

Clarifying Equipment Specifications and Performance Characteristics

To the Editor:—I have concern about the article entitled “Performance Characteristics of Five New Anesthesia Ventilators and Four Intensive Care Ventilators in Pressure-support Mode: A Comparative Bench Study,” which appeared in the November 2006 issue of ANESTHESIOLOGY.¹

Several statements made in the article are incorrect, and we appreciate an opportunity to set the record straight. In table 1 of the article, the Avance (GE-Datex-Ohmeda, Munchen, Germany—which would also apply to our Aestiva, Aisys, and Aespire 7900) is listed correctly as a flow-triggered system, but the specifications for flow triggering are incorrect. Flow triggering on the above GE systems is selectable from 0.2 to 10 lpm, to better address the needs of a pediatric population. Also, the inspiratory-to-expiratory cycle is listed as fixed, 25% of peak flow. Instead, pressure-support ventilation on all of the above systems is adjustable, from 5% to 50% of the peak inspiratory flow, again, to better meet the needs of a pediatric population.

A more serious error, however, is the following statement: “The best characteristics of the pressurization phase for the anesthesia ventilators were obtained with the Fabius, Primus, and Avance under all tested conditions and were comparable with those obtained with the ICU [intensive care unit] ventilators. The Fabius, Primus, and Avance are ‘piston ventilators,’ which use an electric motor to compress gas in the breathing circuit, creating the driving force for mechanical insufflation to proceed. Therefore, they use no driving gas and may be used without depleting the oxygen cylinder in case of oxygen pipeline failure. These features may explain in part that these more recent anesthesia ventilators have comparable performance to modern ICU ventilators.”

The GE Healthcare Avance Anesthesia Carestation does not use a piston, nor do any of the other anesthesia systems from GE Healthcare. We use a microprocessor-driven flow control valve system like all major intensive care unit ventilators (including our own Engstrom critical care ventilator) marketed in the United States. The excellent performance of the Avance in this study is due to the rapid and frequent sensing (0.25 ms) of pressure in the patient’s lungs *via* the flow sensors, and the rapid response of the flow valves in the ventilator.

The SmartVent system uses a variable orifice flow sensor on both the inspiratory and the expiratory side of the breathing system. These flow sensors incorporate pressure sensors on either side of a bidirectional Mylar flap. As gas flows through the sensors and encounters the flap, a pressure difference is created between the two sides of the flap. If there is a lot of gas flowing, the pressure difference between the two sides of the flap is more pronounced. If the gas flow is less, the pressure differential is less pronounced. The Mylar flap flexes more or less (hence the variable orifice attribute) depending on the flow, which

makes the sensor accurate across the complete flow range. This also allows the SmartVent to cover the complete patient range from tiny neonates to obese adults. The SmartVent uses these pressure differential measurements on the inspiratory side to determine the total flow rate (fresh gas and flow from the bellows). This allows for tidal volume compensation, so that the correct tidal volume is delivered, regardless of fresh gas flow, oxygen flush, or compliance losses in the breathing system.

Because the flap is bidirectional, the SmartVent notices which side of flap has a greater pressure and so can determine flow direction, allowing for notification of the clinician if a reverse flow condition occurs in the circle system. The inspiratory flow sensor also communicates pressure changes in the patient’s lungs to correctly deliver pressure-control ventilation, or to limit the airway pressure in volume-control ventilation. The inspiratory flow sensor also responds to negative flow during spontaneous modes of ventilation, such as pressure-support and synchronized mandatory ventilation. The inspiratory flow sensor, microprocessor, and flow valves give perioperative patients the advantages of critical care ventilation. The expiratory flow sensor is an independent monitor that reports the patient’s exhaled tidal and minute volumes, and is not involved in volume compensation, ventilation calculations, or responses.

GE Healthcare Anesthesia Systems also have a multiple-breath, standing bellows, unlike the other anesthesia units cited, which gives visible confirmation of the integrity of the breathing system and the adequacy of fresh gas flow. Fresh gas is delivered directly to the inspiratory limb, not through the breathing bag, as is the design with a decoupled piston ventilator. Therefore, changes made by the user to fresh gas and anesthetic agent settings take effect at the patient much more quickly in the Avance Carestation than in fresh gas decoupled systems that use the breathing bag as a reservoir. The volume of the breathing circuit is only 2.7 l, so the Avance has less than half the volume (and time constant) of fresh gas decoupling systems, especially when those decoupled systems use a 3-l rebreathing bag.

AnnMarie Preece, B.S., GE Healthcare, Madison, Wisconsin.
annmarie.preece@med.ge.com

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(Accepted for publication July 12, 2007.)

In Reply:—We thank AnnMarie Preece for her comments on our article,¹ which help us to clarify the description of some specifications of the Avance ventilator. Indeed, the inspiratory flow triggering on this ventilator is selectable from 0.2 to 10 l/min. However, this point, like the other comments from Ms. Preece, does not modify the reported results of the article about the Avance ventilator. In table 1 of our article, which reported the main characteristics of anesthesia and intensive care unit ventilators tested, some characteristics of pressure-support ventilation in the Avance ventilator

should be corrected as follows: The pressurization phase in pressure-support ventilation allows a slope adjustable from 0 to 500 ms, and the expiratory trigger is adjustable from 5% to 50% of peak inspiratory flow. As stated in the article, the Fabius and Primus are “piston ventilators,” which use an electric motor to compress gas in the breathing circuit, creating the driving force for mechanical insufflation to proceed. However, the Avance is not a piston ventilator as stated in the article, but is one of the last generation of “bellow ventilators” which uses a microprocessor-driven flow con-

trol valve system like all major intensive care unit ventilators combined with a dynamic regulation of the internal compliance of the ventilator. These features may explain in part why these more recent anesthesia ventilators have comparable performance to modern intensive care unit ventilators.

As stated in the editorial by Hossam Tantawy and Jan Ehrenwerth,² this study permitted an important addition to the literature because it shows that technology improvements in traditional intensive care unit ventilators can be transferred to the operating room and the anesthesiologist must understand the pressure-support ventilation indications and limitations, know how to implement it properly, and determine its cost effectiveness.

Samir Jaber, M.D., Ph.D.,* Didier Tassaux, M.D., Mustapha Sebbane, M.D., Yvan Pouzeratte, M.D., Anne Battasti, Xavier Capdevila, M.D., Ph.D., Jean-Jacques Eledjam, M.D., Ph.D., Philippe Jolliet, M.D. *Hôpital Saint Eloi, Montpellier, France. s-jaber@chu-montpellier.fr

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(Accepted for publication July 12, 2007.)

Anesthesiology 2007; 107:1030

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Can 1% Sevoflurane with 5 ng/ml Remifentanyl Prevent Implicit Memory?

