

Treatment of Bleeding in Severe Necrotizing Pancreatitis with Recombinant Factor VIIa

To the Editor:—Recombinant¹ factor VIIa has recently been developed to reduce bleeding complications in hemophilic patients.¹ After injury to the vessel wall, tissue factor (TF) is exposed to the circulating blood and forms complexes with already activated FVII (FVIIa). The complex TF-FVIIa initiates hemostasis at the site of injury by activating FX into FXa, thereby providing the first amount of thrombin.²

It has been argued that FVIIa, in combination with extensive tissue damage, sepsis, and disseminated intravascular coagulation would be contraindicated.³ So far, there are few reports concerning rFVIIa treatment in situations of life-threatening bleeding caused by trauma,⁴ bowel surgery in patients with Crohn disease, and bowel lymphoma and profuse gastrointestinal bleeding.⁵ This report describes a patient successfully treated with rFVIIa in an attempt to control bleeding from necrotizing pancreatitis.

A 50-yr-old woman was admitted to our hospital with severe gallstone-induced pancreatitis. After initial treatment at the intensive care unit she developed necrosis and a pseudocyst of the pancreas. An attempt to drain this endoscopically resulted in massive hemorrhaging from the splenic artery. Immediate laparotomy was performed with repeated packing. She continued, however, to bleed. During reoperation the aorta was clamped and the diseased part of the pancreas resected. Furthermore, a subtotal gastrectomy, splenectomy, and ligation of the gastric and splenic arteries close to the aortic wall was performed. After declamping there was residual oozing from the region of the resected pancreatic necrotic tissue that was covered with an omental patch. At the end of this second laparotomy she had received 19 l of packed red cells; 4.5 l fresh frozen plasma; 300 g of platelets; prothrombin complex concentrate 1,200, *i.e.*, desmopressin 30 µg (Octostim®, Ferring Läkemedel AB, Limhamn, Sweden); anti-thrombin III 2,000; fibrinogen 1,000 mg; antifibrinolytic therapy with

tranexamic acid 1 g; aprotinin 500,000/24 h infusion. Coagulation-fibrinolytic determination showed antithrombin III 47% (normal range > 50%), prothrombintime 32 s (normal range 10–12 s), APTT 35 s (normal range 26–36 s), fibrinogen 1.4 g/L (normal range 2–4 g/L), D-dimer 0.3 mg/ml (normal range < 0.3 mg/ml). She continued, however, to bleed and received another 8 l packed red cells during the next 11 h. She was in a state of circulatory shock.

In response to this uncontrollable hemorrhage a 120 µg/kg dose of recombinant factor VIIa (rFVIIa, NovoSeven®, Novo Nordisk, Bagsvaerd, Denmark) was given and repeated 5 h later. Bleeding decreased after the first dose and ceased after the second, without thromboembolic adverse effects. Coagulation and fibrinolytic parameters normalized. The patient and her hemoglobin stabilized with no recurrence of bleeding. She was reoperated 3 days later with evacuation of residual necrosis and hematoma and the Roux-en-Y gastric jejunostomy was reconstructed without any problems of coagulopathy.

The successful use of rFVIIa in this patient suggests that its use should be considered and studied in patients with massive bleeding from septicemia when other standard therapy has failed.

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This article is accompanied by an Editorial View. Please see: Weiskopf RB: Intraoperative use of recombinant activated coagulation factor VII. *ANESTHESIOLOGY* 2002; 96:1285–6.

Another Possible Mechanism for Bronchospasm after Rapacuronium

To the Editor:—We read with interest the recent case series and editorial on bronchospasm induced by rapacuronium and noted the sudden withdrawal of the drug from the market. Histamine release and muscarinic receptor (M2) antagonism have been suggested as possible mechanisms of action,¹ though adverse reactions occurred without increase in blood histamine level, and histamine release caused by rapacuronium did not necessarily evoke adverse reactions.² More likely, rapacuronium causes M2 blockade as do the structurally similar rocuronium and pancuronium,³ and possibly to a higher degree at clinical doses.

