Continuous Recording of the Ventricular-Fluid Pressure in Patients with Severe Acute Traumatic Brain Injury

A Preliminary Report*

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Measurements of the spinal-fluid pressure by means of lumbar puncture in cases of acute injury of the head have been reported and the value of such measurements has been discussed by Russell,17 Munro,12 Busch,2 Rowbotham16 and several other investigators.1,3 In order to study intracranial dynamics, Ryder et al.18 recorded the spinal-fluid pressure continuously in a few patients with acute injury of the brain. The cerebrospinal-fluid pressure has also been measured in animals after experimentally induced trauma to the head.9,15,20 Obvious interest has thus been shown in the changes of intracranial pressure in traumatic injury of the brain. However, no attempts have been made, either experimentally or clinically, to study, by continuous recording, the variations of this pressure during the acute stage. On the whole, present-day treatment of posttraumatic intracranial hypertension is based on rather fragmentary knowledge.

In our department continuous recording of the ventricular-fluid pressure has been used routinely for several years in a total number of 368 nontraumatic cases of intracranial hypertension. In our experience,10 the technique has proved to be safe and we have found considerable practical value in controlling the ventricular-fluid pressure. On this basis we decided to use continuous control of the ventricular-fluid pressure in cases of traumatic injury of the brain.

This is a preliminary report on the first 30 patients, illustrated by a few representative cases. The recording time ranged between 1 and 15 days (average 7 days). In 3 cases the ventricular cannula was removed because of obstruction or other technical difficulties, and in the remaining cases because further control was judged unnecessary. Daily bacterial cultures and counts of cells in the ventricular fluid disclosed no signs of infection of intracranial structures.

Recording Equipment

The ventricular cannula is connected to a strain-gauge transducer designed to measure pressures within a range of 250 mm. Hg. The impulses from the transducer are amplified by a standard ink-writing potentiometer recorder. The apparatus records the pressure continuously within a range of -10 to +115 mm. Hg on a graduated strip chart with a width of 250 mm. The standard paper speed is 12" per hour. Fig. 1 shows the circuit diagram of the apparatus. Details of the technique have been reported previously.10

Case Reports


H.G.K., a 38-year-old man, was hit by a car and rendered unconscious immediately. He was first admitted to a Department of General Surgery, at which time he was restless and confused, but not comatose.

Examination. On admission to the Neurosurgical Department 4 hrs. after the accident he was comatose and reacted only to pain. An angiogram demonstrated an intracerebral expansive process in the left frontal lobe and a thin subdural haematoma capping the lobe.

Course. During the hours following, the state of consciousness remained unchanged. Occasional attacks of tonic extension of the limbs, forced breathing, and general motor unrest occurred. The pupils were small and equal. Respiration was irregular, and on one occasion the patient had a respiratory arrest of 3–4 min.

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A ventricular cannula was inserted into the right lateral ventricle 19 hrs. after the accident. The ventricular-fluid pressure was initially 20–30 mm. Hg, i.e. moderately increased. This level was interrupted occasionally by spontaneous transient rises in pressure to 80–100 mm. Hg (Fig. 3). These rises were accompanied by tonic extension of the arms and legs and by an increase in blood pressure and pulse rate.

To reduce the intracranial pressure and prevent further sudden elevations, hypothermia (rectal temperature 32°–35°C.) was induced (Fig. 3) and maintained for 4 days. A second angiogram 5 days after admission showed a reduction in the size of

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**Fig. 2. Case 1.** The curve, recorded 24 hrs. after accident, shows the basic level of the ventricular-fluid pressure to be slightly above normal, a sudden spontaneous rise to a high level (about 90 mm. Hg), and after about 20 min. a rapid fall in pressure. The rise in pressure was accompanied by tonic extension of the limbs and cardiovascular disturbances; these symptoms disappeared at once when the ventricular-fluid pressure decreased. The horizontal bar indicates intravenous administration of urea. The small rhythmic oscillations of the pressure curve (1-per-min. waves) were related to the Cheyne-Stokes breathing of the patient.
FIG. 3. Case 1. This curve was recorded 27–33 hrs. after accident, during induction of hypothermia. The rectal temperature is measured in degrees centigrade. The plateau-wave at beginning of the curve was the last one during the time of recording (10 days).
the intracerebral mass, whereas the subdural haematoma had increased somewhat (about 1 cm. thick on the roentgenograms).

