To the Editor.—We read with interest the recent report by Martinez et al. regarding a combined infraclavicular plexus blockade with suprascapular nerve block for humeral head surgery in a patient with severe respiratory failure. However, we have some concerns with regard to the following points: First, it is not clearly stated whether the authors performed an infraclavicular plexus block using the coracoid technique (originally described by Whiffler) or the vertical infraclavicular technique (described by Kilka et al.). In the case report section the authors wrote, “brachial plexus was performed using ‘the coracoid and infraclavicular technique.’” However, figure 1B in the case report of Martinez et al. shows the territories usually blocked by the vertical infraclavicular brachial plexus block. One must be aware of the different extensions of sensory blockade produced by the coracoid technique, the vertical infraclavicular technique, and the modified approach of the Raj technique. Because the coracoid technique approaches the brachial plexus more distally than the vertical infraclavicular technique, the axillary and musculocutaneous nerves are often missed or are not adequately blocked with the former technique. Deleuze et al. described a successful sensory blockade of the axillary nerve by the coracoid technique in only about 22%. Gaertner et al. showed that a multiple injection technique, as compared to a single injection, improved the overall success rate of the coracoid technique from 40% up to more than 70%, without detailing the effect on the axillary nerve.

Martinez performed a suprascapular nerve block to avoid hemidiaphragmatic paresis secondary to interscalene brachial plexus block. In the current case, the possibility of an iatrogenic pneumothorax during this procedure must be mentioned. This theoretical disadvantage is also described for the vertical infraclavicular technique. In the current case, a pneumothorax could have been at least as deleterious as a mediastinal pneumothorax. In the current case, patients undergoing interscalene analgesia with ropivacaine 0.2% versus patient-controlled intravenous analgesia.”

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In Reply.—We read with interest the comments of Blumenthal et al. on our article. In the reported case, the infraclavicular block was performed using a modified approach of the technique reported by Salazar et al. The patient lies supine with the head turned away from the arm to be anesthetized. The puncture is performed 1.5 cm internal to the coracoid process and 1.5 cm caudal to the clavicle, with the arm abducted 45 degrees. The direction of the needle is perpendicular to the operating room table (vertical technique). In general, infraclavicular block techniques attain their effect by diffusion of the anesthetics in an area close to the brachial plexus. Therefore, two groups of techniques can be differentiated according to the site where the infraclavicular plexus is reached. The access between the clavicle and the medial border of the pectoral minor (Salazar et al., Rodríguez et al., Kilka et al., Wilson et al.) was performed in this case. Other techniques describe the access below or distal to the pectoral minor (Kapral et al. and Raj et al.). In the former, the fascicles of the brachial plexus lie grouped along the posterolateral edge of the axillary artery, and a complete block of the upper extremity can be achieved because of the anatomic proximity of the fascicles without arterial interposition. If the infraclavicular plexus is reached more distally, the interposition of the axillary artery and the division of the fascicles into the terminal nerves could lead to an incomplete block. When compared to the interscalene technique, the proposed approach has some advantages, such as the anesthetic effect on the medial fascicle (C8–T1) and the intercostobrachial nerve. However, the anesthetic effect on the superficial cervical plexus or the suprascapular nerve (obtained with the interscalene approach) would not be achieved. For those reasons, the proposed technique (vertical infraclavicular block + suprascapular block) could be even more effective than the interscalene approach.

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With regard to the incidence of complications, the interscalene approach causes phrenic paresis, either by single-shot or continuous local anesthetic infusion, but occurrence of pneumothorax is rare. Supraclavicular block produces phrenic paresis, too, but the feared complication is pneumothorax. On the other hand, the infraclavicular techniques carry a low risk for either complication, as does the suprascapular nerve block. Therefore, we think that the risk-benefit balance of the available techniques supports our election of the infraclavicular block.

The interesting comments of Blumenthal et al. provide us with more information and enable us to consider the infraclavicular technique, which surely will be of increasing interest among anesthesiologists in the future.

Julia Martinez, M.D.,* Xavier Sala-Blanch, M.D., Isabel Ramos, M.D., Carmen Gomar, M.D. CLINIC Hospital Universitari de Barcelona, Barcelona Spain. * jscon@clinic.ub.es

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Mechanisms of Hypotension and Bradycardia during Regional Anesthesia in the Sitting Position

To the Editor—We would like to commend Drs. Campagna and Carter for their informative review1 on the Bezold-Jarisch reflex and mechanisms of heart rate and blood pressure regulation. It is clear from their exhaustive report that these mechanisms are complex, interactive, and, as yet, not fully understood.