To the Editor:—We read with much interest the recent report “When Is a Bispectral Index of 60 Too Low?” by Manyam *et al.*¹ They wrote that 1% sevoflurane is usually sufficient to produce clinically adequate anesthesia with an effect site concentration (Ce) of 5 ng/ml remifentanyl. They used the Observer’s Assessment of Alertness/Sedation scale to assess the sedation level simultaneously measuring the Bispectral Index (BIS) and auditory evoked potential index (AAI), and they concluded that targeting a BIS less than 60 or an AAI less than 30 may result in an excessively deep anesthetic state during sevoflurane-remifentanyl anesthesia.

Previous reports^{2,3} have shown that opioids reduced the Ce of anesthetics at loss of consciousness. We think this phenomenon does not always indicate that opioids decrease the adequate Ce of anesthetics for maintenance of anesthesia. We cannot assess the level of hypnosis at the surgical level of anesthesia with the Observer’s Assessment of Alertness/Sedation scale, because the scale is always 0 when BIS decreases below 50, 40, or less. Currently, there is no direct evidence whether opioids can reduce the Ce of anesthetics to maintain an adequate level of hypnosis for surgery when a medium to high dose of opioids is used. At the surgical level, we cannot tell whether electroencephalogram-derived parameters, such as the BIS, are adequate to indicate the level of hypnosis, because we cannot assess the adequacy of level of hypnosis other than by using the monitors based on electroencephalographic or evoked potentials.

The major goal of general anesthesia is to maintain unconsciousness and amnesia during surgery. And we should pay much attention to memory, especially “implicit memory.” In most cases, the concentration of anesthetic that prevents explicit memory is lower than the minimum alveolar concentration required to follow commands (MAC_{awake}). However, implicit memory formation does occur even when the concentration is above MAC_{awake}. Iselin-Chaves *et al.*⁴ reported that there was evidence of memory for words presented during adequate anesthesia (BIS 41–60) but not during deep anesthesia (BIS 21–40). At this point, there is no consensus about whether we should prevent implicit memory; our view is that we should prevent it because it might have influence on patients’ postoperative emotion or behavior. To prevent memory formation, it is important to block sensory inputs from reaching the brain, especially noxious inputs and auditory inputs. Patients who experience awareness during anesthesia often report pain sensations or hearing sounds or voices during surgery. Remifentanyl, 5 ng/ml, is sufficient to block the noxious stimuli in most cases. As for auditory inputs, we can assess them by auditory evoked potentials. AAI is calculated from the track length of auditory evoked potential waveform in a specific period. There is the possibility

that auditory inputs are processed in the cortex and implicit memory might be retained when AAI values are high. Therefore, it would be better to keep the AAI values less than 30 if we wish to prevent implicit memory. Considering these facts, it is too early to draw the conclusion that targeting a BIS less than 60 with the Ce of remifentanyl at 5 ng/ml results in an excessively deep anesthetic state.

By the way, BIS values become 40–50 when keep the end-tidal concentration of sevoflurane at 1.2–1.4% with 0.2 $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ remifentanyl. At this level, AAI becomes less than 30.⁵ Katoh *et al.*⁶ reported that, during plain sevoflurane anesthesia, the average value of BIS was 60 at 1.0% and 40 at 1.5%. When we keep the end-tidal sevoflurane at 1.2–1.4%, most patients can quickly recover from anesthesia after surgery. Compared with the data by Katoh *et al.*,⁶ BIS values shown in figure 5 or figure 7 seemed to be higher, which suggested that the Ce of sevoflurane would not reach equilibrium in the current study. So the relation between BIS values and end-tidal concentration of sevoflurane would not be correct considering the steady state.

Finally, we think it would be wise to keep the end-tidal concentration of sevoflurane at 1.2–1.4% (a little higher than 1.0%) until enough data are accumulated.

Satoshi Hagihira, M.D., Ph.D.,* Masaki Takashina, M.D., Takahiko Mori, M.D., Takashi Mashimo, M.D., Ph.D. *Osaka University Graduate School of Medicine, Osaka, Japan. hagihira@anes.med.osaka-u.ac.jp

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(Accepted for publication July 13, 2007.)

In Reply:—We appreciate the interest of Dr. Hagihira *et al.* regarding our recent article.¹ We would like to address their concerns regarding our methodology and recommendations.

In our volunteer experiments, we were careful to allow adequate time for equilibration after each step change in sevoflurane to achieve a pseudo-steady state. At each time point, we confirmed a stationary anesthetic level by directly measuring the end-tidal concentration of sevoflurane using a calibrated agent analyzer. An additional indirect confirmation of equilibration was the steady Bispectral Index[®] (BIS[®]; Aspect Medical Systems, Norwood, MA) and Auditory Evoked Potential ARX Index (AAI; Alaris Medical Systems, San Diego, CA) measurements in unstimulated volunteers. This is similar to the methodology used by Katoh *et al.*² Although the “average” BIS values of 1.0% and 1.5% sevoflurane reported by Katoh *et al.*² are different than those that we reported, we believe that both studies had achieved adequate equilibration because the observed and average values of the BIS at each of the modified Observer’s Assessment of Alertness/Sedation scores were similar. In addition, the range of BIS values observed by Katoh *et al.*² when the end-tidal sevoflurane was 1.0% (50–75) and 1.5% (30–55) are similar to what we observed.

The discrepancies between the predictions made by Katoh *et al.*² and those made by our model are most likely due to the differences in the raw observed data range and the mathematical models used to fit the raw data. We believe that fitting a third-order polynomial to a smaller range of measured concentrations (0–2.5%) compared with generating a response surface for a wider range of measured sevoflurane concentrations (0–6%) resulted in the different predictions of the average BIS values at the various sevoflurane concentrations.

Preventing explicit recall is a vital goal of general anesthesia. Multiple investigators have demonstrated that opioids consistently reduce both the minimum alveolar concentration (MAC)^{2,3} and the MAC required to follow commands (MAC_{awake})² in addition to decreasing the effect site concentration of anesthetics required to produce loss of consciousness. There should be no reason why the observed pharmacodynamic interactions we observed in our human volunteer laboratory should not be evident during maintenance of anesthesia. Therefore, moderate to high doses of opioid should allow a persistent decrease in the anesthetic required to prevent explicit recall throughout the perioperative period.

We also agree with Dr. Hagihira *et al.* that the importance of implicit recall of intraoperative events is less clear. Unfortunately, implicit recall is much more difficult to evaluate, and therefore, the literature is full of conflicting data on the anesthetic requirements necessary to abolish implicit memory. The data from Iselin-Chaves *et al.*⁴ are thought provoking, but the large variety of anesthetic techniques used does not allow conclusions to be made on the efficacy of moderate to high opioid concentrations to block implicit memory. However, if implicit recall can be prevented by diminishing noxious stimulation-induced activation of the

auditory centers⁵ or the arousal centers,⁶ then moderate- or high-opioid anesthetic techniques may be useful alternatives to hypnotic-based anesthetics. In addition, if “too deep” anesthesia is indeed associated with a worsening in 1-yr survival,⁷ the use of low or moderate doses of hypnotic with a balance of analgesia (*via* regional anesthesia, systemic opioids, or a combination) may be desirable.

