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INTRACRANIAL AND BLOOD PRESSURE VARIABILITY AND LONG-TERM OUTCOME AFTER ANEURYSMAL SUBARACHNOID HEMORRHAGE

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Abstract

Background—Care of individuals in the intensive care unit (ICU) with brain injury traditionally focuses on maintaining ABP and ICP within prescribed ranges. However research suggests that the dynamic variability of these pressure signals provides additional information about physiologic functioning and may reflect adaptive capacity.

Objectives—The purpose of this study was to examine the ability to predict long-term outcome from arterial blood pressure (ABP) and intracranial pressure (ICP) variability in patients with aneurysmal subarachnoid hemorrhage (SAH).

Methods—ABP and ICP were monitored continuously for four days in 90 patients (74% female; mean age 53 years) in an ICU following SAH. Variability of ABP and ICP signals was calculated at four time scales (24-hour, hourly, 5-minute, and difference of sequential 5second averages). Long-term functional outcome was assessed 6 months post-SAH using the Extended Glasgow Outcome Scale.

Results—Pressure (ABP, ICP) variability indices were better predictors of 6-month functional outcome than mean pressure levels. Indices reflecting faster variability (particularly 5-second) were positively associated with better long-term outcome (typical $p < 0.001$), while greater 24-hour variability was related to poorer outcomes (typical $p < 0.001$), controlling for initial neurologic condition.

Conclusions—Beyond the measurement of ABP and ICP levels in acutely ill patients with SAH, simple measures of variability of these signals provide prognostic information regarding long-term functional outcome. The relationship between outcome and ICP and ABP variability in SAH 2 variability was dependent on the time scale at which the variability was measured. Given its positive association with better outcome, greater faster variability may reflect better physiologic adaptive capacity.

Keywords

adaptation; physiologic; subarachnoid hemorrhage

Monitoring and management of arterial blood pressure (ABP) and intracranial pressure (ICP) are key components of critical care nursing of patients with subarachnoid hemorrhage (SAH) due to a ruptured cerebral aneurysm. Both medical and nursing clinical interventions are directed at maintaining ABP and ICP within set ranges. In keeping with this focus, research examining the association between physiologic variables and outcome has traditionally focused on determining the optimal level or range of values for these variables. However, there is growing interest in examining the dynamics of physiologic variables, such as trends and variability over time. Research has begun to show a relationship between greater physiologic regularity and decreased physiologic complexity, and disease, aging and mortality [1–9]. Decreased physiologic variability is thought to reflect uncoupling of system components normally involved in regulatory processes [10]. The diminished communication between system components results in a decreased ability to respond appropriately to internal or external challenges to the system. In addition to the systemic integration of physiologic system components involved in ABP and ICP regulation, cerebrovascular regulatory mechanisms normally respond to local pressure and metabolic changes in an attempt to ensure adequate cerebral perfusion. ICP variability may also reflect the specific responsiveness of these mechanisms.

Thus consideration of the dynamic variability of ABP and ICP may provide additional information about physiologic functioning, potentially reflecting dimensions of adaptive capacity. Such information could be clinically useful in identifying patients who have decreased capacity to respond to physiologic perturbations, such as a drop in ABP or oxygenation, and are also at greater risk for adverse response to nursing care, such as sustained increased ICP in response to positioning, that may contribute to secondary brain injury and poorer outcome. The purpose of this study is to examine the association between measures of ABP and ICP variability, and outcome in patients admitted to the intensive care unit (ICU) for the management of SAH.

METHODS

Design

This study is a descriptive correlational analysis of physiologic data gathered as part of a randomized clinical trial examining the impact of a highly visible display of cerebral perfusion pressure on cerebral perfusion pressure management and outcome. The methods and results of the parent study have been reported elsewhere[11,12].

Sample and Setting

The study was carried out at an academic medical center. The parent study included patients aged 16 years or older with traumatic brain injury and cerebral aneurysms or arteriovenous malformations who were admitted to an ICU and underwent invasive ABP and ICP monitoring as part of their standard care. Of the 260 patients enrolled in the parent study, 90 had a diagnosis of aneurysmal SAH and were included in this analysis. Patients were enrolled over a 2-year period.

