

Combined Infraclavicular Plexus Block with Suprascapular Nerve Block for Humeral Head Surgery in a Patient with Respiratory Failure: Is an Alternative Approach Really the Best Option for the Lungs?

To the Editor:—We read with interest the recent report by Martinez *et al.*¹ 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 Whiffler²) or the vertical infraclavicular technique (described by Kilka *et al.*³). 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.⁴ 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.*⁵ described a successful sensory blockade of the axillary nerve by the coracoid technique in only about 22%. Gaertner *et al.*⁶ 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.⁷ This theoretical disadvantage is also described for the vertical infraclavicular technique.⁸ In the current case, a pneumothorax could have been at least as deleterious as a phrenic nerve paresis. As shown by Borgeat *et al.*⁹ and Boezaart *et al.*,¹⁰ the decrease of hemidiaphragmatic excursion after interscalene brachial plexus block can be reduced when the block is performed through the interscalene catheter rather than with a single bolus. The catheter technique, either performed at the interscalene or at the

infraclavicular level (where the modified approach of the Raj technique seems to be the optimal solution¹¹), would have also offered good surgical conditions and efficient postoperative analgesia without the danger of a pneumothorax or an insufficient block.

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In Reply:—We read with interest the comments of Blumenthal *et al.* on our article.¹ In the reported case, the infraclavicular block was performed using a modified approach of the technique reported by Salazar *et al.*² 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.³ 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.*,² Rodríguez *et al.*,³ Kilka *et al.*,⁴ Wilson *et al.*⁵) was performed in this case. Other techniques describe the access below or distal to the pectoral minor

(Kapral *et al.*⁶ and Raj *et al.*⁷). 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.

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,⁸ 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.

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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 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 Davrath *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,⁷ 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.

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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 vasodilation 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 vasodilation from the sitting position, increased contractility of the heart secondary to absorbed epinephrine from block mixture, and vigorous contraction of an “empty” ventricle.”¹

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’Alessio *et al.*² 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,³⁻⁷ 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.*⁸ and Davrath *et al.*,⁹ among others,^{3,6} 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.² 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.^{10,11} Because ventricular volumes were quantified, the definition of “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.*¹² 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 reference 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, preexisting 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. A purpose of our review¹ 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.

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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 postoperative morbidity in cardiac surgery patients published by Leal-Noval *et al.* in a recent issue of the Journal,¹ which was accompanied by an editorial view.² The main finding of this study was an association between storage time of packed red blood cells (PRBC) and the acquisition of postoperative 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.¹ 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 two clinical events, the first being a predisposing clinical condition (*e.g.*, a postoperative systemic inflammatory response syndrome) and the second being the transfusion 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.*³ 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, although a significant proportion of patients (135, 16.9%) received platelet transfusion, Leal-Noval *et al.*¹ have not studied the possible association between either the number or the age of platelet concentrates and prolonged mechanical ventilation time. Despite that, because the number of PRBC transfused was significantly higher in patients with mechanical ventilation time greater than 24 h, the development of a certain degree of TRALI, in a dose-dependent manner, may result in a worsening of the pulmonary dysfunction induced by cardiopulmonary bypass with a prolonged mechanical ventilation time and, perhaps, contributes to a higher incidence of postoperative nosocomial pneumonia.⁴

Regarding PRBC storage time, there are several storage-dependent alterations in erythrocytes that, together with transfusion-related immunomodulation, may contribute to the increased risk of nosocomial pneumonia in the transfused patients. The decrease in the ability of erythrocytes to deform and unload oxygen in the peripheral tissues, possibly favoring the degree of visceral ischemia, have been mentioned,^{1,2} but other alterations may account for when erythrocytes are stored together with white blood cells. Leukocyte enzymes, including

neuraminidase, are definitive contributors to the desialylation of erythrocytes during storage and to the exposure of phosphatidylserine residues in the membrane outer leaflet.⁵ These changes make the erythrocytes more adherent to the inflamed endothelium and convert it into a procoagulant surface, thus contributing to the alteration of microvascular blood flow. In addition, these changes are also signals of senescence and enhance erythrocyte phagocytosis by resident macrophages.⁵ All methods in current use for erythrocyte storage result in the maintenance of 90% or more of the *in vivo* recovery for about 2-3 weeks; later an increasing proportion of erythrocytes become nonviable. When old PRBC are transfused, a substantial number of cells (some 10¹² erythrocytes in a four-unit transfusion) must be phagocytosed, which obviously uses part of the capacity of the phagocytic mononuclear system and may compete with infection defense with possible serious consequences, such as postoperative nosocomial pneumonia.⁶

