Bedside Monitoring of Circulating Blood Volume After Subarachnoid Hemorrhage

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***Background and Purpose*—**Maintenance of an adequate intravascular volume is important in the management of patients with subarachnoid hemorrhage (SAH). The purpose of this study was to investigate the circulating blood volume (CBV) after SAH with the use of indocyanine green pulse spectrophotometry.

***Methods*—**CBV and plasma hormones related to stress and fluid regulation were measured 4 times: day 2 to 3, day 4 to 5, day 7 to 8, and day 14 in 50 consecutive patients with SAH surgically treated within 48 hours.

***Results*—**The mean value of CBV was 64 mL/kg on day 2 to 3, which gradually increased to 69 mL/kg on day 4 to 5, 71 mL/kg on day 7 to 8, and 70 mL/kg on day 14 ($P=0.005$) (control, 72 mL/kg). The clinical grades and plasma corticotropin levels were higher in patients with <60 mL/kg of CBV on day 2 to 3 ($P<0.05$ for both). There were no significant differences in other physiological and laboratory parameters such as time for surgery, estimated blood loss, levels of plasma noradrenaline, brain natriuretic peptide, serum sodium, and hematocrit. When CBV was decreased >10% of the former level, there were decreases in hematocrit ($P<0.05$), serum sodium ($P<0.01$), and serum albumin ($P<0.05$) and an increase in urinary sodium ($P<0.05$).

***Conclusions*—**A significant reduction of CBV, especially in patients with poor clinical grades, was noted after SAH and early surgery, which could not be detected by routine examinations. Anemia, central salt wasting, and hypoalbuminemia may be related to a decrease in CBV from the former level. Indocyanine green pulse spectrophotometry may be a powerful tool for the management of patients with SAH. **(*Stroke*. 2003;34:956-960.)**

Key Words: blood volume ■ cerebrovascular circulation ■ hypovolemia ■ indocyanine green ■ spectrophotometry ■ subarachnoid hemorrhage

Maintenance of an adequate intravascular volume is important in the management of patients with aneurysmal subarachnoid hemorrhage (SAH).[1–4] Hypovolemia has been shown to increase the incidence of cerebral ischemia and cerebral infarction in patients with SAH.[2,3,5] Salt wasting accompanied with hyponatremia is well recognized in patients with SAH and is frequently associated with hypovolemia, the result of a marked natriuresis.[6–8] Hypervolemia and hypertensive therapy are believed to prevent ischemic neurological deficits.[8,9] However, no monitoring system has been used for estimating circulating blood volume (CBV) other than the Swan-Ganz catheter and radioactive isotopes.[8–10] Moreover, few studies concerning the serial change of CBV after SAH and the effect of volume expander on CBV have been reported.[10]

Indocyanine green (ICG) pulse spectrophotometry has been developed to facilitate minimally invasive, subsequent measurement of CBV by progressively estimating the arterial concentration of ICG.[11] Recently, using ICG pulse spectrophotometry, we reported that the CBV decreased postoperatively to approximately four fifths of its preoperative value and gradually increased and returned to its preoperative value on day 7 after craniotomy.[11]

The purpose of this study was to investigate the change of CBV after SAH and early surgery with the use of ICG pulse spectrophotometry. The mechanism of the change in CBV after SAH was examined by measuring hormones related to stress and fluid regulation. We also sought appropriate indicators of CBV in routine examinations to estimate the change without measuring CBV.

Subjects and Methods

Patients and Management

Fifty consecutive patients (14 men and 36 women; mean±SD age, 60±13 years) with SAH were investigated in the Department of Neurosurgery, Tokyo Women's Medical University. The study was approved by the Ethical Committee of Tokyo Women's Medical University. Informed consent was obtained from the family of each patient. Eligibility criteria were clipping surgery for aneurysm within 48 hours. We started our protocol on October 1, 1999, and 50 patients were investigated among 77 SAH patients hospitalized by December 2002. Table 1 lists the clinical characteristics of the

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TABLE 1. Characteristics of 50 Consecutive Patients With Subarachnoid Hemorrhage

Characteristics	No. of Patients
Sex	
Female/Male	36/14
Age, y	
<49	11
50–59	12
60–69	14
70<	13
WFNS grade*	
1	19
2,3	14
4	10
5	7
Subarachnoid blood on CT (Fisher)[13]	
2	7
3	42
4	1
Ruptured aneurysm	
ICA	13
MCA	15
AcomA/ACA	17
BA	1
VA	4
Outcome†	
Good	30
Moderate disability	12
Severely disability	5
Persistent vegetative	2
Death	1

ICA indicates Internal carotid artery; MCA, middle cerebral artery; AComA, anterior communicating artery; ACA, anterior cerebral artery; BA, basilar artery; VA, vertebral artery.

