The impact of a highly visible display of cerebral perfusion pressure on outcome in individuals with cerebral aneurysms

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BACKGROUND: Nurses' ability to rapidly detect decreases in cerebral perfusion pressure (CPP), which may contribute to secondary brain injury, may be limited by poor visibility of CPP displays.

OBJECTIVE: To evaluate the impact of a highly visible CPP display on the functional outcome in individuals with cerebral aneurysms.

METHODS: Patients with cerebral aneurysms (n = 100) who underwent continuous CPP monitoring were enrolled and randomized to beds with or without the additional CPP display. Six-month outcome was assessed.

RESULTS: Functional outcome was not significantly different between control and intervention groups after controlling for initial neurologic condition (odds ratio .904, 95% confidence interval 0.317 to 2.573). However, greater time below CPP thresholds (55 to 70 mm Hg) was significantly associated with poorer outcome (P = .005 to .010).

CONCLUSIONS: Although the enhanced CPP display was not associated with significantly better outcome, longer periods of CPP below set levels were associated with poorer outcome. (Heart Lung® 2008;37:227–237.)

After aneurysmal subarachnoid hemorrhage (SAH) brain injury beyond the initial bleed may occur as a result of secondary insults, such as inadequate cerebral blood flow and cerebral perfusion. These insults can contribute to cerebral ischemia and infarction and are associated with poorer outcome.[1-4] The prevention of ischemia is therefore one of the challenges of the acute management of SAH. Cerebral perfusion pressure (CPP), the difference between mean arterial blood pressure (ABP) and mean intracranial pressure (ICP) is commonly monitored in the critical care setting to assess global cerebral blood delivery. Regional cerebral blood flow is decreased after SAH, particularly in those with delayed neurologic deficit.[5-9] As a result of cerebral vasospasm and impaired cerebral autoregulation after SAH,[10-12] cerebral blood flow becomes particularly dependent on CPP. Therefore, maintaining adequate CPP after SAH is one component of preventing or minimizing cerebral ischemia.

Nurses have a key role in the monitoring and management of CPP. Nursing care activities, such as positioning or suctioning, can affect CPP, with transient or sustained activity-related decreases occurring, which may contribute to secondary ischemic brain injury. Moreover, nursing monitoring of CPP is essential for titration of medical management, such as administration of intravenous fluids, mannitol, or vasopressors. Nurses are aware of the need to keep CPP above a critical level, but interventions to do so are often based on hourly or half-hourly recordings of CPP. Although continuous CPP information may be available with current clinical bedside monitoring systems, these systems generally do not provide a highly visible, continuous display of CPP that brings immediate attention to care-related decreases

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in CPP or that clearly displays a trend of decreasing CPP. Nursing maneuvers to manage CPP may be enhanced by heightened awareness of these alterations in CPP, thus allowing for immediate intervention, such as head elevation to decrease ICP or titration of sedation or vasopressor medication. Therefore, a highly visible display of CPP may be expected to contribute to fewer episodes of CPP decreases below the critical level. To the degree that inadequate CPP contributes to secondary ischemic brain injury, preventing or minimizing these drops in CPP may contribute to improved short and long-term functional outcome.

This study was a randomized controlled trial to evaluate, in the context of optimal medical and surgical management, the impact of a highly visible continuous CPP display on CPP management and functional outcome in patients undergoing treatment for cerebral aneurysm. We hypothesized that patients randomized to the highly visible CPP display would have significantly better Extended Glasgow Outcome Scale (GOSE) and Functional Status Examination (FSE) scores at 6 months than those randomized to the control (blank) CPP display. A secondary aim of the study was to examine the effect of the percent of time that CPP was below various threshold levels on outcome after SAH. The hypothesis for this aim was that an increased percent of time below CPP thresholds would be associated with poorer outcome.

METHODS

Participants

Approval for the study was received from the local Institutional Review Board. Patients ≥16 years old who were admitted to an intensive care unit at an academic medical center between 2000 and 2002 with a cerebral aneurysm and who underwent invasive ICP and ABP monitoring were enrolled in the study. The majority presented with aneurysms that had ruptured, resulting in SAH. During the period of this study, a parallel study was carried out with a traumatic brain injury sample. Findings from that sample have been published separately.[13] Subjects were enrolled within 24 hours of ICP monitor insertion. Exclusion criteria were bilateral fixed pupils and impending death. Written consent for follow-up was obtained from the patients and/or a surrogate as appropriate. Outcome assessment was carried out at discharge and again at 3 and 6 months by trained interviewers.

