Intra- and Extra-Cranial ICU Neuromonitoring

Thomas P. Bleck MD FCCM
Louise Nerancy Eminent Scholar in Neurology
Professor of Neurology, Neurological Surgery, and Internal Medicine
Director, Neuroscience Intensive Care Unit
The University of Virginia
Relationships to disclose
Disclosures

- Research support from NIAID, NINDS, Alsius, Pharmos
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- Lecturer for ESP/Pharma, Ortho-McNeil
- I once got a turkey sandwich from Aspect, the manufacturer of the BIS monitor
Continuous EEG Monitoring in the Intensive Care Unit: An Overview

Lawrence J. Hirsch

Abstract: Due to technological advances, it is now feasible to record continuous digital EEG (CEEG), with or without video, in critically ill patients and review recordings remotely. Nonconvulsive seizures (NCSzs) are more common than previously recognized and are associated with worse outcome. The majority of seizures in ICU patients are nonconvulsive and will be missed without CEEG. Factors associated with an increased risk for NCSzs include coma, prior clinical seizures, CNS infection, brain tumor, recent neurosurgery, and periodic epileptiform discharges. In addition to detecting seizures, CEEG is also useful for characterizing paroxysmal spells such as posturing or autonomic changes, detecting ischemia, assessing level of sedation, following long-term EEG trends, and prognosticating. Most NCSzs will be detected in the first 24 hours of CEEG in noncomatose patients, but longer recording periods may be required in comatose patients or in those with periodic epileptiform discharges. EEG patterns in encephalopathic or comatose patients are often equivocal. How aggressively to treat NCSzs and equivocal EEG patterns in these patients is unclear and requires further research. Real-time detection of ischemia at a reversible stage is technologically feasible with CEEG and should be developed into a practical form for prevention of in-hospital infarction in the near future.

Key Words: Continuous EEG monitoring, Critically ill, Intensive care unit, Nonconvulsive seizures, Seizure detection, Ischemia detection.


memory storage capabilities, and the ability to review studies remotely via computer networking.

The most common reason for performing CEEG (Table 1) is to detect nonconvulsive seizures (NCSzs) or nonconvulsive status epilepticus (NCSE) (Fig. 1C). Although previously thought to be uncommon, NCSzs and NCSE are being recognized more frequently. In fact, it is fair to say that anyone who works with critically ill neurologic patients and does not see NCSzs and NCSE on a regular basis is missing the diagnosis.

Continuous EEG monitoring is quite helpful for characterizing spells in intensive care unit (ICU) patients. It is not unusual for comatose or stuporous patients to have sudden posturing, rigidity, tremors, chewing, agitation, or sudden changes in pulse or blood pressure without an obvious explanation. All of these could be seizures, though they are usually not. We have seen patients with paroxysmal spells of whole-body rigidity and tremors/jerking diagnosed as generalized convulsions by the neurologists at the bedside, but which proved to have no EEG correlate (an example of what I refer to as ICU pseudoseizures). EEG recording of a spell such as this would obviously affect diagnosis and management.

In patients that require sedation or paralysis for medical management, CEEG can help assess the level of sedation and identify clinically silent neurologic events. Although anesthe-
EEG in the ICU

• recognized reasons to perform EEG in the ICU
  – status epilepticus
  – diagnosis and management of other seizures
  – workup of encephalopathies
  – determination of the level of awareness of patients
    • receiving NMJ blockade
    • suffering from severe NMJ or peripheral nerve disorders (e.g., Guillain-Barré syndrome)
Technical aspects of EEG in the ICU

- electrical safety
- electrodes
  - stability
  - infection risk
  - CT/MR compatibility
- artifact reduction
- data management
Succinylcholine induced hyperkalemia and cardiac arrest death related to an EEG study

- The authors report the case of a patient who developed cardiac arrest causally related to administration of succinylcholine for reduction of excessive amounts of myogenic artifact during an EEG.
- This case indicates the need for caution when doing an EEG study in an intensive care unit setting.

The ACNS Subcommittee on Research Terminology for Continuous EEG Monitoring: Proposed Standardized Terminology for Rhythmic and Periodic EEG Patterns Encountered in Critically Ill Patients

Lawrence J. Hirsch,* Richard P. Brenner,† Frank W. Drislane,‡ Elson So,§ Peter W. Kaplan,|| Kenneth G. Jordan,¶ Susan T. Herman,# Suzette M. LaRoche,** Bryan Young,†† Thomas P. Bleck,‡‡ Mark L. Scheuer,† and Ronald G. Emerson*

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FIGURE 1. SI-GRDA: Stimulus-induced generalized rhythmic delta activity. In this case, the pattern was elicited by suctioning the patient.
<table>
<thead>
<tr>
<th>Old Term</th>
<th>New Term</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triphasic waves, most of record</td>
<td>Continuous 2/sec GPDs (with triphasic morphology can be added)</td>
</tr>
<tr>
<td>PLEDs</td>
<td>LPDs</td>
</tr>
<tr>
<td>BIPLEDs</td>
<td>BIPDs</td>
</tr>
<tr>
<td>GPDs/PEDs</td>
<td>GPDs</td>
</tr>
<tr>
<td>FIRDA</td>
<td>Occasional brief 2/sec GRDA (if 1–10% of record); frontally predominant can be added</td>
</tr>
<tr>
<td>PLEDS+</td>
<td>LPDs+</td>
</tr>
<tr>
<td>SIRPIIDs w/focal evolving RDA</td>
<td>SI-Evolving LRDA</td>
</tr>
<tr>
<td>Laterализed seizure, delta frequency</td>
<td>Evolving LRDA</td>
</tr>
<tr>
<td>Semirhythmic delta</td>
<td>Quasi-RDA</td>
</tr>
</tbody>
</table>

*Some could have alternative new terms depending on the exact pattern.
SIRPIIDs, stimulus-induced rhythmic, periodic, or ictal discharges.
FIGURE 2. Evolving LRDA: Lateralized rhythmic delta activity that evolves in morphology and frequency. It begins as low-voltage, sharply contoured 1.5-Hz delta in the left parasagittal region, evolves to 3-Hz rhythmic delta, and then slows once again.
FIGURE 3. LRDA-plus: LRDA with superimposed repetitive sharp waves (several marked with asterisks).
FIGURE 5. GPDs-plus: One- to 1.25-per-second GPDs with superimposed low-amplitude quasi-rhythmic sharp fast activity (highlighted in boxes).
EEG stages of SE

1. Discrete seizures
2. Merging seizures
3. Continuous ictal activity
4. Continuous ictal activity with flat periods
5. Periodic epileptiform discharges

Treiman et al. Epilepsy Res 1990;5:49-60
Discrete seizures

Figure 1A. Discrete seizures (Pattern 1)(Patient #1)