We disagree, however, with the authors’ interpretation of previously proposed mechanisms of hypotension and bradycardia during shoulder surgery in the sitting position under peripheral nerve blockade. They state that others have attributed these cardiac events to “peripheral vasodilation from the sitting position” as well as “venous pooling in the setting of peripheral nerve blockade.” However, the mechanism D’Alessio et al.2 proposed to account for activation of the Bezold-Jarisch reflex is in fact “venous blood pooling (induced by the sitting position), and a heightened cardiac contractile state (induced by the β-adrenergic effects of epinephrine).”

During anesthesia under interscalene block, the resulting blockade of sympathetic innervation to the upper extremity results in vasodilation in that arm with minimal, if any, systemic hemodynamic effects. The venous pooling is more likely related to the sitting position only in that arm with minimal, if any, systemic hemodynamic effects. The venous pooling is more likely related to the sitting position rather than the peripheral nerve blockade. There is evidence that during tilt table testing, positive responders have an associated decrease in left ventricular volume due to lower extremity venous pooling.3 It is reasonable to assume some degree of venous pooling in the beach chair position as well.

We believe that the important activating events are central volume depletion and vigorous cardiac contractions. Jacobsen et al.4 evaluated central volume depletion diagnosed echocardiographically secondary to epidural anesthesia and found a relationship with acute bradycardia. Contrary to the current authors’ assertion, the studies by Liu et al.5 and Davarth et al.6 did in fact reveal that patients with a positive tilt response had a decrease in left ventricular volumes, although modest in nature. These reports lend support to the hypothesis that some degree of central volume depletion is involved in this reflex.

We believe that the relative contribution of the central volume depletion to the degree of fractional shortening is the key to activation of reflex slowing. For example, a vigorously contracting, moderately empty ventricle may be as prone to slowing as a normally contracting, severely depleted one. To our knowledge, this has not been formally evaluated, but it may explain some of the contradictions in various published studies.

The reference the current authors cite for “no evidence of increased contractility in response to concentrations of epinephrine seen in local anesthetic mixtures for nerve block”7 actually refers to epidural, not peripheral neural, blockade. Furthermore, that study shows that epinephrine causes complex dose-dependent effects on cardiac output, stroke volume, and vascular resistance in the described situations, making actual determinations of contractility difficult to predict. During shoulder surgery in the sitting position under interscalene block, there are other sources of exogenous epinephrine, in addition to that added to the local anesthetic. These sources include subcutaneous infiltration by the surgeons, and epinephrine added to the irrigating solution used for arthroscopy. This additional epinephrine is likely absorbed during the case and may, as in Bonica et al’s study,8 affect hemodynamic variables.

Finally, the authors cite a case as evidence that the mechanism involved is not the Bezold-Jarisch reflex. A variety of potential causes of the hypotension and bradycardia are observed in this patient, including local anesthetic toxicity, preexisting cardiac pathology, or reflex events. Without additional information, this case offers no insight into the mechanisms of hypotension and bradycardia in this setting.

Taken as a whole, we believe that the literature does support the mechanism of the Bezold-Jarisch reflex in this clinical scenario. It certainly is not conclusive, and further clinical studies must be performed before making definitive statements.

Gregory A. Liguori, M.D.,* Richard L. Kahn, M.D., Michael A. Gordon, M.D., Michael K. Urban, M.D., Ph.D. Hospital for Special Surgery, Weill Medical College of Cornell University, New York, New York. * liguori@hss.edu

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In Reply.—We are pleased to have read and to have the opportunity to respond to the thoughtful comments of Ligouri et al.

We wish to correct the authors in that we did not state that others have attributed the events seen during peripheral nerve block to “peripheral vasodilatation from the sitting position” as well as “venous pooling in the setting of peripheral nerve blockade.” These portions of text were taken from different portions of our review and were presented out of context. In fact, we clearly state that other authors ‘hypothesized that the etiology of the hypotension and bradycardia was based on a combination of peripheral vasodilatation from the sitting position, increased contractility of the heart secondary to absorbed epinephrine from block mixture, and vigorous contraction of an ‘empty’ ventricle.’

We do not suggest a causal relationship between the block and venous pooling; rather, that venous pooling, by any cause (sitting, in this example), in the setting of a nerve blockade, has been suggested by others to be causally related to hypotension and bradycardia. D’Allesio et al. postulate that this event, along with heightened myocardial contractility (secondary to systemically absorbed block mixture epinephrine), is requisite for the hemodynamic embarrassment in question. We are aware of no evidence that maintenance of a sitting position is associated with significant venous pooling and decreased ventricular chamber size (as has been reported with tilt table testing) much less activation of the Bezold-Jarisch reflex (BJR). Even in persons undergoing tilt table testing, such a relationship is in fact highly variable, and, importantly, tilt testing is not a surrogate for either the afferent or efferent limbs of the BJR.

