Correspondence

Success of the Cobra™ after failure of the Fastrach™ in the difficult airway

To the Editor:

We describe successful intubation via the Cobra™ perilaryngeal airway (CPLA; Engineered Medical Systems, Inc., Indianapolis, IN, USA)[1] after unsuccessful intubation via the Fastrach™ intubating laryngeal mask airway (ILMA; Laryngeal Mask Company, San Diego, CA, USA)[2] following failed laryngoscope-guided tracheal intubation.

An obese 42-yr-old male with controlled hypertension was scheduled for an inguinal hernia repair. On examination he had a predicted difficult airway (limited head and neck movement, a thyromental distance < 6 cm, but good mouth opening) and refused both regional anesthesia and an awake tracheal intubation. He had no symptoms of reflux. He was premedicated with temazepam 20 mg *po* and metoclopramide 10 mg *po* and underwent an uneventful gaseous induction with sevoflurane 6% in O_2. Facemask ventilation proved easy and suxamethonium 1 mg·kg^{-1} *iv* was administered; however, laryngoscope-guided tracheal intubation failed after three attempts (Cormack and Lehane 3). A size 5 ILMA was inserted to facilitate lightwand-guided tracheal intubation, a technique which has been shown to be effective for airway rescue.[3] Ventilation with the ILMA was easy and the expired tidal volume was 8 mL·kg^{-1} without an oropharyngeal leak. A size 8 mm internal diameter straight silicone tracheal tube (TT) was primed with a flexible lightwand (FLW) so that the bulb was at the distal end. A supplementary dose of suxamethonium was administered. The TT-FLW was inserted throught the ILMA and advanced 1 cm beyond the epiglottic elevating bar. A bright glow of light was seen in the left lateral area of the neck and the ILMA handle was rotated to the right until the bright glow was in the midline at the level of the larynx. The TT-FLW was advanced 8 cm without tactile resistance, but esophageal intubation occurred, as indicated by an absence of lightglow at the suprasternal notch during advancement.[4] The TT-FLW was withdrawn and readvanced while the ILMA handle was elevated, but esophageal intubation occurred twice more. The size 5 ILMA was replaced with a size 4, but with the same result. The ILMA was then replaced with a size 5 CPLA, which was inserted easily and provided adequate ventilation. On this occasion the TT-FLW advanced into the trachea at the first attempt, as indicated by the lightglow disappearing at the level of the suprasternal notch. The FLW was removed, the TT cuff inflated and correct placement confirmed by movement of the bag and capnography. The minimal SpO_2 was 95% and anesthesia management was otherwise uneventful.

The probable explanation for the success of the CPLA is that it sits in a slightly different position in the pharynx, and the angle at which the TT emerges from the distal aperture is also slightly different. As such, we postulate that there will be scenarios where the CPLA fails and the ILMA succeeds as an airway intubator. If one extraglottic device fails, another may always succeed.

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Anesthetic management of bronchopleurocutaneous fistula – an alternate approach

To the Editor:
A 48-yr-old man with a persistent left bronchopleurocutaneous fistula (BPCF) and empyema thoracis following pneumonectomy presented for bronchial stump closure and left thoracoplasty. The patient refused any attempts at awake intubation.

He was placed in semi-recumbent position with the left side down after topicalization and mask induction. Right-sided double-lumen tube (DLT) placement was attempted while the patient was breathing spontaneously, but was unsuccessful in the absence of adequate muscle relaxation. Attempts to position a left bronchial blocker under fibreoptic guidance resulted in the balloon either slipping into the fistulous opening or backing up into the trachea. Advancing a single-lumen tube into the right main stem bronchus resulted in right upper lobe obstruction and attempted DLT insertion over a tube exchanger was also unsuccessful.

When rescheduled, he was positioned and induced in the same manner as before and intubated with an 8.5 size endotracheal tube. Anesthesia was maintained with air, oxygen and sevoflurane using a Bain circuit, the patient breathing spontaneously. A triple-lumen central line and a transesopheal echocardiograph (TEE) were added to standard monitoring. The patient was positioned in the sitting position and stabilized in the appropriate frame, with his hands abducted and flexed forwards. The surgeon approached the BPCF through a left thoracotomy by transaxillary approach.

Transtracheal jet-ventilation was delivered through the tip of a catheter placed above the level of fistula, to lessen the possibility of barotrauma. Anesthesia was maintained with an infusion of ketamine. During jet-ventilation PaO_2 values were 80 to 90 mmHg while $PaCO_2$ increased to 60 mmHg. The fistula was repaired and leak tested while purulent material from the left chest cavity was suctioned clean. Controlled ventilation restored blood gas levels towards normal. Left thoracoplasty was completed and the patient was extubated in the operating room. Postoperative pain control was achieved by a thoracic epidural catheter with continuous infusion of bupivacaine and fentanyl.