Labar and Barrera J Clin Neurophysiol 2002 in press
Merging seizures

Figure 1B. Waxing and waning merging seizures (Pattern 2) (Patient #1)
Continuous ictal activity

Figure 1C. Continuous ictal activity (Pattern 3)(Patient #1)
Periodic epileptiform activity

Figure 1D. Periodic epileptiform discharges (Pattern 5)(Patient #1)
Residual electrographic SE after control of visible SE in VACSP 265

- 130 overt GCSE patients in whom EEG monitoring was begun within 30 minutes of start of treatment
- 26/130 (20%) remained in electrographic SE after motor movements had stopped (twitchless electrical activity)

Faught *Epilepsia* 1998
Persistent nonconvulsive SE after the control of convulsive SE

- 52% had no after-SE ictal discharges
  - EEG showed generalized slowing, attenuation, PLEDS, focal slowing, and/or burst suppression
- The remaining 48% demonstrated persistent electrographic seizures
  - over 14% manifested NCSE, predominantly CPSE

DeLorenzo et al Epilepsia 1998;39:833-40
Unsuspected NCSE

• 8% of a consecutive series of patients referred for EEG because of coma had NCSE

Burst-suppression myths

- EEG burst-suppression has been demonstrated to be necessary for RSE control
- achieving burst-suppression means that the patient will not have seizures
- the burst-suppression pattern is easily recognized and taught, even for non-neurologists
Depth of EEG suppression and outcome in barbiturate anesthetic treatment for refractory status epilepticus

- Retrospective review of 40 patients with RSE treated with pentobarbital
- 5 died during treatment
- survival correlated best with the etiology of SE

Krishnamurthy and Drislane *Epilepsia* 1999;40:759-762
Depth of EEG suppression and outcome in barbiturate anesthetic treatment for refractory status epilepticus

<table>
<thead>
<tr>
<th>EEG pattern</th>
<th>Slow</th>
<th>S-B</th>
<th>Flat</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>3</td>
<td>12</td>
<td>20</td>
</tr>
<tr>
<td>SE duration</td>
<td>6h</td>
<td>16h</td>
<td>14h</td>
</tr>
<tr>
<td>PB duration</td>
<td>26h</td>
<td>72h</td>
<td>14h</td>
</tr>
<tr>
<td>survival</td>
<td>3 (100%)</td>
<td>3 (25%)</td>
<td>12 (60%)</td>
</tr>
</tbody>
</table>

median durations
Unsuspected electrographic status epilepticus in intensive care units

• 89 patients with electrographic SE (52 with generalized seizures, 37 with focal seizures) over 15 years

• 74 had clinically evident seizures prior to the EEG
  – 45 had clinical SE, however,
  – only 20 had recognized seizures at the time of the EEG
  – 31 of 67 non-anoxic patients (including 15 who were comatose) improved in alertness on antiseizure drugs
  – Patients with focal discharges more likely to respond to antiseizure drugs: 20/37 vs. 11/52 (p < 0.01)

Drislane FW et al *Neurology* 1998;50(Suppl 1)
Prevalence of nonconvulsive status epilepticus in comatose patients

- A total of 236 patients with coma and no overt clinical seizure activity were monitored with EEG as part of their coma evaluation.
- Only cases that were found to have no clinical signs of SE were included in this study.
- EEG showed that 8% met their criteria for the diagnosis of NCSE.

Early prediction of outcome from cerebral trauma by SSEPs

- prognostic value of SSEPs during the first 4 days after severe head injury was studied in a group of 100 ICU patients
- strong association between the presence of bilateral cortical potentials and a good recovery or moderate disability 6 months after injury

Judson JA et al Crit Care Med 1990;18:363-8
Early prediction of outcome from cerebral trauma by SSEPs

- bilateral or unilateral absence of cortical potential was associated with severe disability, persistent vegetative state, or death in a high percentage of patients
- reliable prediction of outcomes was obtained from SSEPs recorded within 24 h of head injury
Table 1. Relationship between category of the first SEP recorded and outcome for the 100 patients

<table>
<thead>
<tr>
<th>Category of SEP</th>
<th>Patients with Favorable Outcome</th>
<th>Patients with Unfavorable Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Category 1 SEP</td>
<td>33</td>
<td>5</td>
</tr>
<tr>
<td>Category 2 SEP</td>
<td>19</td>
<td>7</td>
</tr>
<tr>
<td>Category 3 SEP</td>
<td>3</td>
<td>33</td>
</tr>
</tbody>
</table>
Favorable recovery from bilateral loss of somatosensory evoked potentials

- Four patients with viral encephalitis, CBZ intoxication, head trauma, and left-side, space-occupying hemispheric infarction, respectively.
- Favorable recovery from bilaterally absent cortical SEP

Favorable recovery from bilateral loss of somatosensory evoked potentials

• Three patients had an excellent outcome (GOS 4 and 5).
  – In those three patients, the SEP became completely normal during the clinical course
  – In one patient who remained severely disabled, the SEP became detectable again over the contralateral hemisphere, but remained abnormal
Systematic review of early prediction of poor outcome in anoxic-ischemic coma

• studies concerning patients older than 10 y with anoxic-ischemic coma in which findings from early neurologic exam, EEG, or SSEPs were related to poor outcome
  – defined as death or survival in a vegetative state

Zandbergen EG et al Lancet 1998 Dec 5;352(9143):1808-12
Systematic review of early prediction of poor outcome in anoxic-ischemic coma

• SSEP has the smallest CI of its pooled positive-likelihood ratio and its pooled false-positive test rate.

• Because evoked potentials are also the least susceptible to metabolic changes and drugs, recording of SSEP is the most useful method to predict poor outcome.
Monitoring severe head injury: a comparison of EEG and SSEPs

- long-term monitoring of the EEG power spectrum and SSEPs in 103 patients with severe closed head injury (GCS ≤ 8)
- At 7 of 9 twelve hour time intervals post injury, SSEPs were significantly (p < .05) different between outcome groups using the Glasgow Outcome Score collapsed to 3 categories

Monitoring severe head injury: a comparison of EEG and SSEPs

- The percent delta in the EEG was not significantly different between outcome groups at any time point post injury.
- The total power in the EEG power spectrum differed only at the last time epoch post injury (108 hrs).
- Based on the superior prognostic capabilities of the SSEP, they routinely base management decisions on SSEP values.
Early recovery after closed traumatic head injury: Somatosensory evoked potentials and clinical findings

• Prospective study of 31 patients with SSEPs first obtained within 48 hrs of admission
• 23 of 31 patients had abnormal SEP findings on initial exam
  – 11 recovered clinically, 2 remained vegetative, and 10 died
  – In all 11 patients with clinical recovery, SEP also recovered
• In 8 of 31 patients, initial SEPs were normal and remained normal until discharge; all 8 had a good outcome.
• Initial SEP findings were related with outcome at 6 months (p<.02), and follow-up studies increased the predictive value of SEP

Table 5. Recovery of variables of somatosensory evoked potential at different time intervals in patients recovering from closed head injury$^a$

<table>
<thead>
<tr>
<th>SEP Examination Interval</th>
<th>Latencies</th>
<th>Amplitude</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CCT</td>
<td>N13-P25-IPL</td>
</tr>
<tr>
<td>First 48 hrs</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Days 3–6</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Days 7–14</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Days 15–30</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Days 31–62</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>Mean day of recovery</td>
<td>9.6</td>
<td>15.3</td>
</tr>
</tbody>
</table>

SEP, somatosensory evoked potentials; CCT, central conduction time; N13-P25-IPL, N13-P25-interpeak-latency; IC, intermediate components.

