To the Editor—I read with great interest the recent report by Shore-Lesserson and Reich, detailing diffuse venous thromboembolism in the setting of aprotinin and adult deep hypothermic circulatory arrest. To my knowledge, this is the first reported case of venous thrombosis associated with aprotinin and adult deep hypothermic circulatory arrest in the era of adequate heparinization, as defined by standard-of-care activated clotting time and heparin levels. This case report adds to the recent reports of arterial thrombosis (both pulmonary and systemic) associated with aprotinin in adult cardiac surgery with or without deep hypothermic circulatory arrest.2–4

Therefore, significant life-threatening thrombosis is possible throughout the cardiovascular system during complex cardiac surgery in the setting of aprotinin, despite standard-of-care heparinization. It seems to be uncommon, as evidenced by randomized controlled trials.5

Conceptually, perioperative vascular thrombosis could cause mortality in the intraoperative or postoperative period. The case reports describe intraoperative death in this scenario.1–3 The possibility of death and/or serious morbidity in the postoperative period from vascular thrombosis associated with aprotinin has recently been raised.6,7

Massive vascular thrombosis associated with aprotinin in complex cardiac surgery is rare, but real and catastrophic. The common factor in all the case reports is the onset during or shortly after heparin reversal with protamine, heralded by hemodynamic collapse and ventricular failure.1–5 There may or may not be an identified prothrombotic risk factor including factor V Leiden.1 Clearly, there is a net prothrombotic effect achieved during or after heparin reversal, triggering disseminated major acute intravascular thrombus. On the basis of the existing reports, further clarification of the mechanism is conjecture. However, it is also clear that this phenomenon is not only possible with aprotinin but also with aminocaproic acid.4,8

In complex cardiac surgery, pharmacologic dampening of fibrinolysis reduces alloimmune transfusion and mediastinal recollection for bleeding, an independent predictor for perioperative mortality.9 There is, however, a small but important risk of catastrophic cardiovascular thrombosis in the setting of antifibrinolytic exposure, despite standard-of-care anticoagulation with heparin (monitored by activated clotting time and/or heparin level).

How do we balance these risks? Should the criteria for heparin-based anticoagulation be refined? If so, how and based on what evidence? What about the role of possible concomitant antithrombin deficiency?10 Should patients be screened for underlying procoagulant conditions such as factor V Leiden?11,12 How are all of these considerations modified in the presence of direct thrombin inhibitors, given their arrival as alternatives to heparin for clinical anticoagulation and suppressive effects on thrombin generation?13–15 What is the clinical significance of aprotinin’s effect on protein C activity?15

These case reports together ask many important questions. There is an imperative for further data, not only an international registry but also further clinical trials, to balance the benefits and risks of antifibrinolytics in complex cardiac surgery with cardiopulmonary bypass. The BART study (blood conservation using antifibrinolytics: a randomized trial in a cardiac surgery population) is an important step in this direction.

John G. T. Augusto d 112, M.D., F.A.S.E., The Hospital of University of Pennsylvania, Philadelphia, Pennsylvania. yiandoc@hotmail.com

References


(Accepted for publication November 15, 2006.)
To the Editor.—We read with great interest the report of Shore-Lesserson and Reich1 regarding a fatal case of venous thromboembolism during cardiac surgery with hypothermic circulatory arrest associated with the use of aprotinin in a patient diagnosed, with postmortem analysis, as a carrier of factor V Leiden. The same authors had previously described two fatal cases of intraoperative thrombosis in patients undergoing the same surgical procedure and treated with L-aminocaproic acid: One of the two patients was a postmortem-diagnosed carrier of the factor V Leiden mutation.2 Because of the occurrence over a 3-yr period of four fatal thrombotic events in cardiovascular patients operated on with hypothermic circulatory arrest and treated with antifibrinolytic drugs, the authors of these reports are now screening for the factor V Leiden mutation all patients scheduled to undergo elective surgical procedures requiring hypothermic circulatory arrest to avoid the use of antifibrinolytic drugs in patients who are carriers of the mutation.

