The role of intracranial pressure in the arrest of hemorrhage in patients with ruptured intracranial aneurysm

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Intracranial epidural pressure (EDP) was recorded in 29 patients admitted with ruptured saccular aneurysms, but unfit for immediate surgery. In 10 patients a total of 13 recurrent hemorrhages were recorded; the average time before rerupture was 7.7 days after the last hemorrhage. Ten of the rebleedings started from intracranial pressure levels at or below 400 mm H2O whereas three started from higher prerupture levels. The observations indicate an increasing risk of rebleeding as the epidural pressure decreases toward normal pressure. Most repeat hemorrhages are arrested at EDP levels about that of the diastolic blood pressure. The resulting reduced pressure gradient across the aneurysm wall is important in the arrest of hemorrhage and the maintenance of hemostasis. Measurement of internal carotid artery blood flow during the acute stage of recurrent hemorrhage shows marked changes in blood flow pattern. Arrest of blood flow occurred only at the end of diastole; forward flow occurred only during systole. The effect of intracranial-pressure-buffering mechanisms on the increased EDP after rupture is discussed. Activation of these mechanisms may reduce the EDP to acceptable pressure levels within minutes and should be awaited before decompressive management is considered. Continuous recording of the EDP in patients unfit for immediate aneurysm surgery is important in the selection of the optimal time for operation.

Key Words - intracranial pressure · epidural pressure · carotid artery blood flow · cerebral aneurysm · subarachnoid hemorrhage · hemostasis
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pressure, indicating that specific hemodynamic factors were involved.

In the present paper we discuss the dangerous as well as beneficial effects of the instant increase in ICP at the time of aneurysm rupture, and the importance of continuous ICP recording following subarachnoid hemorrhage (SAH) to evaluate the risk of rebleeding and select the optimal time for surgery.

Material and Methods

A miniature pressure transducer implanted epidurally in the frontoparietal region was used to record intracranial pressure continuously. A Speedomax H or a Honeywell Electronik 194 recorder was used for registration. The probes were implanted for 1 to 29 days. Blood flow measurements were made on the internal carotid artery (ICA) on the side of the aneurysm in two patients selected for graded occlusion of the ICA with a Selverstone clamp. An electromagnetic blood flowmeter (Nycotron, Drammen, Norway) connected to an Elema 81 ink jet recorder was used for this purpose. The electromagnetic flow probes were implanted for 1 and 18 days respectively. Blood pressure was measured by the external brachial method every hour.

We studied 29 patients admitted with SAH due to a ruptured sacular aneurysm; each of these patients was unfit for immediate surgery. The patients suffering recurrent hemorrhage while under observation are presented in Table 1; three of these had two SAH's during the period of registration and are listed twice.

Recurrent hemorrhage was classified as previously described. The so-called SAH Type 1 is characterized by edema and only minimal hematoma, while the SAH Type 2 always has evidence of massive hematoma. The terms "ischemic-edematous lesion" and "hemorrhagic-compressive lesion" have been used for these two conditions respectively. Figure 1 shows the EDP pattern in these two types of recurrent hemorrhage.

