Multimodality Monitoring for Cerebral Perfusion Pressure Optimization in Comatose Patients With Intracerebral Hemorrhage

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Background and Purpose—Limited data exist to recommend specific cerebral perfusion pressure (CPP) targets in patients with intracerebral hemorrhage. We sought to determine the feasibility of brain multimodality monitoring for optimizing CPP and potentially reducing secondary brain injury after intracerebral hemorrhage.

Methods—We retrospectively analyzed brain multimodality monitoring data targeted at perihematomal brain tissue in 18 comatose intracerebral hemorrhage patients (median monitoring, 164 hours). Physiological measures were averaged over 1-hour intervals corresponding to each microdialysis sample. Metabolic crisis was defined as a lactate/pyruvate ratio >40 with a brain glucose concentration <0.7 mmol/L. Brain tissue hypoxia (BTH) was defined as $P_{btO2}$ <15 mm Hg. Pressure reactivity index and oxygen reactivity index were calculated.

Results—Median age was 59 years, median Glasgow Coma Scale score was 6, and median intracerebral hemorrhage volume was 37.5 mL. The risk of BTH, and to a lesser extent metabolic crisis, increased with lower CPP values. Multivariable analyses showed that CPP <80 mm Hg was associated with a greater risk of BTH (odds ratio, 1.5; 95% confidence interval, 1.1–2.1; $P=0.01$) compared to CPP >100 mm Hg as a reference range. Six patients died (33%). Survivors had significantly higher CPP and $P_{btO2}$ and lower ICP values starting on postbleed day 4, whereas lactate/pyruvate ratio and pressure reactivity index values were persistently lower, indicating preservation of aerobic metabolism and pressure autoregulation.

Conclusions—$P_{btO2}$ monitoring can be used to identify CPP targets for optimal brain tissue oxygenation. In patients who do not undergo multimodality monitoring, maintaining CPP >80 mm Hg may reduce the risk of BTH. (Stroke. 2011;42:3087-3092.)

Key Words: brain tissue oxygen ■ cerebral perfusion pressure ■ intracerebral hemorrhage ■ intracranial pressure ■ lactate/pyruvate ratio ■ metabolic crisis ■ pressure reactivity index

High blood pressure after intracerebral hemorrhage (ICH) is associated with hematoma expansion, aggravation of perihematoma edema, and poor clinical outcome.1–3 The American Stroke Association guideline for lowering mean arterial pressure <130 mm Hg and maintaining a cerebral perfusion pressure (CPP) >60 mm Hg in patients with suspicion of increased intracranial pressure (ICP) is based on limited clinical evidence.4 Recent studies have shown aggressive blood pressure control is feasible and might be beneficial in reducing early hematoma expansion in patients with acute ICH.5,6 However, these studies were mainly performed in ICH patients with relatively mild deficits. The benefits of aggressive blood pressure control are even more controversial in severe ICH patients. Brain tissue oxygen tension ($P_{btO2}$) and microdialysis monitoring are used to detect impending ischemia as evidenced by brain tissue hypoxia (BTH) or derangements of oxidative metabolism. BTH and metabolic crisis (MC), defined as elevation of the lactate/pyruvate ratio (LPR) with concurrent brain tissue hypoglycemia, are associated with poor clinical outcome in comatose brain-injured patients.7–9 Poor neurological outcome in brain-injured patients is associated with autoregulatory failure, a phenomenon in which ICP and $P_{btO2}$ levels correlate positively with arterial blood pressure.10,11 Continuous monitoring of cerebrovascular autoregulation, brain tissue oxygenation, and metabolism may help us better-understand the relationship between blood pressure and secondary tissue injury after ICH.12,13 In this

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study, we sought to establish the feasibility of brain multimodality monitoring (MMM) for guiding CPP management in comatose patients with ICH. Specifically, we hypothesized that a CPP threshold exists below which perihematomal BTH and MC increasingly occurs.

Subjects and Methods

Study Population

Nineteen comatose ICH patients underwent MMM between May 2006 and September 2010 in our neurointensive care unit according to a standardized protocol. Patients considered eligible for monitoring had a Glasgow Coma Scale score of 3 to 8 and a supratentorial hemorrhage within 3 cm of the hemorrhage margin whenever possible. Probe location is available (Supplemental Figure I, http://stroke.ahajournals.org). One monitored patient was excluded from the analysis because probes were placed in infarcted tissue. In 2 patients who underwent hemicraniectomy for ICP control, probes were inserted contralateral to the hemorrhage based on the appearance of bilateral IVH and global cerebral edema. This observational study was approved by the Columbia University Medical Center Institutional Review Board.

