Subdural haematoma after the treatment of chronic hydrocephalus by ventriculocaval shunts

R. D. ILLINGWORTH

From the Department of Neurosurgical Studies, The National Hospitals for Nervous Diseases, Queen Square and Maida Vale, London

An extradural or subdural haematoma may occur after lumbar air-encephalography or ventriculography (Khalifeh, van Allen, and Sahs, 1964; Calkins, van Allen, and Sahs, 1967), after ventricular drainage (Haft, Liss, and Mount, 1960), and after administration of intravenous urea (Marshall and Hinman, 1962). The development of a subdural haematoma after the insertion of a ventriculocaval shunt for the relief of hydrocephalus has been reported only occasionally. Foltz and Shurtleff (1963) mention one case and Horwitz and Rizzoli (1967) mention another. Forrest and Cooper (1968) describe briefly two cases of massive subdural haematoma which occurred after shunting in patients with large rigid skulls and thin cerebral cortex. Becker and Nulsen (1968) mention that seven subdural haematomas occurred in their series of 140 patients treated by shunts. However, most of these occurred in infants and none of the cases are described in any detail.

The factor common to these procedures appears to be the sudden reduction of intracranial pressure which results in bleeding into the subdural space, though trauma, which can be minor, may be responsible in some instances.

METHODS

The present paper describes 10 cases of subdural haematoma which occurred after the relief of chronic hydrocephalus by the insertion of a ventriculocaval shunt. Eight of the 10 cases (cases 1 to 8) occurred in a consecutive series of 175 patients treated between December 1958 and December 1968. Two other cases (cases 9 and 10) which occurred outside this series are also reported.

AGE In the whole series of 175 patients, all age groups are represented. There were 45 patients aged under 18 months at the time the valve was inserted, 29 were aged between 18 months and 16 years; there were 101 adults in the series. No children aged under 18 months developed subdural haematoma in the early period after insertion of the valve, but two patients in this age group

were found to have subdural haematomas at the ages of 2½ and 3. Two subdural haematomas were found in children aged 10 and 11; the remaining cases occurred in adults aged between 32 and 67.

DIAGNOSIS All the affected 10 patients had a chronic hydrocephalus. This was due to aqueduct stenosis in five, to communicating hydrocephalus in four, and to fourth ventricular atresia (Dandy-Walker syndrome) in one. These diagnoses were made by ventriculography alone in five and by lumbar air-encephalography alone in three. Two patients had both ventriculography and air-encephalography (AEG). No evidence of any pre-operative subdural haematoma was seen in any of these investigations and two patients had, in addition, a unilateral carotid arteriogram.

VENTRICULAR SIZE In nine patients, the radiographs were available for assessment of ventricular size. All showed a considerable degree of hydrocephalus and this was very marked in eight. In all the ventriculograms, to avoid disturbing cerebral function, only a small amount of air had been injected. This allows only measurement of the frontal mantle as an assessment of ventricular size. This is measured on a lateral brow-up view and is the distance of the frontal horn of the lateral ventricle to the outer table of the skull. This has been found easier to measure than the usual measurement from the frontal horn to the inner table of the skull. For convenience, this measurement has also been used on the AEGs. In the patients who developed subdural haematomas the measurement ranged from 1.5 cm to 4.1 cm with a mean at 2.85 cm (Table).

VALVES Six of these affected patients had Holter valves inserted and four had Pudenz valves. The policy of this neurosurgical department has been to test the valve pressures in most cases before insertion. This is done by setting up a saline-filled manometer to drain through the valve and the pressure at which the flow through the valve stops or becomes barely perceptible is recorded. This routine has been followed for both Holter and Pudenz valve systems. In the six Holter valves, this pressure in millimetres of saline was 140, 70, 60, 60, 50, and 25 and in the Pudenz valves 40 and 30. The pressure of one Pudenz valve was recorded as being 'almost

TABLE

Case no. Hospital no.	1 53370	2 A14848	3 63901	4 64212
Age and sex	2½ F	32 F	57 M	11 F
Initial diagnosis	Aqueduct stenosis	Communicating hydrocephalus	Aqueduct stenosis	Aqueduct stenosis
Initial investigations	Ventriculogram	Ventriculogram	AEG Ventriculogram	Ventriculogram
Ventricular size (Frontal mantle in cm)	2.0	1.5	—	3.3
Shunt and pressure (mm) (see text)	Holter 60	Pudenz*	Pudenz 40	Pudenz 'almost atmospheric'
Interval: shunt to discovery of subdural	7 m.	2 yr.	1 m.	6 w.
Presenting symptoms of subdural haematoma	? valve blocked	Fell. Irritable and ataxic	2 major fits. Coma	Progressive drowsiness and confusion
Investigations	None	Bilateral carotid angiogram	Ventriculogram	Carotid angiogram
Side of subdural	Right	Bilateral	Bilateral	Right
Treatment	Burr hole. Shunt occluded	Burr hole. Shunt occluded. Ventricular drain	Burr holes. Shunt occluded. Ventricular drain	Burr hole. Shunt occluded
Outcome	Well	Well	Survived disabled. Died 18 m. later from status epilepticus	Died

*Pressure not recorded. †Five weeks after revision of caval catheter.

atmospheric' and the pressure of another was not recorded. The mean pressure in the nine valves with recorded pressures was 52.5 mm saline; the mean pressure in the 143 patients without subdural haematomas whose valve pressures were recorded was 56.3 mm saline. In 22 patients, the valve pressure was not recorded. Two patients (cases 3 and 4) initially had Torkildsen's operation performed but these were later converted to ventriculocaval shunts because of difficulty with CSF absorption.