We would like to draw attention to a third possible mechanism that takes account of the fact that bronchospasm after rapacuronium seems to be related to airway manipulation. We have treated severe bronchospasm in three children without any risk factors, who required epinephrine (two times) or repeated doses of albuterol (one time). Two cases occurred after uneventful, atraumatic tracheal intubation. In the third case, the child was intubated after 1.5 mg/kg rapacuronium and

easily ventilated for 15 min, but a second dose, applied during rigid bronchoscopy after the child had coughed slightly, evoked bronchospasm. Hahn pointed out that airway stimulation could elicit a vagotonic response that did not require a reflex arc but that directly fed into the vagal efferents (“cholinergic facilitation”).⁴ Since this mechanism is not centrally mediated it might be less affected by general anesthetics, so that it is still functional when hemodynamic or motor responses, *e.g.*, to intubation, are blunted. We speculate that this mechanism could profoundly enhance a possible M2 receptor blocking effect of the drug.

We would like to encourage further research in this field because we hypothesize that the combination of these mechanisms, *i.e.*, direct vagal stimulation through airway manipulation and drug-induced M2 block, is an important cause of bronchospasm. Since a short duration of action of muscle relaxants can be achieved by low affinity of the drug to the acetylcholine receptor that consequently requires a high

initial blood concentration, this side effect may play an important role for the future development of ultrashort-acting muscle relaxants.

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To the Editor:—Drs. Kron,¹ Naguib,² and Meakin³ discuss cases of bronchospasm associated with rapacuronium. Interestingly, all patients also received propofol. Dr. Kron also states in his discussion that propofol is “usually not associated with bronchospasm.”¹ In the same issue of *ANESTHESIOLOGY*, Brown *et al.*⁴ describe that the metabisulfite preservative used in the newer formulation of propofol does not provide the attenuation in neurally mediated and direct airway smooth muscle-induced bronchoconstriction that is seen with propofol without metabisulfite. None of the reports of bronchospasm specified which formulation of propofol was used. While I believe that there is an association of bronchospasm with rapacuronium, the timing of the administration of propofol and rapacuronium warrants an examination of the propofol used. The release of propofol with metabisulfite in the Spring of 1999 may contribute to the observation of more cases of rapacuronium-associated bronchospasm than was seen during the period of the clinical trials.

In addition, Lewis *et al.*⁵ suggest that the propofol formulation with metabisulfite is less potent than the propofol formulation without metabisulfite. The metabisulfite containing propofol required 10% higher induction boluses and up to 25% higher infusion rates. This suggests that some patients may have been less deeply anesthetized before instrumentation of the airway. Also, while not evident in reading the case reports, there could have been a tendency to intubate prematurely in these patients, possibly contributing to bronchospasm.

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In Reply:—The letter from Dr. Stuth *et al.* raises the ultimate question regarding the mechanism of rapacuronium-induced bronchospasm. They are, however, mistakenly implying that histamine release was suggested in my report as a possible mechanism.¹ I have clearly stated that “...the bronchospasm noted with rapacuronium is mediated *via* mechanisms that do not seem to be related to histamine release.”¹ The description of the three children who developed severe bronchospasm after rapacuronium by Dr. Stuth *et al.* is incomplete. It is, therefore, difficult to make any conclusions regarding rapacuronium-induced bronchospasm in these patients. It seems, however, that their third patient, who coughed during rigid bronchoscopy, might have had inadequate anesthesia. Therefore, it may be difficult to attribute the bronchospasm that occurred in this patient to the second dose of rapacuronium.

Dr. Stuth *et al.* suggested that “cholinergic facilitation”² could explain the rapacuronium-induced bronchospasm. However, they did not specify which receptors needed to be invoked to activate this reflex mechanism. Further, if this reflex mechanism remains active during anesthesia, as suggested by Dr. Stuth *et al.*, one would expect to see a greater incidence of bronchospasm (regardless of the general anesthetic technique used) in the patient population. It is possible that

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Which Formulation of Propofol was Used?

In summary, I believe rapacuronium is associated with bronchospasm, although the contribution of propofol formulation and anesthetic depth at intubation is unknown. I suggest these factors need further investigation.

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there is more than one mechanism involved in triggering severe bronchospasm seen after rapacuronium but the evidence currently available is insufficient to make any definite statement on how rapacuronium induces bronchospasm.

The patient described in my report¹ received 1 mg midazolam, 150 µg fentanyl, and 200 mg propofol for induction of anesthesia. The propofol used was supplied by AstraZeneca Pharmaceuticals (Wilmington, DE). This formulation is known to produce bronchodilation.³⁻⁵ Further, there is no evidence to support Dr. Matsumura's suggestion that the release of propofol with metabisulfite contributed to the higher incidence of bronchospasm seen with rapacuronium. In fact, this higher incidence was noted before the introduction of propofol with metabisulfite into clinical practice.⁶ Similarly, the statement of Dr. Matsumura that “...some patients may have been less deeply anesthetized before instrumentation of the airway” is not substantiated. In such practice, one would see a higher incidence of bronchospasm in the patient population.

