RECENT EXPERIENCES WITH EXTRADURAL HAEMORRHAGE*

F. J. IRSIGLER, M.D.

Neuro-Surgeon, Durban

This paper is based on 20 cases of extradural haemorrhage seen during the last 2 years. They are from a series of 62 intracranial haemorrhages of traumatic origin; among these were 15 intracerebral clots, the remaining cases being subdural haemorrhages.

UNUSUAL LOCATION OF CLOT

In 3 patients the extradural clots were situated at uncommon sites: one in the *posterior fossa* associated with a tear in the left transverse sinus caused by a depressed bone fragment. A profuse bleeding occurred on the removal of the fragment and was finally controlled by a muscle graft. The patient showed unilateral cerebellar signs and recovered without sequelae.

The signs included a coarse nystagmus to the side of the lesion, nuchal rigidity, and a tender swelling in the left mastoid region. The patient was operated upon on the 4th day after admission. This case has but little in common with the syndrome of extradural haemorrhage in the posterior fossa as described by McKenzie,[11] and Coleman and Thomson;[2] these authors mention increasing stupor during an interval of several days, as well as marked hypotonia and areflexia.

Two of the 3 cases had extradural clots in the *anterior fossa*. In one patient a clot was suspected but not encountered by exploratory burr holes in the subtemporal region. In the second case, a boy of 9 who had a small laceration above the right eye, the clot was evacuated on admission—about 30 hours after injury. The haematoma had separated and lifted up the dura entirely from the roof of the right orbit. The dura was conspicuously slack. The patient succumbed shortly after the operation and necropsy revealed an area of infarction extending from the internal capsule down to the mesencephalon on the side of the haemorrhage (Fig. 1).

This is possibly an example of the so-called concussion or blow-out haemorrhages described by Angrist and Mitchell.[1] According to these authors the characteristic features are the following: immediate loss of consciousness (as in concussion); location of the lesion distant from the site of injury—the sites of predilection being the internal capsule and the pons; finally the gross appearance of the lesion, the latter being either a bulky clot or grouped haemorrhages.

Gordy[5] has reported on a young man who suffered a car accident and died 8 days after the injury. Post-mortem examination revealed an extradural clot of considerable size covering the right frontal pole. 'Its presence' said Gordy, 'would not have been disclosed by an approach through the classical temporal incision'. It thus appears that the extradural clots in the frontal and subfrontal regions, especially those covering the frontal poles, are likely to be missed on making exploratory burr holes at the conventional sites.

Two illustrative cases of middle meningeal haemorrhage located in the frontal area were described by Jefferson.[8] Both cases had a most peculiar bilateral extensor rigidity resembling the decerebrate attitude in animals; the operation was unsuccessful.

Gross and Savitsky[6] reported on 3 cases of extradural haemorrhage in the anterior cranial fossa. The conventional temporal approach failed to reveal the clot in one case and and the patient died. In the two other cases the clots were successfully evacuated by a forward extension of the classical incision.

In this connection I want to mention another patient in my series who was admitted in a very poor condition with areflexia and maximally dilated pupils. He died within 2 hours without an operation. There was a depressed fracture of the left frontal bone involving the frontal sinus and an open wound above the left eyebrow. Necropsy revealed gross extradural and subdural haemorrhages in the right occipital area, a contrecoup lesion.

Another striking example of a contrecoup meningeal haemorrhage was found in a child of 9 who fell from a tree, striking the back of her head. She vomited several times. On admission, her level of consciousness was somewhat

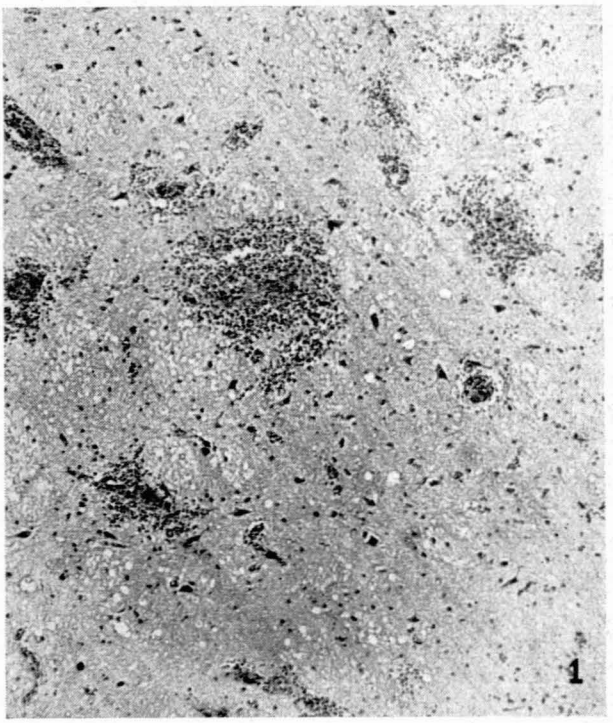


Fig. 1. Microscopic section through dorsal portion of pons. This was a boy of 9 who died after evacuation of a subfrontal extradural haemorrhage. Note ring-shaped petechial haemorrhages and areas of oedema mainly around vessels (haematoxylin and eosin stain). To the naked eye, there was infarction extending from the internal capsule to the midbrain (so-called 'concussion haemorrhage'). Microphoto taken by K. Schulz, Onderstepoort.

