FATAL INTRACRANIAL VENOUS HÆMATOMA FOLLOWING VENTRICULAR DRAINAGE *

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A FATAL complication occurring in the course of a routine procedure must needs be placed on record, and although the title of the present communication contains the gist of the story, the full records of the incidents supply a theoretical interest which equals their practical importance. The three case histories which follow are concerned with the development of clots, extradural in two, intradural and intracerebral in one case, and rapidly fatal in all three. The clots might have been dealt with successfully had they been recognized in time in the second and third instances. It is evident that the speed of production of the clot is of no little importance, and it may well be that once the possibility of the condition is better recognized, successfully treated cases may in the future be placed upon record.

Extradural Hæmorrhages after Lowering Intracranial Pressure (2 cases)

Case 1.—D. R., a schoolgirl aged fourteen, was admitted to the Manchester Royal Infirmary under the care of Professor Jefferson, on the 12/2/40. For the past four years she had suffered from attacks of severe occipital headache accompanied by bouts of vomiting. The first of these attacks, according to her mother, followed a trivial head injury. During the six months preceding her admission to hospital the headache had become more frequent, she had walked unsteadily, and on occasions had experienced attacks in which "things seemed to spin" in front of her eyes. She had fallen in these attacks, but had never lost consciousness. Urgency of micturition was her only other complaint.

Examination showed her to be a well-developed girl, mentally alert and in a good state of general health. A medium degree of chronic œdema without hæmorrhages affected both optic nerve-heads. The visual fields showed no significant alteration other than enlargement of the blind spots, visual acuity was 6/9 in both eyes, the pupillary reflexes were normal and ocular movements full in all directions. There was no nystagmus. Tone was increased in all limbs with exaggeration of deep reflexes, and the plantar responses though not definitely extensor were undoubtedly abnormal. She performed the usual cerebellar tests well, but walked on a broad

base, holding herself stiffly and her head inclined to the right.

X-rays of the skull revealed some increase in the depth of the convolutional markings, flattening of the posterior and shortening of the anterior clinoid processes. Her skull was unusually large, a fact well known to the patient's mother, who for years had experienced difficulty in buying hats for her. The distance between the root of the nose and the external occipital protuberance measured over the vertex was 15½ inches.

The history, clinical state, and X-ray appearances suggested a slowly evolving increased intracranial tension. The absence of crisp signs of a clearly localizing nature and the chronicity of the process argued against the presence of a tumour and the diagnosis most favoured before operation was hydrocephalus due to intermittent obstruction of the iter. This later turned out to be the correct interpretation.

An exploration of the posterior fossa without previous ventriculography was carried out on the 27/2/40. The operation was performed under intratracheal nitrous oxide-oxygen and under local anæsthesia, supplemented by ¼ the basal anæsthetic dose of avertin. Omnopon gr. ¼ and Scopolamin gr. 1/200 were given two hours before the operation. The patient's breathing did not become distressed under the anæsthetic induction. The first step in the operation was puncture of the left lateral ventricle; the pressure of the cerebrospinal fluid was found to be higher than 400 mm. of water. Very slowly fluid was allowed to escape and the pressure reduced to atmospheric. The brain canula was then replaced by a rubber tube with a fine bore. Through this fluid continued to drip while the muscle was stripped from the occipital bone and the arch of the atlas removed. When the latter stage had been completed and before the occipital bone had been removed or even perforated, there was an unexplained and sudden gush of cerebrospinal fluid from the ventricle; it continued for about a minute and then the flow of fluid ceased. At the same moment small vessels in the muscle began to bleed, larger vessels previously coagulated opened up again, and the wound rapidly filled with dark blood. While still engaged in procuring hæmostasis the surgeon was informed by the anæsthetist that the

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patient had stopped breathing and that her systolic pressure had risen from 110 to 150, but her pulse had fallen (Fig. 1). Five minutes later she breathed

FIG. 1.—Chart of the blood pressure, pulse and respiration in Case 1.

again, but her blood pressure continued to rise, and her pulse rate after a temporary decrease climbed steeply. The occipital bone was rapidly removed, but so great was the tension in the posterior fossa that as soon as the dura had been opened for a distance of about 1 cm. the cerebellum bulged through the opening, bursting its surface vessels. No more fluid could be obtained from the left lateral ventricle; several attempts to locate the right posterior horn failed. Attempts to find a cyst by needling the vermis were unsuccessful; prolongation of the dural incision was impossible owing to the hæmorrhage which followed each attempt. The systolic pressure, having reached a peak of 190 mm., began to fall, though the pulse rate continued to rise until it no longer could be accurately counted. The wound was rapidly closed and the patient returned to the ward. She survived the operation for sixteen hours and during that period her temperature rose to 106°. Two intravenous injections of 50 per cent. sucrose caused no change in her condition.