Operation. On the 6th day after the injury the subdural haematoma was removed.

Course. This did not influence the clinical state of the patient or the ventricular-fluid pressure, which at this time was 10–20 mm. Hg. Eventually, the ventricular-fluid pressure decreased and was almost normal when the ventricular cannula was removed on the 10th day. Consciousness had begun to return and during the following weeks the patient made a good recovery.

Comment. In this case the curve showed a considerable rise of the ventricular-fluid pressure during the first days after the accident. This rise was not caused by the subdural haemorrhage but obviously by a contusion of the left frontal lobe followed by oedema of the hemisphere. While the level of ventricular-fluid pressure was high the curve showed gross variations, similar in pattern to those which we have found typical for intracranial hypertension produced by tumours of the brain, i.e. irregularly and spontaneously occurring "plateau-waves" superimposed on a moderately elevated basic level.10 These waves were accompanied by transient signs of increased dysfunction of the brain stem. During induction of hypothermia the ventricular-fluid pressure decreased to a normal level and the plateau-waves disappeared. This occurred at a rectal temperature of 32°–33°C. and cooling below this was judged unnecessary. In most cases a normalizing of the ventricular-fluid pressure is not seen until the temperature drops to about 29°C.C.11


B.K., a 30-year-old woman, had undergone thymectomy some years previously because of myasthenia gravis. She was injured in a traffic accident and immediately rendered comatose.

Examination. On admission to the Neurosurgical Department she reacted only to strong painful stimuli. Her right pupil was dilated and there was a slight tonic extension of the legs.

Roentgenograms of the skull revealed a linear fracture in the left parietal region. An angiogram disclosed an expansion in the right temporal lobe, but no extracerebral haematoma.

Recording of the ventricular-fluid pressure was started 4 hrs. after the accident (Fig. 4). The initial pressure was high, 60–70 mm. Hg, and it rose rapidly to 80–90 mm. Hg. While the patient was being prepared for operation there was a sudden rise above 115 mm. Hg accompanied by respiratory arrest. Artificial respiration and infusion of urea were started. After a short time the ventricular-fluid pressure decreased and spontaneous breathing returned.

Operation. Hypothermia was induced, and at a rectal temperature of 26°C. a partial resection of the contused right temporal lobe was carried out.

Postoperatively the ventricular-fluid pressure remained at a fairly low level without any gross variations until the ventricular cannula was removed on the 9th day. Periods of rhythmic oscillations with a frequency of about 1 per min. coincided with periods of Cheyne-Stokes breathing (Fig. 5). The patient was unconscious for about a fortnight, and then slowly regained consciousness. Eventually, she made a fairly good recovery.

Comment. In this case recording of the ventricular-fluid pressure revealed an acute rise of the intracranial pressure which occurred about 4½ hrs. after the accident and was accompanied by severe disturbances in the function of the brain stem. This rise was considered to be caused by a temporal contusion and a progressive oedema. After infusion of urea and artificial hyperventilation had been started the curve showed a fall followed by a return of spontaneous respiration. However, the effect was short-lived and high-pressure waves reappeared. These waves disappeared when, after the induction of hypothermia, the rectal temperature had dropped below 29°C.

This record of ventricular-fluid pressure facilitated judgment of the intracranial state and assisted in making the decision to induce hypothermia and to postpone craniotomy until the intracranial pressure was under control. The choice of the appropriate time for the operation was also guided by the curve.

