Goal-Directed Fluid Management by Bedside Transpulmonary Hemodynamic Monitoring After Subarachnoid Hemorrhage

Tatsushi Mutoh, MD, DVM, PhD; Ken Kazumata, MD; Minoru Ajiki, MD; Satoshi Ushikoshi, MD; Shunsuke Terasaka, MD

Background and Purpose—Optimal monitoring of cardiac output and intravascular volume is of paramount importance for good fluid management of patients with subarachnoid hemorrhage (SAH). The aim of this study was to demonstrate the feasibility of advanced hemodynamic monitoring with transpulmonary thermodilution and to provide descriptive data early after SAH.

Methods—Forty-six patients with SAH treated within 24 hours of the ictus were investigated. Specific targets for cardiac index (≥3.0 L·min⁻¹·m⁻²), global end-diastolic volume index (700 to 900 mL/m²), and extravascular lung water index (≥14 mL/kg) were established by the single-indicator transpulmonary thermodilution technique, and a fluid management protocol emphasizing supplemental colloid administration was used to attain these targets. Plasma hormones related to stress and fluid regulation were also measured.

Results—A higher cardiac index (mean value of 5.3 L·min⁻¹·m⁻²) and a lower global end-diastolic volume index (555 mL/m²) were observed on initial measurement, for which elevations of plasma adrenaline, noradrenaline, and cortisol were also detected. Cardiac index was progressively decreased (3.5 L·min⁻¹·m⁻²) and global end-diastolic volume index was normalized by fluid administration aimed at normovolemia. The extent of the initial hemodynamic and hormonal profile was greater in patients with a poor clinical status (P<0.05). The extravascular lung water index was mildly elevated but within the target range throughout the study period. No patients developed pulmonary edema or congestive heart failure.

Conclusions—The impact of sympathetic hyperactivity after SAH predisposes patients to a hyperdynamic and hypovolemic state, especially in those whose clinical status is poor. Bedside monitoring with the transpulmonary thermodilution system may be a powerful tool for the systemic management of such patients. (Stroke. 2007;38:3218-3224.)

Key Words: hemodynamic monitoring ■ stress ■ subarachnoid hemorrhage ■ transpulmonary thermodilution

Aneurysmal subarachnoid hemorrhage (SAH) is one of the most striking and devastating neurologic disorders. SAH affects most central nervous system functions, leading to deleterious systemic consequences. It is recognized that SAH is associated with a “catecholamine surge” due to hypothalamic and brainstem activation.1 Neurocardiogenic injuries, such as neurogenic stunned myocardium and neurogenic pulmonary edema, are thought to result from exaggerated sympathetic tone and elevated circulating levels of catecholamines at the site of aneurysm rupture.2,3 Hypovolemia accompanied by cerebral salt wasting and a progressive reduction of extracellular fluid volume also occurs frequently within a few days after symptom onset, possibly through central nervous system–mediated mechanisms.4,5

Brain regions with marginal perfusion and loss of autoregulation, hypotension, and hypovolemia, all of which can exacerbate decreases or reductions in cerebral blood flow, have been shown to increase the incidence of cerebral vasospasm and the related delayed ischemic neurologic deficits resulting from the earlier detrimental effects on blood pressure and cardiac output (CO). In this context, hypervolemia, hypertension, and hemodilution therapy is believed to prevent ischemic events and improve outcome and is central to the medical management of symptomatic vasospasm.6

To ensure appropriate intravascular volume and CO for good fluid management in patients after SAH, an optimal hemodynamic monitoring technique is of paramount importance. However, no bedside monitoring system has been practicable for estimating both CO and volume status simultaneously, other than the pulmonary artery catheter in the intensive care unit setting. Moreover, few studies concerning the serial changes in cardiac performance and circulating

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blood volume early after the insult of SAH, which may predispose these patients to volumetric and hemodynamic impairments, have been documented.