$^a$Of all patients, only initially pathological SEP components with subsequent recovery are documented here.
Early detection of vasospasm after acute SAH using continuous EEG ICU monitoring

- Vespa et al studied 32 SAH patients using cEEG and trending of the quantitative measure, relative alpha (RA), to determine if reductions in RA variability occurred with documented vasospasm.
- In 19/19 patients with angiographically documented vasospasm, found RA variability decreased by a mean of two grades and improved with resolution of vasospasm.
  - In 10/19 this reduction in RA variability preceded the diagnosis of vasospasm by a mean of 2.9 days (SD 1.73).
Poor RA Variability 1

Fair RA Variability 2

Good RA Variability 3

Excellent RA Variability 4
Fig. 2. Temporal profile of RA variability is shown for each of the 19 patients. RA variability scores [4 (excellent), 3 (good), 2 (fair), 1 (poor)] at 3 time points (before vasospasm, during vasospasm and upon resolution of vasospasm) are plotted for each patient. Four patients had only two time points, during vasospasm and resolution of vasospasm. As a group, RA variability declines during vasospasm and improves upon resolution of vasospasm. All RA values were integers (1–4). Graphically, values were slightly dispersed along the y-axis for ease of visual review.
Early detection of vasospasm after acute SAH using continuous EEG ICU monitoring

- positive predictive value 76%
- negative predictive value 100%
- non-diagnostic clinical signs at the time of RA variability reduction and vasospasm were present in 12/19 patients

Vespa PM *EEGJ* 1997;103:607-15
Ion changes in spreading ischaemia induce rat middle cerebral artery constriction in the absence of NO

Olaf Windmüller,1 Ute Lindauer,1 Marco Foddis,1 Karl M. Einhäupl,1 Ulrich Dirnagl,1 Uwe Heinemann2 and Jens P. Dreier1

In rats, cortical spreading hyperaemia is coupled to a spreading neuroglial depolarization wave (spreading depression) under physiological conditions, whereas cortical spreading ischaemia is coupled to it if red blood cell products are present in the subarachnoid space. Spreading ischaemia has been proposed as the pathophysiological correlate of the widespread cortical infarcts abundantly found in autopsy studies of patients with subarachnoid haemorrhage. The purpose of the present study was to investigate whether the extracellular ion changes associated with the depolarization wave may cause the vasoconstriction underlying spreading ischaemia. We induced spreading ischaemia in vivo with the nitric oxide (NO) scavenger oxyhaemoglobin and an elevated K+ concentration in the subarachnoid space while slow potential, pH, extracellular volume and concentrations of K+, Na+, Ca2+ and Cl− were measured in the cortex with microelectrodes. We then extraluminally applied an ionic cocktail (cocktailSI) to the isolated middle cerebral artery in vitro, matching the ionic composition of the extracellular space as measured during spreading ischaemia in vivo. Extraluminal application of cocktailSI caused middle cerebral artery dilatation in the absence and constriction in the presence of NO synthase inhibition in vitro, corresponding with the occurrence of spreading hyperaemia in the presence and spreading ischaemia in the absence of NO in vivo. The L-type Ca2+ inhibitor nimodipine caused the cocktailSI-induced vasoconstriction to revert to vasodilatation in the absence of NO in vitro similar to the reversal of spreading ischaemia to spreading hyperaemia in response to nimodipine in vivo. We found that K+ was the predominant vasoconstrictor contained in cocktailSI. Its vasoconstrictor action was augmented by NO synthase inhibition. Our results suggest that, under elevated baseline K+ as a hallmark of any condition of energy deficiency, the extracellular ion changes represent the essential mediator of the vascular response to spreading neuroglial depolarization. In the presence of NO they mediate vasodilatation and in its absence they mediate constriction.
Spreading and Synchronous Depressions of Cortical Activity in Acutely Injured Human Brain

Anthony J. Strong, DM; Martin Fabricius, DMSc; Martyn G. Boutelle, PhD; Stuart J. Hibbins, MSc; Sarah E. Hopwood, PhD; Robina Jones, MRCS; Mark C. Parkin, BSc; Martin Lauritzen, DMSc

Background and Purpose—Cortical spreading depression (CSD) has been much studied experimentally but never demonstrated unequivocally in human neocortex by direct electrophysiological recording. A similar phenomenon, peri-infarct depolarization, occurs in experimental models of stroke and causes the infarct to enlarge. Our current understanding of the mechanisms of deterioration in the days after major traumatic or ischemic brain injury in humans has not yielded any effective, novel drug treatment. This study sought clear evidence for the occurrence and propagation of CSD in the injured human brain.

Methods—in 14 patients undergoing neurosurgery after head injury or intracranial hemorrhage, we placed electrocorticographic (ECoG) electrodes near foci of damaged cortical tissue.

Results—Transient episodes of depressed ECoG activity that propagated across the cortex at rates in the range of 0.6 to 5.0 mm/min were observed in 5 patients; this rate of propagation is characteristic of CSD. We also observed, in 8 of the 14 patients, transient depressions of ECoG amplitude that appeared essentially simultaneous in all recording channels, without clear evidence of spread.

Conclusions—These results indicate that CSD or similar events occur in the injured human brain and are more frequent than previously suggested. On the basis of these observations, we suggest that the related phenomenon, peri-infarct depolarization, is indeed likely to occur in boundary zones in the ischemic human cerebral cortex. (Stroke. 2002;33: 2739-2744.)
Role of magnesium in the reduction of ischemic depolarization and lesion volume after experimental subarachnoid hemorrhage

Object. Ischemia-induced tissue depolarizations probably play an important role in the pathophysiology of cerebral ischemia caused by parent vessel occlusion. Their role in ischemia caused by subarachnoid hemorrhage (SAH) remains to be investigated. The authors determined whether ischemic depolarizations (IDs) or cortical spreading depressions (CSDs) occur after SAH, and how these relate to the extent of tissue injury measured on magnetic resonance (MR) images. In addition, they assessed whether administration of MgSO₄ reduces depolarization time and lesion volume.