Postoperative recording suggested that the localized contusion in the temporal lobe was the cause of the intracranial hypertension, and also that intracranial hypertension did not play a role in the continuing dysfunction of the brain stem. The “burst” of rhythmic 1-per-min. waves in Fig. 5 illus-
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FIG. 4. Case 2. This curve was recorded 4–7 hrs. after the accident, before and during induction of hypothermia. The first part of the curve shows a gradual rise of ventricular-fluid pressure from 60–70 to 80–90 mm Hg, followed by a sudden rise to >115 mm Hg (flat curve), accompanied by respiratory arrest (arrow 1). Arrow 2: infusion of urea. Arrow 3: cooling started. Rectal temperatures are in degrees centigrade. The lower curve is an immediate continuation of the upper curve.

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B.S., a 27-year-old woman, was injured in a traffic accident. She lost consciousness at once and was still comatose on admission to the Neurosurgical Department.

Examination. Respiration was shallow and irregular and she was in shock. She was bleeding from the pharynx. Roentgenograms of the skull showed fluid levels in the nasal sinuses, but no fracture.

Angiography demonstrated a slight shift of the pericallosal artery to the left, indicating a diffuse swelling of the right hemisphere. There were no signs of a localized haematoma.

Course. Recording of the ventricular-fluid pressure was started approximately 12 hrs. after the injury and the pressure was essentially normal all the time it was being registered (Fig. 6). The patient remained unconscious for about 3 weeks, and when she regained consciousness she showed signs of bilateral palsy of the 6th nerve and a palsy of the left 7th nerve, as well as difficulties of speech, indicating a bulbar lesion.

Comment. This patient had a severe cerebral contusion with roentgenologic signs of some swelling of one cerebral hemisphere.
and neurological signs of dysfunction of the brain stem. Since the ventricular-fluid pressure was normal, it seemed probable that the latter signs were the result of a primary concussion of the brain stem.

A low ventricular-fluid pressure may be explained by leakage of fluid through a fracture of the base of the skull. In this case roentgenologic examination revealed no fracture but there was bleeding in the throat and fluid in the nasal sinuses. Perhaps the low pressure in this case was caused by leakage. In any event the curve of the ventricular-fluid pressure showed that any intervention for reducing the intracranial pressure and relieving the brain stem of mechanical stress was not needed.


S.E.B., a 19-year-old man, crashed into the woods on his motorcycle and was rendered unconscious immediately.

Examination. On admission to the Neurosurgical Department 3 hrs. after the injury he was comatose but reacted to painful stimuli. His breathing was stertorous, his left pupil dilated, and a right hemiparesis was suspected. Roentgenograms showed multiple fractures of the vault, as well as a somewhat depressed fracture in the left temporal region.

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**Fig. 5. Case 2.** The curve was recorded 40 hrs. after resection of contused right temporal lobe. Ventricular-fluid pressure level was slightly elevated but there were no plateau-waves. The rhythmic oscillations (1-per-min. waves) were caused by Cheyne-Stokes breathing, on this occasion from obstruction of the tracheal tube by mucus and relieved by tracheal suction.

**Fig. 6. Case 3.** The curve was recorded about 12 hrs. after the accident. The ventricular-fluid pressure level was within normal ranges. Rhythmic variations of low amplitude indicate periodic respiration.
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Comment. The curve of the ventricular-fluid pressure in Case 4 demonstrated the development of the condition called decompensated intracranial hypertension, i.e. a condition characterized by failure of the mechanisms which secure a sufficient cerebral blood flow by maintaining an adequate balance between systemic blood pressure, cerebral vascular resistance, and intracranial pressure.

This development was probably initiated when the systolic blood pressure fell to 80 mm. Hg (arrow in Fig. 7). With this blood pressure and a level of intracranial pressure of 40 mm. Hg the cerebral blood flow will probably be insufficient to maintain an adequate supply of oxygen to the brain. Hypoxaemia caused by the respiratory arrest may have been a contributory factor. Our observations suggested the following sequence of events: fall in blood pressure, insufficient cerebral blood flow, hypoxia of brain tissue, oedema, increased intracranial pressure, further decrease in cerebral blood flow, etc., i.e. the vicious circle which is supposed to characterize the final stages of fatal intracranial hypertension. The failing cerebral blood flow was clearly demonstrated by the angiograms, and progressive oedema of the brain tissue was seen at the operation (which should not have been performed since circulatory failure in the brain had been diagnosed).