It is important to reemphasize that our data were derived from volunteers where the electroencephalogram-based assessments of depth of anesthesia were performed independent of response to painful stimuli. This is in contrast to what typically occurs in the operating room, where clinicians make electroencephalogram-based and subjective assessments of sedation in the presence of continuous surgical stimuli. It is conceivable that patients may require more sevoflurane and remifentanyl than volunteers to achieve the same degree of electroencephalogram-based sedation. However, based on the current data and the available literature, our recommended anesthetic regimen should prevent explicit recall and may inhibit the formation of implicit memories.

Dhanesh K. Gupta, M.D.,* Sandeep C. Manyam, Ph.D., Ken B. Johnson, M.D., Talmage D. Egan, M.D. *Northwestern University Feinberg School of Medicine, Chicago, Illinois.
dhanesh-gupta@northwestern.edu

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(Accepted for publication July 13, 2007.)

Does Combined Epidural–General Anesthesia Increase Risk over Either Technique Used Alone?

To the Editor:—Having read the editorial views expressed by Heitmiller, Martinez, and Pronovost,¹ I commend the goal of implementing procedures to improve patient safety in the practice of anesthesia. Certainly, changes in teaching practices and techniques that allow safer placement and management of central venous access catheters offer significant opportunities to have a profound impact on serious morbidity and mortality. Changes to connection ports for epidural and intravenous lines to make infusion of unintended substances more difficult also has some potential to decrease risk, although likely on a much smaller and less cost-effective scale when the relatively low incidence of misconnects is taken into

account. Clear and better labeling of epidural catheters may provide nearly equal risk reduction at a lower cost. Further study may help to elucidate the ideal solution.

I do have concern, however, about the authors’ remarks in relation to three deaths in patients receiving combined general–epidural analgesia, specifically regarding the authors’ comment, “This is concerning, although perhaps not surprising,” going on to imply that mortality among other complications may be additive by combining the total risk of the two techniques performed independently. Although it may be true that neurologic injury, failure to intubate, or infusion of an incorrect substance

are all independent risk factors, there is no suggestion in the vast amount of literature published that indicates regional anesthetic techniques either alone or combined with general anesthesia increase mortality or major morbidity. Previous studies,² which demonstrated reduction in mortality with epidural analgesia, may simply have reflected trials conducted before the introduction of routine venous thromboembolism prophylaxis and before changes in the management of patients with coronary artery disease (including perioperative β -blockers). Some recent large trials on high-risk patients with appropriate conventional perioperative management in both groups do not show a decrease in mortality and major morbidity with the addition of epidural analgesia, but also show no increase in mortality or major morbidity with the combined approach.^{3,4} In large studies using the Medicare database, the use of combined general-epidural anesthesia has been associated with a reduction in mortality.^{5,6} Therefore, the authors' suggestion that mortality may be increased by combining epidural with general anesthesia is contrary to the vast body of scientific research.

Neurologic injury, as the authors note, is an independent risk of epidural analgesia, and patients should be given a reasonable informed consent regarding common as well as potentially serious risks of any procedure including the risk of nerve damage from surgical causes, tourniquet, positioning, and stretch, among many other causes. There is unfortunately little consensus on what constitutes appropriate consent, because it is virtually impossible to list every possible complication. Do we go so far as to specify the estimated potential of rare but serious complications such as epidural hematoma (1:150,000),^{7,8} abscess (>1:100,000),⁹⁻¹¹ permanent neuropathy (2.19:10,000),¹² and paraplegia (1:100,000),¹² or is it reasonable to generalize with statements such as "serious events can occur, not limited to death, heart and lung problems, aspiration, allergic reaction, nerve damage, and others"? Do we quote the overall probability of postdural puncture headache or adjust for whether a first-year resident is doing the procedure *versus* a highly skilled practitioner or perhaps offer no estimate of the incidence? Are we equally remiss in offering true informed consent if we do not offer patients the 30-40% reduction in pain conferred by epidural analgesia,¹³ the improvements in patient satisfaction that are consistently noted and potentially decreased mortality?^{5,6} Pain is witnessed on a daily basis and is of significant concern to patients. Serious neurologic injury secondary to epidural analgesia at the estimated rates may not be witnessed by many practitioners in the course of their career. Placement of epidural catheters by anesthesiologist who are not proficient in the technique and the lack of appropriate systems for postoperative management could potentially not only increase risk of serious complication but also greatly reduce any analgesic benefit and would not be recommended.

Despite disagreement with the authors' comment on the risk of mortality with combined epidural-general anesthesia and the sense that there

may be an unjustified bias toward avoiding this practice, I commend the group for bringing to the forefront the importance of improving patient safety. I fully respect the outstanding work these individuals have done to implement strategies that have improved patient safety and their continuing efforts to find new areas for improvement in patient safety.

Jeffrey M. Richman, M.D., The Johns Hopkins University, Baltimore, Maryland. jrjchma1@jhmi.edu

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(Accepted for publication July 26, 2007.)

Anesthesiology 2007; 107:1032-3

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Perioperative Central Venous Cannulation: It Is Time for Action

To the Editor:—I read with great interest the excellent article by Dr. Hove *et al.*¹ detailing their closed claims review of deaths related to anesthesia in Denmark (1996-2004). The accompanying editorial highlights the preventable deaths related to the conduct of central venous cannulation (CVC).^{1,2}

There has been a significant lag in quality improvement for CVC since its role in patient mortality and morbidity was highlighted in 1999 by the Institute of Medicine Report *To Err Is Human*.^{*} It is

time for the worldwide anesthesia community to develop and implement evidence-based guidelines, teaching, and standardization for the practice of CVC.

To my knowledge, there is no published practice guideline for perioperative CVC, despite the fact that it is a common perioperative procedure associated with significant medical error and consequent serious patient risk. The literature review that would be part of this guideline's development would quickly prove the pivotal role of ultrasound guidance. The discussion points at this juncture are not about whether ultrasound guidance is required, but rather about the aspects of its application, such as the superiority of real-time imaging, the role of a needle guide, Doppler imaging, and/or two-dimensional imaging.^{3,4}

* *To Err Is Human: Building a Safer Health System*. Edited by Kohn LT, Corrigan JM, Donaldson MS. Washington, DC, National Academy Press, 1999. Available at: <http://www.nap.edu/catalog/9728.html>. Accessed April 21, 2007.

Furthermore, there are initiatives in progress that will greatly assist in achieving the consistently safe practice of CVC throughout medical practice. One example of these initiatives is instructive procedural videos available through the Internet, such as the series launched recently by the *New England Journal of Medicine*.⁵ Perhaps the leading anesthesia journals would follow this example and include not only CVC but all procedures relevant to perioperative medicine. Another example is the development and validation of standardized teaching methods for CVC such as the tissue model technique from internal medicine.⁶ This avenue of endeavor might be a fruitful application for anesthesia simulation as well.