*Classification according to World Federation Neurological Society SAH grade.[12]

†Classification according to the Glasgow Outcome Scale.[14]

patients, including assessment according to various clinical scales.[12–14]

The height and body weight were between 130 and 176 cm (mean±SD, 155±24 cm) and between 38 and 80 kg (mean±SD, 56±11 kg), respectively. The time for surgery was between 2.7 and 10 hours (mean±SD, 4.7±1.6 hours). The estimated blood loss was between 80 and 1600 mL (mean±SD, 246±252 mL). Ten patients received blood transfusion intraoperatively. Isoflurane with fentanyl was used for general anesthesia. Propofol was infused instead of isoflurane during intraoperative physiological monitoring.

Basic management of postoperative patients followed the previously described procedure.[15] Intracranial hypertension was treated with glycerol and/or cerebrospinal drainage. Blood gas, serum electrolyte, and glucose levels were determined daily until day 14 and promptly corrected if abnormal. Hyponatremia was corrected by adding an ampule(s) of 10% NaCl (20 mL) to the main fluid bag. Anemia (hematocrit <35%) was corrected by red blood cell (RBC) transfusion. When the blood pressure was >200 mm Hg, calcium antagonist was administered. Neither induced hypervolemia nor induced hypertension was used. Nutritional support by parenteral feeding was initiated on the day after surgery. The parenteral nutrition consisted basically of amino acids/electrolytes/hypertonic dextrose solutions (Aminotripa No. 2 plus 10% NaCl 20 mL [920 mL 820 kcal Na 69 mEq/L], Otsuka), multivitamins, and trace elements. Caloric intake was determined by 1.5×basal energy expenditure calculated according to the Harris-Benedict equation (therefore basic fluid intake was determined by the caloric intake). When patients could take nutrition orally during the acute stage, the parenteral nutrition was decreased.

A newly developed drug-delivery system of nicardipine for preventing vasospasm was used for SAH patients when the amount of subarachnoid blood was classified as Fisher group 3.[16] This was developed to be implanted intracranially at the time of surgery for aneurysm clipping.

Twenty-one consecutive patients (8 men and 13 women; mean±SD age, 51±18 years) were studied as controls. Together the patients harbored 11 benign and 6 malignant brain tumors and 4 unruptured aneurysms. All patients were in good condition preoperatively. The heights of the patients ranged from 145 to 175 cm (mean±SD, 157±9 cm), and their body weights ranged from 40 to 78 kg (mean±SD, 55±9 kg).

Methods

The following measurements were performed at least 4 times during the postoperative period, at 2 to 3 days (day 2 to 3), 4 to 5 days (day 4 to 5), 7 to 8 days (day 7 to 8), and 14 days (day 14) after the onset of SAH: CBV; body weight; plasma noradrenaline; plasma corticotropin; plasma cortisol; plasma aldosterone; plasma atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP); RBC, hemoglobin, and hematocrit; albumin; serum sodium and potassium; and urinary sodium.

CBV was measured by ICG pulse spectrophotometry; noradrenaline was measured by high-performance liquid chromatography; corticotropin, cortisol, aldosterone, ANP, and BNP were measured by commercially available radioimmunoassay; and other blood and chemical parameters were measured by automatic analyzer.

ICG Pulse Spectrophotometry

The integrated pulse spectrophotometry monitoring system is composed of a finger probe, a monitoring device, and a computer for recording and printing the results (DDG 2001, Nihon Kohden). The finger probe was applied to the index finger and the baseline was monitored before the injection of the ICG solution (20 mg/8 mL). Monitoring was started and blood concentration was measured at every pulse interval immediately after the administration of a dye. Hemoglobin concentration value was entered into the computer, and final ICG blood concentration time course and estimated value of CBV were printed automatically.[17,18]

Statistical Analysis

All data were stored on a personal computer and analyzed by the StatView software program (SAS Institute Inc). Data that were collected sequentially were examined by repeated-measures ANOVA or paired *t* test. Categorical data were analyzed by χ^2 test. Comparisons between groups were examined by unpaired *t* test when the dispersions of the 2 groups were equal or by Mann-Whitney *U* test. Probability values <0.05 were considered significant. Unless otherwise noted, values are presented as mean±SD.