Measures

Demographic data and information regarding initial neurologic condition (Glasgow Coma Scale [GCS]), SAH severity (Hunt and Hess score), diagnosis, management, and hospital course were recorded. The GCS is a widely used scale that allows for quantification of level of consciousness[13]. Potential scores range from 3 (unresponsive) to 15 (alert and oriented). The Hunt and Hess grade is determined based on clinical neurologic condition and reflects SAH severity[14]. Grades range from 1 (no symptoms or mild headache) to 5 (deep coma,

moribund). Information regarding administration of major classes of medications with the potential to affect ICP or ABP was recorded.

ABP measurements were obtained via intra-arterial catheters connected to fluid-filled pressure transducers (Abbott Laboratories, Abbott Park, IL). ICP measurements were obtained via intraparenchymal Camino transducer-tipped catheters (Integra LifeSciences, Plainsboro, NJ). The ABP and ICP devices were connected to the bedside computer monitoring system (Spacelabs Medical, Redmond, WA) and the signals input from there to the study computer system. ABP and ICP data were saved as five-second averages.

Outcome in the parent study was assessed at six months post-SAH by trained interviewers using the Extended Glasgow Outcome Scale (GOSE) (1 = dead, 8 = upper good recovery) [15,16]. The GOSE was chosen in the parent study as a relevant measure of global functional outcome that could be used consistently across the subgroups of the study, thus a stroke specific outcome measure was not used. Outcome was dichotomized to: 1) survival versus non-survival, and; 2) unfavorable (GOSE 1–4) versus favorable (GOSE 5 to 8).

ABP and ICP variability were calculated at four time scales, including five seconds, 5 minutes, 60 minutes, and 24 hours. At the 5-second time scale variability was calculated as the root mean successive square difference (RMSSD) of adjacent 5-second segments, reflecting the change from one 5-second average to the next. Variability at the other time scales was calculated as the standard deviation (SD). The hourly SD includes variability longer than 5 minutes up to one hour. The 24-hour SD reflects variability longer than 60 minutes up to 24 hours.

Analysis

Correlations between physiologic variables and initial condition and medication use were examined using Spearman correlation coefficients. The relationship between variability measures and outcome was assessed using binary logistic regression, controlling for initial neurologic condition (GCS).

Results

In keeping with the demographics of SAH the sample was predominantly female, with an average age of 53 yrs (Table 1). SAH severity ranged from mild to severe, with the greatest number in the mild to moderate SAH categories (Figure 1). The mean (SD) number of days from aneurysm rupture to initiation of study monitoring was 3.5 (3.3). Almost one-half (49%) had a ventriculostomy inserted for drainage of cerebrospinal fluid. The majority of patients underwent surgical clipping of their aneurysms (Table 1). Nineteen percent underwent a craniectomy. Known pre-existing conditions that could impact ABP included hypertension (54%), diabetes (5%), coronary artery disease (6%), and arrhythmias (6%). Survival at hospital discharge was 80% and at six months was 73%. Six-month outcome by GOSE category is presented in Figure 2. The median GOSE of 5 reflects ability to function independently in the home but inability to return to previous work and considerable restrictions in social and leisure activities.

The values of the physiologic variables averaged over four days of monitoring are presented in Table 2. Both ICP and ABP variability increased as the time scale increased. None of the variability measures differed significantly based on whether the aneurysm was located on the anterior or posterior circulation. To assess if variability differed based on age, the correlation between age and variability was examined. While age was not significantly correlated with any of the ICP variables, it was inversely correlated with mean ABP ($r = -.22$, $p = .035$), ABP 5-second RMSSD ($r = -.22$, $p = .037$), and 24-hour ABP SD ($r = .40$, $p = .000$). As with age,

gender was not significantly associated with any of the ICP variables. However, females had greater 24-hour ABP variability than males (7.63 mm Hg versus 6.34 mm Hg, $p = .038$).

