At-home Recovery after an Anesthetic

To the Editor—With all due respect to the excellent discussion by Ebert et al.1 of recovery from sevoflurane versus isoflurane and propofol, I believe we are continuing to split hairs over aspects of patients’ early (i.e., in-hospital) recovery period while neglecting meaningful and unanswered questions about the recovery process once patients are at home. The article by Ebert et al. adds to an already very large body of literature focusing on the early postoperative period. To our patients, what happens at home would seem to be of more interest, yet it is still an unproven and barely tested assumption that better recovery parameters while patients are in the hospital might actually reflect advantages later in the recovery process.

We still do not have an understanding of many basic questions regarding at-home recovery. What is the natural course of recovery from anesthesia for most outpatient procedures? When does a patient resume their usual at-home activities? How soon are they able to sustain these activities over the course of the entire day? When is cognitive function good enough to allow return to work, and when is it fully restored to baseline? When is a child able to play and eat normally or to return to the usual school or child-care setting so that a parent might return to either work or customary at-home routines? How frequent is postoperative confusion in the elderly outpatient and how long does it last? Are the answers to these questions different for various intravenous and inhaled anesthetic agents?

I am aware of only one study that investigated some of these questions. In 1991, Sung et al.2 noted that patients who underwent general anesthesia for breast biopsies resumed normal activities sooner (7 h vs. 17 h) and returned to work sooner (1.5 days vs. 2 days) after a propofol infusion and nitrous oxide anesthetic versus a pentothal induction and maintenance with isoflurane and nitrous oxide.

From the perspective of the needs of our patients, their families, and their employers, the aforementioned questions would seem to be at least as relevant as the excellent database available to us on times to emergence, orientation, and recovery room and hospital discharge. Moreover, such data should be of great interest to the manufacturers of the new and expensive anesthetic agents, given the pressures so many of us are facing to prove that we are providing “cost-effective care.” In fact, after investigation of at-home recovery, we might find that we have a new and more compelling rationale to support even more widespread use of the short-acting agents; at the very least, this seems plausible enough to deserve further investigation.

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References


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To the Editor.—We read with interest the case report by Chee and Benumof1 and their comments on the advisability of removal of the burned endotracheal tube.

The decision of whether to extubate a particular patient after a fire in an endotracheal tube must include consideration of the risk/benefit ratio. The danger of extubating a patient when reestablishment of the airway is judged to be difficult is considerable. But the risk of commitment to a tube already involved in a fire is also considerable. Before reventilation of the patient, it is absolutely imperative first to assure that the intraluminal flame2 has been totally extinguished. If not, a severe intraluminal and free-end flame can ignite when oxygen is resupplied, and further patient damage will occur. To evaluate the risk of not removing an endotracheal tube involved in a fire, one must also consider the effect of these flames on the integrity of the tube.

Because polyvinyl chloride endotracheal tubes require an oxygen-enriched atmosphere to sustain combustion,3 an extraluminal surface fire can exist when the extraluminal surface is exposed to an oxygen-enriched atmosphere.

An intraluminal fire can exist when the intraluminal surface is exposed to an oxygen-enriched atmosphere. If the tube is ignited while oxygen flows through the tube, the intraluminal fire that develops spreads toward the oxygen flow.

The products of complete oxidation of polyvinyl chloride produced by the intraluminal flame include carbon dioxide, water, and hydrogen chloride. Because oxidation is often incomplete, products of incomplete oxidation are also produced, including carbon monoxide and hydrogen. These products are present in the gases flowing downstream from the intraluminal flame. Notably, because oxidation is often complete, the downstream gases contain no oxygen. Also included in the downstream gases are products of pyrolysis of polyvinyl chloride, such as short and long carbon chains and carbon rings. Some of the downstream gases are capable of further oxidation and can ignite on reaching an oxidizer such as ambient or alveolar air, producing a free-end flame.

If the intraluminal oxygen available exceeds the fuel supply of polyvinyl chloride, or conversely, if the available fuel supply is less than the available oxygen, the intraluminal flame becomes anchored at the distal end of the tube.

The true significance of the hypothesis of the sparing effect on the lung by the “venting” of the flame via the tracheostomy stoma is speculative and requires further investigation.

In addition, safe clinical practice dictates against the concomitant use of an oxygen-enriched atmosphere and the proximate use of the electrosurgical unit in the presence of a polyvinyl chloride endotracheal tube. It must be reinforced that nitrous oxide contributes to the oxygen-enriched atmospheres.

Although our studies demonstrate that the intraluminal flame will extinguish on cessation of intraluminal gas flow, if the decision is made that the risk of extubating is greater than the benefit of not extubating, we strongly support lavage of the intraluminal surface with sufficient water or saline. The sufficient amount will vary with the circumstances. However, to be absolutely certain, direct visual inspection is probably necessary to assure that all potential reignition points are extinguished. Because that is impractical and uncertain, it is advisable initially to reventilate with air. Reventilation with oxygen may rekindle an intraluminal and free-end flame from possible nascent smoldering combustion.