In spite of the fact that the brain tissue
must have been severely and irreversibly damaged by hypoxia, the vasomotor protective mechanism was still functioning after the second operation, as shown by the rise in blood pressure concomitant with the rise in ventricular-fluid pressure. The definite exhaustion of this mechanism, occurring immediately before death, was represented by a steep rise of the ventricular-fluid pressure and the blood pressure, followed within a few minutes by a rapid fall of both pressures to near zero.

Discussion

The main purpose of the present investigation is to assess the importance of variations of intracranial pressure in cerebral dysfunction after acute injury of the head, and to evaluate continuous control of this pressure as a guide for treatment.

The following quotation may be regarded as representative of the commonly accepted opinion: “It would appear from these observations, which have been amply confirmed by others, that, while the intracranial pressure is often raised in cases of head injury of the group under consideration, the rise is seldom very high as compared, for instance, with that which may be observed in a patient with a sub-tentorial tumor whose mental state is alert and wakeful. Further, there is no correlation between the degree of raised pressure and the mental state of the patient.” This view is founded mainly on measurements of lumbar puncture. There are several reasons why such measurements may give misleading results. Leakage of fluid, which often starts along the needle as soon as the needle penetrates the meninges, may continue for several days after withdrawal of the needle. Furthermore, there is a marked tendency for increased intracranial pressure to exhibit rapid spontaneous variations. The futility of single measurements is obvious.

In previous investigations the pattern of the curve of ventricular-fluid pressure in patients with intracranial hypertension caused by intracranial tumours was found to be characterized by three main types of spontaneous variation:

1. Large plateau-shaped waves (usual ranges: height 50–100 mm. Hg, duration 5–20 min.) occurring at varying intervals and often accompanied by headaches and signs of dysfunction of the brain stem appearing paroxysmally. These attacks have been described by a variety of names, such as brain-stem seizures, acute incarceration, acute coning, mesencephalic and diencephalic seizures, cerebellar, decerebrate and tonic fits, etc.

2. Rhythmic oscillations with a frequency of about 1 per min. These waves are related to Cheyne-Stokes breathing.

3. Rhythmic oscillations with a frequency of about 6 per min., probably identical with the Traube-Hering-Mayer waves of the blood pressure.

The plateau-waves are of clinical interest first and foremost because they may cause acute injury to the brain stem, transient or lasting, and because they may be premonitory signs of such injury. Their occurrence signifies that the intracranial hypertension has proceeded to a stage where the forces working to maintain a sufficient cerebral blood flow begin to sag, and decompression is impending. The occurrence of rhythmic 1-per-min. waves may also give useful information since even mild forms of periodic breathing are easily detectable on the curve of the ventricular-fluid pressure.

As illustrated by Figs. 2 and 4 these spontaneous variations occurred in some of our traumatic cases. This confirms our previously expressed opinion that these variations of pressure are phenomena characterizing intracranial hypertension independently of its origin.

For some of our patients with signs of severe dysfunction of the brain stem the curve of the ventricular-fluid pressure showed a normal intracranial pressure (Fig. 6). One explanation is leakage of fluid. However, our observations suggest that patients with severe injury of the brain stem may have a normal intracranial pressure throughout the acute period even without leakage of cerebrospinal fluid. One must assume that

* Concussion and contusion.
the symptoms in such cases are caused exclusively by direct local trauma to the brain stem. It is also clear that there are patients with acute injury of the head in whom dysfunction of the brain stem is caused mainly by tentorial herniation. The importance of distinguishing between these groups is obvious.

From physiological and clinical investigations it is well known that the cerebral blood flow is closely related to the intracranial pressure.\(^6\,9,14\) Noel and Schneider\(^1\) found that the gradient between the mean blood pressure in the cerebral arteries and the intracranial pressure must be above a certain critical limit. In Case 4 (Fig. 7) the transient fall in blood pressure probably reduced this gradient below the critical level causing hypoxia which caused increased oedema of the brain and increased intracranial pressure. Our preliminary experiences suggest that simultaneous recording of the arterial blood pressure and the ventricular-fluid pressure would allow us to judge the efficiency of the cerebral circulation; impending hypoxia because of intracranial hypertension or a fall in systemic blood pressure could thus be predicted and counteracted before the vicious circle described herein is entered upon.