We agree with Ligouri et al. in that Liu et al. and Davrath et al. among others, inform us that the changes in left ventricular volumes during tilt testing are not significant (are “modest”). Importantly, the postulated requirement for triggering the BJR is “increased contractility in the setting of an empty ventricle” with no definition of “empty” offered. This begs the question of what the definition of “empty” is, and whether the modest reductions cited above are consistent with this definition based on experimental data. To our knowledge, the best quantitative evidence showing a relationship between central and ventricular blood volumes and activation of cardio-inhibitory receptors dates from the 1970s. Because ventricular volumes were quantified, the definition for “empty” must be considered, for our purposes, the volume at which those investigators elicited the BJR. Whether the much more “modest” reductions cited above can also elicit the BJR has not been demonstrated.

Ligouri et al. seem to agree with our statement that there is no support in the literature for concentrations of epinephrine resulting from block mixture augmenting cardiac contractility. The Bonica et al. article does focus on epidural epinephrine, but given that the translocation of drugs from the epidural space to the systemic circulation is rapid, it is reasonable to use this pharmacokinetic model as a point of maximum concentration relative to clearance of drug from peripheral locations. The suggestion by Ligouri et al. that in the surgical setting, other sources of epinephrine exist is true, but whether they contribute to hemodynamic instability remains speculative.

The concept that Ligouri et al. cite as the key to activation of the reflex, the degree of fractional myocyte shortening relative to central blood volumes, is well founded in the literature. We share their interest in this concept. Although this notion has never been formally evaluated, we agree that it could explain many of the apparent contradictions in the literature.

Finally, we wish to thank the current authors for pointing out that we neglected to address other potential causes of bradycardia and hypotension in the case cited in the review. This represents a key point of our argument against labeling the BJR as the cause of bradycardia and hypotension in the setting of regional anesthetics. As Ligouri so eloquently stated: “There are a variety of potential causes of the hypotension and bradycardia . . . including local anesthetic toxicity, pre-existing cardiac pathology, or other reflexes. Without other information, this case offers no insight into the mechanisms of hypotension and bradycardia in this setting.” Clearly, our patient illustrates that there are other causes of the hemodynamic embarrassment. For reasons that are unclear, other authors in the literature largely have ignored these other potential causes.

We understand and sympathize with the contention of Ligouri et al. that BJR is responsible for the bradycardia and hypotension observed during regional anesthetics, but we disagree with their assertion that the literature supports such a relationship. We remain wedded to our contention that the literature provides minimal support, aside from conjecture, for this causal relationship and, in fact, may support a mechanism other than the BJR. As a purpose of our review was to draw attention to the fact that many such concepts lack formal evaluation and that, therefore, using them to support the argument that the BJR is causally related to hemodynamic responses seen during regional and neuraxial blockade is specious. We do not disagree about the possibility of a causal relationship between certain observations and the BJR, only that assertions that this relationship exists remain based on speculative notions.

Jason A. Campagna, M.D., Ph.D.,* Christopher Carter, M.D.
Neuroplasticity Research Group, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts. * jcampagna@partners.org

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Erythrocyte Transfusion and Postsurgical Morbidity in Cardiac Surgery Patients: Is It the Storage Time or the Number of Transfused Concentrates that Really Matters?

To the Editor—We read with interest the article about the influence of erythrocyte concentrate storage time on postsoperative morbidity in cardiac surgery patients published by Leal-Noval et al. in a recent issue of the Journal, which was accompanied by an editorial view. The main finding of this study was an association between storage time of packed red blood cells (PRBC) and the acquisition of postsoperative nosocomial pneumonia, but not between the former and prolonged mechanical ventilation time. We would like to comment regarding two points. First, we wish to review some mechanisms by which the relationship between the number of units and pneumonia/ventilation might be connected. Second, we want to point out that the problem may not simply be the age of the transfused blood, but also the absolute numbers of units transfused.

The current authors suggested that allogeneic transfusion may cause some deleterious effects that have not been recognized and which may explain the association between the number of PRBC transfusions and prolonged mechanical ventilation, but not between the duration of storage and prolonged mechanical ventilation. Induction of a subclinical transfusion-related acute lung injury (TRALI) could be one of those frequently underdiagnosed deleterious effects. It has been proposed that TRALI is the result of the mutual interaction of two clinical events, the first being a predisposing clinical condition (e.g., a postoperative systemic inflammatory response syndrome) and the second being the transference of biologically active molecules in stored PRBC and platelets that have neutrophil priming activity. The concentration of some of these molecules (e.g., cytokines, neutral lipids, lysophosphatidylserine) in the stored blood component increases with the product age, whereas that of other molecules (e.g., human lymphocyte antibody class I, class II, and granulocyte antibodies) does not. To this regard, Silliman et al. have found that TRALI was associated with the transfusion of older blood products, especially platelets, and that patients with cardiac disease who required cardiopulmonary bypass were at particular risk to develop TRALI reactions. However, in previous work, this group showed an increase in the incidence of postsoperative nosocomial pneumonia in those patients transfused with more than 4 units. In the present work, patients transfused with more than 4 units. In the present work, patients who developed postoperative nosocomial pneumonia received more PRBC units than those who did not (8.8 ± 4.7 vs. 5.4 ± 4.7, respectively; P < 0.05). Because the authors found that each transfused PRBC unit increased the risk of postoperative pneumonia by 11%, we believe that the implementation of a perioperative blood-saving program, including several alternatives to allogeneic erythrocyte transfusion (e.g., preoperative treatment of anemia, aprotinin in pump-prime, perioperative autologous salvage), together with a restrictive transfusion strategy, would probably be a better option to reduce postoperative morbidity in cardiac surgery patients.