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In Reply:—I appreciate Dr. Matsumura's interest. The propofol formulation in my case did not contain metabisulfite.

Regarding the patient's depth of anesthesia, I would like to point out that despite thorough preoxygenation, she desaturated before any airway instrumentation, suggesting that bronchospasm was evolving before intubation. She had received 1 mg midazolam and 50 μ g fentanyl and approximately 2.5 mg/kg propofol for induction. I doubt that light anesthesia caused her event.

The philosopher David Hume believed that an effect could never be absolutely attributed to a given cause despite their temporal associa-

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Epidural Blood Patch in Obstetric Anesthetic Practice

To the Editor:—We read with interest the article by Safa-Tisseront *et al.*¹ reporting their experience on epidural blood patch (EBP), and would like to comment with regard to obstetric anesthetic practice. These authors reported that large diameter needles (less than 20 gauge) and a short time interval between dural puncture and blood patching (fewer than 4 days) independently predicted failure of EBP. Therefore, considering needle diameter, one must note that only 68 patients (13%) were really at risk of failure, as they underwent dural punctures with Tuohy needles (17-18 gauge). This fact likely contributed to the high success rate of a single EBP reported in the study. It can also explain why only 3.7% of the patients required a second EBP. As spontaneous relief is the natural outcome of postdural puncture headache (PDPH) when small diameter needles are involved,² we can consider that the CSF leak may decrease over time, therefore supporting a lapse of time before performing EBP. This is also probably a strong factor of efficacy if EBP is performed at least 4 days after the dural puncture, assuming that 87% of patients in the study had undergone diagnostic or therapeutic lumbar punctures with small gauge needles.

However, despite the threshold lapse of 4 days, as shown in the study, Safa-Tisseront *et al.*¹ did not recommend to delay EBP. This message is very important for obstetric anesthesiologists, although a higher failure rate must be expected after a single EBP. In our experience of 21 consecutive cases of PDPH complicating epidural procedures with 17 gauge needles, all but one patient had an EBP within 4 days, including 15 women who had their EBP within the first 2 days. Ten patients required a second EBP for headache relief on the day after the first EBP, because although they had experienced complete relief,

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In Reply:—We appreciate the interest and the comments of Dr. Aya *et al.* regarding our article.¹ Our study included all patients treated with an epidural blood patch (EBP) for incapacitating postdural punc-

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tion. Although there were certainly other possible causes for the problems reported by myself and others, their association with rapacuronium, a newly released drug known to produce mild bronchospasm, made it necessary to report these events to the anesthesia community.

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the effect was transient. Several points are in favor of early EBP. Indeed, given that PDPH are usually severe and incapacitating in obstetric patients and prevent women from taking care of their newborns, given that the delay of an effective treatment makes the patient depressive and their family aggressive, and finally, given the low morbidity of EBP, confirmed by the authors, we believe that, at least in the obstetric setting, EBP should be performed as soon as possible and should not be delayed. We therefore strongly support Quaynor and Corbey's assertion:³ "Epidural blood patch: why delay?". Furthermore, obstetric patients undergoing EBP and their families should be informed of the fact that PDPH relief is sometimes transient, and that a second EBP might be required.

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ture headache (PDPH) in our hospital, from all medical and surgical specialties, including obstetrical patients. The incidence of accidental dural puncture during epidural anesthesia (performed with Tuohy

needles) is low. Indeed, only 68 of our patients (13% of 504 treated patients) underwent dural puncture with Tuohy needles in our study. In obstetrical patients alone, the first EBP permitted complete relief of symptoms in 66% (45/68), incomplete relief of symptoms in 13% (9/68), and failure in 21% (14/68) of them, contrary to the global population (including obstetrical patients) where these results are respectively 75%, 18%, and 7%. As noted in the letter by Dr. Aya *et al.*, the low percentage of our patients who had dural puncture with large bore needles probably explains the high success rate of EBP in our study, and explains why only 7% of the patients required a second EBP. Grouping these patients permitted us to perform the multivariate analysis, and to observe that the large diameter of the needle performing the dural puncture was a predictive factor of failure of EBP. This important information would most likely have been missed if only obstetric patients with accidental dural puncture by Tuohy needles had been studied.

