Effective Glycemic Control With Aggressive Hyperglycemia Management Is Associated With Improved Outcome in Aneurysmal Subarachnoid Hemorrhage

Julius Gene S. Latorre, MD, MPH; Sherry Hsiang-Yi Chou, MD; Raul Gomes Nogueira, MD; Aneesh B. Singhal, MD; Bob S. Carter, MD; Christopher S. Ogilvy, MD; Guy A. Rordorf, MD

**Background and Purpose**—Hyperglycemia strongly predicts poor outcome in patients with aneurysmal subarachnoid hemorrhage, but the effect of hyperglycemia management on outcome is unclear. We studied the impact of glycemic control on outcome of patients with aneurysmal subarachnoid hemorrhage.

**Methods**—A prospective intensive care unit database was used to identify 332 patients with hyperglycemic aneurysmal subarachnoid hemorrhage admitted between January 2000 and December 2006. Patients treated with an aggressive hyperglycemia management (AHM) protocol after 2003 (N = 166) were compared with 166 patients treated using a standard hyperglycemia management before 2003. Within the AHM group, outcome was compared between patients who achieved good (mean glucose burden <1.1 mmol/L) and poor (mean glucose burden ≥1.1 mmol/L) glycemic control. Poor outcome was defined as modified Rankin scale ≥4 at 3 to 6 months. Multivariable logistic regression models correcting for temporal trend were used to quantify the effect of AHM on poor outcome.

**Results**—Poor outcome in AHM-treated patients was lower (28.31% versus 40.36%) but was not statistically significant after correcting for temporal trend. However, good glycemic control significantly reduced the incidence of poor outcome (OR, 0.25; 95% CI, 0.08 to 0.80; \( P = 0.02 \)) compared with patients with poor glycemic control within the AHM group. No difference in the rate of clinical vasospasm or the development of delayed ischemic neurological deficit was seen before and after AHM protocol implementation.

**Conclusion**—AHM results in good glucose control and significantly reduces the odds for poor outcome after aneurysmal subarachnoid hemorrhage in glucose-controlled patients. Further studies are needed to confirm these results. (Stroke. 2009;40:1644-1652.)

**Key Words:** critical care • hyperglycemia • intracranial aneurysm • outcome • subarachnoid hemorrhage

The incidence of aneurysmal subarachnoid hemorrhage (aSAH) is 6 to 10 per 100 000 per year or approximately 30 000 people per year, accounting for up to 3% of all strokes and approximately 5% of stroke-related deaths.1 Women are preferentially affected with a mean age at presentation of 55 years.2 Approximately 51% die in the first 30 days. Of the survivors, 33% require lifelong care,3 whereas only 50% achieve good outcome.4 Due to the substantial burden on healthcare resources, strategies to improve outcome is a desirable goal.

There is a growing body of experimental and clinical literature showing a significant association between persistent hyperglycemia and poor outcomes in different acute medical and surgical conditions.5–7 In aSAH, hyperglycemia has been linked to development of clinical vasospasm8 and poor outcome.9 Intensive insulin therapy has been shown to improve outcome in nonselected intubated patients in both medical and surgical intensive care units (ICUs) with an acceptable rate of complications related to hypoglycemia.10,11 Hyperglycemia represents a very common problem in aSAH, occurring in 70% to 90% of the patients.8 Thus, treatment of persistent hyperglycemia has become an attractive management strategy to improve outcomes in patients with aSAH.

Glucose management in patients with acute brain injury is complicated by the complex relationship of systemic and brain-specific factors governing the transport and usefulness of glucose. Because human neurons are insulin-insensitive cells,12 the cellular uptake of glucose by neurons is not increased by insulin and is primarily regulated by supply.13 Lowering systemic glucose levels, for example with intensive insulin therapy, can potentially reduce extracellular glucose levels in the brain.14 Low levels of brain extracellular glucose...
Table 1. Protocol for Aggressive Hyperglycemia Management