**TABLE 1**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>Site of Aneurysm</th>
<th>SAH Type</th>
<th>Before Rupture (mm Hg)</th>
<th>After Rupture* (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>B.P. EDP</td>
<td>B.P. EDP</td>
</tr>
<tr>
<td>1</td>
<td>29</td>
<td>M</td>
<td>anterior cerebral artery</td>
<td>2</td>
<td>145/85 37</td>
<td>225/130 160</td>
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<tr>
<td>2</td>
<td>26</td>
<td>M</td>
<td>middle cerebral artery</td>
<td>1</td>
<td>140/80 12</td>
<td>140/80 85</td>
</tr>
<tr>
<td>3</td>
<td>59</td>
<td>F</td>
<td>anterior cerebral artery</td>
<td>1</td>
<td>140/80 12</td>
<td>230/110 160</td>
</tr>
<tr>
<td>4</td>
<td>33</td>
<td>M</td>
<td>anterior cerebral artery</td>
<td>2</td>
<td>235/135 160</td>
<td>240/110 85</td>
</tr>
<tr>
<td>5</td>
<td>29</td>
<td>M</td>
<td>anterior cerebral artery</td>
<td>2</td>
<td>240/110 85</td>
<td>175/115 140</td>
</tr>
<tr>
<td>6</td>
<td>40</td>
<td>M</td>
<td>internal carotid artery</td>
<td>1</td>
<td>160/90 18</td>
<td>160/90 90</td>
</tr>
<tr>
<td>7</td>
<td>31</td>
<td>M</td>
<td>internal carotid artery</td>
<td>2</td>
<td>160/90 18</td>
<td>230/125 170</td>
</tr>
<tr>
<td>8</td>
<td>52</td>
<td>F</td>
<td>middle cerebral artery</td>
<td>1</td>
<td>140/80 12</td>
<td>140/80 70</td>
</tr>
<tr>
<td>9</td>
<td>38</td>
<td>F</td>
<td>vertebral-basilar artery</td>
<td>1</td>
<td>110/70 23</td>
<td>190/110 150</td>
</tr>
<tr>
<td>10</td>
<td>38</td>
<td>F</td>
<td>anterior cerebral artery</td>
<td>1</td>
<td>150/80 23</td>
<td>185/120 150</td>
</tr>
</tbody>
</table>

* 1 or 2 minutes after rupture.
Results

Satisfactory recordings were obtained in all cases except one in which the transducer broke down 24 hours after implantation. No complications attributed to the implantation of the pressure transducer were observed.

Recurrence Hemorrhage

A total of 13 recurrent hemorrhages verified by CSF tap or operation were recorded (Table 1). Eight of these were SAH Type 1 and five, SAH Type 2. The EDP tracings obtained prior to rerupture are shown in Fig. 2. All of the recorded reruptures except one occurred within 14 days after the last SAH (average 7.7 days). The SAH Type 1 started at an average EDP of 280 mm H$_2$O while the SAH Type 2 ruptured at an average level of 380 mm H$_2$O. The approximate pressure gradients (BP-EDP) can be seen in Table 1. Only two patients had an increased BP prior to rerupture.

Ten of the reruptures occurred at or below a pressure level of 400 mm H$_2$O whereas only three started from higher levels (Fig. 2). The remaining pressure recordings, not culminating in recurrent hemorrhage, are presented in Fig. 3. Since pressures following SAH Type 2 rebleedings are extremely high and usually lead to death within hours, this group will not be discussed further. For a comparable reason, one patient (Case 10) who suffered a SAH Type 1 hemorrhage and died within 4 hours, is not presented in Fig. 3. This leaves a total number of 220 registration days in patients in danger of rebleeding. The patients were considered at risk until the day they died or were treated by intracranial surgery.

The data presented in Fig. 2 (recurrent hemorrhages) and in Table 2 (recurrent hemorrhages) are summarized in Table 2.

### Table 2

<table>
<thead>
<tr>
<th>Group</th>
<th>Days At</th>
<th>Pressure Levels Above 400 mm H$_2$O</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>recurrent hemorrhage</td>
<td>73</td>
<td>27</td>
<td>0.37 (P1)</td>
</tr>
<tr>
<td>no recurrent hemorrhage</td>
<td>147</td>
<td>94</td>
<td>0.64 (P2)</td>
</tr>
</tbody>
</table>

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**Fig. 2.** Record of epidural pressure (EDP) after SAH up to time of rerupture in 13 recurrent hemorrhages. Horizontal interrupted line = 400 mm H$_2$O.
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EDP
mm H₂O

Operation
Cons. treat.
Dead

Fig. 3. EDP records in 23 SAH patients at risk of rebleeding whose pressure sequence did not end in rerupture. F = failure (transducer breakdown). The terminal stage with a BP drop to zero and concomitant EDP fall seen with death is not shown.

hemorrhage) show a total of 73 days of which 27 registration days were at pressure levels above 400 mm H₂O. In Fig. 3 (no recurrent hemorrhage) the figures are 147 and 94 respectively. Table 2 shows the distribution of observation days above and below 400 mm H₂O within these two groups. From these data we can calculate the proportion of observation days with days at pressures over 400 mm H₂O in each group. The difference between these two proportions (p₁ and p₂) has been tested using the Gaussian approximation test. The EDP was higher in the non-rebleeding group, and the difference between the two groups was significant at the 1% level.