Manuel Muñoz, M.D., Ph.D., Aurelio Gómez-Luque, M.D., Ph.D., Enrique Naveira-Abegón, M.D., Ph.D., Gemma Ramírez, M.D. GIEMSA (International Multidisciplinary Autotransfusion Study Group), School of Medicine, University of Málaga, Málaga, Spain. *mmunoz@uma.es

References
In Reply:—We are very thankful to Muñoz et al. for their interest in our recent article.1 We agree with their speculation that transfusion-related acute lung injury might be one of the various causes explaining prolonged mechanical ventilation. However, the relationship between transfusion and transfusion-related acute lung injury and mechanical ventilation is difficult to establish in cardiac surgery patients, who are known to be under the influence of many other causes of pulmonary dysfunction.2 For instance, the association between the number of units transfused and mechanical ventilation may be due to an overload of transfused units, thus favoring the occurrence of pulmonary edema.

In our series, platelet transfusion (as a dichotomous variable) was considered a risk factor for morbidity. However, in the preliminary multivariate analysis, patients transfused with platelets did not show a higher morbidity than those not receiving platelet transfusions. Therefore, platelet storage time was not considered necessary to investigate.

We demonstrated an association existing between nosocomial pneumonia and the number of erythrocyte concentrate units transfused,3 and between nosocomial pneumonia and length of storage of erythrocyte concentrate.1 This is not unexpected, though, as the potential deleterious effects of the storage time may multiply according to the number of units transfused.

We also agree that blood-saving programs and alternatives to allogeneic transfusion are needed. However, the applicability or effectiveness of many methods remains controversial, so preoperative autologous donation programs are effective in decreasing allogeneic blood transfusion but are costly and applicable to elective patients only. The effectiveness of acute normovolemic hemodilution is too unreliable to decrease the risk of allogeneic blood transfusion in elective cardiac surgical patients with normal cardiac function.3 Moreover, among the pharmacologic strategies for blood conservation, antifibrinolytics such as aprotinin are the only agents that have been shown to reduce the risk of mortality after cardiac surgery.5

We believe that the judicious use of rational transfusion guidelines may still be the most simple and cost-effective means of blood conservation. However, blood consumption is still high and is unavoidable, thus justifying the need for studies designed to clarify the potential deleterious effects of blood transfusion, including storage.

Santiago Ramón Leal-Noval, M.D., Ph.D.,* Irene Jara-López, M.D., Ph.D., Ana Marín-Niebla, M.D.
Servicio de Cuidados Críticos y Urgencias, Hospital Universitario Virgen del Rocío, Seville, Spain.
sramon@cica.es

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To the Editor:—Ahmad et al.1 reported on the impact of electroencephalographic Bispectral Index (BIS) monitoring on fast tracking of gynecologic outpatients undergoing laparoscopic surgery. Although these authors concluded that the use of BIS monitoring did not have a significant effect on the ability to fast track outpatients at their institution, their seemingly “unexpected” findings are actually quite predictable given their study design.

In their article, the authors state, “sevoflurane was titrated to maintain a BIS value in the 50–60 range . . . .” Although no actual BIS data were presented, the mean (± SD) concentration of sevoflurane was reported to be 2.14 ± 0.25%. Unfortunately, this concentration of sevoflurane is simply not consistent with BIS values of 50–60. In an earlier study involving a similar outpatient population,2 sevoflurane concentrations of 0.6–1.2% (0.9 ± 0.3%) were associated with BIS values in the range of 55–65 (62 ± 3%!) In this earlier study, sevoflurane concentrations of 1.5–2% (1.8 ± 0.5%) were consistently associated with BIS values of less than 50 (42 ± 8%).2 A more recent study by Hodgson and Liu,3 also published in the Journal, found a linear relationship between the end-tidal concentration of sevoflurane and the BIS value. With sevoflurane concentrations of 1–2%, the BIS values would be expected to range from 50 to 30.

An even more troublesome point is that when Ahmad et al. presented these study results,4 they stated that a BIS target range of 40–50 was maintained in their BIS monitored group. This was also verified when the authors were questioned about their study by Dr. Girish Joshi from the University of Texas Southwestern Medical Center at Dallas in the November 2001 issue of Anesthesiology News (New York, McMillon Publishers).

