The Pain Visual Analog Scale: Linear or Nonlinear?

To the Editor—We read with interest the study reported by Aubrun et al.1 in the June issue of Anesthesiology as well as the accompanying editorial.2

This study is analysis of routine data collected by nursing staff. The postanesthesia care unit nurses in this study followed a routine protocol for the administration of morphine. Most hospitals have standard operating procedures for morphine administration in the PACU. In the study by Aubrun et al., nurses were instructed to ask the patients about their pain and helped them perform a visual analog scale (VAS) every 5 min. Then they administered a 2- or 3-mg dose of morphine (depending on body weight) until adequate pain relief occurred (defined as a VAS < 30). It is very hard to perform 5 to 20 consecutive measurements of respiratory rate, oxygen saturation, arterial blood pressure, Ramsay sedation score, and heart rate while administering morphine every 5 min unless one has no other patient care responsibility. The authors also mentioned that they had discovered that in approximately 17% of the patients, the VAS was not used but instead a verbal or subjective behavioral scale (not defined) was used to collect pain information. Subjects were excluded from the analysis if their pain was not relieved and they required rescue medication. No information is provided regarding what constituted inadequate analgesia requiring rescue. A major limitation of this study is its retrospective nature; therefore, the data provided should be interpreted cautiously.

The authors state that 3,045 patients were analyzed. An assumption is made that all 3,045 patients had relief of pain (VAS < 30) following treatment with morphine, as no information is provided to the contrary. The overall success rate reported by Aubrun et al. is impressive (> 96%). Their table 1 shows that in 39% of patients, severe pain (VAS > 70) was reduced to mild pain (VAS < 30) with 10 mg or less of morphine (in a 70-kg subject). This appears, on the basis of our experience, to be a rather unexpectedly high success rate in patients with severe postoperative pain.

We have been evaluating dose-response of morphine in an ongoing study of patients recovering from total abdominal hysterectomy. In our study, patients are randomized to receive either 12 mg or 21 mg of morphine (5 mg every 3 min) in the PACU, in a double-blind manner. We also defined desired relief as a verbal analog scale of 3 out of 10. We have studied 10 patients so far (unpublished data) and have not achieved the desired relief in any. In another study, we evaluated the analgesic effect of a single 7.5-mg dose of morphine in a double-blind study in patients with lower abdominal procedures and could not measure much analgesia until rescue 10–15 min later. The authors evaluated a diverse group of patients. The data, however, were not analyzed on the basis of surgical type to minimize variations in baseline postoperative pain scores. The authors should have provided basic descriptive statistics such as the number of doses of morphine needed to achieve pain relief based on the initial pain score, type of surgery, and percentage of failure and success, as well as the presence of side effects (if any) and the influence of gender and age. It is equally important to measure the effect of morphine on the affective component of pain, as some patients feel relief despite no change in the intensity of pain.

The editorial accompanying this manuscript stresses that the difference in various procedures translates into lack of standardization of intraoperative anesthetics. We believe diversity of cases reflects a source of baseline variability in pain scores. Furthermore, the editorial states that “opioids side effects exert a major impact on the course of postoperative recovery and limit effective opioids titration in many cases.” We believe that in the majority of cases it is the fear of side effect that limits effective opioids titration. Indeed, at 2 and 5 min after administration of a single 7.5-mg dose of morphine, we observed no significant hemodynamic change.

References


(Accepted for publication November 20, 2003.)

To the Editor—I have spent some time considering the conclusions reached by Aubrun et al.1 in their study investigating the relationship between the pain visual analogue scale (VAS) and morphine requirements in the postanesthesia care unit. They demonstrated that the Hill equation could be used to derive a sigmoid relationship between the pain VAS and morphine dosage, and concluded that this relationship is not linear. In their discussion, they referred to an earlier study in which I and colleagues had found a linear relationship between pain intensity and the pain VAS.2 and they seemed to suggest that their study contradicted our earlier results.

The results of the Aubrun et al. study could have been readily predicted from their methodology, in that the Hill equation is a standard approach used to generate a sigmoid curve using population pharmacokinetics. This approach generates a typical dose–response curve. Such a curve could be derived for the dose of a hypotensive drug and blood pressure, or the dose of a sedative drug and hypnotic state. Yet, the effect of interest—be it blood pressure, hypnotic state, or pain intensity—can still be linear phenomena. The results of the study data analyzed by the authors do not preclude the possibility that the pain VAS has ratio scale properties and is linear. In fact, if the pain VAS is linear then it would have a sigmoid relationship with morphine dosage.

I have one other concern regarding the conclusions drawn by the authors. In their Methods, they arbitrarily defined “severe pain” as when the patient received a total dose of intravenous morphine greater than 0.15 mg/kg. Because they found an association between an initial VAS score of 70 or greater (VAS70) and morphine dosage greater than 0.15 mg/kg, they concluded that VAS70 was indicative of severe pain. But VAS70 itself may not signify severe pain. Opioid dose–response has substantial interindividual variability, with a five- to tenfold range in requirements. Patients may fail to respond to a specific dose not because they have greater pain intensity but because they have had a lesser response to that dose.

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References


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Although our European counterparts have reported better success with larger doses, even these doses may not be sufficiently large. Evaluation of higher doses (i.e., 5 mg) at 3- to 5-min intervals may demonstrate a better relief profile in most patients. The frequency of adverse events should increase with dose; the anticipated occurrence of these side effects, however, may be exaggerated because of our lack of experience. Clinicians are often too concerned about the respiratory effect of rather small doses of morphine (1-2 mg). In some of our patients undergoing abdominal procedures, tachypnea may have represented intentional defense against pain (to avoid deep breaths). We have noticed a significant drop in respiratory rate to a more normal range after relief of pain in some of these patients.

