ACUTE SUBDURAL HAEMATOMA

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The main purpose of this paper is to urge a more optimistic approach to the problem of acute subdural haematoma and to point out a modification of the usual operation for this condition. This modified approach is based on a somewhat different opinion from that commonly held regarding the origin of the haemorrhage.

For many years it has been stressed that extradural haematoma represents an extreme operative emergency, but little has been said regarding the acute subdural haematoma. Too often the impression has been given that those patients with acute subdural haemorrhage always have associated brain damage (contusion, laceration, etc.) of such severity that the situation is hopeless. Authority after authority remarks on the uselessness of searching for the origin of the haemorrhage. A certain number of these patients have little or no brain damage other than that secondary to compression by clot and present a picture in the accident room indistinguishable both in clinical signs and also in the rapidity of downhill progression from extradural haemorrhage. With immediate action and in some cases with a modified operative approach many of these patients can be salvaged.

This surgeon feels very strongly that any patient seen in the emergency ward following head trauma who shows definite progression in his neurological picture and deepening of his state of unconsciousness merits exploratory burr holes immediately. It is not felt that normal pulse rate or blood pressure or respiratory rate should influence one in determining to let the patient "ride along." Patients may die with an operable intracranial haematoma with a pulse rate in the normal range up to within a few moments of exitus. Bilateral neurological signs, dilated fixed pupils, or tonic fits should not deter one from operative intervention if there has been definite progression in the picture. If a patient is not seen until an hour or several hours following trauma, it is not safe to assume that the picture is due to intrinsic brain damage, but on the contrary intracranial haematoma must be ruled out. The responsibility falls squarely on the shoulders of the surgeon—and too many patients with head injuries still die who might have been saved.

The papers on the subject usually ascribe the origin of acute subdural haematomas to laceration of the veins crossing to the longitudinal sinus. One author has the following to say: "Acute subdural hematomas usually result from extensive laceration of the veins crossing to the sinus [? longitudinal]. As it is impossible to learn which of these many veins is involved it is impossible to control them and surgical treatment of this condition is

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rarely successful."² Rowbotham¹ mentions as points of origin of the bleeding: lacerated dural venous sinus, cortical vein with associated tearing of arachnoid, or, most commonly, short communicative veins draining the cortical vessels into the sinuses. His figures show tearing of veins to longitudinal sinus. Browder reported that in 75 per cent of his cases there was a laceration or contusion of the brain implicating the cortical vessels.¹ He mentions that there is a fairly constant vein at the anterior aspect of the Sylvian fissure crossing the subdural space where it joins the sphenoparietal venous sinus which is frequently a source of bleeding. He makes a point, which should be emphasized, that at autopsy it is difficult to impossible to definitely identify the origin of the clot. There is a tendency to talk of the blood trickling to the most dependent parts of the subdural sac and accumulating over and under the temporal lobes, whereas it has been my experience in cases of massive acute subdural haematoma that the blood has welled up from the base at operation rather than originating at the vertex. This discussion is not concerned with thin layers of blood a few mm. in thickness but only with clots large enough to cause signs and symptoms by compression. In the latter type of case the bleeding is more often than not from the inferior cerebral veins running from the inferior surface of the temporal and occipital lobes to the transverse sinus (vein of Labbé, etc.), sphenoparietal sinus, and superior petrosal sinus (middle cerebral vein). These veins may be lacerated or even torn from the sinus with severe rapid bleeding.

The treatment usually recommended, with a suggestion of hopelessness, consists of a subtemporal decompression approach (with no mention made of locating bleeding points), evacuation of clot, and drainage of the subdural space (40 per cent mortality rate).² ⁴ ⁵ ⁶ ⁸ Dott, Alexander and Ascroft² advocate treatment by “evacuation of clot and arrest of bleeding if still in progress.” Aspiration of the haematoma and drainage of the cavity is also advocated with the admonition that no attempt be made to find the bleeding point, the sealing of which is left to natural processes. Other writers condemn the use of a wide osteoplastic flap or a wide subtemporal decompression with the comment that in an acute head injury the simpler the operation the greater the chances of recovery. The bleeding points that are left to nature unfortunately have a habit of bleeding more profusely than ever with the clot removed. Drainage of the subdural space, unless the “clot” is almost pure cerebrospinal fluid, is highly ineffective—if it were effective many of the patients would be exsanguinated on the table. One would not turn one’s back on severe venous or dural sinus bleeding at the time of tumor removal and I see no reason to do so in traumatic cases. In many instances, at least, the search for the bleeding point or points will be successful and fewer cases will come to autopsy with findings such as the following—"R died of a ruptured pial emissary vein on the left side of the head which caused a subdural haematoma. There were no lacerations of the brain of any sort." No surgical exploration was carried out on this patient although he lived for hours following trauma.