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Coupling of Local Cerebral Blood Flow to Local Cerebral Glucose Utilization during Isoflurane and Sevoflurane Anesthesia

To the Editor—In the December 1998 issue of ANESTHESIOLOGY, Lenz et al.1 reported that close coupling between local cerebral blood flow (LCBF) and glucose utilization (LCGU) is preserved in animals anesthetized with 1 minimum alveolar concentration (MAC) of isoflurane or sevoflurane (fig. 2).1 This conclusion seems to have been based on linear regression of the mean values of LCBF against LCGU for each of the regions examined and evaluation of the derived correlation coefficients (fig. 2).1

Regression analysis of LCBF and LCGU values derived from autoradiographic experiments has been criticized on methodologic and statistical grounds.2 "Blood flow–metabolism coupling" conventionally refers to changes in blood flow within a brain region in response to changes in metabolism in that region.3,4 Evaluation of LCBF in relation to LCGU in many different brain regions under one particular set of conditions does not fit this concept.2

Table 1. LCBF/LCGU Ratios during Isoflurane and Sevoflurane Anesthesia

<table>
<thead>
<tr>
<th>Condition</th>
<th>Mean LCGU (μmol · 100 g⁻¹ · min⁻¹)</th>
<th>Mean LCBF (ml · 100 g⁻¹ · min⁻¹)</th>
<th>LCBF/LCGU</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conscious</td>
<td>64 ± 23</td>
<td>106 ± 38</td>
<td>1.7 ± 0.2</td>
</tr>
<tr>
<td>Isoflurane 1 MAC</td>
<td>38 ± 16</td>
<td>130 ± 44</td>
<td>3.7 ± 0.9†</td>
</tr>
<tr>
<td>Isoflurane 2 MAC</td>
<td>33 ± 21</td>
<td>176 ± 70</td>
<td>6.3 ± 2.0†</td>
</tr>
<tr>
<td>Sevoflurane 1 MAC</td>
<td>44 ± 18</td>
<td>119 ± 50</td>
<td>2.8 ± 0.6†</td>
</tr>
<tr>
<td>Sevoflurane 2 MAC</td>
<td>33 ± 23</td>
<td>141 ± 64</td>
<td>5.0 ± 1.7†</td>
</tr>
</tbody>
</table>

Mean values (± SD) of LCGU μmol · 100 g⁻¹ · min⁻¹ and LCBF ml · 100 g⁻¹ · min⁻¹ for the 40 brain structures listed in tables 2 and 3.1 Kruskal-Wallis analysis was performed on LCBF/LCGU ratios (H = 143, df = 4, P < 0.001) with post hoc Student-Neuman-Keuls test to identify individual differences.

LCGU = local cerebral glucose utilization; LCBF = local cerebral blood flow; MAC = minimum alveolar concentration.

* P < 0.05 versus control values.
† P < 0.05 versus lower concentrations of the same anesthetic and equivalent concentrations of the alternate anesthetic.

Regression analysis of LCBF on LCGU examines the homogeneity of the ratio of LCBF to LCGU among the various brain regions examined.2

The results of the study by Lenz et al.1 suggest that isoflurane and sevoflurane alter the LCBF/LCGU ratio. We compared the LCBF: LCGU ratios for the 40 structures studied in the five conditions from their data (tables 1 2 and 3). Isoflurane and sevoflurane both increased the mean ratio of LCBF/LCGU in a dose-dependent fashion, with isoflurane producing the most marked effect (table 1). This analysis remains open to the statistical criticism2 that the variability between animals was eliminated by using mean values of LCBF and LCGU, thereby understimating the real uncertainty in the LCBF: LCGU relation.

The experimental design used by Lenz et al.1 does permit exploration of blood flow–metabolism coupling through analysis of the relation between LCBF and LCGU within specific brain regions as the LCGU is depressed by increasing concentrations of anesthetic. Figure 1 (based on data1 from their tables 2 and 3) shows the LCBF response to increasing anesthetic concentrations in two brain regions, with similar mean values for LCGU and LCBF in the conscious animals. The data suggest that LCBF in these two brain regions responds differently to increasing concentrations of isoflurane and sevoflurane, because, despite similar decreases in LCGU with increasing MAC multiples, LCBF appears to decrease in auditory cortex but to increase in the inferior colliculus. The analysis of these data2 could not be attempted with the information provided in the original report.1

In summary, further analysis of the data reported by Lenz et al.1 supports previous observations that inhalational anesthetics increase the ratio of mean cerebral blood flow/cerebral metabolic rate for oxygen (CBF/CMRO2) in a dose-dependent fashion.1 Inspection of the data for individual brain regions suggests that a detailed analysis2 of flow–metabolism coupling may reveal significant regional differences.
In Reply—We thank Drs. Archer and Pappius for their critical comments about our article. They raise essentially three points, which we are happy to comment on.

Coupling of Blood Flow to Metabolism

Archer and Pappius correctly state that “blood flow–metabolism coupling conventionally refers to changes in blood flow within a brain region in response to changes in metabolism in that region.” This is certainly a definition that is generally accepted. However, it is rather common to use the term coupling also in a broader sense to describe the long-term adjustment of local cerebral blood flow to the local metabolic rate for each brain structure.1

Statistical Methods

Archer and Pappius propose a statistical analysis for specific brain regions and make specific statements concerning auditory cortex and inferior colliculus. In light of the criticism specified in the last paragraph (Statistical Methods) we would hesitate to definitely come to such a specific conclusion about discrepant trends in different structures as raised by Archer and Pappius. We believe that such conclusions are heavily dependent on the kind of statistical analysis used and therefore may not be unequivocal. In spite of the existence of different methods of statistical analysis, we believe that none of them can be used without raising some criticism when multiple data of local blood flow and metabolism are compared during different anesthetic conditions.