Methods. By means of the endovascular suture model, experimental SAH was induced in 52 rats, of which 37 were appropriate for analysis, including four animals that underwent sham operations. Before induction of SAH, serum Mg²⁺ levels were measured and 90 mg/kg intravascular MgSO₄ or saline was given. Extracellular direct current potentials were continuously recorded from six Ag/AgCl electrodes, before and up to 90 minutes following SAH, after which serum Mg²⁺ levels were again measured. Next, animals were transferred to the MR imaging magnet for diffusion-weighted (DW) MR imaging. Depolarization times per electrode were averaged to determine a mean depolarization time per animal.

No depolarizations occurred in sham-operated animals. Ischemic depolarizations occurred at all electrodes in all animals after SAH. Only two animals displayed a single spreading depression-like depolarization. The mean duration of the ID time was 41 ± 25 minutes in the saline-treated controls and 31 ± 30 minutes in the Mg²⁺-treated animals (difference 10 minutes; p = 0.31). Apparent diffusion coefficient (ADC) maps of tissue H₂O, obtained using DW images approximately 2.5 hours after SAH induction, demonstrated hypointensities in both hemispheres, but predominantly in the ipsilateral cortex. No ADC abnormalities were found in sham-operated animals. The mean lesion volume, as defined on the basis of a significant ADC reduction, was 0.32 ± 0.42 ml in saline-treated controls and 0.11 ± 0.06 ml in Mg²⁺-treated animals (difference 0.21 ml; p = 0.045). Serum Mg²⁺ levels were significantly elevated in the Mg²⁺-treated group.

Conclusions. On the basis of their data, the authors suggest that CSDs play a minor role, if any, in the acute pathophysiology of SAH. Administration of Mg²⁺ reduces the cerebral lesion volume that is present during the acute period after SAH. The neuroprotective value of Mg²⁺ after SAH may, in part, be explained by a reduction in the duration of the ID of brain cells.
Increased incidence and impact of nonconvulsive and convulsive seizures after traumatic brain injury as detected by continuous electroencephalographic monitoring

Paul M. Vespa, M.D., Marc R. Nuwer, M.D., Ph.D., Valeriy Nenov, Ph.D., Elisabeth Ronne-Engstrom, M.D., Ph.D., David A. Hovda, Ph.D., Marvin Berge-Steiner, M.D., Daniel F. Kelly, M.D., Neil A. Martin, M.D., and Donald P. Becker, M.D.

Division of Neurosurgery and Department of Neurology, University of California at Los Angeles School of Medicine, Los Angeles, California; and Department of Neurosurgery, Uppsala University, Uppsala, Sweden

Object. The early pathophysiological features of traumatic brain injury observed in the intensive care unit (ICU) have been described in terms of altered cerebral blood flow, altered brain metabolism, and neurochemical excitotoxicity. Seizures occur in animal models of brain injury and in human brain injury. Previous studies of posttraumatic seizures in humans have been based principally on clinical observations without a systematic approach to electroencephalographic (EEG) recording of seizures. The purpose of this study was to determine prospectively the incidence of convulsive and nonconvulsive seizures by using continuous EEG monitoring in patients in the ICU during the initial 14 days post-injury.

Methods. Ninety-four patients with moderate-to-severe brain injuries underwent continuous EEG monitoring beginning at admission to the ICU (mean delay 9.6 ± 5.4 hours) and extending up to 14 days postinjury. Convulsive and nonconvulsive seizures occurred in 21 (22%) of the 94 patients, with six of them displaying status epilepticus. In more than half of the patients (52%) the seizures were nonconvulsive and were diagnosed on the basis of EEG studies alone. All six patients with status epilepticus died, compared with a mortality rate of 24% (18 of 73) in the nonseizure group (p < 0.001). The patients with status epilepticus had a shorter mean length of stay (9.14 ± 5.9 days compared with 14 ± 9 days [t-test, p < 0.03]). Seizures occurred despite initiation of prophylactic phenytoin on admission to the emergency room, with maintenance at mean levels of 16.6 ± 2.8 mg/dl. No differences in key prognostic factors (such as the Glasgow Coma Scale score, early hypoxemia, early hypotension, or 1-month Glasgow Outcome Scale score) were found between the patients with seizures and those without.

Conclusions. Seizures occur in more than one in five patients during the 1st week after moderate-to-severe brain injury and may play a role in the pathobiological conditions associated with brain injury.
Increased incidence and impact of nonconvulsive and convulsive seizures after traumatic brain injury as detected by continuous EEG monitoring

- **Purpose:** to determine prospectively the incidence of convulsive and nonconvulsive seizures using continuous EEG monitoring in patients in the ICU during the initial 14 days post-injury.
- **94 patients with moderate-to-severe brain injuries underwent continuous EEG monitoring beginning at admission to the ICU (mean delay 9.6±/−5.4 hours) and extending up to 14 days postinjury.

Vespa PM et al *J Neurosurg* 1999 Nov;91(5):750-60
Increased incidence and impact of nonconvulsive and convulsive seizures after traumatic brain injury as detected by continuous EEG monitoring

- Convulsive and nonconvulsive seizures occurred in 21 (22%) of the 94 patients, with six of them displaying SE.
- In more than half of the patients (52%) the seizures were nonconvulsive and were diagnosed on the basis of EEG studies alone.
Increased incidence and impact of nonconvulsive and convulsive seizures after traumatic brain injury as detected by continuous EEG monitoring

- All six patients with SE died, compared with a mortality rate of 24% (18 of 73) in the nonseizure group (p<0.001).
- The patients with SE had a shorter mean length of stay
  - 9.14±5.9 d compared with 14±9 d, p<0.031.
- Seizures occurred despite initiation of prophylactic PHT on admission to the ED, with maintenance at mean levels of 16.6±2.8 mg/dl.
Increased incidence and impact of nonconvulsive and convulsive seizures after traumatic brain injury as detected by continuous EEG monitoring

- No differences in key prognostic factors (GCS score, early hypoxemia, early hypotension, or 1-month GOS) between patients with seizures and those without.
- Conclusions: Seizures occur in more than one in five patients during the 1st week after moderate-to-severe brain injury and may play a role in the pathobiological conditions associated with brain injury.
The BIS™ monitor provides a direct measure of the effects of sedatives on the brain.

In the Critical Care setting, the BIS monitor is commonly used to allow objective assessment of sedation during:

- Mechanical ventilation
- Bedside procedures
- Barbiturate coma
- Neuromuscular blockade

The BIS is most useful for patients who are chemically paralyzed and/or moderately to deeply sedated. Muscle activity may interfere with reliable BIS performance.