We agree with Dr. Aya *et al.*'s remarks concerning obstetrical patients who have severe PDPH complicating epidural anesthesia. No study (neither Loeser *et al.*'s study,² nor our's¹) can actually support the claim that delaying EBP from dural puncture could increase its effectiveness. However, it is important to inform patients in advance of the average success and failure rates of a first EBP, and of the possible

necessity of performing a second EBP. This information must be tailored according to the diameter of the needle that is used to perform the dura mater puncture. This information might allow for a better acceptance of EBP failure by patients, and therefore, a better acceptance of a second EBP. In our opinion, these elements and the safety of the EBP favor early EBP realization in patients with severe PDPH after dural puncture with a large bore needle, including obstetrical patients.

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Postoperative Visual Loss, Still No Answers Yet

To the Editor:—Drs. Lee and Lam recently reported a case of unilateral blindness in a patient after prone lumbar spine surgery.¹ This unfortunate complication of surgery in the prone position has been reported in the surgical literature,²⁻⁵ and less commonly in anesthesia journals.^{6,7}

Drs. Roth and Barach write in the accompanying editorial⁸ that there still are no answers as to the etiology of postoperative visual loss (PVL), although researchers are currently in the process of collecting much-needed prospective data to establish the cause(s) of PVL. In the meantime, we as anesthesiologists must be aware that PVL can occur despite our best efforts at prevention. Should we routinely inform patients of this risk? Our answer is yes, although there are some that would beg to differ. It is, however, an indisputable point that we should routinely ask patients questions about their vision in the recovery room and during the postoperative check. Although there is no definitive treatment for PVL, early detection with subsequent consultation with ophthalmologists, and rapid correction of possible causes of decreased oxygen delivery (e.g., anemia) may be the greatest hope for a patient who has suffered PVL.

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Multifactorial Etiology of Postoperative Vision Loss

To the Editor:—The thoughtful discussion of the case report "Unilateral Blindness after Prone Surgery" by Drs. Lee and Lam¹ reasonably excluded systemic hypotension and anemia as independent etiologic factors. However, the discussion was deficient in three respects. First, although they claim that the eyes within the soft foam cushion (manufacturer and model unreported) were intermittently "checked" every 30 min, conventional eye checks in the prone position can only be performed by peeling or pressing the foam cushion away from the eyes to visualize them. However, peeling or pressing the foam cushion away from the eyes necessarily changes the relationship of the foam cushion to the eyes; *i.e.*, the test itself changes the results. The eyes can also be checked by palpation, but this is a blind procedure, which also requires the fingers to peel or press away the foam cushion from the eyes. Furthermore, since the patient's face at

the end of procedure was "extremely edematous," the physical relationship of the eyes to the foam cushion must have been continuously changing in the direction of increasing intraocular pressure. The point in stating these limitations of the traditional eye check is that the new Prone-View foam cushion system (Dupaco) allows the eyes to be continuously and directly visually monitored (by mirror image) without the need to manipulate the foam cushion.²

Second, the authors failed to comment on the height of the nasal bridge of the patient. A low nasal bridge allows the medial aspect of the eyes to experience greater contact and pressure with the foam cushion, and as the periorbital area becomes relatively more edematous than the nasal bridge, the medial aspect of the eyes will press harder into the foam cushion.

Third, the 15° head down tilt and compression of the abdomen and thorax (not commented upon) of this 80 kg patient (height and body mass index were unreported) may have contributed to increased venous and intraocular pressures, facial edema, and decreased eye perfusion. As the authors point out, the selective effect of neosynephrine infusion on eye venous and arterial hemodynamics is unknown. It will be very important to elucidate the effect of neosynephrine infusion on the vascular supply of the eye since awareness of postoperative vision loss in the anesthesia community is rapidly increasing, and efforts to prevent hypotension by neosynephrine infusion will likely increase.

In summary, we agree that the cause of postoperative prone spine surgery vision loss is multifactorial. Drs. Lee and Lam have done a good job of ruling out systemic hypotension and anemia as independent causative factors in their particular case. We would be greatly interested in their thoughts about the multiple factors of technically inadequate eye checks, the relationship of their patient's eyes to the bridge

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In Reply:—Drs. Cheng and Tempelhoff are correct that postoperative visual loss (PVL) occurs in the absence of negligence by the anesthesiologist. The origin of PVL appears to be multifactorial, and further studies are needed to define the mechanisms. Hence, we opined in the editorial that more data are needed to design strategies for prevention of this devastating complication. Because of the poorly-defined nature of this injury, whether patients should be informed of the risk preoperatively remains a matter of judgment for the individual anesthesiologist and surgeon.

