Intracranial hypertension in patients with ruptured intracranial aneurysm

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Simultaneous continuous recording of intracranial pressure (ICP) and systemic blood pressure was carried out in 26 patients admitted within 1 week after subarachnoid hemorrhage (SAH) due to a ruptured intracranial aneurysm. The patients were graded as described by Hunt and Hess. Recordings were made for 1 to 5 days. The more impaired the consciousness, the higher the rate of ICP. In Grade III, IV, and V patients, the mean ICP level was in the range of 15 to 40 mm Hg, 30 to 75 mm Hg, and exceeded 75 mm Hg, respectively. A definite correlation between vasospasm shown by cerebral arteriogram and the clinical grade was not observed. In our series of ICP recordings, we never observed a typical plateau wave. The variations of ICP seen in Grade III and IV patients were the B- and C-waves (15 to 45 mm Hg in amplitude) described by Lundberg, and those in Grade V patients were the high amplitude monotonous waves synchronous with the arterial pulses (15 to 40 mm Hg in amplitude). These phenomena may indicate that Grade III and IV patients with SAH are in a condition of cerebral vasomotor instability, and Grade V patients have cerebral vasomotor paralysis.

KEY WORDS subarachnoid hemorrhage clinical grade intracranial aneurysm intracranial hypertension vasomotor instability vasomotor paralysis

Since the microtechnique has been introduced for intracranial aneurysm surgery, the trend has been toward operating for ruptured aneurysm very soon after subarachnoid hemorrhage (SAH). However, the surgical risk is still closely related to the patient's condition, particularly the level of consciousness at the time of surgery.11,26 Operations performed soon after SAH have met with problems such as intracranial hypertension, cerebral vasospasm, and intracerebral hematoma formation.

The purpose of this investigation was to study the correlations between the level of consciousness, the level of intracranial pressure (ICP), and the prognosis in patients with recently ruptured intracranial aneurysm.

Clinical Material and Methods

The series consists of 26 patients admitted within 1 week after SAH due to a ruptured intracranial aneurysm. Each of these patients was unfit for immediate surgery. Patients were graded according to the method of Hunt and Hess.6 Five patients were in Grade V during recordings, eight in Grade IV, and 13 in Grade III.

Cerebral arteriography was usually performed within the first 24 hours of admission.
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and the location of the aneurysm was determined as well as the occurrence of cerebral vasospasm and the formation of intracerebral hematoma. Cases complicated with hematoma were omitted from this report.

Intracranial pressure was recorded through an indwelling ventricular and/or lumbar subarachnoid catheter connected with a pressure transducer.* Ventricular fluid pressure was recorded in three Grade III patients and all 13 Grade IV and V patients. The method used is based on that described by Lundberg.\(^{19}\) Lumbar subarachnoid pressure was recorded in 10 Grade III patients. It has been reported that lumbar subarachnoid pressure does not always show the actual ICP in patients with severe intracranial hypertension.\(^{17}\) In our series, however, the patients recorded through an indwelling lumbar subarachnoid catheter had no signs of obstruction in the cerebrospinal fluid (CSF) pathways. The systemic blood pressure (SBP) was recorded simultaneously in all patients through an intraarterial catheter placed in the femoral artery. The ICP and SBP were continuously recorded on a two-channel paper-chart recorder for between 1 and 5 days.

A direct intracranial operation was carried out in 22 patients using microsurgical technique within 1 week. The remaining four patients, all of whom were in Grade V, only underwent continuous ventricular drainage.

Results

Satisfactory recordings were obtained in all patients (Table 1). There were no complications attributable to the indwelling catheters, such as intracerebral hemorrhage or infection.

**Mean Intracranial Pressure and Cerebral Vasospasm**

Figure 1 shows a correlation between the clinical grade and mean ICP. The mean ICP levels ranged from 15 to 40 mm Hg in Grade III patients, from 30 to 75 mm Hg in Grade IV patients, and exceeded 75 mm Hg in Grade V patients. One of the Grade III patients died of diffuse vasospasm, which developed after surgery; the others survived and were categorized as a good result (that is, they were able to return to their preoperative activities with no neurological deficit or minimal and non-disabling neurological deficits). All the Grade IV patients survived, but three of them had severe residual neurological deficit. All the Grade V patients died of severe intracranial hypertension within 5 days of the last rupture.

Cerebral vasospasm was observed at arteriography in 12 of our 26 patients (46%): one Grade V patient, five Grade IV patients, and six Grade III patients. We observed only localized vasospasm. There was no definite

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*Pressure transducer model MP-4 and MPU-0.5 manufactured by Nihon Koden Kogyo Co., Ltd., Nishi-ochiai 1-31-4, Shinjuku-ku, Tokyo, Japan.