We agree with Leal-Noval *et al.*¹ with regard to the need for continued research of the possible deleterious effects of old PRBC transfusion. However, in previous work,⁴ this group showed an increase in the incidence of postoperative nosocomial pneumonia in those patients transfused with more than 4 units. In the present work,¹ 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%,¹ 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,⁷⁻⁹ would probably be a better option to reduce postoperative morbidity in cardiac surgery patients.

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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.⁴ 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.

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Bispectral Index Monitoring and Fast Tracking after Ambulatory Surgery: An Unexpected Finding?

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 (\pm SD) concentration of sevoflurane was reported to be $2.14 \pm 0.25\%$. 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% ($0.9 \pm 0.3\%$) were associated with BIS values in the range of 55-65 (62 ± 3)! In this earlier study, sevoflurane

concentrations of 1.5-2% ($1.8 \pm 0.3\%$) 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, McMahon Publishers).

Of importance with respect to the negative findings reported by Ahmed *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^{5,6} have confirmed our earlier findings² 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.*⁷ 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 2001,⁸ 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. . . ."

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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 study¹ 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,² 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 bypassing the phase II recovery area is only one 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 perioperative 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 use³ 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

estimator to determine the effect size (W). We determined that 3,000 subjects would be required with this same study design to demonstrate that the difference between BIS and non-BIS monitored subjects is statistically significant, at a power of 0.8 and an alpha of 0.05. Furthermore, if one extrapolates the outcome results observed in this study, for the entire 3,000 study subjects, the bias would favor the non-BIS monitored group as having a better outcome.

It remains for the individual reader to decide whether a study of this magnitude is warranted to prove whether the BIS[®] monitor statistically affects the ability of the subjects to bypass the phase I recovery area following gynecologic surgery. We believe that our study demonstrated the importance of the choice of the appropriate anesthetic technique in this patient population. It is possible to fast track the majority of patients following careful titration of the newer volatile agents, and the use of the BIS monitor is not mandatory for patients to successfully bypass the phase I recovery area.

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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 conduction anesthesia. 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 akinetic eye, 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 dysrhythmia 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 tech-

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niques are inversely proportional to education and experience. This is affirmed by several reports of adverse sequelae of ophthalmic anesthetics 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.

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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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In Reply:—The purpose of the study at Auckland was to show that in a large study group and with a range of operator experience, the sub-Tenon block (STB) is very effective and has a low potential for sight-threatening complications. The article stated that it was not statistically possible to show that the incidence of ocular injury is lower with a sub-Tenon technique than any other.

With regard to topical *versus* regional anesthesia, the choice depends on three primary factors: surgeon, patient, and anesthesiologist preferences. Auckland hospital is a teaching hospital with a variable level of expertise for both anesthesiologist and ophthalmologist. Most of the surgeons in our institution have a preference for regional anesthesia for the majority of our patients, because the akinesia and loss of orbicularis tone provides superior operating conditions¹ and the best environment to teach junior surgeons. Anesthesiologists prefer regional anesthesia because it is associated with superior intraoperative analgesia.¹⁻³ The patient's cooperation is more important for topical anesthesia, and many elderly patients have difficulty maintaining a steady eye. Furthermore, there is evidence that the majority of patients prefer regional anesthesia to topical when given the choice.¹

The choice of regional technique will depend on the skill, experience, and personal preference of the operator as well as patient and surgical factors. Although it would be ideal for all anesthesiologists to be experts in all ophthalmic anesthetic techniques, in practice this is not feasible. We have chosen STB as our predominant technique because it provides an excellent degree of analgesia and akinesia, is virtually painless to perform, has a low injectable volume, avoids passage of sharp needles into the orbit, and has an extremely low incidence of significant complications. Performance of the block takes

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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 Svetcic *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

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less than 2 min and is well suited to "high-volume" lists. We believe that STB is least likely to result in ocular trauma in the hands of operators with a variable level of expertise. Although it is impossible to be certain that this technique has a lower incidence of complications than other regional techniques, particularly peribulbar anesthesia, there are only a handful of reports of complications from STB in a denominator that can be estimated to be in the hundreds of thousands. Anecdotally, more than 25,000 STB have currently been performed in hospital and "office-based" settings in the Auckland region, with no sight-threatening complications.