Drs. Cheng and Tempelhoff think it is “indisputable” that we should routinely ask patients about their vision postoperatively. While this seems an easy procedure to implement and a worthwhile recommendation, data are lacking to mandate such a practice. Patients waking up after anesthesia may not be fully responsive, pupil signs can be erroneous, and visual fields might not be assessable. Anesthesiologists do not routinely perform fundoscopy to examine the optic nerve. In the setting of posterior ischemic optic neuropathy, the fundoscopic exam will, early on at least, likely be inconclusive. Also complicating any postoperative visual examination is the fact that symptom onset may occur anywhere from immediately postoperatively to several days later. Serial examinations would be needed. In our opinion, the inavailability of qualified personnel, the questionable cost-benefit ratio, and the low incidence of PVL render this approach, currently, generally not feasible.

The main thrust of Drs. Benumof and Mazzei's comments relate to the device used to position the head during spine surgery. Anesthesiologists are well aware of the necessity to avoid external pressure on the patients' eyes. Benumof and Mazzei argue that the device they

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In Reply:—We appreciate the interest of Drs. Benumof and Mazzei in our case report of visual loss after an uneventful spine surgery in the prone position in an otherwise healthy patient.¹ They expressed concerns about several issues. In particular, their emphasis on “adequacy” of eye-check and padding provides us with the opportunity to address an important issue that we perhaps did not adequately explain in our report, leading to their misunderstanding. Adequate padding of the head in the prone position to prevent pressure on the eyes is important, and in the case we reported, we used a standard foam support (Gentletouch Headrest Pillow, Orthopedic Systems Inc., Union City, CA), with a cut-out for the eyes. Eye-checks can be performed accurately by pressing down on the side of the foam cushion without altering the correct positioning. The patient is of Caucasian extraction with a high nasal bridge. Moreover, the nasal bridge is of no conse-

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of the nose, and the effect of 15° head down tilt and increased venous pressures on the etiology of vision loss in their patient.

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developed allows continuous observation of the eyes without the need to manipulate the foam cushion. Having used this headrest, we agree that it has this advantage. However, we feel that the anesthesiologist should consider also periodically palpating the eyes directly, every 5-30 min, and documenting this on the patient's operative record. With respect to the conventional square foam headrest, this device is, in fact, suitable for the majority of patients. Placing a hand under the foam to intermittently feel the eyes is easily done. That the foam has to be “peeled” or “pressed away” as Benumof and Mazzei point out, is not a major problem because the eyes are in the open portion of the headrest. Moreover, one could argue that the process of moving the foam and perhaps even lifting the head from time to time adds further assurance that the eyes are not subjected to external pressure. In any event, we think these comments regarding the headrest distract the focus from the primary issues raised in the case report of Drs. Lee and Lam, because direct pressure is rarely the cause of postoperative visual disturbances.

We agree with Drs. Benumof and Mazzei that data on the effects of head down tilt and neosynephrine on the ocular circulation are not available. Whether it is advisable to use vasoconstrictors to maintain higher perfusion pressures to the eye in the prone position cannot currently be stated.

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quence provided that the eyes are positioned correctly in the cut-out portion of the foam cushion.

Of utmost concern is their perseveration regarding the type of foam cushion, the height of the nasal bridge, and the method of eye-checks, which underscores a fundamental lack of understanding, and a common misconception about the pathophysiology of visual loss after spine surgery.² The ophthalmologic diagnosis in our case report, similar to most cases of visual loss after prone spine surgery, was posterior ischemic optic neuropathy. The lesion lies posterior to the lamina cribosa, sparing the retina, and has never been shown to be related to pressure on the globe.³ Visual loss from pressure on the globe, on the other hand, is secondary to central retinal artery occlusion with or without anterior ischemic optic neuropathy. These patients will frequently show signs of external periorbital bruising or proptosis, with

evidence of retinal ischemia, which our patient did not. Moreover, in some patients who develop visual loss after prone spine surgery, Mayfield head pins were used instead of foam cushions, removing all doubts about pressure on the eyes. (ASA Postoperative Visual Loss Registry, unpublished data, 2001). The emphasis on pressure on the eyeballs in the context of postoperative visual loss is akin to the man looking for his keys under the lamppost after dropping them on the lawn; he sees a bright spot but he won't find the keys. While we can all applaud efforts to improve patient safety with foam cushions of better designs (the Dupaco Prone-View foam cushion is certainly a good one), overemphasis on this aspect will divert our attention and focus away from the real pathophysiology and prevention of postoperative visual loss from ischemic optic neuropathy.