I congratulate Dr. Hove *et al.* on their excellent article, which adds further emphasis to the perioperative imperative to make CVC safe for all of our patients all of the time.

John G. T. Augoustides, M.D., F.A.S.E., Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania. yiandoc@hotmail.com

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(Accepted for publication July 26, 2007.)

Anesthesiology 2007; 107:1033

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Medication Error: A Leading Cause of Anesthesia-related Morbidity and Mortality

To the Editor:—We read with great interest the analysis of anesthesia-related deaths registered by the Danish Patient Insurance Association.¹ Hove *et al.*¹ are to be congratulated for reporting these important results. The authors categorized 24 fatal cases by their underlying causes: airway management, ventilation management, placement of a central venous catheter, medication errors, transfusion error, infusion pump problems, and regional blockade. We noted that 8 of the 24 anesthesia-related deaths described were most likely attributable to a drug error: 4 overdoses (benzodiazepines, methohexital, thiopental, nitroglycerine), 3 infusion pump errors, and 1 patient likely received a large intrathecal dose of mepivacaine. Therefore, the frequency of medication-related incidents exceeded the 4 deaths that resulted from loss of the airway and 4 from complications related to central venous line insertion. It seems that the single most common cause of anesthetic-related death was a drug error.

These findings are consistent with an analysis from the Canadian Medical Protective Association of closed medicolegal claims against anesthesiologists.² The Canadian Medical Protective Association provides malpractice insurance for most physicians in Canada. From 1998 to 2002, there were 232 closed legal actions against anesthesiologists. Medication error was the most common cause involving 52% of the claims. It is noteworthy that the American Society of Anesthesiologists Closed Claims Project reports the proportion of drug errors as 4%.² This number has been consistent throughout the 1980s and 1990s. Reasons for the discrepancy in the relative frequency of medication errors reported in the American Society of Anesthesiologists Closed Claims Database from those in Denmark and Canada requires further exploration but may be attributed, in part, to differences in the categorization of root causes.

The impact of drug error in anesthetic practice is not new and will not surprise experienced anesthesiologists. A survey by the Canadian Anesthesiologists' Society found that 85% of participants had experienced at least one drug error or "near miss."³ Most of these errors were of minor consequence; however, 1.8% resulted in major morbidity

(cardiac arrest, stroke, permanent injury) or death. The misidentification of a syringe was the most common cause. In 1984, Cooper *et al.*⁴ published a classic analysis of critical incidents in anesthesia management. Breathing circuit disconnect was the most common identified factor; however, reanalyzing their data set indicates that medication-related events far exceeded airway and ventilation problems. Of a total of 507 incidents, 169 were attributed to errors or problems in drug administration. Equally important, when incidents with "substantive negative outcomes" were further analyzed (defined as mortality, cardiac arrest, cancelled operative procedure, or extended recovery room, intensive care unit, or hospital stay) approximately 25% of them resulted from a drug error.

Together, these studies suggest that the impact of medication error has been underestimated by the lack of a common taxonomy for anesthesia-related adverse events. More importantly, the data beseech us to acknowledge the problem and develop innovative strategies to reduce the likelihood of a drug error in anesthetic practice.

Rachel Meyer, M.D.,* Beverley A. Orser, M.D., F.R.C.P.C., Ph.D., Robert Byrick, M.D., F.R.C.P.C. *University of Toronto, Toronto, Ontario, Canada. rachel.meyer@utoronto.ca

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(Accepted for publication July 26, 2007.)

Anesthesiology 2007; 107:1034

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In Reply:—We thank Dr. Richman for his thoughtful letter in response to our editorial¹; however, we believe he misinterpreted our statement regarding risk of combined general and regional anesthesia. We never said, or intended to imply, that mortality *per se* is increased with a combined general and regional anesthetic technique when compared with a single technique. Rather, we said that basic probability tells us that the risk of complications will be greater when two techniques are combined as compared with each technique alone. We agree that combined techniques may result in benefits, and those benefits may offset the risk.

When deciding whether a patient should have a combined anesthetic technique *versus* a single technique, the patient and anesthesiologist must decide whether the benefit outweighs the risk. The patient should have the opportunity to make an informed choice. As an example, for a patient who requires general anesthesia for a procedure and is considering the addition of a regional technique, is the benefit of 2 days of modestly improved pain control with a combined anesthetic technique (visual analog scale pain score of 3 with parenteral opioids *vs.* 2 with epidural analgesia)² worth the risk of nerve injury? Although our current knowledge of true estimates of risk is imprecise, we do know that some risks of regional anesthesia are very rare, such as epidural hematoma, but others, such as nerve injury, are more common. Because long-term complications are generally identified by retrospective surveys of clinicians, claims, or chart reviews rather than prospective clinical follow-up of patients, the true risk of injury may be higher than that published for any given anesthetic technique. Are patients informed that they may be trading a potential long-term complication for a short-term gain?

Anesthesiology 2007; 107:1034

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In Reply:—We gratefully acknowledge the interest that Dr. Augoustides and Dr. Meyer *et al.* have shown in our article on closed claims review of deaths related to anesthesia in Denmark.¹

Dr. Augoustides makes an insightful comment in noting the lack of a practice guideline for perioperative central venous cannulation. We can only agree that such a guideline would improve the patient safety.

Dr. Meyer *et al.* indicate that medical errors have been underestimated. We agree that medical errors probably have been underestimated in previous reports, but we have to point out that our data do not allow us to conclude the fraction of medical errors of the total because not all deaths from anesthesia are reported to the Danish Patient Insurance Association.

Anesthesiology 2007; 107:1034-5

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Epidural Complications across the Globe

To the Editor:—We read with interest C. M. Cameron *et al.*'s review of epidural morbidity,¹ having recently published a similar survey of our own.² As in their review, we examined more than 8,000 cases of patients receiving epidural analgesia after surgery over a 6-yr period in our hospital. This compares with their 16 yr, perhaps reflecting not only the different sizes of our two centers, but also differences in practice. Despite this, the results are remarkably similar. We demonstrated an epidural abscess rate of 1:1,350 (*vs.* 1:1,368) and an epidural hematoma rate of 1:2,700 (*vs.* 1:4,105). However we also identified 3 cases of meningitis (1:2,700). Although this is a recognized complication of postoperative epidural infusions, Cameron *et al.* do not discuss meningitis and seem not to

have searched their database for this significant complication. We assume that this was an oversight.

