

Arterial Oxygen Desaturation after Induction of Anesthesia

To the Editor:—Naguib *et al.*¹ 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.² 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.³ We showed convincingly that desaturation was more likely in obese patients and 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.*⁴ 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.*⁵ 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.

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In Reply:—I was aware of the study by Drummond and Park,¹ and as Dr. Drummond indicated in his letter, his study bears no direct resemblance to ours.² The aims and methods of both studies were different. Dr. Drummond studied the effect of different preoxygenation techniques on hemoglobin saturation in patients anesthetized with 3–5 mg/kg thiopental. Neuromuscular block was established with 100 mg succinylcholine. Incremental doses of thiopental and succinylcholine were administered to maintain anesthesia and apnea for 3 min. Dr. Drummond then evaluated the changes in hemoglobin saturation over a period of 3 min. The Hewlett Packard ear oximeter used in Dr. Drummond’s study¹ was not only “clumsy,” as characterized by Dr. Drummond, but also inaccurate (see Douglas *et al.*³).

Dr. Drummond disagrees on where to put the emphasis on mechanisms underlying the observed hypoxic trend in my report. He suggested that reduction in functional residual capacity has a more important contribution to hemoglobin desaturation than shunting in anesthetized obese patients. I have described the effect of anesthesia on functional residual capacity in my article.² I still believe that shunting played a significant role in my observations. In reality, as I stated in the Discussion, “reduced functional residual capacity . . . will result in an increase in shunt fraction because of collapse of alveoli.” Further, Gunnarsson *et al.*⁴ reported that 87% (39 of 45) of their patients had development of atelectasis and shunting during anesthesia and concluded that “during anesthesia, shunt influenced PaO₂ most.” Although the mechanisms underlying my results may be open to disagreement, the lesson is that the risk of desaturation in the immediate postinduction period is much greater than most clinicians recognize if there were failure to intubate and ventilate our patients.

Dr. Drummond also stated that the model prediction of Hardman *et al.*⁵ . . . was more complete than the model studies” cited in my report. I do not agree. I cited the work of Hardman *et al.* Nevertheless, in the study of Hardman *et al.*, the authors used a simulator to examine the onset and course of hypoxemia during apnea. They evaluated one factor at a time (and this does not reflect the clinical reality). They

concluded that the time to 50% oxyhemoglobin saturation varied from 11 to 8 min in an open and an obstructed airway apneic patient model, respectively. This prediction is at variance with my observations² and those of others.⁶ Although none of patients in my study² or the volunteers in the study of Heier *et al.*⁶ were allowed to have hemoglobin saturation decreased less than 90% and less than 80%, respectively, times to 50% hemoglobin saturation in the aforementioned studies would have been shorter than that predicted by Hardman *et al.*⁵

Models, by definition, attempt to simplify reality. Models can be useful and effective teaching tools, but ultimately, they require validation. The observations in my investigation were much more alarming than conventional wisdom (and modeling) would predict. When observed data conflicts with theory, it is the theory that must be challenged.

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Cricothyroidotomy: Do Not Compare Small Apples to Big Oranges

To the Editor:—Schaumann *et al.*¹ 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,² 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 tracheostomy 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) 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 93), 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 any 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.

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Surgical Cricothyroidotomy Technique

To the Editor:—Schaumann *et al.* are to be congratulated on their large and detailed study on cricothyroidotomy techniques.¹ However, the suggestion that their results favor the Seldinger technique as a method of inserting a surgical airway is misleading. The control technique used was inappropriate; the airways used were not comparable, and uncuffed narrow bore tubes may not be suitable as emergency airways. The clinical applicability of the study is therefore limited. The authors did not discuss these deficiencies in their article.