So far, the ventricular-fluid pressure has not been recorded in patients with an extradural haematoma before its removal. This, of course, is because of the acute need for immediate angiography and/or trephining. However, in patients from whom a haematoma has been removed, or else when shortage of time is no hindrance, it would be advantageous to start recording the ventricular-fluid pressure as soon as possible. If a haematoma has been removed, a recurrence or another expanding lesion could be detected early. The greatest value of recording the ventricular-fluid pressure is the information it gives in cases of severe injury of the brain without haematoma. In these cases, intervention to decrease intracranial pressure by such means as hypertonic solutions, hyperventilation, hypothermia, drainage of fluid and removal of localized contusions, may be more rationally applied, since "le régime tensionel" is known. This is especially the case in deeply comatose patients in whom intracranial hypertension may develop without other conspicuous clinical changes. The effect of the measures used can also be controlled more precisely than by means of the usual "pressure symptoms."

The tolerance of the brain to high intracranial pressures varies widely. Rises up to 100 mm. Hg may be endured by one patient without cerebral disturbances, whereas another patient may suffer respiratory arrest with relatively smaller increases of pressure, e.g. from 15 to 40 mm. Hg.\(^12\) The presence of a tentorial herniation, the degree of incarceration and other factors influencing intracranial dynamics as well as cardiovascular or respiratory disturbances may affect this tolerance. Consequently, no fixed limit between a tolerable and a nontolerable ventricular-fluid pressure can be established. However, in our clinical work we apply the rule that levels of ventricular-fluid pressure above 40 mm. Hg and large plateau-waves should always be regarded as potentially dangerous and an indication for measures to reduce pressure. Failure of urea to lower a high level of ventricular-fluid pressure may be caused by insufficient cerebral circulation and usually means a poor prognosis.

Our technique permits recording of the ventricular-fluid pressure for long periods practically without risk of infection of intracranial structures.\(^1\)\(^8\) It is an added advantage that fluid may be drained from and gas introduced into the ventricular system at any moment without extra intervention, and under control of pressure. Ventriculograms with small amounts of oxygen may be done repeatedly and have had no untoward side effects. Continuous drainage of fluid has in some cases been used successfully together with other pressure-reducing aids.

Our preliminary experiences suggest that continuous recording of the ventricular-fluid pressure can be done during the acute stage in cases of severe injury of the brain without exposing the patient to unchanged risk. The information gained by this procedure greatly facilitates the judgment of the intracranial state, and offers a more rational basis for
treatment than conventional measures of control. Our experiences, therefore, challenge the widely accepted view as expressed by Evans: "Emphasis should be placed on the failure of the cerebrospinal fluid pressure readings, even continuously recorded, to serve as a reliable guide to changes of a space-consuming character."

Summary

A technique for continuous recording of the ventricular-fluid pressure, hitherto used routinely for the control of intracranial pressure in patients with intracranial hypertension of a nontraumatic origin, has recently been applied in a series of patients with acute injury of the head (30 cases). Our experiences in this preliminary trial suggest the following conclusions:

1. Continuous recording of the ventricular-fluid pressure can be done throughout the acute stage of severe injury of the brain without exposing the patient to excessive risk.

2. In intracranial hypertension resulting from acute injury of the brain the ventricular-fluid pressure shows the same tendency to spontaneous variations and varies according to the same patterns and within the same ranges as in intracranial hypertension of a nontraumatic origin.

3. Variations in intracranial pressure seem to be of greater importance in the development of cerebral dysfunction after acute injury of the head than is generally supposed.

4. Distinguishing between cases of dysfunction of the brain stem with intracranial hypertension and such cases with a normal intracranial pressure is of clinical importance.

5. Continuous recording of the ventricular-fluid pressure in cases of severe traumatic injury of the head facilitates the evaluation of intracranial dynamics and offers a more rational basis for treatment than do conventional control measures.

References