<table>
<thead>
<tr>
<th>Patient Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Blood glucose monitoring every 4 to 6 hours.</td>
</tr>
<tr>
<td>2. Intravenous insulin infusions in patients with admission glucose &gt;7.8 mmol/L (140 mg/dL) or persistent hyperglycemia (glucose &gt;7.8 mmol/L on 2 consecutive draws) to achieve blood glucose 4.4 to 7.8 mmol/L (80 to 140 mg/dL).</td>
</tr>
<tr>
<td>3. Regular insulin administered subcutaneously using a sliding scale to maintain blood glucose 4.4 to 7.8 mmol/L (80 to 140 mg/dL).</td>
</tr>
<tr>
<td>4. No dextrose-containing fluids.</td>
</tr>
<tr>
<td>5. Feeding started as soon as feasible with a goal to achieve 90% caloric requirement by Day 3.</td>
</tr>
</tbody>
</table>

Table 1. Protocol for Aggressive Hyperglycemia Management

have been associated with worse neurological outcome. Current recommendations advocate for a less restrictive target for systemic glycemic control in acutely brain-injured patients. This study investigated the effects of aggressive insulin therapy and resultant tight glycemic control on patient outcomes after aSAH.

Methods

Study Design

Using a prospectively collected Institutional Review Board-approved database of patients admitted to the Massachusetts General Hospital for the management of subarachnoid hemorrhage (SAH), we retrospectively identified all patients with SAH who were hospitalized between January 1, 2000, and December 31, 2006, and met the following inclusion criteria: (1) age >18 years; (2) survival through the first 72 hours of hospitalization; (3) documented aneurysm as the cause of SAH; (4) aneurysm repair by either endovascular coiling or surgical clipping within 72 hours of ictus; and (5) hyperglycemia defined as blood sugar >11.1 mmol/L (200 mg/dL) on admission or the first 24-hour mean blood sugar >7.8 mmol/L. Two patient groups were subsequently identified: (1) patients admitted before 2003 and treated with standard hyperglycemia management (SHM), targeting the blood glucose levels to <11.1 mmol/L (200 mg/dL); and (2) patients treated after 2003 and treated with aggressive hyperglycemia management (AHM), targeting the blood glucose levels between 4.4 and 7.8 mmol/L (80 to 140 mg/dL). Before 2003, all patients were treated with SHM. In 2003, we developed a new institutional protocol (Table 1) for aggressive hyperglycemia management. No other major changes in the management of patients with aSAH occurred during the study period. Patients admitted during the year 2003 were excluded from the study to reduce the differential effects of process implementation and intensive care staff learning curve. Patients who received AHM were further categorized as achieving good glycemic control (mean glucose burden <1.1 mmol/L [20 mg/dL] or poor glycemic control (mean glucose burden ≥1.1 mmol/L). Comparisons between the SHM and AHM groups were performed to establish any direct benefits of tight glycemic control on patient outcome. A secondary within-group analysis comparing patients who achieved good glycemic control with those who failed to achieve the target glucose level was also performed.

Study Population

A total of 332 patients met the inclusion criteria for the study out of 1139 patients admitted for management of SAH during the aforementioned time period. Of the 807 patients excluded, 261 had nonaneurysmal SAH, 174 were treated beyond 72 hours from symptom onset, 161 were admitted in 2003 during protocol development, 139 died in the ICU within 72 hours, 53 had normal blood sugar on admission and/or in first 24 hours, and 19 had incomplete records.

Standardized Clinical SAH Treatment Protocol

At our hospital, all patients with SAH are treated according to a standardized clinical protocol. Patients are assigned a Hunt and Hess (HH) clinical score and Fisher grade based on their initial clinical presentation and head CT imaging, respectively. Patients with known intracerebral aneurysm are treated with either surgical clipping or endovascular coiling within 24 hours of admission. The decision to clip or coil is based on collective decision among neurosurgery, neurointerventional, and neurocritical care teams. Patients with no visible aneurysm on conventional 4-vessel cerebral angiography undergo repeat diagnostic cerebral angiography 7 days postpresentation. All patients are monitored closely in the neurointensive care unit until resolution of vasospasm or posthemorrhage Day 10 (if they do not develop vasospasm). Patients receive continuous blood pressure monitoring with an arterial catheter and continuous central venous pressure monitoring with a central venous catheter. Extraventricular drains are placed before or during aneurysm repair in patients with clinical and radiographic evidence of hydrocephalus. Phenytoin is administered on hospital admission and discontinued after the aneurysm has been secured or if the patient is awake and following commands. All patients are treated with a 21-day course of oral nimodipine. Hyperthermia is treated with acetaminophen and surface cooling to euthermia. Euvolemia with target central venous pressure of 8 to 10 mm Hg is maintained. Patients are kept nothing by mouth and nonglucose-containing intravenous fluids are given in the first 24 hours. Enteral nutrition is started as soon as possible after the aneurysm is secured.

