Intracranial pressure changes following aneurysm rupture

Part 3: Recurrent hemorrhage

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Continuous monitoring of intraventricular pressure (IVP) was performed before and during 13 recurrent hemorrhages occurring in 10 patients between the 3rd and 14th day after the initial rupture of an intracranial saccular aneurysm. Before re-rupture, nine patients were of Hunt and Hess' clinical Grade III or IV. Severe angiographic vasospasm was demonstrated in six patients. In the period between ruptures, IVP and mean arterial blood pressure were significantly increased compared to pressures in patients who did not rebleed. Ventricular drainage of cerebrospinal fluid (CSF) to a level of 25 mm Hg did not increase the rate of rebleeding (17% of patients). On the other hand, the use of drainage while the repeat rupture was taking place seemed to exert a deleterious effect on the natural mechanisms that lead to arrest of hemorrhage. In five patients with CSF drainage during their rebleed, the steady-state IVP level after the repeat rupture was significantly increased, and four patients died from large intracerebral hemorrhages. These results suggest that drainage of CSF should be avoided during recurrent hemorrhage, and should not be resumed until a steady-state IVP level has been reached.

KEY WORDS • intracranial aneurysm • subarachnoid hemorrhage • intracranial pressure • cerebral vasospasm • recurrent hemorrhage

In patients with recent rupture of an intracranial saccular aneurysm, a recurrent hemorrhage is a frequent and serious complication. Depending on the anatomical location of the aneurysm, rates of rebleeding vary between 14% and 30% during the first 2 weeks after the initial hemorrhage. Recurrent hemorrhage carries a high mortality. In the Cooperative Aneurysm Study, 85% of patients died after a repeat hemorrhage.

Under experimental conditions, bleeding from an intracranial artery has been shown to be a self-limiting and survivable event because of an interplay between several mechanisms: 1) intracranial counterpressure; 2) clot formation; 3) autoregulation of cerebral blood flow (CBF); and 4) outflow of cerebrospinal fluid (CSF). In cases of repeated subarachnoid hemorrhages (SAH), the steady state of CSF pressure will increase with each bleed, eventually resulting in a sustained high intracranial pressure (ICP) threatening vital brain functions.

The course of ICP during the rupture of an aneurysm has been studied in patients by continuous monitoring, both in the preoperative phase and during operation. The risk of provoking a recurrent hemorrhage during reduction of an elevated ICP by means of drainage of CSF has been discussed.

Parameters that might permit identification of patients who are liable to rebleed would be of clinical significance. Continuous monitoring of the intraventricular pressure (IVP) in aneurysm patients yields information of therapeutic and prognostic significance. We have recorded the IVP during recurrent hemorrhage in 10 patients. The aim of this paper was to evaluate the relationship between clinically measurable parameters in the period between ruptures (specifically, blood pressure, IVP, clinical condition, and degree of vasospasm), and the possible influence of drainage of CSF on the pathophysiological mechanisms taking place during recurrent hemorrhage.

Clinical Material and Methods

The patient population and methods have been described in detail in the first part of this study.
Intracranial pressure changes after aneurysm rupture

**TABLE 1**

**Summary of clinical and pathophysiological changes in 10 patients with rebleeding from an intracranial saccular aneurysm***

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Site of Aneurysm</th>
<th>Vasospasm</th>
<th>Day After 1st SAH</th>
<th>Ventricular Drainage</th>
<th>Clinical Grade</th>
<th>Mean IVP (mm Hg)</th>
<th>MABP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>13</td>
<td>50, F</td>
<td>ICA</td>
<td>slight</td>
<td>7</td>
<td>no</td>
<td>III/IV</td>
<td>19/36</td>
<td>112/118</td>
</tr>
<tr>
<td>17</td>
<td>52, F</td>
<td>PCA</td>
<td>severe</td>
<td>12</td>
<td>no</td>
<td>III/IV</td>
<td>13/16</td>
<td>110/118</td>
</tr>
<tr>
<td>22</td>
<td>64, F</td>
<td>ACoA</td>
<td>slight</td>
<td>12</td>
<td>yes</td>
<td>III/III</td>
<td>16/20</td>
<td>125/136</td>
</tr>
<tr>
<td>27</td>
<td>64, F</td>
<td>ICA</td>
<td>severe</td>
<td>9</td>
<td>no</td>
<td>III/IV</td>
<td>27/33</td>
<td>108/125</td>
</tr>
<tr>
<td>30</td>
<td>39, F</td>
<td>PCA</td>
<td>severe</td>
<td>8</td>
<td>yes</td>
<td>IV/V</td>
<td>25/52</td>
<td>104/45</td>
</tr>
<tr>
<td>33</td>
<td>51, F</td>
<td>ACoA</td>
<td>none</td>
<td>3</td>
<td>no</td>
<td>III/IV</td>
<td>20/22</td>
<td>156/158</td>
</tr>
<tr>
<td>34</td>
<td>46, F</td>
<td>ACoA</td>
<td>severe</td>
<td>5</td>
<td>yes</td>
<td>III/III</td>
<td>32/50</td>
<td>137/140</td>
</tr>
<tr>
<td>38</td>
<td>43, F</td>
<td>basilar</td>
<td>slight</td>
<td>11</td>
<td>yes</td>
<td>IV/V</td>
<td>18/60</td>
<td>103/110</td>
</tr>
<tr>
<td>53</td>
<td>57, M</td>
<td>ACoA</td>
<td>severe</td>
<td>14</td>
<td>no</td>
<td>IV</td>
<td>15/22</td>
<td>116/132</td>
</tr>
<tr>
<td>54</td>
<td>66, F</td>
<td>ICA</td>
<td>severe</td>
<td>9</td>
<td>no</td>
<td>II/IV</td>
<td>14/28</td>
<td>106/120</td>
</tr>
</tbody>
</table>