ONSET OF SYMPTOMS The interval between the insertion of the valve and the discovery of the subdural haematoma varied in eight patients from 15 days to three months, with the mean about five weeks. In one patient (case 8), subdural haematomas were found 2½ years after the original valve insertion, but five weeks after routine lengthening of the caval catheter. In the other patient (case 1), a unilateral subdural haematoma was found seven months after the original valve insertion when a revision was performed for obstruction of the ventricular catheter. The same patient was found to have bilateral subdural hygromas 18 months later when she became irritable after a fall. On this occasion, the fluid, though

containing 3.8 and 6.6g% of protein, resembled CSF and had obviously been present for a long time.

SYMPTOMS In six patients (cases 1, 3, 4, 5, 7, and 10) the presentation suggested that the valve was obstructed or not functioning properly. The usual symptoms were gradually increasing headache, and/or drowsiness and confusion. One of these patients became suddenly unconscious after some days of intermittent headache of increasing severity and another with a similar history of slow deterioration was in coma by the time of readmission. One patient (case 3) complained of headache on the side of the subdural haematoma and had a mild contralateral hemiparesis. In one patient (case 6), a massive bilateral subdural haematoma caused rapid descent into coma within minutes of a fall and another patient (case 2) had a similar course after two major epileptic convulsions. One patient (case 8) was drowsy and irritable after a routine revision of the caval catheter at another hospital, and then developed left-sided focal fits which progressed to prolonged status epilepticus. When this was controlled, he had a severe left hemiparesis. Bilateral subdural haematomas were present five weeks after the revision, the right being larger than the left.

TABLE—continued

5 64456	6 64749	7 A42946	8 A29815	9	10
62 M	60 M	10 M	3 M	66 M	59 M
Communicating hydrocephalus	Hydrocephalus, Dandy-Walker	Aqueduct stenosis	Aqueduct stenosis	Communicating hydrocephalus	Communicating hydrocephalus
AEG	AEG Ventriculogram	Ventriculogram	Ventriculogram	AEG	AEG
3.5	3.0	3.2	2.2	4.1	3.0
Pudenz 30	Holter 70	Holter 25	Holter 60	Holter 50	Holter 140
7 w.	1 m.	17 d.	2½ yr.†	3 m.	15 d.
Failure to improve. Slight increase in dementia	Fell out of chair. Coma	Lumbar puncture pressure raised with shunt working	Drowsy and irrit- able. Left focal fits, then status. Left hemiparesis	Drowsy and demented. Coma	Increasing drowsiness. Fell
Carotid angiograms	None	Carotid angiogram	Carotid angiogram	Carotid angiogram	None
Right	Bilateral	Bilateral	Bilateral	Left	Right
Burr holes. Shunt occluded. Craniotomy and excision of membranes	Burr holes. Shunt occluded	Burr holes. Shunt occluded. Ventricular drain. Valve changed	Burr holes. Subduro-peritoneal tubes	Burr holes. Craniotomy and excision of membranes	Burr holes
Survived disabled	Died	Well	Well	Died	Well

INVESTIGATION Seven patients had carotid angiography as the initial investigation to show the subdural haematoma, although in some of these patients malfunction of the valve was felt to be a likely alternative. Two patients (cases 2 and 6) with acute symptoms had immediate burr holes made without any initial diagnostic procedure. Electroencephalography was performed in three patients but was not helpful in two. In two patients with unilateral subdural haematoma, plain radiograph of the skull showed the tip of the ventricular catheter to be displaced from its position in the immediate post-operative period. In one case (Fig. 1), the catheter tip was displaced across the midline and this was also shown in one case of bilateral subdural haematomas in which one haematoma was larger than the other. This sign is, however, sometimes seen in patients without subdural haematoma and too much significance should not be attached to its presence. Ventriculography was performed in two patients with massive bilateral subdural haematomas; in both the previously grossly dilated ventricles were reduced to irregular slits (Figs. 2 and 3). In four patients, the subdural haematomas were unilateral. Three were on the right and one was

on the left. Five were bilateral and one (case 1) was initially unilateral and later bilateral.