We continue to believe that postoperative pain is undertreated because of the caregiver’s fear and lack of experience. It has been known for quite some time that a VAS of 70 or 80 should be considered severe pain, and that severe pain requires more morphine for treatment than mild or moderate pain. Although postoperative treatment should be individualized, the safety and efficacy of higher doses of morphine must be determined prospectively in patients with moderate-to-severe postoperative pain.

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References

1. Aubrun F, Langeron O, Quesnel C, Coriat P, Riou B: Relationships between measurement of pain using visual analog scale and morphine requirements during postoperative intravenous morphine titration. ANESTHESIOLOGY 2003; 98: 1415-21
2. Berde CB, Brennan TJ, Raja SN: Opioids: More to learn, improvements to be made (editorial). ANESTHESIOLOGY 2003; 98: 1309-12

(Accepted for publication November 20, 2003.)

In Reply.—We thank our colleagues for their comments and criticisms of our study, and we wish to offer the following responses.

First, it is not hard to measure respiratory rate, oxygen saturation, and arterial blood pressure during morphine titration because monitors perform those functions well. Moreover, determination of Ramsay score is also very rapid, because an awake patient who is able to rate his or her visual analog scale (VAS) can be easily quoted 1 on the Ramsay score. Concerning the verbal and subjective behavioral scale used by our nurses, the reader could refer to our recent published study. The definition of inadequate analgesia requiring rescue was subjective: the anesthesiologist decided to stop morphine titration and use another analgesic technique, usually when more than 10-15 bolus of morphine were administered.

We do not accept the term “retrospective” used by Larijani and Goldberg. Indeed, the design of our study was clearly defined after a pilot study, a special data sheet was implemented (although this data sheet is now that used in routine practice in our unit), and all consecutive patients who fulfilled the criteria for inclusion and did not fulfill the criteria of exclusion were included. Thus, we think that this study was prospective.

We agree with Larijani and Goldberg that we need more information on the relationship between morphine requirement and the type of surgery. The influence of gender is real, but its magnitude seems not to be very important. Last, we previously demonstrated that age does not modify morphine requirement during intravenous morphine titration, whereas subcutaneous morphine requirement during the initial 24 hours significantly decreased in elderly patients. Larijani and Goldberg suggest that higher-bolus doses could be more rapidly efficient. We agree with this hypothesis, but the incidence of adverse outcomes may also increase. Only a randomized study could provide the response, and we have begun such a study.

Larijani and Goldberg report that their patients who had abdominal hysterectomy required a higher dose of morphine. In our study, 35 women underwent abdominal hysterectomy and they actually required 14 ± 8 mg (0.23 ± 0.14 mg/kg) morphine during intravenous titration, indicating that this surgery induced severe postoperative pain requiring a greater dose of morphine. However, comparison of the morphine doses during postoperative intravenous titration must be done cautiously, because the anesthetic regimen during the preoperative period, including the type and dose of opioids administered, may markedly interfere with that dose. The interest of our study is only to provide data from a large population to help to recognize some important relationships between the measurement of pain using VAS and morphine requirements. Further studies are required to precise the preoperative and postoperative variables associated with morphine dose in the postoperative period, as recently studied by Dahmani et al.

In our opinion, the definition of severe pain (i.e., VAS > 70) may not be very useful for a given patient. In contrast, we have explained that this definition may help to identify a population of patients that could benefit from morphine titration in less supervised clinical conditions, such as emergency medicine, or may help to stratify the severity of pain during clinical trials.

We do not agree with the comment from Dr. Myles concerning the linear versus sigmoid nature of the relationship between visual analog scale and morphine dose. In our study, we provided several statistical points of evidence that this curve is better described using a sigmoid curve than a straight line. These data include both the initial (30-40) and final (80-100) parts of the VAS range. The important question is whether or not this relationship reflects the VAS-pain relationship. In our study, we suggest that measuring the morphine dose required to obtain pain relief may be another way to assess the severity of pain. However, we agree that we did not take into account the complex nature of pain, which cannot be summarized only by its intensity, and that opioid-dose response has substantial interindividual variability.

In conclusion, we hope that our recent studies on intravenous morphine titration will favor the development on more clinical research on this important topic.

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References

1. Aubrun F, Langeron O, Quesnel C, Coriat P, Riou B: Relationships between measurement of pain using visual analog scale and morphine requirements during postoperative intravenous morphine titration. ANESTHESIOLOGY 2003; 98: 1415-21

(Accepted for publication November 20, 2003.)
To the Editor:—Based on previous research reporting that electromyo-
graphic activity has been shown to elevate the bispectral index (BIS) in
patients not receiving neuromuscular blockade, Vivien et al.1 investi-
gated the magnitude of the decrease in BIS value following adminis-
tration of neuromuscular blocking drugs (NMB) in sedated intensive
care unit patients. They concluded that BIS might be lower with
paralysis for an equivalent degree of sedation because of high muscular
activity. Therefore, Vivien et al.1 ascribe the BIS value modification
only to suppression of muscular activity due to the use of NMB.
Nevertheless, the effect of administration of a neuromuscular blocker
on BIS and electromyographic activity was highly variable among the
45 patients studied, and in 13 of the 45 patients there was no change
in BIS range assessment. These considerations could support the hy-
pothesis that BIS modification may be related not only to muscular
activity suppression but also to NMB properties.