Figure 3–Figure 4 present time series plots of ICP and ABP over 24 hours demonstrating variability at the different time scales. The x-axis represents time in hours and the y-axis represents mm Hg. The top frame is the original time series of 5-second averaged data points. The subsequent frames break the variability into the different time components. The second frame shows slow variability that occurs over a period longer than one hour. The third frame shows variability that occurs over longer than 5 minutes but less than one hour. The bottom frame shows faster variability that occurs over longer than 5 seconds up to 5 minutes. Figure 3A shows high fast (5-second, 5-minute) ICP variability in an individual who had a good six-month outcome. Figure 3B shows low fast ICP variability in an individual who had an unfavorable outcome. Figure 4 illustrates high ABP fast variability while Figure 4B reflects low ABP fast variability. These patterns were associated with favorable and unfavorable outcome, respectively.

The association between variability measures and SAH severity, as reflected by initial GCS score and Hunt and Hess score, is presented in Table 3. Greater faster variability (5-second RMSSD and 5-minute SD) was significantly associated with better initial clinical condition (higher GCS score and lower Hunt and Hess score). This association was strongest in relation to ABP variability. The differences in variabilities are highlighted when groups with more severe SAH (Hunt and Hess score of 4 or 5) and less severe SAH (Hunt and Hess of 1 to 3) are compared (Table 3).

There was a significant positive association between ICP median level and all ICP variability indices except 24-hour variability ($r = .243$ to $.288$, $p = .021$ to $.006$). Thus, ICP level was included as a control variable in the regression models. Given that there was not a similar association between ABP level and ABP variability indices, ABP level was not included in the regression models.

The association between variability measures and cerebral vasospasm was examined. Vasospasm was categorized as present versus absent and as severe, defined by transcranial Doppler criteria, versus other. Although ICP level differed significantly between those with and without vasospasm (mean (SD) = 12.6 (7.4) versus 7.3 (4.3) respectively), after controlling for level there were no significant differences between the two groups on any of the ICP variability measures. When subgroups with severe vasospasm versus all others were considered there were no significant differences in relation to ICP level or ICP variability measures. ABP level and variability measures did not differ significantly between groups based on presence or absence of vasospasm or severe vasospasm versus others.

Figure 5 presents the odds ratios for 6-month survival by ICP level and variability indices, controlling for initial clinical condition (GCS score) and additionally for ICP level for the variability indices. While the association between ICP level and survival was not significant, ICP variability at the 5-second, 5-minute, and 24-hour time scales were all significant predictors of survival. Whereas greater faster variability (5-second, 5-minute) was associated with greater odds of being alive at six months, greater slower variability (24-hour) was associated with lower odds of six-month survival.

The pattern of greater faster (5-second, 5-minute) variability and better odds of survival was also present in relation to ABP (Figure 6). As with 24-hour ICP variability, greater 24-hour ABP variability was also associated with significantly lower odds of six-month survival.

To further examine whether the association between ABP and ICP variability and outcome predicts not only survival, but also functional ability at six months, the above analysis was

repeated dichotomizing six-month outcome to favorable (GOSE >4) versus unfavorable (GOSE ≤4). As with survival, median ICP level was not significantly associated with functional outcome (Figure 7). ICP variability at both the 5-second and 5-minute time scales was significantly associated with greater odds of favorable outcome at six months. While the relationship between 24-hour ICP variability and favorable outcome was in the same direction as that of 24-hour variability and survival, it showed only a strong trend and did not reach statistical significance.

ABP level was not a significant predictor of six-month survival, however higher ABP level over four days of monitoring was associated with significantly lower odds of favorable outcome at six months (Figure 8). Greater fast (5-sec RMSSD) ABP variability was associated with significantly greater odds of favorable outcome. Conversely, greater slow (24-hour) ABP variability was associated with significantly lower odds of favorable outcome.