During the time of this study, standard medical management of CPP was targeted at both ICP and ABP. There was no specific CPP management protocol used at the time of the study, and targets for CPP management were typically set on an individual basis. Specific SAH management included administration of nimodipine; cerebrospinal fluid drainage; early aneurysm obliteration whenever possible; daily transcranial Doppler sonography; and use of pulmonary artery catheters with hypertensive, hypervolemic, hemodilution therapy, and balloon angioplasty as indicated for cerebral vasospasm.

Interventions

Patients were randomized to beds with either a highly visible CPP display (intervention) or a blank CPP display (control), positioned on a stand at the head of the bed. The active display consisted of a bar graph reflecting CPP during the previous 30 minutes, with bars displayed in blue or red depending on whether CPP was above or below a threshold of 70 mm Hg, respectively. Given the absence of an established critical lower CPP threshold for patients with SAH, 70 mm Hg was selected. This was the recommended lower threshold for the critical care management of traumatic brain injury at the time of the study.[14] The numeric CPP value was also visible on the active CPP display. Update of the display occurred every 5 seconds. The blank display consisted of a black screen with the message "Program Running. Please Do Not Adjust."

Measurement

ABP was measured using intra-arterial catheters connected to Abbott Transpect pressure transducers (Abbott Laboratories, Abbott Park, IL). ICP was measured using intraparenchymal Camino transducer-tipped catheters (Integra LifeSciences, Plainsboro, NJ). Analog signals were input to the display computer from the bedside monitoring system (Spacelabs Medical, Redmond, WA). CPP was calculated as mean ABP minus mean ICP.

Data collection

Demographic (age and sex), diagnostic (computed axial tomography, angiography, transcranial Doppler sonography), and management (pharmacologic, surgical, or radiologic intervention) data were recorded. Hunt and Hess grade was documented. This grade reflects SAH severity based on clinical neurologic condition, with higher grades indicating more severe SAH.[15] Grade I reflects no symptoms or mild headache, whereas grade V reflects deep coma and moribund appearance. Individuals with unruptured aneurysms were assigned a grade of 0. Glas-

gow Coma Scale (GCS) scores were also recorded. The GCS score provides a means to quantify the level of consciousness and ranges from 3 (unresponsive) to 15 (alert and oriented).[16] Five-second summaries of ICP and ABP data were saved to the display computers.

Outcomes

The primary outcome measure was functional outcome, as evaluated using GOSE and FSE scores at 6 months. A secondary end point was survival at 6 months. The GOSE divides each of the severe-disability, moderate-disability, and good-recovery categories of the original Glasgow Outcome Scale into upper and lower categories,[17,18] resulting in an 8-point scale. A GOSE score of 1 represents death, and a score of 8 represents upper good recovery. The FSE assesses the changes in physical, social, and psychological functioning and financial independence that are attributable to acute brain injury.[19] Ten categories reflecting functional status are each scored from 0 to 3, with 0 indicating no change from pre brain injury functioning and 3 indicating complete dependence. Scores are summed to yield a final score ranging from 0 to 30, with a score of 31 assigned for death. Compared with the GOSE, where lower scores reflect poorer functioning, higher scores on the FSE reflect poorer functioning. Both GOSE and FSE scores were obtained using a standardized interview format.

Sample size

A sample size of 50/group allowed for detection of a true group difference of $\geq$0.8 on the GOSE with 80% power, controlling for age, sex, and SAH severity.

Randomization

Randomization was carried out using a block randomization procedure, with block size randomly varying between 4 and 6. Randomization assignments were generated, placed in consecutively numbered envelopes, and sealed. As patients were enrolled, the research nurse selected the next consecutive envelope and set up the active or blank display as indicated. Given the visibility of the display computer, care providers, patients, and families could not be blinded to randomization group assignment. However, individuals assessing outcome were blinded.