While anesthetizing the patient with BPCF in the sitting position, oxygenation can be assured with jet-ventilation, and gravity aids in draining away purulent secretions from the trachea.[1] CO_2 accumulation can be managed by limiting the surgical time and establishing controlled ventilation at the earliest opportunity. Hypotension can be corrected with crystalloid infusion. The hazard of air embolism can be monitored with TEE and treated with a triple lumen central venous catheter.[2]

When conventional methods of pulmonary isolation fail, anesthesia and surgery pose unusual challenges during the operative management of BPCF.[3,4] Careful planning and meticulous anesthetic management can transform a difficult case into a manageable one.

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"The critical airway"

To the Editor:
In their editorial of March 2005, Murphy *et al.* propose a new paradigm for the difficult airway (DA) approach in the operating room (OR) centred on ventilation and oxygenation rather than intubation.[1] We extend this concept to the emergent airway management outside the OR and describe the "critical airway" (CA). The CA is defined as the airway management outside the OR in a patient requiring emergent oxygenation and ventilation secondary to pathology. It is the antithesis of the OR airway management as the

situation is uncontrolled, tools are suboptimal and the patient, by definition, is critically ill.

Establishing an emergent airway in a remote location is "inherently difficult" as the routine airway assessment and management are changed producing failure rates much higher than would be acceptable in the "elective" setting. The OR paradigms of airway management need to be reassessed.

1) The anesthesiologist is not formally trained to manage the airway outside the OR. Predicting difficult laryngoscopic intubation in a critically ill unresponsive patient requiring emergency airway management is not practical as more than half of the clinical airway examination components suggested by the "Practice guidelines for management of the difficult airway" need a cooperative patient.[2]

2) The patient presents with acute comorbidities, a "full stomach", in immediate need for oxygenation and ventilation. A rapid sequence induction and intubation is not always indicated or applicable.[3]

3) The airway devices available are limited and the work environment suboptimal.

4) The time available to prepare and to successfully achieve ventilation and oxygenation is measured in seconds.

The goal of the CA management is efficient ventilation and oxygenation with no stomach inflation, regurgitation, and aspiration and without inducing comorbidities in an already critically ill patient (hypotension, hypertension, bradycardia, tachycardia, hypoxemia, hypercarbia, cardiac arrest, cervical injury).[4,5]

The inability to efficiently bag mask ventilate and the persistence to intubate a "full" stomach patient are at the core of most complications in the remote setting.[3] The CA is a dynamic concept that demands the anesthesiologist to be skilled in "all four dimensions" of the airway management techniques: bag mask ventilation (BMV), supraglottic airway (SGA; laryngeal mask airway, Combitube, laryngeal tube...), glottic airway (GA; laryngoscopy, endotracheal intubation, Eschmann), or infraglottic airway (IGA; cricothyroid membrane or surgical techniques); any of the techniques can be used as the first option (Figure).

The CA receives minimal attention in the anesthesiology literature, during residency training or workshops. We use the CA concept to train our residents by emphasizing the differences from the OR routine. The ASA DA algorithm is a tool primarily used to avoid crisis in a controlled environment (OR); the CA concept applies to crisis in progress in a remote location. The classic "cannot intubate-cannot ventilate" becomes "cannot ventilate with BVM and a supraglottic device and cannot intubate after an optimal attempt".

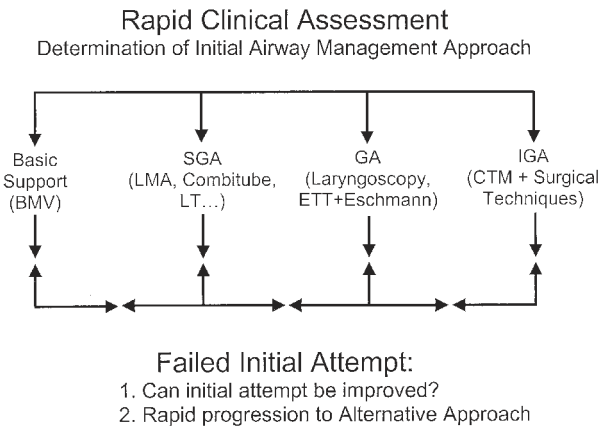

FIGURE The management of the critical airway. BMV = bag mask valve; SGA = supraglottic airway; GA = glottic airway; IGA = infraglottic airway

It is ironic that when we should function at our best as "airway consultants," we relay on suboptimal or no airway assessment. Can we define predictors for airway management "difficulty" in "all four dimensions" outside the OR? Can we define optimal use of specific devices: BVM, SGA, GA, and IGA? How does the clinical status of the patient impact on our choice of airway devices?