The results do not necessarily support the use of a Seldinger technique but rather demonstrate that their standard technique of cricothyroidotomy is more time consuming. Their standard technique is more complex than that originally described for elective situations:² It involves, in addition, both vertical and horizontal incisions and also use of both dilation and a tracheal hook. Other techniques have been developed for emergency situations. These include that of the Advanced Trauma Life Support course³ and the rapid four-step technique.⁴ Expert reviewers have recommended such techniques.⁵ It may be possible to secure an airway in 32 s,⁴ as opposed to 109 s with the Arndt airway¹ or 137 s¹ or 114 s⁴ for a standard technique. Although these techniques may have their own problems,^{4,6} they have been

shown to work in clinical practice.⁷⁻¹⁰ Comparison of a Seldinger airway for emergency use with one of these techniques would have been more valid. Elective techniques have previously been used as a control in studies of a new emergency technique,^{5,11} and this has been criticized.^{12,13}

The study assessed only the Arndt airway. The Seldinger technique is used with other airway devices. The Arndt airway is an uncuffed device of 3 mm ID. Subjective ease and objective speed of insertion of the Arndt airway may be a consequence of its narrower diameter when compared with the control airway: 5 mm ID plus a cuff. This may also account for the differences in injuries to the larynx. It has been shown that larger airways require an increased force for insertion.¹⁴ It would have been more appropriate to have used a Seldinger cricothyroidotomy airway with a diameter comparable to that of the tracheostomy tube used. When the Cook Melker airway was compared with a standard elective technique, there was no difference in time of insertion.¹⁵

Reoxygenation and ventilation of the patient must also be considered in the assessment of a novel airway device. Clearly, this is a limitation of cadaver studies. It is likely that the performance of uncuffed narrow bore tubes depends on the degree of upper airway obstruction.¹⁶ Their use as emergency airways has been criticized.¹⁷⁻¹⁹

It would have been 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.

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In Reply:—We appreciate the great interest in our article.¹ In response to Drs. Dulguerov and Gysin, referring to the question of an uncuffed *versus* a cuffed canula, although it seems obvious that it is easier to insert a smaller canula, the difference is not as great as expected. However, our aim was to compare two different recognized methods of cricothyroidotomy and not a cuffed *versus* an uncuffed canula. Although ventilation is not comparable between a 3- and a 5-mm tube, oxygenation may be sufficient during the first few minutes. Neither method is assumed to serve as a long-time device.

Referring to the authors' second question, we do not fully understand the "additional 70%": The few seconds (10-17) spent were necessary to inflate the cuff, to connect the valve of the breathing bag, and to deliver the first squeeze of the bag. Although one assumes that everything goes faster, we often need more time in the real world—at least in this study.

Regarding the failures (page 9, paragraphs 3 and 4), there is confusion of Drs. Dulguerov and Gysin between accurate placement and injuries: There was a failure rate of 11.8% in group 1 (including the four misplacements) and a failure rate of 16% in group 2. The punctures of the thyroid vessels in group 2 are not listed as failures but as injuries and did not necessarily prevent insertion of the canula.

The "Viennese tracheal dilator" is a piece of our standard equipment. We agree that the authors can use the hook or other device they are familiar with.

We are concerned about the authors' use of the aggressive terms of "frivolous" and "misunderstanding." Having performed more than 500 cricothyroidotomies in corpses ourselves, we are sure that a mean difference of 1.6 cm in neck circumference is not clinically relevant. If someone has performed more cricothyroidotomies in corpses, we are ready for discussion.

In response to Dr. Price, we could not find a major time difference whether a vertical and horizontal incision was used or only horizontal incisions. The reason for a vertical incision is that in wide necks, it is easier to find the cricothyroid membrane.

With regard to time, Holmes *et al.*² state,

A surgical airway was established in 28 of 32 attempts with the use of the rapid four-step technique (88%); the average time elapsed

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before tube placement was 43 s. Thirty of 32 attempts involving the standard technique (94%) were successful; the average time to tube placement was 134 s (95% confidence interval for a difference of 91 s, 63 to 119; $P < 0.001$). Complications were identified in 12 attempts involving the standard technique (38%; 1 considered major) and in 12 involving the rapid four-step technique (38%; 3 considered major).

The time varies between 4 and 134 s. Furthermore, this study did not include first ventilation. In addition, major complications occurred very often in this study (38% in both groups). So, the fastest time was accompanied by severe complications.