The percentage of subjects receiving major classes of medication with potential effects on ABP or ICP is presented in Table 4. All subjects received calcium channel blockers as the standard of care for management of SAH. In addition, all subjects received analgesia during the period of monitoring and a large majority received anxiolytics/sedatives/hypnotics. Given the highly variable amounts of PRN medications administered to subjects, the median number of doses (based on standard average doses) received over the four days of monitoring was calculated for analgesics (11.0, range 1–76), anxiolytics/sedatives/hypnotics (1.8, range 0–466), and diuretics (.9, range 0–38). The high end of the range of anxiolytics/sedatives/hypnotics was related to high-dose propofol administration to a number of subjects.

In relation to ICP, median ICP was significantly correlated with the number of doses of analgesics ($r = .277$, $p = .008$) and diuretics ($r = .294$, $p = .005$). There were no significant correlations between ICP 5-second RMSSD and 5-minute SD and any of the major classes of medications. ICP hourly SD was significantly correlated with anxiolytic/sedative/hypnotic dose ($r = .311$, $p = .003$) and diuretic dose ($r = .212$, $p = .045$). Daily ICP SD was also significantly correlated with diuretic dose ($r = .278$, $p = .008$).

Median ABP was significantly correlated with anxiolytic/sedative/hypnotic dose ($r = .252$, $p = .016$) and corticosteroid dose ($r = .385$, $p = .000$). ABP 5-second RMSSD was significantly correlated with antiarrhythmic (yes/no) ($r = -.224$, $p = 0.034$), diuretic dose ($r = -.227$, $p = .031$), and vasodilator dose ($r = .245$, $p = .020$). Five-minute ABP SD was not significantly correlated with any of the major drugs classes. Hourly ABP SD was significantly correlated with anxiolytic/sedative/hypnotic dose ($r = .375$, $p = .000$), diuretic dose ($r = .221$, $p = .036$), and vasodilator dose ($r = .22$, $p = .036$). Twenty-four hour ABP variability was significantly correlated with adrenergic blockers administration ($r = .208$, $p = .049$), antiarrhythmic administration ($r = .226$, $p = .032$), and anxiolytic/sedative/hypnotic dose ($r = .220$, $p = .037$).

Discussion

While adaptive mechanisms normally act to maintain physiologic variables within ranges compatible with maintenance of system integrity and functioning, these measures are not static and variability is a feature of healthy systems. However variability outside the normal healthy range in either direction is likely to reflect ineffective adaptive mechanisms in the face of disease or injury. ABP and ICP variability following SAH may provide clinically important information regarding physiologic responsiveness of the cardiovascular and cerebrovascular systems that reflect impaired functioning. Individuals with impaired physiologic responsiveness, as reflected by abnormal variability, could be identified as being at high risk for further physiologic derangement.

In addition to intrinsic adaptive mechanism functioning, external factors contribute to physiologic variability, including environmental influences and medical and nursing interventions. However the degree to which these factors positively or negatively impact variability is not well documented. This study measures the overall variability of ICP and ABP related to all factors early following SAH. The independent contributions of disease severity and medication administration are examined.

ABP variability can be related to both physiologic and pathophysiologic factors and has been examined as an indicator of cardiovascular control and for its potential prognostic significance. Overall ABP variability results from a number of different factors acting at different time scales from seconds to days. This includes fluctuations related to intrinsic cardiovascular regulation, activity, and a 24-hour circadian variability [15]. In addition, disease and pharmacologic agents also influence ABP variability.

ABP variability has been examined in relation to hypertension and cardiovascular and cerebrovascular risk. Higher measures of ABP variability are associated with carotid artery damage [18,19], increased cardiovascular mortality [7], cerebral white matter lesions [20], cardiac structure damage [21,22], stroke [23,24] and myocardial infarction [25], particularly in hypertensive individuals. Findings of this study are in agreement with a general hypothesis of an association between greater ABP variability and more severe disease manifestation that is supported by previous research. However, these findings have not previously been documented specifically in a population of critically ill individuals with SAH.

There is also little research examining simple indices of ICP variability, such as SD, in acute SAH. In a study of a small group of patients ($n = 8$) with various cerebrovascular disorders, ICP SD was evaluated over a period of four minutes. ICP SD was elevated (6–10 mm Hg), particularly during periods of decompensation, except in the course of cerebral vasospasm, when it was low [24]. In the current study ICP variability is not significantly associated with cerebral vasospasm. While cerebral vasospasm decreases the amplitude of individual intracranial pressure waveforms, this effect may not be reflected in 5-second ICP SD averages.