As airway specialists, anesthesiologists should address through research and training the specific issues of the CA thus improving the outcome of the critically ill. We agree with Murphy et al. that we have "to change how we think about airway management" and to shift from intubation to oxygenation and ventilation. This principle applies both inside and outside the OR.

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"Keep out of trouble" airway algorithm

To the Editor:

In his editorial, "Predicting the difficult laryngoscopic intubation: are we on the right track"[1] Dr. Murphy asks a relevant question and I agree with his answer. In truth, every intubation is potentially difficult, suggesting that the current mystique surrounding the difficult airway is both exaggerated and beside the point. Early in my career, I noticed that critical incident reports about difficult airways usually involve "losing the airway" (this is not surprising: a "lost" airway is rapidly lethal so those patients may not reach our care). Therefore, not losing the existing airway should be the first goal of management. All of the techniques of intubation are much easier with stable gas exchange and a bit of time.

With this in mind, many years ago I developed an algorithm for teaching medical students how to quickly assess the airway for potential complications in order to avoid making a bad situation worse (Figure). This includes the "dimensions" suggested by Dr. Murphy plus a few more. Its strengths are: relative simplicity; a preventative point of view; and quick identification of situations where speed matters. In the last circumstance, admittedly uncommon, everyone has to develop his or her own best intubation technique (I favour one involving the lighted sytlet), and usually the option of surgical airway exists. If a surgical airway is not possible, then early identification is even more crucial.

Although this approach has not been compared in a randomized double-blind study with consensus approaches (they also have not been validated) due to the low incidence of events as noted by Dr. Murphy, I can only say that it has been uniformly successful in my practice.

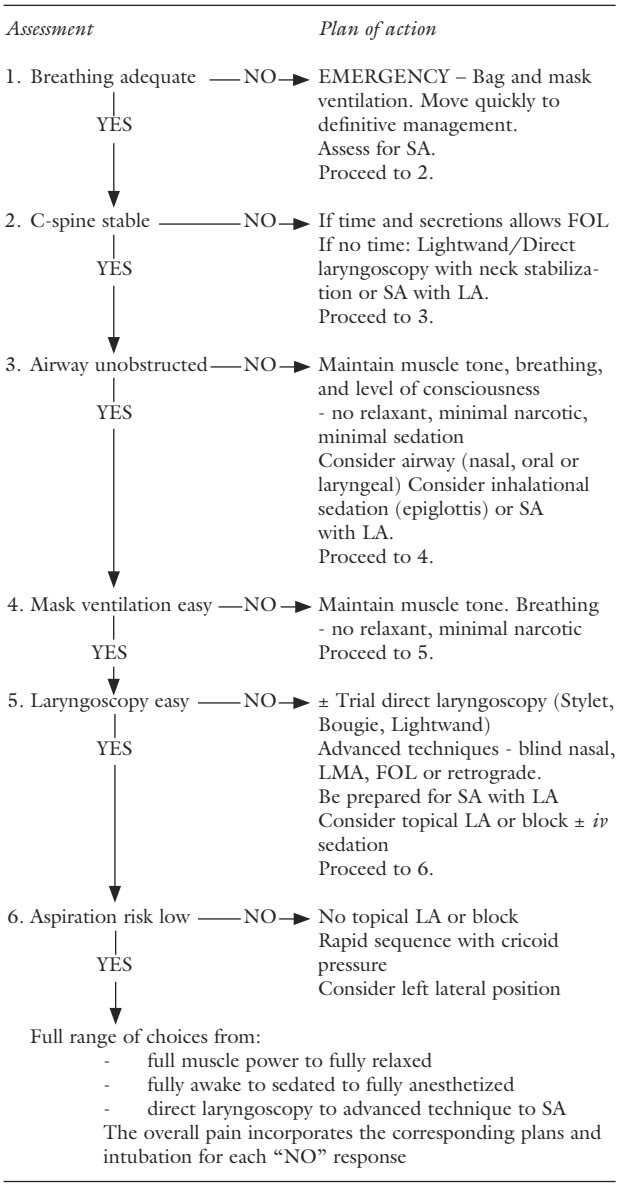

FIGURE K.O.O.T. (keep out of trouble) airway algorithm. An aide in choosing a safe technique of airway management. SA = surgical airway; FOL = fibreoptic laryngoscopy; LA = local anesthetic; LMA = laryngeal mask airway.

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Reply:

We must thank Drs. Matioc, Arndt, and Lee for their insightful comments. It is heartening to hear anesthesiologists echoing what we have been teaching residents and other practitioners in airway courses and workshops for more than a decade.

Additionally, these same sentiments and concepts form the foundation of our objectives in educating anesthesia practitioners to alternative ways of thinking and acting to address just the kind of "critical airways" that Matioc and Arndt described. The use of simple but practical strategies to rapidly predict and identify difficulty in all of its dimensions (bag mask ventilation, supraglottic devices, tracheal intubation and a surgical airway) in an emergency, is fundamental to our overall teaching goal.