Statistical methods

Group differences in continuous variables reflecting baseline characteristics were assessed using the independent sample Student *t* test. $\mathbf{X}^2$ was used to assess group differences in categoric variables. The percent of time that CPP was below the thresholds was first calculated per day, and then the values for the first 4 days were averaged into 1 single value. This data-collection period encompassed a time during which subjects were at risk for low CPP and was thus a period during which it would be expected that the CPP display would be of greatest value. Binary logistic regression and analysis of variance were used to examine the primary intervention effect on outcome, controlling for age, sex, and measures reflecting SAH severity. Covariates used to statistically control for SAH severity included initial GCS score, Hunt and Hess grade, and initial presence of intraventricular hemorrhage and hydrocephalus. The outcome variables used in the binary logistic regression analyses were survival and GOSE, dichotomized to favorable outcome (lower and upper moderate disability, lower and upper good recovery) and unfavorable outcome (dead, vegetative, lower and upper severe disability). There is no established cut-off point at which to dichotomize the FSE to reflect favorable versus unfavorable outcome. The FSE was therefore treated as an interval variable for the purpose of statistical analysis.

The secondary purpose of this study was to explore the relationship between CPP and outcome, regardless of intervention or control group assignment. Little literature has specifically addressed the impact of CPP levels on outcome after SAH, so thresholds were selected within a range of CPP levels likely to be encountered clinically. The degree to which CPP below a particular threshold contributed to cerebral ischemia may depend not only on the threshold but also on the length of time below the threshold. We therefore examined the impact on outcome of the length of time CPP was below the threshold level. For each CPP threshold level (55 to 80 mm Hg, in 5–mm Hg increments), a dichotomous variable was created using receiver operating characteristic analysis to identify a cut-off percent of time that CPP was below threshold that identified subgroups at lower risk or higher risk for poorer outcome. The cut-off percent times used to create the dichotomized variable of greater or less time below the thresholds are presented in Appendix 1. As the CPP threshold decreased, the cut-off percent time also decreased, reflecting the greater risk for cerebral ischemia with shorter excursions below the

Table I
Demographic and clinical characteristics

Characteristics	Control (n = 49)	Intervention (n = 51)	*P*
Age in years (mean ± SD)	55 ± 12	52 ± 13	.37
Female sex (%)	76	77	NS
GCS-PR (mean ± SD)	12.4 ± 3.9	11.6 ± 3.8	.26
Median GCS-PR	15	13	
Hunt and Hess score (mean ± SD)	2.1 ± 1.4	2.9 ± 1.3	.01
No. Hunt and Hess score (%)			
0	6 (12)	3 (6)	
I	10 (20)	4 (8)	
II	16 (33)	13 (26)	
III	8 (16)	14 (29)	
IV	6 (12)	11 (22)	
V	3 (6)	6 (12)	
No. CAT findings (%)			
IVH	33 (67)	38 (75)	
ICH	10 (20)	11 (22)	
Hydrocephalus	30 (61)	34 (67)	
Aneurysm site (%)			
Anterior circulation	86	77	
Posterior circulation	14	24	
No. aneurysm management (%)			
Ventriculostomy	14 (29)	30 (59)	
Clipping	44 (90)	44 (87)	
Coiling	5 (10)	4 (8)	
Angioplasty	9 (18)	16 (31)	
No. vasospasm (%)	34 (69)	42 (82)	

CAT, Computed axial tomography; *GCS-PR,* postresuscitation Glasgow Coma Scale score; *ICH,* intracerebral hemorrhage; *IVH,* intraventricular hemorrhage.

lower thresholds. Although the percent times were relatively low, they reflected cumulative time during 4 days of monitoring. Times that CPP was below the different thresholds ranged from 16.3 hours (17%) to 6 minutes (0.1%).

For each CPP threshold level, analysis of variance was conducted, modeling the GOSE and FSE (dependent variables) by more or less percent time below CPP threshold, controlling for age, sex, and initial severity. In addition, binary logistic regression was performed using the dichotomous dependent variable survival (alive or dead) by more or less percent time below CPP threshold subgroup, again controlling for age, sex, and measures of SAH severity.

