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Does Vasopressin Infusion Improve the Outcome of Severe Septic-Shock without any Adverse Effects?

To the Editor:— We read with great interest the report by Patel *et al.* on beneficial effects of short-term infusion of vasopressin for severe septic shock.[1] The authors reported that for 24 patients with severe septic shock, 4 h infusion of vasopressin spared norepinephrine to maintain mean arterial pressure and improved urine output and creatinine clearance compared with the control group (norepinephrine infusion) in a double-blinded, randomized, controlled fashion. We were impressed with the results showing that low-dose vasopressin therapy may increase survival of septic shock. However, there was no information about the outcome of their patients. To survive from severe septic shock, there may be a need for long-term vasopressin infusion to maintain stable cardiovascular status. Dunser *et al.* demonstrates 72-h vasopressin infusion brings about a significant increase in liver enzymes and total bilirubin concentration and a significant decrease in platelet count.[2]

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In Reply:— We agree with Dr. Iijima *et al.* who reemphasize that our study of a short-term vasopressin infusion in patients with severe septic shock does not address the key issue of survival. Furthermore, they point out that longer duration infusions may be associated with potentially adverse effects. Our current study was not designed to address survival. Nevertheless, we found that 1 of 11 patients who received norepinephrine infusion survived to hospital discharge while 5 of 13 patients who received vasopressin infusion survived. We think that the results of our 4 h study are not sufficient by themselves to advocate the use of vasopressin in patients with severe septic shock. A properly powered, randomized, controlled trial with an important primary endpoint, such as survival, is required.

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Depth of Anesthesia Monitors: Status Quo

To the Editor:— A recent editorial[1] proposes some interesting ideas, and places into perspective some of the many issues that have arisen since the introduction of the so-called "depth of anesthesia" monitors. The authors ask two main questions: (1) What are we trying to accomplish with these monitors? and (2) should these monitors be based on measurements derived from spontaneous electrophysiologic activity or evoked responses of the brain?

There are some conceptual hurdles that must be cleared when studying the depth of anesthesia. One of these is actually defining depth of anesthesia. Is depth of anesthesia equivalent to "level of consciousness?" Or should we go back to the classic definition of anesthesia, separating depth of hypnosis, depth of analgesia (as a measure of autonomic response to stimulus or stress), and level of muscle relaxation?

Maybe we should think about what is expected from the depth of anesthesia monitors. The BIS® (Aspect Medical Systems Inc., Newton, MA, USA) and A-line® (Alaris Medical Systems, Inc., San Diego, CA, USA) devices are designed to monitor the hypnotic state of the patient. Both process information from the cortex, although the A-line® also indirectly assesses the subcortical and brainstem auditory pathways. It is not surprising then, that the indices they derive are poor predictors of either movement (a spinal chord response, as suggested by Struys *et al.* in their study[2]) or autonomic and hemodynamic reactions, which are poorly related with the cortical response of the brain.[3]

The same idea is reflected when the authors mention the usefulness of the mid-latency auditory evoked potentials (MLAEP) monitors to predict the effect of benzodiazepines and narcotics on the level of hypnosis. With respect to benzodiazepines, it is correct that some publications state that they have little effect on MLAEP.[4] However, most of the studies carried out recently agree on the good response obtained with those monitors on patients anesthetized with benzodiazepines.[5,6]

Similarly, there is a substantial distinction between analgesia and hypnosis. Opioids are used to blunt the response to painful stimulation. However, no study has demonstrated that narcotics induce hypnosis by themselves, although they may reduce the amounts of other drugs needed to obtain a certain level of hypnosis. The old "Pure Analgesic Technique" from De Castro *et al.* applied in Europe during the mid-seventies comes to mind.[7,8] It used only huge doses of fentanyl plus neuromuscular blocking agents in order for patients to be in "no pain but able to answer." Because analgesia can be dissociated from hypnosis, it is thus illogical to expect monitors to "track" analgesia, or alternatively, changes induced by opioids may not reflect depth of hypnosis.

We therefore agree with the authors that both of these monitors provide only partial information about the anesthetic state of the patient. While we hope that there may someday be a true depth of analgesia monitor, currently we must use the information provided by depth of hypnosis monitors in combination with other relevant clinical data.