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In conclusion, we performed a detailed statistical analysis of our data. However, we are not sure about the real impact of hundreds of comparisons and therefore followed the suggestion to omit the statistical part.

We are grateful to Drs. Archer and Pappius for giving us the opportunity to clarify several important aspects of our work.

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Intramuscular Opioid Injections: A Step in the Wrong Direction

To the Editor—We refer to the recent study by Choinière et al.,1 which contrasted the efficacy and costs of patient-controlled analgesia (PCA) with regularly administered intramuscular (IM) opioid therapy. The conclusion that PCA is more costly and does not have clinical advantages for pain management after hysterectomy deserves comment. The limitations of on-demand nurse-administered IM opioid therapy as a method of controlling postoperative pain are well recognized.2 PCA was introduced into clinical practice in the early 1980s as a means of overcoming these limitations. Personal control, rapid onset of pain relief, and timely effective analgesic therapy at the bedside are important aspects of PCA use.3 The technique adjusts for interpatient and intrapatient variations in opioid requirements. Today, many consider PCA therapy the “gold standard” of parenteral opioid administration for the control of postoperative pain. Consequently, alternative techniques of opioid administration must at least demonstrate comparative efficacy to PCA use.4 In our experience, a large majority of patients who have had a chance to compare IM injections and PCA prefer the latter. Unfortunately, patients in this study received only one form of treatment. Allowing patients to compare both techniques by a crossover design might have shown higher satisfaction with PCA. Furthermore, many patients report significant discomfort from repeated IM injections in the buttock area.4 Patients administered IM opioids in this study received at least 12 injections. Of interest, patients who received scheduled IM morphine required significantly more morphine in a 48-h period (132 ± 37 vs. 93 ± 50 mg; P < 0.0001) to obtain equivalent pain relief, significantly more rescue doses (30% vs. 0%), and significantly more morphine dose adjustments (63% vs. 15%; P < 0.0001). Given that both patient groups had similar outcomes in terms of analgesic efficacy, it should be noted that the IM patients received more morphine, required more rescue doses and dose adjustments, and consumed more nursing time than the PCA patients. This is perhaps a testament to the individualization, ease of administration, and overall success of PCA therapy. Although pain scores were equal in both groups, pain on movement was not measured during the first 24 h of this study. If it had been, a difference between the two techniques might have been observed. The assessment of efficacy of an analgesic technique should also include a measure of convenience of that technique to both patient and staff. The authors failed to comment on the ease of applicability of the IM regimen and on how successful the nursing staff was with regard to administering drug on time and dealing with apparently frequent problems of inadequate pain control in the IM group, particularly during the first 24 h postoperatively. In the somewhat artificial environment of a study, it may be possible to administer timely and appropriate IM injections. However, previous clinical experience indicates that this is a major issue in providing adequate postoperative pain relief and results in significant patient dissatisfaction.5 Nurse-administered IM opioid injections require adequate staffing levels to minimize delay between request and injection. In this climate of ever-shrinking health care dollars, we question whether there are sufficient numbers of nursing staff to follow this proposed IM protocol. Indeed, in many hospitals in the United States, the move seems to be toward providing higher numbers of less-skilled workers (medical assistants and licensed practical nurses rather than registered nurses) to care for patients in the postoperative setting. The burden imposed on the nursing staff in determining the

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success of such an IM regimen is not defined in this study. The saying "if it isn’t broken don’t fix it" may well apply to this study. IM injection of opioids has had its day and failed. Let us not return to the dark days of postoperative pain management without just cause or reason.

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In Reply.—We appreciate the comments of Dr. Fitzgibbon et al. regarding our study, which shows that regularly nurse-administered intramuscular (IM) injections of morphine are less costly and provide comparable analgesia to patient-controlled analgesia (PCA) after hysterec¬omy.1 We welcome the opportunity to elaborate on important issues that we could not address in our article because of space limitations. We want to emphasize from the outset that we did not propose to "return to the dark days of postoperative pain management." Similarly, their concluding remark, that “IM injections of opioids has had its day and failed miserably," applies solely to on-demand scheduling of administration, which we believe is irrelevant to our findings. In our study, we purposely chose not to compare PCA with on-demand IM injections precisely because of the well-recognized inadequacies of this mode of administration.2,3 Instead, we chose to assess the comparative benefits of PCA versus regular IM dosing in terms of efficacy and cost.

Our results showed that the two techniques were equally effective, but the patients on IM therapy received more morphine, required a greater number of dose adjustments, and consumed more nursing time. Fitzgibbon et al. argue that this is perhaps a testament to the individualization, ease of administration, and overall success of PCA. They are right in a way, but they omit several important points, including the cost of the technique, a crucial element to consider in these days of increasing budget constraints in our healthcare system. Our study is the first to provide a thorough cost analysis of PCA. This analysis showed that PCA was more costly than regularly nurse-administered IM injections, although the cost for the pumps themselves were counted as zero. Considering that the two techniques were equally efficacious, one must ask whether one wants to pay extra for something that is no better.