It has been proposed to classify the major hereditary prothrombotic conditions in two major groups, including hereditary deficiencies of natural anticoagulants and hereditary disorders associated with increased function of coagulation factors.3 The factor V Leiden mutation, which renders activated factor V resistant to proteolysis by activated protein C, belongs to the second group of inherited prothrombotic conditions and is frequently observed in white but not in Asian or African people.4 Whereas many subjects with deficiency of natural anticoagulants experience venous thromboembolism before the age of 60 yr, only a minority of factor V Leiden carriers will ever develop thromboembolic events.5 If factor V Leiden plays a contributory role in the development of intraoperative thrombosis in patients undergoing cardiac surgery with hypothermic circulatory arrest and receiving antifibrinolytic drugs, then patients with deficiency of natural anticoagulants should be at even greater risk, also given the effect of hemodilution. In addition, screening for the prothrombin G20210A mutation should also be recommended, because the associated hyperprothrombinemia has been shown to inhibit plasma fibrinolysis through a TAFI-mediated mechanism.5 On a cost–benefit basis, screening of the general population for thrombophilia defects is ineffective, and it is currently a matter of debate whether such screening should be performed even in patients with venous thromboembolic events.6 In the absence of evidence-based data, caution against screening for factor V Leiden patients undergoing cardiovascular surgery with hypothermic circulatory arrest has already been suggested.7 Before depriving patients at high risk for bleeding of the proven antihemorrhagic effect of antifibrinolytic drugs,8–10 we suggest the institution of an international registry of severe thrombotic complications occurring during cardiac surgery to study the prevalence and the possible causes of this surely underestimated phenomenon. If thrombophilia plays an important role, one would expect history of thromboembolism to be associated with an increased occurrence of this devastating complication of cardiac surgery.

Valter Casati, M.D.,* Piero Paolo Zanetti, M.D., Armando D’Angelo, M.D.* Policlinico di Monza, Monza, Italy. valter.casati@policlinicodimonza.it

References

2. Fanashawe MP, Shore-Lesserson L, Reich DL: Two cases of fatal thrombosis after aminocaproic acid therapy and deep hypothermic circulatory arrest. ANESTHESIOLOGY 2001; 95:1525–7

(Accepted for publication November 15, 2006.)

In Reply.—We are pleased to respond to the comments that address our case report “A Case of Severe Diffuse Venous Thromboembolism Associated with Aprotinin and Hypothermic Circulatory Arrest in a Cardiac Surgical Patient with Factor V Leiden.”1 We agree with Dr. Augoustides that thrombosis after cardiac surgery is a rare event, yet often fatal. When it is reported, it is temporally related to the reversal of heparin with protamine, presumably because the anticoagulant effect of heparin therapy is being neutralized and thus any “protective” effect from thrombosis is removed. Dr. Augoustides’ suggestion that this may be the first report of venous thrombosis in association with antifibrinolytic therapy is true with respect to the published literature; however, we know this complication to be dramatically underreported. Furthermore, it remains to be proven that thrombosis of the pulmonary artery is truly “arterial” pathology. Often, this complication is the result of venous thromboembolic phenomena that present as pulmonary thromboembolism.

The sheer volume of cardiac surgical procedures that are performed using antifibrinolytic therapeutic agents where no occult thrombosis occurs, further affirms the hypothesis put forth. That is, when life-threatening thrombosis occurs in association with cardiac surgery and...
antifibrinolytic therapy, there should be some other hypercoagulable predisposition responsible for tipping the balance in favor of thrombosis. This delicate balance between bleeding and thrombosis is steadied by procoagulant factors, anticoagulant factors, fibrinolysis, and platelet-related factors. Many of these previously undiagnosed adverse thrombotic outcomes are now prospectively being identified as heparrin-induced thrombocytopenia type 2, as a result of better diagnostic techniques. This addresses Dr. Augoustides’ question regarding the use of direct thrombin inhibitors. We would agree that better suppression of thrombin formation coupled with the avoidance of heparin would reduce the occurrence of many of these adverse thrombotic events. The suggestion for an international registry for reporting of thrombotic events is commendable and would be supported by us. An international registry for deep hypothermia and circulatory arrest is also currently under investigation.