RESULTS

A total of 100 patients were enrolled. Patient demographics and clinical variables are presented in Table I. Control and intervention groups did not differ significantly with regard to age or sex. However, despite randomization, SAH severity differed between the intervention and control groups, with greater severity in the intervention group. The intervention group had a significantly higher mean Hunt and Hess grade ($P = .010$). The mean initial GCS score for the intervention group was lower than for the control group, although the difference was not statistically significant. Whereas 59% of the control group presented with a GCS score of 15, only 39% of the intervention group had an initial GCS score of 15 ($P = .046$). Almost one half (49%) of the intervention group was intubated on admission versus 29% of the control group ($P = .036$). The intervention group was significantly more likely to have a ventriculostomy during their course of treatment ($P = .002$). In addition, the intervention group was more likely to have intraventricular hemorrhage, ce-

Table II
Discharge status

Status	Control (n = 49)		Intervention (n = 51)	
Disposition	Actual	Predicted	Actual	Predicted
Home	22	22	7	10
Rehabilitation	11	12	18	20
Skilled nursing facility	10	7	8	5
Died	4	8	14	16
Other	2	0	4	0

Predicted values were determined using multinomial logistic regression and reflected numbers that would be expected in each category controlling for age, sex, and subarachnoid hemorrhage severity.

Table III
Outcome at 6 months by randomization group

ANOVA of GOSE scores at 6 months by randomization group[1]

Group	Control (n = 49)	Intervention (n = 51)	*F*	*P*
Mean GOSE (SEM)	4.13 (0.25)	3.82 (0.24)	.749	.389
Mean FSE (SEM)	18.46 (1.24)	19.02 (1.12)	.103	.749

Binary logistic regression prediction of favorable outcome at six months by randomization group[2]			Binary logistic regression prediction of 6-month survival by randomization group[2]		
OR	**95% CI**	***P***	**OR**	**95% CI**	***P***
.904	.317–2.573	.850	.600	.157–2.296	.456

Controlling for age, gender, and subarachnoid hemorrhage severity.
[1]Values adjusted for age, sex, and SAH severity.
[2]Controlled for age, sex, and SAH severity.

rebral vasospasm, and angioplasty, although these differences were not statistically significant. These variables all reflect poorer initial neurologic status, greater SAH severity, and a higher incidence of subsequent complications associated with more severe SAH in the intervention group.

Discharge disposition of study participants is listed in Table II. The actual numbers, along with the predicted numbers, in each category are listed. The distribution of the predicted values was determined using multinomial logistic regression, controlling for baseline demographic and SAH severity information, the latter of which differed significantly between the control and intervention groups.

Six-month mean adjusted GOSE and FSE scores are listed in Table III. These values indicated, on average, recovery to a level requiring daily dependence on others for at least some activities. Analysis of variance showed no significant difference in GOSE or FSE scores between the control and intervention groups after controlling for age, sex, and SAH severity. For logistic regression analyses, the odds ratios reflected the odds of a favorable outcome or survival at 6 months for subjects in the intervention group compared with those in the control group. There was not a statistically significant difference between groups in relation to those achieving a favorable outcome (OR 0.904, 95% con-

Table IV
Physiologic variables based on averages over the first 4 days of monitoring

Variables[1]	Control (n = 47)	Intervention (n = 50)	*P*
Mean CPP (mm Hg) (SD)	91.5 (13.6)	92.5 (11.0)	NS
% Time CPP <80	25.1 (24.2)	24.70 (23.8)	NS
% Time CPP <75	16.0 (21.4)	16.1 (18.7)	NS
% Time CPP <70	9.7 (16.6)	9.3 (12.5)	NS
% Time CPP <65	5.3 (12.0)	4.6 (6.9)	NS
% Time CPP <60	3.4 (10.5)	2.0 (3.1)	NS
% Time CPP <55	2.5 (9.5)	0.8 (1.5)	NS
Mean ABP (SD)	102.3 (12.6)	105.3 (10.2)	NS
Mean ICP (SD)	10.7 (6.8)	12.6 (7.2)	NS
Mean mannitol (mL) (SD)	55 (104)	144 (237)	.017
Mean packed red cells (mL) (SD)	36 (84)	81 (114)	.026

[1]For physiologic data, the sample size was 97 because data were incomplete for 3 subjects.

fidence interval [CI] .317 to 2.573) or 6-month survival (OR 0.600, 95% CI .157 to 2.296).

Intermediate variables that would reflect interventions carried out by nurses to manage CPP were examined for differences between the control and intervention groups. Mean ABP, ICP, and CPP, and percent of time CPP below various thresholds during the first 4 days of monitoring are listed in Table IV. There were no statistically significant differences in any of these measures between the intervention and control groups. However, individuals in the intervention group received, on average, significantly greater amounts of mannitol and packed red blood cells.

