

# **Relationship between Intracranial Pressure and the Development of Vasospasm after Aneurysmal Subarachnoid Hemorrhage**

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## **Abstract**

The relationship between intracranial pressure (ICP) and the development of vasospasm after subarachnoid hemorrhage caused by the rupture of an intracranial aneurysm was investigated. Eleven patients were divided into high (6 cases) and low (5 cases) ICP groups based on ICP data obtained during the perioperative period by continuous ICP monitoring. Transcranial Doppler ultrasonography was performed every 24 hours for 7 days and the severity, distribution, and duration of vasospasm were assessed. The high ICP group tended to have severe, prolonged, and diffuse vasospasm compared with the low ICP group. However, only duration of vasospasm was statistically different. The relationship between cerebral perfusion pressure (CPP) and the development of vasospasm was also examined. CPP had a less significant effect than ICP although similar tendencies for high ICP and low CPP were observed. High ICP worsens vasospasm and treatment for decreasing ICP with perioperative ICP monitoring has potential for avoiding the development of vasospasm.

Key words: intracranial pressure, transcranial Doppler ultrasonography, vasospasm

## **Introduction**

Although the outcome of patients with aneurysmal subarachnoid hemorrhage (SAH) has been improved with acute microsurgical surgery and the use of nimodipine,<sup>1,15,17</sup> delayed ischemic deficits after the onset of vasospasm are still a major problem.<sup>12,13,15</sup> There are a considerable number of factors which influence vasospasm, including age,<sup>5,22,25</sup> the amount of blood observed on the initial computed tomography (CT) scan,<sup>10,22</sup> and preoperative grade.<sup>10,22,25</sup> Although measuring intracranial pressure (ICP) has become a standard technique for patients who suffer from increasing ICP,<sup>6,21</sup> the effect of ICP on the development of vasospasm has not been reported, with the exception of the relationship between ICP and traumatic SAH.<sup>30</sup> This study analyzed the relationship between ICP and the development of vasospasm in patients with aneurysmal SAH.

## **Subjects and Methods**

Twenty-three patients were admitted to the Neurosurgical Service at Harborview Medical Center for SAH due to intracerebral aneurysmal rupture confirmed by cerebral angiography between January 1, 1997 and March 31, 1997. Eleven patients, four males and seven females aged 23 to 66 years (mean 47.5 years), who underwent surgical clipping of the aneurysm within 72 hours after the onset of SAH were entered into this study (Table 1). The aneurysms were located as follows: five on the anterior communicating artery, three on the middle cerebral artery (MCA), one at the basilar tip, one at the bifurcation of the vertebral artery (VA) and the posterior inferior cerebellar artery, and one at the bifurcation of the basilar artery (BA) and the superior cerebellar artery.

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**Table 1** Characteristics of patients

Case No.	Age	Sex	Location of aneurysm	Grade*	VD	CT group**	ICP group	CCP group
1	42	M	lt MCA	2	—	2	low	high
2	54	F	lt VA-PICA	2	preop.	2	high	high
3	39	M	AcomA	2	POD3	2	high	low
4	63	M	AcomA	1	—	3	high	low
5	66	F	lt MCA	4	preop.	3	high	low
6	50	F	AcomA	2	—	3	high	low
7	43	F	AcomA	5	preop.	3	low	high
8	50	F	AcomA	2	—	3	low	low
9	42	F	lt MCA	2	—	2	low	low
10	51	F	BA top	3	preop.	3	high	high
11	23	M	lt BA-SCA	3	—	2	low	high

\*According to Hunt and Hess classification. \*\*According to Fisher grade. AcomA: anterior communicating artery, BA: basilar artery, BA-SCA: basilar artery-superior cerebellar artery, CCP: cerebral perfusion pressure, CT: computed tomography, ICP: intracranial pressure, MCA: middle cerebral artery, POD: postoperative date, preop.: preoperatively, VA-PICA: vertebral artery-posterior inferior cerebellar artery, VD: ventricular drainage.