* A paper presented at the South African Medical Congress, Durban, September 1957.

depressed but she was responsive. In the left occipital area there was an abrasion of the scalp. The neurological findings were normal throughout. The child died a few hours later from sudden respiratory failure. Autopsy disclosed a fracture of the left occipital bone which had not been visualized on X-ray, with a small extradural haemorrhage beneath, not of surgical proportions. In the right frontal region there was a big subdural clot and in the underlying frontal lobe an intracerebral haemorrhage the size of a walnut, with smooth, shining walls; this clot had apparently ruptured into the subdural space but not into the ventricle. There was a considerable shift of the midline to the left.

DIAGNOSIS

Carotid Angiography

Carotid angiography may aid considerably in the diagnosis of extradural haemorrhage and its differential diagnosis from intracerebral haematoma. Occasionally, I found the *venous phase* of the angiogram most useful because it brought out the extent of the clot more clearly than the arterial phase—especially on stereoscopic viewing—by showing up a conspicuous *avascular area*, as in the case illustrated in Fig. 2, in which a big extradural clot on the left side was washed out through a parieto-occipital burr hole. Contrasting with this, Fig. 3 shows an intracerebral haematoma in the right pre-Rolandic area in a left-handed patient of 22; the clot was aspirated through a single burr hole on the 6th day after the injury.

The diagnosis of extradural clots in the posterior parietal and occipital areas may be difficult because of absence of pyramidal signs and pupillary changes. Even the cerebral arteriogram may fail to show up a displacement clearly; in such cases the venous and capillary phases of the angiogram usually are more helpful in diagnosis.

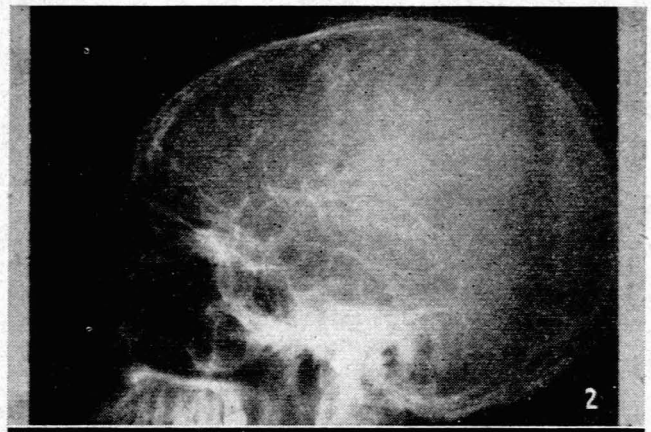
I would like to point out, however, that in cases with clear-cut signs of tentorial coning carotid needling may not be without danger; I have seen 2 cases of fatal pulmonary oedema during or immediately following this procedure. The one was a case of extradural clot beneath a depressed fracture in the left parietal bone associated with a big intracerebral haemorrhage adjacent to the fracture. The other patient was a chronic alcoholic who during an attack of delirium tremens suffered multiple head injuries and was admitted deeply unconscious. He had a bilateral subdural haematoma with softening in the distributary area of the posterior cerebral arteries due to tentorial coning.

The risks associated with angiography in patients with severe head injury, especially those requiring sedation or anaesthesia, were emphasized by Webster, Dawson and Gurdjian;[14] they also pointed out that arteriography is unreliable in cases of suspected extradural or subdural collection in the parietal and occipital areas. On the other hand, Kristiansen[10] has advocated what appears to be an almost indiscriminate use of angiography in cerebral trauma.

In my own experience, exploratory burr holes should be used rather than angiography in all patients deeply unconscious or in violent stupor, and in the presence of clinical signs of pressure cone. If needed, a ventricular estimation or ventriculography may be added as a further diagnostic step. In carrying this out collections in the occipital and posterior parietal regions present themselves for immediate evacuation.