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**Considerations for Use in the ICU**

- Reliance on the BIS alone for sedative management is not recommended.
- Clinical judgment should always be used when interpreting the BIS in conjunction with other available clinical signs.
- BIS readings should be interpreted over time and in response to stimulation, and in the context of patient status and treatment plan.
- Movement may occur with low BIS values.
- Movement (EMG) may indicate inadequate analgesic level.
- Artifacts and poor signal quality may lead to unreliable BIS values. Potential artifacts may be caused by poor skin contact, muscle activity or rigidity, head and body motion, sustained eye movements, improper sensor placement or skin preparation, and unusual or excessive interference.
- BIS values should be interpreted cautiously in patients with known neurological disorders, in those taking psychoactive medications and in children less than 1 year old.
- Natural sleep cycles may affect the hypnotic level.
Detection of brain death onset using the bispectral index in severely comatose patients

• (this is a bad idea)

Is the bispectral index appropriate for monitoring the sedation level of mechanically ventilated SICU patients?

• Design and setting: Prospective convenience sample in a 12-bed anesthesiological-surgical ICU of a university hospital.

• Patients: 19 consecutive patients without any central neurological diseases requiring mechanical ventilation for more than 24 h.

Is the bispectral index appropriate for monitoring the sedation level of mechanically ventilated SICU patients?

- Measurements: BIS version 3.12 and clinical depth of sedation assessed by the
  - modified Observers’s Assessment of Alertness/ Sedation Scale,
  - modified Glasgow Coma Scale,
  - modified Ramsay Scale,
  - Cook Scale, and
  - Sedation-Agitation Scale

- Measured twice daily while patients were intubated and once daily after extubation until discharged from ICU.
BIS patients

Filled circles = ketamine used for sedation

'BIS patients'
'non-BIS' patients
Is the bispectral index appropriate for monitoring the sedation level of mechanically ventilated SICU patients?

- There was a moderate correlation between BIS and each sedation score in 11 patients (58%, “BIS patients”) and no correlation in 8 patients (42%, “non-BIS patients”).
- Found no parameters distinguishing between these two groups.
- On average eight measurements were necessary to establish a statistical correlation.
Is the bispectral index appropriate for monitoring the sedation level of mechanically ventilated SICU patients?

- **Conclusions:** BIS is correlated only in some ICU patients with the clinical assessment of their sedation level as based on various scores.
- At deeper sedation levels the interindividual differences increase.
- There were no criteria found to distinguish patients with and without correlation.
- This suggests that the BIS is not suitable for monitoring the sedation in a heterogeneous group of surgical ICU patients.
Sedation modulates recognition of novel stimuli and adaptation to regular stimuli in critically ill adults

- Studied 22 endotracheally intubated, mechanically ventilated patients chemically sedated with narcotics and a benzodiazepine (n=12), or narcotics and propofol (n=10) using sedation protocols.
- Evaluated the level of sedation using an automated auditory or visual P300 device.

Clifford and Buchman *Crit Care Med* 2002 30:609-616
Solid=freq stim
Dashed=rare stim
auditory

fentanyl, > 200 mcg/hr
propofol, < 25 mg/hr

visual

fentanyl, > 200 mcg/hr
propofol, > 25 mg/hr

milli-seconds
Continuous measurement of cerebral blood flow velocity using transcranial Doppler reveals significant moment-to-moment variability of data in healthy volunteers and in patients with subarachnoid hemorrhage

Continuous TCD

- Prospective study in 10 normals and 8 SAH patients
- There was significant moment-to-moment variability in both volunteers (-31% to 58%) and in patients (-38% to 78%).
- There was a greater number of observations exceeding 10% moment-to-moment variability in the patient group with regard to systolic and diastolic velocities compared with volunteers (8% vs. 2%, p < .001).
Continuous TCD measurement of mean MCA flow velocity in a patient after SAH
ICP and CPP monitoring

- ICP monitoring techniques
  - ventriculostomy
  - epidural transducer
  - subarachnoid bolt
  - parenchymal monitors
    - fiberoptic (Camino)
    - strain gauge (Codman)

- CPP calculation
  - position of arterial transducer
Cerebral perfusion pressure

- CPP=MAP-ICP
  - recommendations for CPP are extrapolated from effects of changing CPP in normals, who have intact autoregulation
- ‘CPP management’ stresses primacy of CPP over the actual ICP value
- the ‘Lund protocol’ is diametrically opposed, lowering MAP to decrease edema production
- both claim improved outcome compared to historical controls
Rosner view of cerebral blood flow
Rosner proposes that decreasing vessel caliber will lower intracranial blood volume and hence ICP without compromising perfusion
Improved outcome after severe head injury with a new therapy based on principles for brain volume regulation and preserved microcirculation

Christer Eker, MD; Bogi Åsgeirsson, MD, PhD; Per-Olof Grände, MD, PhD; Wilhelm Schalén, MD, PhD; Carl-Henrik Nordström, MD, PhD

Objective: To assess the new “Lund therapy” of posttraumatic brain edema, based on principles for brain-volume regulation and improved microcirculation.

Design: A prospective, nonrandomized outcome study over a 5-yr period on severely head-injured patients with increased intracranial pressure, comparing the results with a historical control group with the same selection criteria for patients who were treated according to conventional principles.

Setting: General intensive care unit of a university hospital.

Patients: Fifty-three consecutive head-injured patients with a Glasgow Coma Score of <8, and with increased intracranial pressure (>25 mm Hg), despite conventional treatment.

Interventions: Interstitial fluid resorption was obtained by lowering intracapillary hydrostatic pressure, by preserving normal colloid osmotic pressure, and by maintaining a normovolemic (normal albumin/serum and hemoglobin/serum), not overtransfused patient. Intracapillary pressure was reduced by the combination of precapillary vasoconstriction (low-dose thiopental, dihydroergotamine) and reduction of mean arterial pressure, the latter attained with a β₁-antagonist (metoprolol 0.2 to 0.3 mg/kg/24 hrs iv) and an α₂-agonist (clonidine 0.4 to 0.8 μg/kg x 4 to 6 iv). Clonidine, in combination with normovolemia, also improves microcirculation by reducing catecholamines in plasma. Intracranial blood volume was reduced by arterial (low-dose thiopental sodium and dihydroergotamine) and large-vein (dihydroergotamine) vasoconstriction. The start dose of dihydroergotamine (maximum 0.9 μg/kg/hr) was successively reduced toward discontinuation within 4 to 5 days.

Measurements and Main Results: There were 8% of patients who died and the neurologic conditions of 13% remained severely damaged, compared with 47% and 11%, respectively, for the control group.

Conclusions: The low mortality compared with previous outcome studies strongly indicates that this therapy improves outcome for severe head injuries. However, a randomized, controlled study is needed to reach general acceptance of this new therapy. (Crit Care Med 1996; 26:1881-1886)

Key Words: α₂-agonist; β₁-antagonist; blood-brain barrier; cerebral edema; cerebral microcirculation; cerebral perfusion pressure; cerebral vasoconstriction; head injury; intracranial pressure; outcome
Cerebral vasomotor reactivity testing in head injury: the link between pressure and flow

E W Lang, J Lagopoulos, J Griffith, K Yip, A Yam, Y Mudaliar, H M Mehdorn, N W C Dorsch

Background: It has been suggested that a moving correlation index between mean arterial blood pressure and intracranial pressure, called PRx, can be used to monitor and quantify cerebral vasomotor reactivity in patients with head injury.