TREATMENT In all cases, the haematomas were initially evacuated through burr holes and the subdural spaces were then tapped daily with blunt needles until dry. It is worth noting that, in one patient with bilateral frontal subdural haematomas, nothing abnormal was found when the posterior parietal burr holes made for ventriculography were reopened. In seven patients, to assist the brain to expand and obliterate the subdural cavity, the shunt tubing was occluded where it passed behind the ear. This was done with a strong ligature or by placing a Scoville clip across the tubing. Even in patients with communicating hydrocephalus, it was usually necessary to insert a ventricular drain to control the ventricular pressure and prevent it rising to dangerous levels while the shunt was occluded in this way. As soon as the subdural effusions had resolved, the ligature or Scoville clip was removed from the tubing and the ventricular drain was removed. No trouble was experienced from blood clot or debris blocking either end of the shunt system while it was occluded. One patient (case 7), whose

FIG. 1. Carotid arteriogram: Large subdural haematoma displacing the ventricular catheter (case 9).

FIG. 2. Ventriculogram: Ventricles reduced to irregular slits by massive bilateral subdural haematomas. Air is also present in the left subdural space (case 2).

FIG. 3. Ventriculogram: The same patient as in Fig. 2 after the ventricles had re-expanded (case 2).

initial valve pressure of 25 mm saline was felt to be too low, had this changed to one opening down to 110 mm. In one patient (case 8), persistent bilateral subdural haematomas were rapidly cleared by the insertion of a subduroperitoneal tube. The ventriculocaval shunt was left patent and not occluded in this instance.

RESULTS

Three patients died from the effects of the haematoma. All were deeply comatose at the time the diagnosis was made and in two this state had developed rapidly. Two patients survived with increased disability. One was severely demented with a mild hemiparesis before the shunt operation and was left mentally unchanged with a severe hemiparesis. The other, also severely demented before operation, was left with a moderate hemiparesis and severe dysphasia and died 18 months later from status epilepticus. No residual haematoma was found at necropsy. The remaining five patients survived without any apparent sequelae. The better results in these patients were probably due to earlier diagnosis and in some our previous experience led to an active search for a subdural haematoma as an alternative diagnosis to poor function of the shunt.

DISCUSSION

It seems most unlikely that any of these patients could have had subdural haematomas before the

shunt was inserted. All had had an air-study, seven had had burr holes which were usually bilateral and two had had carotid angiography. Rapid reduction in intracranial pressure can provoke extracerebral haematoma formation and this seems the likely explanation in these cases. It is known that the establishment of a functioning shunt system does cause reduction in ventricular size and, in cases of severe hydrocephalus, this could cause the surface of the brain to tend to fall away from the dura and open the subdural space. The rupture of bridging veins, or some other mechanism, could then result in the formation of a haematoma. It is interesting that no cases developed initially in the 45 children under 18 months of age. These numbers are too small for definite conclusions to be drawn, but it may be that before the anterior fontanelle has closed the skull has sufficient elasticity to prevent a marked disparity developing between the skull and its contents after a shunting operation. Trauma certainly played a part in one case, and possibly in two others, and may have contributed to two cases in which severe epileptic attacks occurred. In most cases the onset was insidious and was tentatively ascribed to poor functioning of the shunt. The possibility of a coexistent subdural haematoma should certainly be considered if cerebral symptoms develop in a patient with a valve which can be shown to be functioning.

The discovery of moderate sized subdural haematomas in a patient with virtually no symptoms (case 1), is of interest and it seems likely that the fluid had been present for a very long time. This suggests that some sort of balance can be reached between the pressure in the subdural fluid and in the shunted ventricles.

It has been pointed out (Adams, Fisher, Hakim, Ojemann, and Sweet, 1965; Adams, 1966) that a chronic symptomatic hydrocephalus can occur with an intracranial pressure which is well within the accepted normal range. These patients can be improved by the insertion of a ventriculocaval shunt which reduces the intracranial pressure to even lower levels. It may be that such an adult patient with a large hydrocephalus drained by a relatively low pressure valve is particularly at risk of developing a subdural haematoma. The relationship between the valve pressure as tested before insertion and the intracranial pressure after operation is clearly of importance. In this unit, we have attempted to measure and record a valve passage pressure and in many patients the lumbar puncture pressure has also been measured after the valve insertion. In most instances, the pressures of the valve and the lumbar cerebrospinal fluid have been approximately the same with differences of up to 30 mm of fluid. From

this it appears that at least in the lateral decubitus position the intracranial pressure after shunting is the same as the valve pressure in most instances. However, it has been reported (Corkery and Zachary, 1967) that the pressure of the valve can sometimes alter either upward or downward after insertion, and our experience confirms this.

It appears from the experiences described here that the treatment of chronic hydrocephalus by a ventriculocaval shunt does carry some risk of producing subdural haematomas. More information is required about the alteration in intracranial pressure which occurs after the insertion of ventriculocaval shunts, and about the ideal pressures at which the ventricles should be drained.

SUMMARY

Ten cases of subdural haematoma occurring after the insertion of a ventriculocaval shunt are described. This serious complication resulted in the death or disablement of five of these patients and seems more likely to occur after the treatment of patients with severe long-standing hydrocephalus. The diagnosis and treatment are discussed.

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