Patients receive daily transcranial Doppler ultrasound screening for vasospasm. All patients with clinical, transcranial Doppler, or angiographic vasospasm are treated with hemodynamic augmentation (systolic blood pressure >160 mm Hg) and hypervolemia (central venous pressure >8 cm H2O) until the resolution of clinical and angiographic vasospasm. Those with medically refractory vasospasm are treated with balloon angioplasty or intra-arterial infusion of vasodilators (nicardipine and/or milrinone).

Data Collection

Electronic medical records of included patients were reviewed. The following demographic and clinical characteristics were recorded: age, sex, HH grade, Fisher group, aneurysm location and size, number of aneurysm, medical history of hypertension, diabetes, heart disease and stroke, presenting symptoms of sentinel headache, seizure, loss of consciousness and prehospital worsening, admission CT evidence of infarction, intraparenchymal bleed, hydrocephalus, cerebral edema, intraventricular hemorrhage and midline shift, and admission glucose level.

Important aspects of patient management were also documented and included: need for emergent extraventricular drain, interval between symptom onset and aneurysm repair, method of aneurysm repair (clipping versus coiling), blood transfusion, hemodynamic augmentation therapy, perioperative steroid use, and pattern of insulin use.

Outcome Measures

Good and poor outcomes were defined as modified Rankin scale <4 and ≥4, respectively, and data were obtained at 3 to 6 months follow-up. In patients who did not have a follow-up record, the outcome was based on physical therapy notes recorded at the time of discharge. The occurrence of symptomatic vasospasm (defined as any transcranial Doppler peak velocity >200 cm/s or radiological evidence of vessel narrowing ≥25% from baseline or >50% from normal if no baseline study associated with clinical deterioration in the absence of other causes such as worsening hydrocephalus, rebleeding, seizures, infection, other systemic illness) and delayed ischemic neurological deficits (defined as persistent neurological abnormality and/or new CT or MRI evidence of ischemic infarction) were documented. Complications developing during hospitalizations were identified. Rebleed was defined as new or increased intracranial blood content from baseline; seizure was defined as clinical convulsive event or electroencephalogram evidence of rhythmic epilepto-
genic activity; cardiac dysfunction was defined as new evidence of congestive heart failure (documented by abnormal wall motion contractility or hypokinesia and ejection fraction <40% with clinical or radiographic evidence of pulmonary congestion) and/or development of acute myocardial infarction (documented by new electrocardiographic evidence of myocardial injury associated with troponin elevation >0.1 ng/mL and/or new segmental hypokinesia on echocardiography); respiratory failure was defined as hypoxemia requiring ventilatory support or failure to extubate within 24 hours postoperatively; infection was defined as development of pneumonia (radiological evidence of air space disease associated with pathological bacterial sputum growth and systemic inflammatory response) or central nervous system infection (cerebrospinal fluid culture positive for pathological bacteria/fungi); renal failure was defined as >50% elevation in baseline creatinine or azotemia requiring renal replacement therapy; venous thromboembolism was defined as sonologic or radiological evidence of deep venous thrombosis or development of pulmonary embolism. Hypoglycemia was defined as blood glucose <3.88 mmol/L (70 mg/dL). Hospital and ICU length of stays were also documented.

**Glucose Burden**

Admission blood glucose was recorded in all patients. Daily mean blood glucose was calculated by averaging the measured blood glucose for the day between Day 1 and Day 14. Overall mean blood glucose during ICU stay was calculated by averaging the daily mean blood glucose in the ICU. Daily mean glucose burden was defined as the daily mean blood glucose in excess of 7.8 mmol/L (140 mg/dL). The reference glucose value of 7.8 mmol/L was chosen based on our protocol and on prior studies.9 Total mean glucose burden was calculated by averaging the daily glucose burden. Poor glycemic control was defined as total mean glucose burden in excess of 1.1 mmol/L (20 mg/dL) above the target glucose level.