No Recurrent Hemorrhage

Figure 4 shows that the “no-rebleed” group has a comparatively high number of observations during the period where there is an increased risk of rebleeding (5th - 11th day\(^{15}\)). This excludes the possibility that the observed difference is due to a reduced number of observations during the critical period. In contrast, the dip in the number of patients with a recurrent hemorrhage is caused by that incident itself.

Autopsy showed that edema was the main

Fig. 4. Number of patients monitored per day after SAH.
cause of pressure increase in the six patients that died. Schutta and coworkers²⁹ have shown how formation of edema may induce high ICP.

**Decompressive Management at Rupture**

Five of the patients with recorded recurrent SAH Type 1 were not subjected to any kind of decompressive management during the early period following rebleeding. These patients all had single pressure peaks in their records.

Three patients with a SAH Type 1 hemorrhage were subjected to decompressive management. Figure 5 shows in Case 7 the effect of immediately reducing the EDP from 1100 to about 625 mm H₂O by lumbar CSF tap (5 ml). This was followed by an instant increase to more than 2100 mm H₂O, forming a second pressure peak in the record. The tracing thereafter shows gradual fall in EDP again.

The patient in Case 10 was given 75 gm of mannitol intravenously during the period of the pressure peak (Fig. 6). The pressure then rapidly decreased below the pre-rupture pressure level. From this low level, never previously seen in this patient, the tracing developed a succession of pressure peaks.
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Fig. 7. Case 4. Three sequences presenting epidural pressure, internal carotid artery blood flow, and brachial blood pressure. Rebleeding occurred during the second and third sequence. A and B refer to the original flow tracings in Fig. 8. Electromagnetic flow recording was made discontinuously. Note vasopressor response after the first but not the second rehemorrhage.

Blood Flow Pattern at Rupture

The patient in Case 4, who had been selected for graded occlusion of the ICA with an electromagnetic blood flow probe implanted, had a recurrent hemorrhage before occlusion could be accomplished. The ICA blood flow (aneurysm side) was 160 ml/min before rerupture at an EDP of 250 mm H₂O (18 mm Hg) and a BP of 140/100 mm Hg (Fig. 7). The tracings go on to show pressure changes as high as 1850 mm H₂O (136 mm Hg) at the time of hemorrhage; BP measurements revealed a vasopressor response to 180/125 mm Hg. The registered blood flow during this period of EDP elevation was about 60 ml/min, as indicated by B in Fig. 7. The original flow record with tracings for mean flow and pulsatile, instantaneous flow from this sequence is shown in Fig. 8 together with the flow record (A) before rerupture. The tracing representing instantaneous blood flow shows forward flow mainly during systole and flow arrest at the end of diastole.

The patient was deeply comatose; after 15 minutes without change in EDP or clinical condition a minor spinal CSF tap was made. The subsequent fall in EDP and increase in blood flow to about 120 ml/min only resulted in a slight clinical improvement. One hour later the patient suffered his second rehemorrhage. This time there were no signs of vasopressor response. When the flowmeter immediately was connected, the ICA flow was zero. This time a lumbar puncture with removal of 5 ml of CSF had no effect on the intracranial pressure or the ICA flow. The EDP, ICA, and BP together clearly indicated a state of “brain tamponade” which resulted in a fatal outcome within minutes.

Discussion

When the ICP approaches the systolic BP at rupture, both aneurysm leakage and cerebral blood flow are arrested. This is the ultimate pressure state in what we have designated the “hemorrhagic-compressive”
lesions seen in SAH Type 2. Nonfilling of the cerebral vessels during angiography has been observed under similar conditions and the term “brain tamponade” is descriptive of this picture.

Our data show that a majority of rebleedings are arrested at EDP levels of approximately the diastolic BP. The EDP then returns to considerably lower levels within minutes. This pressure pattern is typical of SAH Type 1. The pressure gradient across the aneurysm wall, as well as the cerebral perfusion pressure, must be markedly reduced during the period of the pressure peak, and is presumably about zero during diastole. The diastolic arrest of the internal carotid artery blood flow seen in Fig. 7 strongly supports this concept.