NMB are reported to potentiate the effects of anesthetics. Despite
the emphasis that all NMB are completely devoid of analesic prop-
ties,2 some elicit analgesia. Laudanosine, the atracurium and cisatrac-
urium metabolite, elicits dose-dependent analgesia in the mouse,
attenuated by coadministration of μ1- and μ2-selective antagonists,
indicating a μ-related mechanism for analgesic properties of lau-
danosine.3 A cross-tolerance between laudanosine and morphine has
also been observed.3 Moreover, at clinical concentrations reported in
cerebrospinal fluid, atracurium and laudanosine are able to activate the
central α4β2 nicotinic acetylcholine subtype receptors.4 Agonists for
these receptors showed analgesic activity. Epibatidine, a potent ligand
identified at the α4β2 nicotinic acetylcholine receptor subtype, dis-
played a potent nicotinic activity and a strong nicotinic analgesic
effect.5 Taken together, these data indicate that the coadministration
of the NMB atracurium or cisatracurium in sedated patients could po-
tiate the effects of midazolam and sufentanil sedation, and therefore
decrease BIS value. This hypothesis is also consistent with a study
reporting that mivacurium, a NMB without analgesic properties, does
not alter hypnotic level during propofol anesthesia.6

Nevertheless, it is not possible to support or deny the suggested
hypothesis; interestingly, the NMB used in the study of Vivien et al.1
were not reported. A list of NMB administered to the patients could
maybe offer an aid, pro or conra.

The clinical relevance is that the modification of BIS detected by
Vivien et al.1 in sedated intensive care unit patients after administration
of NMB may be related not only to muscular activity block, as master-
fully demonstrated, but also to other cofactors related to NMB or their
metabolites’ properties.

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References
1. Vivien B, Di Maria S, Ouattara A, Langeron O, Coriat P, Riou B: Overesti-
mation of bispectral index in sedated intensive care unit patients revealed by
administration of muscle relaxant. Anesthesiology 2003; 99:9–17
2. Prielipp RC, Cousin DB: Applied pharmacology of common neuromuscular
3. Fodale V, Santamaria LB: Laudanosine, an atracurium and cisatracurium
4. Fodale V, Santamaria LB: The possible neuroprotective effect of lau-
danosine, an atracurium and cisatracurium metabolite (letter). Acta Anaesthesiol
Scand 2003; 47:780–1
5. Belluardo N, Mudo G, Blum M, Fuxe K: Central nicotinic receptors, neuro-
rophic factors and neuroprotection. Behav Brain Res 2000; 113:21–34
DI: Muscle relaxation does not alter hypnotic level during propofol anesthesia.
Anes Analg 2002; 94:604–8

(Accepted for publication November 25, 2003.)

In Reply.—We appreciate the interest of Drs. Fodale and Santamaria
in our article examining the decrease in bispectral index (BIS) follow-
ing the administration of a neuromuscular blocker1 and would like to
reply to their comments. Above all, we must correct a mistake in the
letter: The neuromuscular blocker used in our study was clearly re-
ported as 0.5 mg/kg atracurium in every patient studied. Otherwise,
we think that the analysis of the authors is simply invalid for several
reasons.

First, Peat et al.2 reported that the prolonged use of atracurium is not
associated with excessive accumulation of laudanosine, so long as
the renal and hepatic functions are normal. On the other hand, Grigore
et al.3 also reported that laudanosine accumulation may occur in patients
with both fulminating hepatic and renal failures, but it is not associated
with any measurable central neurologic effect. In our study, we inves-
tigated either a single standard dose of atracurium or the beginning of
a longterm administration. Therefore, we do not think that we could
have observed central neurologic effects due to high concentrations of
laudanosine in our patients.

Second, an analgesia-mediated property was described experimen-
tally for laudanosine through a μ1 mechanism4 and stimulation of the
central α4β2 nicotinic acetylcholine receptor.5 However, BIS is less
influenced by analgesic than by sedative drugs,6 and consecutive BIS
change because of laudanosine should have been very limited.

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References
1. Vivien B, Di Maria S, Ouattara A, Langeron O, Coriat P, Riou B: Overesti-
mation of Bispectral Index in sedated intensive care Unit patients Only Related to Electromyographic Activity?

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Anesthesiology 2004; 100:746

Third, the authors reported in a recent review that data about the
evidence of the depressive effects of laudanosine on the central ner-
vous system in humans are not yet available.7 They also reported that
the laudanosine administration would either stimulate the central ner-
vous system or have convulsive effects.8,9 Such effects would logically
and expectedly result in an increase in BIS values,10 in contrast to that
suggested in the letter from the authors.

Finally, even if a BIS decrease following atracurium administration
would have been due to the sedative effect of laudanosine produced
after atracurium administration, it seems unlikely that BIS recovery
after laudanosine elimination would have been exactly parallel to
electromyographic activity recovery after atracurium elimination, as
observed in our patients.

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References
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References
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References
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Management of Patient Body Temperature Is Challenging

To the Editor.—We thank Gali et al. for their description of what they consider to be a pressure/burn injury following the use of the Allon thermoregulation system.1 Management of patient body temperature and successful maintenance of normothermia in such a procedure is challenging and, as noted by the authors themselves, the patient maintained normothermia and her temperature at the end of the procedure was 36.3°C.

The incidence of pressure ulcers during surgery is well documented in the literature. Overall incidence is around 8%, depending on type of surgical procedure and length of surgery. The incidence rate is positively correlated to the length of the procedure and exceed 13% in procedures lasting longer than 7 h.2-5

As Gali et al. state, this patient had numerous risk factors that predisposed her to pressure-related injury during the operative procedure, including prolonged surgery (6.5 h), advanced age, severe muscle wasting, poor nutritional status, end-stage liver disease, and intraoperative hypotension. We therefore beg to differ with the authors favoring the injury as being primarily due to a burn. This interpretation is based on the surgeon’s impression and on the patient’s postoperative complaint of a burning sensation in the affected area.

It is nearly impossible to differentiate by observation alone whether a skin lesion is due to pressure or a burn, whereas a burning sensation is certainly not specific to burn victims. Furthermore, many factors may contribute to the pressure sores between the operating table and the ThermoWrap. Therefore, it is understandable that the Allon system itself was checked and found to be functioning properly.