The single thermal indicator transpulmonary dilution system is a device for continuous CO measurement combined with cardiac preload volume and extravascular lung water (EVLW) monitoring. It computes CO with an arterial pulse-contour analysis algorithm after calibration by means of the transpulmonary thermodilution method to measure the volumetric preload parameter that includes the total volumes of the cardiac atria and ventricles, as well as part of the systemic vascular blood volume. Bedside monitoring with this system offers the facility to measure CO with only central venous and arterial catheters; it has numerous clinical advantages in patients with hemodynamic instability or critical illness in whom continuous measurement of CO is required, and it avoids the well-documented risks associated with the use of the pulmonary artery catheter. 7

We hypothesized that compared with conventional pressure-derived preload assessment, volumetric preload determination by the transpulmonary thermodilution system would better reflect left ventricular filling, thereby giving a better estimate of the safety and efficacy of volume therapy to minimize associated cardiopulmonary complications, such as pulmonary edema or congestive heart failure. Hence, the aim of this study was to investigate the serial changes in cardiac performance and volume status in patients with SAH treated postoperatively with normovolemia guided by the transpulmonary thermodilution technique. The mechanism of change in CO and circulating blood volume early after SAH was examined by measuring hormones related to stress and fluid regulation.

Methods

Patients

Forty-six consecutive patients (14 men and 32 women; mean ± SD age, 65 ± 11 years) with aneurysmal SAH were investigated in the Department of Neurosurgery, Teine Keijinkai Medical Center. The study protocol was approved by the institutional ethics committee, and informed consent was obtained from each patient or appropriate designee. Patients entered the study on the day after surgical clipping or intravascular coiling for aneurysm within 24 hours of the onset of symptoms (designated study day 0). Exclusion criteria included the following conditions: (1) both good clinical grade (World Federation of Neurological Surgery [WFNS] grade I) and modest bleeds (Fisher CT grade ≤ 2); (2) renal disease (creatinine level > 2.0 mg/dL); or (3) death within 7 days of bleeding. Fifty-six patients were screened, of whom 46 met the inclusion criteria and were enrolled between April 2005 and October 2006. Neurologic outcome was assessed by the modified Rankin scale score for all patients after 1 month. A summary of clinical data is given in the Table.

Experimental Procedures

General Management

All patients had a 7F central venous catheter inserted into the femoral vein postoperatively and received a baseline infusion of crystalloid (1500 to 3000 mL/d) for up to 14 days after onset of SAH. The patients were maintained on bed rest with intravenous fluids and oral food intake if possible. Intracranial hypertension was treated with glycerol and/or cerebrospinal drainage. Hyponatremia (defined as a serum sodium level of < 135 mEq/L for at least 2 consecutive days) was corrected by adding an ampule(s) of 10% NaCl (20 mL) to the main fluid bag. If hyponatremia persisted, fludrocortisone (0.3 mg/d) or hydrocortisone (1200 mg/d) was given as necessary. 8 Blood transfusion was performed only when the hematocrit level was < 30%. If the maximal systolic blood pressure was > 200 mm Hg, a calcium antagonist was administered. Nimodipine was not used (this drug is unavailable in Japan).

Patients were followed up by transcranial Doppler sonography daily or every other day according to the standard criteria for angiographic vasospasm. 9 Delayed ischemic neurologic deficit was defined as a worsening of the neurologic condition that could not be attributed to rebleeding or systemic or postoperative complications. Surveillance angiography was performed when patients did not respond to medical treatment. When the caliber of any artery was < 50% of that observed on the admission angiogram, ischemia due to cerebral vasospasm was diagnosed. 10

<table>
<thead>
<tr>
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<td>Sex, M/F</td>
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<tr>
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<tr>
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<td>II</td>
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<td>Other</td>
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</tr>
<tr>
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mRS indicates modified Rankin Scale; ACA, anterior cerebral artery; AcoA, anterior communicating artery; MCA, middle cerebral artery; and ICA, internal carotid artery.