The ABP is the primary input for ICP. There is a significant positive association in this study between ABP and ICP variability across-subjects at the 5-second ($r = .502$, $p = .000$), 5-minute ($r = .332$, $p = .001$), and 24-hour ($r = .318$, $p = .002$) time scales. However, this does not address the within-subject impact of ABP variability on ICP variability. ICP variability is affected by numerous factors other than ABP level, including the degree of transmission of ABP to ICP, venous drainage from the head, cerebrospinal fluid circulation, and cerebral autoregulation. Cerebral autoregulation is a mechanism whereby cerebral blood vessels constrict or dilate in responses to changes in ABP to ensure a consistent blood flow to the brain. Changes in cerebral blood vessel diameter can lead to changes in blood volume, resulting in changes in ICP. Cerebral autoregulation is normally very rapid, with compensatory changes beginning within seconds.

Variability of ICP and ABP early following SAH significantly predicts six-month outcome. However, the relationship between variability and outcome is dependent on the time scale over which variability is assessed. While greater fast variability is associated with increased odds of both survival and good outcome, conversely, greater slow variability is associated with decreased odds of survival and good outcome. Greater five-second variability of both ICP and ABP were the strongest predictors of survival and good outcome, thus this faster variability may reflect more effective functioning of adaptive mechanisms. Given its significant association with poorer outcome, greater slow variability (longer than an hour) may reflect decreased integrated ability of adaptive mechanisms to respond to physiologic alterations or challenges to the system. ABP and ICP variability in this study were measured relatively early

after SAH. It is not known if this variability would change as recovery occurs or in the long term.

The minimum resolution of data points in this analysis is 5 seconds so the function of mechanisms effecting very rapid changes in ICP and ABP is not reflected. This includes beat-by-beat fluctuations related to the cardiac cycle, most respiratory cycle fluctuations (for respiratory rates of 12 per minute or faster), and very rapid autonomic nervous system modulation of ABP. The impact of sympathetic nervous system activity and Mayer waves would be compatible with fluctuations represented in the ABP 5-minute SD [27]. Further study of the effect of circulating catecholamines on ABP and ICP variability following SAH would be of interest. Other contributors to both ABP and ICP variability over longer than 5 seconds include nursing care, environmental stimuli, and patient activity. Factors affecting longer-term ABP variability, e.g., hourly, include functioning of longer-term feedback mechanisms, such as the renin-angiotension system.

When the association between major medication group administration and ICP variability was examined, all significant correlations were in a positive direction. In relation to ABP, diuretic and antiarrhythmic medication administration were associated with less 5-second ABP variability. All other significant correlations between medications and ABP variability reflected greater variability with medication administration. While the administration of a number of medications could decrease ABP and ICP variability, such as sedation that decreases activity level, this was not the overall direction of the association seen. The medications may actually have little effect on variability, may increase variability, or the reason for which they are given may continue to have a greater impact on increasing variability than the medications' effect to decrease variability. This relationship is also apparent in relation to level, for example, higher doses of analgesics and diuretics, both given to manage increased ICP, are associated with higher median ICP.

The observation of trends over periods longer than that routinely displayed on bedside clinical monitors is informative in relation to physiologic state and prediction of outcome. While the calculation of the SD of physiologic measures such as ABP and ICP is simple and provides quantification of pressure variability, even visual records of trends over various periods of time provide meaningful qualitative information regarding faster and slower variability. The original ABP and ICP time series in the top frames of Figure 3 and Figure 4 that display trends over 24 hours clearly demonstrate differences in both faster and slower variability between these two individuals.

