the x-ray table of this axis of rotation can be varied manually by turning a worm screw. As a result, the manual moving of the tube arm in one direction causes the film to move in the opposite direction so that all objects in one selected plane will occupy the same position, thereby putting objects in all other planes out of focus. The arm is pushed sufficiently slowly to cause an approximate 4-second exposure when the tube describes a 60 to 90 degree arc over the area being x-rayed.

* Roentgenologist, Beverly Hills, California.