The second question, whether monitors should rely on spontaneous or evoked electrical activity, may actually be unimportant. Different anesthetic drugs act in different areas of the brain with different mediators. We believe that what matters is not whether the signals are spontaneous or evoked, but whether we can determine if the variation of those responses is caused by an increase in the depth of hypnosis brought upon by hypnotic drugs or due to an abolition of stressful stimuli caused by analgesic drugs. Something must also be said about the methods commonly used in most of the studies that analyze the response of the depth of hypnosis monitors. Many of them compare the response of the monitors against clinical signs like the loss of eyelash reflex or the OAA/S scale (Observer's Assessment of Alertness/Sedation).[9] This is in fact correct, however, it should be pointed out that to assess consciousness using the OAA/S scale, the patient has to be stimulated repeatedly. These stimuli have an effect on their hypnotic state. For example, OAA/S level 3 (which approximately corresponds to a BIS® value of 65 or an A-line® of 30, as in the study by Struys *et al.*[2]) is reached when the patient "responds only after his name is called loudly and/or repeatedly." This loud/repeated calling is a stimulus that could move the patient into a lighter hypnotic state, and the monitors will reflect this change with an increase in the index value. The same can be said when the patient is more deeply asleep, as in OAA/S 2 ("responds only after mild prodding or shaking"). The patient is again stimulated and could "lighten hypnotically." Which monitor value then corresponds to the OAAS/S, the value recorded before or after the stimulus? It could be argued that the value of the monitor should be registered only after the OAA/S level has been assessed. Maybe this helps to explain the overlapping values in the study of Struys *et al.*[2]

Again, we agree with the authors that these devices monitor the hypnotic level and not the depth of anesthesia, which is a much more complex phenomenon. Nevertheless, these devices are a great step forward. Very few of us want to go back to the time when the controversy was, Automatic blood pressure monitoring, or the finger of the anesthesiologist on the patient's radial artery. . .?

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In Reply:— We thank Dr. Litvan for his thoughtful comments about our recent editorial on the issue of "depth of anesthesia" monitors.[1] We agree that at best these devices should be considered "hypnosis" monitors, because by definition "adequacy of anesthesia" also implies adequate analgesia. The issue of prediction of movement has received considerable interest since Rampil *et al*. showed that an intact brain is not a prerequisite for determination of minimal alveolar concentration (MAC) in rats.[2] However, we feel that the old misconception that blurs these two aspects of anesthesia has now been replaced by a new one: namely that analgesia and hypnosis are entirely separate entities without any interrelation. Several observations suggest that this may not be the case. Patients who remain conversant after a low dose of benzodiazepine do fall asleep when given large doses of opioids; similarly, when opioid levels are moderately high, patients will not respond to verbal command even when propofol or sevoflurane concentrations have decreased to levels that, in isolation, would yield only minimal sedation. We are just scratching the surface of the pharmacodynamic interaction between hypnotics and opioids. Fortunately the issue is now receiving considerable attention in well-designed clinical studies. As Dr. Litvan points out, even the "gold standard" assessment of sedation/hypnosis by verbal or tactile stimuli (Observer's Assessment of Alertness/Sedation) is an example of a measurement instrument affecting the measured variable.

Dr. Litvan seems not to be concerned about whether we should measure spontaneous or evoked cortical electrical activity, as long as the electrophysiological variable that is chosen tracks hypnotic level or anesthetic concentration. Recent data from the UK[3] and Belgium[4] suggest that the MLAER reacts in a different way to a changing anesthetic concentration than does the electroencephalogram. This apparent difference in the slope of the concentration-effect curve may have implications for the usefulness of the monitor at hand. Whereas the BIS® variable appears to begin to increase almost immediately after discontinuation of the hypnotic, the MLAER is likely to remain unchanged until just before the transition from unconsciousness to the awake state. Both response patterns may contain information useful to the clinician.