A similar observation has been made by Greenfield and Tindall who used an electromagnetic flowmeter in patients with intracranial tumors. They observed arrest of carotid blood flow at the end of diastole when the CSF was artificially elevated to about 950 mm H₂O (70 mm Hg) at a diastolic BP of about 80 mm Hg.

We were concerned about the high ICP during the pressure peaks, which was the reason for the decompressive management instituted in four patients. The records show how such decompression instantly or within minutes is followed by new pressure peaks supposed to be hemorrhagic outbursts from the aneurysm. However, it is not proved that these secondary peaks are caused by the extravasation of blood alone. They may possibly be due to rapid variations in cerebral blood volume caused by vasomotor instability. We have previously described pressure peaks unrelated to aneurysm leakage, so-called “warning episodes.”

The lesson of our experience with acute decompressive management during the period of pressure peak in SAH is that the effects of physiological buffer mechanisms should be awaited before such treatment is considered. As seen from the records these mechanisms often dramatically reduce even very high intracranial pressures within minutes after the SAH. This EDP pattern is attributed to two factors: one is the arrest of hemorrhage, and the other is maintenance of hemostasis during a period when pressure-buffering mechanisms markedly reduce the ICP. The main buffering mechanism is supposed to be an outflow of CSF and extravasated blood along the craniospinal axis.

Two different mechanisms are generally considered of importance in securing cere-
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Intracranial pressure plays a significant role in the arrest of subarachnoid hemorrhage (SAH), primarily through autoregulation and the vasopressor response. A third mechanism, the pressure buffering mechanism of the intracranial compartments, is also critical.

The reduced pressure gradient over the aneurysm wall caused by increased ICP at rupture may be of special importance in stanching hemorrhage, because an aneurysm has a defective muscle layer that probably does not possess the ability to contract. However, the aneurysm wall contains collagen fibrils, and it has been shown that exposed collagen fibrils initiate platelet aggregation. It is reasonable to assume that the hemodynamic state described with reduced pressure gradient and the diastolic arrest of leakage, is an important factor in the initial mooring of platelets at the site of rupture and for the subsequent formation of a platelet plug.

Experimentally, it has been shown that the platelet plug builds up in the course of 1 or 2 minutes at the site of vessel injury. In the following minutes, the bleeding may occur through channels in the plug during the time the plug is gradually increased in strength by platelets becoming tightly adherent to each other. Permanent hemostasis is obtained when the strength of the platelet plug and the surrounding pressure can counterbalance the forces of aneurysm blood pressure. A stable BP and ICP during this critical period is important to maintain hemostasis.

It should be pointed out that during the first 24 hours after a platelet plug has been formed, fibrin is deposited in the platelet mass. If the coagulation system acts normally, this process appears to increase the hemostatic efficiency of the plug, which by this time rather should be called the hemostatic clot.

Dissolution of the hemostatic clot during the first 2 weeks due to normal fibrinolytic activity is considered an important cause of recurrent hemorrhage. At the third week, granulation tissue provides a far better support and probably accounts for the lower frequency of rebleeding.

Furthermore, arterial spasms are supposed to aid hemostasis by reducing aneurysmal blood pressure. The damping effect on the pulsatile components of flow and pressure caused by the constriction of feeding vessels has been demonstrated by Jain and Tominaga. These data support the idea that the risk of rebleeding during the first 2 weeks after aneurysmal rupture increases as the ICP falls. It is reasonable to believe that the relatively low frequency of recurrent hemorrhage during the first week after SAH may be due to the strength of the hemostatic clot, the presence of arterial spasms, or the support contributed by the increased intracranial pressure. The specific tendency for rebleeding to occur between the 7th and the 14th day after SAH may thus be caused by reduced clot strength due to fibrinolysis, release of spasms in feeding arteries, and the return of the intracranial pressure to normal.

Klafta and Hamby have reported improved surgical results when aneurysm surgery was delayed until lumbar CSF pressure had fallen to about 200 mm Hg, even though there had been no change in the clinical status of the patient. These findings are in accordance with our observations.

Thus, the continuous registration of epidural pressure in these patients helps define the optimal state balancing these opposing considerations. We conclude that it seems useful to consider an epidural pressure of about 400 mm Hg as a guideline in the timing of aneurysm surgery.

References


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