We also embrace the comments of Dr. Casati et al. in that they have also suggested a registry for the reporting of adverse thrombotic events. However, we do continue to support the screening of elective deep hypothermia and circulatory arrest patients in our institution. This represents a very small subset of cardiac surgical patients at any institution. The cost is therefore not prohibitive, and the accuracy of testing is extremely high. Both the factor V Leiden mutation and the prothrombin mutation G20210A occur with a prevalence of 1–8% in the European population and are even less prevalent in Asian and African-American persons. Therefore, the number of patients identified as positive will be small. Donahue et al. have shown that patients with factor V Leiden can undergo cardiopulmonary bypass safely even with the use of antifibrinolytic drugs. In fact, these patients have less bleeding and may not need the benefit of antifibrinolytic agents, irrespective of the safety of this practice. Therefore, the question arises: Is a patient with a genetic predisposition to hypercoagulability one that would be considered to benefit from the use of antifibrinolytic agents?

Dr. Casati et al. suggest that patients with a deficiency of anticoagulant activity would be at greater risk than those with an excess of procoagulant activity due to hemodilution. We do not necessarily agree with this conclusion and think that it is difficult to conjecture which groups of patients would be at highest risk for thrombosis. The hemostasis system is rich with feedback mechanisms and protective pathways that act as fail-safe mechanisms to ensure normal clotting. When a patient has excess thrombotic activity, it is the fibrinolysis system that acts to restore the balance. If fibrinolysis is inhibited, coagulation can proceed unchecked. However, a patient with deficient fibrinolysis (such as PAI-1 excess or the prothrombin mutation G20210A) already has ineffective fibrinolysis and thus relies on other functional anticoagulant pathways that are not pharmacologically inhibited to restore the balance. In cardiac surgical patients where the perturbations of the hemostasis system are extreme, we support that patients with a history of hypercoagulability should be screened so that appropriate hematologic management can be instituted.

Linda Shore-Lesserson, M.D.*, David L. Reich, M.D.* Montefiore Medical Center, Bronx, New York. ishore@montefiore.org

References


(Submitted for publication November 15, 2006. Accepted for publication November 15, 2006.)

To the Editor—I read with interest the article titled ‘Intraoperative Tidal Volume as a Risk Factor for Respiratory Failure after Pneumonectomy’ by Fernández-Pérez et al.1 This is an important article because the traditional approach to one-lung ventilation has been to deliver 10–12 ml/kg tidal volume. As the authors pointed out, two previous studies reported that high intraoperative airway pressures during one-lung ventilation were associated with postoperative acute lung injury. The study by Fernández-Pérez et al.1 showed that larger tidal volumes were associated with a higher risk of postoperative respiratory failure. However, the largest tidal volume recorded on the chart was used in the analysis. This would most likely have been during two-lung ventilation, even if the tidal volume had been reduced during one-lung ventilation. If the tidal volume is not adjusted when initiating one-lung ventilation, the airway pressure will increase due to reduced compliance. It is possible that the ventilator will not deliver the full tidal volume, and then the largest recorded tidal volume would be the two-lung tidal volume. Data were missing in more than 20% of the cases in this study, but it may still have been useful to examine what data were available, because this is the critical time period, and apparently there still would have been more than 100 cases to analyze. It would be important to follow up this study with either a prospective, randomized study using different tidal volumes during one-lung ventilation or even a retrospective study in which the tidal volumes can be definitely correlated with one-lung ventilation.

The authors stated that their most interesting finding was the association of both large tidal volume and greater fluid administration with postoperative respiratory failure. This makes sense in that the larger fluid administration can lead to pulmonary edema, once there is a capillary leak from a ventilator-induced injury. The authors hypothesized that the larger tidal volumes might have led to hypotension, which ‘forced’ the anesthesiologists to administer more fluid. Although it is possible that resulting hypotension could have been treated with fluid, an alternative would have been infusion of a vasoconstrictor, such as phenylephrine. A more likely possibility is simply that the anesthesiologist who does not limit the tidal volumes or airway pressures during one-lung ventilation is less likely to be vigilant in limiting fluid administration. I do not understand, based on their data, how the authors concluded that even brief exposure to such ventilator settings could cause the postoperative complications.

Steve Neustein, M.D., Mount Sinai Medical Center, New York, New York. steve.neustein@msnyuhealth.org

Anesthesiology 2007; 106:875–6

Copyright © 2007, the American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.
In Reply:—We thank Dr. Neustein for his comments and appreciate the opportunity to reply.