As stated above, we wanted to compare a standard technique to another commercially available kit. Of course, other methods may be similarly useful. We agree that a device with a larger cuff may require more time; however, if it is possible to shorten the time, allowing adequate oxygenation, even without adequate ventilation, may be valuable for patients in the first few minutes.

We thank Dr. Price for the idea of comparing the cuffed Cook Melker airway with a cuffed tracheostomy tube with the rapid four-step technique. If our time allows, we will investigate these devices in the future.

Again, we thank both readers for their valuable comments and helpful criticism. However, we cannot agree that the clinical applicability of our study is limited.

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Protamine Contributes to Myocardial Ischemia

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. Crystalloid 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.³ Reduction in perfusion after protamine administration may depress ventricular function, contributing to systemic hypotension and pulmonary hypertension.

In a large multicenter study,⁵ 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.⁵ This suggests that most of the MIs were triggered in this period.⁵ MI diagnosed by a new significant Q wave^{3,4,6} or increased creatine kinase MB^{3,4,6,7} 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.⁸ 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 homeostasis.

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.

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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 in-hospital mortality. His comments resemble our own mechanistic speculations and outline plausible pathophysiologic 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

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

Clinical evidence does not support Dr. Jain's speculation that overt short-term myocardial depression is primarily responsible for the findings of our study. Our experience with intraoperative transesophageal echocardiography indicates that new segmental wall motion abnormalities 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 administration. In this regard, we do not believe there is value in further dissecting 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 challenge 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.

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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 malfunctions before intubation, because there may be occlusion, incorrect attachment of the sampling tube,² or other problems. Unfortunately, the capnogram can still be lost intraoperatively as a result of mechanical 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 frustration because the anesthesia provider must investigate the source of

the occlusion after appropriate patient ventilation is confirmed. Occasionally, 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 sampling 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 appropriately 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 elbow 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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Fig. 1. Threading the sampling tube through the unused elbow.

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Utility of Fiberoptic Assessment in the Differential Diagnosis of Glottic Impaction versus Reflex Glottic Closure with the Laryngeal Mask Airway

To the Editor:—Impaction¹ 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.¹ There is little information about the incidence, with one study reporting that it occurred in 4% of anesthetized patients² 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.

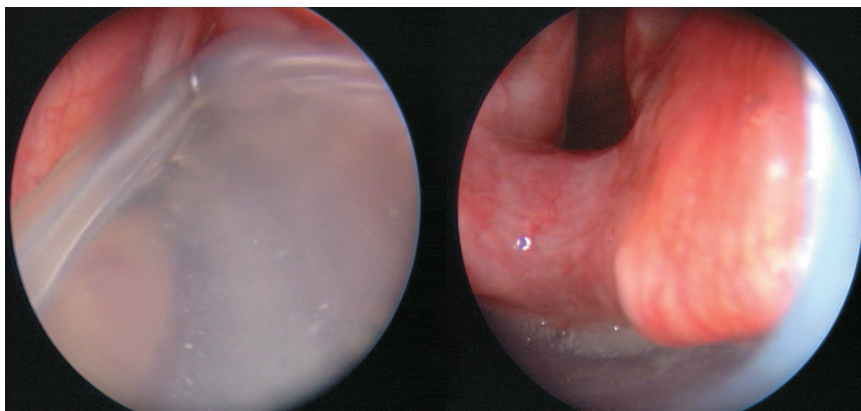
The first patient was a 34-yr-old man with an American Society of Anesthesiologists physical status of I who was undergoing minor general surgery with no anticipated airway problems. Induction was with 3 mg/kg propofol and 1 μg/kg fentanyl. A size 5 disposable LMA-

*Unique*TM (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 H₂O. 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-*Unique*TM 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 (SpO₂) 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.

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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 μ g/kg alfentanil. A size 4 *LMA-Classic*TM (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 ap-

proach does not correct the problem of glottic impaction, the simplest solution is to place a guide in the esophagus and railroad the *LMA-ProSeal*TM (Laryngeal Mask Company Ltd., Nicosia, Cyprus) into position along its drain tube.^{4,5}

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