Of importance with respect to the negative findings reported by Ahmad et al., the anesthetic, analgesic, and muscle relaxant drug dosages were all identical in both their control and BIS-monitored groups. It is obvious that the anesthesiologists caring for patients in the BIS group failed to use the BIS monitor to make decisions regarding

The authors have no conflicts of interest regarding the subject of this letter.

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their use of any of these anesthetic drugs. Therefore, the negative findings are totally predictable. Of interest, at least two more recent studies have confirmed our earlier findings that cerebral monitoring improves the titration of volatile anesthetics and, thereby, can facilitate a fast-track recovery.

Another flaw in the study design that could also have biased the results against BIS monitoring relates to the anesthetic management at the end of the operation. Although sevoflurane was discontinued when the laparoscope was removed from the abdominal cavity in both study groups, nitrous oxide was added in the BIS-monitored group to "maintain the BIS value below 60 ..." On the other hand, in the control group nitrous oxide was only added if the blood pressure and/or heart rate increased to more than 20% above the awake values! The authors failed to mention how many subjects received nitrous oxide in each group. Therefore, this practice would have biased the emergence data in favor of the control (vs. BIS-guided) group.

The criteria used for fast tracking was the standard Aldrete scoring system, and it is not clear that any effort was made by the investigators to implement a paradigm shift in the recovery process by educating the anesthesia personnel and nursing staff. In a recent study by Apfelbaum et al., a multidisciplinary educational program was believed to be critically important in increasing the PACU bypass rate and, thereby, facilitating the fast-tracking process.

Because the authors’ institution was apparently able to fast track more than 85% of their cases without using a cerebral monitor, the study was in fact seriously ‘underpowered’ with respect to their primary outcome variable. Interestingly, the authors failed to present the actual “bypassed” (vs. nonbypassed) recovery times. The only data presented was the percentage of patients who actually bypassed the PACU after satisfying the bypass criteria during a 10-min observation period following discontinuation of the anesthetic drugs. Of interest, the mean length of their stay in the phase II recovery area averaged over 200 min.

Finally, the authors state that a recent study in a similar population reported fast-track rates of only 8–11%. However, in a study published in 2001, we reported fast tracking 80% of our gynecologic laparoscopy patients undergoing general anesthesia at a public teaching hospital in Dallas, Texas (Parkland Memorial Hospital).

Rather than clarifying the issue with regard to BIS monitoring and fast tracking, this article has simply ‘muddied the water.’ We would agree that this study successfully ‘eliminated the possibility of contamination of the study results by anesthesiologists who learned from their experience using the BIS® monitor ...’ because they obviously failed to change their standard anesthetic practice based on the information provided by the cerebral monitor. By requiring ‘strict adherence’ to their standard anesthetic practices, it is not unexpected that they achieved a negative result. We believe the results of this study are misleading, and we strongly disagree with the authors’ conclusion that “this study clearly demonstrates that the application of the BIS® monitor does not have a significant impact on the ability to successfully fast track outpatients following general anesthesia ...”

Paul F. White, Ph.D., M.D.*, Dajun Song, M.D., Ph.D. University of Texas Southwestern Medical Center at Dallas.
* paul.white@utsouthwestern.edu

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Anesthesiology 2004; 100:195–6

In Reply—Thank you for the opportunity to respond to the letter from Drs. White and Song. There is an important distinction between the subjects in our study and those in the reports quoted by them. Our patients underwent inhalation inductions with sevoflurane, which consisted of priming the anesthesia circuit with an oxygen flow rate of 8 l of oxygen and 8% sevoflurane prior to induction of anesthesia, having the patients breathe this mixture until loss of response to verbal command (“Open your eyes”), and then reducing the concentration to 3.5% until tracheal intubation. This technique resulted in initial high end-tidal concentrations of the volatile agent and the resultant mean concentrations we reported.

We apologize for the typographical error in our abstract, which we failed to notice until after the meeting; however, we have reported the correct Bispectral Index (BIS) range in our publication. The dosages of opioid and muscle relaxant were not significantly different in the two groups because our subjects were similar in size and the procedures were of similar duration. We chose to standardize the dose of opioid so that the only variable would be the dose of volatile agent and resultant depth of hypnosis, which in turn should be reflected by the BIS value. The time from removal of the laparoscope to the completion of surgery was approximately 10 min, and we did not believe that inhalation of nitrous oxide for 5–10 min would have a significant impact on arousal time.

We agree with Drs. White and Song that by bypassing the phase II recovery area is one only aspect of fast tracking, which consists of the process of efficiently conducting patients through the perioperative period. At our institution, before the start of our study, a clinical pathway was instituted for patients undergoing gynecologic laparoscopy to facilitate the process. This involved education of not only the healthcare team but also the patients, so that they had realistic expectations regarding postoperative care. The gynecologists at our institution still require their patients to void prior to discharge, and this was the usual cause for the extended stay in the phase II recovery area.