Results

The mean value of CBV on day 2 to 3 was 64 mL/kg, which was increased to 69 mL/kg on day 4 to 5, 71 mL/kg on day 7 to 8, and 70 mL/kg on day 14 (Figure 1). There was a significant change over time (P=0.005). The mean±SD value of CBV in control patients was 72±15 mL/kg.

In the early postoperative stage (day 2 to 3), CBV varied mostly between 32.1 and 97.3 mL/kg. The grade for World Federation of Neurological Surgery (WFNS), time for surgery, estimated blood loss, and physiological and laboratory

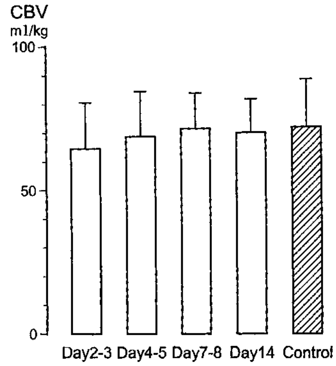

Figure 1. Serial change in CBV on day 2 to 3, day 4 to 5, day 7 to 8, and day 14 after SAH and early surgery. There was a significant change over time in CBV ($P=0.005$). Compared with control, there was a significant difference only in day 2 to 3 ($P<0.05$).

parameters were compared in patients with CBV <60 and ≥60 mL/kg (Table 2). There were significant differences in the WFNS grades ($P<0.05$) and corticotropin levels ($P<0.05$) between the 2 groups. There were no significant differences in other parameters between the 2 groups. There were also no significant differences in the laboratory and physiological parameters between <60 and ≥60 mL/kg of CBV on day 4 to 5, day 7 to 8, and day 14.

The laboratory and physiological parameters were compared when CBV levels were decreased and increased >10% of the former level. The level of CBV was decreased between day 2 to 3 and day 4 to 5 in 6 patients, between day 4 to 5 and day 7 to 8 in 7 patients, and between day 7 to 8 and day 14 in 4 patients. No RBC was transfused in these periods. Table 3 shows the physiological and laboratory parameters at the 2 time points. The mean value of CBV of 71 mL/kg was decreased to 57 mL/kg ($P<0.0001$). There were significant changes in hematocrit ($P<0.05$), serum sodium ($P<0.01$), urinary sodium ($P<0.05$), and serum albumin ($P<0.05$) between the 2 time points. No significant change was noted in central venous pressure (CVP).

The level of CBV was increased between day 2 to 3 and day 4 to 5 in 17 patients, between day 4 to 5 and day 7 to 8 in 12 patients, and between day 7 to 8 and day 14 in 7 patients. Among the total 36 episodes, RBC was transfused in 12. The physiological and laboratory parameters were compared between the 2 time points (Table 4). The mean value of CBV of 55 mL/kg was increased to 72 mL/kg ($P<0.0001$). There were no significant changes in hematocrit, serum sodium, urinary sodium, serum albumin, or CVP between the 2 time points.

Twenty-four patients received RBC transfusion. RBC was transfused between day 2 to 3 and day 4 to 5 in 8 patients, between day 4 to 5 and day 7 to 8 in 12 patients, and between day 7 to 8 and day 14 in 12 patients. The average volume of RBC transfusion was 3.2±1.0 units (1 unit=RBC from 200 mL blood) between the 2 time points. The mean value of CBV was significantly increased from 61 to 70 mL/kg ($P<0.0001$). The mean values of hematocrit before and after blood transfusion were 33.6% and 35.0% (Figure 2). There was no significant difference in hematocrit level.

No delayed ischemic neurological deficits and cerebral infarctions were experienced except in 2 patients, probably because of the newly developed drug-delivery system. The CBV of these patients was kept within a normal range.