At the post-mortem examination a large extradural clot was found overlying and compressing both frontal lobes. It extended upwards from near the floor of the anterior fossa to within about a centimetre of the coronal suture. Its maximum thickness was 4 cm. and more blood was present on the right side than the left. The lateral and third ventricles were greatly dilated, and at the level of the anterior quadrigeminal bodies the iter of Sylvius could not be identified. Histological studies later revealed that the lumen of the iter at this level had been replaced by proliferations of astrocytes enclosing narrow channels and extending into the central gray matter. It may be presumed that during the lifetime of the patient these channels allowed the occasional passage of small quantities of cerebrospinal fluid. Tentorial and foraminal herniations were present.

Comment.—The story of the operation, truly a

surgical nightmare, the chart with its eloquent graphs of blood pressure and pulse rate, and finally the necropsy findings form a picture which can gain but little in completeness by further comment. The chart brings to mind the results of animal experimentation concerning the effects of raised intracranial tension (Cushing, 1902), but in cases of rapidly expanding intracranial hæmorrhage the rising arterial pressure and the cerebral vasodilatation combining to restore the cerebral circulation, must add *pari passu* to the volume of the effusion and establish a vicious circle. The combination of supratentorial pressure with infratentorial decompression peculiar to the present case produced tentorial and foraminal herniation, thus further embarrassing vital centres. The greatly increased pressure in the posterior fossa mentioned in the description of the operation has its corollary in the rapid rise of the patient's pulse rate indicating failure of the vagus centre. It is interesting that the diastolic pressure did not for the first thirty minutes follow the upward trend of the systolic pressure, and for the first fifty minutes its curve closely followed that of the pulse rate.

Case 2.—E. P., a girl aged 10 years, was transferred to the Neurosurgical Department on the 31/12/41, having previously been under the care of Dr. F. R. Ferguson. For the past year she had suffered from headache, more severe and accompanied by vomiting in the past six months. Two months ago her sight deteriorated rapidly; she could no longer see the writing on the blackboard, and she ceased to attend school.

She was a well-developed girl in a fair state of nutrition. Her head was enlarged, its greatest circumference measured 23 inches. No gross mental defect was observed, but on some days she was apathetic and unduly sleepy. Both optic discs were affected by secondary atrophy, and she could barely count fingers at a distance of 18 inches. Her visual fields were generally constricted; her eyes moved freely in all directions without nystagmus. When she stood or walked she swayed and stumbled both to the right and left, but when she lay in bed her limbs disclosed no striking dysfunction apart from a slight terminal tremor which affected the movements of both hands.

On the basis of these clinical findings a definite diagnosis could not be made, though it was considered that her condition was more likely to be due to an inflammatory than a neoplastic obstruction of the flow of cerebrospinal fluid from the ventricles. Ventriculography was performed on the 7/11/41, and 110 c.c. of fluid were replaced by air. The films showed gross symmetrical dilatation of the lateral and third ventricles, and only the first three millimetres of the iter were outlined. A fine-bore rubber tube was introduced into the left lateral ventricle to provide a temporary safety valve against any rise in intracranial pressure and the child was returned to the ward. Her condition was good, though she was listless, and when questioned com-

plained of headache. About 1½ hours later the child developed a series of convulsions and became deeply unconscious. She was slightly cyanosed, her pulse rate was 180 and her respirations, peculiarly strained, were at the rate of 40 to 50 per minute. The fits followed each other in rapid succession, sometimes without a pause, and at first they were mainly of a tonic "decerebrate" variety. Each attack began with opisthotonus which lifted the patient's back off the bed; her arms became extended and overpronated, and spasm of the adductor and extensor muscles of the thighs determined the posture of the legs. No change in the attitude of the limbs occurred when the head was passively moved. Later another type of attack was observed: it consisted of loose-jointed flexion and extension movements at the left hip, knee, and elbow reminiscent of hemiballism. Occasionally the patient's eyes slowly moved from side to side and a fine horizontal nystagmus was present. The pupils were dilated and did not react to light. The flow of cerebrospinal fluid from the ventricular drain had ceased, and from the opposite ventricle only a few drops of fluid escaped under pressure.

