To the Editor.—We read with interest the case report by Albertin et al. who reported a bilateral subdural hematoma (SDH) in a parturient after epidural anesthesia. In a previous report, we reviewed 47 cases of cerebral hemorrhage after spinal (26 cases) and/or epidural (21 cases) anesthesia (table 1). Almost half of the patients developed bilateral subdural hematoma after dural puncture by an epidural needle. In contrast, subdural hematoma after spinal anesthesia occurred most frequently on the left side of the brain. It is possible that the increased incidence of bilateral subdural hematoma after epidural (11 of 21) versus spinal anesthesia (4 of 26) can be attributed to the greater loss of cerebrospinal fluid volume after inadvertent dural puncture with a large-bore epidural needle. Cerebrospinal fluid leak through a large dural hole might lead to marked cerebral hypotension associated with excessive traction on the bridging veins on both sides of the brain, resulting in bilateral subdural hematoma formation.

Table 1. Cerebral Subdural Hematoma after Spinal versus Epidural Anesthesia

<table>
<thead>
<tr>
<th></th>
<th>Spinal Group</th>
<th>Epidural Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>26</td>
<td>21</td>
</tr>
<tr>
<td>Bilateral subdural hematoma</td>
<td>4</td>
<td>11</td>
</tr>
<tr>
<td>Left subdural hematoma</td>
<td>14</td>
<td>6</td>
</tr>
<tr>
<td>Right subdural hematoma</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>Intracerebral hematoma</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Pregnant patients</td>
<td>6</td>
<td>17</td>
</tr>
<tr>
<td>Blood patch</td>
<td>—</td>
<td>8</td>
</tr>
</tbody>
</table>

Further analysis of the 47 cases of SDH showed that half of the published cases were in obstetric patients (22 of 47 cases); 6 cases were reported after spinal anesthesia, and 17 cases followed epidural...
In Reply.—We appreciate the interest of Drs. Zeidan and Baraka in our report and enjoyed reading their articles on the subject.1,2 Indeed, we previously described the formation of bilateral intracranial subdural hematomas in a parturient who received epidural analgesia and who presumably encountered an occult dural tear.3 The data presented by Plourde et al.2 on the brain’s response to different forms of auditory stimuli during sedation and anesthesia. This holistic approach is the only rational option to understand such phenomena as learning during anesthesia and perioperative dreaming.

One of the most interesting findings from this study is the extreme diminution of cortical response to auditory stimuli during the first episode of “sedation” before anesthesia. The cortical response to auditory stimuli during the period of “sedation” before anesthesia was much less than at baseline or at an equivalent blood level of sedation during recovery. The authors offer the explanation of acute tolerance to propofol in the interpretation of these results, which I would suggest is unnecessary.

We too have noted an increase in brain activation during propofol sedation, most evident in the statistical maps used to define brain activity in neuroimaging studies. During propofol or thiopental sedation, a very visible increase in statistical activation to auditory stimulation is present, although in actuality a small decrease in regional cerebral blood flow occurs.2 Plourde et al. also document a similar discrepancy between blood oxygenation level–dependent signal magnitudes, which remain similar in many regions to baseline, and the increase in statistical significance, from roughly f = 14 at baseline to 19 in recovery, clearly visible in their brain map figures. There is no doubt that the subjects in the study of Plourde et al. were awake and sedated during recovery, because they remembered some 60% of the stimuli presented to them. Therefore, the phenomenon of increased statistical significance seems to be a marker of the effect of a sedative drug on auditory stimulation in an awake cortex. The new, paradoxical finding in the study of Plourde et al., however, is that this situation is absent in the initial “sedation” condition, where both blood oxygenation level–dependent signal and significance are similar to the anesthesia condition. The only behavioral measure prospectively collected in this study is memory performance, and it too is noticeably absent in the initial “sedation” condition, being the same as during anesthesia. However, at recovery, the memory performance is at the expected 60% of control. As noted by Plourde et al., these sedative concentrations of propofol should produce performance of approximately 50% of baseline/control, as we have documented in our studies.3

The most parsimonious explanation of the paradoxical findings during the initial “sedation” condition is that the subjects were indeed asleep, whereas they were awake and truly sedated in the recovery condition. The contention that sedation was equivalent at the begin-

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References

(Accepted for publication August 17, 2006.)

