CEREBELLAR EXTRADURAL HÆMATOMA*

BY

F. K. KESSEL

THE clinical features of an extradural hæmatoma due to rupture of the middle meningeal artery are well known; the "classical" picture may, however, be frequently absent or overshadowed by the signs produced by single or multiple cerebral lesions by a second extradural or a subdural hæmatoma. Besides the meningeal arteries the great venous sinuses can produce intracranial hæmorrhages. If a tear involves such a sinus and gives rise to an extradural hæmatoma overlying one of the more or less "silent" cortical areas, early diagnosis may be very difficult. To this latter group belongs the extradural hæmatoma of the posterior fossa; probably the rarest form of traumatic intracranial bleeding. The present paper contains a description of such a case, which was under the writer's care in spring, 1938.

A girl, aged 24, was thrown off her bicycle by a blow from an opening door of a stationary car, which she was about to pass. She struck the back of her head heavily, fell, and lost consciousness for a few minutes. Later she vomited, but on admission to hospital shortly after the accident she only complained of severe headache. There were no external injuries and no neurological abnormality was detected. X-ray pictures showed a slight separation of the left half of the lambdoid suture. About 30 hours later an experienced ward-sister observed the patient in a series of typical "cerebellar fits." In these her head was well retracted and her extremities rigidly extended; she was cyanosed, her breathing was irregular and she rapidly became unconscious. The first attack lasted about 2 minutes, a second one occurring a few hours later, and a third prolonged and severe attack was observed on the morning of the third day. After the first attack the patient's condition, which had been satisfactory until then, changed for the worse. She became increasingly restless and finally comatose. A lumbar puncture was performed, but only a few drops of clear cerebro-spinal fluid were obtained, the flow then ceased abruptly. A second puncture, performed 2 hours later, yielded the same result. There was no nystagmus and the tendon reflexes were equal on both sides.

The history of trauma, the separation of the left half of the lambdoid suture, the cerebellar fits, the cerebro-spinal fluid block, and the increasing coma all pointed to a hæmorrhage in the posterior fossa, probably extradural. Owing to the patient's deterioration operation could no longer be delayed.

Operation.—The usual skin-muscle flap for cerebellar exposures was employed and a burr-hole made over each cerebellar hemisphere. On the left side an extradural hæmatoma was found, and in order to evacuate it, the greater part of the bone on this side had to be nibbled away. A hole was then discovered in the left transverse sinus, situated at about the middle of its lower border—undoubtedly the source of the bleeding. The tear had already been closed by clot, and no further bleeding occurred. The hæmatoma had covered the greater part of the dura over the left cerebellar hemisphere, which was flattened and shifted to the right, thus blocking the cisterna magna. In order to relieve the increased intracranial tension, the cisterna was opened wide and fluid escaped under considerable pressure. Future events were to cause anxiety as regards this opening. The patient's general condition rapidly improved towards the end of the operation.

The healing of the wound was troublesome. Two days after the operation necrosis of the superficial layers of the scalp at the back of the head began and soon a great portion of the incision was involved in this process. A persistent leakage of cerebro-spinal fluid ensued, and apart from frequent dressing of the wound little could be done to check the flow of fluid. Fortunately the fistula closed spontaneously, and within a few weeks the patient left the hospital in good condition.

Comment

When first confronted with this case we had, like McKenzie (1938) under similar circumstances, been unaware of the occurrence of extradural hæmatomata in the posterior fossa, but our doubts regarding the diagnosis were dispelled by the striking signs presented by the patient.*

The most outstanding clinical feature was, of course, the patient's fits, which, like the occurrence of extradural cerebellar hæmatomata and the prognosis of a rupture of a transverse sinus, need some further comment.