Our sample size and power analysis were based on published reports on BIS use and our clinical experience. Unexpectedly, the number of subjects who successfully bypassed the phase I recovery area was not statistically different between the BIS and non-BIS monitored groups. Based on these results, we agree with Drs. White and Song that our study is underpowered. Power analysis is most critical when trying to demonstrate that no difference exists between two independent treatment methods; the smaller the difference, the greater the number of subjects who must be evaluated. Post hoc, we used a chi-square test.
Sub-Tenon Techniques Should Be One Option among Many

To the Editor—We read with interest the article on sub-Tenon (STB) regional anesthesia by Guise in the Journal.1 The article confirms the usefulness and safety of the technique to provide good ocular conductions. Our concern is, however, that the author’s conclusion that STB is safer and preferable to other ocular anesthetic techniques is based solely on a comparison of this approach to a series of retrobulbar anesthetics, without consideration of the many other factors that may determine the choice of ocular anesthesia. As reported previously,2 cannula-based STB techniques should be considered as one option among many for accomplishing adequate ocular anesthesia. In our practices at a major university hospital (Bascom Palmer Eye Institute, Miami, Florida) and an outpatient community facility (Tampa Eye and Specialty Surgery Center, Tampa, Florida), we use a variety of ocular anesthetic techniques, including topical anesthesia, intracameral injection, peribulbar anesthesia, STB, and even general anesthesia.

Selection of the appropriate anesthesia technique should consider many factors that pertain to the patient, surgery, surgeon, anesthesia provider, and operative venue. Patient considerations include the cooperativeness of the patient, anatomic factors such as increased axial length, existence of a staphyloma, previous scleral buckle surgery, and coagulation status. Surgical considerations include the surgical approach (i.e., scleral tunnel vs. clear corneal). Surgeons’ preferences also must be considered: whether they require an anesthetic, or if chemosis affects their approach (e.g., lateral corneal incisions). The STB technique may not be the best choice in all of these situations. Furthermore, anesthesia providers should perform the techniques in which they are trained, and many are not prepared to use the STB approach. The operative venue is important because, for example, in a community setting where cataract surgery takes 15 min or less, the STB approach may not be efficient.

In Guise’s study, retrobulbar anesthesia complication rates were compared to the STB technique. Of note, the stated retrobulbar complication rate is higher than some quoted in the literature.3–5 Most clinicians practicing ophthalmic regional anesthesia currently use peribulbar anesthesia rather than retrobulbar because this technique carries a lower complication rate.6 Although peribulbar anesthesia affords greater safety, the possibility of scleral perforation, hemorrhage, brainstem anesthesia, and dysphoria still exist. The same is true for STB blocks. The author details in his article that all of these complications have been reported with STB anesthesia.1,7–8

Finally, we would note that the risks of all ocular anesthetic techniques are inversely proportional to education and experience. This is affirmed by several reports of adverse sequelae of ophthalmic anesthesia rendered by inadequately trained/educated personnel in the early 1990s.8–11 In a survey of 284 directors of anesthesiology and ophthalmology programs, no formal training/education in ophthalmic regional anesthesia was provided to anesthesia residents in the vast majority of programs.12 We strongly advocate that before performing any method of ocular anesthesia, attending anesthesiologists obtain adequate training/education through a suitable university program or via an organization such as the Ophthalmic Anesthesia Society.

Steven Gayer, M.D., M.B.A.* Gary D. Cass, M.D.* Bascom Palmer Eye Institute, University of Miami, Miami, Florida.

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Shireen Ahmad, M.D.,* Meltem Yilmaz, M.D., R-Jay Marcus, M.D., Silas Glisson, Ph.D., Annette Kinsella, R.N. Northwestern University Feinberg School of Medicine, Chicago, Illinois.

* sah704@northwestern.edu

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**Topical Anesthesia Is the Technique of Choice for Routine Cataract Surgery**

To the Editor:—I read with interest the recent report by Guise on sub-Tenon anesthesia for cataract surgery.\(^1\) The author’s technique was to incise the fused conjunctiva and anterior tenon capsule and insert a catheter in the posterior sub-Tenon space for the injection of 4 ml of local anesthetic. However, subconjunctival hemorrhages were noted in 7% of the cases, and 4.1% of the patients required an additional facial nerve block for persistent orbicularis tone. Moreover, 0.8% required a retrobulbar top-up block.

Guise’s technique for cataract surgery seems extremely complicated, considering that several surgeons at our facility almost exclusively use a topical technique, usually with tetracaine eyedrops\(^2,3\) or a topical technique supplemented with intracameral lidocaine.\(^4\)

I would appreciate the author’s thoughts regarding the reasons he advocates the sub-Tenon approach, rather than simple topical anesthesia.