I would agree that appropriate training in regional anesthesia of the orbit is a pivotal factor in the safety of any regional technique.

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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.³ 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,^{4,5} 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 Svetcic *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 anesthesia approach is more practical. Whether the contin-

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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

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uous ketamine infusion is superior to the patient-controlled anesthesia delivery is yet to be demonstrated by a randomized controlled trial.

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Spinal Cord Temperature

thoracoabdominal aneurysm repair was $26.7 \pm 3.2^\circ\text{C}$.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 SSEP 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 $33.5 \pm 0.3^\circ\text{C}$ for a minimum of 4 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.

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In Reply:—We thank Harrison *et al.* for their comments and interest in our recent article.¹ We appreciate that they considered important our observation that surface hypothermia to a core temperature of 32°C did not depress median nerve somatosensory evoked potentials (SSEP) amplitude but rather prolonged latency. We also agree that this has implications for patients undergoing certain orthopedic and vascular surgical procedures in which SSEPs are monitored.

Harrison *et al.* remarked that we did not state the mean total duration of induced hypothermia. Note that duration of hypothermia with an esophageal temperature of approximately 32°C depended on surgical resection time, which, of course, varied between individuals. However, total duration of marked hypothermia of 32°C, as required for resection of choroidal melanoma, averaged 101 min ± 40 (SD). Thus, there was ample time for temperature equilibration across the body, including the spinal cord.

We agree that it would be interesting to know precisely how rapidly spinal cord and esophageal temperatures equilibrate in patients undergoing certain surgical procedures during hypothermia, and to exactly what degree spinal cord temperature influences SSEPs. Obviously, however, intraspinal cord temperature measurements with a thermistor-tipped pulmonary artery catheter, as suggested by Harrison *et al.*, are hardly feasible in humans undergoing eye surgery. However, as reported by Kumar *et al.*, there is a close correlation between intrathecal and core temperatures ($r = 0.873$; $P < 0.001$) during proximal

aortic surgery,² and during aortic clamping, intrathecal temperature is close to intraaortic temperature in rats.³

Thus, esophageal temperature during hypothermia, when maintained for some time, is likely similar to intrathecal temperature. Regardless, our data show that surface hypothermia to an esophageal temperature of 32°C does not depress median nerve somatosensory evoked potentials amplitude but rather prolongs their latency.

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Catastrophic Failure of Aestiva 3000 Absorber Manifold

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 baclofen pump 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

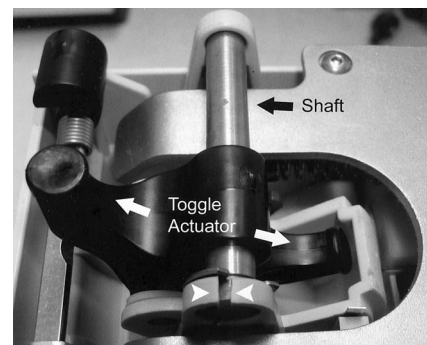


Fig. 1. Bag-ventilator selector switch mechanics. White arrows point to the crack in the bracket holding the shaft of the toggle actuator.

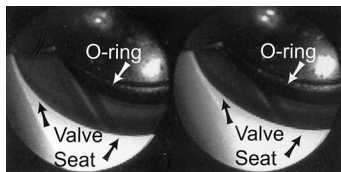


Fig. 2. Images of the ventilator-exclusion valve obtained by passing a fiber-optic bronchoscope through the absorber manifold. The image on the left shows the valve with the bag-ventilator selector switch set to ventilator mode. The image on the right shows the valve with the bag-ventilator selector switch set to bag mode; with the switch in this position, the O-ring should make contact with valve seat, closing the valve.

the pop-off valve). The exclusion valves are plunger-type valves; to seal, a pliable O-ring must make contact with the lip of the valve orifice. The crack in the support bracket allowed enough play in the rocker-arm actuator that the ventilator-exclusion valve did not seal when the selector was set to bag mode (fig. 2).