Patient satisfaction is obviously an important aspect to consider in such a context. According to Fitzgibbon et al., if we had used a crossover design so that our patients had the chance to compare the two techniques, they might have preferred PCA. They are probably right. IM injections are indeed painful, but it is common clinical practice to rotate the injection site to different areas (buttocks, arms, thighs) to minimize patient discomfort. Our suggestion,4 to replace the IM injections by subcutaneous injections administered through a winged infusion set, is an even better solution that probably would enhance patient satisfaction. But even if patients persist in preferring PCA to nurse-administered subcutaneous injections, two issues still must be considered. First, the preference for PCA should be of such a magnitude that it overwhelms the cost advantage of IM therapy (mild preference in the face of important cost disparities is not a compelling argument). Based on previous observations,5–6 this seems doubtful, but further research is clearly needed in this field.7 Patient satisfaction ratings are usually very high irrespective of the analgesic regimen used, and it is by no means clear what such ratings represent.8–10 As pointed out by Egen and Ready,8 patient satisfaction with postoperative analgesic care is a complex issue. It encompasses and reflects many factors such as personal preference, patient expectation about pain relief, communication with healthcare providers, perceived compassion, etc.8–10 Progress in this area requires more studies on the sources of patient satisfaction (or dissatisfaction) with PCA and other types of analgesic regimens. However, these satisfaction ratings should not be used in isolation from other data such as postoperative pain scores and medication intake because they could lead to erroneous conclusions about the quality of pain management.7

A second and perhaps more important issue is that patient preference for a given analgesic method is far from being a guarantee of its efficacy. Several studies9,10 have shown that patients reported being highly satisfied with their analgesic treatment despite the fact that they experienced relatively high pain levels. Patient satisfaction does not equate to pain relief. In the PCA literature, several reports11–15 indicate that patients who use this technique rarely medicate themselves to optimal or complete pain relief. Reasons are numerous and include fear of experiencing drug side effects, concern about drug addiction, educational attitudes toward pain, and religious beliefs. Nevertheless, these same patients report being highly satisfied with the “machine” and, paradoxically, some of them are even proud of saying that they

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did not use it very much. In our study, PCA patients consumed less morphine than those who received IM therapy, and it is interesting to note that the mean VAS scores on all of the measures assessing pain at rest and with movement were slightly but consistently higher in the PCA group, even during the first 24 h of the study. Results on the pain relief measure and the recovery parameters also tended to not favor PCA therapy (tables 3 and 4 in our original article). The group differences reached the fixed level of statistical significance on one of the measures where the PCA patients were found to take significantly longer to be able to sit in a chair without assistance. A tendency also emerged for them to report more pain when walking than IM patients (the difference was significant at 0.03 but did not meet the Bonferroni’s corrected alpha level).† These observations are interesting and we were tempted to mention them in our article, but we wanted to avoid any suggestion that the group differences were real differences (as defined by achieving statistical significance). However, the best estimates of the differences did not favor PCA therapy.

With regard to the ease of applicability of the regular IM regimen, we did not collect any measures, although it would have been interesting to do so. However, administering the drug on time did not seem to be a problem. Rescue dose and dosage adjustments were time-consuming, but the procedure was made easier by the use of a standardized protocol for increasing or decreasing the medication, which was part of the prescription order included in patients’ medical files.

In the real-life situation (as opposed to the somewhat artificial environment of a study), regular IM (or subcutaneous) dosing can probably be made easier (and less nurse-intensive and consequently cheaper yet) by synchronizing the injections with other fixed duties (e.g., vital signs), which was not the case during the course of our study. PCA certainly requires less nursing time, but it does not guarantee adequate pain control and its expense overwhelms any nursing cost disadvantages of regular IM injections. Dr. Reedy himself has suggested elsewhere† that proper PCA use is not without its own share of nursing time commitment: “There is a widespread misconception that pain relief with PCA is completely automatic. In fact, PCA can only be used optimally when it is accompanied by regular, expert nursing and medical supervision.” So, the argument presented in the letter that hospitals will eventually move toward providing less-skilled workers to care for postoperative patients (supposedly to the sole detriment of the regular IM injections regimen) also holds for PCA.

In conclusion, all of us are in search of better pain management, not merely pushing favorite methods in the face of significant contrary evidence. PCA has its advantages and disadvantages. The same is true for regularly nurse-administered analgesia. However, it is incorrect to argue that regular IM injections of opioids for controlling postoperative pain are “a step in the wrong direction.” Their use is a step in a different direction that provides value commensurate with resource outlays. Until such time as PCA is able prove itself to have such a superiority in patient preference (including strength of preference, not just direction of preference) that it overwhelms its cost and perhaps efficacy disadvantages, it will seem (at least in this indication) to be a high-tech solution where none is needed. Such solutions are often falsely attractive.

**References**


**CORRESPONDENCE**
To the Editor—Here we report on a malfunction of the volume monitor sensor on an Ohmeda Modulus CD series anesthesia machine. This failure occurred after the overhead surgical lights were directed toward the sensor.

A 29-yr-old black man was scheduled for a cystoscopy, ureteral stent placement, and lymphocele drainage approximately 3 months after he underwent a cadaveric renal transplant. After satisfactory induction of anesthesia and tracheal intubation, the patient was mechanically ventilated with 50% nitrous oxide/oxygen and 3% desflurane. Both an Ohmeda 7850 ventilator and an Ohmeda modulus CD series anesthesia machine were used (Datex-Ohmeda, Madison, WI). The patient was then placed in the lithotomy position. Bilateral breath sounds were confirmed by auscultation, end-tidal carbon dioxide reading was noted to be 35 mmHg, and the endotracheal tube cuff was palpated in the sternal notch. The SpO₂ was 97%, the tidal volume was noted to be 700 ml, respiratory rate was 10/min, and the peak inspiratory pressure was 20 cm H₂O, both before and after positioning.