With the on-going emphasis in airway management research and education, we are confident that patient outcome will improve.

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Bilateral foot drop: looking for the needle in the wrong haystack?

To the Editor:
We read with interest the description of a case of bilateral foot drop following spinal anesthesia.[1] We have described the etiology of such postoperative deficits (preoperative injury, co-existing neuropathy, surgical trauma, tourniquets, positioning and anesthetic techniques).[2] The case mentioned requires further clarification.

1. Documentation of neurovascular deficits in any limb with fractures is considered part of basic trauma (orthopedic) examination. Further, trauma to the lumbosacral spine may also result in bilateral foot drop.

2. Did the patient have other co-existing disease with subclinical neuropathy (e.g., diabetes)? Pre-existing neuropathies have been identified as risk factors in nerve palsies.[3]

3. Does the plural "tibial pins" suggest that these were inserted on both sides?

4. Was the femur alone operated? How were the leg fractures managed? If they were also operated upon, the foot drop could be related to the surgery.[4]

5. What was the position of the patient during surgery? Femoral traction on a hip table (supine) may stretch the sciatic nerve. In the lateral position the sciatic nerve of the dependent side may be compressed.[5]

The authors erroneously identify paralysis of foot flexors as the cause of foot drop. Neurological examination is more important to rule out neurological deficits following the trauma rather than preceding the anesthetic (as they recommend). Prior to searching for iatrogenic causes, if they had documented the presence or absence of bilateral foot drop (at admission or after the traction pin application), they may not be faulted for looking for the metaphoric needle in the wrong anesthetic haystack!

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Reply:

This is in response to the letter from Dr. Naveen Eipe and Dr. Nihar Ranjan, regarding the above mentioned article in the May 2005 issue of the Canadian Journal of Anesthesia. I thank Dr. Naveen for his comments, and would like to clarify the points raised by him.

There was no documentation of preoperative neurological deficit in the basic trauma examination. It is

quite true that trauma to the lumbosacral spine can result in bilateral foot drop, but this was ruled out in the preanesthetia visit and the magnetic resonance imaging done postoperatively. The patient did not have any other coexisting disease with subclinical neuropathy (diabetes), although pre-existing neurological disease should also be ruled out e.g., lumbar disc syndrome or spondylitis or recent herpes zoster in such cases. The tibial pins were inserted on both sides, and the femur alone was operated during the first stage. The legs were surgically reduced in the second stage, so the foot drop could easily be related to the surgery. The patient was operated in the lateral position, no femoral traction was applied (on the hip table). We agree that the sciatic nerve compression in the lateral position could again be a factor (but unilateral).

Since the patient had no neurological deficit preoperatively, a neurologist's opinion was not sought. Consultation was undertaken postoperatively, after the patient developed foot drop. There was no documentation of the presence of foot drop at admission or after the insertion of tibial pins (bilateral) so the etiology was most likely related to surgery or spinal anesthesia. Bilateral foot drop after spinal anesthesia is possible only if lumbar puncture is performed above the third lumbar segment, which is uncommon. Considering the angle of entry of the spinal needle, trauma to one side of the conus is possible.

We emphasize that this case is not meant to attribute blame, but to highlight that this complication, though rare, does occur, and has its own medicolegal implications.

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Does bilateral thoracic sympathectomy predispose to reflex bronchospasm following tracheal intubation?

To the Editor:

Thoracic endoscopic sympathectomy has become the technique of choice for treating intractable essential hyperhidrosis.[1] We report severe bronchospasm following tracheal intubation in a patient with a previous history of bilateral thoracic sympathectomy. Consent for publication was obtained from the patient.

A 31-yr-old non-smoking woman, 60 kg, without a history of allergy, or asthma, was scheduled for left knee arthroscopy. Two months previously, she had an uneventful general anesthetic for bilateral thoracoscopic sympathectomy to treat essential hyperhidrosis. Preoperative examination revealed clear lungs. Following preoxygenation, anesthesia was induced with propofol 2.5 $mg \cdot kg^{-1}$ *iv*, vecuronuim 0.15 mg kg^{-1} *iv*, and fentanyl 2 $\mu g \cdot kg^{-1}$ *iv*. Ventilation by facemask was easy. Following onset of neuromuscular block, tracheal intubation was easily performed by direct laryngoscopy. Immediately following intubation, ventilation became difficult. Chest auscultation revealed bilateral expiratory wheezing associated with decreased air entry and increased airway pressure up to 60 cm H_20. Oxygen saturation, as monitored by pulse oximetry, decreased from 100% to 80%. Anesthesia was deepened by inhalation of sevoflurane (4–8% inspired concentration) in 100% oxygen. Six doses of 20 µg ipratropium bromure and five doses of 100 µg salbutamol were administered via the endotracheal tube. During the following five minutes, ventilation became progressively easier, oxygen saturation increased to 100%, and breath sounds normalized. Anesthesia was continued with 2% to 4% sevoflurane. Throughout this event, no cutaneous flushing was observed, blood pressure ranged between 110/90 mmHg and 130/95 mmHg, with pulse rate between 65 and 115 $beats \cdot min^{-1}$. Surgery and recovery proceeded uneventfully. Twelve weeks after surgery, an atopy skin patch test was negative.