It is physiologically plausible that both faster variability and slower variability reflect physiologic adaptive capacity. Given the findings of this and other studies of a relationship between greater slow ABP variability over 24 hours and higher cerebrovascular and cardiovascular morbidity, greater long-term variability may reflect poorer functioning of adaptive mechanisms and decreased physiologic regulatory capacity. In addition, the association in this study between greater faster variability and better outcome may suggest that fast physiologic variability reflects a regulatory system capable of responding rapidly and more effectively to internal and external perturbations that occur in a critical care clinical situation. Thus, real-time visual assessment of pressure variability may be useful clinically to identify critically ill individuals who have decreased physiologic adaptive capacity and are therefore less likely to be able to compensate for both physiologic and pathophysiologic insults. Nursing care for identified high-risk individuals could then be specifically targeted to minimize such insults. The development and testing of such interventions requires further study.

In addition, further research is needed to determine the most appropriate and meaningful measures to assess dynamic variability of physiologic measures in the clinical setting. Further

study is also needed to better understand the relationship between physiological variability, adaptive capacity, and outcome. Whether nursing or medical intervention directed specifically at changing ABP or ICP variability can be effective, and whether this alteration is ultimately associated with changes in outcome also remains to be studied.

Conclusion

Beyond ABP and ICP level, simple measures of variability of ABP and ICP provide prognostic information regarding 6-month survival and functional outcome. The direction of the association depends on the time scale at which the variability is measured. Greater fast ABP and ICP variability as measured in this study in patients early following SAH may reflect relatively fast cardiovascular and cerebrovascular physiologic responsiveness and are associated with better outcome. Conversely, there is an association between greater slow ABP and ICP variability and poorer outcome.

Summary of Key Points

The critical care management of individuals in the intensive care unit with subarachnoid hemorrhage (SAH) has traditionally focused on maintaining ICP and ABP within prescribed ranges. However, research suggests that assessment of the dynamic variability of these pressure signals can provide additional information about physiologic functioning and may reflect adaptive capacity. The variability of ICP and ABP at different time scales (5-second to 24-hour) was measured in patients with subarachnoid hemorrhage and the association between variability measures and clinical condition and outcome was examined.

- Measures of ICP and ABP variability were better predictors of 6-month functional outcome than mean pressure levels.
- Greater fast ICP and ABP variability were associated with less severe SAH, as reflected by Glasgow Coma Scale score and Hunt and Hess score.
- Greater fast ICP and ABP variability were associated with better outcome, even after controlling for SAH severity.
- Greater slower ICP and ABP variability were associated with poorer outcome, even after controlling for SAH severity.
- Observation of ICP and ABP trends over periods longer than that routinely displayed on bedside clinical monitors is informative in relation to physiologic state and prediction of outcome.

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Figure 1.
Hunt and Hess Scores

Figure 2.
Percent in Extended Glasgow Outcome Scale Categories at Six Months

Figure 3A. ICP Variability over 24 Hours at Four Time Scales*: High variability; Favorable outcome (GOSE = 5)

Figure 3B. ICP Variability over 24 Hours at Four Time Scales*: Low variability; Unfavorable outcome (GOSE = 1)

*Top panel: original time series of 5-second data points
 Second Panel: hourly variability
 Third Panel: 5-minute variability
 Bottom Panel: 5-second variability

Figure 3.

Figure 4A. ABP Variability over 24 Hours at Four Time Scales*: High ABP 5-second RMSSD, 5-second SD variability; Favorable outcome (GOSE = 5)

Figure 4B. ABP Variability over 24 Hours at Four Time Scales*: Low ABP 5-second RMSSD, 5-minute SD; Unfavorable outcome (GOSE = 1)

*Top panel: original time series of 5-second data points
 Second Panel: hourly variability
 Third Panel: 5-minute variability
 Bottom Panel: 5-second variability

Figure 4.

Figure 5.