*Polyrecorder model EPR-3T manufactured by Toa Electronics Co., Ltd., Suwa-machi 235, Shinjuku-ku, Tokyo, Japan.
TABLE 1
Clinical course and recording data in 26 patients with ruptured intracranial aneurysm

<table>
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<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>Site of Aneurysm*</th>
<th>Day Begun after SAH</th>
<th>Total No.</th>
<th>Method†</th>
<th>Vasospasm</th>
<th>Clinical Grade during Recording</th>
<th>Operation</th>
<th>Results</th>
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<td>clipping</td>
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* MCA = middle cerebral artery; ACoA = anterior communicating artery; PCoA = posterior communicating artery.
† VFP = ventricular fluid pressure; LSP = lumbar subarachnoid pressure.

correlation between the severity of vasospasm and the clinical grade.

Pressure Patterns

The continuous ICP recording in the Grade III and IV patients showed a marked tendency to rapid variations superimposed upon the increased ICP baseline. The ICP increased recurrently to values of 15 to 20 mm Hg in Grade III and 30 to 45 mm Hg in Grade IV patients. Analysis of the waves revealed only two kinds of variations: one related to
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FIG. 2. Case 13, Grade III. Recording of intra-cranial pressure (ICP) on the fifth day after sub-arachnoid hemorrhage. Frequent ICP variations with moderate amplitude are seen. The abscissa indicates the date after the rupture.

periodic breathing of the Cheyne-Stokes type (B-wave), and the other to Traube-Hering-Mayer waves of SBP (C-wave) as described by Lundberg. In our series of ICP recordings, we never observed a typical plateau wave.

Figure 2 shows the ICP recording in a Grade III patient (Case 13). The simultaneous recordings of ICP and SBP at point A and B in Fig. 2 are shown in Fig. 3. The ICP curve fluctuated between 20 and 45 mm Hg, sometimes irregularly (Fig. 3 A) and sometimes rhythmically (Fig. 3 B); the duration of the waves was 30 seconds to 1 minute. The ICP variations caused slight changes in SBP. A rise in ICP to about 40 mm Hg or over caused the patient to become confused, while a fall in ICP to about 20 mm Hg made the patient alert.

Figure 4 shows the ICP recording in a Grade IV patient (Case 8). The ICP curve fluctuated with extremely high amplitude from 40 to 75 mm Hg. These waves were invariably accompanied by fluctuations of the patient’s level of consciousness. The rise in ICP to between 65 and 75 mm Hg coincided with developing deep coma, while the fall in ICP to about 40 mm Hg coincided with developing confusion or semicoma. Figure 5 shows the simultaneous recording of ICP and SBP at point A in Fig. 4. Frequent ICP variations were seen, and a marked rise in SBP was seen concomitant with ICP variations.

FIG. 3. Case 13, Grade III. Recordings of intra-cranial pressure (ICP) and systemic blood pressure (SBP) at point A and B shown in Fig. 2. Paper-chart speed is 2 cm/min. Pressure variations in ICP are sometimes irregular (A) and sometimes rhythmic (B).

FIG. 4. Case 8, Grade IV. Recording of intra-cranial pressure (ICP) on the fourth day after sub-arachnoid hemorrhage. Frequent ICP variations with extremely high amplitude are seen.

FIG. 5. Case 8, Grade IV. Recordings of intra-cranial pressure (ICP) and systemic blood pressure (SBP) at point A shown in Fig. 4. Paper-chart speed is 2 cm/min. Both ICP and SBP show marked fluctuations.
Figure 6 shows the trace of ICP in a patient (Case 6) during deterioration from Grade IV to V. Frequent pressure variations were seen at the Grade IV clinical level, whereas at the Grade V level the pressure variations were suppressed and replaced by high amplitude monotonous waves synchronous with the arterial pulses; these waves were registered upon the extremely increased ICP baseline which exceeded 75 mm Hg. Tachycardia and tachypnea were usually seen in this stage. Figure 7 shows the simultaneous recording of ICP and SBP at point A in Fig. 6 while the patient was in a Grade V clinical condition. Stable, high amplitude monotonous waves of ICP and SBP were registered. This high level of ICP dropped concomitantly with falling SBP in the terminal stage. This ICP pattern was seen in all Grade V patients, and indicated a state of cerebral vasomotor paralysis.

Intracranial Pressure during Subarachnoid Hemorrhage

In Case 7 the aneurysm ruptured during the ICP recording. The patient deteriorated to Grade IV after the first attack, and continuous ventricular drainage kept the ICP constant at about 20 to 35 mm Hg (Fig. 8 A). Twenty hours after the ventricular drainage, the ICP showed a steep rise to about 75 mm Hg (Fig. 8 B), and effusion of fresh blood from the ventricular catheter was seen. The patient suddenly became deeply comatose, and ICP showed high amplitude monotonous waves derived from the arterial pulses. The patient died and subsequently the ICP decreased concomitantly with the falling SBP.