After preparing the patient, the surgical lights (Skytron [IN3022EC], Grand Rapids, MI) at high intensity (5 on a scale of 1–5) were directed toward the head of the operating room table and anesthesia machine, away from the operative site. Within 1 min the spirometer of the anesthesia machine indicated “apnea volume” with no tidal volume or respiratory rate indicated on the monitor. Auscultation of the chest showed clear bilateral breath sounds, and an end-tidal carbon dioxide reading of 34 mmHg with a normal capnogram was noted. The chest was rising symmetrically, the peak inspiratory pressures were unchanged at 20 cm H₂O, the SpO₂ was 97%, and water condensation was noted in the endotracheal tube. The volume monitor vanes were rotating. Because the tidal volume monitor operated partially on optical readings, we thought perhaps that the overhead light shining on the volume monitor of the anesthesia machine was distorting the reading, much like fluorescent lights interfere with pulse oximeters.1–3 Within 1 min after redirecting the overhead lights, the spirometer recorded our initial tidal volume and respiratory rate readings.

Discussion

Volume measurements, including tidal volume, expired minute volume, and respiratory rate, are measured by both an optical and mechanical sensor placed on the expiratory limb of the breathing circuit. In the Ohmeda Modulus CD, a series of three vanes located within a transparent cartridge constitute the mechanical portion of the volume detector. Two of the vanes are stationary and are positioned before and after a rotating vane. The stationary vanes are shaped much like six-spoked wheels. As gas moves through the cartridge, the stationary vanes channel the gas to move the middle rotating vane. A pair of optical sensors on a plastic clip, each of which consists of an infrared light emitting diode and photosensitive detector, converts the motion of the rotating vane into electrical signals. As the vane in the transparent cartridge spins, it momentarily blocks the path of the infrared light traveling to the optical detector. Each time the vanes pass an optical detector, an electrical pulse is sensed by the monitor’s microprocessor. The microprocessor counts the pulses to determine the gas flow direction, volume, and respiratory rate.4

The orientation of the photodetector on the transparent cartridge is crucial for this described malfunction to occur. During this incident, the open part of the clip was directed upward with a greater exposure to the surgical light. The spirometer failure will not occur with the photodetectors directed downward toward the floor. Unfortunately, this is not the “neutral” position the sensor takes because of traction from the attached electrical cord. In addition, the intensity of the overhead surgical lights is important. In this report, the lights were set at the highest intensity. The malfunction did not occur when the lights were dimmed to a lower intensity (< 2 on a scale of 1–5).

Our observation demonstrates that intense lighting, such as overhead surgical lighting, can affect the Ohmeda Modulus CD series volume monitor sensor and lead to erroneous data. No warnings were noted in the Ohmeda Modulus CD series manual regarding this possible malfunction. Such failures are especially likely to occur more frequently as video-assisted surgical procedures become more commonplace and the overhead operating light(s) are directed toward the head of the table to provide the anesthesiologist’s otherwise dark work area with light. This reported failure occurs with this design of spirometry sensor (Ohmeda 7850 ventilator); it would not occur with the newer models of Ohmeda spirometry (e.g., the 7900 ventilator).

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An Unusual Case of Epidural Catheter Obstruction

To the Editor:—A 48-year-old man presented for a colostomy take-down. His medical history was significant for Crohn’s disease, necessitating colon resection and colostomy. Combined epidural–general anesthesia was planned.

After achieving intravenous access, localization of the epidural space was achieved via the L1–L2 interspace with the patient in the sitting position. A Perfix Continuous Anesthesia kit was used (B. Braun Medical Inc., Bethlehem, PA). A Tuohy–Schliff epidural needle (18-gauge × 10 cm) was placed in the epidural space without difficulty using the loss-of-resistance-to-injection-air technique. A radiopaque polyamide epidural catheter was inserted through the epidural needle. After the removal of the epidural needle over the catheter, a screw-cap connector was fixed to the distal end of the epidural catheter in the usual fashion.

It was then observed that injection of the test dose via the catheter was impossible. Incremental withdrawal of the catheter did not correct this situation. The epidural catheter was eventually withdrawn completely. Subsequent attempts to flush the catheter proved futile. Close scrutiny of the epidural catheter assembly unit showed that the screw-cap connector revealed a complete absence of the lumen within it (fig. 1). It is interesting to note that a simple naked-eye examination of the epidural screw-cap connector would have been sufficient to avoid the need for a second attempt at epidural catheterization. This coupled with an ‘injection test’ of the epidural catheter and epidural screw-cap connector assembly would eliminate several mishaps of this nature. Support was provided solely from institutional and/or departmental sources.

Support was provided solely from institutional and/or departmental sources.

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Difficult or impossible injection via the epidural catheter can be a result of several causes, resulting in mechanical obstruction of the epidural catheter at various levels. Apart from accidental kinking, knotting, axial torsion, and malposition of the catheter, occasional manufacturing defects of the catheter (e.g., catheter without terminal helical “eyes”) can lead to this problem. As far as we are aware, this is the first report of such a manufacturing defect of the screw-cap connector.