Finally, we agree that the information derived from a dedicated electroencephalogram or MLAER monitor may represent a step forward in helping the anesthesiologist to titrate anesthetics to the desired level. We also agree that if BIS® or AAI guided titration can serve to decrease unnecessary overdosing then both physiologic stresses and wasteful anesthetic consumption might be avoided. However, we remain unconvinced that there is a magic number that clinicians can "ride" that will simultaneously allow a reduction in anesthetic consumption while at the same time reducing the incidence of intraoperative awareness with explicit recall. In fact, we harbor the concern that the practice of "riding the numbers" might actually have the potential to increase the incidence of awareness, if some very specific threshold, *e.g.*, a BIS® of 60, were applied too aggressively.

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In Reply:—We appreciate the comments of Drs. Litvan and Paniagua regarding our article[1] and accompanying editorial.[2] We realize that it is impossible to validate whatever monitor of "anesthetic depth" is used within one study due to the complexity of the phenomenon itself. The hypothesis of our study was based on the concept of anesthetic depth as described by Dr. Glass in a recent editorial in this Journal.[3] He concluded that what is called "general anesthesia" is a process requiring a state of unconsciousness of the brain (produced primarily by the volatile anesthetic or propofol). If only unconsciousness is achieved, a noxious stimulus needs to be inhibited from reaching higher centers (called an arousal reaction). This is achieved by the action of the opiate at opiate receptors within the spinal cord (or, local anesthetics on peripheral nerves, or volatile anesthetics on the spinal cord when administered at concentrations equal to the minimum alveolar concentration (MAC). Previously, I. Kissin[4] already stated that the diversity of pharmacological actions that in combination provide anesthesia make it almost impossible to determine the potency of different actions with one measurement. It has not been our intention to investigate the complete "spectrum of anesthetic depth." Therefore, we only have concluded in our article that both BIS®, AAI and propofol effect-site concentration were accurate indicators for the level of sedation and loss of consciousness (LOC) but poor indicators for predicting response to noxious stimulus. As propofol was given without opiates, a poststimulus arousal reaction was detected at these hypnotic levels too low to block these reactions. Both BIS® and AAI were able to detect these arousal reactions as plotted in figure 9 of our article.

We disagree with Drs. Litvan and Paniagua when they state that the measures of anesthetic depth could have been influenced by the assessments of the OAA/S scale (Observer's Assessment of Alertness/Sedation) and might explain the overlapping values seen in figure 3 of our study. As described in our methodology and validated by others before,[5] all "electronic" measures were and have to be recorded before the assessment of the clinical scores and could not cause bias in the data. Of course, these clinical assessments were responsible for the poststimulus arousal phenomenon.

Regarding the so-called "overlapping values," Drs Litvan and Paniagua might have been misled by the authors of the editorial.[2] Based on figures 3A and B of our original article,[1] the editorial focuses on an overlap between OAA/S level 3 and 0. Although present, this doesn't explain an overlap between consciousness and unconsciousness, because level 0 of the OAA/S scale measures "no reaction to a trapezius squeeze," being a painful stimulus. We are aware of the potential limits of the OAA/S scale. Below level 2, this scale is only based on reaction to painful stimulus. To avoid bias in our methodology, we have defined LOC as the transition between OAA/S levels 3 and 2. It might have been more correct that the authors of the editorial would have compared OAA/S levels 3 and 2 where nearly no overlap is seen. One has also to realize that the box and whisker plots are not showing Gaussian distributions (!). Because of the asymmetry in the data, some overlap in the figures doesn't have to result in an equal amount of overlap in the population. In their editorial, the authors conclude, "many patients with BIS scores between 50 and 60 must have been responsive to voice command or to minimal prodding or shaking." They have not followed our interpretation of LOC because "minimal prodding or shaking" is defined as OAA/S level 2 (being "nonresponsive" or "unconscious"). They also have not interpreted figure 7 of our article, otherwise they should have observed that only two patients had a conscious level at BIS® levels lower than 60 (also observable in table 5). One lost consciousness at a BIS® level of 55 and the last one at 53. As also seen in figure 7, the overlap in values originates more in the unconscious than in the conscious data. This means that these monitors of anesthetic depth lose power in the indication of a too excessive level of hypnosis, making the complete reasoning of this editorial questionable.