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*(Accepted for publication August 2, 2003.)*

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**Combination of Morphine with Ketamine for Patient-controlled Analgesia: Is Ketamine Plasma Concentration Adequate?**

To the Editor:—We read with much interest the article of Sveticic et al.\(^1\) who designed a study to optimize combinations of morphine and ketamine, and a lockout interval for patient-controlled analgesia, using a direct search model. Opioids may activate N-methyl-D-aspartate pain facilitatory processes, leading to hyperalgesia and enhanced postoperative pain.\(^2\) Concomitant administration of the N-methyl-D-aspartate antagonist ketamine may therefore allow reduction in the dose of morphine. The optimization procedure converged to a morphine-to-

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ketamine ratio of 1:1 and a lockout interval of 8 min, allowing a reduction in mean pain scores with a low incidence of side effects. Ketamine plasma concentration of 60 ng/ml is the smallest concentration known to counteract hyperalgesia while producing minimal side effects. However, when ketamine is combined with morphine for patient-controlled analgesia, the dose of ketamine administered depends on the dose of morphine required. Interindividual variability in drug requirement to achieve satisfactory analgesia is well known, and there is no evidence that the dose of ketamine required to achieve optimal plasma concentration is linked to similar interindividual variability. Whether the optimal plasma concentration of ketamine has been achieved is therefore unknown.

Given that an initial bolus dose of ketamine 0.5 mg/kg followed by a continuous infusion of 2 µg/kg per min achieves a plasma concentration close to 60 ng/ml without side effects, we believe that concomitant continuous infusion of low-dose ketamine with patient-controlled analgesia morphine is the optimal technique for an opioid-sparing effect. Dosage of ketamine plasma concentration would have been of great interest in the study of Sveticic et al. Further study would be valuable in this field.

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In Reply:—We thank Van Elstraete et al. for their interesting remarks concerning our article.1 We agree that a continuous infusion of low-dose ketamine during patient controlled analgesia with morphine may be better than a patient-controlled approach alone. However, when applied to the clinical settings, problems arise. At best, an additional electronic pump would be required. This would, however, increase costs. Alternatively, a simple intravenous perfusor may be used. We find this approach less safe in the ward. Furthermore, the addition of another intravenous line and device is inconvenient and reduces patient’s mobility.

Thus, although from a pharmacologic point of view the observation by Van Elstraete et al. is correct, in our opinion the pure patient-controlled analgesia approach is more practical. Whether the continuous ketamine infusion is superior to the patient-controlled anesthesia delivery is yet to be demonstrated by a randomized controlled trial.

Gorazd Sveticic, M.D.,* Urs Eichenberger, M.D., Michele Curatolo, M.D., Ph.D., Inselspital, Bern, Switzerland.
* gorazd.sveticic@insel.ch

Reference


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Spinal Cord Temperature

To the Editor—In their confirmatory study, Kottenberg-Assenmacher et al. concluded that surface hypothermia to a core temperature of 32°C did not depress median nerve somatosensory-evoked potentials (SSEP) amplitude, but did prolong latency.1 This is an important observation, as patients undergoing certain orthopedic and vascular surgical procedures that may result in spinal cord ischemia have SSEPs monitored. Because these patients are often mildly to moderately hypothermic, it is important to know if the spinal cord temperature is at the same core temperature and, if so, what affect spinal cord hypothermia has on SSEPs.

In their study, the authors induced hypothermia, and although the mean total duration of the surface hypothermia was not stated, the article inferred that it was acute and transitory. To determine whether there were effects of hypothermia on the spinal cord that affected the SSEPs, we recommend the following:

1. Measure the temperature of the spinal cord. This can be approximated clinically by inserting a thermistor-tipped 4-French pulmonary artery catheter via a needle into the subarachnoid space at the lumbar level. Using this technique, the lowest mean cerebrospinal fluid temperature in a case series of patients undergoing spinal cord cooling for thoracoabdominal aneurysm repair was 26.7 ± 3.2°C.2 This technique will provide an indirect, but clinically relevant, measurement of temperature at the spinal cord level. If this were undertaken, the tibial nerve SSEP would need to be measured to determine the effect of spinal cord hypothermia on the SSEP.

2. Measure SSEPs in patients with acquired poikilothermia in whom steady-state hypothermia can be maintained. MacKenzie et al. published a case series of four patients with acquired poikilothermia who had spontaneous hypothermia maintained at 35.5 ± 0.3°C for a minimum of four days.3 The inference is that in poikilothermia, the patient’s central core steady-state temperature is more likely to reflect the local spinal cord temperature. In these patients, hypothermia increased the latency of the central conduction times; however, in this group, only the latencies could be reliably identified.

As interest in moderate hypothermia as a neuroprotective technique increases, it is important to definitively answer the question concerning the effect of spinal cord hypothermia on SSEPs.

