Chronic Venous Epidural Hematoma at the Vertex

GEORGE C. STEVENSON, M.D., HOWARD A. BROWN, M.D., AND WILLIAM F. HOYT, M.D.
Division of Neurosurgery and Department of Ophthalmology, University of California San Francisco Medical Center, and Department of Neurosurgery, Franklin Hospital, San Francisco, California

The fact that epidural hemorrhage constitutes an immediate threat to life has been accepted since the early reports of Hill18 (1772) and of Cock21 (1842). Callender,7 in 1867, wrote that all treatment of epidural hemorrhage was hopeless and advised against futile trephination of the skull. Gross23 (1873), however, basing his opinion on his experiences during the Battle of Shiloh on April 6, 1862, urged immediate evacuation of extradural hematomas by trephination. Gross, and later Erichsen17 in 1878, believed that this epidural bleeding was of venous origin. Jacobson38 (1886), while agreeing with Gross on treatment, suggested an arterial origin of these hematomas in his classical treatise.

In 1912, the anatomist, Jones,39 demonstrated that the grooves in the inner table of the parietal bones are formed by the middle meningeal veins or sinuses, not by arteries. His studies at necropsy of 3 cases of epidural hemorrhage revealed lacerations of these veins; the middle meningeal arteries were not injured.40 Vance,72 during his tenure as medical examiner of New York City, compiled a list of an impressive number of epidural hematomas which included examples of lacerations of the venous sinus. Rowbotham66 considered bleeding from diploic veins as the most frequent cause of epidural hemorrhage. Ver Brugghen,74 Reichert and Morrissey,64 Gurdjian and Webster,25 King and Chambers44 and others11,17,19,34,42,80 also have emphasized the various venous sources of epidural bleeding.

The literature contains several reports of intracranial hemorrhage following laceration of the superior sagittal, straight and transverse venous sinuses.6,10,33,45,56,58,63,65,66,67,79 Subsequent to the beautifully illustrated and documented papers of Wharton77 and Holmes and Sargent36 dealing with injuries to the superior sagittal sinus, Gurdjian and Webster67 collected all existing reports of extradural hematomas after injury of the dural sinus. The first report of an epidural hematoma that compressed the superior sagittal sinus was that which occurred in a "dragoon . . . wounded at the battle of Salamanca by a musket-ball in the body, which caused him to fall from his horse, injuring the top of his head."77 Operative findings of epidural hematomas that have stripped the superior sagittal sinus from the inner table of the skull have been described by Reichert and Morrissey64 and Raaf.63 Rowbotham has published a color plate of such a lesion.66

The initial report of a venous epidural hematoma in the posterior fossa is credited to McKenzie49 (1938) although lacerations of the lateral venous sinuses with resulting hemorrhage which "pressed in the cerebellum" were noted by Vance73 in 1927. Three years after McKenzie's report, Coleman and Thompson44 reported the first successful removal of a similar epidural hemorrhage in the posterior fossa of a 9-year-old boy. Epidural bleeding in the posterior fossa arises from the torcular Herophili and/or transverse sinuses.2,14,19,21,30,41,45,46,49,58,70 The clinical signs and symptoms are usually acute, but may be chronic.

Unique chronicity occurred in an epidural hematoma which was first recognized and removed 6 years following injury; it was invested by a calcified membrane.26 Intervals of 2 to 6 weeks from injury to diagnosis have been reported by Chambers35 and others.4,41,42,52,57,61,65,66,67,72,78 Recognition of an epidural hematoma may be hindered by (1) cerebrospinal-fluid rhinorrhea15 and (2) surgical ventricular drainage,78,79 both of which would provide for continuous decompression of the cerebrospinal fluid. In children, the chronicity is fostered by the more ready stripping of the dura mater from the skull which allows a thinner, more widespread extravasation of blood.5,25,56

The following case report exemplifies the clinical feature of chronicity in a hematoma at the vertex which caused inferior dislocation and compression of the superior sagittal sinus.

Case Presentation

While working on a cement loading dock, a 35-year-old lumberman was caught beneath an iron door as it fell from a box car. When the door was lifted 10 min. later, the patient was found to be unconscious. He was transported in this condition to a local hospital where roentgenograms disclosed a biparietal ring fracture of his skull (Fig. 1). After 3 days of profound coma, his sensorium began to clear and he was fully conscious by the 8th day following trauma. At the end of the 3rd week he was discharged from the hospital. He complained only of a mild suboccipital headache and horizontal diplopia on left lateral gaze. Neurological findings were normal except for minimal weakness of the left lateral rectus muscle. He had no papilledema.

Received for publication December 9, 1963.
Two weeks later, the patient's headaches had disappeared, but he began to experience obscurations of vision. These fleeting episodes of visual blurring always occurred in both eyes simultaneously and seemed unrelated to activity or time of day. Their duration was less than 1 min. The episodes persisted and re-examination of his fundus 6 weeks after injury revealed bilateral papilledema (3 D.) with hemorrhages. He was referred immediately to the Franklin Hospital in San Francisco for neurosurgical evaluation.

Examination. The patient was alert, cooperative and oriented. His blood pressure was 110/70. His pulse rate was 76/min. and was regular. He had no visible evidence of head injury. There was minimal weakness of the left lateral rectus muscle. He had no ptosis or nystagmus and his pupils reacted normally and were equal in size. Visual acuity was 20/20 in each eye and visual fields were normal except for enlargement of his blind spots. Papilledema was present in both eyes and was notable because of marked hemorrhagic changes located both superficially and deep within the nerve-fiber layers of the optic discs. Distension of retinal veins was moderate and venous pulsations were absent. Localizing or lateralizing neurological signs were not found.

Special Studies. Roentgenograms of the skull confirmed the presence of a nondepressed linear coronal fracture of the skull which extended obliquely across the midline into both parietal bones (Fig. 1). There was no evidence of increased intracranial pressure or of displacement of the pineal body. Electroencephalography provided no evidence of focal or generalized abnormality of the brain except for a "generalized nonspecific dysrhythmia" after hyperventilation. Carotid arteriograms revealed no lateral displacement of midline structures. Inferior displacement of the pericallosal arteries was evident in the arterial phases of this study. The venous phases demonstrated inferior displacement of the superior sagittal sinus from the inner table of the skull (Figs. 2 and 3). These studies indicated an epidural space-occupying lesion which was interposed between the skull and the parasagittal portions of both cerebral hemispheres. This finding, the history of trauma, and the findings of papilledema, palsy of the lateral rectus muscle, and fracture of the skull indicated an epidural hematoma at the vertex.

Operation. An organized epidural hematoma (0.5 cm. in thickness) with vascular membranes was encountered at each of 4 burr holes placed symmetrically over the vertex. Burr holes were located bilaterally both anterior and posterior to the parietal fracture approximately 3 cm. from the sagittal suture. A moderate amount of xanthochromic cerebrospinal fluid was removed from the subdural space subsequent to incision of the dura mater beneath the 4 burr holes.

Course. The retinal hemorrhages had begun to resolve by the 4th postoperative day. The optic discs were virtually normal by the 19th day after operation. The patient no longer had diplopia. Although the visual obscurations had disappeared, he noted transient left-sided photopsias for almost a month. The cerebrospinal-fluid pressure remained elevated during the first 4 weeks after operation (230–250 mm. water) and did not return to normal until the 8th postoperative week. The patient, at the present time, is asymptomatic and is gainfully employed.

Discussion

The majority of epidural hematomas located at the vertex are problems of acute and urgent clinical management although occasional prolonged clinical histories have been reported as in Alexander's series of patients. Alexander purported that the medial location of the extradural...