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Anesthesia Should Not Be Blamed for All Perioperative Complications

To the Editor:— With great interest, we read the article by Newland *et al.*[1] reporting the incidence of anesthesia-related cardiac arrests in their department over a 10-yr period. Unfortunately, several drawbacks may invalidate the conclusions of this study. We would have liked to know more details on the factors that led to cardiac arrest in each of the fifteen reported cases.

First of all, we do not know who provided the anesthesia. As Biboulet *et al.*[2] point out in their study, human error has been considered the leading factor contributing to anesthesia-related cardiac arrest. It is important to know if an experienced anesthesiologist or only a trainee was involved in these cases.

The following cases illustrate the difficulty in deciding whether a cardiac arrest is caused by anesthesia or not:

Patients 1 and 7

It is inconceivable that administration of an overdose of morphine postoperatively in the PACU may lead to cardiac arrest. The close monitoring available in the PACU should have prevented it. If it occurred because the nurse did not monitor the patient according to normal standards, then it cannot be attributable to anesthesia.

Patient 15

In a 65-yr-old patient, ASA physical status III, 1 mg of midazolam led to cardiac arrest? We are not able to even imagine a mechanism responsible for this complication in an elective patient. Did this patient get another drug instead of midazolam?

Patients 6 and 8

An "unknown" or "probable" vagal reaction should not have been included in this group of patients. Moreover, cardiac arrest occurred in the pediatric intensive care unit (PICU) where many other factors might have been involved.

Patient 14

During the procedure of implantable cardioverter-defibrillator (ICD) placement, there are always times when ventricular fibrillation is induced as a test. Other arrhythmias may occur during the placement of the ICD leads through a central vein. A cardiac arrest resulting from this procedure should be surgery-related and not anesthesia-related.

Patient 11

What led to this postoperative myocardial infarction? Hypotension, tachycardia, hypoxia. . ..? This important information is missing.

The few mentioned examples highlight how difficult it is to attribute a cardiac arrest to anesthesia. A more detailed description of the cases would have given the readers a better notion of the real incidence of this complication.

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In Reply:—We thank Drs. Avidan and Gozal for their interest in our article reporting on anesthetic-related cardiac arrest and its mortality.[1] They are concerned that lack of detail about the fifteen reported cardiac arrests we determined to be attributable to anesthesia may invalidate the conclusion of the study, namely that our results more accurately reflect the risk of perioperative cardiac arrest and the real risk of anesthesia. Our study design was to prepare a case abstract from data obtained from the medical records using a standardized data collection form and submitting this abstract to a study commission. Using this information the study commission then judged whether anesthesia was attributable or contributory to the cardiac arrest. The fifteen cases exemplify the study commission's best judgment. Privacy and other considerations preclude provision of additional case details.

The question of who provided anesthesia care was raised. As noted in our article, anesthesia was provided by faculty, residents, and certified registered nurse anesthetists. It was our practice that an anesthesia faculty member was immediately available and responsible for every case.

To address concerns if a relative overdose of narcotic can lead to cardiac arrest in the PACU or the patient's room, the study commission judged that it could, based on the information available to them, and that the anesthesia provider was responsible or contributory. Neither event was related to nursing care.

In the case of the 65-yr-old patient, ASA physical status III who received 1 mg of midazolam as premedication and went on to cardiac arrest in the ambulatory surgical unit, these events did occur and the patient had not received any additional drugs prior to the arrest. The patient did have a complex medical history and had undergone several major operations in the past.

The two cases attributed to probable vagal reaction occurred with an anesthesia provider in attendance (even in the pediatric intensive care unit shortly after transport from the operating suite) and recovered uneventfully. No other cause could be determined.

Because general anesthesia is used in cases for AICD placement it is possible for dysrhythmias to occur related to anesthesia and before any placement of leads or elective induction of cardiac arrest.

Last, based on the information available to the study commission, it was their judgment that anesthesia was contributory to the perioperative myocardial infarction.

We would encourage other investigators and institutions to do similar studies of their patients receiving anesthesia and report the findings. In the future perhaps a national database, gathering information from each institution, will allow comparison between institutions as well as provide national statistics that are meaningful and accurate.