Objectives: To validate this index and study its relation with cerebral blood flow velocity and cerebral autoregulation; and to identify variables associated with impairment or preservation of cerebral vasomotor reactivity.

Methods: The PRx was validated in a prospective study of 40 head injured patients. A PRx value of less than 0.3 indicates intact cerebral vasomotor reactivity, and a value of more than 0.3, impaired reactivity. Arterial blood pressure, intracranial pressure, mean cerebral perfusion pressure, and cerebral blood flow velocity, measured bilaterally with transcranial Doppler ultrasound, were recorded. Dynamic cerebrovascular autoregulation was measured using a moving correlation coefficient between arterial blood pressure and cerebral blood flow velocity, the Mx, for each cerebral hemisphere. All variables were compared in patients with intact and impaired cerebral vasomotor reactivity.

Results: No correlation between arterial blood pressure or cerebral perfusion pressure and cerebral blood flow velocity was seen in 19 patients with intact cerebral vasomotor reactivity. In contrast, the correlation between these variables was significant in 21 patients with impaired cerebral vasomotor reactivity, whose cerebral autoregulation was reduced. There was no correlation with intracranial pressure, arterial blood pressure, cerebral perfusion pressure, or interhemispheric cerebral autoregulation differences, but the values for these indices were largely within normal limits.

Conclusions: The PRx is valid for monitoring and quantifying cerebral vasomotor reactivity in patients with head injury. This intracranial pressure based index reflects changes in cerebral blood flow and cerebral autoregulatory capacity, suggesting a close link between blood flow and intracranial pressure in head injured patients. This explains why increases in arterial blood pressure and cerebral perfusion pressure may be useful for reducing intracranial pressure in selected head injured patients (those with intact cerebral vasomotor reactivity).
Intact autoregulation

A  Pressure passive dilatation  |  Zone of autoregulation  
  • Vasoconstriction  |  Pressure passive dilatation

Cerebral blood volume compartment

Cerebral blood flow (ml/100 g/min)

Arterial blood pressure (mm Hg)
Example of intact autoregulation
Defective autoregulation
Example of defective autoregulation
Over half of head injured patients have impaired autoregulation

<table>
<thead>
<tr>
<th>Variable</th>
<th>Impaired CR (n=21)</th>
<th>Intact CR (n=19)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Range</td>
</tr>
<tr>
<td>ABP</td>
<td>87 (22)</td>
<td>60 to 160</td>
</tr>
<tr>
<td>CPP</td>
<td>72 (21)</td>
<td>38 to 143</td>
</tr>
<tr>
<td>ICP</td>
<td>16 (7)</td>
<td>5 to 25</td>
</tr>
<tr>
<td>CBFV</td>
<td>64 (29)</td>
<td>22 to 129</td>
</tr>
<tr>
<td>PRx</td>
<td>0.55 (0.17)</td>
<td>0.31 to 0.9</td>
</tr>
<tr>
<td>Mx*</td>
<td>0.43 (0.23)</td>
<td>-0.05 to 0.93</td>
</tr>
<tr>
<td>ΔI/r Mx</td>
<td>0.16 (0.25)</td>
<td>0.07 to 1.01</td>
</tr>
</tbody>
</table>

*Significant difference between impaired and intact CR (p<0.001).

ABP, mean arterial blood pressure (mm Hg); CBFV, cerebral blood flow velocity (cm/s); CPP, mean cerebral perfusion pressure (mm Hg); CR, cerebrovascular reactivity; ICP, intracranial pressure (mm Hg); I/r, left/right; Mx, moving correlation coefficient between arterial blood pressure or cerebral perfusion pressure and cerebral blood flow velocity; Prx, index comparing arterial blood pressure and intracranial pressure.
Efficacy of hyperventilation, blood pressure elevation, and metabolic suppression therapy in controlling intracranial pressure after head injury

Matthias Oertel, M.D., Daniel F. Kelly, M.D., Jae Hong Lee, M.D., M.P.H., David L. McArthur, Ph.D., M.P.H., Thomas C. Glenn, Ph.D., Paul Vespa, M.D., W. John Boscardin, Ph.D., David A. Hovda, Ph.D., and Neil A. Martin, M.D.
### TABLE 1

**Results of global vasoreactivity tests**

<table>
<thead>
<tr>
<th>Subject of Test</th>
<th>No. of Studies</th>
<th>Parameter</th>
<th>Mean ± SD*</th>
<th>Abnormal (%)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>baseline CBF</td>
<td>55</td>
<td>global CBF (ml/100 g/min)</td>
<td>39 ± 13†</td>
<td></td>
</tr>
<tr>
<td>hyperventilation</td>
<td>57</td>
<td>CO₂ reactivity (mm Hg)</td>
<td>3.2 ± 1.5</td>
<td>12.5</td>
</tr>
<tr>
<td>induced hypertension</td>
<td>55</td>
<td>pressure autoregulation (%)</td>
<td>64 ± 70</td>
<td>55.4</td>
</tr>
<tr>
<td>metabolic suppression</td>
<td>43</td>
<td>metabolic reactivity (%)</td>
<td>16 ± 11</td>
<td>88.4</td>
</tr>
</tbody>
</table>

* SD = standard deviation.
† Percent of studies with abnormal vasoreactivity. Normal CO₂ reactivity is defined as 3.7 ± 0.5%/mm Hg PaCO₂; normal pressure autoregulation is defined as a PAI greater than 70%; and normal metabolic suppression is defined as a decrease in CO₂-corrected V_{MCA} of 30% or more after the patient has undergone administration of high-dose propofol, according to Lee, et al.

‡ Of the baseline ¹³³Xe CBF studies, data from one study (1.8%) demonstrated global ischemia, which was defined as global CBF lower than 20 ml/100 g/min, and data from 10 studies (18%) showed absolute hyperemia, which was defined as global CBF higher than 55 ml/100 g/min, according to Kelly, et al., 1996, and Obrist, et al.
Oxygenation monitoring

- jugular bulb catheter
  - jugular venous blood oxygen saturation
    - A-V differences in saturation, content, lactate
- direct cortical oxygen sensors
  - Licox
  - Neurotrend (going out of business)
Oxygenation monitoring

• jugular bulb catheter
  – jugular venous blood oxygen saturation
    • A-V differences in saturation, content, lactate
  – provides an average of the venous oxygen in the vascular territory drained
  – cannot detect local problems with oxygenation

• direct cortical oxygen sensors (Licox; Neurotrend no longer to be marketed)
  – data from only a few mm$^3$

• PET
Cerebral tissue PO$_2$ and SjvO$_2$ changes during moderate hyperventilation in patients with severe traumatic brain injury

ROBERTO IMBERTI, M.D., GUIDO BELLINZONA, M.D., AND MARTIN LANGER, M.D.