Clinical Management

All patients were treated according to a standardized management protocol. An ICP goal of <20 mm Hg was maintained using a stepwise management strategy.4,14 CPP was targeted at >60 mm Hg at all times and was directed at higher target levels on a case-by-case basis as needed to optimize PbtO2 based on daily review of previous 24-hour data. All patients were ventilated to achieve an arterial oxygen saturation ≥95% and PCO2 of 30 to 40 mm Hg. PbtO2 measurements were excluded from this analysis when the fraction of inspired oxygen (FIO2) exceeded 50%.

Data Acquisition

A high-resolution data acquisition system (BedmasterEX; Excel Medical Electronics) was used to acquire digital data every 5 seconds. ICP monitoring was performed using a parenchymal ICP probe (Camino System; Integra Neurosciences), PbtO2 was measured with a Clark type probe (Licox System; Integra Neurosciences), and microdialysis monitoring was performed with a 20K Dalton cut-off catheter with 10-mm membrane length (CMA Microdialysis). Cerebrovascular pressure reactivity index (PRx)15 and oxygen reactivity index16 were calculated post hoc as the running 200-second Pearson correlation coefficient between ICP and mean arterial pressure (PRx) and PbtO2 and CPP (oxygen reactivity index). PRx and oxygen reactivity index values range from +1 to −1, with more positive values indicating impaired autoregulation.

Radiological Image Analysis

Admission brain CT scans were analyzed using MIPAV software (Medical Image Processing, Analysis, and Visualization, version 4.3; National Institutes of Health).13 Regions of hemorrhage on CT scan were outlined slice-by-slice using a semiautomatic threshold approach by a rater blinded to all clinical information.18 Parenchymal hematoma and IVH volumes were calculated.

Statistical Analysis

All physiological variables were averaged over the time period corresponding to each microdialysis sample (usually every hour). MC was defined as a LPR >40 and brain glucose <0.7 mmol/L.19 BTH was defined as PbtO2 <15 mm Hg.20,21 Optimal CPP for autoregulation (CPPPRx) for each day was defined as the CPP point that the daily observed CPP was higher than the CPPPRx.22 Delta CPP was defined as the mean daily CPP-CPPPRx. A positive delta CPP means that the daily observed CPP was higher than the CPPPRx.

Univariate comparisons of pooled data were performed using a generalized linear model using a binomial distribution and logit link graf

<table>
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<th>N</th>
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<th>Sex</th>
<th>Location</th>
<th>Etiology</th>
<th>GCS</th>
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<th>Percent of MMM With BTH</th>
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<th>IVH Volume (mL)</th>
<th>Onset to MMM (hr)</th>
<th>Duration of MMM (hr)</th>
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<td>HT</td>
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</tbody>
</table>

AA indicates amyloid angiopathy; BTH, brain tissue hypoxia; F, female; GCS, Glasgow Coma Scale; HT, hypertension; IVH, intraventricular hemorrhage; M, male; MC, metabolic crisis; MMD, Moyamoya disease; MMM, multimodality monitoring.
function and were extended by generalized estimating equations using the autoregressive process to handle repeated observations within subject. SPSS 18 software (SPSS) was used for data analysis. \( P < 0.05 \) was considered statistically significant.

### Results

#### Demographics

Among 18 comatose and ventilated ICH patients, 9 were women and the median age was 59 years (interquartile range, 42–67; Table 1). Median parenchymal ICH volume was 37.5 mL (interquartile range, 1.8–62.3), median IVH volume was 27 mL (interquartile range, 3–49), median Glasgow Coma Scale score was 6 (interquartile range, 4–8), and the median MMM time was 164 hours (interquartile range, 88–204). Thirteen patients had deep hemorrhages, and all but 1 had coexisting IVH. No complications occurred as a result of MMM probe insertion. Six patients (33%) died in the hospital; all 12 survivors were discharged to either acute or subacute rehabilitation facilities. Life support was actively withdrawn in 5; 1 patient (patients 16) died of brain death related to ICP crisis.

