To the Editor—Naguib et al. have presented a clinically relevant study of arterial saturation values after preoxygenation and different doses of suxamethonium, and discuss their results using the extensive reports relevant to this topic. Unfortunately, they perhaps did not search the published work back to times before the pulse oximeter was introduced. Before the pulse oximeter, the Hewlett Packard ear oximeter (Waltham, MA) was used to measure arterial oxygen saturation continuously. Although clumsy, it was accurate and gave reliable results. We used this device to be the first to report changes in oxygen saturation, measured continuously, at induction of anesthesia. We recorded arterial oxygen saturation in patients who were given thiopental and suxamethonium (although in greater doses than those used by Naguib et al) and described the effects of preoxygenation. We showed convincingly that desaturation was more likely in obese patients than that even a small mask leak markedly impaired the efficacy of oxygenation.

Naguib et al. suggested “shunting” as a reason for the hypoxemia they found, particularly in obese patients. This is not the most probable explanation. As they acknowledge, in anesthetized obese subjects, functional residual capacity is small, often close to residual volume, as shown in the study by Damia et al. In these circumstances, oxygen stores are small, and desaturation occurs more quickly as the small alveolar oxygen content decreases. This effect is shown clearly in the model predictions of Hardman et al. This model, which was more complete than the model studies considered by Naguib et al., showed much more rapid desaturation with a small functional residual capacity than with a greater pulmonary shunt.

Gordon B. Drummond, F.R.C.A., Royal Infirmary, Edinburgh, United Kingdom. g.b.drummond@ed.ac.uk

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To the Editor—Schaumann et al. are to be congratulated for a nice study regarding cricothyroidotomy. The duration and complications of cricothyroidotomy performed by emergency physicians in cadavers was evaluated. They specifically compared a surgical technique and the Arndt cricothyroidotomy set. As in a previous study from the same institution, the assessment of the cutaneous-tracheal tract by a pathologist is a valuable methodologic aspect. However, we have to disagree with their conclusions because of several methodologic problems.

The most important methodologic issue, which should be obvious to someone familiar with tracheostomy, is that a cuffed tracheostomy tube was used in the surgical group, whereas the canula in the Arndt cricothyroidotomy set is without a cuff. A cuffed canula, although much better for ventilation, is much more difficult to insert. In addition, the canula provided in the Arndt set is of a smaller diameter, an ID of 3 mm versus 5 mm for the Mallinckrodt tracheotomy tube. It is easier to insert a smaller, smooth object into tissues, and I doubt that many anesthesiologists will find the ventilation through a 3-mm tube to be equivalent to that through a 5-mm tube!

A second related question is the additional 70% of time spent for the connection of the ventilation equipment (10.1 s in the Seldinger technique group and 17.4 s in the surgical technique group) in the surgical cricothyroidotomy group. Because in both cases the tracheal tubes used ended with a connection piece specifically made to fit standard ventilation equipment, our unique potential explanation is that time is taken to inflate the cuff. The authors do not discuss this difference in the text, and a thorough explanation seems necessary.

Probably the most bothersome aspect of this study is the way the authors interpret the failures of the trials. In the Seldinger group, there were seven cases that were classified unsuccessful, to which should be added four cases where the tube was found by the pathologist in the subcutaneous tissues. To our knowledge, placing the tube in front of the trachea can hardly be considered as a successful placement allowing ventilation. Therefore, the failures in this group amount to 11.8% (11 of 95), which should be compared with a failure rate of 6.4% (6 of 94) in the open cricothyroidotomy group. A simple statistical test shows this difference to be highly significant. Contrary to what might be argued by the authors, the injury of vessels is rare in the area of the cricothyroid membrane and is far less important than the misplacement of the canula.

Another point of lesser importance is the use of the ‘Viennese tracheal dilator’ in the surgical cricothyroidotomy. We doubt that most of the readers of ANESTHESIOLOGY are familiar with this tool, and without further description, it is unclear why such a dilator would provide any advantage over classic surgical tracheal–laryngeal hooks for spreading of the tissue. What it certainly does is increase the time spent for the insertion of the endotracheal tube and ventilation, the two main outcomes the authors chose to evaluate.

Finally, it is surprising that in a randomized study, the cadavers were significantly heavier and with larger necks in the surgical group. Stating that ‘the differences in weight and circumference of the neck were not clinically relevant’ is either frivolous or represent a misunderstanding of the risk factors for this operation.

In conclusion, this study, which seems exemplary at first glance, suffers from major methodologic flaws. Doubling of the failure rates should be an obvious reason to prefer a procedure, especially when a failure for cricothyroidotomy means a probable death for the patient. Overlooking these data and basing the conclusion on the duration of the procedure seems bewildering. Furthermore, these delays (time to tube insertion and time to first ventilation), although seeming objective, are somewhat subjective because they were performed by an unblinded and hopefully unbiased observer.