Barry A. Harrison, M.B., B.S.,* Timothy S. J. Shine, M.D., Martin L. De Ruiter, M.D., Michael J. Murray, M.D., Ph.D. Mayo Clinic, Jacksonville, Florida. * barry.barry@mayo.edu

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To the Editor:—We wish to report a potentially catastrophic failure mode of the absorber module of the Aestiva 3000 anesthesia machine (Datex-Ohmeda, Madison, WI). During anesthesia for removal of a basolophen in a 17-yr-old girl, the surgeon requested a Valsalva maneuver to confirm adequate dural seal. On switching from ventilator mode to bag mode, it was not possible to generate positive pressure in the breathing circuit; during efforts to troubleshoot the problem, the patient’s depth of anesthesia lightened to the point that she aroused and self-extubated. Following reintubation, the patient was successfully ventilated using the ventilator. At the conclusion of surgery, the patient was allowed to breathe spontaneously from a Mapleson D (Vital Signs, Totowa, NJ) breathing circuit and auxiliary oxygen source, as it remained impossible to generate positive pressure with the anesthesia machine in bag mode.

Examination of the machine immediately after the episode revealed a massive leak in the bag mode; activation of the oxygen flush with the bag-ventilator switch set to bag mode resulted in expansion of the ventilator bellows. The machine was pulled from service and the absorber assembly was disassembled and inspected, revealing a crack in the bracket holding the shaft of the bag-ventilator switch toggle actuator (fig. 1). A review of the design of the Aestiva absorber shows how this defect produced the failure.

In the Aestiva absorber, the bag-ventilator switch uses a rocker-arm toggle actuator (fig. 1) to close either the ventilator-exclusion valve (in bag mode) or the bag-exclusion valve (in ventilator mode). In the event that both valves are open, compression of the bag results in diversion of gas to the ventilator assembly, where it is vented through the open ventilator pop-off valve to the scavenger system, resulting in a massive leak. Activation of the ventilator with both exclusion valves open results in diversion of the inspired tidal volume to the breathing bag and/or the scavenger system (depending on the pressure limit set on the ventilator).

Fig. 1. Bag-ventilator selector switch mechanics. White arrows point to the crack in the bracket holding the shaft of the toggle actuator.
In Reply:—Thank you for the opportunity to respond to the letter from Gunter et al., in which they correctly describe both the function of the bag-to-ventilator switch and the results produced by the broken rocker-holding mechanism. The entire control panel assembly from the Aestiva used during this case was returned to Datex-Ohmeda, where the cause of the failure was determined. Further investigation into the root cause of the breakage determined no design, manufacturing, assembly, or service issues that may have resulted in this break. Although every attempt is made to design and manufacture robust and reliable products, occasionally a part may fail for reasons that remain indeterminable.

The authors continue by describing sporadic episodes during which the bag-to-ventilator switch seems to incompletely exclude the ventilator when switched into the bag mode. Unfortunately, these additional events have not been reported to Datex-Ohmeda. In the absence of such reports, no investigations have been implemented. We will, however, begin an internal review of the bag-to-ventilator switch.

Datex-Ohmeda agrees with the authors regarding the need for an auxiliary method to provide ventilation during administration of each anesthetic, as required by the U.S. Food and Drug Administration, and wishes to thank the authors for their report. In addition, Datex-Ohmeda encourages all anesthesia providers and anesthesia technicians to report possible problems with any piece of equipment to the respective manufacturer so that proper investigations may be conducted.

Michael Mitton
Datex-Ohmeda, North America, Madison, Wisconsin. michael.mitton@us.datex-ohmeda.com

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To the Editor—Clinicians performing airway management are at risk of exposure to airborne respiratory pathogens. Ever since the severe acute respiratory syndrome outbreak, most anesthesiologists in Hong Kong have been wearing extra protection, including a full-face shield. On many occasions, we have witnessed the sharp lower edge of the shield to be pressing on the patient’s eye(s), potentially causing serious injuries (Fig. 1).

Other objects that the anesthesiologist wears could also injure patients’ eyes, including photo-identity badges and wristwatch bands. Applying eye protection in patients before intubation has been suggested, but the process may be cumbersome and time-consuming and may interfere with other aspects of airway management (e.g., eye goggles on the patient could interfere with bag-mask ventilation). Although removing a watch or photo-identity badge before intubation is an attractive option, removing the face shield may not be. The best protection remains the awareness that the patient’s eyes are vulnerable, and that not only what we do (or not do), but also what we wear, could cause serious injuries.

Anthony M-H. Ho, M.S., M.D., F.R.C.P.C., F.C.C.P.*, Geoffrey C. S. Lam, M.B.,Ch.B., Manoj K. Karmakar, M.B.B.S., M.D., F.R.C.A. The Chinese University of Hong Kong, Prince of Wales Hospital, Hong Kong, Special Administrative Region of the People’s Republic of China. * hoamh@hotmail.com

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