#### Relationship of CPP to Brain Tissue Oxygenation and Metabolic Crisis

Analysis of 24-hour data frequently revealed linear relationships between \( P_bO_2 \) and CPP, which tended to resolve over time (Supplemental Figure II, http://stroke.ahajournals.org). The probability of BTH increased significantly from 21% to 58% as CPP declined from \( 90 \) to \( 50 \) mm Hg (Figure 1). A less pronounced relationship existed between CPP and MC; the probability was 17% when CPP was \( 70 \) mm Hg and steadily declined to 3% when CPP was \( 110 \) mm Hg.

### Table 2. Univariate Analysis of Risk Factors for Brain Tissue Hypoxia and Metabolic Crisis

<table>
<thead>
<tr>
<th></th>
<th>Brain Tissue Hypoxia</th>
<th></th>
<th>Metabolic Crisis</th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td>OR (95% CI)</td>
<td>( P )</td>
<td>OR (95% CI)</td>
<td>( P )</td>
</tr>
<tr>
<td><strong>Demographics</strong></td>
<td></td>
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<tr>
<td>Female</td>
<td>1.63 (0.46–5.71)</td>
<td>0.45</td>
<td>1.88 (0.25–14.35)</td>
<td>0.54</td>
</tr>
<tr>
<td>Age, per y</td>
<td>1.00 (0.97–1.04)</td>
<td>0.91</td>
<td>0.98 (0.92–1.03)</td>
<td>0.39</td>
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<tr>
<td><strong>Medical history</strong></td>
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<tr>
<td>Hypertension</td>
<td>0.83 (0.29–2.36)</td>
<td>0.73</td>
<td>0.57 (0.09–3.78)</td>
<td>0.56</td>
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<tr>
<td>Diabetes mellitus</td>
<td>2.13 (0.54–8.39)</td>
<td>0.28</td>
<td>2.66 (0.34–21.02)</td>
<td>0.35</td>
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<tr>
<td>Smoking</td>
<td>1.58 (0.45–5.52)</td>
<td>0.47</td>
<td>4.25 (0.71–25.52)</td>
<td>0.11</td>
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<tr>
<td><strong>Baseline clinical</strong></td>
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<tr>
<td>ICH volume, per mL</td>
<td>0.99 (0.97–1.02)</td>
<td>0.79</td>
<td>1.01 (0.98–1.03)</td>
<td>0.57</td>
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<tr>
<td>IVH volume, per mL</td>
<td>1.00 (0.97–1.04)</td>
<td>0.76</td>
<td>0.93 (0.87–1.00)</td>
<td>0.06</td>
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<tr>
<td>Glasgow Coma Scale</td>
<td>1.12 (0.80–1.56)</td>
<td>0.50</td>
<td>0.98 (0.85–1.11)</td>
<td>0.34</td>
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<tr>
<td>APACHE II subscore*</td>
<td>0.95 (0.86–1.04)</td>
<td>0.23</td>
<td>0.95 (0.82–1.09)</td>
<td>0.45</td>
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<tr>
<td><strong>Daily variables</strong></td>
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<tr>
<td>ICH day</td>
<td>0.90 (0.73–1.11)</td>
<td>0.32</td>
<td>1.06 (0.93–1.20)</td>
<td>0.40</td>
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<tr>
<td><strong>Hourly Variables</strong></td>
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<tr>
<td>CPP, per 10 mm Hg</td>
<td>0.82 (0.69–0.96)</td>
<td>0.014</td>
<td>0.98 (0.90–1.07)</td>
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<tr>
<td>ICP, per mm Hg</td>
<td>1.02 (0.98–1.07)</td>
<td>0.31</td>
<td>0.99 (0.97–1.01)</td>
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<tr>
<td>ETCO(_2), per mm Hg</td>
<td>0.85 (0.78–0.93)</td>
<td>&lt;0.001</td>
<td>0.95 (0.90–1.01)</td>
<td>0.11</td>
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<tr>
<td>Serum glucose, per mmol/L</td>
<td>1.01 (1.00–1.02)</td>
<td>0.06</td>
<td>1.00 (0.99–1.02)</td>
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<tr>
<td>Prx &gt; 0.2</td>
<td>3.05 (1.47–6.31)</td>
<td>0.003</td>
<td>0.98 (0.76–1.27)</td>
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<td>ORx &gt; 0.2</td>
<td>0.89 (0.45–1.75)</td>
<td>0.75</td>
<td>1.12 (1.02–1.23)</td>
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</table>