Pavel Dulguerov, M.D., P.D.D.* Claudine Gysin, M.D. Geneva University Hospital, Geneva, Switzerland. pavel.dulguerov@hcuge.ch

References


(*Geneva University Hospital, Geneva, Switzerland. pavel.dulguerov@hcuge.ch

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It would be more appropriate for the study to have compared the cuffed Cook Melker airway to a cuffed tracheostomy tube inserted with the rapid four-step technique.

Richard J. Price, F.R.C.A., Gartnavel Hospital, Glasgow, United Kingdom. rjprice@doctors.org.uk

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To the Editor—Welsby et al.1 found that among patients who underwent cardiopulmonary bypass (CPB) for coronary artery bypass graft surgery, systemic hypotension, pulmonary hypertension, or both after protamine administration were associated with in-hospital mortality. Their data do not suggest a mechanism for this.

We have reported that ischemic ST-segment elevation occurs in temporal association with protamine administration.2 Angiographically detected vasospasm toward the end of surgery and early in the postoperative period has been reported to be a cause of myocardial infarction (MI) in several case reports, even in the patients who were not known to have vasospasm other than during the perioperative period.2–4 The right coronary artery is most commonly involved, even when it is not stenosed or grafted.2–4

The coronary endothelium is abnormal as a result of atherosclerosis. It is further damaged because of ischemia, which may occur before, during, and after CPB. Ischemia may lead to reduction in the anticoagulant and vasorelaxing properties of the coronary microcirculation, predisposing to small-vessel closure and myocyte injury. Crystallloid potassium cardioplegia also damages the endothelium. Potassium in the cardioplegia may also lead to vasoconstriction. Further damage occurs on reperfusion after release of aortic occlusion.

In this milieu, various stimuli, especially the administration of protamine, may trigger thrombosis and vasospasm. Thrombosis may also accentuate vasoconstriction. Vasomotion may significantly contribute to the occurrence of MI.3,4 Thrombosis may be potentiated by low concentrations of antithrombin III after CPB. Protamine also inhibits the neutralization of thrombin by antithrombin III. The occurrence of vasospasm and thrombosis may lead to myocyte injury and necrosis. Protamine administration may lead to thrombosis even if the endothelium is intact. Protamine may also interact with plasma proteins to form complexes that may partially obstruct the coronary circulation. As Welsby et al.1 point out, protamine administration leads to increased concentrations of complements C3a and C4a. The salutary effect of heparin on the endothelium may be lost on the administration of protamine.

If reduction in myocardial perfusion after protamine administration is persistent, myocyte necrosis may occur within hours after protamine administration. Hemodynamic instability and hemodilution may also contribute to the necrosis.3–5 Toward the end of surgery and in the early postoperative period, echocardiographic abnormalities and systolic dysfunction have been reported.5 Reduction in perfusion after protamine administration may depress ventricular function, contributing to systemic hypotension and pulmonary hypertension.

In a large multicenter study,7 we reported that among the patients who met Q-wave criteria for MI after coronary artery bypass graft surgery, a new Q wave was first recorded immediately after surgery in 39% of patients and by the morning of the first postoperative day in 80% of patients. Peak serum creatine kinase MB concentration occurred within 16 h of release of aortic occlusion in approximately 70% of the patients who had MI. There was a strong association with these MIs of episodes of ST-segment deviation occurring minutes to hours after the release of aortic occlusion.6 This suggests that most of the MIs were triggered in this period.5 MI diagnosed by a new significant Q wave3,4,6 or increased creatine kinase MB3,4,6,7 are reported to be associated with increased mortality.

We found that electrocardiographic changes occurring intraoperatively after CPB and duration of post-CPB hypotension were independent predictors of MI.5 We also found that intraoperative hypotension and other hemodynamic abnormalities occurring randomly do not correlate with ischemia.6 Hypotension is likely to contribute to MI when occurring during a period of ischemia such as after protamine administration. In our studies, hemodynamic changes occurred despite attempts to maintain hemodynamic support.

Welsby et al.1 may be able to determine whether the patients who expired in their study had ischemia or MI and whether this was considered to be the primary cause of death. Many of these patients are likely to have ischemic changes on 12-lead electrocardiogram recorded at the time of arrival at the intensive care unit.

Uday Jain, M.D., Ph.D., St. Mary’s Medical Center, San Francisco, California. uday_jain@yahoo.com

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In Reply—We thank Dr. Jain for his interest in our article,1 in which we note an association between a spectrum of hemodynamic perturbations after protamine administration and post-cardiac surgery inhospital mortality. His comments resemble our own mechanistic speculations and outline plausible pathophysiological scenarios whereby protamine administration could provoke coronary thrombosis and myocardial infarction.2 It is of interest that Dr. Jain’s group has also observed a relation between protamine administration, hypotension after cardiopulmonary bypass, and poor outcome.