Rigid Fits

The patient's fits were similar to those observed in cases of cerebellar tumours, described for the first time by H. Jackson (1906) and commonly called "cerebellar fits"; they are mostly considered as a special form of decerebrate rigidity caused by a

* In the discussion following the reading of this paper Prof. Cairns mentioned a case of his own in which the clinical picture suggested cerebellar bleeding; the condition, however, resolved spontaneously. He went on to suggest that the Queckenstedt test might yield useful information in cases of this type, particularly when the lateral sinus has been ruptured. In the present case this test could not have been carried out owing to the almost complete block which existed.

* Read at the 28th meeting of the Society of British Neurological Surgeons (20th July, 1941, Oxford).

functional decerebration at a level near the junction of pons and medulla. In Sherrington's (1897, 1898) opinion fits of this kind produced in animals are release phenomena arising from the interruption of cerebellar efferent impulses from the anterior lobe and its nuclei. Why these attacks are paroxysmal is not known. Webster and Weinberger hold the opinion that "cerebellar fits" are an extreme form of physiological decortication due to a temporary cortical ischemia. Penfield and Erickson (1941) state that "cerebellar" fits have nothing to do with the cerebellum itself and result from activity within the brainstem, namely a sudden neuronal discharge within the upper pons or lower mid-brain.

Besides this type of decerebrate rigidity two other forms can be distinguished in man and higher animals, namely, a "high" and a "low" decerebration; their characteristics and origin have been thoroughly discussed by Fulton (1938). Their essential symptomatic difference lies in the attitude of the upper extremities, which in a "high" decerebration are semiflexed and which show the tonic neck and labyrinthine reflexes of Magnus and de Kleijn, whereas in a "low" decerebration the upper extremities are in a rigid extension with pronation, and the rigidity may be so intense that the above-mentioned reflexes are difficult to elicit. Moreover, Bailey, Buchanan, and Bucy (1939) have recently stressed the important fact that an intercollicular ("low") decerebration results in relatively the same picture in all mammals—i.e. a generalized and severe extensor rigidity and an absence of the righting reflexes—but that the production of a high decerebrate syndrome demands a quite different level of section in the carnivora and the primates. In primates the high decerebration is essentially produced by decortication restricted to the areas 4 and 6, and the picture thus produced is, therefore, identical with a complete bilateral hemiplegia.

After Sherrington's description of decerebrate rigidity in 1897 and 1898 more than two decades passed before these results of experimental research were correlated with, and applied to, clinical observations. Apart from a few scattered reports, Wilson (1920) was the first to analyse certain postures in terms of decerebrate rigidity. Since then a number of cases of brain tumour showing decerebrate (or decorticate) syndromes have been reported (e.g. Walshe (1923), Davis (1925), Bailey (1933), Bailey, Buchanan, and Bucy (1939), and others), the tumour being mostly situated in the diencephalon or the hypothalamus. Certainly more cases of this kind have been observed than have been published; thus the present writer has seen the phenomenon under discussion in two cases of pineal tumour.

In cases of head injuries, also, states of muscular rigidity can be observed. It is perhaps worth mentioning that Jacobson's (1885) classical paper on middle meningeal hæmorrhage contains two cases with general muscular rigidity. Jefferson in 1921 described two cases with anteriorly placed clots derived from the middle meningeal arteries. The patients exhibited bilateral limb rigidities. Jefferson

was apparently the first to describe this condition as a definite entity after head injury, and regarded it as the nearest approach to true Sherringtonian decerebration possible in man. Since then further observations of this kind have been made (Babcock (1928), Woodhall and co-workers (1941), Penfield and Erickson (1941)), and a careful study of the extensive literature on head injuries would probably bring to light many more.

As to the cause of decerebrate rigidity in cases of intracranial hæmorrhage different explanations have been given: anoxæmia or change in intracranial circulation produced by the injury (Jefferson 1921, Babcock (1928), Penfield); or a temporal pressure cone (Jefferson, 1938); or compression of the mesencephalon (Reid and Cone, 1939). In my case, the space-occupying hæmatoma in the posterior fossa and the at least partial obstruction of one lateral sinus must have disturbed the intracranial circulation to a considerable extent, and this could, therefore, very well explain the whole phenomenon if one is not inclined to believe that the indirect pressure of the hæmatoma upon the anterior lobe of the vermis had a release effect in Sherrington's sense.