Servizio di Anesthesia e Rianimazione II, IRCCS Policlinico San Matteo, Pavia, Italy

Object. The aim of this study was to investigate the effects of moderate hyperventilation on intracranial pressure (ICP), jugular venous oxygen saturation ([SjvO$_2$], an index of global cerebral perfusion), and brain tissue PO$_2$ (an index of local cerebral perfusion).

Methods. Ninety-four tests consisting of 20-minute periods of moderate hyperventilation (27–32 mm Hg) were performed on different days in 36 patients with severe traumatic brain injury (Glasgow Coma Scale score ≤ 8). Moderate hyperventilation resulted in a significant reduction in average ICP, but in seven tests performed in five patients it was ineffective. The response of SjvO$_2$ and brain tissue PO$_2$ to CO$_2$ changes was widely variable and unpredictable. After 20 minutes of moderate hyperventilation in most tests (79.8%), both SjvO$_2$ and brain tissue PO$_2$ values remained above the lower limits of normality (50% and 10 mm Hg, respectively). In contrast, in 15 tests performed in six patients (16.6% of the studied population) brain tissue PO$_2$ decreased below 10 mm Hg although the corresponding SjvO$_2$ values were greater than 50%. The reduction of brain tissue PO$_2$ below 10 mm Hg was favored by the low prehyperventilation values (10 tests), higher CO$_2$ reactivity, and, possibly, by lower prehyperventilation values of cerebral perfusion pressure. In five of those 15 tests, the prehyperventilation values of SjvO$_2$ were greater than 70%, a condition of relative hyperemia. The SjvO$_2$ decreased below 50% in four tests; the corresponding brain tissue PO$_2$ values were less than 10 mm Hg in three of those tests, whereas in the fourth, the jugular venous O$_2$ desaturation was not detected by brain tissue PO$_2$.

The analysis of the simultaneous relative changes (prehyperventilation − posthyperventilation) of SjvO$_2$ and brain tissue PO$_2$ showed that in most tests (75.5%) there was a reduction of both SjvO$_2$ and brain tissue PO$_2$. In two tests moderate hyperventilation resulted in an increase of both SjvO$_2$ and brain tissue PO$_2$. In the remaining 17 tests a redistribution of the cerebral blood flow was observed, leading to changes in SjvO$_2$ and brain tissue PO$_2$ in opposite directions.

Conclusions. Hyperventilation, even if moderate, can frequently result in harmful local reductions of cerebral perfusion that cannot be detected by assessing SjvO$_2$. Therefore, hyperventilation should be used with caution and should not be considered safe. This study confirms that SjvO$_2$ and brain tissue PO$_2$ are two parameters that provide complementary information on brain oxygenation that is useful to reduce the risk of secondary damage. Changes in SjvO$_2$ and brain tissue PO$_2$ in opposite directions indicate that data obtained from brain tissue PO$_2$ monitoring cannot be extrapolated to evaluate the global cerebral perfusion.
Moderate hyperventilation lowered ICP and improved CPP...
...but produced ‘critical’ tissue hypoxia despite preserved SjO$_2$
Regional cerebrovascular and metabolic effects of hyperventilation after severe traumatic brain injury

MICHAEL N. DIRINGER, M.D., TOM O. VIDEEN, PH.D., KENT YUNDT, M.D., ALLYSON R. ZAZULIA, M.D., VENKATESH AIYAGARI, M.D., RALPH G. DACEY, JR., M.D., ROBERT L. GRUBB, JR., M.D., AND WILLIAM J. POWERS, M.D.

Departments of Neurology and Neurological Surgery, and Radiology, Neurology/Neurosurgery Intensive Care Unit, Washington University School of Medicine, St. Louis, Missouri

Object. Recently, concern has been raised that hyperventilation following severe traumatic brain injury (TBI) could lead to cerebral ischemia. In acute ischemic stroke, in which the baseline metabolic rate is normal, reduction in cerebral blood flow (CBF) below a threshold of 18 to 20 ml/100 g/min is associated with energy failure. In severe TBI, however, the metabolic rate of cerebral oxygen (CMRO₂) is low. The authors previously reported that moderate hyperventilation lowered global hemispheric CBF to 25 ml/100 g/min but did not alter CMRO₂. In the present study they sought to determine if hyperventilation lowers CBF below the ischemic threshold of 18 to 20 ml/100 g/min in any brain region and if those reductions cause energy failure (defined as a fall in CMRO₂).

Methods. Two groups of patients were studied. The moderate hyperventilation group (nine patients) underwent hyperventilation to PaCO₂ of 30 ± 2 mm Hg early after TBI, regardless of intracranial pressure (ICP). The severe hyperventilation group (four patients) underwent hyperventilation to PaCO₂ of 25 ± 2 mm Hg 1 to 5 days postinjury while ICP was elevated (20–30 mm Hg). The ICP, mean arterial blood pressure, and jugular venous O₂ content were monitored, and cerebral perfusion pressure was maintained at 70 mm Hg or higher by using vasopressors when needed. All data are given as the mean ± standard deviation unless specified otherwise. The moderate hyperventilation group was studied 11.2 ± 1.6 hours (range 8–14 hours) postinjury, the admission Glasgow Coma Scale (GCS) score was 5.6 ± 1.8, the mean age was 27 ± 9 years, and eight of the nine patients were men. In the severe hyperventilation group, the admission GCS score was 4.3 ± 1.5, the mean age was 31 ± 6 years, and all patients were men. Positron emission tomography measurements of regional CBF, cerebral blood volume, CMRO₂, and oxygen extraction fraction (OEF) were obtained before and during hyperventilation. In all 13 patients an automated search routine was used to identify 2.1-cm spherical nonoverlapping regions with CBF values below thresholds of 20, 15, and 10 ml/100 g/min during hyperventilation, and the change in CMRO₂ in those regions was determined. In the regions in which CBF was less than 20 ml/100 g/min during hyperventilation, it fell from 26 ± 6.2 to 13.7 ± 1 ml/100 g/min (p < 0.0001), OEF rose from 0.31 to 0.59 (p < 0.0001), and CMRO₂ was unchanged (1.12 ± 0.29 compared with 1.14 ± 0.03 ml/100 g/min; p = 0.8). In the regions in which CBF was less than 15 ml/100 g/min during hyperventilation, it fell from 23.3 ± 6.6 to 11.1 ± 1.2 ml/100 g/min (p < 0.0001), OEF rose from 0.31 to 0.63 (p < 0.0001), and CMRO₂ was unchanged (0.98 ± 0.19 compared with 0.97 ± 0.23 ml/100 g/min; p = 0.92). In the regions in which CBF was less than 10 ml/100 g/min during hyperventilation, it fell from 18.2 ± 4.5 to 8.1 ± 0 ml/100 g/min (p < 0.0001), OEF rose from 0.3 to 0.71 (p < 0.0001), and CMRO₂ was unchanged (0.78 ± 0.26 compared with 0.81 ± 0.32 ml/100 g/min; p = 0.61).