The severe bronchospasm experienced by the patient may have been secondary to an anaphylactic reaction to the induction agents. In a recent French survey of anaphylaxis,[2] the most common features were cardiovascular manifestations (71.5%), followed by cutaneous symptoms (69%). The least frequent feature was bronchospasm which only occurred in 44.2% of the cases, and was associated with a history of atopy or asthma. In our patient, there was no history of atopy or reactive airway disease and the bronchospasm was not associated with any cardiovascular or cutaneous manifestations. The severe bronchospasm occurred immediately following tracheal intubation, suggesting that it may have been a reflex response which was triggered by instrumentation of the airway under a light level of anesthesia. Other causes of bronchospasm such as unrecognized aspiration, carinal irritation, secretions, or chemical irritants that may have contaminated the tracheal tube were also considered and excluded.

Patients with essential hyperhidrosis have sympathetic overactivity, associated with compensatory high parasympathetic tone. Sympathectomy results in

a decrease of plasma norepinephrine,[3] and parasympathetic predominance[4] which may increase airway resistance.[5] Intraoperative bronchospasm is usually cholinergically-mediated. Thus, patients with essential hyperhidrosis who have undergone bilateral thoracic sympathectomy, may be more liable to develop reflex bronchospasm under light levels of anesthesia.

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Retained and cut stimulating infraclavicular catheter

To the Editor:
We report the case of a stimulating infraclavicular catheter that was retained and subsequently cut, requiring a surgical incision for removal. Consent for publication of personal information was obtained from the patient according to our local institutional guidelines.

A 52-yr-old American Society Anesthesiologists class 1 woman had an open reduction and internal fixation of her left radius under general anesthesia. Postoperatively she was given narcotics for analgesia but experienced severe nausea and vomiting. She agreed to the insertion of a continuous infraclavicular brachial plexus block for pain control. Using Wilson's landmarks,[1] an Arrow StimucathTM Continuous Nerve catheter (Arrow International, Reading, PA, USA) was placed uneventfully in the left infracoracoid area. The catheter was advanced easily under stimulation 4 cm beyond the tip of the needle. Its proximal end was tunnelled under the skin leaving a small skin bridge at the initial insertion site of the needle. The patient received a bolus of 30 mL bupivacaine 0.25% followed by a continuous infusion (10 $mL \cdot hr^{-1}$ ropivacaine 0.2%) with excellent pain relief.

The following day, the local anesthetic infusion was stopped to allow a neurological assessment of the operated limb by the surgeon. As her pain was well controlled with acetaminophen and codeine, the patient requested that her catheter be removed. After experiencing resistance upon removal of the catheter, the patient's nurse cut the catheter at the skin bridge. When she pulled on the distal end of the catheter, the sheath came off, leaving the stimulating wire in place. We were then notified. After assessment, the patient was taken to the operating room. With fluoroscopy, the wire was visualized and knotting was ruled out. Under local anesthesia, a small incision was made and the wire removed (Figure).

Inability to remove perineural catheters due to knotting or breakage has been described after femoral,[2] fascia iliaca,[3] sciatic,[4] and axillary[5] blockade. Most of these cases required surgical extraction for catheter removal. We are the first to describe a retained stimulating infraclavicular catheter. The latter became separated from its stimulating wire after being cut. In this institution nurses on the ward remove all nerve catheters. We feel that it is important to instruct the nursing staff and ambulatory patients never to cut stimulating perineu-

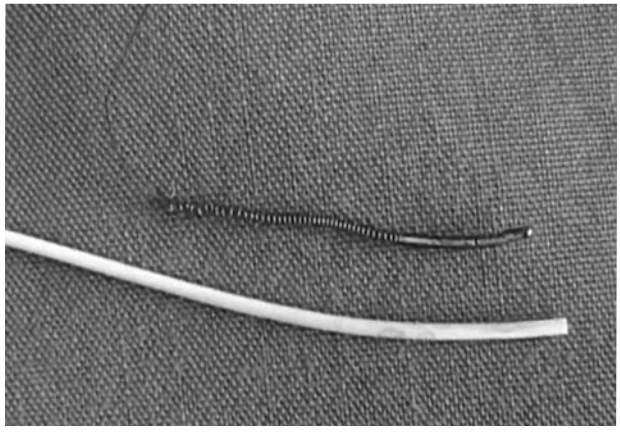

FIGURE Distal end of catheter sheath and wire.

ral catheters. Furthermore, patients should be told to contact the anesthesia department immediately if they encounter any difficulty during catheter removal.