*Abbreviations: SAH = subarachnoid hemorrhage; MABP = mean arterial blood pressure; ICA = internal carotid artery; PCA = posterior cerebral artery; ACoA = anterior communicating artery; IVP = intraventricular pressure.

Briefly, the series consisted of 52 patients (35 females and 17 males) admitted to the University Clinic of Neurosurgery in Aarhus within 1 week of their initial SAH from an intracranial saccular aneurysm. Their mean age was 46 years (range 15 to 68 years). The diagnosis was confirmed by angiography on admission. Based on daily clinical assessment, the patients were graded according to the system of Hunt and Hess, and divided into three categories: Grades I–II comprised 13 patients, Grades II–III 19 patients, and Grades III–V 20 patients. The degree of cerebral vasospasm was measured on angiograms taken on admission and repeated approximately 7 days later. Severe spasm was defined as a reduction of the arterial caliber of more than 50% and slight spasm as a reduction of more than 25%.

Continuous monitoring of the IVP according to the method of Lundberg was carried out in all patients from admission for an average period of 8 days (range 2 to 16 days). Ventricular drainage of CSF was carried out when IVP exceeded 25 mm Hg. Samples of ventricular CSF were collected repeatedly and analyzed for lactate and pH. Measurements of diastolic and systolic blood pressure, pulse rate, respiratory rate, and temperature were performed hourly during the period of study. All pressures were expressed as mean pressures (diastolic pressure + one-third of the pulse pressure).

Recurrent hemorrhage occurred 11 times in nine of the 52 patients. One of these was excluded from the present study because of a failure to record IVP properly during rebleeding. Later, two other patients who rebled during continuous monitoring of IVP were included (Cases 53 and 54). Thus, this study comprises 10 patients who rebled 13 times within the first 14 days after the initial SAH. Their mean age was 53 years (range 39 to 66 years).

**Results**

**Clinical and Angiographic Findings**

The patients' age and sex, the site of the aneurysm, the degree of vasospasm, and the clinical grade before and after the repeat rupture in the 10 patients are shown in Table 1. Recurrent hemorrhage was seen more often in poor clinical condition than in good condition. Rebleeding occurred only once in patients of Grade II, eight times in Grade III patients, and four times in Grade IV patients.

Angiographic vasospasm was demonstrated in nine of the 10 patients. Slight spasm was found in three patients, and severe spasm in six (60%). In patients who did not rebleed, only 31% (13 of 42 patients) exhibited severe spasm. The difference in the percentages is significant (p < 0.001).

**Pressure Changes before Recurrent Hemorrhage**

Patients who rebled had a high and unstable blood pressure. The relationship between the courses of mean IVP and mean arterial blood pressure (MABP) in patients who rebled and in patients who did not rebled is shown in Fig. 1. Expressed as mean values for the whole period, both parameters were significantly higher in patients who rebled (p < 0.025). The frequency of arterial hypertension diagnosed before the primary aneurysm rupture was slightly higher in the patients who rebled (40%) compared to patients without rebleeding (19%), but this difference is statistically insignificant.

Sudden, short-lasting increases in IVP without concomitant clinical changes, so-called “warning episodes,” were recorded in four patients. In one of them (Case 13), this episode was followed by repeat SAH 12 hours later. Values of CSF lactate and pH, pulse rate, respiratory rate, and temperature measured...
before repeat rupture did not show any special trends that might predict a recurrent SAH.