**Statistical Analysis**

All data were analyzed with SAS for Windows software, Version 9.1.3 (SAS Institute Inc, Cary, NC). Categorical data were analyzed using Fisher exact test or χ² tests as appropriate. Continuous data with normal distribution were reported as mean±SD and analyzed with Student t test, whereas those with nonnormal distribution were reported as median [interquartile range] and analyzed with Wilcoxon 2-sample test. The primary outcome was poor clinical outcome. Secondary outcomes included clinical vasospasm, delayed ischemic neurological deficits, medical complications, and length of stay in the hospital and neurointensive care unit.

The crude effect of aggressive hyperglycemia management on dichotomized primary and secondary outcome was determined using a χ² analysis. Multivariable adjusted logistic regression models were then used to evaluate the relationship of aggressive hyperglycemia management on poor outcome. A separate exploratory analysis was performed to evaluate the influence of glucose control in each group with Student’s t test as appropriate. Continuous data expected and consistent with other reports,10,11 there were more hypoglycemic episodes in the AHM group compared with the SHM group (22 [13.3%]); however, other aspects of patient treatment were similar between groups (Table 3).

**Glucose Control**

Admission glucose did not differ between the 2 groups. However, patients in the SHM group had significantly higher overall mean blood glucose (8.9 mmol/L [161.0 mg/dL] versus 7.7 mmol/L [138.4 mg/dL], P<0.01), overall mean glucose burden (1.2 mmol/L [26.5 mg/dL] versus 0.5 mmol/L [9.4 mg/dL], P<0.01), and longer hyperglycemia duration (7 days versus 5 days, P<0.01) compared with the AHM group. In addition, daily mean glucose values were significantly higher in SHM group versus AHM group from Day 1 to Day 14 (Figure 1). The proportion of patients achieving target glucose control was significantly higher in the AHM group compared with the SHM group (80.1% versus 52.4%, P<0.01).

Hypoglycemia occurred in 25 patients and all except one were receiving insulin during the hypoglycemic episode. As expected and consistent with other reports,10,11 there were no immediate hypoglycemia-related complications were seen and none of the patients who developed hypoglycemia had clinical sequela.

**Primary Outcome**

The proportion of poor outcome among patients treated with AHM after 2003 was significantly lower compared with patients treated with SHM before 2003 (28.3% versus 40.4%, P<0.01) and remained statistically significant after adjusting for age, sex, HH grade, Fisher group, method of aneurysm repair, history of hypertension, CT sign of infarction, and postoperative steroid use. However, using interrupted time series analysis, the difference could not be explained entirely by AHM protocol implementation after correcting for tem-
Older age (adjusted OR, 1.07; 95% CI, 1.04 to 1.10; \( P = 0.01 \)), poor HH grade (adjusted OR, 5.73; 95% CI, 2.03 to 16.19; \( P < 0.01 \) for HH Grade 3; OR, 23.31; 95% CI, 8.67 to 62.68; \( P < 0.01 \) for HH Grade 4 and 5), and admission CT evidence of infarction (adjusted OR, 5.27; 95% CI, 1.20 to 23.07; \( P = 0.03 \)) were found to be independently associated with poor outcome after multivariable adjustments (Table 5).

Admission glucose (10.4 mmol/L [186.9 mg/dL] versus 9.0 mmol/L [161.2 mg/dL], \( P < 0.01 \)), mean glucose (9.0 mmol/L [162.6 mg/dL] versus 8.3 mmol/L [148.5 mg/dL], \( P < 0.01 \)), duration of hyperglycemia (7.7 versus 5.9 days, \( P < 0.01 \)), and mean glucose burden (1.5 mmol/L [27.6 mg/dL] versus 1.0 mmol/L [17.1 mg/dL], \( P < 0.01 \)) were all significantly higher in patients with poor outcome. However, none of these glucose parameters were independent predictors of poor outcome after multivariable adjustments.

Among patients treated with AHM, achievement of good glycemic control significantly reduced the chance of poor outcome (adjusted OR, 0.25; 95% CI, 0.08 to 0.80; \( P = 0.02 \)) compared with patients who had poor glycemic control (Table 6).
More patients did not have a follow-up record in the SHM group compared with the AHM group (46 versus 17, respectively). However, the result of the analysis did not change when modified Rankin Scores obtained at hospital discharge were used.