Although the percent time CPP was below the various thresholds was not statistically significantly different between the intervention and control groups, it was lower in the intervention group at the lower CPP thresholds, particularly 55 and 60 mm Hg. For example, for a threshold of 60 mm Hg, CPP was below the threshold 3.4% of the time for the control group versus 2.0% of the time for the intervention group. During a period of 4 days, this difference of 1.4% translates to the intervention group on average having CPP below the threshold for 80 minutes less than the intervention group. For a CPP threshold of 55 mm Hg, subjects in the intervention group on average had CPP below the threshold for 98 minutes less than those in the control group. The highly visible display may have been a contributing factor to these differences.

The effect on 6-month survival of more or less time with CPP below the specified CPP thresholds, controlling for age, sex, and SAH severity, was assessed using binary logistic regression (Table V). ORs reflected the odds of survival for subjects in the subgroup with CPP below the threshold for more time compared with those having CPP below the threshold for less time. The odds of 6-month survival were lower in those with more time below the CPP thresholds for all threshold levels, although this did not reaching statistical significance at the 75– and 80–mm Hg thresholds.

Table V
Binary logistic regression prediction of 6-month survival by more versus less percent time CPP was below specified thresholds[1]

Time CPP (mm Hg)	OR	95% CI	*P*
<80	.650	.186–2.267	.499
<75	.337	.097–1.169	.086
<70	.168	.043–.656	.010
<65	.136	.033–.555	.005
<60	.124	.029–.528	.005
<55	.131	.032–.534	.005

[1]Controlled for age, sex, and SAH severity.

GOSE and FSE scores at 6 months for those with more versus less percent of time below the specified CPP thresholds are presented in Figs 1 and 2, respectively. At all thresholds, average GOSE and FSE

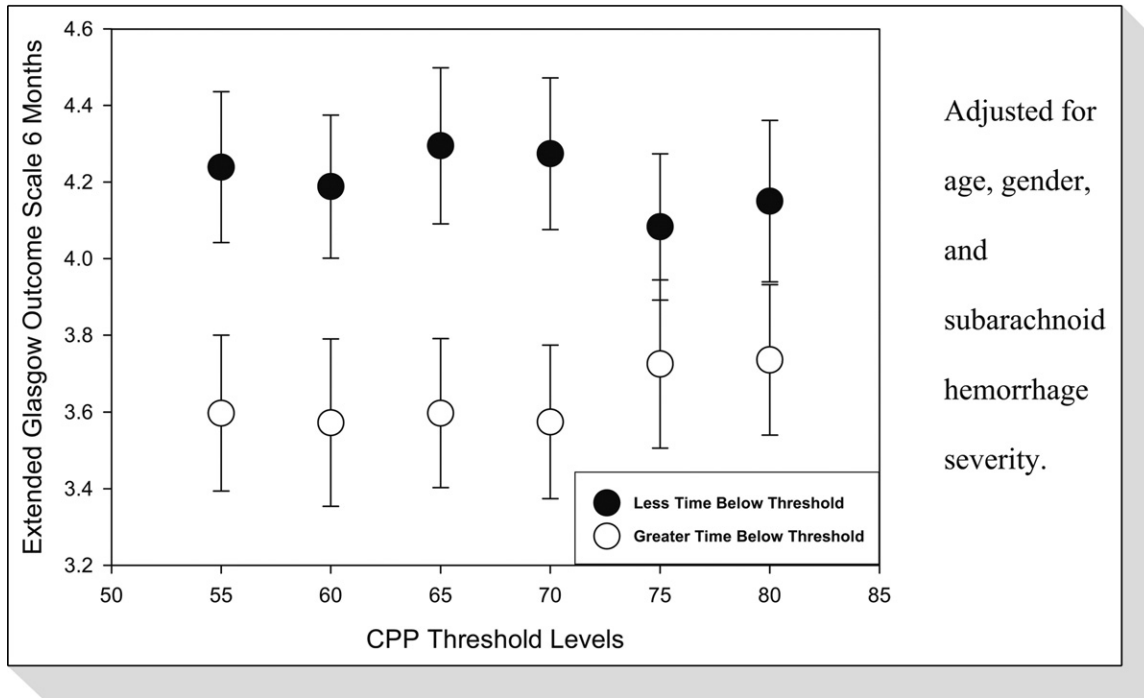

Fig 1 GOSE scores at 6 months by more versus less time below CPP thresholds. Scores were adjusted for age, sex, and SAH severity.

scores (controlling for age, sex, and SAH severity) reflected poorer outcome in the subgroups with more CPP time below threshold. Analysis of variance of 6-month outcome by more versus less CPP time below threshold shows that the differences in 6-month GOSE scores were statistically significant for thresholds ≤70 mm Hg (Table VI). Differences in FSE scores were not significantly different.