We have also experienced recurrent episodes in which it has been temporarily impossible to deliver positive pressure with the breathing bag on the Aestiva anesthesia machine; these events have typically occurred after switching from ventilator mode to bag mode and have been consistent with a massive circuit leak. All affected machines were reported to have had minimal leak during the anesthesia machine checkout procedure. During at least one of these episodes, activation of the oxygen flush with the bag-ventilator switch in the bag mode resulted in expansion of the ventilator bellows along with the breathing bag, as in the event described above. These episodes have resolved spontaneously during troubleshooting, and despite extensive examination by anesthesiologists, hospital biomedical engineers, and company representatives, no physical defects or mechanical problems were ever identified. We believe that these sporadic, self-correcting events occurred when minor alterations in the handling or function of the bag-ventilator switch permitted the ventilator-exclusion valve to fail to seal; manipulation of the switch during troubleshooting then corrected the problem.

In an effort to confirm that failure of the ventilator-exclusion valve

would result in the observed failure, we attempted to reproduce the failure mode by modifying the ventilator-exclusion valve. Removal of the ventilator-exclusion valve O-ring resulted in the ability to generate, but not sustain, positive pressure in the breathing bag. Removal of the entire ventilator-exclusion plunger assembly reproduced the failure mode as outlined above (inability to generate positive pressure with the bag, filling of the ventilator bellows with the oxygen flush when in bag mode, and proper function of the ventilator). It is interesting that it remained possible to generate positive pressure using the ventilator despite the laxity introduced into the switch actuator by the cracked bracket; examination of the valve fiber-optically (as in fig. 2) revealed that the bag-exclusion valve did indeed seal (not shown). This observation leads us to speculate that there is some difference in the operation of the bag- and ventilator-exclusion valves. This speculation is supported by the fact that all of the sporadic, self-correcting episodes mentioned above have involved the inability to generate positive pressure using the breathing bag and never the converse (*i.e.*, inability to generate positive pressure with the ventilator).

In conclusion, we have presented a failure mode of the Datex-Ohmeda Aestiva absorber assembly that has presented in both a catastrophic mode and in a sporadic, self-correcting mode. We speculate that both failure modes can be attributed to failure of the ventilator-exclusion valve to seal when the bag-ventilator selector switch is set to bag mode. Anesthesiologists experiencing loss of circuit integrity in an Aestiva anesthesia machine when switching from ventilator mode to bag mode should respond initially by cycling several times between bag and ventilator mode, which should correct the problem. If repeated cycling of the selector switch does not resolve the situation, then the patient should either be returned to the ventilator or be ventilated using an auxiliary oxygen source and breathing circuit. The possibility of unexpected loss of circuit integrity despite an adequate preanesthesia checkout demonstrates again the importance of having an auxiliary oxygen source and breathing circuit during administration of each anesthetic.

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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 addi-

tional 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.

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Potential Eye Injury due to Protective Face Shields

To the Editor:—Clinicians performing airway management are at risk of exposure to airborne respiratory pathogens.¹ 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.² Applying eye protection in patients before intubation has been suggested,² 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.

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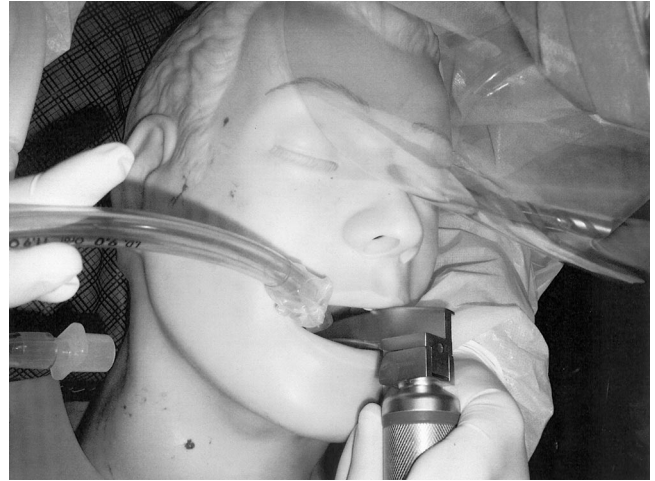


Fig. 1. The bottom edge of the full-face shield is almost pressing on the "patient's" eyes.

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