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References

In Reply:—Recently, we were notified that an article entitled, “An unusual case of catheter obstruction” was submitted for publication in ANESTHESIOLOGY. The article was written by Dr. Dharmender Chandhok of Beth Israel Deaconess Medical Center, who experienced some difficulties with one of our epidural catheter connectors. Our subsequent examination of the returned product confirmed that the inner lumen of the connector was occluded because of an isolated molding anomaly.

We are dedicated to providing our customers with products of the highest quality and continuously monitor incident reports to ensure that we can quickly isolate and correct any problems that may occur. B. Braun Medical, Inc. has been manufacturing this product for more than 15 yr, with more than 4 million catheter connectors produced annually. To date, we have never received a report of this nature for this product. This is also supported by the author, who concurs that he has not previously encountered this condition. We would agree with Dr. Chandhok that his difficulties may have been avoided by a cursory visual examination of the product and sincerely appreciate his bringing this matter to our attention.

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Fig. 1. Screw cap connectors, (left) without lumen, (right) normal lumen, as indicated by the arrows.
Simple Modification of the Ovassapian Fiberoptic Intubating Airway

To the Editor—For orotracheal fiberoptic intubation, an Ovassapian fiberoptic intubating airway has been used to provide an open oropharyngeal space and to introduce a fiberoptic bronchoscope at the midline of the oropharynx. When using this device with proper application of the jaw-thrust maneuver and extension of the head and neck, laryngeal exposure is usually easy, even in anesthetized, paralyzed patients. However, in some patients (e.g., patients with obesity or with limitations of head and neck extension), the space between the pharyngeal surface of the intubating airway and the soft palate is narrow, despite performance of an adequate jaw-thrust maneuver by an experienced assistant. In these cases, a fiberoptic view is obstructed and identification of the midline is difficult.

We pasted a black line on the midline of the pharyngeal surface of the airway (fig. 1). This line facilitates identification of the midline and advancement of the fibroscope along the midline when the space between the intubating airway and the soft palate is narrow (fig. 2). We have used this modified intubating airway in more than 50 adult paralyzed patients and believe that it is valuable for trainees and instructors in teaching fiberoptic intubation. We believe that this black line is helpful for experienced endoscopists, especially in patients with morbid obesity or in those with limited head and neck extension.

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Fig. 1. An Ovassapian airway with a black line pasted on the midline of the pharyngeal surface.

Fig. 2. Fiberoptic view in an obese patient. The space between the pharyngeal surface of the Ovassapian airway (OA) and the soft palate (SP) is narrow, despite adequate performance of the jaw-thrust maneuver and extension of the head and neck by an experienced assistant. A black line on the airway facilitates identification of the midline. An arrow indicates the base of the uvula.

References

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Crimping of a Laser Tube Resulting in Hypoxemia

To the Editor:—Laryngeal surgery involving a laser often necessitates the use of special endotracheal tubes (ETTs) to avoid an airway fire.1,2 Many companies, including Rüsch (Duluth, GA), manufacture these tubes. We describe an anesthetic complication with a laser ETT constructed from rubber, wrapped with foil, and overwrapped with fabric.3

The patient was a 90-kg, 170-cm, 65-yr-old male smoker who was scheduled for direct laryngoscopy and vocal cord laser therapy. After uneventful induction of general anesthesia and paralysis, the patient was intubated via direct laryngoscopy with a 6.0-mm ID Rüsch Laser-tubus (lot #CE0124) without difficulty. The patient had bilateral clear breath sounds, arterial oxygen saturation was 100%, and end-tidal CO2 was demonstrated. The ETT was secured with 1-inch silk tape at 23 cm at the lips. The operating room table was then turned away from the anesthesia machine, and the surgeons extended the neck and head to position the patient for the surgical procedure. Within 30 s, the peak inspiratory pressures began to increase, and both the ETCO2 and SpO2 started to decrease. No devices had been inserted into the patient’s mouth, and there was no tension on the circuit or ETT. The patient’s head was returned to the neutral position, but bilateral auscultation showed minimal to no breath sounds bilaterally. The patient was hand-ventilated with 100% oxygen, and the sevoflurane was increased to 8%. SpO2 decreased to 70%, and no ET CO2 was evident by capnography. Immediate direct laryngoscopy confirmed correct ETT placement. Because inspection of the ETT showed no kinks or obvious obstructions, working diagnoses included the presence of either bronchospasm or a mucus plug in the tube. The patient received an inhaled β-agonist and subcutaneous terbutaline without improvement. We attempted to pass a suction catheter through the tube but met resistance at approximately 20 cm. Because initial intubation was easy and the patient was not responding to medical therapy, we decided to exchange the ETT for a PVC Portex (Keane, New Hampshire) Softseal 7.0-mm ID (nonlaser tube), which was inserted to 23 cm. Ventilation was achieved, and within 10 breaths, the patient’s SpO2 returned to 100%. Surgery proceeded, and it was decided that the laser portion of the operation was not necessary. Emergence and extubation were uneventful.

We inspected the Lasertubus and found the tube crimped under the tape (fig. 1). Although this was only a small defect not obvious to cursory inspection, it resulted in complete obstruction to airflow when tested with the breathing circuit. Experimenting with unused tubes showed that after bending the Lasertubus, a weakness within the wall of the tube remained, predisposing to crimping with minimal force and complete obstruction to airflow. All anesthesia providers need to be aware of this potential complication.