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Anesthetic Preconditioning: Target the Right Patients

To the Editor:— Perhaps the greatest shortcoming in our specialty is that anesthesia has little or no direct therapeutic benefit for the patients for whom we care. Over the years our specialty has diversified into fields such as critical care, pain management, patient safety, and quality assurance, all of which involve the therapeutic management of patients. Thus, the recent observations that certain anesthetics may possess therapeutic effects[1] is greeted with pleasure by most of us. However, we must remember that these anesthetics (in particular the volatile agents) have been in use for years and that their administration has never been demonstrated to decrease morbidity or mortality.[2,3]

Warltier *et al.* in their editorial[1] suggest that the anecdotal suggestions of a reduced frequency of ischemic events and pharmacological or mechanical support after cardiac surgery is caused by the "relatively greater use of volatile anesthetics" because these agents posses cardioprotective properties.

However, cardiac surgery appears an inappropriate field in which to study this question. Aortic clamping and unclamping are dependent on the surgeon (intentional or unintentional ischemic preconditioning) and the array of cardioplegia cocktails, anterograde, retrograde, continuous, intermittent, cold, warm perfusia, and so on, are but a few of the factors that affect outcome and are difficult to control. Thus, to demonstrate the clinical benefits of anesthetic preconditioning, the study of high-risk surgical patients undergoing noncardiac surgery procedures appears potentially more fruitful.

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In Reply:— We read with considerable interest the letter of Dr. D'Attellis concerning our recent editorial about anesthetic preconditioning (APC)[1]. Importantly, Dr. D'Attellis correctly notes that volatile anesthetics have been used for decades, but have never been shown to reduce morbidity, mortality, or adverse cardiac events, especially in patients undergoing coronary artery bypass graft (CABG) surgery. He emphasizes that many variables affect the extent of ischemia during cardiac surgery, and these factors may confound interpretation of results of studies conducted in this patient population. He further suggests that clinical investigations to assess the impact of APC in high-risk patients undergoing noncardiac surgery may be preferable to studies conducted on patients undergoing CABG.

Dr. D'Attellis' comments certainly have merit. The array of cardioplegia cocktails, the adequacy of myocardial protection, the presence or absence of cardiopulmonary bypass, the duration of aortic cross-clamping, and, of course, the technical success of the surgical anastomoses all represent factors that may diminish the relative impact of APC. This complex picture may represent a potential reason why previous studies of CABG patients have been unable to demonstrate the clear benefits of volatile anesthetics. However, there may be several other reasons as well. The use of sulfonylurea oral hypoglycemic agents for the treatment of adult-onset diabetes mellitus is common in patients with coronary artery disease. These drugs are known to inhibit the ATP-dependent potassium channel, an important component of the signal transduction cascade responsible for APC.[2] Recently, results from our laboratory[3] have also shown that the adequacy of control of blood glucose concentration affects APC. High blood glucose concentrations antagonize APC in the presence and absence of diabetes. To complicate matters further, morphine was often used as an anesthetic adjuvant for patients undergoing CABG surgery before the widespread use of synthetic opioids. Morphine also exerts direct cardioprotective effects that are potentiated by volatile anesthetics.[4] Factors such as these may be controlled in a prospective clinical trial to determine if volatile anesthetics are truly beneficial in patients with coronary artery disease but are difficult to even assess in retrospective studies.

The use of patients with coronary artery disease undergoing noncardiac surgery for future clinical studies of APC in humans certainly deserves consideration, but is also potentially problematic. The perioperative use of ($beta_1$-adrenoceptor antagonists is a new standard of care, and coronary revascularization before other surgical procedures may limit or completely eliminate the frequency and extent of subsequent ischemic events. Thus, the classic "patient with coronary artery disease undergoing noncardiac surgery" population described in older studies is dwindling as a result of aggressive perioperative management of ischemic heart disease. Myocardial protection produced by volatile anesthetics should ideally be assessed by mortality related to cardiac events. Such outcome data would require a large number of patients but such difficulties are not insurmountable. Clinical investigation of APC in a large patient population with or at risk for coronary artery disease should be undertaken.

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Continuous Spinal Anesthesia Redux

To the Editor:— The article by Malinovsky *et al.* was quite interesting.[1] I am writing not because of any criticism of the paper, but to revisit the issues of lidocaine neurotoxicity, "transient radicular irritation (TRI)," and continuous spinal anesthesia.