The presenting symptoms did vary between the patients in the two surveys. All of our patients with epidural abscesses displayed back pain, and most had features of meningitic irritation. In contrast, both of these symptoms were rare in their patients. A key point to note from both studies is the delay before clinical presentation of an epidural abscess (6–31 and 5–11 days, respectively). Therefore, almost all patients develop symptoms after the epidural catheter has been removed, and many after discharge home. One of our patients with an epidural abscess did not make a full neurologic recovery. This patient developed symptoms of spinal cord compression at home but did not

Eugenie Heitmiller, M.D.,* Elizabeth Martinez, M.D., Peter J. Pronovost, M.D. *Quality and Safety Research Group and Johns Hopkins School of Medicine, Baltimore, Maryland. eheimil@jhmi.edu

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(Accepted for publication July 26, 2007.)

Lars Dahlgaard Hove, M.D.,* Jacob Steinmetz, M.D. *Rigshospitalet, Copenhagen, Denmark. larshove@dadnet.dk

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(Accepted for publication July 26, 2007.)

return to the hospital until paraplegic. We now provide written advice to patients receiving epidural analgesia, detailing specific signs and symptoms that require medical investigation should they occur after discharge.

Chlorhexidine in alcohol has been our standard solution for skin preparation for many years. We note that in 2004, Cameron *et al.* reverted from chlorhexidine back to an iodine solution for skin preparation. Since then, their incidence of insertion site infections seems to have increased. We wonder whether they think this increase is significant enough to justify returning to an alcoholic chlorhexidine solution.

Common with most of the published data on epidural infections, the implicated organism in their patients was *Staphylococcus aureus*. What was striking in our data was the high incidence of methicillin-resistant *S. aureus*, which was cultured from five of the nine patients with infective complications. Unfortunately, routine methicillin-resistant *S. aureus* screening was not performed during our survey. We were thus unable to investigate whether methicillin-resistant *S. aureus* colonization of the patient, the staff, or the ward predisposed to these complications. We would now, however, be reluctant to insert an epidural catheter in any patient colonized with methicillin-resistant *S. aureus*.

Cameron *et al.* discovered that epidural abscesses were associated with epidural site infection that in turn was related to the duration of epidural catheterization and abdominal or thoracic surgery (odds ratio, 3.3). Interestingly, in our series, insertion of an epidural at a thoracic level did not increase the risk of developing either an epidural abscess or meningitis (odds ratio, 1.18; 95% confidence interval, 0.3–4.7).

Anesthesiology 2007; 107:1035

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In Reply:—We thank Drs. McCabe and Christie for their letter, which describes adverse neuraxial outcomes from their own clinical practice,¹ published in an article almost simultaneously with our own report.² It is indeed reassuring to find that most of their conclusions are consistent with our experience, and overall we hope this will heighten awareness of the insidious problem of epidural space infections and help to avoid long-term adverse outcomes.

In their article, they describe three cases of meningitis as separate adverse outcomes after analgesic epidural infusions, all being diagnosed within 5 days of catheter removal. We did not report any discrete episodes of meningitis, and on searching our database specifically for the term, we have not discovered any such events. It would seem that meningitis *per se* after epidural analgesia is an uncommon event.

The reason for our change to alcoholic Betadine solution for skin preparation was because we found it difficult to see the light pink alcoholic chlorhexidine on the skin, to ensure full coverage of the area. The apparent increase in incidence of site infections subsequent to this is being monitored and will be reviewed at the end of this year. Clinical recommendations tend to favor alcoholic chlorhexidine skin preparation,³ and if our trend is not simply an aberration, we will adopt a return to alcoholic chlorhexidine.

The second spinal hematoma was an intrathecal bleed in patient 3. An L2–L3 epidural was placed for analgesia after removal of a femoral nail and insertion of a hip screw. A “bloody tap” on the first pass complicated insertion of the epidural, which was resited and managed effectively for 2 days. The catheter was dislodged and subsequently removed after 1 h after 5,000 U dalteparin, and the patient subsequently developed neck stiffness and nausea. Magnetic resonance

Perhaps, as they suggest, it is the duration of the epidural infusion that is the more critical factor. We recommend that epidurals are removed within 3 days of insertion.

Although Cameron *et al.* identified two spinal hematomas, only one was epidural. We would be interested to know where the other hematoma was located in relation to the dura and how it presented (*i.e.*, whether it affected patient 1 or patient 3).

When we presented the results of our survey, both locally and nationally, the frequency with which these complications occur came as a surprise to many. It is reassuring that on the other side of the globe, the incidence seems to be similar. Only through continued audit of our practice can we determine the true risk of the interventions we undertake to benefit rather than harm our patients. The Royal College of Anaesthetists has recently started a national audit of these complications in the United Kingdom, and we would commend to others to consider a similar register.

Shane E. T. McCabe, M.B.B.S., F.R.C.A.,* Iain W. Christie, F.R.C.A. *Christchurch Hospital, Christchurch, New Zealand. shanemccabe@totalise.co.uk

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(Accepted for publication August 20, 2007.)

imaging of the spine revealed intrathecal blood from L1 to L3. The patient's symptoms resolved rapidly with no specific management.

We support the recommendations of Drs. McCabe and Christie for auditing these complications. As part of a statewide government initiative commissioned by the Victorian Quality Council, Department of Human Services (Melbourne, Victoria, Australia), we have developed a toolkit for measuring Acute Pain management outcomes.* Included in this is a pro forma that includes specific fields for recording adverse neuraxial outcomes after epidural analgesia as well as other adverse events such as respiratory depression necessitating naloxone treatment associated with systemic opioids. This is being implemented throughout the state of Victoria and is hoped to lead to a more accurate and balanced understanding of the incidence of all serious adverse outcomes relating to acute pain management.

Finally, the provision by Drs. McCabe and Christie of written advice at discharge to patients who have had an epidural is an initiative to be commended.

David A. Scott, M.B., B.S., Ph.D., F.A.N.Z.C.A., F.F.P.M.A.N.Z.C.A.,† Christie M. Cameron, M.B., B.S., F.A.N.Z.C.A., Michael J. Davies, M.B., B.S., M.D., F.A.N.Z.C.A., Wendy M. McDonald, B.N., Grad. Dip. (Crit. Care). †St. Vincent's Hospital Melbourne, Victoria, Australia. david.scott@svhm.org.au

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Etomidate Unlikely to Have Induced Pacemaker-mediated Tachycardia

To the Editor:—Although I agree that the case “Etomidate-induced Pacemaker-mediated Ventricular Tachycardia”¹ could represent an important contribution to our literature, I am concerned that the data presented in this article cannot justify the conclusion that myoclonus from the etomidate was actually responsible for this pacemaker-driven tachycardia (this is not actually an arrhythmia). Therefore, this article might be cited by future practitioners when, in fact, etomidate likely made little or no contribution to the ventricular pacing shown at 140 beats/min.