It is, however, important to realize that other patterns of fits may be caused by a lesion in the posterior fossa. Jackson (1906) quoted reports of clonic convulsive movements in patients with cerebellar tumours and other cases have been described since. Lately Webster and Weinberger have shown that in cases of cerebellar tumour practically all the kinds of fits usually observed in supratentorial lesions may occur.

The occurrence of rigid fits in head injuries emphasizes the seriousness of the prognosis (Babcock (1928), Woodhall (1941)) and indicates the urgent need for surgical intervention.

Extradural Venous Hæmorrhages

There are three possible sources of extradural hæmorrhages, namely, the meningeal arteries, the diploic veins, and the venous sinuses. Extradural hæmatomata of venous origin are by no means rare. Wood-Jones (1912) went so far as to say that from the published accounts of operations undertaken for middle meningeal hæmorrhage and from his own investigations it would seem that in the majority of such cases the blood was issuing from a ruptured venous sinus rather than from an artery. Surgical experience since then has not confirmed this view. Nevertheless, not a few cases have been reported illustrating the occurrence of venous extra-dural hæmorrhage (Erichson (1878), Jacobson (1885), Verbruggen (1937), McKenzie (1938), Reichert and Morrissey (1941), Munro and Maltby (1941), and others).

Only very few cases of extradural cerebellar hæmatoma have been made known; none of them had been successfully operated upon, at the time I encountered my case. Since then, Coleman and Thomson (1941) have reported a further case.*

* As these authors state they have not found a case in the literature that recovered, reference should be made of a previous communication (Jaeger and Kessel, 1938), in which the present case was mentioned (p. 140).

The following cases of extradural hæmatoma in the posterior fossa have been found in the literature: Lecount and Apfelbach, Naz and Jaboulay, Vance, McKenzie, Coleman and Thomson. It will be seen that not all of these cases can be considered as true "extradural cerebellar hæmatomata." A few other cases of hæmorrhage in the posterior fossa, complicated by other lesions, will be mentioned later.

Lecount and Apfelbach (1920) reported on 504 autopsied cases of injuries of the brain and the cranial bones. One hundred and ninety-nine cases showed extradural hæmatomata, 104 of them, weighing 20 to 246 g., had been large enough to produce an appreciable compression of the brain. There were no less than 8 cases with fractures of the posterior fossa with hæmorrhages from one of the transverse sinuses, the blood clot in none of these cases weighing more than 50 g. Unfortunately no further details about these 8 cases can be found in this very valuable paper. I very much doubt that all of them could be correctly designated as "extra-cerebellar hæmatomata," if this term means a hæmorrhage for the most part or wholly restricted to the cerebellar fossa proper. If out of 104 major extradural hæmatomata 8 were situated over the cerebellum, a ratio of 12 supratentorial hæmorrhages to 1 infratentorial would exist in the authors' collection. This is in striking contrast to all clinical experiences and to all other reports concerning extradural bleeding. I assume, therefore, that at least some, perhaps the majority, if not all, of these 8 cases of Lecount and Apfelbach were actually cerebellar-occipital hæmatomata. But it is noteworthy that all these 8 hæmorrhages were due to rupture of one of the transverse sinuses and that all of them were accompanied by fractures of the skull.

In his monograph Duret (1922) mentions 2 cases of "hématomes extra-dure-mériens des fosses cérébelleuses," one observed by Bousquet (1901) and another by Naz and Jaboulay (1910). In all probability, however, these hæmorrhages were not in the cerebellar fossa proper but over the occipital lobes.

Vance (1927) reported on 512 autopsies of skull fractures, 106 of them with extradural hæmorrhages; 4 of the hæmatomata were caused by a ruptured transverse sinus and had "pressed on the cerebellum and occipital lobes." This description seems to show rather clearly that these cases were not pure "extra-cerebellar hæmatomata." But it is again remarkable that all these cases had fractures of the posterior fossa and ruptures of the transverse sinuses.