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To the Editor.—During a preoperative interview, it is not unusual for a patient to list in their drug history a medication by a trade name that is unfamiliar to an anesthesiologist. A reasonable response to this situation is often to review the package insert of the drug in question. Unfortunately, these inserts do not undergo periodic review and, therefore, it is not uncommon to read outdated, questionable, or even incorrect information for older drugs, especially if they are beyond patent protection. On the other hand, newer drugs should be expected to be associated with more current and thorough information. I recently read the product information for Coreg (carvedilol; SmithKline Beecham, Philadelphia, PA), which was presented in an advertisement that appeared in JAMA (volume 280, number 18, November 1998). The indication for Coreg is “hypertension and mild or moderate heart failure NYHA II or III,” and it seemed to me that this might represent an area of possible anesthetic interaction. The manufacturer apparently agreed and added a specific caution concerning “anesthetic agents that depress myocardial function, such as ether, cyclopropane and trichloroethylene.” I was struck by the combination of a relatively new drug (1995) with anesthetist agents no longer in use in the United States. This prompted me to search, via the website www.pdr.net/physician, for the Physicians Desk Reference (Medical Economics, Montvale, NJ), for mention of any of these three anesthetics in this compendium of package inserts. The response rates were 5 for trichloroethylene, 13 for cyclopropane, and 156 for ether. It was also possible to find basically identical wording from the Coreg advertisement used with another drug from another company.

My inferences from this are several: (1) despite the general caution, there are no reliable data concerning interactions between Coreg and currently used inhalation anesthetics, otherwise they would have been mentioned; (2) a significant number of inserts, once created, seem to remain frozen in time; and (3) the oversight of package insert accuracy seems lacking.

Until every operating suite has access to near-instantaneous electronic data retrieval, package inserts will remain the primary quickly available preoperative source of information about drugs. In fact, even after such time, most on-line references will inevitably be largely based on manufacturer-supplied data. This is especially true for recently released drugs, and although novel or recent data have next-day availability electronically, it can remain no less difficult to erase erroneous original information, no matter how it is presented.

Therefore, I strongly request that both manufacturers and the Food and Drug Administration exert increased efforts to ensure that package insert information is timely, relevant, and accurate. I would also suggest that the addition of phrases suggesting generally increased concern or caution should be discouraged unless adequately supported by experience. Such additions are well-meaning but misguided and may have unintended consequences. Conversely, when such experience has been obtained, especially after marketing, the insert requires prompt revision.

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To the Editor.—Various methods have been used to detect the onset of lumbar epidural blockade (LEB). A predictor of successful LEB not requiring any direct communication with the patient would be particularly useful. One possibility is the use of a plethysmographic pulse oximeter. Photoelectric digital plethysmography has been shown to detect successful sympathetic block, as evidenced by an increase in pulse amplitude resulting from an increased blood flow to the treated limb.1,2 We, therefore, investigated whether a pulse oximeter could be used to detect successful epidural blockade.

Eighty-eight adult patients, American Society of Anesthesiologists physical status I or II, aged 22–51 yr, weighing 57 ± 14 kg (mean ± SD), of both sexes undergoing lower abdominal procedures during epidural anesthesia (alone or in combination with general anesthesia) were prospectively studied. The protocol was approved by our institutional ethical committee for human research and informed consent was obtained from all patients. Patients with a history of hypertension, diabetes mellitus, or peripheral vascular disease were excluded from the study.

After arrival in operating areas, axillary temperature was measured and two pulse oximeter probes were attached to the big toes. Oximetry was recorded on a Siemens Sirecust 1261 monitor (Siemens Medical Electronics, Inc.), which incorporates plethysmographic pulse oximeter waveform (PPWF) monitoring in its display. Amplitude of the waveform was proportional to the analog signal, not scaled. An epidural catheter (Perifix LOR, B. Braun Melsungen AG, Germany) was inserted at the L2–L3 lumbar interspace and advanced 3 cm cephalad. With the patient supine, a mixture of 10 ml bupivacaine, 0.5% (ASTRA-IDL, Bangalore, India), and 5
ml lidocaine, 2% (ASTRA-IDL), was injected into the epidural space. Maximum height of the PPWF displayed on the two different monitors were measured separately with the help of calipers and a measuring scale before administration of epidural drug mixture (a mixture of lidocaine and bupivacaine; baseline value) and then at 5, 10, 15, 20, 25, and 30 min after epidural drug administration. Simultaneously, the sensory blockade was also tested using the loss-of-cold-sensation test by applying ice cubes from the thoracic to the sacral dermatomes bilaterally at 5-min intervals until 30 min after administration of the epidural drug mixture. The operative procedure began after 30 min of epidural drug administration with or without supplemental general anesthesia. Patients were kept warm throughout. Any change in the height of PPWF from baseline value was computed as a percent of baseline.

Epidural blockade was successful in 82 of 88 patients and failed in 4. In the four patients without evidence of blockade, we saw no change in amplitude. In the 82 with good blocks, we saw a rapid increase in amplitude (fig. 1). In one patient, failure of the pulse oximeter prevented study. In another patient, one limb showed significant increase in the PPWF, whereas in other limbs no such change in the height of PPWF was observed, and subsequent sensory test (loss of cold sensation) confirmed a unilateral LEB.