**Note:** APACHE indicates Acute Physiology and Chronic Health Evaluation; CI, confidence interval; CPP, cerebral perfusion pressure; ETCO\(_2\), end tidal carbon dioxide; ICH, intracerebral hemorrhage; ICP, intracerebral pressure; IVH, intraventricular hemorrhage; OR, odds ratio; ORx, oxygen reactivity index; Prx, pressure reactivity index.
Table 3. Multivariable Model for Predicting Brain Tissue Hypoxia

<table>
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<tr>
<th>Brain Tissue Hypoxia</th>
<th>Survivors (N=12)</th>
<th>Nonsurvivors (N=6)</th>
<th>P</th>
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<td>ETCo2 &lt; 34 mm Hg</td>
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<td>&lt;0.01</td>
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<td>Ranges of CPP</td>
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<td>&gt;100 mm Hg</td>
<td>Reference group</td>
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<td>90–100 mm Hg</td>
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<td>&lt;60 mm Hg</td>
<td>1.8 (1.3–2.4)</td>
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CI indicates confidence interval; CPP, cerebral perfusion pressure; ETCo2, end-tidal carbon dioxide; OR, odds ratio; PRx, pressure reactivity index.

Predictors of Brain Tissue Hypoxia

Univariate analysis showed that patients were 18% less likely to experience BTH for every 10-mm Hg increase in CPP (odds ratio, 0.82; 95% confidence interval, 0.69–0.96; P<0.01; Table 2). ETCo2 and impaired pressure autoregulation (PRx > 0.2) were also significantly associated with BTH. In a multivariable generalized estimating equations model, low CPP and ETCo2 levels <34 mm Hg (dichotomized on median value) were significantly associated with BTH after adjusting for age, admission Glasgow Coma Scale, Acute Physiology and Chronic Health Evaluation (APACHE) II subscore, and PRx > 0.2 (Table 3). The odds for BHT reached statistical significance when CPP was <80 mm Hg compared to CPP ≥100 mm Hg as a reference (odds ratio, 1.5; 95% CI, 1.1–2.1).

Predictors of Metabolic Crisis

Univariate analysis showed that patients were 12% more likely to have MC when their oxygen reactivity index was >0.2 (odds ratio, 1.12; 95% confidence interval, 1.0–1.2; P=0.02). However, in a multivariable model no variables were statistically significant for predicting MC (Table 2).

Factors Associated With In-Hospital Mortality

No baseline characteristics, including age, Glasgow Coma Scale score, and ICH or IVH volume, were associated with in-hospital mortality. By contrast, all but 1 of the continuously recorded variables were significantly different in survivors and nonsurvivors. Patients who survived had lower ICP and higher CPP values with similar mean arterial pressure levels, slightly higher Pao2 and microdialysis glucose values, and lower LPR values (Table 4). Delta CPP was significantly greater in survivors compared to those who died. Both PRx and oxygen reactivity index were significantly lower in survivors, indicating greater preservation of autoregulation. Analysis of time-series data comparing survivors and nonsurvivors showed that survivors had significantly higher CPP and Pao2 levels starting on postbleed day 4 and lower ICP values starting on day 5 (Figure 2). By contrast, survivors had significantly lower PRx and LPR values, indicating preserved pressure autoregulation and aerobic metabolism, respectively, throughout the entire monitoring period.

Discussion

We retrospectively analyzed data to determine whether Pao2 and microdialysis monitoring can identify CPP thresholds that might minimize the risk of secondary brain injury. We found that the risk of BTH increased significantly when CPP decreased to <80 mm Hg compared to >100 mm Hg as a reference range. Nonsurvivors had early and sustained impairment of pressure autoregulation (high PRx) and aerobic metabolism (high LPR), and with delayed reductions in CPP and Pao2 in the setting of increased ICP.