The fits were first controlled by an ether anaesthetic and later 8 c.c. of a 10-per-cent. solution of sodium pentothal were introduced rectally. No further attacks occurred after this, but she died five hours after the onset of convulsions.

At the post-mortem examination an extradural clot was found overlying both frontal lobes. Its vertical extent from the floor of the anterior fossa was 15 cm., at its centre it was 5 cm. thick, and it measured 11 cm. from side to side. Careful scrutiny of the dura failed to reveal any indication as to a likely source of the bleeding. There were bilateral tentorial and foraminal herniations, and on sectioning the mid-brain the iter was found to be obstructed by a rounded tumour about the size of a cherry. Histologically the tumour was later proved to be an astrocytoma.

Comment.—For 1½ hours after her return to the ward following the ventriculography and institution of ventricular drainage the child was thought to be asleep. The fits first attracted the attention of the nursing staff. It is possible that she had been unconscious for some short time before the onset of the seizures which by their character—decerebrate rigidity and hemiballistic movements—suggested that a lesion had occurred at the level of the mid-brain. This interpretation was anatomically correct, for the herniated temporal lobes compressed the mid-brain. Jefferson (1921) for the first time described decerebration occurring in extradural hæmorrhage and in a more recent paper (1938) explained how tentorial herniation may bring about decerebrate rigidity.

Subdural and Intracerebral Hæmorrhage after Lowering Intracranial Pressure (1 Case)

Case 3.—A. B., female aged 24 years, was referred to Professor Jefferson on the 13/4/42. Excruciating headache and unsteadiness had troubled this patient

for four months before her admission. The headache affected the sides and back of her head and radiated into the neck, making all head movements painful. On four occasions her legs had given way, she had fallen, but had not lost consciousness nor been dizzy. On each occasion she got up almost at once and walked on. During the month before her admission the headache had become much more severe. Temporary loss of vision accompanied some of the attacks of pain. Her vision had deteriorated so that she no longer could read ordinary small print, and she had experienced double vision.

She gave this history readily though her statements regarding significant dates were not reliable. Both optic discs were swollen and there were scattered retinal hæmorrhages. The right external rectus was completely and the left partially paralysed. Ocular movements in all other directions were normal and there was no nystagmus. Her pupils reacted briskly to light and on convergence. Examination of her arms and legs yielded little positive information. The left hand was a trifle less steady than the right, and in performing the "heel-knee" test she overshot the mark a little with both heels. The tendon reflexes were depressed in all four limbs, but both plantar responses were extensor. The presumptive diagnosis was hydrocephalus due to cerebellar cyst.

On the day following her admission a fine-bore rubber tube was inserted into the posterior horn of the lateral ventricle because of the severity of her headache; the ventricular pressure was 200 mm. In the afternoon of that day she felt much better, but in the evening her head was retracted and she called out, apparently in great pain. The next morning she complained of severe frontal headache and screamed with pain every few minutes. She had difficulty in finding the words to convey even a simple statement. Her lips were slightly cyanosed; her pupils were equal and reacted to light, but her neurological state had not otherwise altered. No more C. S. F. escaped from the drain. Her temperature was 101.2°, her pulse rate 76. About an hour after this examination she suddenly stopped breathing. Artificial respiration was instituted at once and continued until her circulation failed five hours later.