As for their final point, we would advise Drs. Benumof and Mazzei to read the original discussion in our case report again¹, where we had raised similar questions, the answers to which are currently unavailable, and should form the focus of concerted research efforts.

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With Technology Comes Responsibility: Intraoperative Failure of an Anesthetic Vaporizer

To the Editor:—As new technology enters the operating room, integrated computers “automatically” manage functions and parameters normally controlled by the anesthesiologist. While our background and training in pharmacology allows us to fully understand the implications of using a new drug, our relative lack of training in mechanical and electrical engineering forces us to “assume” new technology brought to the operating room is safe. In this letter I would like to report an episode of incompatibility between the automated systems of a Datex-Ohmeda D-Tec “Plus” Desflurane vaporizer (Datex-Ohmeda, Inc., Madison, WI) and a Draeger Medical Julian anesthesia machine (Draeger Medical, Inc., Telford, PA) that resulted in the intraoperative failure of the vaporizer.

The episode occurred after the uneventful induction and intubation of an otherwise healthy 36-yr-old woman presenting for a laparoscopic tubal ligation. The Julian ventilator was programmed for volume control ventilation with a tidal volume of 600 ml, a respiratory rate of 8 breaths/min and an I:E ratio of 1:2.5 with a 50% oxygen in air mixture at 2 l total flow. The Desflurane vaporizer was initially set at 8% and then reduced on reaching an expired concentration of 6%. Approximately 20 min into the case (10 min of mechanical ventilation), the Desflurane vaporizer alarm labeled “no output” went off, and the Desflurane concentration in the anesthesia circuit began to fall. The Desflurane vaporizer was immediately turned off and isoflurane was added to the inspired gas flow to ensure adequate anesthetic depth. After several seconds the Desflurane vaporizer automatically reset, and a second attempt was made to use the vaporizer. Within several minutes the same no output error occurred. To rule out the possibility that the failure was caused by obstruction at the vaporizer attachment site, the vaporizer was removed and repositioned in another site. Once again the vaporizer would work for approximately 5 min before alarming no output. To determine if this was a faulty vaporizer, the vaporizer was exchanged for a new one, however, the problem persisted. It was hypothesized that the no output condition might be secondary to the current ventilator settings. Despite changes to the tidal volume, flow rate, % inspiratory pause, and I:E ratio the Desflurane vaporizer continued to fail within minutes of being started. By this time the surgery was coming to a close, so the use of Desflurane was abandoned and the surgery was completed using isoflurane. The patient had an uneventful wake-up with no recollection of intraoperative events.

In an effort to determine if the vaporizer failure was caused by the mode of ventilation, the next patient in the room was ventilated using

pressure control ventilation with settings that would result in ventilatory parameters similar to those used in the previous case. The patient was once again started on Desflurane, however, this time the vaporizer worked without any problem. To validate our finding an attempt was made mid case to switch to volume control ventilation. The no output alarm was triggered within 30 s of switching ventilation modes. After reinitiating pressure control ventilation and allowing the Desflurane vaporizer to reset, the remainder of the surgery proceeded without incident.

To better understand what had occurred, technical representatives from both Draeger and Datex-Ohmeda were contacted. The companies supplied the following information that helps to clarify the cause of the failure of the vaporizer in volume control ventilation mode. The new Draeger Julian ventilator is capable of operating in three different modes, spontaneous ventilation (or hand-bagging with pop-off valve), volume control ventilation, and pressure control ventilation. The ventilator controls are entered *via* a digital interface. When in volume control mode, the interface allows the anesthesiologist to set the tidal volume he wishes the patient to receive and automatically compensates for changes in fresh gas flow. The ventilator compensates for fresh gas flow by decoupling the fresh gas flow from the ventilator circuit during the inspiratory phase of ventilation. As a result, during the inspiratory phase of the ventilatory cycle, there is no fresh gas flow into the patient circuit. A Draeger representative explained that the way decoupling works is that all the fresh gas is delivered during expiration. The fresh gas flow set by the anesthesiologist is actually the average flow over the course of the respiratory cycle. The actual gas flow is zero during inspiration, and higher than the set flow during expiration, so the average is what that user has set. For example, at an I:E ratio of 1:2 and a set gas flow of 4 l/min, actual gas flow is 6 l/min during expiration and zero during inspiration, thus averaging 4 l/min. This results in a period of time where there is no flow past the vaporizer.