Conclusions. After severe TBI, brief hyperventilation produced large reductions in CBF but not energy failure, even in regions in which CBF fell below the threshold for energy failure defined in acute ischemia. Oxygen metabolism was preserved due to the low baseline metabolic rate and compensatory increases in OEF; thus, these reductions in CBF are unlikely to cause further brain injury.
...but HV didn’t produce energy failure

**Fig. 2.** Bar graphs showing regional cerebrovascular hemodynamics in patients with TBI before (*black bars*) and during hyper-ventilation (*stippled bars*). *p < 0.0001.*
Or does it?

Effect of hyperventilation on cerebral blood flow in traumatic head injury: Clinical relevance and monitoring correlates*

Jonathan P. Coles, FRCA; Pawan S. Minhas, FRCS; Tim D. Fryer, PhD; Peter Smielewski, PhD; Franklin Aigbirhiio, PhD; Tim Donovan, BSc; Stephen P. M. J. Downey, MSc; Guy Williams, PhD; Dot Chatfield, BSc; Julian C. Matthews, PhD; Arun K. Gupta, FRCA; T. Adrian Carpenter, PhD; John C. Clark, DSc; John D. Pickard, FRCS; David K. Menon, PhD

Objective: To investigate the effect of hyperventilation on cerebral blood flow in traumatic brain injury.

Design: A prospective interventional study.

Setting: A specialist neurocritical care unit.

Patients: Fourteen healthy volunteers and 33 patients within 7 days of closed head injury.

Interventions: All subjects underwent positron emission tomography imaging of cerebral blood flow. In patients, Paco2 was reduced from 36 ± 1 to 29 ± 1 torr (4.8 ± 0.1 to 3.9 ± 0.1 kPa) and measurements repeated. Jugular venous saturation (Sjvo2) and arteriovenous oxygen content differences (AVDO2) were monitored in 25 patients and values related to positron emission tomography variables.

Measurements and Main Results: The volumes of critically hypoperfused and hyperperfused brain (HypoBV and HyperBV, in milliliters) were calculated based on thresholds of 10 and 55 mL-100g−1·min−1, respectively. Whereas baseline HypoBV was significantly higher in patients (p < .05), baseline HyperBV was similar to values in healthy volunteers. Hyperventilation resulted in increases in cerebral perfusion pressure (p < .0001) and reductions in intracranial pressure (p < .001), whereas Sjvo2 (>50%) and AVDO2 (<9 mL/mL) did not exceed global ischemic thresholds. However, despite these beneficial effects, hyperventilation shifted the cerebral blood flow distribution curve toward the hypoperfused range, with a decrease in global cerebral blood flow (31 ± 1 to 23 ± 1 mL-100g−1·min−1; p < .0001) and an increase in HypoBV (22 [1–141] to 51 [2–420] mL; p < .0001). Hyperventilation-induced increases in HypoBV were apparently nonlinear, with a threshold value between 34 and 38 torr (4.5–5 kPa).

Conclusions: Hyperventilation increases the volume of severely hypoperfused tissue within the injured brain, despite improvements in cerebral perfusion pressure and intracranial pressure. Significant hyperperfusion is uncommon, even at a time when conventional clinical management includes a role for modest hyperventilation. These reductions in regional cerebral perfusion are not associated with ischemia, as defined by global monitors of oxygenation, but may represent regions of potentially ischemic brain tissue. (Crit Care Med 2002; 30:1950–1959)

Key Words: head injury; positron emission tomography; cerebral blood flow; ischemia; cerebral oxygenation; hyperventilation
Baseline
PaCO₂
35 mmHg

HV
PaCO₂
26 mmHg
Fig. 2. (A) A schematic diagram of the Licox polarographic oxygenation probe. The numbered components of the diagram are: (1) polyethylene tube diffusion membrane; (2) polarographic gold cathode; (3) polarographic silver anode; (4) cell filled with electrolyte; and (5) cerebral tissue. (B) A schematic diagram of the Licox probe illustrating placement via a cranial bolt into the cerebral tissues. Placement is similar to ICP monitoring and is often used through the same bolt.
Reduced mortality rate in patients with severe traumatic brain injury treated with brain tissue oxygen monitoring

Michael F. Stiefel, M.D., Ph.D., Alejandro Spiotta, M.D., Vincent H. Gracias, M.D., Alicia M. Garuffe, M.S.N., Oscar Guillamondegui, M.D., Eileen Maloney-Wilensky, M.S.N., Stephanie Bloom, M.S.N., M. Sean Grady, M.D., and Peter D. LeRoux, M.D.

Object. An intracranial pressure (ICP) monitor, from which cerebral perfusion pressure (CPP) is estimated, is recommended in the care of severe traumatic brain injury (TBI). Nevertheless, optimal ICP and CPP management may not always prevent cerebral ischemia, which adversely influences patient outcome. The authors therefore determined whether the addition of a brain tissue oxygen tension (PO$_2$) monitor in the treatment of TBI was associated with an improved patient outcome.

Methods. Patients with severe TBI (Glasgow Coma Scale [GCS] score < 8) who had been admitted to a Level I trauma center were evaluated as part of a prospective observational database. Patients treated with ICP and brain tissue PO$_2$ monitoring were compared with historical controls matched for age, pathological features, admission GCS score, and Injury Severity Score who had undergone ICP monitoring alone. Therapy in both patient groups was aimed at maintaining an ICP less than 20 mm Hg and a CPP greater than 60 mm Hg. Among patients whose brain tissue PO$_2$ was monitored, oxygenation was maintained at levels greater than 25 mm Hg. Twenty-five patients with a mean age of 44 ± 14 years were treated using an ICP monitor alone. Twenty-eight patients with a mean age of 38 ± 18 years underwent brain tissue PO$_2$-directed care. The mean daily ICP and CPP levels were similar in each group. The mortality rate in patients treated using conventional ICP and CPP management was 44%. Patients who also underwent brain tissue PO$_2$ monitoring had a significantly reduced mortality rate of 25% (p < 0.05).

Conclusions. The use of both ICP and brain tissue PO$_2$ monitors and therapy directed at brain tissue PO$_2$ is associated with reduced patient death following severe TBI.

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DID YOU INTEND THE PRESENTATION TO BE INCOMPREHENSIBLE, OR DO YOU HAVE SOME SORT OF RARE "POWERPOINT" DISABILITY?