Single-Indicator Transpulmonary Thermodilution

A 4F thermistor-tipped arterial catheter (PV2014L16, Pulson Medical Systems, Munich, Germany) was inserted into the brachial artery. The arterial catheter and a central venous catheter were connected to pressure transducers and to the single thermal indicator dilution system (PiCCO, Pulson Medical Systems) for monitoring. Continuous CO calibration, global end-diastolic volume (GEDV),...
and EVLW were determined by triplicate central venous injections of 15 mL of ice-cold saline (<8°C). CO was calculated by analysis of the thermodilution curve followed by pulse-contour analysis for continuous monitoring. GEDV was calculated from the difference of mean indicator transit time and exponential indicator downslope time and from CO. EVLW was calculated from GEDV based on a fixed algorithm established from data obtained from earlier double-indicator transpulmonary thermodilution. The basis of this method can be accessed in the supplemental material, available online at http://stroke.ahajournals.org.

Hemodynamic values were indexed to body surface area by means of the DuBois formula: body weight (in kilograms) × body length (in centimeters)²/₃ × 1.84. In this study, cardiac index (CI), GEDV index (GEDVI), and EVLW index (EVLWI) were calculated.

**Measurements**

The following measurements were performed during the postoperative period (days 1 to 14) after the onset of SAH: CI (normal values, 3.0 to 5.0 L·min⁻¹·m⁻²), GEDVI (680 to 800 mL/m²), EVLWI (3 to 7 mL/kg), central venous pressure (CVP), plasma adrenaline, noradrenaline, cortisol, aldosterone, antidiuretic hormone (ADH), and brain natriuretic peptide (BNP). Water balance was calculated from the difference between the total amount of water intake (sum of transvenously infused water and orally ingested water) and water losses (sum of urine, transpired water, and various drainage fluids). Metabolized water and water included in the stool were not included in this study.

The CI, GEDVI, EVLWI, and CVP were measured at least twice daily until day 14; plasma adrenaline, noradrenaline, cortisol, ADH, and BNP were measured initially on admission, at 12 hours, and daily until day 3 by commercially available radioimmunoassay; and other blood and chemical parameters were measured daily or every other day until day 14 by automatic analyzer. Water balance was measured every 8 hours until day 14.

**PicCO-Guided Fluid Management**

Basic fluid management was aimed at the maintenance of CO, which was our primary method to increase cerebral blood flow medically, and prevention of hypovolemia and cardiopulmonary complications.6-11 Hemodynamic stability was defined as a CI ≥3.0 L·min⁻¹·m⁻², a GEDVI ≥700 mL/m² (lower limits were defined at lower values as the normal 680 to 800 mL/m²), and EVLWI ≤14 mL/kg (upper limits were defined at a higher risk of mortality with pulmonary edema when EVLWI >14 mL/kg).12 Patients were assigned to receive intravascular volume expansion with 6% hydroxyethylstarch (500 to 1500 mL) if CI fell below the target level (<3.0 L·min⁻¹·m⁻²) due to hypovolemia (GEDVI <700 mL/m²). When hydroxyethylstarch was ineffective in raising GEDVI above the lower target values and CI was persistently lower than 24 hours, additional 25% albumin solution (50 to 100 mL) was administered. If the low CI persisted even under hypervolemia (GEDVI ≥900 mL/m², EVLWI ≤14 mL/kg) along with fluid therapy for at least 24 hours, inotropic support with dobutamine (3 to 15 μg·kg⁻¹·min⁻¹) or milrinone (0.25 to 0.75 μg·kg⁻¹·min⁻¹) was started to maintain the CI above target levels. When the patient had an elevated EVLWI (≥15 mL/kg) and any sign of congestive heart failure or pulmonary edema (eg, bilateral pulmonary infiltrates and/or cardiomegaly observed with a cardiothoracic ratio >50% on chest radiography), furosemide (5 to 20 mg) was administered until EVLWI was reduced to <14 mL/kg. This fluid management protocol was strictly adhered to throughout the entire study period unless symptomatic vasospasm was diagnosed.

Patients who become symptomatic with delayed ischemic neurologic defect due to vasospasm were managed by enhancement of their cardiac contractility (“hyperdynamic therapy”) by the use of inotropes to titrate the CI above normal limits (>5.0 L·min⁻¹·m⁻²) to the level at which the deficit resolved or until a maximal systolic blood pressure of 200 mm Hg was achieved.