DISCUSSION

Critical care nurses have a key role in the monitoring and management of CPP after SAH. We are unaware of other studies that have examined the impact of the presentation of CPP information on nurses' ability to manage CPP and on patient outcome. We hypothesized that the presence of a highly visible CPP display would enhance nurses' ability to manage CPP, resulting in fewer episodes of CPP below a critical threshold and, to the extent that CPP below a critical threshold contributes to poorer outcome, would be associated with better outcome. However, the presence of a highly visible, real-time display of CPP was not associated with significantly better functional outcome at 6 months. A number of factors may have contributed to the lack of a difference in outcome between subjects with the active versus the blank displays.

Despite the randomization protocol, there was a randomization imbalance in relation to SAH severity, which is an identified predictor of outcome. Several measures of initial clinical condition, including Hunt and Hess grade, initial GCS score, and initial intubation status, all reflected greater SAH severity in the intervention group. Although these variables were included as covariates in the statistical analyses, this control may have been inadequate and may not have captured additional relevant latent differences between the control and intervention groups, such as difficulty of aneurysm obliteration. In addition, outcome for those with the most severe SAH may have been poor regardless of CPP management. Therefore, the likelihood of an intervention effect would be decreased given the higher percentage in the intervention group of those with the most severe SAH. To prevent such an imbalance in future studies, stratified randomization based on SAH severity is recommended. The majority of subjects had aneurysms that had ruptured, resulting in SAH. However, the percentage of subjects with unruptured aneurysms, who would be considered at lower risk for ischemic injury and poorer outcome, was higher in the control group. This may also have diluted an overall beneficial effect of the intervention.

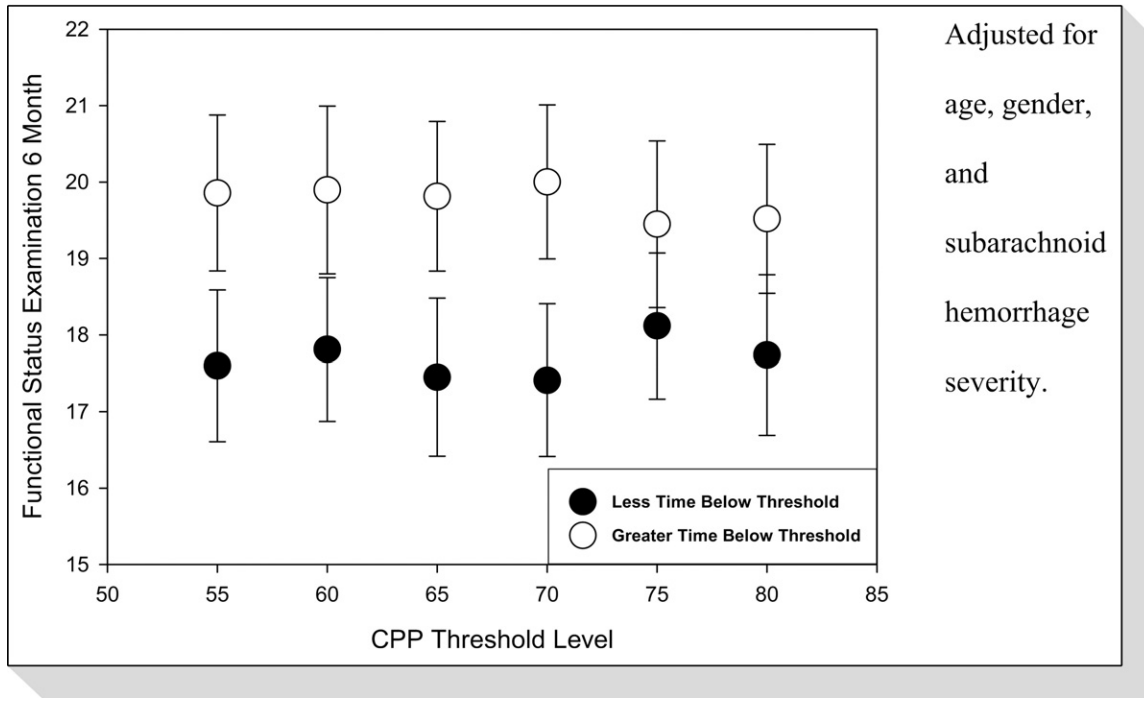

Fig 2 FSE scores at 6 months by more or less time below CPP thresholds. Scores were adjusted for age, sex, and SAH severity.