The rate-responsive mechanism in this Medtronic Prodigy pacemaker (St. Paul, MN) is a piezo crystal attached to the case of the pacemaker.² It is activated by case deformation (pressing on the case). The rate-responsive algorithm has a number of programmable settings that determine how the pacemaker will change the pacing rate (called activity-indicated rate) due to stimulation of this crystal. These settings include the following (not specified in the article): lower pacing rate, upper activity rate, activity threshold, activity rate response, acceleration time, and deceleration time. In the default setting, the acceleration time is 0.5 min, so in 30 s, the pacemaker will achieve 90% of the difference between the current paced rate and the higher (calculated) activity-indicated rate consistent with the activity level.

The default deceleration time is 5 min and is of interest here. Upon abrupt cessation of activity, the pacing rate begins decreasing almost immediately, and it will decrease over 5 min by 90% from the current (high) paced rate to the new, lower activity-indicated rate. In this case, assuming that the lower pacing and upper activity rates had been programmed to 70 and 140 beats/min, respectively, the actual pacing rate would have been approximately 122 beats/min at 30 s, 109 beats/min at 1 min, and 92 beats/min at 2 min (fig. 1). No time intervals were reported in the article, but 30 or more seconds likely elapsed from the last patient movement to the reprogramming of the pacemaker.

Therefore, I believe that the continued pacing at 140 beats/min suggests that something else was affecting the pacemaker. It might have been the weight of the programming head. In fact, the Prodigy manual warns: “Clinical studies of activity rate responsive pacemakers have reported instances of . . . an increase in the pacing rate up to the programmed Upper Activity Rate . . . by pressing the programmer head over the pacemaker.”

The stability of the heart rate (determined by the intervals between the QRS complexes) in the author’s published figure 2 suggests that the pacemaker was sensing continuous mechanical activity. If the mechanical activity had ceased, the pacing rate would have been slowing, which would result in the successive lengthening of the intervals between the QRS complexes.

This article is an important reminder that pacemakers (and implanted cardioverter-defibrillators) are mere computers that respond to electrical and environmental signals in a predictable fashion. The authors should have emphasized that these devices can produce unusual pacing behavior that might be misinterpreted by clinicians (likely true in this case) and, therefore, induce inappropriate treatment, which did not happen here. For example, pressure on the chest over a pacemaker or defibrillator due to instruments or surgical personnel, as well as mechanical activity over a pacemaker or defibrillator from surgical site preparation, can lead to a paced tachycardia approaching the upper activity rate. In fact, misinterpretation of pacemaker

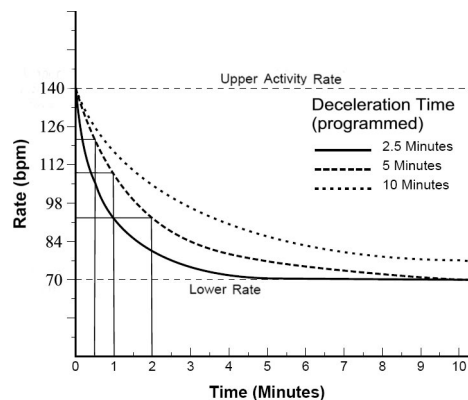


Fig. 1. The deceleration operational graph from the Medtronic Prodigy Pacemaker Manual on page 11-9 was adapted to the presumed programmed parameters for this case to predict the pacing rate after abrupt cessation of activity. For this adaptation, the lower rate was assumed to be 70 beats/min, the upper activity rate was assumed to be 140 beats/min, and the deceleration time was assumed to be 5 min, the default setting. The vertical lines at 30 s, 1 min, and 2 min were added to show that the expected paced rate would have been 122, 109, and 92 beats/min, respectively, after the cessation of mechanical activity detected by the pacemaker and interpreted as patient activity. Original artwork copyright 1995 by Medtronic Corporation, Minneapolis, Minnesota, and reproduced with permission of Medtronic, Inc. The Medtronic Corporation takes no responsibility for the validity or accuracy of any modification to the illustration.

(pseudomalfun) behavior is common,³ and sometimes it leads to disastrous results with patient injury.⁴

Finally, the comment in this article that “It is essential that the pacemaker should be programmed to an asynchronous mode” is not justified by any part of the case presentation, the discussion, any literature, or any manufacturer recommendation. Pacemaker and implantable cardioverter-defibrillator manufacturers do recommend a comprehensive interrogation of any device that has “seen” an external cardioversion or defibrillation.

Marc A. Rozner, Ph.D., M.D., University of Texas M. D. Anderson Cancer Center, Houston, Texas. mrozner@mdanderson.org

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(Accepted for publication August 31, 2007.)

Anesthesiology 2007; 107:1037

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In Reply:—We thank Dr. Rozner for his interest in our case report, “Etomidate-induced Pacemaker-mediated Ventricular Tachycardia,”¹ and we appreciate the opportunity to address some of the points he raises in his letter.

He first questions our use of the term *pacemaker-mediated ventricular tachycardia*, asserting that the rhythm we reported is not actually an arrhythmia. We agree that the rhythm we observed was indeed not a clinical arrhythmia. Perhaps a more accurate description might be his term, *abnormal pacemaker-driven tachycardia*.

Dr. Rozner then refers to the programmable settings that affect the rate-responsive function. In our patient, the lower pacing rate and upper activity rates were 70 and 160 beats/min, activity threshold was medium, acceleration time was 0.5 min, and deceleration time was 5 min. Dr. Rozner’s discussion of the rate-responsive mechanism demonstrates his well-known expertise² in the area of implantable cardiac devices, but his calculation using the deceleration time is not relevant to our case. His letter examines the situation where the stimulus activating the rate-responsive function terminates, and the pacing rate thus begins to decrease gradually as he describes. However, we have stated that the rapid rhythm (shown in fig. 2 of our case report) ceased immediately after the rate-responsive function was disabled. That is, the stimulus (myoclonus) did not stop, but instead the pacemaker was reprogrammed to stop responding to the stimulus. Once the rate-response function is disabled, a pacemaker will immediately revert to its baseline settings (VVI at the lower pacing rate of 70 beats/min, in our case) as shown in figure 2.

Therefore, we agree with Dr. Rozner’s statement that “the pacemaker was still sensing activity,” but we maintain that etomidate-induced myoclonus is a much more plausible explanation than the weight of the programming head without any additional pressure being applied. If the programming head were solely responsible for the rapid ventricular pacing (as Dr. Rozner suggests), we would expect to have gotten the same result each time the rate-responsive function was restored, not just the first time.

We have considered other possible causes for tachycardia in this setting, such as erroneous programming of the pacemaker (e.g., if the oper-

ator accidentally turned off the mode switch in a patient with atrial fibrillation, the device might pace at the upper activity rate) or inappropriate atrial sensing (e.g., 1:2 sensing of an atrial rate of 280 beats/min). However, we believe that the observed myoclonic activity led to this case of pacemaker-driven tachycardia. This abnormal rate-response is clearly corroborated by the data shown in figure 2. Our hypothesis is also supported by the fact that there were no further episodes of rapid ventricular pacing with reactivation of the rate-responsive function, after waiting to ensure complete cessation of the myoclonus. Persistent myoclonic activity for this amount of time is reasonable; etomidate-induced myoclonus has been reported to last up to 8 min.³

Finally, Dr. Rozner takes exception to our recommendation that the pacemaker be set to an asynchronous mode. Although we agree that this statement is not valid for a cardioversion, it remains sound advice for anesthesiologists who take patients to the operating room, where unipolar electrocautery is routinely used, and we apologize for any confusion this generalized statement may have caused.