Discussion

The study shows an increase (also visually appreciable) in the amplitude of PPWF after successful LEB. This correlated well with sensory examination (loss of cold sensation). Further, this method detected the onset of LEB earlier than the usual sensory test (via loss of cold sensation).

Different sensory examinations to confirm a successful LEB have been described. However, they can not be used in situations in which verbal communication with the patient is difficult. Our method could be useful in such situations. Further, because the amplitude changes are so large, our method may be easier to use than the manual assessment of peripheral temperatures, as described by Asato et al. Laser Doppler flowmetry has been used to detect epidural blockade. However, apart from its high cost, it is not widely available in the operating room, and its use and interpretation require more skill to learn. The widespread availability of the pulse oximeter in the operating room and its dependence on pulsatile blood flow makes it a simple and safe tool that is easy to interpret.

At 10 min after epidural injection, an amplitude increase of 200% was seen in 79 of 82 patients who underwent successful epidural anesthesia. In contrast, loss of cold sensation at this point was noted in only 14 of 82 patients. Because the sympathetic fibers are first to be blocked after epidural anesthesia, the plethysmographic pulse oximeter helps in early detection of onset of LEB, saving precious operating room time in addition to ensuring pain relief. Even failed blockade could be detected early, allowing early institution of appropriate management, such as catheter readjustment or replacement or changing to general anesthesia. Subsequently, the plethysmographic signals could be used to monitor the effect of a repositioned catheter or when a “top-up” dose is to be given.

In conclusion, plethysmography pulse oximetry is useful in predicting successful epidural blockade. Considering its simplicity and effectiveness, we recommend its use to predict successful LEB, especially when verbal communication with the patient is difficult.

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Inverse arterial blood flow when the injection is made at a pressure that exceeds arterial pressure. The following case is illustrative.

Local anesthetics can result in central nervous system toxicity. Most anesthesiologists are familiar with reports of convulsive activity after inadvertent intraarterial injection of local anesthetics during the performance of stellate ganglion and interscalene blocks. What is not readily appreciated by many is that accidental intraarterial injection of very low doses of local anesthetics at distant locations, including the brachial and femoral arteries, may also result in toxic central nervous system concentrations. The explanation for this phenomenon is reverse arterial blood flow when the injection is made at a pressure that exceeds arterial pressure. The following case is illustrative.

A 47-year-old woman, American Society of Anesthesiologists physical status I, presented for a right metacarpal fusion. After placement of a pulse oximeter, noninvasive blood pressure and electrocardiography monitors, supplemental oxygen via nasal cannulae was administered. A right axillary brachial plexus block via the transarterial approach was attempted. The local anesthetic mixture used was 1% lidocaine with epinephrine 1:200,000 (Astra USA, Inc., Westborough, MA) and tetracaine crystals (Abbott Laboratories, North Chicago, IL) diluted in lidocaine to a concentration of 0.2%. The needle used was a 1.5-in, 22-gauge, blunt-bevel needle (Sherwood Medical, St Louis, MO). Needle penetration of the axillary artery was confirmed by aspiration of bright red blood. The needle was advanced through the posterior wall of the artery until blood was no longer aspirated. Twenty milliliters of the solution was injected in small aliquots after repeated negative aspirations. There was no change in the heart rate or in the patient’s mental status. The needle was then withdrawn until blood reappeared. At this point, the needle was withdrawn further. Subsequent aspiration revealed residual blood. The needle was withdrawn further, and, after an apparent negative aspiration, 5 ml of the solution was injected. Within 30 s, the patient became dysphoric with evidence of muscle twitching in the face and distal upper extremities. Soon thereafter, the patient became unresponsive. Ventilation was immediately assisted with a Jackson-Rees circuit, and 50 mg sodium thiopental was administered intravenously. The patient became responsive within a few minutes but complained of a headache. The scheduled procedure was then performed using general anesthesia.

Aldrete et al. showed that toxic concentrations of lidocaine could be measured in the internal carotid artery and jugular vein within seconds after injection into the brachial or femoral arteries of laboratory animals. They concluded that local anesthetic drugs injected into these arteries might reach the cerebral circulation after a centripetal pathway and thus produce central nervous system toxic responses. Moreover, Downs et al. demonstrated the possibility of reversed arterial flow traveling distances greater than 60 cm when volumes of 3 ml contrast media, used to irrigate radial artery cannulae, allowed visualization of the subclavian and vertebral arteries. These observations help explain how local anesthetics injected into peripheral arteries may gain access to the brain where the threshold for toxicity is low.

In our case, an accidental intraaxillary artery injection of a small dose of local anesthetics (lidocaine, 30 mg; tetracaine, 6 mg) resulted in clinical evidence of central nervous system toxicity.

In conclusion, practitioners must be aware of the possibility of reverse arterial flow as a mechanism of local anesthetic central nervous system toxicity when performing regional anesthetic blocks to the extremities.

To the Editor.—Accidental intraarterial injection of very low doses of local anesthetics can result in central nervous system toxicity. Most anesthesiologists are familiar with reports of convulsive activity after inadvertent intraarterial injection of local anesthetics during the performance of stellate ganglion and interscalene blocks. What is not readily appreciated by many is that accidental intraarterial injection of local anesthetics at distant locations, including the brachial and femoral arteries, may also result in toxic central nervous system concentrations. The explanation for this phenomenon is reverse arterial blood flow when the injection is made at a pressure that exceeds arterial pressure. The following case is illustrative.

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