Local Injury to Scalp and X-rays

In this series the diagnosis of extradural haemorrhage was missed in 2 patients because due attention was not paid to a local injury to the scalp. Without complete shaving and close examination of the whole head a bruise, a minute cut or a circumscribed area of 'puffy' oedema may easily be overlooked. Equally essential for early diagnosis are X-rays of the skull. In 17 of these 20 cases (85%) a skull fracture was present; in one instance the fracture was not visualized on the plates, but was found on necropsy. In 7 cases there was a linear fracture of the calvaria; in 8 cases a depressed fracture was present. Gurdjian and Webster[7] in their series



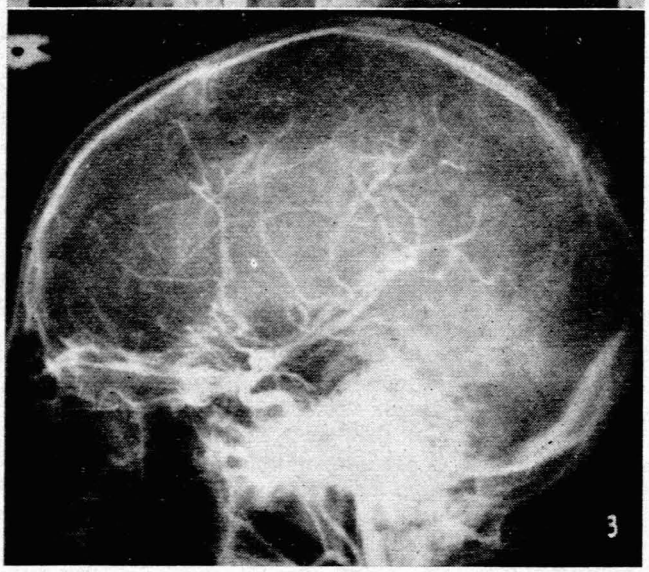


Fig. 2. Venous phase of carotid angiography in a man with a left-sided parieto-occipital extradural clot, which is outlined by the extension of an area of avascularity. The clot was subsequently washed out through an occipital burr hole.

Fig. 3. Carotid angiography (Arterial phase) in a case of intracerebral clot in the right pre-Rolandic region following closed head injury. The clot was successfully aspirated on the 6th day after the injury. The ascendent branches of the Sylvian group are splayed out indicating site of the clot. The anterior cerebral artery appears 'ironed out' suggesting shift across the mid-line which was clearly visible on the a-p. view. The 'arteriae insulares' of Krayenbühl and Richter[9] are compressed by the haematoma.

of 30 cases, found a fracture in every case; in 20% the fracture was depressed. In 4 cases of my series, the bone fracture involved the orbital and frontal regions of the skull which should always be studied on the plates with great care. In one case, a comminuted fracture extended into the frontal sinus (which was very large in this patient) resulting in a subcutaneous emphysema of the left forehead. In another patient, autopsy disclosed multiple fractures of the base of the skull, viz. both orbital roofs and the right petrous bone; this patient developed a recurrent extradural bleed (see below). Kristiansen[10] has aptly pointed out that the haematoma may be on the opposite side to the skull fracture; 2 such contrecoup haematomas were found in my series.

In only 2 cases in my series did examination of the scalp together with the X-ray plates fail to give an indication of the presence of an intracranial clot.

Neither a blood-stained lumbar fluid nor complete absence of pyramidal signs exclude an extradural clot; this point of great practical importance was brought out also by Gurdjian and Webster[7] in their comprehensive study published in 1942.

The so-called high decerebration attitude, i.e. tonic extensor rigidity due to release of subcortical levels, even if present from the onset, should not prevent investigation by exploratory burr holes or angiography as long as corneal and pupillary reflexes can be elicited. In one case I misinterpreted an early and consistent bilateral extensor rigidity as being due to primary brain-stem damage, only to be taught by the autopsy that the patient had a large parietal extradural haemorrhage extending into the floor of the middle fossa on the left side.

A *traumatic mydriasis* due to direct force applied to the eye (commonly resulting in a retrobulbar haematoma and exophthalmos with restriction of all ocular movements) may conceal an oculomotor palsy indicative of an extradural clot on the same side.

Cases Manifested Late

In the present series there were 3 cases of extradural haemorrhage of the slow-developing type, with clinical symptoms of increasing or fluctuating intensity after the 3rd day. At that stage, extradural haemorrhage may simulate a chronic subdural clot or an intracerebral haematoma of the kind recently described by Courville.[4] Furthermore, in the absence of a reliable history a differential diagnosis from typhoid fever, tuberculous meningitis, or cerebral venous thrombosis and the like, may have to be made. An example of this kind has been reported by Rowbotham and Whalley.[13] In one case I removed a big extradural clot from the left middle fossa extending into the occipital region in a Native boy of 8 who on the 10th day after injury developed a bilateral papilloedema and a dilated and fixed pupil.