In anticipated long-duration procedures and in chronically ill, high-risk patients, the usual precautions of meticulous attention to adequate temperature. With regard to the pressure relief algorithm, the only information that we could find is one unpublished trial of three patients, which revealed pressures of up to 200 mmHg (measurement limited because of 200 mmHg as the maximum range of calibration).

Although the Allon system may help maintain normothermia, it may do so at some risk to patient well-being.


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References

5. Schulz A: Etiology and incidence of pressure ulcers in surgical patients. AORN J 1999; 70:434–49
7. Stewart TP, Magnano SJ: Burns or pressure ulcers in the surgical patient? Decubitus 1988; 1:36–40

Accepted for publication September 28, 2003.)
To the Editor.—We read with interest the excellent review article of randomized, controlled trials involving the effects of nonsteroidal anti-inflammatory drugs (NSAIDs) on bleeding after tonsillectomy.1 Although the results of this meta-analysis suggest “that the use of NSAID therapy after tonsillectomy should be abandoned both at the hospital and at home,”1 we must remind the readers that the perioperative use of cyclooxygenase (COX)-2 selective NSAIDs may offer a safer alternative in the management of posttonsillectomy pain. Previous data have suggested that celecoxib,2 rofecoxib,3 and valdecoxib4 have analgesic effects similar to conventional NSAIDs when used for acute pain. Further, because platelets express only COX-1 and are incapable of expressing COX-2,5 selective COX-2 inhibitors do not inhibit platelet function. We have found that the preoperative administration of rofecoxib possesses a more favorable pharmacokinetic profile than the other COX-2 selective NSAIDs in the management of pediatric tonsillectomy pain. Rofecoxib provides onset of clinical analgesia within 27 min8 and has an elimination half-life of 17.5 h; therefore, it can be administered once daily.3 Rofecoxib is available as a strawberry-flavored oral suspension (containing 12.5 mg or 25 mg of rofecoxib per 5-ml solution), which makes pediatric dose titration easy to accomplish.

We recently evaluated the analgesic efficacy and safety of administering rofecoxib (1 mg/kg) prior to pediatric tonsillectomy.7 This study revealed no significant increase in measured intraoperative surgical bleeding or in the likelihood of reoperation for bleeding. Furthermore, the preoperative administration of rofecoxib resulted in a significant decrease in postoperative pain and more than a threefold reduction in the incidence of postoperative nausea and vomiting. The reduction in postoperative nausea and vomiting may be attributable to either the improved analgesic efficacy of rofecoxib or to the centrally mediated action. It has been demonstrated in an animal model that activation of the medullary vomiting center involves prostaglandins, and the preemp-tive administration of COX inhibitors significantly decreased lipopolysaccharide-induced emesis.6 NSAIDs with significant penetration into the central nervous system may be advantageous in reducing postoperative nausea and vomiting. Previous studies in rats have revealed that 35% of rofecoxib plasma concentrations penetrated into the cerebrospinal fluid; this relative penetration into the central nervous system was independent of dose (≤ 150 mg/day).10 Therefore, we believe the perioperative administration of rofecoxib, a selective COX-2 inhibitor, may offer significant advantages over conventional NSAIDs in the management of tonsillectomy pain.

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References

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Effects of Postoperative Nonsteroidal Antiinflammatory Drugs on Bleeding Risk after Tonsillectomy

To the Editor.—As stated by Marret et al.,1 posttonsillectomy hemorrhage (PTH) can be life-threatening, and an incidence of 4.5% per year in the United States with a reoperation rate of 1–5.5% is not insignificant, particularly when associated with concerns surrounding the administration of general anesthesia under such circumstances. Nonsteroidal antiinflammatory drugs (NSAIDs) are, however, now widely used not only in the operating room and ward but also at home, and they are widely believed to be safe and effective. Unfortunately, Marret et al. considered just seven investigations that satisfied their chosen criteria, none of which in fact reported a significant difference between the groups studied. Simply combining the results of these into a single analysis to create the reported result does not provide an adequate answer to the concerns expressed. In addition, anesthesiologists are aware that the chosen operative technique, together with the skill and experience of the operating surgeon, can be of significant importance. The total number of patients in the seven studies is small, with marked variability, including the NSAID chosen, the number of doses administered, the surgery undertaken, and whether a primary or secondary hemorrhage occurred. An additional qualifying study was not included.2

In 1984, Carrick3 reported the potential adverse consequence of increased risk by salicylate administration on platelet function when PTH occurred in 1 of 350 patients versus just 1 of 355 patients receiving acetaminophen. NSAIDs, including aspirin (acetylsalicylic acid), inhibit platelet cyclooxygenase and prolong bleeding time by preventing the biosynthesis of thromboxane A2, leading to reduced platelet aggregation. NSAIDs are very widely used, and because they are available without prescription many patients take them perioperatively. To overcome this problem, the New York Eye and Ear Infirmary has instructed patients not to use aspirin or products containing aspirin, as well as other NSAIDs, for at least 7 to 10 days before and after surgery.4 This, as well the standard of specialist medical care, is
Nonsteroidal Antiinflammatory Drugs and Hemorrhage following Tonsillectomy: Do We Have the Data?

To the Editor—Marret et al. provided a meta-analysis of nonsteroidal antiinflammatory drugs (NSAIDs) and bleeding risk after tonsillectomy using return to the operating room as their primary endpoint. The authors carefully reviewed the literature and selected seven articles for inclusion. We are concerned that the conclusions reached are invalid and that the wording in the current Marret et al. article provides fodder for lawyers without adequate scientific knowledge to support these admonitions.

The authors have combined repeated single-dose intramuscular administration, continuous infusion, and chronic administration of NSAIDs to contribute to an increased incidence of PTH. The effects on platelet function can be of long duration and would be expected to follow even a single dose given perioperatively. Administration at or before the time of surgery may beneficially allow hemostasis to be secured while the drug is active. This study has, however, usefully served to signal the urgent need for the performance of further randomized, controlled trials with both NSAID administration and surgical factors tightly controlled in an effort to bring clarity to this issue. NSAIDs were effective in these patients, and the cyclooxygenase-2 inhibitors are currently being promoted for perioperative use.