To Sleep, Perchance to Decode?

To the Editor.—Plourde et al.3 recently reported the effects of propofol on the brain’s response to different forms of auditory stimuli during sedation and anesthesia. This holistic approach is the only rational option to understand such phenomena as learning during anesthesia and perioperative dreaming.

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CORRESPONDENCE

In Reply.—We thank Dr. Veselis for his insightful comments on our article.1 His first point is that the lack of specific auditory activation during the light sedation stage was most probably caused by physiologic sleep. The interactions we had with the subjects during the experiment and the informal debriefing between runs make us think this is unlikely: Subjects after the light sedation run seemed more “chatty” than sleepy. In addition, as we point out in the article, normal sleep does not abolish responses to complex sounds. Hence, even if the subjects had fallen asleep, this would not per se explain the lack of auditory responses. Despite these arguments and as indicated in our article, we agree that we have no way to unambiguously demonstrate that subjects were not sleeping during the light sedation recordings. In future experiments, we will have to monitor subjects’ level of arousal more closely, although even asking subjects to perform an active task might not be sufficient. Lack of response would not necessarily indicate sleep; it could also indicate attentional, motivational, motor, or memory alterations due to the anesthesia, for example. The most objective means is probably monitoring of an ongoing electroencephalograph, a complicated task in a magnetic resonance imaging suite.

Irrespective of the sleep issue, we still believe the evidence of increased neural excitability (or disinhibition) after anesthesia with propofol because the level of activation during recovery (propofol plasma concentration: 0.64 μg/ml) significantly exceeded baseline levels. The critical question is, Does the development of this hyperexcitability require previous exposure to a higher concentration of propofol? Veselis believes that the answer is no, based on a study2 that revealed a visible increase in statistic activation to auditory stimulation during sedation (not preceded by exposure to higher concentration) with propofol or thiopental. The statistical significance of that effect was not reported. We proposed acute tolerance as the most likely explanation for the increased excitability seen during recovery in our study because we have observed an analogous phenomenon with the 40-Hz auditory steady state response: The amplitude during recovery from propofol anesthesia significantly exceeded baseline amplitude.3 This phenomenon was not observed during propofol sedation not preceded by exposure to a higher concentration4 or during prolonged recordings in nonanesthetized subjects (unpublished observations, Gilles Plourde, M.D., M.Sc., Professor, Department of Anesthesia, Montreal Neurologic Hospital, Montreal, Quebec, Canada, 2002). We believe that this topic deserves further inquiry.

Dr. Veselis’ second point is that we were not able to demonstrate higher level processing during anesthesia because of insufficient power. We did discuss in the article the factors that could have led to a relative loss of signal during the anesthesia and hence affected our ability to pick up a signal. However, we disagree that the lack of response during anesthesia for higher level processing is explained on the basis of a lack of power:

First, we did have sufficient power to detect speech- and voice-related responses during baseline (and recovery). During anesthesia, we showed, using the same functional magnetic resonance imaging sampling protocol, that these responses were absent with mean signal amplitudes very close to zero and one-sample t tests yielding P > 0.2. Naturally, neither we nor anyone else can prove the null hypothesis of no activation, regardless of how large a sample is studied or how many data points are acquired. However, as we also point out in the article, decreased but still present brain activity in response to voice or speech during anesthesia would not likely be detected in this study.

Plourde et al. have presented a more refined evaluation of auditory processing during sedation and anesthesia than has heretofore been published. The interesting questions raised by these findings clearly elucidate paths for further study. However, the conclusions drawn from the current study are limited by lack of behavioral data and by issues of statistical significance. I would suggest that the evidence for acute tolerance to propofol is confounded by sleeping subjects. The issue of inhibition of higher order but not general auditory processes by propofol is still an open one. It seems likely that decoding of auditory stimuli during anesthesia is still functioning at some level, because numerous studies have demonstrated at least an implicit form of memory for these stimuli, albeit in much larger sample sizes (e.g., Deeprose et al.5).

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our conclusion is consistent with electrophysiologic data from higher order visual cortex in monkeys.