**Statistical Analysis**

All data were stored on a personal computer and analyzed by commercially available software (SPSS version 15.0, SPSS Inc, Chicago, Ill). Data that were collected sequentially with a data acquisition system (PicCO-VoLEF-Win version 6.0, Pulmus Medical Systems) were examined by repeated-measures ANOVA with a post hoc Bonferroni-Dunn correction or paired t test. Categorical data were analyzed by χ² test. Comparisons between groups were examined with an unpaired t test when the dispersions of the 2 groups were equal or by the Mann-Whitney U test. The Pearson correlation was established for absolute values between stress hormones and CI. Statistical significance was claimed when the probability of a type I error was <.05. All values were expressed as mean ± SD.

**Results**

**Stress Response and Hemodynamic Outcomes After SAH**

The time-course changes of CI, GEDVI, and EVLWI obtained from the single-indicator transpulmonary thermodilution technique in 46 patients after SAH and early surgery are shown in Figure 1. High CI values (5.3 ± 0.4 L·min⁻¹·m⁻²) were detected on day 1, which progressively fell to a minimum of 3.5 ± 0.2 L·min⁻¹·m⁻² on day 5 (P < .05). Conversely, the GEDVI was low (555 ± 27 mL/m²) on day 1 but normalized to 740 ± 47 mL/m² (P < .05) by day 3 by titrating fluid administration aimed at normovolemia. Throughout the study period, EVLWI remained slightly raised (10.4 ± 2.3 mL/kg).

Initial plasma noradrenaline concentrations sampled on admission were 1.6 times higher (0.79 ± 0.15 ng/mL) than the reference level but then returned to close to the upper normal limits (0.39 ± 0.05 ng/mL) after 24 hours. Plasma adrenaline concentrations were mildly elevated (0.11 ± 0.02 ng/mL) on admission but also returned to within the normal range after 24 hours. Plasma cortisol was also high (24.8 ± 2.0 μg/dL) on admission but gradually returned to the normal range within 48 hours. There were no statistically significant time-course changes in plasma aldosterone, ADH, and BNP throughout the study period (P > .05). Linear-regression analysis between the stress hormones and CI showed significant correlations for adrenaline (r² = 0.390, P < .0001), noradrenaline (r² = 0.453, P < .0001), and cortisol (r² = 0.388, P = .0001; Figure 2).

**Comparison of Hemodynamic Outcomes Between Good and Poor Grades**

Initial measurements after SAH without the effect of fluid regulation were compared between clinical grades (WFNS grades I–III versus grades IV and V; Figure 3). Higher CI and plasma concentrations of adrenaline (0.06 ± 0.02 versus 0.13 ± 0.02 ng/mL), noradrenaline (0.46 ± 0.10 versus 1.06 ± 0.25 ng/mL), and cortisol (21.8 ± 2.4 versus 29.1 ± 3.0 μg/dL) and lower GEDVI values were present in SAH patients with a poor clinical grade (P < .05 for each). Although in patients with a poor clinical grade the EVLWI values tended to be higher, the difference was not statistically significant (P = .07). There were no statistically significant differences in plasma aldosterone, ADH, and BNP between good and poor grades (P > .05).
Overall Performance of Transpulmonary Thermodilution System

In 43 patients (93%), the hemodynamic targets (CI ≥ 3.0 L min⁻¹ m⁻², GEDVI ≥ 700 mL/m², and EVLWI ≤ 14 mL/kg) were obtained by using the fluid management protocol until day 3 ± 2 (mean ± SD; range, 1 to 8) after SAH onset. However, 3 patients failed to reach the established target values. In all cases CI and EVLWI were within the target range, but a low GEDVI (< 700 mL/m²) persisted throughout the study period.

For fluid management, 4 patients (9%) were given maintenance fluid infusion only and oral intake; 38 patients (83%) were administered supplemental hydroxyethylstarch for volume expansion; and 26 patients (57%) were given 25%

Figure 1. Time-course changes of CI, GEDVI, and EVLWI for 14 days in 46 patients after SAH. Lower hatched box on the vertical axis indicates the normal range of each variable.