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Effectiveness of bolus landiolol on paroxysmal atrial tachycardia

To the Editor:
Landiolol is a ß-adrenergic receptor antagonist with a short elimination half-life which has recently been developed.[1,2] We and others have documented its efficacy as a ß-adrenergic receptor antagonist, when administered as an iv bolus.[3,4] Here, we show that an elderly patient with paroxysmal atrial tachycardia was successfully treated with bolus landiolol *iv*.

An 84-yr-old male patient (161 cm, 68 kg) was scheduled for repair of a right femoral fracture under spinal anesthesia. His preoperative electrocardiogram showed a complete right bundle-branch block, and he had a history of palpitations. Holter monitoring demonstrated frequent supraventricular arrhythmias, and these were successfully treated with the class Ia antiarrhythmic agent cibenzoline 150 mg·day^{-1} before surgery. Spinal anesthesia was performed using isobaric bupivacaine 15 mg at L3/4. Thirty minutes later, he abruptly became tachycardic (190 beats·min^{-1}) and was

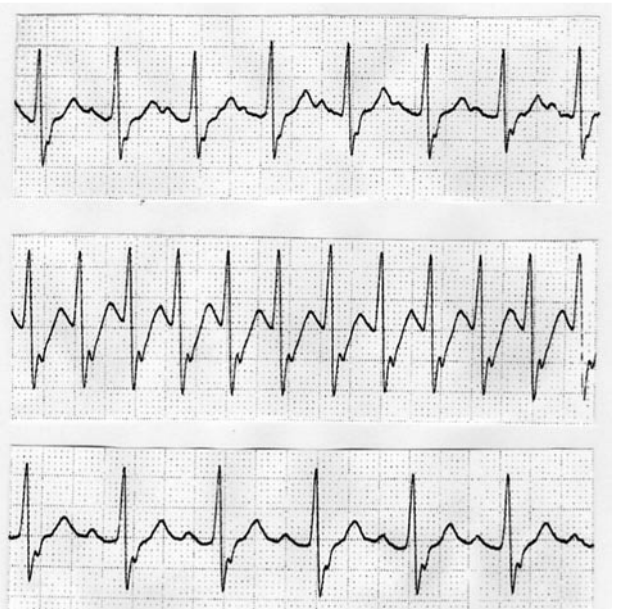

FIGURE Electrocardiogram: (a) before spinal anesthesia; (b) immediately before the administration of landiolol; (c) nine minutes after initial administration of landiolol 5 mg *iv* (total 15 mg *iv* for nine minutes).

treated with cibenzoline 70 mg *iv* over two minutes. Until that time, his blood pressure and heart rate had been stable (140–160/90–100 mmHg and 110–115 beats·min^{-1}, respectively). However, the tachycardia did not respond to this treatment. We subsequently administered three boluses of landiolol 5 mg *iv* (over ten seconds each). After a cumulated 15 mg *iv* dose of landiolol, his heart rate decreased to 95 beats·min^{-1}, normal sinus rhythm (Figure), without any hypotensive episode (blood pressure 135/90 mmHg). The remainder of the anesthetic was uneventful, whereas two hours after surgery, he again suffered a similar episode of tachycardia, for which cibenzoline 30 mg *iv* was ineffective. In this instance, iv volume replacement therapy including blood transfusion gradually restored normal heart rate.

Bolus landiolol (0.1–0.3 mg·kg^{-1} *iv*) is effective to prevent tachycardia in response to tracheal intubation without affecting blood pressure.[3] For our patient, we administered the higher dose of landiolol to treat the tachyarrhythmia. This drug was effective without inducing hypotension even in such an elderly patient. Importantly, the arrhythmia we managed was insensitive to a class Ia antiarrhythmic agent, indicating that landiolol can be used as an alternative when tachyarrhythmia is refractory to conventional therapy.

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Mean arterial blood pressure estimation and its limitation

To the Editor:

Mean arterial blood pressure (MAP) has clinical and physiologic significance in both the representation of perfusion pressure and its utilization in the calculation of hemodynamic variables. The accurate MAP is defined as the zero frequency (direct current) component following Fourier analysis of the arterial waveform,[1] or as the time-weighted integral of the instantaneous pressures derived from the area under the curve of the pressure-time.[2] However, clinicians have yet to find an easier way that may be applied in clinical practice. Some researchers continue to investigate more accurate MAP estimation methods.[2–4]

Even though we sometimes forget the limitations in using these equations,[5] we should keep in mind that they will not have the same MAP if they have different arterial pressure waveform morphologies, no matter what are the causes of the differences.[1] The following is an example.