Second, and perhaps more important, we successfully demonstrated significant negative activation during anesthesia in the words-versus-scrambled words contrast. This suggests that the protocol had adequate sensitivity, because with the same number of trials, we picked up an atypical response difference between the two stimulus classes. If the lack of normal positive response to words or voice stimuli were simply due to low signal and/or high noise, we would not have detected the unusual negative response either.

Third, the reduced level of significance for the words versus scrambled words and vocal versus nonvocal stimuli was expected because we were comparing activations produced by different classes of stimuli (instead of a stimulus vs. silent baseline). This is therefore not an indication of inadequate signal acquisition.

We look forward to additional research on this topic from Dr. Veselis’ group and others, because much clearly remains to be learned from the study of neural responses during anesthesia.

Gilles Plourde, M.D., M.Sc.,* Pascal Belin, Ph.D., Robert J. Zatorre, Ph.D. “McGill University, Montreal, Quebec, Canada. gilles.plourde@staff.mcgill.ca

References


In Reply:—Reuttershan et al.1 reported on the finding of reduced endotoxin-induced acute lung injury in mice pretreated with isoflurane. In their investigation of the underlying cause of this phenomenon, the authors focused on neutrophil recruitment and chemokine concentrations. They found a reduction of neutrophil recruitment and CXCL1 and CXCL2/5 chemokine concentrations in the lungs of isoflurane-treated mice. Previous studies have not established a causal link between chemokine levels or the associated neutrophil accumulation and endotoxin-induced lung injury. There is, however, evidence that the cytokines tumor necrosis factor and interleukin 1 are involved in a reduction of alveolar ion and the associated fluid transport and hence pulmonary edema clearance.2,5 Particularly tumor necrosis factor has been strongly implicated in the pathogenesis of pulmonary edema, and its effect is mediated by nitric oxide.2,4 Tumor necrosis factor–induced interleukin 1–dependent formation of pulmonary edema might be one might occur without the other.2 For example, increased vascular permeability and PMN recruitment are two key factors in lung injury, there is evidence that regulation of both is different and one might occur without the other.2 For example, vascular protein leakage is dependent on inducible nitric oxide syn-

Role of a Reduction of Cytokine Levels in Isoflurane-mediated Protection from Endotoxin-induced Lung Injury

To the Editor:—Reutershan et al.1 reported on the finding of reduced endotoxin-induced acute lung injury in mice pretreated with isoflurane. In their investigation of the underlying cause of this phenomenon, the authors focused on neutrophil recruitment and chemokine concentrations. They found a reduction of neutrophil recruitment and CXCL1 and CXCL2/5 chemokine concentrations in the lungs of isoflurane-treated mice. Previous studies have not established a causal link between chemokine levels or the associated neutrophil accumulation and endotoxin-induced lung injury. There is, however, evidence that the cytokines tumor necrosis factor and interleukin 1 are involved in a reduction of alveolar ion and the associated fluid transport and hence pulmonary edema clearance.2,5 Particularly tumor necrosis factor has been strongly implicated in the pathogenesis of pulmonary edema, and its effect is mediated by nitric oxide.2,4 Tumor necrosis factor–induced nitric oxide leads thereby to a reduction of the activity of the alveolar epithelial sodium channel and the basolateral sodium potassium adenosine triphosphatase, which are essential for alveolar sodium and fluid transport.2 Isoflurane has been shown to reduce plasma interleukin 1β and tumor necrosis factor levels significantly in endotoxemic rats.5 The underlying cause for this effect has been investigated and found to be an inhibition of nuclear transcription factor KB.6 The reduction in chemokine levels and subsequently neutrophil accumulation may be only a reflection of this inhibition of nuclear transcription factor KB rather than causally related to lung injury reduction, because chemokine genes are part of the array of genes regulated by this transcriptional factor.7 Future studies on the protective effect of isoflurane against lung injury must correlate its effect on tumor necrosis factor and interleukin-1 levels with measures of pulmonary edema clearance.