Discussion

The increase in ICP following the rupture of an aneurysm contributes to the arrest of the hemorrhage.21,22 When patients tolerate the high ICP at the time of the hemorrhagic attack, ICP falls subsequently to a considerably lower level with little or no hematoma formation, but a slow increase in ICP occurs in the course of the next few hours.22 Our study shows that this second increase in ICP continues for several days.
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considered as separate processes. Nornes and Magnaes considered that edema formation derived from tissue hypoxia was due to increased ICP or arterial spasm following SAH. Obrador and Pi-Suñé, Langfitt, et al., and Hekmatpanah all emphasized the role of vascular dilatation in producing acute brain swelling. In our study, vasodilatation is implicated as the cause of increased ICP by the spontaneous fluctuations in ICP in Grade III and IV patients and the phenomenon of cerebral vasomotor paralysis seen in the Grade V patients. In the latter, particularly, ICP appears to reflect SBP, that is, the marked oscillating waves synchronous with the arterial pulses registered on an extremely increased ICP which declines concomitantly with the fall of SBP in the terminal stage.

On the other hand, blood in the sub-arachnoid space causes an aseptic inflammatory response or thickening of the meninges, producing arachnoid adhesions at the base of the brain. The meningeal reaction blocks the CSF pathways and increases ICP. Suzuki, et al., report that continuous ventricular drainage improved the level of consciousness. These indicate that the edema formation, the increase in intracranial blood volume and CSF accumulation are all significant in producing the secondary rise in ICP after SAH.

Nagai, et al., reported that when ICP exceeded 40 mm Hg in dogs, the cerebral blood flow decreased slightly, and at the pressure of approximately 75 mm Hg, the blood flow in the brain stem began to decrease and brain function became irreversibly damaged. Langfitt, et al., stated that the acute elevation of ICP causes cerebral vasodilatation and the vasopressor mechanism fails in the terminal stage. Our data show that impaired consciousness in patients with recently ruptured aneurysms is much more dependent on the increased ICP than the cerebral vasospasm. The mean ICP levels were in the range of 15 to 40 mm Hg in Grade III patients, 30 to 75 mm Hg in Grade IV patients, and exceeded 75 mm Hg in Grade V patients. It is likely that the impairment of consciousness is slight and the autoregulatory mechanism of the cerebrovascular system may still be preserved in a Grade III patient, the level of consciousness is moderately impaired and the cerebral blood flow may be reduced in a Grade IV patient, and in Grade V the cerebral blood flow is markedly reduced, losing the autoregulatory mechanism in the last stage.

Marked fluctuations in ICP in Grade III and IV patients were related to periodic breathing of the Cheyne-Stokes type (B-wave), and to the variations of SBP (C-wave) described by Lundberg and those in Grade V patients were the high amplitude monotonous waves derived from the arterial pulses. On the other hand, the so-called plateau waves were never observed in the acute stage of patients with ruptured aneurysms. In general, B- and C-waves seen in chronic intracranial hypertension are of less amplitude and of little clinical significance. We found in acute intracranial hypertension in Grade III and IV patients, however, that the more impaired the consciousness, the more frequent and the higher the amplitude of B- and C-waves. The B- and C-waves are believed to be related to an intrinsic rhythmic activity of the medullary centers, released from the influence of higher centers in the upper brain stem, and regarded as a sign of brain-stem dysfunction. The fact that the frequent and continuing B- and C-waves were seen in Grade III and IV patients suggests that the cerebral vasomotor instability may play an important role in these stages. Such vasomotor instability may still be reversible. On the other hand, the high amplitude ICP changes derived from the arterial pulses seen in Grade V patients indicate the loss of the cerebral vasomotor mechanism, that is, the vasoparalytic state, which is not reversible.

Klafta and Hamby recommended that surgery in Grade III and IV patients should be delayed until CSF pressure returns to 15 mm Hg and the clinical status has improved and stabilized. Nornes also claimed that an epidural pressure at about 30 mm Hg is an indication for the timing of surgery. Our experience supports these findings. The quality of survival in Grade III patients was good with one exception. However, the quality of survival was poor in Grade IV patients in whom the mean ICP exceeded 30 mm Hg. Surgical procedures in patients with ICP exceeding 30 mm Hg may possibly produce more impairment to the brain structure, leading to an irreversible state. We stress that continuous recording of ICP in patients with a recently ruptured intracranial aneurysm
shows not only the course of the disease but also the grade of cerebral vascular dysfunction such as vasomotor instability and paralysis.

References

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