We analyzed a patient's direct arterial blood pressure data, which was continuously checked by an anesthesia monitor, sampled at 250 Hz, and stored in a computer through analogue output. The data length was about one hour. We divided the data in two-second intervals, so the total data sections numbered around 1,800. In each section, we calculated the mean using the Fourier transformations by a computer software program (Matlab®, Natick, MA, USA). The maximum difference of MAP within the same systolic arterial pressure and diastolic arterial pressure pairs was sought. We then compared the two waveforms with the maximum MAP difference. The MAP differences were originated mainly from, in my case, an early reflected wave (Figure). However, there may be other sources of waveform differences, for example, the existence of a dicrotic notch, high inotropic pressure pulse,[6] and a double dicrotic notch.

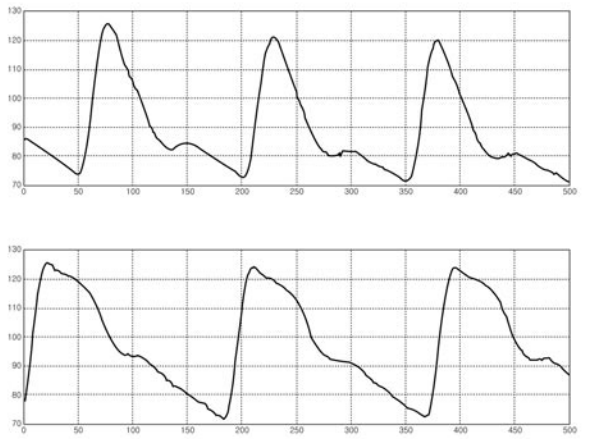

FIGURE Different mean arterial pressures (MAP) of one patient. The X-axis is arbitrary time; the Y-axis is blood pressure in mmHg. Both readings have the same systolic and diastolic pressures, but have a 10 mmHg difference in MAP.

As clinicians, we need an easily calculable determinant of MAP, but we should understand its limitations as applied to clinical practice, especially where the arterial waveform could be different from the one used to derive the simplified equation. Factors which may alter arterial waveform, and hence MAP determination include increasing age, atherosclerosis, and changes in blood volume status.

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Continuous renal replacement therapy

To the Editor:

We read the article by Jacka *et al.*[1] with interest. This manuscript is provocative and novel but raises potentially significant methodological questions.

First, the issue of crossovers between treatment groups makes it difficult to conclude which modality was responsible for favourable or adverse outcomes. Was continuous renal replacement therapy (CRRT) still associated with better renal recovery if only patients treated with a single modality were included?

Second, comparing renal recovery in survivors is of questionable validity as the sole renal outcome. Specifically, the "advantage" of CRRT was observed only after excluding those who died – and as Jacka *et al.* note, death in the intensive care unit (ICU) was significantly more frequent in the CRRT group. Since death and dialysis-dependence are competing risks, the composite of both outcomes would seem more appropriate. This outcome does not appear to significantly differ between the two modalities, which is unsurprising. Although the largest randomized study to date actually showed significantly increased in-hospital mortality due to CRRT use,[2] this may not have resulted from the effect of treatment per se. As noted by Jacka *et al.* in their article, the underlying illness probably influences prognosis to a far greater extent than the dialytic modality.

Third, impaired kidney function at baseline is strongly associated with the need for chronic dialysis in people with acute renal failure.[3] In the study by Jacka *et al.*, patients who received intermittent hemodialysis (IHD) had significantly higher serum creatinine at ICU admission, suggesting that they were more likely to have pre-morbid chronic renal insufficiency. Unfortunately, analyses evaluating renal recovery by treatment modality did not control for this difference, which may have influenced the findings.

Finally, a pooled analysis of four randomized studies including more than 400 patients showed no renal benefit of CRRT (and a slight trend towards harm).[4] Although meta-analysis has its limitations, none of the four included studies showed a renal benefit of CRRT. Jacka et al. do not discuss why their retrospective study might differ from the available randomized trials. We speculate that the discrepancy is due to bias resulting from the non-randomized design.

Multiple non-randomized studies over the last 20 years have been used to support the theoretical benefits of CRRT, and to justify its higher costs. However, randomized studies have not demonstrated that CRRT is superior. Even for surrogate outcomes such as intradialytic hypotension, no good quality data support the use of this indisputably more expensive treatment.

We agree with Jacka *et al.* that larger randomized trials should be performed, but respectfully disagree that their article helps to inform debate in the interim. Since the best available data do not indicate that dialytic modality influences outcome in critically ill patients, we suggest that the least costly therapy should be used until new randomized trials demonstrate otherwise.