Established clinical predictors of mortality, including age, Glasgow Coma Scale score, and volume of hemorrhage were not significantly different in those who died or survived because of the similar severity of illness across patients we studied. By contrast, our findings indicate that mortality was associated with impaired cerebral autoregulation, lower CPP and higher ICP values, and a greater burden of BTH and anaerobic tissue metabolism. Nonsurvivors had persistently positive PRx values (Figure 2), indicating impaired autoregulation. The prognostic meaning of PRx has been addressed by other groups, showing that low CPP contributed to poor outcome among ICH patients with impaired cerebrovascular reactivity (PRx >0.2).24 We also found persistent extracellu-
lar LPR elevations and lower brain tissue glucose levels among those who died. Cerebral lactate and LPR elevation been associated with mortality after severe traumatic brain injury and poor-grade subarachnoid hemorrhage, but to our knowledge it has not yet been linked to poor outcome after ICH. The lack of association between MC with CPP in our study supports the concept that persistent mitochondrial dysfunction may play a more important role than hypotension as a cause of impaired energy metabolism.26

Survivors had a progressive reduction in ICP and increase in CPP after day 3 (Figure 2). Even among the nonsurvivors, mean ICP was maintained at <20 mm Hg throughout the entire monitoring period, which likely reflects the aggressive management protocol that we adhered to. Survivors in our study not only had higher CPP values overall, but also higher delta CPP (CPP-CPPPRx) values, indicating less likelihood for CPP to decline below the threshold for optimal pressure reactivity. Further studies are needed to determine whether goal-directed CPP optimization aimed at minimizing BTH can improve outcome after ICH.

Our study has several important limitations. The subject cohort was small, and generalizability is hampered by the fact that all patients were treated in a single facility. Hospital mortality is widely accepted as a reliable and valid end point for clinical research, but there is increasing awareness that decisions to withdraw care can influence survival. Because caregivers were not blinded to the MMM results, it is conceivable that these data could have affected decision-making, but we feel that this is unlikely. Life support was actively withdrawn in all but 1 of the 6 patients who died. Decisions to continue or withhold life support are an important potential source of bias that may have influenced our mortality analysis. Unfortunately, we did not measure long-term survival and functional recovery in this retrospective hospital-based study. Given the fact that almost every MMM variable that we recorded was associated with mortality, larger studies are needed to better-understand their relative importance for predicting outcome.

**Conclusions**

In conclusion, our findings suggest that MMM is a feasible method for optimizing perfusion and possibly minimizing secondary injury in comatose ICH patients. Because this analysis was viewed as exploratory and hypothesis-generating, our findings clearly require independent confirmation in a larger multicenter patient population.

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Disclosures

None.

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Multimodality Monitoring for Cerebral Perfusion Pressure Optimization in Comatose Patients with Intracerebral Hemorrhage

Running head: MMM in intracerebral hemorrhage

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Keywords: intracerebral hemorrhage, cerebral perfusion pressure, intracranial pressure, brain tissue oxygen, metabolic crisis, lactate/pyruvate ratio, Pressure reactivity index,
S1. Example of MMM probe placement in a patient with putaminal intracerebral hemorrhage

Note that the monitoring probe was placed in the perihematomal tissue in the frontal lobe (arrow) (patient 16 in Table 1).
S2. 24-hour plots of $P_{bt}O_2$ versus CPP in patient 6.

On Day 1 a linear relationship between CPP and $P_{bt}O_2$ is evident, indicative of autoregulatory failure. On Days 2 and 3 the curve begins to flatten as higher $P_{bt}O_2$ and CPP levels are maintained. On day 4 the $P_{bt}O_2$ and CPP relationship is completely flat, indicating restoration of normal autoregulation.
뇌내출혈을 가진 환자에서 뇌관류압 최적화를 위한 다중 감시 장치

**Abstract 7**

**Multimodality Monitoring for Cerebral Perfusion Pressure Optimization in Comatose Patients With Intracerebral Hemorrhage**

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(*Stroke*. 2011;42:3087-3092.)

**Key Words:** brain tissue oxygen ■ cerebral perfusion pressure ■ intracerebral hemorrhage ■ intracranial pressure ■ lactate/pyruvate ratio ■ metabolic crisis ■ pressure reactivity index

배경과 목적
뇌내출혈(intracerebral hemorrhage)을 가진 환자들에서 특정 뇌관류압(cerebral perfusion pressure)의 수치를 제한하는 연구는 매우 드물다. 저자들은 뇌관류압을 최적화하고 뇌내출혈 후 발생할 수 있는 이차적인 뇌손상을 줄이기 위한 뇌 다중 감시 장치의 실행 가능성을 검토하고자 하였다.