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References


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Hypotension, Heart Rate Variability, and Altered Autonomic Function

To the Editor—The elegant studies by Hanss et al.1,2 demonstrate that alterations in heart rate variability (HRV) may predict hypotension after spinal anesthesia. The authors found a significant association between an increased ratio of low-frequency HRV (LF) to high-frequency HRV (HF) (i.e., LF/HF) and subsequent hypotension. A less predictive but still significant relation was found between decreased HF and subsequent hypotension.3

In their discussion, the authors briefly compare their results with our previous results4 investigating altered autonomic function and hypotension after induction of general anesthesia. Both of these studies demonstrated an association between decreased HF and subsequent hypotension. Although the stronger relation that we found with decreased HF and subsequent hypotension may simply reflect obvious differences in the study conditions (e.g., general anesthesia vs. spinal anesthesia, different patient populations), it might also relate to the different methods used to quantitate HF.

Heart rate variability measurements in the study by Hanss et al.1 are presented using normalized units, whereas the power in a specific frequency band (e.g., HF) is normalized by the total power in the HRV spectrum (total HRV) for that patient (i.e., HF in normalized units = HF [in absolute units]/[total HRV in absolute units]). A patient with decreased HF expressed in absolute units might have a normal value of HF when expressed in normalized units if the total HRV in his or her HRV power spectrum is also reduced (which is often the case in patients with autonomic dysfunction from multiple causes). For this reason, we used absolute measurements of HRV in our study. We found both a significant predictive value of decreased HF for postinduction hypotension and a strong correlation between HF and other measures of autonomic dysfunction. Historically, both absolute and normalized measurements of HRV have been used by different investigators.5 Normalized units have advantages when HF is used to examine “sympathovagal balance” (a major focus of the study of Hanss et al.), whereas absolute units have advantages when reflex integrity or “gain” (a major focus of our study) is examined.

Did the authors of the current study6 examine any possible relation between absolute measurements of HF and subsequent hypotension (or is such an analysis possible with their data)? The relation that they observed with postspinal hypotension and normalized measurements of HF might be even stronger using absolute measurements of HF. A stronger relation, if it exists, would argue that preexisting alterations in reflex integrity or gain might also play a role in hypotension after spinal anesthesia (similar to previous findings with general anesthesia6–8). Furthermore, if a significant predictive relation between absolute measurements of HF and postspinal hypotension does exist, fairly simple clinical tests of autonomic function (e.g., the change in heart rate with six vital capacity breaths, which is highly correlated with absolute measures of HF9) might also have significant predictive value.

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References


Accepted for publication August 18, 2006.
In Reply.—We thank Dr. Latson for appreciating our interesting comment on our study. As he stated, we demonstrated that the ratio of low frequency (LF) to high frequency (HF) of heart rate variability (HRV) predicts postspinal hypotension with high sensitivity and specificity.1,2 These findings were confirmed in the control group of another study.3 In addition, we demonstrated significant differences of normalized HF spectral power before subarachnoid block. These results may indicate that baseline HF is of predictive value as well, confirming the conclusions of Latson et al.4

We agree with the author that the type of HRV analysis may crucially influence the results. Latson et al. investigated absolute values, whereas we analyzed the different frequency bands of the power spectrum as normalized units.

Currently, HRV-derived data are processed and presented in different ways. First, absolute HRV values are frequently analyzed and, as the author stated, may have advantages in the analysis of autonomic reflex integrity. Nevertheless, total power of HRV and its different parts decrease with increasing age, underlying cardiovascular disease, and impaired autonomic regulation.5–7 Thus, an absolute HF of, for example, 1,000 ms²/Hz may be considered as below normal in young and healthy patients, normal in older healthy patients, and even above normal range in patients with coronary artery disease. Therefore, absolute values do not reflect the specific impact of the parasympathetic or the sympathetic activity of the autonomic regulation. Second, HRV data are normalized to total power. If the total power is high (e.g., 10,000 ms²/Hz), an HF of 1,000 ms²/Hz is only 10%, reflecting a low parasympathetic activity in the individual. If the total power is only 2,000 ms²/Hz, the same absolute HF value would reflect a considerably higher parasympathetic activity. The aim of our studies was to identify a general predictor of postspinal hypotension; therefore, we investigated the different frequencies in normalized units and cannot comment on absolute HF values in our patients. Absolute values were analyzed as well, but demonstrated only a trend toward higher HF in normotensive patients compared with hypotensive patients. Therefore, we cannot recommend the analysis of absolute values for prediction of postspinal hypotension based on our data.