At post-mortem both the right and left frontal lobes were covered and compressed by subdural blood clots, the one on the left being the larger (Fig. 2). In addition an intracerebral hæmorrhage into the white matter of the left occipital lobe had occurred in the track of the ventricular drain (Fig. 3). There was slight tentorial herniation on the left, but no foraminal prolapse. All four ventricles and the iter were greatly enlarged, and the foramen of Magendie appeared to be occluded by a membrane of thickened pia-arachnoid. To the naked eye the basal arachnoid throughout appeared tougher and more opaque than is normal, but microscopy failed to reveal inflammatory changes in the lepto-meninges. Histological sections of the brain area adjacent to the intracerebral hæmorrhage

A

B

FIG. 2.—The subdural clots from Case 3 photographed after formalin fixation. The larger clot was on the left side.

showed intense hyperæmia of all vessels, many of which were surrounded by hæmorrhages. These changes were most marked in the white matter although cortical and surface vessels were also affected (Fig. 4). In the mid-brain only an occasional hæmorrhage was seen and the state of filling of the vessels was not excessive.

Comment.—In this case ventricular drainage was performed as an emergency measure in the hope that it might improve the patient's condition

sufficiently to allow an operation to be undertaken after a day or two and to relieve the patient's headache. After a temporary improvement the headache returned in its former intensity, and shortly before the patient lost consciousness dysphasia was observed as the only additional neurological abnormality. Its significance did not sufficiently impress itself on the observers because it was not very obtrusive in so ill a patient who soon lost consciousness. She was thought to have a posterior

FIG. 3.—Section through the left occipital lobe from Case 3. Note that smaller hæmorrhages have occurred near the ventricle and elsewhere in the white matter.

FIG. 4.—Photomicrographs of sections through left parietal lobe from Case 3. (a) Dilated vessels in a sulcus. (b) Perivascular hæmorrhages in white matter.

fossa cyst and worsening of her condition was clinically attributed to a change in the tumour or in its neighbourhood. No striking circulatory changes were recorded in this patient, but her pulse rate—76—was relatively slow when her temperature had risen to 101.2° .

The three patients whose stories have been told died from intracranial hæmorrhage following ventricular drainage. Internal hydrocephalus with raised intracranial tension was present in all three; two had an obstruction of the iter near its commencement, in the third patient there was adhesive arachnoiditis at the foramen of Magendie. The hæmorrhage was extradural in two cases, subdural and intracerebral in Case 3. Although the vessels responsible for the hæmorrhage could not be identified, the situation of the clots and the absence of arterial disease or injury made it likely that the bleeding was venous. The extradural clots were probably derived from a number of dural veins, the subdural hæmorrhage from the rupture of a vein which crossed the subdural space on its way to the longitudinal sinus. All three extracerebral clots overlay and compressed the frontal lobes.

Discussion

Ventricular taps to reduce intracranial pressure during operations designed for posterior fossa lesions was introduced by Cushing and has greatly added to the safety with which many neurosurgical operations may be carried out. Following a suggestion by J. R. Learmonth, ventricular drainage was extended into an operation in its own rights at the Mayo Clinic, and E. R. Schmidt (1938) reported 24 cases in whom the ventricles had been continuously drained for periods up to 79 days. Only nine of these patients survived eventually, but since it may be assumed that either the pathology of the

lesion or the patients' general state precluded an attempt at a radical cure, the high mortality rate does not disparage the value of the method in a selected group of cases. As a method of rapid and prolonged decompression it is attractive for tiding a patient over an acute phase and for making bad cases more fit for operation. It is especially attractive after ventriculography if a general anæsthetic is to be given. An indwelling drain relieves the patient of the possibility of an acute pressure catastrophe during the stage of induction when anoxæmia and hypercapnia may introduce cerebral circulatory difficulties that would excite a fatal mid-brain hæmorrhage undetected at the time and credited to the operation itself in the after-event.

In this clinic the method has been extensively employed without ill effects until the present series of fatalities occurred. These have naturally prejudiced us against the method, but possibly we have recently been using too wide a bore tube. The introduction of a ureteric catheter was originally advocated, but the difficulties of rendering gum-elastic material sterile caused us to change over to the use of a fine-bore rubber tube. Its end should be half closed with a stitch so that fluid just drips slowly from the opening. We have not always done that; it was not done in Case 3.

The first time an extradural clot occurred it was thought to be ill-fortune, but when two further cases made their appearance it was clear that these hæmorrhages were something important. It is not difficult to trace a number of factors which could cause intracranial hæmorrhages when hydrocephalic ventricles are drained, but it is puzzling to understand why the employment of the method under entirely similar circumstances in the past did not more frequently result in this complication.