**Secondary Outcomes**

There was no difference in rate of clinical vasospasm, delayed ischemic neurological deficit, and development of clinical complications before and after AHM protocol implementation (Table 4). ICU and hospital length of stays were comparable between the 2 treatment groups. Surgical aneurysm repair and poor HH grade significantly increased ICU (2.5 ± 1.0 days, \( P < 0.01 \), and 2.2 ± 0.5 days, \( P < 0.01 \), respectively) and hospital length of stay (4.9 ± 1.9 days, \( P = 0.01 \) and 3.2 ± 0.9 days, \( P < 0.01 \)).

Consistent with prior studies, Fisher group (adjusted OR, 3.97; 95% CI, 1.66 to 9.48; \( P < 0.01 \)) and poor HH grade...
In this study, we determined the impact of glucose control on outcome after aSAH in a cohort of patients using a prospectively collected database. We have shown that aggressive hyperglycemia management targeting a systemic blood glucose level of 4.4 to 7.8 mmol/L (80 to 140 mg/dL) is feasible and effective in achieving target glucose level with acceptable rates of hypoglycemia. Furthermore, we have demonstrated that among patients treated with aggressive hyperglycemia management, achievement of target glucose level was independently associated with good outcome (Figure 2).

Similar to a prior study, we did not find any association between admission hyperglycemia and poor outcome after multivariable adjustments suggesting that it is likely a marker of disease severity representing generalized catecholamine surge. However, failure to achieve the target glucose level in patients treated with aggressive hyperglycemia management was found to be significantly associated with poor outcome, highlighting the deleterious effect of persistent systemic glucose elevation.

**Discussion**

Intensive insulin therapy is currently advocated for use in general critical care practice based on 2 landmark studies by van den Berghe. The first study was conducted in surgical critically ill patients showing significantly reduced ICU-related complications and mortality with intensive insulin use. The second study conducted in the medical ICU reduced morbidity but did not have any impact on mortality. The results of these studies suggest that there may be differential effects of insulin therapy among various types of...
critically ill patients, including those with acute brain injuries. A recent report showed no difference in mortality, functional outcome, and occurrence of vasospasm among patients with aSAH treated with intensive insulin therapy compared with conventional glucose management. The study included 78 patients and only 69% of patients randomized to intensive insulin therapy achieved target glucose level. The small number of patients and the inability to achieve target glucose may have reduced the power of the study to detect significant difference. Oddo summarized the results of small clinical

<table>
<thead>
<tr>
<th>Variable</th>
<th>Good Control (N=133)</th>
<th>Poor Control (N=33)</th>
<th>Adjusted OR (95% CI)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary outcome</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poor outcome defined as mRS ≥4, N (%)</td>
<td>32 (24.06)</td>
<td>15 (45.45)</td>
<td>0.25 (0.08–0.80)</td>
<td>0.02</td>
</tr>
<tr>
<td><strong>Secondary outcome</strong></td>
<td></td>
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</tr>
<tr>
<td>Symptomatic vasospasm, N (%)</td>
<td>46 (34.59)</td>
<td>15 (45.45)</td>
<td>0.75 (0.33–1.71)</td>
<td>0.49</td>
</tr>
<tr>
<td>DINDs, N (%)</td>
<td>72 (54.14)</td>
<td>23 (69.70)</td>
<td>0.73 (0.31–1.72)</td>
<td>0.48</td>
</tr>
<tr>
<td><strong>Complication rates</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rebleed, N (%)</td>
<td>11 (8.27)</td>
<td>2 (6.06)</td>
<td>1.85 (0.34–10.06)</td>
<td>0.47</td>
</tr>
<tr>
<td>Intracranial hypertension, N (%)</td>
<td>59 (44.36)</td>
<td>15 (45.45)</td>
<td>1.10 (0.47–2.57)</td>
<td>0.83</td>
</tr>
<tr>
<td>Intracranial hemorrhage, N (%)</td>
<td>21 (15.79)</td>
<td>6 (18.18)</td>
<td>1.16 (0.40–3.40)</td>
<td>0.79</td>
</tr>
<tr>
<td>DVT/PE, N (%)</td>
<td>14 (10.53)</td>
<td>8 (24.24)</td>
<td>0.49 (0.17–1.43)</td>
<td>0.19</td>
</tr>
<tr>
<td>Cardiac dysfunction, N (%)</td>
<td>38 (28.57)</td>
<td>14 (42.42)</td>
<td>0.55 (0.23–1.32)</td>
<td>0.18</td>
</tr>
<tr>
<td>Seizure, N (%)</td>
<td>5 (3.76)</td>
<td>3 (9.09)</td>
<td>0.35 (0.07–1.81)</td>
<td>0.21</td>
</tr>
<tr>
<td>Infection, N (%)</td>
<td>62 (46.62)</td>
<td>18 (54.55)</td>
<td>1.00 (0.42–2.40)</td>
<td>0.99</td>
</tr>
<tr>
<td>Respiratory failure, N (%)</td>
<td>71 (53.38)</td>
<td>24 (72.73)</td>
<td>0.39 (0.12–1.22)</td>
<td>0.11</td>
</tr>
<tr>
<td>Renal failure, N (%)</td>
<td>6 (4.51)</td>
<td>4 (12.12)</td>
<td>0.50 (0.11–2.35)</td>
<td>0.38</td>
</tr>
<tr>
<td><strong>Length of stays</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital LOS, median (25%, 75%)</td>
<td>17 (13, 25)</td>
<td>21 (12, 28)</td>
<td>−2.93 (2.28)†</td>
<td>0.20‡</td>
</tr>
<tr>
<td>ICU LOS, median (25%, 75%)</td>
<td>13 (10, 16)</td>
<td>14 (9, 18)</td>
<td>0.78 (1.14)†</td>
<td>0.49‡</td>
</tr>
</tbody>
</table>