References


(Accepted for publication October 29, 2003.)

Table 1. Patients Undergoing Tonsillectomy at Glan Clwyd Hospital

<table>
<thead>
<tr>
<th>Year</th>
<th>Patients (No.)</th>
<th>PTH</th>
<th>No.</th>
<th>%</th>
<th>NSAID</th>
<th>No.</th>
<th>%</th>
<th>Senior Surgeon (%)</th>
<th>Senior Anesthetist (%)</th>
</tr>
</thead>
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<td>1991</td>
<td>596</td>
<td>0</td>
<td>0</td>
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<td>637</td>
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<td>136</td>
<td>21.4</td>
<td>38.8</td>
<td>60.6</td>
</tr>
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<td>510</td>
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<td>0.8</td>
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<td></td>
<td>92</td>
<td>18.0</td>
<td>32.4</td>
<td>65.3</td>
</tr>
<tr>
<td>1994</td>
<td>545</td>
<td>2</td>
<td>0.4</td>
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<td></td>
<td>57</td>
<td>10.5</td>
<td>21.5</td>
<td>63.9</td>
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<tr>
<td>1995</td>
<td>573</td>
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<td>0.4</td>
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<td></td>
<td>143</td>
<td>25.0</td>
<td>17.1</td>
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<tr>
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<td>572</td>
<td>6</td>
<td>1.0</td>
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<td></td>
<td>104</td>
<td>18.2</td>
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<td>46.7</td>
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<td>1997</td>
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<td>1.0</td>
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<td>166</td>
<td>32.0</td>
<td>19.1</td>
<td>54.7</td>
</tr>
<tr>
<td>1998</td>
<td>526</td>
<td>2</td>
<td>0.4</td>
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<td></td>
<td>221</td>
<td>42.0</td>
<td>17.7</td>
<td>58.0</td>
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<td>1999</td>
<td>456</td>
<td>5</td>
<td>1.1</td>
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<td></td>
<td>384</td>
<td>84.2</td>
<td>40.1</td>
<td>42.5</td>
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<tr>
<td>2000</td>
<td>451</td>
<td>5</td>
<td>1.1</td>
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<td></td>
<td>262</td>
<td>58.1</td>
<td>19.7</td>
<td>54.7</td>
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<tr>
<td>2001</td>
<td>313</td>
<td>9</td>
<td>2.9</td>
<td></td>
<td></td>
<td>181</td>
<td>57.8</td>
<td>28.8</td>
<td>47.9</td>
</tr>
<tr>
<td>2002</td>
<td>151</td>
<td>2</td>
<td>1.3</td>
<td></td>
<td></td>
<td>110</td>
<td>72.9</td>
<td>41.1</td>
<td>68.2</td>
</tr>
</tbody>
</table>

Senior surgeon and senior anesthetist are trained and experienced specialists.

NSAID = nonsteroidal antiinflammatory drug administered intraoperatively; PTH = posttonsillectomy hemorrhage requiring reoperation.

reflected in their reported low incidence for PTH of 0.9% between 1992 and 1996.

Postoperative nausea and vomiting is also a relevant contributory factor. When compared with opioids, the value of NSAIDs in reducing this and avoiding the need for antiemetic administration is borne out by the only statistically significant difference reported in one of the seven included studies.7

Since the late 1980s, the first author’s practice has been to administer diclofenac for pain control parenterally at the time of surgery, initially alone but subsequently together with morphine (after the now widely adopted combination had been shown to yield significant benefit). An initial audit for the years 1991 to 1994, when 2,136 tonsillectomies were performed in our hospital, revealed 10 cases requiring a return to the operating room for control of PTH (0.47%). Of the 378 patients who had received NSAIDs intraoperatively, only 1 was included in the 10 cases reported. These results suggested the practice to be safe but are in marked contrast to those subsequently published by Robinson and Ahmed8 describing a 5.5% incidence of PTH following tonsillectomy. The conclusion of Marret et al.9 is supported in their low incidence for PTH of 0.9% between 1991 and 1996.

The recorded incidence of PTH requiring reoperation is reassuringly low over the whole period except for the year 2001 (2.9%). Our figures do not suggest that the experience of either surgeon or anesthesiologist is a critical factor. We have no specific record of the frequency of use of NSAIDs in the postoperative period, but we know that they have been and continue to be widely prescribed. Our data could be interpreted as showing a weak relationship between the frequency of intraoperative use and PTH. The conclusion of Marret et al. that postoperative conventional NSAIDs increase the risk of reoperation for hemostasis and should not be used after tonsillectomy is too broad; it ignores the benefits provided by a reduction in both postoperative nausea and vomiting and postoperative pain, which themselves may contribute to an increased incidence of PTH. The effects on platelet function can be of long duration and would be expected to follow even a single dose given perioperatively. Administration at or before the time of surgery may beneficially allow hemostasis to be secured while the drug is active. This study has, however, usefully served to signal the urgent need for the performance of further randomized, controlled trials with both NSAID administration and surgical factors tightly controlled in an effort to bring clarity to this issue. NSAIDs were effective in these patients, and the cyclooxygenase-2 inhibitors are currently being promoted for perioperative use.


Table 1. Patients Undergoing Tonsillectomy at Glan Clwyd Hospital
NSAIDs. Unlike aspirin, the effects of most NSAIDs on platelet function are very short-lived. For pediatric patients, the half-lives of ketorolac and ibuprofen are approximately 2 h and 1.5 h, respectively.6,10 Because significant effects on platelet function are reversible and related in part to a blood concentration of NSAIDs, virtually no antiplatelet effect would be present after 5 to 6 drug half-lives. Therefore, any bleeding attributable to single doses of these medications would have to occur in the immediate postoperative period, and certainly within the first 24 h.11,12 Hemorrhage that occurs after 24 h is unlikely to be caused by these medications unless they are administered after hospital discharge; we do not know if this is different with infusions or intramuscular administration.