Figure 2. Correlation coefficients between stress hormones and CI in 46 patients after SAH. Hormonal data were sampled on admission, and CI was obtained immediately after application of the transpulmonary thermodilution system.
This study provides new evidence for ongoing hemodynamic albumin. To obtain a CI ≥ 3.0 L · min⁻¹ · m⁻², 3 patients (7%) required inotropic support with dobutamine (4%, n = 2) or milrinone (2%, n = 1). Hyponatremia developed in 24 patients (52%; n = 10 in grades I–III and n = 14 in grades IV and V); in most, their condition was corrected by adding 10% NaCl to the main fluid, 2 patients needed oral fludrocortisone, and 3 required intravenous hydrocortisone. For the correction of anemia (hematocrit < 30%), 11 patients (24%) required blood transfusion. Furosemide (10 mg/d for 3 days) was used in 1 patient to treat an elevated EVLWI due to neurogenic pulmonary edema.

The daily fluid intake of all patients was gradually increased (eg, 2661 ± 547 mL/day; range, 1516 to 3807 mL/day until day 3 and 4336 ± 907 mL/day; range, 2324 to 5959 mL/day after day 4; n = 46), and patients with a poor clinical grade required significantly greater fluid administration on day 5 (P < 0.05; Figure 4). Net daily fluid balance and CVP were not significantly different throughout the study period nor between clinical grades (P > 0.05).

Of the 17 patients (37%) who met the standard criteria for angiographic vasospasm, 9 (20%) remained asymptomatic, whereas 8 (17%) showed a delayed ischemic neurologic defect and had direct angiographic verification of vasospasm. Patients who experienced symptomatic neurologic deterioration due to vasospasm were switched to hyperdynamic therapy with dobutamine during the study. At the onset of symptomatic vasospasm (9 ± 2 days; range, 6 to 12 days after SAH onset), the CI was 3.3 ± 0.3 L · min⁻¹ · m⁻² (within the lower limit of normal), and all of them were started on dobutamine at a maximum mean ± SD dose of 6.7 ± 2.4 µg · kg⁻¹ · min⁻¹ (range, 3 to 12 µg · kg⁻¹ · min⁻¹) to attain a CI of 5.1 ± 0.6 L · min⁻¹ · m⁻² until 10 ± 2 days (range, 7 to 13 days) after SAH onset. Cerebral infarctions secondary to a vasospasm-induced delayed ischemic neurologic defect occurred in 4 patients (9%).

In this study, no patient developed pulmonary edema or congestive heart failure during fluid therapy, except for the presence of neurogenic pulmonary edema in 4 patients (9%) and neurogenic stunned myocardium (“Tako-tsubo” cardiomyopathy) in 1 patient (2%) diagnosed on admission.

**Discussion**

This study provides new evidence for ongoing hemodynamic monitoring of cardiac performance and volume status with the single-indicator transpulmonary thermodilution technique. We have demonstrated that CI increases progressively and significantly early after SAH, which was strongly associated with stress-induced sympathetic hyperactivity at the site of aneurysmal rupture, especially in patients with a poor clinical grade. Bedside monitoring with the single-indicator transpulmonary thermodilution system may be a powerful tool for systemic management.

An incremental relation was observed between the WFNS grade, which is widely used in assessing the severity of neurologic injury after SAH, and the hyperdynamic/hypovolemic state early after SAH. It is known that the release of norepinephrine induces peripheral and splanchnic vasoconstriction, a major contributor to the maintenance of central organ perfusion, whereas reduced vagal activity increases heart rate and CI. Our finding is consistent with previous studies that showed that patients with more severe grades of SAH had a higher incidence of catecholamine release, as estimated by an increased stress index obtained from a combination of blood sugar level/serum potassium concentration or by direct measurement of plasma catecholamine levels. Those investigations demonstrated that the plasma catecholamine level was extremely high in the supracranial stage (within an hour of bleeding) but decreased fairly quickly at 24 hours to the normal range. Given the higher correlation and similar time-course changes observed between the stress hormones (epinephrine, norepinephrine, and cortisol) and CI, acute stress caused by SAH can contribute to the sympathetic hyperactivation that induces a hyperdynamic state at an early stage, depending largely on the intensity of the initial stress. Adverse physical (eg, burn trauma, infection, or sepsis) and psychological conditions can activate the sympathetic nervous system, the hypothalamic-pituitary-adrenal axis, immune cells, and cytokines, which can initiate the stress-response cascade that regulates myocardial and adrenal transcription/translation genes and culminates in cardiac contraction/relaxation defects or an impairment of fluid/sodium retention. Such actions and others may negatively affect the heart in several ways.