Table VI
ANOVA of 6-month outcome by more versus less time below CPP thresholds

GOSE score[1]			FSE score[1]		
CPP threshold	***F***	***P***	**CPP threshold**	***F***	***P***
55 mm Hg	5.144	.026	55 mm Hg	2.513	.116
60 mm Hg	4.569	.035	60 mm Hg	2.073	.153
65 mm Hg	6.100	.015	65 mm Hg	2.743	.101
70 mm Hg	6.153	.015	70 mm Hg	3.320	.072
75 mm Hg	1.498	.224	75 mm Hg	.835	.362
80 mm Hg	2.068	.154	80 mm Hg	1.541	.218

[1]Adjusted for age, sex, and SAH severity.

The severity of SAH represented in this sample may have been impacted by concurrent intervention studies. The inclusion criteria for the CPP study were broader with fewer exclusion criteria than the other studies, so some subjects were available for enrollment in the CPP study by virtue of not meeting inclusion criteria of other studies. This may have biased SAH severity in subjects in the CPP study in the direction of greater severity.

Whether patients were randomized to an active or a blank CPP display, CPP was monitored in all patients in the study, although with the standard bedside monitoring system it was not readily visible during care because of poor ergonomics. The CPP display was therefore an adjuvant to increase nurses' awareness of and ability to rapidly respond to decreases in CPP, not the sole source of CPP information. It is possible that as a result of the study, nurses had a greater awareness of CPP in all patients whether they had an active or a blank display. The lack of direct measurement of nurses' response to the CPP display is a limitation of this

study, and conclusions as to whether nursing management of CPP actually changed as a result of the CPP display are therefore speculative. A smaller study (n = 17) was carried out during this study to examine nursing response to CPP changes in patients in a traumatic brain injury sample of the study.[20] Patients with an active display, a blank display, or a usual bedside display were included. Sustained decreases in CPP were found to occur mostly when the nurse was out of the patient's room. Therefore, although the active display provided information about decreases occurring during the preceding 30 minutes that was unavailable with the blank or usual displays, it did not confer additional benefit to allow early responses to CPP decreases if the nurses were not in the room to see the decreases in real time. Incorporating an audible alarm to alert nurses that CPP was below a critical threshold would help address this issue.

Although the differences in percent of time CPP was below the various thresholds in the intervention and control groups were not statistically significantly, CPP was below the thresholds of 60 and 55 mm Hg, on average, 80 and 98 minutes less, respectively, in the group with the highly visible display than in the control group. This amount of time could be clinically significant in relation to secondary brain injury. It is not known, however, whether the intervention group had less time with CPP below these thresholds related to action by nurses in response to presence of the active display or related to other physician or patient factors influencing CPP.

The effects of ICP and ABP after SAH have typically been examined independently rather than as the combined parameter of CPP. Therefore, this study, which examined the impact of CPP on patient outcome, adds to the research literature beyond the independent effects of ICP and ABP on patient outcome. Considered independently, increased ICP is known to contribute to cerebral ischemia and negatively influence outcome after SAH.[21] Mean arterial blood pressure <70 mm Hg is also independently associated with death or severe disability at 3 months after SAH.[22] The simplified acute physiology score (SAPS) II has been used to examine the ability of 12 physiologic variables, age, type of admission, and three underlying disease characteristics to predict outcome and the occurrence of delayed cerebral ischemia after SAH.[23] The SAPS II value was the strongest and only independent predictor of poor outcome at 3 months after SAH in the multivariate analysis. In the univariate analysis, systolic blood pressure outside normal ranges was significantly associated with poor outcome. The SAPS II value was also the only independent predictor of delayed cerebral ischemia. Enblad examined the occurrence and impact on outcome of secondary brain insults, including CPP and ≤70 mm Hg, in patients with SAH.[24] CPP ≤70 mm Hg was the most frequently occurring secondary insult in the first week after SAH, with 48 episodes of at least 3 hours in length occurring in 55 patients (maximum count of 1/d). The effect of each insult was not analyzed separately, but the total number of insults was a significant independent predictor of outcome. The measures and analyses used in the previously mentioned studies do not allow for a direct comparison of findings with those of the current study; however, they do support a role of the components of CPP in outcome after SAH. In addition, the study by Enblad highlights the substantial number of potentially preventable secondary brain insults that occur despite intensive care management and the need for more precise monitoring and further examination of the impact of secondary insults on outcome. More visible and meaningful displays of relevant physiologic variables may contribute to increased detection and more rapid management by nurses, thus preventing or minimize the occurrence of secondary insults.