When examining the duration of treatment (table 1 of the Marret et al. review), studies by St. Charles et al.7 and Harley and Dattolo8 involved patients who received treatment for 2 weeks. Both patients who had bleeding in the Harley and Dattolo study did so 2 to 7 days after the operation, so it is possible that their bleeding could be attributed either to the continued use of the NSAIDs or to the loss of eschar after the operation because of lack of cooperation.† Likely that bleeding occurred more than 24 h after surgery. § chi-square P = 0.209 (underpowered with α < 0.8).

<table>
<thead>
<tr>
<th>Drug Type</th>
<th>Bleeding</th>
<th>Control</th>
<th>NSAIDs</th>
<th>Bleeding</th>
<th>Control</th>
<th>NSAIDs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bailey³</td>
<td>Ketorolac IM × 24 h (adults)</td>
<td>0/43</td>
<td>0/37</td>
<td>1/43</td>
<td>2/37†</td>
<td></td>
</tr>
<tr>
<td>Gunter⁵</td>
<td>Ketorolac IV × 1 dose</td>
<td>0/47</td>
<td>1/49</td>
<td>0/47</td>
<td>1/49</td>
<td></td>
</tr>
<tr>
<td>Harley⁶</td>
<td>Ibuprofen × 2 wk</td>
<td>0/11</td>
<td>0/16</td>
<td>0/11</td>
<td>2/16</td>
<td></td>
</tr>
<tr>
<td>Rømsing⁴</td>
<td>Ketorolac IV × 1 dose</td>
<td>0/15</td>
<td>1/15†</td>
<td>Unknown</td>
<td></td>
<td></td>
</tr>
<tr>
<td>St. Charles⁷</td>
<td>Ibuprofen × 2 wk</td>
<td>0/55</td>
<td>1/55†</td>
<td>Unknown</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Solanen¹⁴</td>
<td>Ketorolac × 24 h; infusion × 24 h (adults)</td>
<td>0/25</td>
<td>1/40</td>
<td>Unknown</td>
<td></td>
<td></td>
</tr>
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<td>Sutters⁷</td>
<td>Ketorolac IM × 1 dose</td>
<td>0/42</td>
<td>0/45</td>
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<td>0/198</td>
<td>4/257§</td>
<td>1/198</td>
<td>5/257</td>
<td></td>
</tr>
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</table>

Date are expressed as numbers.

* Time not described: the first 15 patients were not included because five hemorrhages were attributed to poor surgical technique. † Patient returned to the operating room because of lack of cooperation. § Likely that bleeding occurred more than 24 h after surgery. ** chi-square P = 0.209 (underpowered with α < 0.8).

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IM = intramuscularly; IV = intravenously; NSAID = nonsteroidal anti-inflammatory drug.

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References


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was still documented with the use of NSAIDs (odds ratio: 4.0; 95% confidence interval: 1.4, 11.3; \( P = 0.009; P = 0.98 \) for the heterogeneity test).

Finally, Drs. Lake and Khater concluded that urgent, randomized, double-blind trials are needed to clarify the risk of reoperation with NSAIDs use. In fact, there is now sufficient evidence to demonstrate an increased risk of bleeding after tonsillectomy with NSAIDs. Regarding this risk of reoperation, we consider that the use of NSAIDs should be abandoned.

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References


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Carotid Sinus Mechanical Properties

To the Editor:—Boyce and Peters1 describe the interesting case of complete vasomotor collapse during resection of a recurrent right carotid body tumor involving the right carotid bifurcation requiring excision and autologous vein grafting. The authors suggest mechanical or electrical stimulation of the right carotid sinus nerve as the initiating event leading to vasomotor collapse. Rarely, however, does a significant increase in afferent activity from a single baroreceptor site result in the described persistent profound hypotension because of compensation from other baroreceptor sites, even in the absence of a right vagus nerve.

Alternatively, if in this patient the tumor had infiltrated the right carotid sinus wall such that no deformation (strain) of the baroreceptors could take place as can occur in severe carotid atherosclerosis,2 then there would be no afferent neural input to the hindbrain from this site. Under these circumstances, the vasomotor integration centers in the hindbrain would not recognize zero input from the right carotid sinus as a legitimate null signal, particularly in the presence of normal afferent signals from other baroreceptor sites.3 Subsequently, sudden significant afferent activity in the right carotid sinus nerve, either from exogenous deformation of the sinus wall or direct stimulation, would probably be interpreted as a very significant signal by the hindbrain because of the neural history from this site, resulting in the reported dramatic decrease in blood pressure. A similar phenomenon is occasionally seen during carotid endarterectomy when severe atherosclerotic plaque is suddenly removed from the carotid sinus area.4,5

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References


(Accepted for publication September 23, 2003.)
To the Editor:—Most of the preventable incidents in anesthesia practice involve human error, with breathing circuit disconnections featuring prominently. Overt equipment failures constitute a small proportion of preventable incidents, but equipment design is contributory in many categories of human error.¹⁻⁴ We herein report a case of a breathing circuit obstruction resulting from failure to completely remove the transparent packaging.