TABLE 2. Comparison of Clinical and Laboratory Data in Subarachnoid Hemorrhage Patients With Circulating Blood Volume Between <60 mL/kg and >60 mL/kg on the Early Postoperative Stage (day 2 to 3)

CBV (mL/kg)*	CBV<60 mL/kg 50±8	CBV≥60 mL/kg 78±11
Female/Male	17/9	19/5
Age	62±11	56±16
WFNS grade*		
1	5	14
2,3	8	6
4	8	2
5	5	2
Time for surgery, h	4.6±3.0	4.7±2.0
Estimated blood loss, mL†	278±333	212±119
Hematocrit, %	37±4	37±4
Plasma albumin, g/dL	3.4±0.4	3.5±0.3
Serum sodium, mEq/L	140±4	140±4
Urine sodium, mEq/d	204±108	304±117
Plasma noradrenaline, ng/mL	0.94±0.52	0.83±0.53
Plasma corticotropin, pg/mL*†	45±34	19±18
Plasma cortisol, μg/dL†	29±16	22±8
Plasma aldosteron, ng/mL	6.1±3.6	5.7±3.1
Plasma ANP, pg/mL	46±39	57±38
Plasma BNP, pg/mL†	65±44	168±261
CVP, cm	5.6±4.3	8.1±2.9

*Classification according to World Federation Neurological Society (WFNS) SAH grade.[12]

†Mann-Whitney *U* test.

There is a significant difference between 2 groups in WFNS grade ($P<0.05$), circulating blood volume (CBV) ($P<0.0001$), and corticotropin ($P<0.05$).

ANP indicates atrial natriuretic peptide; BNP, brain natriuretic peptide; CVP, central venous pressure.

Discussion

A significant decrease in CBV was noted in the postoperative stage in patients with SAH treated by early surgery. The mean value of CBV was 64 mL/kg on day 2 to 3, which gradually increased to 69 mL/kg on day 4 to 5 and 71 mL/kg on day 7 to 8 under the present management protocol. We previously reported a similar result after craniotomy.[11] CBV decreased postoperatively to approximately four fifths of its preoperative value and gradually increased and returned to its preoperative value on day 7. There were significant differences in the clinical grades and plasma corticotropin levels between patients with <60 and ≥60 mL/kg of CBV during the early postoperative stage (day 2 to 3).[11] There were no significant differences in other parameters between the 2 groups. These results suggested that the initial decrease in CBV might be related to the intensity of the initial stress in addition to the surgical stress. A reduction of CBV after stress is caused by the shift of fluid to the interstitial space. The initial decrease after SAH and early surgery is probably due to the same

TABLE 3. Laboratory and Physiological Changes When Circulating Blood Volume Was Decreased 10% More Than the Former Level in Subarachnoid Hemorrhage Patients

	Former	When Decreased	Mean Difference	*P* Value
CBV, mL/kg	71±17	57±15	14	$P<0.0001$
Hematocrit, %	37±5	35±5	2	$P<0.05$
Serum sodium, mEq/L	140±5	138±5	2	$P<0.01$
Urine sodium, mEq/d	245±149	306±132	60	$P<0.05$
Serum albumin, g/dL	3.6±0.5	3.2±0.5	0.3	$P<0.05$
CVP, cm	6.0±5.2	4.7±4.2	1.3	NS

CBV indicates circulating blood volume; CVP, central venous pressure.

The comparison was made between day 2 to 3 and day 4 to 5 in 6 patients; between day 4 to 5 and day 7 to 8 in 7 patients; and between day 7 to 8 and day 14 in 4 patients.

mechanism.[2,3] No routine examinations were found for estimating the CBV during the postoperative stage (day 2 to 3), which varied between 32.1 and 97.3 mL/kg.

The initial reduction of the mean value of CBV returned to within the normal range (72 mL/kg) on day 7 to 8 under the present management protocol.[10] The return of the fluid from the interstitial space to the intravascular space several days after surgery is known as the stage of postoperative polyuria in general surgery. We may expect a similar phenomenon after SAH and early surgery because our management protocol of early parenteral nutritional support and prompt correction of hyponatremia and anemia may help this phenomenon.

When CBV was decreased >10% of the former level, there were decreases in hematocrit, serum albumin, and serum sodium and an increase in urinary sodium. These results suggested that depleted RBC, salt wasting, and hypoalbuminemia are related to the cause of a decrease in CBV after the initial reduction. Kudo et al[2] suggested that depleted RBC volume was more responsible for neurological deterioration than was lowered plasma volume. Not only blood loss at surgery but bedrest, supine position, negative nitrogen balance, decreased erythropoiesis, and iatrogenic blood loss may cause a decrease in RBC volume. The significant increase in CBV by the transfusion of RBC supported this hypothesis.[19] Natriuresis was reported to be one of the major factors of volume contraction several days after SAH.[5–8] We did not obtain any correlations between a decrease in CBV and hormonal changes such as mineralocorticoid deficiency or excessive release of ANP and/or BNP.[8,20] The reduction of CBV after SAH may not be a simple phenomenon that can be explained by a single hormonal change.