### I. ICP monitoring

ICP was measured continuously using a Camino OLM ICP monitoring kit (Camino Laboratories, San Diego, Calif., U.S.A.). The device was inserted preoperatively in all patients with the exception of Case 4 with the symptom of grade I, in whom the device was placed immediately after aneurysm clipping. ICP monitor calibration was performed more than once a day. The ICP monitor usually remained in place for more than 7 days following surgery, and until the vasospasm diagnosis with transcranial Doppler (TCD) ultrasonography improved to at least mild. Two patients continuously revealed no vasospasm in any arteries, were alert and totally orientated, with no focal deficits observed for 5 days following surgery. In these patients, the ICP monitor was removed earlier to start physical therapy out of bed. The mean ICP over 6 hours prior to aneurysm clipping was used as the preoperative ICP. Postoperative ICP was recorded every hour and the mean ICP during every 6 hour period was calculated.

### II. TCD ultrasonography

TCD was performed daily in these patients to detect vasospasm. Details of this technique have already been reported.<sup>20)</sup> Flow velocities were recorded from the main trunk of the MCA ( $M_1$  segment), proximal branches of the MCA ( $M_2$  segment), cavernous and terminal portions of the internal carotid artery (ICA), precommunicating segment of the anterior cerebral artery (ACA  $A_1$  segment), precommunicating segment of the posterior cerebral artery (PCA  $P_1$  segment), proximal postcommunicating segment of the PCA ( $P_2$ ), the BA, and the VA. In order to

calculate the Lindegaard ratio,<sup>16)</sup> flow velocity was measured in the retromandibular extracranial ICA.

For evaluation of the MCA and ICA, time averaged maximum velocities (TAMV) of 120 cm/sec or more was considered as mild vasospasm, 150 cm/sec or more as moderate vasospasm, and velocities of 200 cm/sec or greater as severe vasospasm. The Lindegaard ratio was also used. A value of less than 3.0 indicated no vasospasm, a ratio of 3.0–5.9 indicated mild to moderate vasospasm, and a ratio of 6.0 or more indicated severe vasospasm.

For the ACA, a TAMV of 130 cm/sec was considered to show vasospasm. However, in the setting of ICA/MCA vasospasm, the ipsilateral ACA may show increased flow velocity due to action as a collateral, so the interpretation of vasospasm versus collateral flow was considered.

For the PCA, a TAMV of 110 cm/sec or more was considered to show vasospasm. Again in the setting of ICA/MCA vasospasm, the ipsilateral PCA may increase in flow velocity due to action as a collateral, so the interpretation of vasospasm versus collateral flow was considered.

The degree of vasospasm is difficult to categorize in the VA and BA due to the variations in the caliber of these arteries and the tortuosity which influences the angle of insonation and the calculation of velocity. Vasospasm was considered to be present if the TAMV was 80 cm/sec or greater in the VA and 95 cm/sec or greater in the BA. Caution must be used, especially in the presence of vasospasm in the anterior circulation, as the vertebrobasilar system may also act as collaterals. The distal 1/3rd of the BA is

often technically difficult to insonate due to the depth at which it is located. TCD was performed for at least 10 days and until two successive diagnosis with normal flow velocities in all vessels were obtained.

### III. Treatment protocol

Patients with SAH usually had a Camino ICP monitor inserted into the subdural space at the time of admission. If significant hydrocephalus or a high ICP was observed, a ventriculostomy was performed before cerebral angiography. Cerebral angiography was done within one day of admission and craniotomy with aneurysm clipping was performed within 72 hours after the onset of SAH. Postoperative management included nimodipine, 240 mg/day for 3 weeks. Central venous pressure was measured and maintained at more than 8 cmH<sub>2</sub>O by administering 500 ml of 5% albumin bolus if necessary. Cerebral perfusion pressure (CPP) was kept at 70 mmHg or greater by use of a dopamine drip. If ICP exceeded 20 mmHg, a 2 g/kg mannitol drip was used every 6 hours and hyperventilation was also started. If this protocol did not work well, the ventricular drainage set at 10 cm above the external auditory meatus was lowered to the same height. Patients might undergo ventriculostomy at that time, if not performed before. During follow-up with TCD, cerebral angiography was performed for the diagnosis of severe vasospasm.