A 36-yr-old, 185-cm, 86-kg healthy man with no history of previous anesthesia underwent uneventful induction of general anesthesia for endoscopic sinus surgery conducted in a standard manner with midazolam, fentanyl, propofol, and vecuronium. Tracheal intubation was accomplished with an 8.0-mm-ID oral cuffed endotracheal tube. Proper endotracheal tube placement was confirmed by auscultation of bilateral breath sounds and end-tidal carbon dioxide. Endotracheal tube depth was confirmed and secured. At the request of the surgeon, the breathing circuit was briefly disconnected and the table was rotated 180 degrees. The existing 90-degree gas sampling connector was removed, and a straight gas sampling connector (SIMS Portex, Inc., Fort Myers, FL) was removed from its plastic packaging and placed in-line. Chest excursion immediately ceased and the ventilator failed to cycle. Hand ventilation with the anesthesia machine was attempted and failed. An Oxygen E-Cylinder with a Mapleson circuit (Vital Signs, Totowa, NJ) was used to ventilate the patient without difficulty with intravenous propofol infusion to maintain anesthesia. Auscultation of the chest revealed normal breath sounds. Careful inspection of the breathing circuit revealed a taut, transparent diaphragm of plastic packaging obstructing the straight connector at its interface with the breathing circuit (fig. 1). The clinician’s thumb had inadvertently retained this transparent obstruction on removal from its packaging. Following removal of the obstruction, the circuit was reconnected and the case proceeded uneventfully.

In summary, we suggest that as an additional measure of safety, packaging for airway components be considered both for its visibility and potential for airway circuit obstruction (fig. 2). We contacted the manufacturer (Portex, Inc., Keene, NH) and MEDWATCH (the U.S. Food and Drug Administration Safety Information and Adverse Event Reporting Program) to alert them of our experience.

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References

(Accepted for publication September 26, 2003.)

After numerous failed attempts to acquire a Reply to this Letter, it is being published without a response. —Michael M. Todd, Editor-in-Chief.
Revenue Gain for Academic Anesthesiology Departments if the Centers for Medicare and Medicaid Services Provide Full Reimbursement to Teaching Physicians

To the Editor—Following the 1996 publication of new rules regarding payment to teaching physicians, the Centers for Medicare and Medicaid Services (then known as the Health Care Financing Administration) reduced by 50% the payments made to teaching anesthesiologists for medical direction of residents at a ratio of one faculty member to two residents. After 1996, the Centers for Medicare and Medicaid Services paid 100% of allowable charges to a teaching anesthesiologist only when the physician either personally performed the care or medically directed only one resident. Because resident care could not be billed to the Centers for Medicare and Medicaid Services, this change in the teaching rules resulted in a significant reduction in payments to academic anesthesiology groups.1 Recently, the Centers for Medicare and Medicaid Services were asked to reexamine these teaching rules by both anesthesiology societies and Senator Hillary Clinton (written communication to Mr. Thomas Scully, April 2003).

Using data from a previously published study,2 we examined the potential financial impact that a change to full reimbursement would have on two of the four academic anesthesiology groups examined in that investigation (the database for the other two groups did not identify Medicare and other payers). Groups A and D (as designated in the previous study) provided care to 2,549 and 7,096 Medicare beneficiaries undergoing surgery during the 1-yr study period. For purposes of analysis, we used the conversion factor for the Galveston, Texas, area ($17.25) and assumed that all anesthetic procedures were performed by anesthesiologists supervising residents at a 1:2 ratio.3 We calculated that the annual revenue lost by the current rules, versus the pre-1996 rules, was $550,000 for group A and $1,070,000 for group D (table 1). The estimated lost revenue per case was similar between the two groups ($137 and $150 for groups A and D, respectively).

If the previous rules for full payment (which are similar to the current rules applied to other medical specialties) were applied, the increase in revenue would help academic anesthesiology departments successfully meet their economic challenges.

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

Table 1. Impact of Teaching Rules on Academic Anesthesiology Departments

<table>
<thead>
<tr>
<th>Cases (No.)</th>
<th>2,549</th>
<th>7,096</th>
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</thead>
<tbody>
<tr>
<td>Total ASA units billed (No.)</td>
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<td>124,100</td>
</tr>
<tr>
<td>Total allowable</td>
<td>$700,000</td>
<td>$2,140,000</td>
</tr>
<tr>
<td>50% not paid because of faculty supervision of resident care at 1:2 ratio</td>
<td>$350,000</td>
<td>$1,070,000</td>
</tr>
<tr>
<td>Revenue lost per case</td>
<td>$137</td>
<td>$150</td>
</tr>
</tbody>
</table>

Medicare cases billed in 1996 by two different academic anesthesiology departments; allowable calculated using the 2003 Galveston, Texas, conversion factor ($17.23). Estimates are based on the assumption that all cases would be performed using the staffing model of one teaching anesthesiologist to two residents. This information is based on data from a previous study by Abouleish et al.2

ASA = American Society of Anesthesiologists.

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References


(Accepted for publication October 10, 2003.)

Deviation of the Cauda Equina by Changing Position

To the Editor—We have obtained interesting information about spinal puncture using magnetic resonance imaging. It is important to understand the anatomy of the cauda equina when performing spinal anesthesia. Previous studies using computed tomography or magnetic resonance imaging have shown that in the supine position, the cauda equina lies symmetrically in the dorsal subarachnoid space.1–3 However, a patient is usually placed in the lateral decubitus position during spinal puncture. Thus, it is necessary to obtain detailed anatomic information about the cauda equina in the lateral decubitus position. Magnetic resonance imaging reveals interesting and important information about the anatomy of the cauda equina. In seven healthy volunteers, axial views of magnetic resonance imaging of the cauda equina during both the supine and left lateral decubitus positions were obtained and compared. In all subjects, a movement of the cauda equina was observed by changing position. The cauda equina lay symmetrically at the dorsal side of the subarachnoid space when the patient is in the supine position (fig. 1A). However, it moved to the left side of the subarachnoid space when the patient was placed in the left lateral decubitus position (fig. 1B). Our observations are similar to those that Fink et al. reported in an abstract at the 1993 Annual Meeting of the American Society of Anesthesiologists (published in ANESTHESIOLOGY 1993; 79:AR28). These results suggest that the cauda equina has considerable mobility in the cerebrospinal fluid. During the lateral decubitus position, it may dynamically move to the gravity-dependent side. This phenomenon may alter our thought on spinal anesthesia. First, we should care about cauda equina syndrome. If a spinal needle is inserted downward in the lateral decubitus position, it may increase the possibility of injury to the cauda equina. Second, it is necessary to consider the specific gravity of local anesthetics for spinal anesthesia. Differences in anesthetic effects between hyperbaric and...
To the Editor—New anesthesia machines incorporate novel designs of familiar components to provide additional functionality. The current literature and the U.S. Food and Drug Administration’s MAUDE database* contain periodic reports of difficulties in operating this modern equipment. The onus is on anesthesiologists to remain vigilant to possible new problems with this equipment and to new manifestations of old problems. We recently experienced a condition in which the design of the Drager Fabius GS (Draeger Medical Inc., Telford, PA) anesthesia machine’s breathing circuit exposed a loss of redundancy that was heretofore present in anesthesia machines.

