Extradural Hematoma
Experience with 167 Patients

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For generations, physicians have been taught to recognize extradural hemorrhage by the rapid march of symptoms and signs which occur after a seemingly trivial blow to the head. The onset of deepening coma, especially after a lucid interval, together with an enlarging pupil, contralateral hemiplegia, and bradycardia all portray the well-known picture of hemorrhage into the cranial extradural space. When the initial brain injury has been of no consequence, the clinical significance of such a rapid deterioration following an injury to the head is rarely overlooked. In such cases, the diagnosis is usually established quickly, and when appropriate surgical measures are taken the prognosis will be fairly good.

That the syndrome of extradural hematoma often can be puzzling and complex does not seem to be generally known. Little attention has been given to the difficulties in diagnosis that occur so often in those who develop a hematoma in the presence of significant amounts of associated brain injury. In such patients, the changing clinical picture caused by the accumulating blood clot frequently is obscured by the clinical alterations produced by the associated brain insult alone. The existence of significant grades of brain damage influences not only the clinical picture but also the surgical prognosis.

It is difficult to assess the over-all clinical variations of extradural hematoma except by a review of a large number of patients. This is particularly true when one attempts to examine the syndrome in the midst of varying amounts of associated brain injury. In this paper, we are presenting our experience with 167 patients who had an extradural hematoma. The pathological mechanisms as well as the principal syndromes as encountered among the patients in this series are also reviewed. These patients were treated on the Neurosurgical Service of Kings County Hospital, Brooklyn, New York, during the 25-year period ending with 1960. So far as we are aware, this is the largest clinical series of patients with extradural hemorrhage reported up to this time.

Pathological Mechanisms

In patients who have received a blow to the head and who are destined to develop an extradural hematoma, two immediate pathological events occur. One is the fracture of the skull and the other is the disturbance suffered by the intracranial contents.

In almost all cases of extradural hematoma there is fracture that involves some branch of the middle meningeal vessels. In this series, fracture of the skull was demonstrated by x-ray surgery, or autopsy in 152 patients (91%). A review of the 15 patients in whom fracture was not found showed that nine of them had no fracture by x-ray nor had it been found at operation. Since all of the nine survived after surgery, it is conceivable that a fracture did exist which had escaped the notice of both the radiologist and the surgeon. The remaining six patients without clinical evidence of fracture died and at autopsy no fracture was discovered. The exact source of the extradural bleeding in those individuals was never established.

Freytag, reporting the autopsy findings in 211 patients with extradural hematoma, found a fracture in all but six. Munro and Maltby, reporting 44 patients with extradural hematoma, stated that they had never encountered a case without fracture. Gurdjian and Webster said that all of their 30 patients had fractures. However, isolated instances of extradural hemorrhage without fracture of the skull have been reported. Thus, McKenzie in his study of 20 patients mentioned one case without fracture. In our experience, it is a mistake to place complete reliance on x-ray studies alone when searching for frac-
tions. For example, of the 152 patients in this series in whom fracture was eventually demonstrated, 12 had no fracture by x-ray but a fracture later was demonstrated either by operation or by autopsy. Because a fracture involving the cranial bones at the base is notoriously difficult to demonstrate by x-ray, bleeding from the nose or ear should be viewed as presumptive evidence of fracture.

Since the middle meningeal vessels are the usual source of hemorrhage in extradural hematoma, a review of them and their anatomic relationships is appropriate. The middle meningeal artery, after arising from the external carotid, enters the base of the skull through the foramen spinosum and then turns laterally on the floor of the middle cranial fossa until it reaches the squamous portion of the temporal bone. Here it divides into large anterior and posterior trunks with the branches of each distributed like a fan over the lateral aspect of the calvarium between the dura and the inner table. The branches of the artery reach as far out as the frontal and occipital regions where they become reduced to rather tiny dimensions. The artery is accompanied throughout all of its divisions by the middle meningeal vein and the vein drains into the pterygoid plexus.

For a long time middle meningeal hemorrhage was considered to be synonymous with bleeding from the artery alone and the pathologic significance of the middle meningeal vein was overlooked. However, it has been shown that the anatomical position of the vein renders it even more vulnerable to rupture than the artery. In 1723, James Keill\(^2\) mistakenly postulated that the vascular markings on the inner table of the skull were produced by the pulsations of the artery against it. This view was held until 1912 when Jones\(^1\) showed that these grooves contained the middle meningeal vein and that in fact the artery usually took a position entirely outside of the groove. Besides demonstrating the true position of the vein, the same investigator reported three fatal cases of extradural hemorrhage which had occurred as a result of rupture of the vein alone. In an autopsy study he injected contrast medium into the middle meningeal artery and vein separately and found that in each instance the vein alone had been torn while the artery had remained intact.

At operation the surgeon may find it difficult to identify the exact source of bleeding in cases of extradural hemorrhage. Active bleeding usually can be seen where the fracture crosses the middle meningeal grooves but even so it may be difficult to tell whether the artery, or the vein, or both have been ruptured. At operation accessory sources of hemorrhage may sometimes be seen coming from the cut edges of the fractured bone as well as from multiple spots on the dura. Occasionally laceration of the sagittal or the lateral venous sinus may prove to be the primary source of the hematoma rather than the middle meningeal vessels.

The strength of the dural attachments to the skull plays a major part in the development of the hematoma. Regarding these dural attachments, John Erichsen\(^5\) in 1779 said this: "I think that there can be little doubt that the detachment of the dura mater is a result of the blow on the head and the filling up is the consequence of that detachment and could not take place if the detachment had not previously occurred."

This important clinical observation remained largely forgotten until 1816 when Sir Charles Bell\(^1\), using cadavers, showed that when the cranium was struck with a wooden mallet, the blow caused the dura to become separated from the skull directly beneath the site of the impact. Because we considered Bell's observations so important, we repeated his work in our Pathologic Laboratory and corroborated his findings. Once the hemorrhage has begun, the gathering clot fills the extradural pocket first and as the bleeding progresses the dura becomes stripped away in an everwidening perimeter. In this manner the hematoma can grow to enormous proportions extending all the way from the frontal to the occipital regions. It has long been known that age has an influence on the strength of the dural attachments to the inner table. In most age groups the dura can be separated from the skull rather easily. However, in infants and in the elderly, the dura is so strongly adherent to the bone that it is difficult to separate the dura away from it and this undoubtedly explains why extradural hematoma is uncommon in the very young and in the aged.

In addition to the fracture, the other pathologic event of consequence following a blow to the head is the disturbance and damage