We agree with Dr. Neustein that the absence of reliable data on airway pressures and tidal volumes during one-lung ventilation is a major limitation of our study. Indeed, we are currently undertaking a prospective cohort study in patients at high risk for postoperative pulmonary complications such as those undergoing lung resection to evaluate the cumulative exposure to potentially harmful intraoperative ventilator settings. This study includes a precise calculation of exposure during one-lung ventilation.

Our study design did not allow us to determine the mechanism of the observed interaction between the intraoperative tidal volume and fluid administration.1

A definitive trial to prove that intraoperative mechanical ventilation per se causes acute lung injury in humans would be difficult to design. Short-term large tidal volume mechanical ventilation during anesthesia has been associated with worsening pulmonary inflammatory response in experimental animal models.2,3 Despite some controversies, corresponding human data support the hypothesis that even brief exposure to high-tidal-volume ventilation influences the inflammatory and coagulation response in the lung.4–6 In our study, postoperative respiratory failure in the group of patients receiving larger tidal volume was observed during surgery as short as 244 min (25% interquartile range). Although optimal ventilator settings in patients undergoing pneumonectomy are yet to be determined, we believe that to maximize patient safety, routine use of very large tidal volumes (>10 ml/kg predicted body weight) during two- and in particular during one-lung ventilation is potentially harmful and should be avoided.7–9

References


Therapy in Postpuncture Meningitis

To the Editor:—We read with great interest the comprehensive overview by Baer4 regarding post-dural puncture (bacterial) meningitis (PDPM). However, some questions can be raised regarding treatment recommendations for patients with PDPM.

The recommendation for empiric treatment that is made in this article follows Infectious Diseases Society of America guidelines for Streptococcus pneumoniae meningitis.2 In these guidelines, use of vancomycin plus the third-generation cephalosporins ceftriaxone or cefotaxime is recommended with the addition of ampicillin in patients older than 50 yr.2 Of the 179 reviewed cases in this article, indeed, almost half had meningitis due to viridans streptococci; however, relatively high rates of Staphylococcus aureus (9 patients, 5%), Pseudomonas aeruginosa (8 patients, 4%), and Enterococcus faecalis (3 patients, 2%) were also found.1 This specific distribution of species stresses that PDPM should be regarded as a specific category of patients that most resembles the ‘standard’ category of patients with recent neurosurgery.3,4 In this category, recent guidelines recommend vancomycin plus cefazidime. Cefazidime, and not ceftriaxone or cefotaxime, has shown efficacy in several studies of patients with Pseudomonas meningitis.2,5 Alternatively, one can use cefepime, which also has greater in vitro activity than the third-generation cephalosporins.2,5

In addition, Baer recommends the use of adjunctive dexamethasone in patients with PDPM.1 In a recent European randomized clinical trial, adjunctive dexamethasone therapy reduced mortality from 15% to 7%.6 Of the total of 301 included patients in this trial, 9 (3%) had meningitis...
due to viridans streptococci, and were partly described previously. One of the 5 patients included in the placebo group died, versus 1 of the 4 in the dexamethasone group. Both patients died of S. milleri meningitis; others had meningitis due to S. salivarius (n = 4), S. sanguis (n = 2), and S. oralis (n = 1) (van de Beek, unpublished, October 2006). In a recent quantitative review of the effect of adjunctive dexamethasone in adults with community-acquired bacterial meningitis that included the results of five clinical trials, treatment with dexamethasone was associated with a significant reduction in mortality and neurologic sequelae. On basis of these data, dexamethasone is recommended in the subgroup of patients with PDPM.

**References**


(Accepted for publication December 8, 2006.)

**Think about Room Air**

Rather than go through the inconvenience of learning the feel of the saline method, those practitioners continue to subject the patient to the risk of infection. This ban should include the hanging drop method. Although less air is entrained, why allow any? It seems clear that the data show that room air has contaminated droplets and that injecting them into the epidural space is unnecessary and of greater risk than using saline. Of even more concern is how many other issues are out there, easily discovered, if we only followed up with our patients and accurately measured outcomes as we are morally obligated to, but rarely, do.