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Reply:

We thank Tonelli et al. for their interest in our study showing improved renal recovery among patients treated with continuous renal replacement therapy (CRRT).[1]

As we described, ours was a non-randomized observational study. The 'issue of crossover' should not affect interpretation, even adopting a 'worst case scenario'. Crossovers were very few and occurred only among patients who had stabilized on CRRT from the indications precluding intermittent hemodialysis (IHD). Crossovers would be a concern in a randomized trial, among patients assigned to IHD but who required CRRT due to hemodynamic instability, and who would probably die without renal replacement.

Second, the risks of dialysis-dependence and death might be an appropriate composite outcome measure in a prospective randomized analysis. In our study, as we pointed out in the methods and discussion, CRRT was applied to patients who had sufficient hemodynamic instability, intracranial hypertension, or liver failure that made use of IHD impossible. The finding of any survivors among these CRRT patients supports the use of CRRT, making randomized evaluation unethical.

Third, most studies comparing CRRT and IHD have used mortality and renal recovery as separate outcome measures, rather than composites. Tonelli et al. have suggested that studies comparing modes of RRT should concentrate on renal recovery.[2] *No study has found hospital mortality 'due' to CRRT as implied by Tonelli.*

Fourth, although serum creatinine was higher at intensive care unit admission among IHD patients, careful examination of our tables shows that at the time of institution of RRT, and as we pointed out in our discussion, serum creatinine was similar between groups.

Fifth, we agree that meta-analysis has limitations. Although Tonelli failed to find a benefit from CRRT, Kellum showed lower mortality with CRRT when patients were stratified according to severity of illness.[2,3]

Sixth, CRRT has been shown to have specific advantages over IHD. CRRT minimizes hemodynamic fluctuation in unstable patients and prevents further elevation of intracranial pressure in patients with fulminant liver failure.[4] *CRRT is superior in correcting azotemia and acidosis and is recommended for patients with severe sepsis.*[5,6]

While we support Tonelli et al. in their advocacy of the least costly alternative, insistence on minimizing cost in the face of evidence of benefit represents an inappropriately regimented approach. Finally, we thank Tonelli et al. for helping us to stimulate and inform debate on the issue of renal replacement among the critically ill, along with our descriptive study.

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Sulfadiazine-induced methemoglobinemia in a boy with thalassemia

To the Editor:

Drug-induced methemoglobinemia has been well documented,[1–3] but an acute episode of sulfadiazine-induced arterial desaturation during emergence from anesthesia has never been reported. A three-year-old boy (14 kg, 90 cm) suffered scalding injury (55% of body surface area) and was scheduled for debridement. Family history was positive for a dominant ß-thalassemia trait. Physical examination revealed no

cardiac murmurs, abnormal breath sounds or cyanosis. Preoperative hemoglobin and hematocrit were 79 $g \cdot L^{-1}$ and 25%, respectively. Acetaminophen had been prescribed for analgesia. Routine induction and maintenance of anesthesia were performed with atropine, thiamylal, atracurium, sevoflurane and oxygen. At the end of surgery, silver sulfadiazine ointment (1%) was applied for wound dressing. During emergence from anesthesia, an acute onset of arterial desaturation was documented by pulse oximetry (SpO_2: 92%–96%). After excluding sputum impaction, lung collapse and malposition of endotracheal tube as possible causes, the patient was extubated. Upon arrival in the postanesthesia care unit, SpO_2 decreased to 63% and severe cyanosis was observed. The patient was re-intubated and SpO_2 increased to 86% in the presence of pure oxygen. An emergency chest x-ray revealed no remarkable findings, although the patient remained cyanotic. The blood gas analysis revealed a PaO_2 of 428 mmHg with an estimated saturation of 99.6%. However, the blood was dark chocolate in colour, and desaturated (SpO_2: 85%) as measured by the pulse oximeter. Both hypothermia and acidosis were excluded. An emergency co-oximetry analysis revealed a high methemoglobin concentration of 27.7%. After being treated with methylene blue (1.0 $mg \cdot kg^{-1}$, *iv*), the patient became acyanotic with SpO_2 of 99%. The fraction of methemoglobin decreased to 1% within an hour, and the patient was extubated the next morning.

Both congenital (e.g., sickle cell trait, α-thalassemia) and acquired (e.g., acetaminophen, sulfamethoxazole, nitrate, benzocaine) methemoglobinemias have been well documented.[1–3] However, it has not previously been reported that silver sulfadiazine ointment may cause methemoglobinemia. In this pediatric patient with an extensive burn injury, a large area of skin debridement might have provided a route for sulfadiazine into systemic circulation. In addition, ß-thalassemia trait or disease might have predisposed to methemoglobinemia due to less resistance to oxidative stress. The dramatic therapeutic effect of methylene blue excluded sulfhemoglobenemia in this patient.[4] In conclusion, anesthesiologists should be aware that silver sulfadiazine ointment may cause methemoglobinemia in association with coexisting burn injury and thalassemia.

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