Odds of Survival at Six Months by ICP Variables

Controlling for initial Glasgow Coma Scale Score

Variability indices controlling for initial Glasgow Coma Scale Score and median ICP

Figure 6.
Odds of Survival at Six Months by ABP Variables
Controlling for initial Glasgow Coma Scale Score

Figure 7.
 Odds of Extended Glasgow Outcome Scale > 4 at Six Months by ICP Variables
 Controlling for initial Glasgow Coma Scale Score
 Variability indices controlling for initial Glasgow Coma Scale Score and median ICP

Figure 8.
Odds of Extended Glasgow Outcome Scale > 4 at Six Months by ABP Variables
Controlling for initial Glasgow Coma Scale Score

Table 1
Demographics, Clinical Condition, Management, and Outcome

Age in years	
Mean (SD)	53.4 (12.7)
Range	27–83
Gender	
Female (%)	74
Glasgow Coma Scale	
Mean (SD)	11.7 (3.9)
3–8 (%)	24
9–12 (%)	20
13–15 (%)	56
Aneurysm location (%)	
Anterior circulation	74
Posterior circulation	16
Ventriculostomy (%)	49
Craniotomy, aneurysm clipping (%)	87
Coiling (%)	10
Craniectomy (%)	19
Vasospasm (%)	
Present	82
Severe	33
Angioplasty (%)	28
Days in ICU	
Mean (SD)	13.8 (8.5)
Median	12.9
Range	0–63
Days in hospital	
Mean (SD)	20.7 (11.9)
Median	18.3
Range	2 – 63
Glasgow Coma Scale discharge (survivors)	
Mean (SD)	13.7 (1.8)
3–8 (%)	1
9–12 (%)	17
13–15 (%)	82
Discharge disposition (%)	
Home	24
Rehabilitation	30
Skilled nursing facility	20
Other	6
Died	20

Table 2

Physiologic Variables Over Four Days of Monitoring

Variability measure	ABP (mm Hg)	ICP (mm Hg)
Mean (SD)	103.2 (10.7)	11.6 (7.3)
Range	76.8 – 131.4	–1.7 – 42.2
5-second RMSSD		
Mean (SD)	3.5 (1.5)	1.2 (.87)
Range	1.6 – 10.1	.4 – 6.1
5-minute SD		
Mean (SD)	3.7 (1.1)	1.6 (.9)
Range	1.6 – 7.9	.5 – 5.3
5-minute to 60-minute SD		
Mean (SD)	3.9 (1.1)	1.7 (.7)
Range	2.3 – 8.6	.7 – 4.2
60-minute to 24-hour SD		
Mean (SD)	7.3 (2.6)	3.6 (1.7)
Range	2.7 – 21.3	.5 – 8.3

Table 3
Correlations between SAH severity and variability measures

Variability Measure	GCS score		Hunt & Hess	
	r	p	r	p
ABP RMSSD	.407	.000	-.463	.000
ABP 5-minute SD	.387	.000	-.360	.000
ICP RMSSD	.278	.008	-.329	.002
ICP 5-minute SD	.269	.010	-.318	.002
	Hunt & Hess		Hunt & Hess	
	1–3		4–5	
ABP RMSSD (mm Hg)				
Mean (SD)	3.8 (1.6)		2.8 (1.0)	
Range	1.9 – 10.1		1.6 – 6.0	
ABP 5-minute SD (mm Hg)				
Mean (SD)	3.9 (1.1)		3.1 (0.9)	
Range	2.2 – 7.9		1.6 – 5.3	
ICP RMSSD (mm Hg)				
Mean (SD)	1.4 (1.0)		0.9 (0.3)	
Range	0.4 – 6.1		0.4 – 1.7	
ICP 5-minute SD (mm Hg)				
Mean (SD)	1.8 (1.0)		1.2 (1.6)	
Range	0.6 – 5.3		0.5 – 3.0	

Table 4
Percent Subjects Receiving Major Medication Groups

Medication Group	Percent Receiving
Adrenergics (dopamine, dobutamine, levophed, neosynephrine, norepinephrine)	37
Adrenergic Blockers (esmolol, labetolol, metoprolol)	21
Analgesics (dilaudid, fentanyl, ibuprofen, morphine, oxycodone, acetaminophen)	100
Antiarrhythmics (amiodarone, disopyramide)	5
Anxiolytic/Sedative/Hypnotic (lorazepam, midazolam, propofol)	82
Corticosteroids (dexamethasone, fludrocortisone)	31
Diuretics (furosemide, mannitol)	50
Sodium Chloride Tablets	24
Vasodilators (apresoline, nitro paste, nipride)	16