Michael D. Altose, M.D., Ph.D.,* Elias León-Ruiz, M.D. *University Hospitals Case Medical Center, Cleveland, Ohio. michaelaltose@yahoo.com

The authors thank Robert N. Goldstein, M.D. (Assistant Professor of Medicine, Director, Cardiac Device Clinic, Division of Cardiology/Electrophysiology, University Hospitals Case Medical Center, Cleveland, Ohio), for his assistance in the preparation of the manuscript.

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(Accepted for publication August 31, 2007.)

Anesthesiology 2007; 107:1037–8

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Potential Hazard Associated with a Laryngoscope Blade

To the Editor:—A problem was recently encountered with one of our laryngoscope blades (Classic Mac 3 FO; Heine Optotechnik, Herrsching, Germany) that could have resulted in significant consequences. During cleaning and high-level sterilization of the blade, it was noted that the distal, rounded tip of the blade had become loose to the point where it could easily be detached from the body of the blade (fig. 1). Most anesthesiologists are under the impression that the metal blade is made of one single piece, but in fact the rounded tip is welded onto the blade.

This defective blade was produced in February 2003 and has been used at our hospital on a regular basis since. However, it is not known how many times this particular blade has been used. It is difficult to determine whether the defect resulted from a manufacturing problem or from rough handling (e.g., dropping of the blade to the floor). The finding was reported to Heine Canada, Health Canada, and the Federal Drug Administration, and none of them is aware of a similar case.

Independent of the cause, the potential implications for the patient remain the same.

If the blade had been used without the protective tip, this could easily have lead to severe mucosal trauma because the edges of the unprotected

blade tip are remarkably sharp and rugged. In the case where the tip would still be attached to the blade upon insertion of the laryngoscope blade into the hypopharynx, the small metal tip could be dislodged during



Fig. 1. The loose tip of the laryngoscope blade (partially reattached for illustration purposes) as noticed after cleaning and sterilization.

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

intubation and projected into the tracheobronchial tree, requiring rigid bronchoscopy for removal (if detected immediately) or leading to a significant range of complications, such as segmental atelectasis, obstructive emphysema, pneumonia, or even perforation.

After the detection of the problem, all blades at our institution were inspected, but none was found to have the same defect. Although this seems to be a rare finding, it is recommended to check the integrity of the

laryngoscope blade on a regular basis. A quick inspection with manual pulling on the tip may help to prevent serious complications.

Igor Luginbuehl, M.D.,* Keith Matthews, R.T. *University of Toronto, Hospital for Sick Children, Toronto, Ontario, Canada. igor.luginbuehl@sickkids.ca

(Accepted for publication July 3, 2007.)

Anesthesiology 2007; 107:1038

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In Reply:—We were most concerned to learn of the issue with the Fiber Optic Laryngoscope blade at the Hospital for Sick Children in Toronto. Both Mr. Matthews and Dr. Luginbuehl were very helpful in bringing the information to our attention and coordinating matters to ensure proper findings.

It is important to note that the blade was sent to us for further examination. We tested the blade completely and examined it completely through a high-power microscope. We were not able to determine exactly why the blade tip became separated; however, we believe that there may not have been sufficient soldering to hold the tip in place if the blade were to receive a shock of some kind. We can only speculate due to the fact that soldering matter could have fallen out after the tip separated.

It is important to note that Heine Optotechnik (Herrsching, Germany) has been manufacturing this product since 1983. Since that time, we have manufactured several million units. This is the first and only reported incident worldwide. We have investigated whether any

possible claim was registered with the Federal Drug Administration, Health Canada, and European authorities. None was found.

Although our blades are single-piece blades, meaning that there is no disassembly possible, the blades actually have 12 parts. As with most products, this one has several components that come together to form the whole. Heine has never claimed otherwise.

We see this incident as isolated and have taken additional precautions to verify all current inventories. Further, we are placing specific emphasis on this part of the manufacturing process to ensure this incident does not repeat itself.

It is important to note that Heine manufactures all products to the highest International Organization for Standardization Quality Standards. Our commitment is to manufacture the best products in class in the marketplaces we serve.

Ben St. Jean, Heine USA Ltd., Dover, New Hampshire. www.heine.com

(Accepted for publication July 3, 2007.)

Anesthesiology 2007; 107:1038-9

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Misleading Behavior of Masimo Pulse Oximeter Tone during Profound Bradycardia

To the Editor:—Pulse oximetry is a basic monitoring technology that we are all familiar with and can, at times, take for granted. In an operating room (OR), we can gain a great deal of information from the sound of the tone generated by the pulse oximeter without looking at the monitor. We can tell the patient's saturation and easily detect any changes with it; we can tell the heart rate and detect any sudden changes. We can even catch arrhythmias if we have some experience and are paying careful attention. In recognition of the value of continuous audible information, a recent revision to the American Society of Anesthesiologists Standards for Basic Anesthetic Monitoring requires that the "variable pitch pulse tone" be audible to the anesthesiologist when pulse oximetry is used.

After a recent change in pulse oximetry technology, we encountered a major problem that was not apparent during the trial, conversion period, or our early fully installed experience. We write this letter because we believe that this problem significantly erodes the reliability of this pulse oximeter as a monitor in the OR, and hence is of general interest to your readers.

The problem has been noticed only in the OR and documented twice, both times in September 2006. In these cases, the patient experienced a profound bradycardia, followed by a pause, and accompanied by a steady heart rate tone being produced by the pulse oximeter. In simple terms, the patient's heart stopped and the pulse oximeter tone did not change, giving the anesthesiologist auditory input indicating that an asystolic patient was not having a cardiac arrest. During the entire period, the saturation displayed did not change. This behavior has only been observed

in the OR, perhaps because the OR is the only place in our facility where the pulse oximeter is monitored acoustically. The electrocardiographic monitor correctly displayed a flat line during this period of cardiac arrest, but was not being monitored acoustically.

This problem can be easily duplicated with an automatic noninvasive blood pressure cuff. With a blood pressure cuff on the same arm as the pulse oximeter probe, we have found that the tone will continue through the noninvasive blood pressure cycle, even when the plethysmograph is flat and there is no palpable pulse. For a more quantitative trial, we have used a cuff and a manometer to occlude the arterial inflow to the arm. The oximeter pulse tone will continue for at least 8 s after the cuff has been inflated to a pressure 200 mmHg above the systolic pressure. Thus, there could be 8 s of asystole with no audible indication from the monitor to tell there has even been a change in the heart rate, let alone that the heart has stopped. Our bench testing indicates that if there is any motion artifact, this time is longer and may go on indefinitely under some motion conditions.