To achieve normovolemia during vasospasm in SAH patients, depleted RBC volume can be treated by RBC transfusion; hypoalbuminemia can be treated by addition of human albumin; and central salt wasting can be treated by replacement of sodium hydrochloride with fluid or administration of fludrocortisone.[21] Presently, CBV is measured at the postoperative stage by ICG pulse spectrophotometry and is measured repeatedly when the initial CBV is <60 mL/kg. CBV is also measured when a transfusion of RBC and albumin is considered, when hyponatremia shows progression, and when symptoms of vasospasm are encountered. Thus, useless transfusion of RBC and albumin could be avoided for increasing CBV.

ICG, a tricarbocyanine dye, has been used in the clinical diagnosis of hepatic function and in the indicator dilution technique for estimation of cardiac output. It is firmly bound to plasma proteins and is believed to be confined to the plasma and not subjected to extravascular distribution. The measurement of blood ICG concentrations by pulse spectrophotometry operates on the same principle as the monitoring of SpO_2 by currently available pulse oximeters. The CBV can be computed as follows: $CBV=I_0$ (ICG dose administered)/ Cd_0 (initial blood ICG concentration). Arterial ICG concen-

TABLE 4. Laboratory and Physiological Changes When Circulating Blood Volume Was Increased 10% More Than the Former Level in Subarachnoid Hemorrhage Patients

	Former	When Increased	Mean Difference	*P* Value
CBV, mL/kg	55±13	72±13	17	$P<0.0001$
Hematocrit, %	35±4	36±4	1	NS
Serum sodium, mEq/L	140±4	138±5	1	NS
Urine sodium, mEq/d	261±102	273±119	12	NS
Serum albumin, g/dL	3.3±0.5	3.2±0.5	0.1	NS
CVP, cm	6.2±3.5	7.0±4.7	0.7	NS

CBV indicates circulating blood volume; CVP, central venous pressure.

The comparison was made between day 2 to 3 and day 4 to 5 in 17 patients; between day 4 to 5 and day 7 to 8 in 12 patients, and between day 7 to 8 and day 14 in 7 patients.

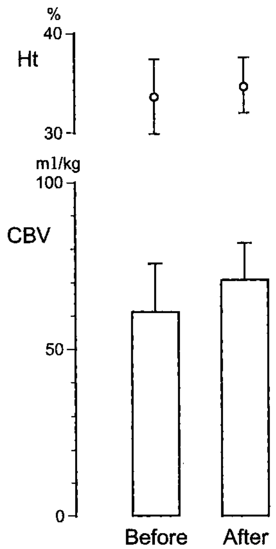

Figure 2. Effect of RBC transfusion on CBV and hematocrit (Ht). There was a significant difference before and after blood transfusion in CBV ($P<0.0001$) but not in hematocrit.

tration decreases exponentially. The regression line between 2.5 and 5.5 minutes after mean transit time was back-extrapolated to the time point of the mean transit time for the first circulation of ICG.[17,18]

Measurement of CBV is useful for monitoring fluid distribution. Radioactive isotopes and the Swan-Ganz catheter have been used to measure CBV, but these methods are not suitable for routine bedside use. The integrated pulse spectrophotometry monitoring system is less invasive and can be applied repeatedly within a short period without blood sampling. This methodology is useful for estimating CBV of neurosurgical patients with a single bolus injection of ICG. A CVP value is generally used as a guide for fluid balance. Although CVP showed some correlation to CBV in the present study, it did not reach statistical significance. A recent randomized controlled trial of CVP-guided volume expansion failed to demonstrate an increase in CBV.[22] By serially measuring CBV with ICG pulse spectrophotometry, effective hypervolemic therapy can be achieved without any complications and without using an invasive Swan-Ganz catheter.[23] ICG pulse spectrophotometry may be a powerful tool for the management of patients with SAH.

In conclusion, a significant reduction in CBV, especially in patients with poor clinical grade, was noted after SAH and early surgery, which could not be detected by routine examinations. Depleted RBC, salt wasting, and hypoalbuminemia may be related to the cause of the decrease in CBV from the former level. RBC transfusion significantly increased the level of CBV. ICG pulse spectrophotometry may be a powerful tool for the management of patients with SAH.

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