Medical management of this particular patient population was often targeted at maintaining specific levels of ICP, arterial blood pressure, and pulmonary capillary wedge pressure rather than specifically targeting CPP. Hypertensive–hypovolemic–hemodilution (triple-H) therapy[25-31] was used to treat cerebral vasospasm. Although hypertension and/or triple-H therapy have been shown to increase CBF[9,32] and reverse delayed ischemic deficits from cerebral vasospasm,[9,33] triple-H therapy is not without complications, and its value remains controversial.[26,30] However, as a result of its use in this study sample, overall mean CPP was >90 mm Hg, and subjects were not typically at risk for CPP decreases greater than what would generally be considered critical ischemic levels. Therefore, the beneficial effect of the active display in this study may have been limited to those subjects with low CPP levels who were at particular risk for cerebral ischemia.

Although most subjects did not have a high percentage of time with CPP below the various thresholds examined, within subjects, a relatively small percentage of CPP time below thresholds up to 70 mm Hg was significantly associated with poorer outcome. In the presence of cerebral vasospasm, triple-H therapy generally dictates the upper target

for ABP, and therefore, to a large degree, CPP. However, given the occurrence of low cerebral blood flow after SAH, the minimal CPP threshold is also of interest in defining risk for cerebral ischemia. This is particularly so when triple-H therapy is not used or has not yet been initiated. In this study, the critical time below CPP threshold levels that was associated with poorer outcome was threshold dependent, decreasing as the CPP threshold level decreased, reflecting less ability to tolerate CPP below lower thresholds. Whereas short periods of time of CPP below thresholds examined in this study may be tolerated in general, the study demonstrated that decreases occurring for longer periods, or cumulative decreases, were associated with poorer outcome. Individual patient circumstances are likely to determine the exact critical CPP thresholds, and the critical times below the thresholds, that are associated with poorer outcome. The neurologic impact of a decrease in CPP may not be readily apparent at the time of the decrease; however, nurses must be aware of potential cumulative effects beyond the immediate period.

CONCLUSION

Although the presence of a highly visible CPP display was not associated with better outcome in this study, an association between periods of low CPP and poorer outcome was found. Overall, the incidence of poor outcomes in individuals suffering aneurysmal SAH remains significant, and current management strategies are based on limited studies, many which suffer from methodologic weaknesses.[34] This leaves considerable room for improvement. Further research is needed to understand the impact of physiologic derangements, including low CPP, and their management on patient outcome after SAH. Given the complex multimodality physiologic monitoring in current intensive care management and the high incidence of secondary insults, improving the ergonomics of clinical monitoring systems can potentially contribute to nurses' ability to better control physiologic parameters, which may ultimately improve patient outcome after SAH.

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APPENDIX 1

The cut-off percent times used to create the dichotomized variable of more or less time below the threshold for each CPP threshold level are as follows:

CPP <80 mm Hg: 17% (54% had CPP <80 mm Hg for >17% of monitored time)
CPP <75 mm Hg: 13% (43% had CPP <75 mm Hg for >13% of monitored time)
CPP <70 mm Hg: 4% (50% had CPP <70 mm Hg for >4% of monitored time)
CPP <65 mm Hg: 1.3% (53% had CPP <65 mm Hg for >1.3% of monitored time)
CPP <60 mm Hg: 0.8 % (42% had CPP <60 mm Hg for >0.8% of monitored time)
CPP <55mm Hg: 0.1% (52% had CPP <55 mm Hg for >0.1% of monitored time).

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