THE ROLE OF INTRACRANIAL HYPOTENSION

In 2 cases in the series, intracranial hypotension due to loss of ventricular fluid following fracture of the base of the skull or extensive ventricular tap may have added to the fatal outcome. If the patient is in the supine position cerebrospinal fluid is likely to leak unobserved through a crack in the cribriform plate or in the petrous bone into the nasopharynx. A markedly *flabby dura* at operation obviously may imply the possibility that meningitis may develop; in addition, a low pressure state is likely to obscure the clinical picture and even may facilitate a recurrent or a contrecoup bleed. This applies equally to extradural and subdural collections. This point has been brought out nicely in 4 cases recently reported by Connolly.[3]

LUMBAR PUNCTURE

Sudden relief of pressure by lumbar tap was possibly a factor in the fatal outcome in one case of the series in which the patient died on the 5th day after injury from a recurrent extradural bleed in the posterior parietal region. On admission an extradural clot had been removed by the subtemporal approach. Lumbar puncture in the presence of an unsuspected extradural haemorrhage sometimes has disastrous effects; impressive cases of this type with fatal outcome have been reported by McKenzie,[11] Mock[12] and others.

OPERATIVE TECHNIQUE

The routine operative approach to an extradural haemorrhage is through a small subtemporal craniectomy which may be extended forward or backward as required so as to give sufficient access to the frontal and subfrontal area as well as the parietal area. The bone need not be replaced.

Occasionally an extradural clot may be encountered and should be washed out—like a subdural collection—through a single occipital burr hole, using the same site as for routine ventriculography.

Only in exceptional cases, when a solid clot of considerable thickness extends over the greater part of a hemisphere and cannot easily be washed out because of its firm adherence to the dura, will an enlarged trephine opening be necessary, and it is then advisable to replace the bigger bone fragments. If the brain does not expand after evacuation of the clot, and the dura remains slack, the dura should be sutured to the periosteum so that it lies snugly against the bone; this is certainly the best means to prevent a recurrent bleed.

MORTALITY RATE AND RESULTS

Ten patients—half of this series—fall into what could be suitably termed the group of blow-out haemorrhages without a well-marked lucid interval. Of these, 6 died soon after admission without regaining a normal level of consciousness. (Parenthetically, inebriation may interfere with the sequence of events.) Careful study at necropsy revealed that these failures were due to the following causes:

1. In 2 cases the patients were seen in a terminal stage, i.e. in profound unconsciousness with absence of all movements and of reflexes, including the nociceptive reflexes, and with dilated pupils. The only chance for such cases is that a suitably trained surgeon should be available in the casualty service to make emergency burr holes, if necessary under artificial respiration.

2. In 3 cases there was, on admission, an additional intracranial complication such as extensive fracture of the base of the skull with primary damage to the adjacent brain stem as evidenced by fixed pupils, bilateral extensor plantar response, over-breathing and occasionally hyperthermia; or a laceration of the brain with blood flooding the subdural space. In 2 cases there was, in addition to the extradural haemorrhage, an unsuspected intracerebral clot, of which I have given an example above.

Of the remaining 10 cases, all survived with the 2 exceptions

already mentioned, viz. one with the clot in the anterior fossa, and another with a recurrent extradural haemorrhage —the only one, by the way, seen in this series.

REFERENCES

1. Angrist, A. and Mitchell, N. (1943): Arch. Surg., **46,** 265.
2. Coleman, C. C. and Thomson, J. L. (1941): Surgery, **10,** 985.
3. Connolly, C. (1956): Brit. Med. J., **2,** 1154.
4. Courville, C. B. (1957): Arch. Neurol. Psychiat., **77,** 464.
5. Gordy, D. (1948): J. Neurosurg., **5,** 294.
6. Gross, S. W. and Savitsky, N. (1942): Ann. Surg., **116,** 821.
7. Gurdjian, E. S. and Webster, J. E. (1942): Surg. Gynec. Obstet., **75,** 206.
8. Jefferson, G. (1921): Brit. Med. J., **2,** 683.
9. Krayenbühl, H. and Richter, H. R. (1952): *Die Zerebrale Angiographie*, pp. 15 *et seq.* Stuttgart: Georg Thieme Verlag.
10. Kristiansen, K. (1948): Surgery, **24,** 755.
11. McKenzie, K. G. (1938): Brit. J. Surg., **26,** 346.
12. Mock, H. E. (1950): *Skull Fractures and Brain Injuries.* Baltimore: The Williams and Wilkins Company.
13. Rowbotham, G. F. and Whalley, N. (1952): J. Neurol. Neurosurg. Psychiat., **15,** 64.
14. Webster, J. E., Dawson, R. and Gurdjian, E. S. (1951): J. Neurosurg., **8,** 368.