*P values from logistic regression except Hospital LOS and ICU LOS.
†Parameter estimate (SE).
‡P value from linear regression model.

mRS indicates modified Rankin scale; DINDs, delayed ischemic neurologic deficits; DVT, deep venous thrombosis; PE, pulmonary embolism; LOS, length of stay.

Figure 2. Patient outcome by treatment group and glucose control.
outcome studies comparing intensive insulin therapy with conventional glucose management and showed no significant effect on neurological outcome and mortality.16

A major concern with intensive insulin therapy for neurocritically ill patients is the danger of brain tissue hypoglycemia. The human brain is an obligate glucose consumer. Because neuronal tissues are insulin-insensitive, cerebral glucose uptake and metabolism is likely supply-driven in humans.13 There are experimental18 and clinical14,19 evidence that systemic lowering of glucose reduces brain tissue glucose concentration. Reduced cerebral tissue glucose in turn has been associated with elevated peri-ischemic cortical depolarization18 and with poor neurological outcome.14 However, there is also convincing evidence that persistent hyperglycemia exacerbates secondary brain injury and independently predicts poor outcome.8,9,20–22 The challenge in critical care for patients with SAH is in determining the optimal blood glucose target after acute brain injury. Because achievement of normoglycemia during insulin therapy may be associated with critical reduction in brain tissue glucose concentration, the lower limit of glucose target may need to be addressed in future studies.

Acute hyperglycemia in critically ill patients causes adverse effects from multiple mechanisms with positive feedback further upregulating the destructive processes.23 Although it has been postulated that insulin has direct beneficial effects for outcome after SAH,24,25 our study is consistent with previous clinical studies in showing that blood glucose control rather than the use of insulin is the main reason for improvement in patient outcomes.26

Our study has a number of limitations, most of which are related to its retrospective nature. First, we were unable to quantify the amount of insulin given to each patient. Second, we were unable to quantify the daily caloric load of each patient in relation to the glucose levels. However, all patients were managed in a standardized fashion with appropriate nutritional support and uniform use of normal saline without dextrose. Hence, we do not believe significant differences in nutritional intake would be found in the 2 groups. Third, because of the extended period of the study, secular trend may have influenced the outcomes. This was addressed using an interrupted time series analysis with the inclusion of the year of admission as one of the predictor variables in the multivariable model.

Conclusion

Effective aggressive glucose management to maintain blood glucose <140 mg/dL is associated with better neurological outcome in patients with aSAH. Further studies are needed to validate our results and to explore the feasibility and safety of aggressive glucose management in a broader patient population in the neurointensive care unit.

Acknowledgments

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Disclosures

None.

References


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