The first certain extradural cerebellar hæmatoma was described by McKenzie (1938). McKenzie's collection of 20 cases of extradural hæmorrhage contained an account of one case of extradural hæmorrhage over the right cerebellar hemisphere. This patient, a child, sustained a slight head injury. There was a lucid interval of 30 hours, and marked flaccidity of all limbs was the only abnormal neurological feature. The cerebrospinal fluid was

clear. Forty-four hours after the injury and 14 hours after the onset of stupor the patient died from respiratory failure during an epileptic seizure beginning on the right side of the body and followed by opisthotonus. Necropsy disclosed a fracture of the occipital bone one inch long, which had not been seen on the X-ray films, and a clot compressing the right cerebellar lobe. Commenting on this case, McKenzie remarked that an operation might have saved the patient's life. He does not discuss the source of the bleeding, but clearly it could not have come from the middle meningeal artery, and may have been venous.

The case described by Coleman and Thomson (1941) is of great interest. The patient, a negro child, aged 9, had fallen from a moving truck, striking the back of his head, without becoming unconscious. He was brought to the hospital, but discharged, as there were no other signs present but a small hæmatoma in the left occipital region. The boy was returned to hospital 24 hours later because of headache and some drowsiness; he had vomited once or twice. The examination was again negative and the child was again discharged. The following night he was brought the third time to the hospital; he was moderately drowsy, occasionally very restless, complained of headache, and had vomited several times. The patient was then admitted to the hospital for further observation.

Fifteen hours later (on the morning of the third day following the accident) the boy was very drowsy, his neck was stiff and all limbs showed definite hypotonia. The biceps, triceps, knee and ankle jerks were absent. X-ray examination of the skull showed a long linear fracture in the mid-line, extending from the lambdoid suture downward into the foramen magnum.

Operative intervention revealed the presence of some old blood and of an early organized extradural hæmatoma over both cerebellar hemispheres, apparently due to a laceration of the torcular Herophili. The hæmatoma was removed and the patient recovered completely.

In this remarkable case more than 60 hours passed before the first symptom—drowsiness—appeared. Coleman and Thomson explain this long interval by the fact that the pressure within the sinus is only 4 to 6 mm. Hg., and that therefore a hæmatoma from a torn sinus develops slowly. A rather long latent period may therefore intervene between the time of injury and the onset of symptoms—an important point in view of the diagnosis.

Coleman and Thomson quote in their communication an unpublished case observed by Mayfield; the patient was suspected to have a cerebellar clot, but died before the posterior fossa could be explored. The post-mortem examination revealed an extradural cerebellar hæmatoma.

This short review of the literature shows that uncomplicated extradural cerebellar hæmatomata are extremely rare. The cases of McKenzie, Coleman and Thomson, Mayfield, and my own case, belong certainly to this group, but the cases quoted

by Duret are most probably not cerebellar hæmatomata, and it seems to be very unlikely that all 8 cases mentioned by Lecount and Apfelbach were pure posterior fossa hæmorrhages. None of Vance's cases apparently belongs to the group under discussion.

No other cases of uncomplicated extradural cerebellar hæmatoma could be found in the literature, but cases have been reported in which the extradural hæmatoma in the posterior fossa constituted only part of the pathological findings. Duret quotes a few cases of this kind. Two further cases of special interest as to the source of the bleeding may shortly be mentioned. Munro and Maltby (1941) in their series of 44 cases of extradural hæmorrhage reported one cerebellar hæmatoma found at post-mortem examination. Reporting this case in an earlier publication Munro (1938) stated that the hæmorrhage was caused by rupture of tributary veins to the left transverse sinus. Complicating this condition were: a right supratentorial subdural hæmatoma, contusion and laceration of the brain, fracture of the left occipital bone and medullary oedema. The clinical signs were naturally manifold and led to an operation on the subdural hæmatoma, while the bleeding in the posterior fossa remained unsuspected. The patient died 39 hours after the injury.