The Desflurane vaporizer made by Datex-Ohmeda has an imbedded circuit board that senses output from the vaporizer and turns off the vaporizer if no output is detected. According to the technical personnel at Datex-Ohmeda, the vaporizer monitors the power source, temperature, tilt, fluid level, and pressure differentials between the input and output ports. If any of these parameters deviate from “normal” the vaporizer shuts down. The engineers I spoke with reported that the changes made between the original Tec 6 Desflurane vaporizer, used during the certification of the Julian ventilator, and the Tec-D Desflur-

rane Plus vaporizer were made to help increase the functional life expectancy of the vaporizer and minimize maintenance requirements. However, the changes were felt to have minimal impact on the overall function of the vaporizer and therefore did not require FDA recertification. There was agreement among representatives from both companies that the circumstances pointed to the vaporizer sensing the decoupling of flow in volume control mode. When pressure control or spontaneous ventilation modes are used on the Julien ventilator, fresh gas flow is continuous, as there is no need to compensate for fresh gas flow during inspiration. In these modes, the vaporizer senses continuous flow and no error is triggered. This would explain the uneventful nature of the second case where pressure control ventilation was used.

This case serves to emphasize the importance of fully understanding the equipment we use every day. Even seemingly minor upgrades in a previously approved piece of equipment can result in unforeseen problems. If a drug company were to present a new drug, we would

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In Reply:—In his letter entitled “With Technology comes Responsibility: Intraoperative Failure of an Anesthetic Vaporizer,”¹ Dr. Kimatian describes a situation where a Datex-Ohmeda D-Tec “Plus” (Datex-Ohmeda Inc., Madison, WI) vaporizer mounted to a Julian anesthesia workstation (Draeger Medical Inc., Telford, PA) ceased to deliver Desflurane because of the fresh gas decoupling feature of the Julian, which stops fresh gas flow during inspiration. This problem occurred despite certification of an earlier model Desflurane vaporizer (Datex-Ohmeda D-Tec Desflurane vaporizer) with the Julian anesthesia system.

After retesting both of the vaporizers, it was determined that under certain ventilation settings, the D-Tec Plus would indeed cease output because of the fresh gas decoupling feature of the Julian anesthesia system. As a result, Datex-Ohmeda modified the D-Tec Plus software to make it equivalent to the original D-Tec software. Subsequent testing revealed that at higher respiratory rates, it is still possible for fresh gas decoupling to cause the vaporizer to cease output. Draeger has revised the operator’s manual to caution against the use of respiratory rates greater than 30 breaths/min when using a D-Tec vaporizer with the Julian in volume ventilation mode. Pressure ventilation and manual ventilation will not affect the Desflurane vaporizer.

Dr. Skimatian is absolutely correct that clinicians should understand the features of anesthesia delivery equipment that might lead to patient injury. Indeed, new anesthesia workstations offer new features and capabilities that require training to be used effectively. It is not clear, however, that in this case such knowledge would have avoided the incident described, because the Julian with fresh gas decoupling had

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In Reply:—Thank you for allowing us to respond to the Letter to the Editor, “With Technology Comes Responsibility: Intraoperative Failure of an Anesthetic Vaporizer.”¹

Datex-Ohmeda would like to compliment the author for correctly identifying the underlying cause of this particular failure. Indeed, the original design of the D-Tec Plus, with its improved alarm handling and logging software, does sense differences between the inlet and outlet ports of the vaporizer. This feature was included to prevent any backward flow through the vaporizer, which can significantly alter the function of the vaporizer.

The D-Tec Plus is a variant of the Datex-Ohmeda Tec 6 Plus desflu-

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want to know its mechanism of action, route of delivery, metabolism, excretion, side effects, indications, and contraindications before administering it to a patient. However, we routinely accept new pieces of technology into the operating room that we do not fully understand, and as this case points out, Food and Drug Administration approval does not guarantee compatibility with other pieces of equipment. Since identifying this problem to Datex-Ohmeda, they have recalled, modified, and replaced our Desflurane vaporizers so that they are now compatible. As technology in the operating room continues to become “smarter,” understanding the mechanisms behind the interfaces is imperative.