There are opportunities to investigate the mechanisms proposed by ourselves and Dr. Jain’s group. If the complement pathway is important in the pathophysiology of this phenomenon, existing data from complement inhibitor trials may demonstrate reduced postprotamine hypotension in treatment arms.3 Similarly, bradykinin antagonists4 may

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prove useful in characterizing the role of bradykinin. To our knowl-
edge, no such comparisons have been performed. This area of research
also has interesting direct clinical implications for therapies that influ-
ence circulating bradykinin activity (e.g., aprotinin, angiotensin-con-
verting enzyme inhibitors).

Clinical evidence does not support Dr. Jain’s speculation that overt
short-term myocardial depression is primarily responsible for the find-
ings of our study. Our experience with intraoperative transesophageal
echocardiography indicates that new segmental wall motion abnormal-
ities or deterioration of myocardial function after cardiopulmonary
bypass are infrequently detected, whereas we observed a predefined
metric of hypotension after protamine administration in 12.4% of
patients, and more than 25% of patients had a recorded systolic blood
pressure less than 70 mmHg in the 30 min after protamine administra-
tion. In this regard, we do not believe there is value in further dissec-
ting an associative database study; we intentionally examined ‘‘hard’’
outcomes such as mortality and digitally recorded hemodynamic data
to avoid some of the limitations of a retrospective study. The ideal
investigation to assess for causality awaits comparison of protamine
with an (inert) alternate heparin reversal agent. However, the chal-
lenge lies in finding a suitable alternative.

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A New Technique for Obtaining Large-bore Peripheral Intravenous
Access

To the Editor:—I have devised an effective technique for placing
large-bore intravenous catheters in patients considered to be “hard
sticks.” The idea occurred to me after a morning of performing Bier
blocks for upper extremity procedures.

After placing a tourniquet on the upper arm, the provider introduces
a small-bore (20- to 24-gauge) catheter into the dorsal hand or volar
wrist. Without removing the tourniquet, a volume of crystalloid (I have
used 60 ml normal saline, but the volume could vary according to the
circumstances) is injected via the catheter. The saline will distend the
arm veins to the point where a large-bore catheter can be placed easily.

I recently applied this technique in the case of a patient with a
known placenta accreta who was scheduled to undergo cesarean
delivery. She seemed to have poor peripheral venous access, so I
distended the veins in her right arm by injecting saline via a 20-gauge
catheter in the back of her hand. I had no trouble placing an 18-gauge
catheter in her “intern’s vein.” I was then able to change the catheter
via Seldinger technique to an 8.5-French Rapid Infusion Catheter
(Arrow International, Inc., Reading, PA). The patient was thus spared
the risks associated with central venous catheter placement.

Jonathan I. Stein, M.D., York Hospital, York, Pennsylvania.
j.i.stein@worldnet.att.net

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Using the Circuit Elbow to Prevent Capnometer Tube Kinking

To the Editor:—Capnography is an important monitor in anesthesia
care that assists with detection of esophageal intubation and with
assessment of ventilation. Preoperatively,1 it is important to check the
capnograph system on the anesthesia machine to detect any malfunc-
tions before intubation, because there may be occlusion, incorrect
attachment of the sampling tube,2 or other problems. Unfortunately,
the capnograph can still be lost intraoperatively as a result of mechan-
ical failures of the sampling tube. We find that in cases with the bed
turned 90° or 180° from the anesthesiologist, the sampling tube can
kink at the connection with the breathing circuit Y-piece as a result of
weight from draping or the surgical field. This results in loss of the
capnogram because of occlusion, which causes both alarm and frus-
tation because the anesthesia provider must investigate the source of
the occlusion after appropriate patient ventilation is confirmed. Occa-
sionally, brief interruption of the surgeon or scrub nurse is necessary.
Traditionally, we have taped the sampling tube in a half-loop at this
connection point; this prevents kinking somewhat with axial loads but
still cannot prevent lateral motion and subsequent occlusion.

During a maxillofacial surgery case in which the capnometer sam-
ppling tube kinked multiple times, we discovered that the circuit elbow,
normally set aside when flexible corrugated tubing is attached to the
endotracheal tube, can be used to protect the sampling tube at the
attachment with the Y-piece. Fortunately, the elbow piece is appro-
priately sized to allow passage of the tube while also creating a rigidly
protected and controlled 90° bend (fig. 1). Tape is used to secure the
elevator firmly against the Y-piece. This technique has become popular at
our institution for helping prevent occlusion of the capnometer sampling
tube and seems to provide better protection than taping the tube directly.