The portions of Malinovsky's paper that refer to lidocaine show once again that intrathecal 5% lidocaine can be neurotoxic. The neurotoxicity of lidocaine in animals has recently and very nicely been reviewed.[2] There is no question that in some circumstances lidocaine can damage the spinal cord of patients during spinal or epidural anesthesia.[3–8]

More controversial is the TRI that occurs after otherwise uncomplicated single injection lidocaine spinal anesthesia. It is my opinion that TRI is a manifestation of lidocaine neurotoxicity. Others believe that because TRI is self-limited (lasting only a few days) that it is not toxic.[9]

But, Malinovsky *et al.* found in their study that while only two of ten rabbits injected with lidocaine showed "behavioral disturbances," at least four (maybe all, but this is unclear from the paper) of the ten lidocaine-treated rabbits had neural histopathologic changes:

> "Rabbits receiving intrathecal 5% lidocaine presented with signs of local neurotoxicity. Two rabbits presented with areas of loss of myelin or with necrosis in spinal cord and two others presented with axonal degeneration, endoneuronal edema, and perivascular lymphocytosis infiltration in spinal nerves."

This raises the important question of whether something similar is happening with TRI. While TRI patients have no long-lasting symptoms or "behavioral changes," other than pain and dysesthesias for a few days, do they have axonal degeneration, endoneural edema, and perivascular lymphocytosis infiltration like Malinovsky's rabbits?

The reams of publications that followed the reports of cauda equina syndrome associated with continuous spinal anesthesia[4,5] fail to show that spinal microcatheters caused the neural injury, *per se*. In fact, cauda equina syndrome has since been reported after single shot spinal anesthesia[7,8] and accidental intrathecal injection with intended epidural anesthesia.[5,6] The cause is the injection of large amounts or poor distribution of certain neurotoxic local anesthetics (principally lidocaine and tetracaine). Yet, there is still a ban on the use of spinal microcatheters, and the method of continuous spinal anesthesia (with larger catheters) has essentially been eliminated from the anesthesiologist's armamentarium.

This is too bad, because epidural anesthesia is the only alternative. When compared with continuous spinal anesthesia, epidural anesthesia is more difficult to perform, requires the use of much larger doses of local anesthetics and narcotic, which increases the risks for systemic toxicity, and it is less reliable. In fact, nearly 30% of epidural anesthetics used for postoperative analgesia have problems.[10] Furthermore, the target for the drugs injected epidurally is in the intrathecal space. Doesn't it make more sense to make the injection where the receptors are and avoid the barriers that diminish effectiveness?

Continuous spinal anesthesia is a valuable technique. I believe it deserves another chance. The reams of publications that I mention herein demonstrate that continuous spinal anesthesia can be safe. We need only avoid lidocaine and tetracaine, use isobaric solutions whenever possible, and avoid repeated injections when the desired effect is not achieved after injecting an amount that would be sufficient with the single shot method.

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A Preventable Cause of Brachial Plexus Injury

To the Editor:—The article by Coppieters *et al.*, "Positioning in Anesthesiology Toward a Better Understanding of Stretch-Induced Perioperative Neuropathies"[1] was enlightening. We thought the readers of ANESTHESIOLOGY might also be interested in a preventable cause of perioperative brachial plexus injury—operating room (OR) armboard malfunction.

A 66-yr-old, ASA 3, man was scheduled for abdominal-perineal resection. The patient's arm was secured to the armboard with a Velcro strap; the arm was abducted approximately 75°. After general anesthesia was induced in the supine position, he was repositioned for the surgical procedure. During repositioning, when the patient was moved caudally on the OR table, the armboard on which the patient's left arm was secured fell. Even though the incident was witnessed and the patient's arm was immediately supported, the weight of the OR armboard, about 3 kg, transiently pulled on the patient's left arm.

The operation proceeded uneventfully. Upon awakening, however, the patient complained of left arm numbness and weakness. Examination revealed neurologic deficits in the left C5–C7 nerve roots; 0/5 arm flexion, 2/5 arm extension, 2/5 hand grip, and numbness of the fingertips. One month after the event, an EMG/NCV study showed acute denervation of the left C5–6 nerves. With treatment, the patient's left arm sensation and function returned and matched his right arm in 24 months.