**Pressure Changes during Recurrent Hemorrhage**

The recurrent hemorrhages recorded occurred between the 3rd and 14th day after the initial SAH (mean 9.8th day). Before re-rupture, intermittent ventricular drainage was carried out in eight patients. However, when the rupture took place the drainage system was open in only three cases. In three additional cases it was opened immediately upon rupture.

An example of the development of IVP in a patient subjected to drainage during rebleeding is shown in Fig. 2. The mean IVP values recorded minute by minute in all six cases undergoing drainage during re-rupture are seen in Fig. 3. In 1 to 2 minutes, IVP rose to a level between the diastolic and systolic blood

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**Fig. 1.** The relationship between intraventricular pressure (IVP) and mean arterial blood pressure (MABP) in 31 patients who did not rebleed (broken lines) and 10 patients who rebled (solid lines) during the first 12 days after the initial aneurysmal rupture. Bars indicate mean ± standard error.

**Fig. 2.** Recording of the intraventricular pressure during recurrent hemorrhage in a 64-year-old woman with a right internal carotid artery aneurysm (Case 27). Arrow indicates the start of cerebrospinal fluid drainage.

**Fig. 3.** The course of the intraventricular pressure (IVP) in six cases of recurrent hemorrhage subjected to cerebrospinal fluid drainage during rerupture. Bars indicate mean ± standard error.
Intracranial pressure changes after aneurysm rupture

Fig. 4. Left: Recording of the intraventricular pressure during recurrent hemorrhage in a 50-year-old woman with a left internal carotid aneurysm (Case 13). Right: The course of the intraventricular pressure (IVP) in seven cases of recurrent hemorrhage not subjected to cerebrospinal fluid drainage. Bars indicate mean ± standard error.

pressure. In three instances in which the drainage system was open before the onset of re-rupture, the pressure rise was of shorter duration. A steady-state pressure level considerably higher than before the rebleed was reached in approximately 5 minutes.

An example of the development of IVP in a patient not subjected to drainage (Case 13), and the mean IVP values during all “spontaneous” rebleeds are seen in Fig. 4. The pressure rise lasted 2 to 3 minutes, whereupon the pressure decreased slowly to reach a steady state at about the prerupture level in approximately 10 minutes.

The difference between the two patterns of IVP and their relationship to the MABP, which was measured shortly before and immediately after rebleed, is depicted schematically in Fig. 5. Even though drainage shortened the duration of the pressure peak, the subsequent steady-state pressure level was significantly higher than after “spontaneous” rupture. Consequently, the range between IVP and the limit of the critical perfusion pressure was considerably reduced.

The Final Outcome

Recurrent hemorrhage had a deleterious effect on the prognosis. All five patients subjected to drainage during rebleeding died. In four of them (Cases 17, 27, 34, and 38), autopsy disclosed a large intracerebral hemorrhage. The fifth patient (Case 30) died from another re-rupture 2 days after the first one.

Three of the five patients not subjected to drainage died. One of them (Case 13) died from increasing IVP, indicating progressive cerebral edema. Autopsy was not performed on this patient. The two others (Cases 22 and 53) slowly improved after repeat SAH but died from a second re-rupture 5 and 14 days later, respectively. The two last patients in this group (Cases 33 and 54) improved to Grade III and were operated on but remained permanently disabled.

Discussion

The primary purpose of operating on a ruptured cerebral saccular aneurysm is to prevent recurrent hemorrhage by definite obliteration of the aneurysmal sac. There is no general agreement as to the optimum time for surgical intervention. Early surgery has been proposed to reduce the incidence of rebleeding. However, patients in clinical Grade III or worse are usually not considered fit for operation. Despite reduction in early rebleeding by antifibrinolytic therapy, recurrent hemorrhage still constitutes a major cause

Fig. 5. Schematic drawing of the typical course of the intraventricular pressure during recurrent hemorrhage with (Drainage) and without (Spontaneous) the use of cerebrospinal fluid drainage in relation to mean arterial blood pressure (MABP). Shaded area represents the region of critical perfusion pressure.
of death in such patients. Attempts have been made to identify objective factors that might distinguish patients who will re-bleed from patients who will not. Richardson, et al., listed eight factors of prognostic significance in predicting re-rupture, including age, sex, level of consciousness, diastolic and systolic blood pressure, direction of the aneurysm, the length-breadth ratio of the aneurysm, and the time elapsed since the initial rupture. Based on a necropsy series of 89 patients who had suffered repeat rupture, Crompton showed that the size, form, and location of the aneurysm were of importance. Furthermore, certain clinical signs, attributed to minor leakage of blood, have been suggested to portend a major hemorrhage.