### IV. Statistical analysis

To analyze the relationship between ICP and the development of vasospasm, the 11 patients were divided into two groups: six patients with ICP exceeding 20 mmHg more than once (high ICP group) and five patients with ICP continuously lower than 20 mmHg (low ICP group) (Table 2). To analyze the relationship between CPP and the development of vasospasm, the patients were again divided into two groups: six patients with CPP less than 70 mmHg (low CPP group) and five with CPP more than 70 mmHg continuously (high CPP group) (Table 3). The ICP and CPP groups were compared, showing two

patients had high ICP and high CPP, four had high ICP and low CPP, three had low ICP and high CPP, and two had low ICP and low CPP.

Vasospasm was considered from three aspects: severity, duration, and distribution. The severity was expressed by the most severe TCD diagnosis on the ICA, MCA, or ACA within 7 days. The duration of vasospasm was determined by the number of consecutive days for which the TCD diagnosis of moderate or severe vasospasm of the MCA, ACA, or ICA was made. The distribution of vasospasm was based on division of all vessels measured by TCD into three areas: the first and second consisting of the ICA, MCA, and ACA on each side, and the third consisting of the posterior circulation including the bilateral PCAs, VAs, and the BA. If at least one vessel was diagnosed with vasospasm in any of the three areas, that territory was considered to have vasospasm.

In each ICP group, the following parameters were determined: the number of patients with severe vasospasm, the number of patients with duration of vasospasm exceeding 6 days, and the number of patients with vasospasm involving all three areas. The same data was determined for the CPP groups. These data were analyzed statistically using a contingency table with Fisher's exact probability test. Fisher's exact probability < 0.05 was considered significant.

## Results

Overall changes in ICP are seen in Fig. 1. Table 2 shows the difference between the high ICP and the low ICP groups. Severe vasospasm was observed in only three patients in the high ICP group. Four patients had moderate vasospasm lasting more than 6 days in the high ICP group. There were no such cases in the low ICP group. A statistically significant difference was observed relative to the duration of vasospasm ( $p = 0.045$ ). Five of six patients in the high ICP group were affected by vasospasm in all three areas, but only one of five patients in the low ICP group.