The guidelines of the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology do not favor any one of the mathematical techniques, and each may have advantages and disadvantages.8

However, the ratio of LF to HF was demonstrated to be the most valuable parameter of HRV in terms of prediction of postspinal hypotension.1,3 This parameter reflects the balance of the autonomic nervous system and is independent of relative or absolute values because the ratio of absolute and normalized values is mathematically identical. Risk stratification based on the analysis of this autonomic balance was demonstrated to successfully guide prophylactic therapy.5

We conclude that in a homogenous group of patients, analysis of absolute values of HRV parameters is probably the best method to determine reflex integrity or “gain.” If the trial’s patient population demonstrates a large variation with respect to age, sex, and underlying diseases, relative values may be more appropriate. LF to HF ratio may be particularly useful for HRV analysis because it is independent of this difference.

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References

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Downloaded From: http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931044/ on 04/08/2017
In Reply:—We thank Dr. Lacassie for and appreciate his valuable comment on our study.¹ We agree with his statement that if prophylactic treatment of postspinal hypotension is based on measurement of heart rate variability (HRV), timing of the analysis is crucial. Measurement of HRV is performed to analyze autonomic nervous system regulation. It was clearly demonstrated by Chassard and Bruguerolle² as well as Takase et al.³ that autonomic nervous system activity demonstrates circadian rhythm. Therefore, timing of the measurement is important and should be comparable in all patients. In our previous studies, baseline analysis of HRV on the day of surgery directly before volume preload and regional block was demonstrated to identify patients at risk of hypotension in the course of subarachnoid block,⁴,⁵ whereas the analysis on the day before surgery did not correlate with postspinal hypotension.

Events of the current study were defined based on these findings. Baseline analysis identifying patients at risk was the analysis on the day of surgery before volume preload and anesthesia. Because cesarean deliveries were performed at 8:00 A.M., all analyses were performed at the same time. The measurement on the day before surgery was performed according to our previous studies and confirmed that there was no correlation between HRV parameters at that event and postspinal hypotension. We conclude that HRV changes during a 24-h period. Therefore, under study conditions, analyses should be performed at the same time to secure comparability of results, as was done in our study.

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In Reply:—The ProSeal™ laryngeal mask airway (PSLMA; Laryngeal Mask Company Limited, Mahe, Seychelles) is not designed as an intubation device. However, we may need to intubate the trachea with a PSLMA in place, using it as an intubation conduit with fiberoptic guidance. This situation may occur in an unanticipated difficult intubation, when a PSLMA is used to ventilate the patient. The PSLMA might then be preferred to the LMA-Classic™ when a potentially full stomach is a concern.¹–³

On the LMA North America Web site,* which includes PSLMA specifications, it is mentioned that it is possible to introduce a 4.5-mm-ID uncuffed endotracheal tube into a size 1.5 or 2 PSLMA. We tested this information with a 4.5 uncuffed Mallinckrodt (MM) S.A. de C.V., a Mallinckrodt Company, Juarez, Mexico) tube, and we realized that this tube does not fit into a size 1.5 PSLMA. Moreover, a size 2 PSLMA is also too small to accept easily a 4.5 uncuffed tube. However, it is possible to introduce a 4.0 uncuffed tube into a 1.5 or 2 PSLMA.

Support was provided solely from institutional and/or departmental sources. After numerous failed attempts to acquire a manufacturer reply to this letter, it is being published without a response. —Michael M. Todd, M.D., Editor-in-Chief

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We believe that the problem may be related to variations in the outer diameter of endotracheal tubes from one company to another, inner diameter being the same.

In our opinion, this misinformation on the LMA North America Web site should be corrected because many anesthesiologists transcribe it on their difficult airway chart for emergency airway management.

Marie-José Colas, M.D., F.R.C.P.C.,† René Martin, M.D., F.R.C.P.C., †University of Sherbrooke, Sherbrooke, Quebec, Canada. marie-jose.colas@usherbrooke.ca

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