Natriuresis was reported to be 1 of the major factors of volume contraction several days after SAH. Most patients who demonstrated hyponatremia showed a decrease in plasma volume by > 10% or failed to maintain CVP within...
the target range (8 to 12 cm H2O), suggesting the presence of hypovolemia, consistent with the cerebral salt-wasting syndrome. However, we did not find any correlations between a decrease in GEDVI and hormonal changes, such as mineralocorticoid deficiency or an excessive release of BNP. The reduction in GEDVI after SAH may not be a simple phenomenon that can be explained by a single hormonal change. A reduction in circulating blood volume after stress is caused by the shift of fluid to the interstitial spaces, which may in part explain the initial depression of GEDVI and the need for optimizing hemodynamics early after SAH.

Patients with pulmonary complications after SAH are more prone to vasospasm and are at higher risk for mortality and neurogenic morbidity. Furthermore, patients who are subjected to administration of large amounts of fluid as well as vasopressors to maintain a higher CO associated with hypertension, and hemodilution therapy may have a higher risk of pulmonary edema. The transpulmonary thermodilution system has been shown to quantify EVLW with a very high correlation to conventional gravimetric measurement of EVLW, which allowed us to minimize pulmonary edema and/or congestive heart failure by directing adequate volume status after SAH. In the present study, the decreased intravascular volume that developed at an early stage, largely in patients with a poor clinical grade, was able to establish hemodynamic stability safely and appropriately before the onset of cerebral vasospasm without overloading, according to the protocol for this method.

Good fluid management of cerebral vasospasm involves knowing how much hydration patients will tolerate before developing complications such as pulmonary edema and congestive heart failure due to fluid overloading. Serial volume measurements as estimated by GEDVI and EVLWI with the transpulmonary thermodilution method proved to be sufficiently practical and accurate and would be useful for monitoring patients after SAH. The system also allows continuous assessment of CO, which gave us the opportunity to respond rapidly to hemodynamic changes. So far, this system has not been validated in patients with SAH except for a case report, wherein the catheters was placed a few days after the occurrence of vasospasm. Although this study included a small number of patients, we believe that our initial experience with SAH patients indicates a potentially effective monitoring technique for the management of cerebral vasospasm. Future studies should continue to focus on safety and better definition of the optimal protocol and overall efficacy of this therapy.

Our study has shown that this methodology is useful and can be applied repeatedly for estimating cardiac performance and the volume status of neurosurgical patients with a bolus injection of cold saline solution. A recent randomized, controlled trial of CVP–guided volume expansion also failed to demonstrate an increase in circulating blood volume in patients after SAH or sepsis. Cardiac filling pressures also afforded a poor prediction of fluid responsiveness after the early phase of sepsis. Although a comparison between the transpulmonary thermodilution technique and conventional monitoring to guide fluid therapy in SAH patients has not been performed and should be confirmed in the future, the finding that neither net fluid balance nor CVP changed significantly throughout the study period (Figure 4) may support the concept that these parameters are poor predictors of volume expansion. In fact, several studies have demonstrated that volumetric cardiac preload measurements assessed by transpulmonary thermodilution better reflect left ventricular filling than pulmonary artery catheter–derived pulmonary artery occlusion pressure to estimate left ventricular end-diastolic volume, indicating the superiority of

![Figure 4](http://stroke.ahajournals.org/)

**Figure 4.** Daily fluid intake/output, fluid balance, and CVP changes for 14 days in SAH patients with a good (WFNS grades I–III, n=23; shown in white bars/circles) and a poor (grades IV and V, n=23; shown in black bars/circles) clinical grade. *P<0.05 vs grades I–III.
volumetric monitoring of cardiovascular volume status over conventional pressure monitoring.

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Disclosures

None.

References

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