We have had discussions with Masimo Corporation (Irvine, CA) about the problem. Doug Harding, V.P. for Quality Assurance at Masimo, told us in November of 2006 that when their algorithm detects a low signal-to-noise ratio, it uses a calculated pulse rate to generate the pulse tone. This allows a tone to be generated even in low-signal (*i.e.*, "noisy") conditions such as motion or low perfusion. A side effect of this design choice is that a tone will continue to be generated even when there is no pulse for up to 8 s. Masimo have specified that their algorithm will detect asystole within 8 s and that the behavior we observed meets that specification. However, anesthesiologists depend on the pulse tone in the OR for a near-instantaneous alert to arrhythmias including sudden severe bra-

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

dyscardia and asystole. The current Masimo technology no longer provides this function, but since they have been informed of the problem, they have begun working on an algorithm adjustment to correct the problem. The new algorithm is expected to appear in stand-alone devices in early 2007. For integrated monitoring systems, this change will occur more slowly.

When new technology is introduced clinically, it may behave in ways that have not been anticipated. This behavior may not be discov-

ered, even after an extensive and thorough clinical trial. Vendors must understand this and be willing to improve the design of their products to improve safety in all environments.

Ryan E. R. Forde, B.S.,* Frederic M. DeBros, M.D., Emily L. Guimaraes, M.D., Warren S. Sandberg, M.D., Ph.D.
*Massachusetts General Hospital, Boston, Massachusetts.
rforde1@partners.org

(Accepted for publication July 3, 2007.)

Anesthesiology 2007; 107:1039-40

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In Reply:—As described in the letter, after initial communication from Massachusetts General Hospital (MGH) on this topic, we provided MGH with a software update that allows the user to select either SmarTone (Masimo Corporation, Irvine, CA), which uses Masimo SET's sophisticated signal processing to enable saturation tones even during low signal-to-noise conditions, or a more traditional pulse tone algorithm, which uses signal morphology only, causing breaks in the tone in the presence of low perfusion or signal interference. We have been offering SmarTone as a user-selectable feature on our monitors for some time now. However, we believe it is important that users understand the value of SmarTone so that they can decide which pulse tone to choose for their particular needs.

The American Society of Anesthesiologists standard that Mr. Forde *et al.* at MGH cite as being diminished by the Masimo SmarTone algorithm is exactly the standard that many other clinicians believe we have enhanced. This American Society of Anesthesiologists standard was established in the context of oxygenation, which implies that tracking the pitch of the tone and thereby indicating changing saturation values was the true intent of the standard. With this understanding and the knowledge that interruptions in the pulse tone caused by low perfusion, motion, and electrical or other interference are common, causing frequent "false alarms" and decreasing the amount of time that the tone was available, we concluded that it would be of clinical value to decrease these interruptions in the pulse tone.

The pulse tone feature on many pulse oximeters is related to the morphology of the plethysmographic waveform, emitting a tone only when a clean pulse signal is recognized. In the presence of low signal-to-noise conditions, the arterial pulse waveform can be virtually impossible to distinguish when looking at the raw plethysmograph. In these instances, most other pulse oximeters either discontinue the tone until a valid pulse signal is recognized or sound a tone based on the noise frequency, one that is not indicative of the patient's pulse. This leads to frequent, long periods without audible information on oxygenation status or false low-saturation indications.

Masimo SET's unique signal processing algorithms, which include five distinct signal processing engines working in parallel, enable identification of the arterial pulse wave under far more of these difficult clinical conditions. This allows Masimo to provide a variable pitch saturation tone during periods of signal disturbance that would cause loss of signal or false saturation tones in conventional pulse oximeters. The SmarTone feature uses real-time signal morphology, similar to

conventional pulse oximetry, to create the tone during periods in which the pulse can be clearly recognized. During periods wherein the pulse signal becomes obscured, Masimo uses its advanced signal processing algorithms to identify the pulse and provides a tone indicative of true oxygen saturation. Feedback from current and prospective customers has been overwhelmingly positive toward this feature.

The case reported in the MGH letter focuses narrowly on one aspect of the SmarTone feature. Because of the sophisticated signal processing involved, during periods of signal disturbance or low perfusion there can be up to an 8-s delay before the cessation of the pulse tone. This short delay is well within internationally recognized performance requirements for heart rate meter response to asystolic events.*

The letter makes reference to a bradycardic period preceding the asystole, during which the pulse tone continued. It is not clear that the surgical team was able to observe whether the pace of the tone was consistent with the rate of the bradycardia. It is likely that the Masimo device tracked the bradycardia, as evidenced by several independent and objective studies that have demonstrated the superior ability of Masimo SET to track sudden changes in heart rate.^{1-3,†‡}

The letter also suggests that a simulation using a blood pressure cuff reproduces this clinical scenario. Although these two scenarios may seem similar, they are actually very different because in one case the heart has stopped, whereas in the other a pressure cuff is occluding the flow from a beating heart. Our own pressure cuff testing, which is supported by the literature,⁴⁻⁶ has proven that it is very difficult to occlude 100% of the blood pulsations on certain patients. Because Masimo SET has been shown to have superior low perfusion performance,⁷⁻¹² it may be the only pulse oximeter to continue to read during cuff inflation when weak arterial pulsations are present. We recently performed more than 100 cuff inflations on various subjects, using a dual bladder tourniquet inflated to 250 mmHg with a Masimo Radical pulse oximeter and a Nellcor N-600 (Nellcor-Covidien, Mansfield, MA) attached to the test arm. In every case, the Radical would display a reading during cuff inflation only if there was a visible arterial pulse wave in the plethysmograph, and it would zero out after the plethysmograph became flat. Overall, the results were inconsistent, with no clear pattern of either monitor (Nellcor or Masimo) zeroing out before the other, and neither monitor consistently zeroed out within 8 s of cuff inflation. We have captured a number of these cases on video and are pleased to share these results upon request.

As discussed above, after initial communication from MGH on this topic, we provided MGH with a software update that allows the user to select either SmarTone or a more traditional pulse tone algorithm. Initial feedback regarding this new software has been positive. Since early this year, SmarTone has been offered as a user-selectable feature on all of our products, allowing clinicians to decide for themselves which pulse tone to use, based on their clinical scenario.

We value the feedback and suggestions of our customers in helping us make our products the safest and best pulse oximeters in the world, and we appreciate the useful comments of the MGH team. We trust that giving clinicians the option of choosing whether to activate the SmarTone feature will provide them greater flexibility in making informed decisions regarding the clinical management of their patients.

* AAMI/IEC EC-13. Available at: <http://www.aami.org/publications/>. Accessed August 23, 2007.

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Michael T. Petterson, R.R.T., Masimo Corporation, Irvine, California. mpetterson@masimo.com

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(Accepted for publication July 3, 2007.)