Inspection of the OR armboard revealed that the metal bracket which contacted the OR table rail system was damaged, resulting in an insecure connection. Inspection of all armboards in the OR disclosed four damaged armboards. These damaged armboards were removed from the OR.

We recommend that the readers of ANESTHESIOLOGY inspect the OR armboards that they use. When attaching or manipulating an armboard, we suggest that it is a good idea to test the armboard's security by gently leaning on the armboard and attempting to move the armboard out of position, before securing the patient's arm to the armboard.

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A Rash Decision

To the Editor:—A patient admitted to our hospital for a scheduled elective cesarean section had a medical history significant for severe skin reactions to adhesive tapes, causing exfoliating "burn-like" lesions, resulting in permanent skin scarring. Of note, "paper tape" caused her no adverse skin reaction. Further history taking revealed that contact with electrocardiogram electrode pads left her with erythematous "cigar burns" over the entire portion of her skin contacted by the adhesive of the pad; these lesions were painful and took several weeks to resolve.

In an effort to minimize, and possibly eliminate, any adverse skin reaction caused by contact with the electrocardiogram pad adhesive, we removed the paper backing of the electrocardiogram pad (Red Dot Electrocardiogram Electrode, 3 M Health Care Products and Services Division, London, Ontario), cut out a small (approximately 0.3 cm) central circle and reapplied the paper to the pads (fig. 1). These modified electrocardiogram pads were then placed in proper position on the patient and secured in place with paper tape. The resultant electrocardiogram trace showed normal amplitude without any evidence of interference.

This minor adjustment to the electrocardiogram pads allowed for the electrode and conductive gel to be in contact with the patient, while minimizing the adhesive contact with the patient's skin. Postoperative examination revealed no adverse skin reactions.

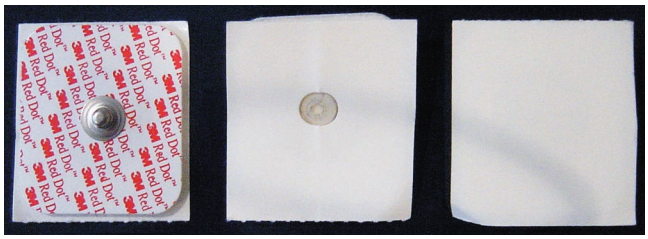

Fig. 1. (*Left*) Back of 3 M Red Dot pad (Red Dot Electrocardiogram Electrode, 3 M Health Care Products and Services Division, London, Ontario); (*center*) Front of 3 M Red Dot pad with hole punch through center of paper adhesive covering to expose only limited gel portion and electrode; (*right*) Front of 3 M Red Dot pad with nonmanipulated paper adhesive covering.

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Buprenorphine Contains Glucose

To the Editor:—We report the unnecessary removal of a correctly inserted epidural catheter. A 47-yr-old man underwent colostomy. After the surgery, a continuous epidural infusion of 0.25% bupivacaine (95 ml), to which buprenorphine 0.5 mg (2.5 ml) and droperidol 2.5 mg (1.0 ml) were added, was started at a rate of 2 ml/h. Before the patient left the postanesthesia care unit to the ward (2 h after the start of epidural infusion), we did an aspiration test, and 2.5 ml of clear fluid was relatively easily aspirated through the catheter. The glucose concentration of the aspirated fluid was 83 mg/dl and the blood glucose concentration was 73 mg/dl. Two hours later, 1.8 ml of clear fluid was again easily aspirated through the catheter. It contained 100 mg/dl of glucose. Although there was no definite motor block of the lower limbs, the epidural catheter was removed because subarachnoid catheter migration could not completely be ruled out due to the high glucose concentration of the fluid. After removing the epidural catheter, we checked the insert packed with the buprenorphine. It stated that this commercial buprenorphine contained "50 mg per ml of glucose." The glucose concentration of the fluid in the infuser we used was calculated to be 127 mg/dl (125 mg of glucose in 98.5 ml). Buprenorphine used in the United States and United Kingdom also contains 50 mg/ml of glucose.

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