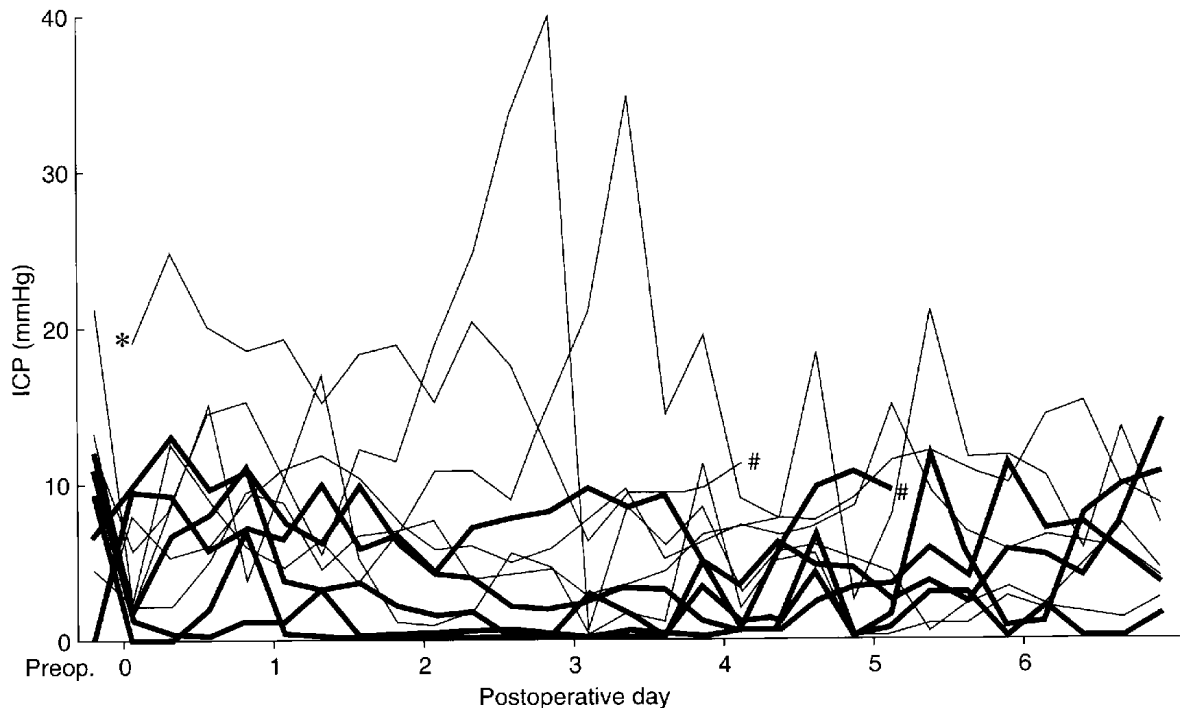
**Table 2 Comparison of intracranial pressure (ICP) groups**

	Severity		Duration*		Distribution	
	Severe	Less than moderate	≥ 6 days	≤ 5 days	All areas	≤ 2 areas
High ICP group (6 cases)	3	3	4	2	5	1
Low ICP group (5 cases)	0	5	0	5	1	4

\* $p = 0.045$ .

**Table 3 Comparison of cerebral perfusion pressure (CPP) groups**

	Severity		Duration*		Distribution	
	Severe	Less than moderate	$\geq 6$ days	$\leq 5$ days	All areas	$\leq 2$ areas
Low CPP group (6 cases)	3	3	3	3	3	3
High CPP group (5 cases)	0	5	1	4	3	2



**Fig. 1** Sequential intracranial pressure (ICP) changes in all patients. ICP values are the mean over 6 hours. Low ICP group patients (thick lines) tended to have an ICP is lower or around 10 mmHg, whereas high ICP group patients (thin lines) tended to have a spike of high ICP. \*Preoperative data not obtained. #Discontinuation of ICP monitoring.

The differences between the low CPP group and the high CPP group show the tendency for the low CPP group to have severe, prolonged, and diffuse vasospasm, but no statistical significance was obtained (Table 3).

The treatment protocol for lowering ICP was successful and ICP decreased to less than 20 mmHg within one day. In Case 3, the ventriculostomy was performed on the 3rd day after the clipping and was effective to decrease ICP. Three patients with severe vasospasm diagnosed by TCD (Cases 3, 5, 6) had cerebral angiography on the same day, and the presence of vasospasm was confirmed. Two patients (Cases 3, 5) developed progression of neurological deficits consistent with the vasospasm area after the

diagnosis of severe vasospasm with TCD. No progression of neurological deficits was observed in the other nine patients.