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been shown to work with an earlier model Desflurane vaporizer. Although the incident is described as a failure, the vaporizer was actually working as it was designed, that is, to detect when output from the vaporizer ceases. The new model of the vaporizer was more sensitive to cessation of vaporizer output than the earlier model, hence the incompatibility.

It is notable, however, that despite a failure of the vaporizer, there was no adverse effect on the patient. This positive outcome underscores the importance of design features that minimize the potential for patient injury. In this case, the “no output” alarm on the Desflurane vaporizer alerted the anesthetist to the malfunction, which initiated corrective action. Had the problem continued for any length of time, low anesthetic agent concentration alarms would have also alerted the anesthetist to a problem.

Anesthesia delivery system manufacturers pursue rigorous testing and thorough risk analysis in the process of designing equipment. Despite these high standards, equipment malfunctions will occur in the process of delivering anesthesia. Patient injury can be avoided, as it was in this case, by using equipment with appropriate alarm technology, recognizing a problem when it occurs, having a backup plan, and taking corrective action in timely fashion.

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rane vaporizer, designed specifically for use with Dräger anesthesia machines. Before the release of the new design, Datex-Ohmeda provided the D-Tec Plus to Dräger Medical so that the vaporizer could be validated on Dräger designed and manufactured anesthesia machines. The effect of the pulsating flows that occur during volume mode ventilation in the Julian design was not recognized during this validation.

After notification of the events described in the Letter to the Editor, Datex-Ohmeda, along with Dräger, worked quickly to fully understand the implications of this pulsating fresh gas flow and its impact upon the alarm management of the D-Tec Plus. As a result of this investigation,

the D-Tec Plus software has been upgraded. This upgrade has been provided to all the D-Tec Plus vaporizers in use and is currently being used during the manufacture of all new D-Tec Plus vaporizers.

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An Unusual Presentation of an Airway Foreign Body Involving Dentures

To the Editor:— A 78-yr-old woman was admitted to the hospital for repair of a fractured wrist, sustained in a fall. A rapid sequence induction was performed and her dentures were left in place until laryngoscopy. An uneventful general anesthetic was administered, and the patient was successfully extubated and taken to the recovery room in satisfactory condition. Several minutes after admission to the recovery room, her SpO₂ decreased from 99% to approximately 78%. She subsequently had a severe episode of coughing and expectorated a foreign body later identified as a Seabond® (Combe Inc., White Plains, NY) denture adhesive sheet.

Dental appliances, discovered by patient interview or examination, are left in place before intubation to aid in the performance of mask ventilation, should this prove necessary. Local pharmacies carry 15 varieties of dental adhesives, three of which use paraffin impregnated flannel sheets (two brands are shown in fig. 1). The Seabond® adhesive system consists of two separate sheets impregnated with a water-based adhesive which, when moistened, is applied to the denture and forms an efficient suction bond with the roof of the mouth. The patient expectorated one of these sheets; it is not known where the other went.

It is theorized that the sheet of adhesive, affixed to the roof of the mouth and to the denture, stayed in the mouth. It then adhered to the endotracheal tube as it passed through the oral pharynx, and was deposited in the airway. The obstruction became manifest in

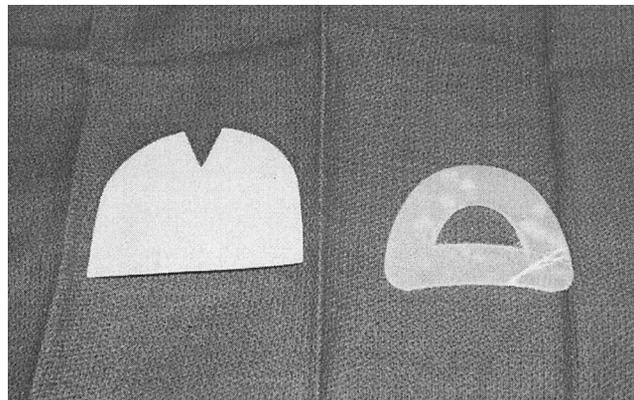


Fig. 1. Seabond® denture adhesive (*left*); EZO® paraffin impregnated flannel sheet (*right*).

recovery and was dislodged by the patient's coughing, without further sequelae.

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