The obvious mechanical effect of ventricular drainage is twofold: reduction of intracranial

pressure and diminution of the volume of cerebrospinal fluid. As the intracranial pressure falls the dura, no longer subjected to tension, may contract by its elasticity and in some places become detached from the bone. Traction exerted on the dura via the venous anchorage of the brain presumably aids this process with the result that veins passing between the membrane and the skull are torn. The initiation of extradural clots may therefore depend on the ease with which the dura strips from the bone and on the original degree of ventricular dilatation; their incidence should be highest in the young in whom cerebellar tumours, strictures of the iter and adhesive arachnoiditis are common causes of very complete ventricular blocks and in whom the dura is loosely attached.

The emptying of the ventricles must affect the venous circulation, for the venous and cerebrospinal fluid pressures are closely related and an alteration in the one produces a corresponding change in the same direction in the other (Hill, 1896; Weed and Hughson, 1921). Thus ventricular drainage will cause the venous pressure to fall and, as the volume of cerebrospinal fluid decreases, blood will be retained in the intracranial veins. Fig. 4 demonstrates the hyperæmia which may occur under these circumstances. Possibly veins that have for long been compressed by increased intracranial tension have suffered anoxæmic damage and their walls may not be equal to the strain now imposed on them. The greatest burden may fall on the veins which connect the dural sinuses with the brain, for they are not supported by parenchyma and are dragged on as the ventricles empty. Once a vein has ruptured a clot will form and expand as long as the venous pressure exceeds the cerebrospinal fluid tension (Wegefärth, 1914). This condition is fulfilled in ventricular drainage. Presumably after a single tap the cerebrospinal fluid pressure soon rises again, thus preventing extensive separation of the dura and the formation of a hæmatoma, but should the pressure remain low a clot could result from one ventricular tap. Similarly intracerebral hæmorrhage in the track of the canula, as occurred in Case 3, might follow a single ventricular puncture if the cerebrospinal fluid pressure remained lower than the venous pressure. David *et al.* (1935) reported three cases in whom fatal intracerebral hæmorrhage followed the operative relief of longstanding intracranial pressure. The authors explained the hæmorrhages as being due to mechanical factors coupled with hypothetical damage to the hypothalamus. Their argument that the trauma of ventricular puncture was not a causative

factor is not convincing. Only one case of extradural hæmatoma following ventricular puncture can be found in the literature (Peet, 1940), but no details concerning it are available.

A common experience of neurosurgical practice illustrates the mechanism which produces extradural hæmorrhage. When a bone flap has been turned and the intracranial pressure lowered the dura follows the collapsing brain and by its elastic shrinkage separates from the edges of the bone defect. The fall in intracranial tension allows dural veins to fill with blood and many bleeding points now make their appearance on a dura which may not have bled previously. Particularly severe bleeding may be encountered from beneath the bone edge if a nearby meningeal vein or venous sinus becomes detached from the bone. Stitches then have to be inserted to pull the dura against the bone and muscle grafts applied to bleeding points.

In conclusion some practical points that emerge from the experience of these three cases may be stated. Intracranial bleeding may result from open ventricular drainage. Young individuals whose ventricles are greatly enlarged are particularly prone to this complication. A deterioration in a patient's state of responsiveness following the reduction of intracranial pressure should raise the suspicion that an extracerebral hæmorrhage has occurred, and the diagnosis should be established by exploration through burr holes. One burr hole on either side should be placed over the frontal pole. Cessation of the flow of cerebrospinal fluid was observed in all three patients when their clinical state had taken a turn for the worse, and in two patients no fluid could be obtained from the opposite ventricle. If present this sign should confirm the suspicion of intracranial hæmorrhage; its absence should not delay exploration. Air may remain for many days following ventriculography in ventricles which have no communication with the subarachnoid space—e.g. in cases of iter stricture. In these patients a rapid and decisive answer to the question whether a clot is present or not might be obtained from an X-ray examination.

Summary

1. Ventricular drainage caused fatal intracranial hæmorrhage in three patients.
2. Young individuals with chronic hydrocephalus appear to be most prone to this complication.
3. Detachment of the dura and venous hyperæmia are offered as explanations of the hæmorrhages.

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