Pringle (1938) reported 71 cases of intracranial hæmorrhage. One of these (No. 267) was unconscious when admitted to hospital. A hæmatoma was suspected, but not found at operation. Autopsy disclosed two separate clots, one over the left occipital lobe and another over the left cerebellar hemisphere. Several fractures of the skull, cerebral contusion, an intracerebral and a subdural hæmorrhage were also present. Obviously no exact clinical diagnosis could have been arrived at in so complicated a case.

In another quite different type of case, hæmorrhage from the middle meningeal artery may extend backwards over the cerebellum. Kroenlein (1895), McKenzie (1938), and other authors have described such cases. Most of these patients died.

Munro (1938), Ricard (1929), and Voris (1940) have each observe and successfully operated upon a case of extradural hæmatoma originating from a tear in the upper edge of the transverse sinus. These clots, situated above the tentorium, showed the signs of a hæmorrhage from the middle meningeal artery.*

Conclusions

Reviewing the cases of the literature and my own, we can draw some important conclusions. *First*, that isolated extradural cerebellar hæmatoma are apparently always due to a laceration of the transverse sinus or one of the tributary veins. Duret

stated that rupture of the posterior meningeal artery could produce a cerebellar hæmatoma, but this, of course, is not true. Coleman and Thomson believe that a hæmatoma of the posterior fossa does not occur without skull fracture. My own case showed a separation of the left part of the lambdoid suture, a condition in its kind and effect not much different from a fracture. It is, however, important to note that a fracture can be invisible in the X-ray pictures, as in McKenzie's case.

The *second* conclusion is the fact, underlined by Coleman and Thomson, that owing to the relatively low pressure within the transverse sinus the development of a cerebellar hæmatoma may take rather a long time. More than 60 hours passed in Coleman's case before drowsiness as the first clinical symptom appeared; 30 hours elapsed in my case before the first fit occurred. In McKenzie's case there was a lucid interval of 30 hours' duration, but during this time hypotonia of the limbs was found.

It is doubtful if the few clinically observed cases permit a certain syndrome of cerebellar hæmatoma to be established.

Coleman and Thomson describe the diagnostic criteria for recognition of an extradural hæmorrhage in the posterior fossa as follows: History of a blow on the back of the head severe enough to produce a fracture of the skull, with or without unconsciousness; gradually increasing headache, usually accompanied by nausea and vomiting; progressive drowsiness and restlessness; forced position of the head; neck stiffness; nystagmus (which may be absent); disappearance of deep reflexes; hypotonia; when the drowsiness changes into unconsciousness, pulse and respiration become irregular, and death is imminent.

The symptomatology and the course of McKenzie's case and my own were, however, somewhat different from this description. It is certainly of importance that "cerebellar fits" can be the outstanding feature of the clinical picture. In such a case the diagnostic problem is not a difficult one; if no fits occur, as in Coleman and Thomson's case, certainly only repeated neurological examination and careful observation will deliver early enough conclusive symptoms for a correct diagnosis.

The *prognosis* in a case of ruptured sinus is necessarily bad. Of 42 cases with and without fracture of the skull found in the literature and reported by Ricard (1929) one-half died. Nine patients in this series had no fracture of the skull; they all died, and the diagnosis was established only at the post-mortem examination. Only the very clear symptoms of the case above reported saved her from death.

Summary

What appears to be the first case of extradural cerebellar hæmatoma successfully operated upon is described and discussed. The literature relative to the subject is discussed.

* While this paper was in the press, Gurdjian and Webster published a case of extradural hæmatoma over the cerebellum and occipital lobes due to a penetrating wound of the skull; the patient was operated upon and cured. *Surg. Gynec. Obstet.*, 75, *Internat. Abstracts*, 206.

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