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To the Editor—Impaction\(^1\) of the distal cuff with the glottic inlet is a well-recognized complication of laryngeal mask airway insertion, but it is rarely reported because it is frequently mistaken for and/or associated with reflex glottic closure and requires fiberoptic assessment for confirmation.\(^4\) There is little information about the incidence, with one study reporting that it occurred in 4% of anesthetized patients\(^2\) and another reporting that it occurred in 2% of sedated patients.\(^3\) In principle, it is more likely to occur if the distal cuff follows an anterior path, the larynx is posteriorly placed, or the cuff presents a broad leading edge. We describe three cases of airway obstruction after laryngeal mask insertion where rapid fiberoptic assessment differentiated between impaction and glottic closure and facilitated prompt and appropriate treatment.

The first patient was a 34-yr-old man with an American Society of Anesthesiologists physical status of I who was undergoing minor general surgery with no anticipated airway problems. Induction was with 3 mg/kg propofol and 1 \(\mu\)g/kg fentanyl. A size 5 disposable LMA-\(^\text{Unique}\) (Laryngeal Mask Company, Ltd., Mahe, Seychelles) was easily inserted by a skilled user using the standard technique, and the cuff was inflated to an intracuff pressure of 60 cm H\(_2\)O. Ventilation was impossible, with airway obstruction and an audible oropharyngeal air leak at high airway pressures. There was no coughing or bucking. Fiberoptic inspection was completed within 30 s and showed that the distal cuff had impacted with the glottic inlet and the vocal cords were open (fig. 1). The LMA-\(^\text{Unique}\) was removed and reinserted, and a clear airway was obtained. Repeat fiberoptic assessment showed that the distal cuff was sitting in its correct position in the hypopharynx (fig. 1). There were no other complications, and the minimal oxygen saturation measured by pulse oximetry (Sp\(_O_2\)) was 95%.

The second patient was a 41-yr-old man undergoing minor urologic surgery. Induction, insertion technique, and cuff inflation and type/size of the laryngeal mask airway were identical to the first case, but in addition, the patient coughed during insertion. Fiberoptic inspection was completed within 30 s and showed that the distal cuff had impacted with the glottic inlet and the vocal cords were open (fig. 1). The LMA-\(^\text{Unique}\) was removed and reinserted, and a clear airway was obtained. Repeat fiberoptic assessment showed that the distal cuff was sitting in its correct position in the hypopharynx (fig. 1). There were no other complications, and the minimal oxygen saturation measured by pulse oximetry (Sp\(_O_2\)) was 95%.

The second patient was a 41-yr-old man undergoing minor urologic surgery. Induction, insertion technique, and cuff inflation and type/size of the laryngeal mask airway were identical to the first case, but in addition, the patient coughed during insertion. Ventilation was impossible with an audible oropharyngeal leak at low airway pressures. The fiberoptic finding, management, and outcome were identical to the first case.

**Fig. 1.** Threading the sampling tube through the unused elbow.

**Fig. 1.** Impaction of the distal cuff of the disposable laryngeal mask airway with the glottic inlet (left) and the distal cuff sitting in its correct position (right).
The third patient was a 56-yr-old woman with an American Society of Anesthesiologists physical status of II who was undergoing minor orthopaedic surgery with no anticipated airway problems. Induction was with 2.5 mg/kg propofol and 10 μg/kg alfentanil. A size 4 LMA-Classic™ (Laryngeal Mask Company, Ltd.) was easily inserted by a skilled user using the standard recommended technique, and the cuff was inflated with 15 ml air. Ventilation was impossible, with airway obstruction and an audible oropharyngeal air leak at high airway pressures. There was no coughing or bucking, but there was a slight movement of the foot during insertion. Fiberoptic inspection was completed within 20 s and showed that the distal cuff was sitting in its correct position in the hypopharynx but the vocal cords were closed. An additional 100 mg propofol was administered, continuous positive airway pressure was applied, and the airway obstruction was relieved within a minute. There were no other complications, and the minimal SpO₂ was 98%.

We strongly recommend the rapid use of the fiberoptic scope to assess the etiology of airway obstruction after the insertion of a laryngeal mask, because the treatment for impaction is reinsertion and the treatment for reflex glottic closure is to deepen anesthesia or give a muscle relaxant, unless associated with a transient swallow, in which case, the glottis will spontaneously reopen within 20–30 s. On occasion, impaction will also lead to reflex glottic closure, in which case both treatments may be required. If reinsertion using a different approach does not correct the problem of glottic impaction, the simplest solution is to place a guide in the esophagus and railroad the LMA-ProSeal™ (Laryngeal Mask Company Ltd., Nicosia, Cyprus) into position along its drain tube.4,5


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