## Discussion

TCD ultrasonography is an accepted modality to detect vasospasm.<sup>8,11,26</sup> Problems with this technique include the difficulty in performing the examination<sup>31</sup> and interpretation of the results.<sup>20,31</sup> Analysis of the relationship between ICP and vasospasm should consider the fact that pronounced increases in ICP can lead to false-negative results.<sup>14</sup> We should also consider that, especially after SAH, increased blood volume due to hemodilution, loss of autoregulation,

and cerebral vasodilation may occur.<sup>30)</sup> Calculating the ratio of the velocity between the extracranial ICA and the MCA can correct for hyperdynamic flow.<sup>16)</sup> In our institution, this ratio is also used for grading the severity of vasospasm.<sup>19)</sup>

The velocity of the ACA may increase in the presence of ipsilateral MCA and/or ICA vasospasm as the ACA functions as a collateral source to that hemisphere.<sup>16,31)</sup> Therefore, simultaneous vasospasm of both the MCA/ICA and ACA on the same side is difficult to distinguish from vasospasm only of the MCA/ICA associated with high ACA velocities due to ACA collateral flow. For this reason, we divided the cerebral arteries into three areas to assess the distribution of vasospasm, although the velocities of the MCA and the ACA can be detected independently.

Posterior circulation vasospasm is more difficult to localize and quantify with TCD.<sup>27)</sup> This study used highly specific criteria, VA  $\geq$  80 cm/sec and BA  $\geq$  95 cm/sec, for the presence of vasospasm. The grading of the severity of vasospasm in the posterior circulation has not yet been established, so only data regarding the distribution of posterior circulation vasospasm was used for this study.

This study only included a few cases, but did reveal a relationship between high ICP and the development of vasospasm, and the duration of vasospasm. Previously, no relationship was observed between traumatic SAH and ICP.<sup>30)</sup> However, only the severity of vasospasm in the MCA was analyzed. We should also consider that the ICP in head-injured patients is affected by several factors such as contusion or other types of traumatic hemorrhage. Our study assessed vasospasm from three aspects, severity, duration, and distribution. Severity of vasospasm is not the only factor determining the development of delayed ischemic deficits. The duration of ischemia is also important, and diffuse vasospasm may lead to clinically significant symptoms more often.

There are a considerable number of factors that increase ICP following aneurysmal SAH. First, the normal flow of cerebrospinal fluid (CSF) is disrupted by the presence of blood clot and obstruction occurs, resulting in increased ICP.<sup>4)</sup> In our patients, a ventriculostomy was performed if the head CT revealed hydrocephalus. However, in some patients with perturbation of CSF, hydrocephalus may not be evident on CT and these patients may also have elevated ICP.<sup>9)</sup> Even after ventriculostomy, ICP cannot always be well controlled.<sup>2,3)</sup> In our series, three of six patients in the high ICP group underwent preoperative ventriculostomy. This failure to control ICP may be due to several factors. Disruption of the blood-arterial wall barrier may result in vasogenic edema and raised ICP.<sup>24)</sup> Vasoparalysis, observed with CSF

acidosis,<sup>29)</sup> may cause high ICP due to the resultant hyperemia. Finally, neurochemical effects following SAH, including atrial natriuretic factor increase,<sup>7)</sup> which are involved in increased ICP. It is important to understand that all of these predisposing factors, presence of blood clot, blood-arterial wall barrier disruption, CSF acidosis, and neurochemical substances, can also cause vasospasm without an increase in ICP. On the contrary, there is some evidence that increased ICP can be the direct cause of vasospasm. A significant association was found between high ICP and systemic hypovolemia due to the increase in sympathetic activity, which increases the incidence of cerebral ischemia.<sup>18)</sup> Experiments to find the effect of a transient elevation in ICP on subsequent infarction demonstrated that even a transient elevation in ICP enlarged the size of an infarction, showing that high ICP may relate to worsening vasospasm.<sup>28)</sup>

We suggest that both predisposing factors and the direct mechanism of elevated ICP must influence the development of vasospasm following aneurysmal SAH. Treatment to prevent high ICP should be mandatory for all patients, whether ICP relates to vasospasm directly or not. In our results, although no strong tendency was observed between CPP and the development of vasospasm, maintaining low ICP and high CPP with a general protocol<sup>23)</sup> is desirable, and such a treatment protocol may also prevent vasospasm from increasing in severity.