After induction of anesthesia, mechanical ventilation and 2% desflurane in nitrous oxide/oxygen were provided with a Drager Fabius GS anesthesia machine. Set gas flows were 2 l/min of nitrous oxide and 1 l/min of oxygen. The anesthesia machine had been completely checked according to the manufacturer’s directions immediately before the case. This included leak tests of the circuit, breathing bag, and ventilator, all of which showed satisfactory results. Immediately after induction, we realized that the patient was latex-allergic and replaced the breathing bag with a nonlatex bag. A few minutes later, we noted that the breathing bag was empty. This is never a normal condition with the Fabius GS, as this machine has a piston ventilator and uses the breathing bag as the gas reservoir during mechanical ventilation. After changing the nonlatex bag, the machine was pressure tested and mechanical ventilation and delivery of anesthetics were resumed. Thereafter, gas monitor values were consistent with machine settings. Afterwards, the patient denied recall of intraoperative events.

The oxygen sensor on the machine also showed that less oxygen was being delivered than the machine was set for. We inferred that the machine was entraining room air, as it is designed to do under conditions of insufficient fresh gas flow, but this appeared to be inconsistent with the 3 l flows set on the machine, and we immediately suspected a breathing circuit leak. We disconnected the patient from the anesthesia machine, switched to manual ventilation mode, closed the adjustable pressure-limiting valve, and attempted to pressurize the circuit. This revealed a large hole hidden in the folds of the nonlatex breathing bag. The breathing bag was quickly replaced, the circuit was successfully pressure-tested, and mechanical ventilation and delivery of anesthetics were resumed. Thereafter, gas monitor values were consistent with machine settings. Afterwards, the patient denied recall of intraoperative events.

This is not the first report of a sudden inability to pressurize the breathing circuit of the Fabius GS. In 2002, several reports were made to the Food and Drug Administration of an inability to pressurize the breathing circuit, sometimes after successfully completing a leak test. This problem was traced by Drager’s engineers to a small change in a proven design, and the problem has been definitively addressed.

The Drager Fabius GS uses a piston-driven ventilator, and the piston is not the breathing circuit gas reservoir. Instead, the breathing bag hypobaric local anesthetics may contribute to the deviation of the cauda equina by changing position. Although additional investigations concerning the anatomy of the cauda equina are necessary, this observation transforms our knowledge about spinal anesthesia.

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References


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Novel Breathing Circuit Architecture: New Consequences of Old Problems

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


Fig. 1. Anesthesia machine settings during the event described. Inset shows contemporaneous gas analyzer readings. The difference was due to room air entrained through a large hole in the breathing bag.

Fig. 1. Axial view of the cauda equina. Magnetic resonance imaging (T2 weighted, spin echo, TR 2000/TE, 100 ms) at L2–3 level in the same subject were obtained in the supine position (A) and in the left lateral decubitus position (B). After changing position, the cauda equina markedly moved to the left side of the subarachnoid space. L = left; R = right.

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serves as the reservoir during both mechanical and manual ventilation. Breathing bag failure compromised the function of our anesthesia machine in both ventilation modes: the inability to pressurize the circuit in manual mode and decreased oxygen and anesthetic concentrations in mechanical mode.

Our failure was not so catastrophic that the patient could not be ventilated (at least mechanically), and it had nothing to do with the anesthesia machine itself. Adequate mechanical ventilation continued throughout the event. However, the anesthesia machine entrained room air presumably freely through the hole in the breathing bag. This path of air entrainment explains another important feature of this event: no alarms sounded to alert clinicians of the failure.

The Fabius GS has a “low fresh gas flow” alarm to alert users that insufficient gas is entering the circuit to make up for removal due to uptake and leaks. With inadequate fresh gas flow, the piston empties the breathing bag and then room air is entrained via a one-way valve into the piston chamber so that the ventilator delivers the set tidal volume. A small negative pressure is generated when the ventilator attempts to draw from an empty breathing bag, triggering the alarm. In our case, the circuit was always in continuity with the atmosphere because of the large hole in the breathing bag, no negative pressure was developed, and no alarms sounded, but room air was entrained. Instead, we detected and corrected a massive circuit leak (induced after machine checkout) by observation of a constellation of inconsistencies. Despite improved alarm systems, previously secondary monitors such as agent analyzers are sometimes the only alerts to impending failures (in this case, awareness).4

Relative to older designs, subtle changes in the external operation of the Fabius GS conceal radical internal changes that both add functionality and completely alter its response to failure conditions. The use of a piston ventilator with room air entrainment as a backup gas source is safe because it ensures adequate ventilation. However, this design could lead to awareness5 without the use of agent analysis or some measure of anesthetic depth, and is also of concern when considering the use of the machine to care for patients dependent on high inspired oxygen concentrations.

**References**


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