The present small series was characterized by a large percentage of poor-risk patients. This is in accordance with recent results of the Cooperative Aneurysm Study, in which a rebleed rate of 25% was reported in patients in poor condition as opposed to only 9.2% in patients in good condition. One explanation for the preponderance of poor-risk patients may be the presence of vasospasm. Nine of our 10 patients showed angiographic vasospasm. It has been suggested that arterial spasm should be a protective mechanism against further major bleeding. However, in a series of 174 patients, Du Boulay and Gado did not find statistical evidence to support this theory. Our results indicate that vasospasm might even be a factor associated with an increased risk of rebleeding. In fact, the most severe hemorrhages associated with concomitant hematoma were observed in patients with severe spasm. Even though acute spasm may be a protective mechanism, the persistence of irregular narrowing of the arterial lumen in delayed vasospasm might theoretically influence the blood stream in the aneurysm-bearing artery by initiating turbulence that may increase the stress on the aneurysmal wall, thereby increasing the likelihood of re-rupture. Furthermore, we have previously reported a significantly diminished CBF and impaired autoregulation in patients with severe spasm. which indicates that the maintenance of an adequate CBF during the pressure peak of a re-rupture is probably not possible.

Patients who rebled had a raised and unstable blood pressure. Changes in systemic blood pressure are reflected by comparable changes in intra-aneurysmal pressure, and the probability of rupture therefore increases with an increase in MABP, and in particular with sudden increases. Consequently, a reduction and stabilization of MABP seems important to obtain in these patients. It is likely that a raised MABP in most of our patients was secondary to a raised IVP. A reduction of the blood pressure alone will, in the case of unaltered IVP, involve a concomitant and undesirable reduction of the cerebral perfusion pressure. Thus, a reduction of an increased IVP alone or in combination with antihypertensive therapy would be rational.

The risk of provoking a recurrent hemorrhage by reducing a raised ICP toward the normal level has been stressed previously. However, Sundbärg and Ponten, in their large series of 127 patients who underwent continuous ventricular drainage to a level of 15 mm Hg, reported a rebleed rate of 16%, which did not suggest that repeat SAH was provoked by drainage. In the present study in patients with intermittent drainage of CSF to a level of 20 to 25 mm Hg, we found a rebleed rate of 17%. It is remarkable that recurrent hemorrhage occurred almost exclusively in patients with a raised IVP and, further, that the drainage system was open at the time of rerupture in only three of 13 instances. Thus, our results do not suggest that cautious drainage of CSF through a ventricular catheter implies an increased risk of rebleeding.

The changes in IVP recorded during recurrent hemorrhage showed a sudden steep rise reaching a level between systolic and diastolic blood pressure in 1 to 2 minutes, followed by a gradual decrease to a steady-state level in about 10 minutes. A transient rise in MABP was often seen (Cushing response). These observations are in accordance with experimental findings. Nornes and Magnaes described two different patterns of ICP recorded during aneurysm rupture. Type 1 represented a pressure peak followed by a return of ICP to the prerupture level, after which the pressure slowly increased over the following days due to cerebral edema. Type 2 was a more severe bleed with formation of a hematoma and a subsequent sustained high-pressure level until death. The use of drainage in the present study makes a comparison between the two series difficult. Yet, the development of IVP in our "spontaneous" rebleedings was very much like Type 1 of Nornes and Magnaes, while drainage of CSF appeared to elicit a pressure pattern comparable to their Type 2, and to increase the volume of extravasated blood.

One of the important physiological factors operative during aneurysm rupture is the outflow of CSF. The stanching effect of the counterpressure, which builds up during bleeding, is probably diminished and delayed if CSF is vented suddenly and in large amounts. Our results showed that a steady-state IVP was reached sooner and at a higher level in patients subjected to drainage of CSF. Furthermore, large fresh hematomas were found at autopsy in most of these patients. These facts suggest that ventricular drainage performed while rerupture is in progress impairs the natural mechanisms leading to arrest of hemorrhage.

The clinical implications of our findings may be to reduce and stabilize both a raised ICP and a raised blood pressure during the initial phase after rupture of an aneurysm. Furthermore, drainage of CSF should probably not be performed until elapse of 10 to 15 minutes after a recurrent hemorrhage when ICP has reached a steady-state level.
Intracranial pressure changes after aneurysm rupture

References


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