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## Commentary

The authors described the relationship between intracranial pressure (ICP) and the development of cerebral vasospasm in patients with aneurysmal subarachnoid hemorrhage (SAH). They concluded that high ICP should worsen vasospasm, based on the fact that patients with high ICP during the perioperative period tended to have a high incidence of vasospasm assessed by transcranial Doppler ultrasonography. Such a study would be expected to provide useful information in the management of SAH patients. However, in this study, the correlation between ICP and the time course of vasospasm was not clear, making the results difficult to interpret. Increased ICP, especially that which could not be eliminated by ventriculostomy, might be due not only to primary SAH insults but also to ischemic brain edema resulting from vasospasm. Additionally, it is also unclear whether the control of high ICP by the authors could improve vasospasm.

In general, the number of analyzed cases is not adequate to evaluate the proposition conclusively. Further clinical studies are required.

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TCD has been used to assess vasospasm, which is still a major problem in the management of the patients with SAH, because of the clear correlation between the velocity of blood flow and the vessel diameter. However, the ICP,  $PCO_2$ , or the hematocrit are also influences. In the study of TCD evaluation of vasospasm and ICP by Klingelhofer et al. (1991), an increase in ICP decreased the mean flow velocity due to a reduction of the blood flow, leading to false negative results. So, it is proposed that the index of cerebral circulatory resistance should be considered to evaluate the TCD flow velocity, when vasospasm is present with the increased ICP (over 20 mmHg). This means that a decreased flow velocity reflects a rise in ICP rather than a reduction in vasospasm if the resistance index is above the value of 0.6. It is also conceivable that there can be cases in which both high ICP and high mean flow velocity, i.e. vasospasm, are present simultaneously. Klingelhofer et al. (1991) reported there was no such cases. In my opinion I think this can be observed in cases with a mildly increased ICP. This paper provides examples of such cases in which vasospasm was detected with a high velocity on TCD in the high ICP group, which had an intermittent rise over 20 mmHg.

This paper suggests that there is a close relationship between ICP and the development of vasospasm, and ICP control may be helpful for the management of vasospasm. This gives an additional meaning to ICP control, in the approach to controlling vasospasm in SAH patients.

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This is an interesting and provocative article that suffers only from the fact that very few patients were studied. Certainly we expect CBF to be altered with significant elevations in ICP and this alteration should be aggravated by reactive vascular narrowing and everything that results from the impaired flow. That is, narrowed arterial vessels will reduce CBF; compressed outflow vessels (veins and venules) from increased ICP will also decrease CPP and CBF. The combination of arterial vasospasm and elevated ICP would be expected to have an additive (but not necessarily linear) impact on cerebral perfusion. I don't believe the authors have convinced me that "High ICP worsens vasospasm . . ." But high ICP certainly worsens the impact of vasospasm.

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The authors investigated the relationship between intracranial pressure (ICP) measured by an ICP monitor inserted into the subdural space, and the development of cerebral vasospasm detected by TCD. The study showed that the duration of cerebral vasospasm was statistically associated with high ICP, and concluded that the high ICP caused the development of vasospasm. This study is interesting because of its unique viewpoint.

However, the volume of subarachnoid blood is known to be an essential factor affecting the incidence of cerebral vasospasm, and massive subarachnoid hemorrhage also causes the increase of ICP per se. The volume of subarachnoid blood, therefore, is an independent factor that can induce both vasospasm and high ICP. In this study, the evaluation of subarachnoid blood was done by Fisher's grading which is the most prevailing method of classification, but 4 of 6 patients with Fisher grade 3 showed high ICP and the other 2 showed low ICP (Table 1 of this article). Further detailed analysis of the degree of subarachnoid blood should be considered for an evaluation of this sort. The number of cases in the study is also insufficient. Further investigation with more

*cases is necessary to make statistically supported conclusions about the relationship between cerebral vasospasm and ICP.*

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