방법
저자들은 18명의 환자 뇌내출혈 환자에서 후향적으로 출혈 주변 뇌 조직에서 측정한 뇌 다중 감시 자료(중간값, 164시간)를 분석하였다. 생리학적 치료들이 각각의 미세투석법(microdialysis) 제료당 1시간 간격의 평균값으로 제시되었다. 대사적 위기(metabolic crisis)는 식약성-피루브산 비율(lactate/pyruvate ratio)이 40 초과, 뇌 당(glucose) 농도가 0.7 mmol/L 미만일 때로 정의되었다. 뇌 조직 저산소증(hypoxia)은 뇌 조직 산소 분압이 15 mm Hg 미만일 때로 정의되었다. 또한, 압력 반응 지수(pressure reactivity index)와 산소 반응 지수(oxygen reactivity index)가 계산되어 제시되었다.

결과
뇌 내출혈은 50세, 글레스코혼수질환(Glasgow Coma Scale) 점수의 중간값은 6점, 뇌내출혈 부위의 중간값은 37.5 mL이었다. 뇌 조직 저산소증의 위험도는 뇌관류압이 낮을수록 증가하였고, 대사적 위기인 뇌 조직 저산소증보다는 적은 정도였지만 뇌내출혈과 연관성이 있었다. 당뇨병 분석에서 80 mm Hg 미만의 뇌관류압은 100 mm Hg 초과의 뇌관류압에 비해 뇌 조직 저산소증의 위험도 증가가 유의하게 관찰되었다(OR=1.5: 95% CI, 1.1~2.1; P=0.01). 6명의 환자(33%)가 사망하였는데, 생존자는 뇌내출혈 4일째부터 시작하여 유의하게 높은 뇌관류압은 뇌 조직 저산소 중상 및 뇌두개내압을 가졌다. 반면에, 식약성-피루브산 비율과 압력 반응 지수는 지속적으로 높았는데, 이는 산소부화와 압력 자동조절능의 보존을 시사하는 것으로 볼 수 있었다.

결론
뇌 조직 산소 분압 감시는 뇌 조직 산소공급의 최적화를 위한 뇌관류압의 목표를 설정하기 위해 사용할 수 있다. 다중 감시 장치를 적용하지 않는 환자에서 80 mm Hg 이상의 뇌관류압의 유지는 뇌 조직 저산소증의 위험도 감소시킬 수 있다.
昏睡状態の脳内出血患者における脳灌流圧最適化のための
多元的モニタリング

Multimodality Monitoring for Cerebral Perfusion Pressure Optimization in Comatose Patients
With Intracerebral Hemorrhage

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Abstract

脳内出血患者における脳灌流圧（CPP）については、具体的な目標値を推奨するデータが限られて
いる。本研究は、CPPの最適化に加え、脳内出血後の二次的な脳損傷を低減するための脳の多元的モニタリングの
実施可能性を明らかにすることを目的とした。

方法：脳死状態の脳内出血患者18例（モニタリングの中央値：164時間）における血圧変動の脳組織を対象とした
脳の多元的モニタリングのデータを後向きに分析した。

生理的測定結果は微小透析の各検体に応じて、1時間
間隔で平均化した。電極モニター・クライシスは、乳酸
とビリルピン酸比が40かつ脳内のグルコース濃度が0.7
mmol/Lと定義した。脳組織低酸素症（BTH）はP_{iO_2}が
15 mm Hgと定義した。血圧反応性指数および酸素反応性
指数を算出した。

結果：年齢の中央値は59歳、グラスゴー・コマスケー
ルスコアの中央値は6、および脳内出血容積の中央値は
37.5 mLであった。BTHのリスク、および程度は低いが
高率に示された。多変量解析では、CPPが80 mm Hg
は、基準範囲のCPPが100 mm Hgと比較して、BTHのリスクを増
加させた（オッズ比1.5、95%信頼区間：1.1～2.1、p =
0.01）。患者6例が死亡した（33%）が、生存した患者は、
CPPおよびP_{iO_2}有意に高く、出血後4日目にICPが
低値になり始めたが、乳酸/ビリルピン酸比および血圧反応性
指数が低値を維持し、好気性代謝および血圧自己調節能
の保持が示された。

結論：P_{iO_2}のモニタリングは、最適な脳組織酸素化のため
のCPPの目標値を特定する目的に有用である。多元的モニタリングを実施していない患者では、CPPが
80 mm Hg
に維持することでBTHのリスクを低減できると考えられる。

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