**References**


(Accepted for publication December 8, 2006.)

**Gloved and Masked—Will Gowns Be Next? Let the Data (Not Logic) Decide This Issue**

To the Editor—The editorial “Gloved and Masked—Will Gowns Be Next?” by Hepner is both interesting and disconcerting. The main reason surgeons resist neuraxial anesthesia is because “It takes too long!” For this reason, it is frightening to see the rubric “Will gowns be next?” Demanding that anesthesiologists wear gowns to perform neuraxial anesthesia will, in my opinion, be the death knell for spinal and epidural anesthesia. Locating and donning a gown for these procedures will obviously not make doing them less time-consuming. More importantly, is it even necessary?

Despite outcome data demonstrating a 1:10,000 to 1:50,000 risk of post–dural puncture meningitis, Dr. Hepner bases his recommendations for using gowns during neuraxial anesthesia on logic: “we must...
Parturients Expect Safe and Clean Regional Anesthesia

To the Editor—The largest single group of patients receiving central blockades worldwide is undoubtedly parturients. Fortunately, serious complications are rare, as pointed out in two recent studies published in Anesthesiology.1,2

Ruppen et al. found six epidural hematomas and justly considered these numbers inadequate to produce a robust estimate of event rates. Because of their study design, several cases reported in the literature are excluded from the statistical calculation performed in the meta-analysis. For example, the Closed Claims project included three spinal hematomas in obstetric patients.3 Many other excluded cases occurred in patients with impaired coagulation due to preeclampsia or large blood loss, conditions that might predispose even to a spontaneous spinal hematoma.4-6 Nonetheless, despite frequent bloody taps and thrombocytopenia, spinal hematoma is rare in obstetric patients.7,8

The compliant spinal canal in a young individual permits the introduction of volumes that may cause severe symptoms and permanent damage in an older person with a stenotic spinal canal.

However, a healthy parturient receiving epidural blockade for pain relief might overnight turn into a patient with severe coagulopathy. The calculated low risk then no longer applies to this patient. Such was the case with one patient in our study9: After delivery, the epidural catheter was removed before transferring the patient to the intensive care unit for treatment of the syndrome of hemolysis, elevated liver enzymes, and low platelets, and the subsequent spinal hematoma was diagnosed with delay. The perception of low incidence in the healthy parturient contributes to underestimation of risk when she becomes affected by coagulopathy.

Whereas patient characteristics seem to influence development and outcome after some severe complications, such as spinal hematoma and epidural abscess, no predisposing conditions are found for iatrogenic meningitis. Purulent meningitis will develop in any patient, however healthy, if bacteria are introduced into the spinal fluid, devoid of any immunologic defense. Dr. Baer’s comprehensive review of post–dural puncture meningitis (PDPM) includes reports of three tragic and unnecessary deaths in previously healthy obstetric patients. These cases could reflect a higher incidence of PDPM due to lower hygienic standards in the obstetric setting, lower diagnostic preparedness, or higher motivation to report these cases. Finally, the parturient could be at higher risk of developing PDPM. The reason being that α-hemolytic streptococci are normally part of the flora in the genitourinary tract and fluids containing these bacteria obviously will cover the lower backs of many parturients. As a consequence, faulty skin disinfection before performing a central blockade in the parturient could be more hazardous than in the average surgical patient.

The author and the editorial comment both address the incidence of PDPM.10 Important as this may seem, we would like to quote a recent editorial by Prof. Reynolds: “there can be no such thing as a ‘true incidence’ of an infective complication, . . . as its occurrence will vary with the number of risk factors both in local clinical practice and among the patient population.”11

We agree with Dr. Baer that these three mortalities, the increasing antimicrobial resistance of viridans streptococci, and oropharyngeal presence of more invasive pathogens should be sufficient for every anesthetist to adopt truly aseptic technique whenever performing a central blockade.

Analyzing literally every aspect of PDPM, the author does, however, leave one question unanswered: Why do physicians have such an aversion to facemasks? The editorial comment proposes the remedy, advocating shift in culture, guided by responsible leadership and compelling guidelines. To this we can only totally agree. We would also like to add a further question: Shouldn’t all cases of PDPM be subject to epidemiologic evaluation and thorough scrutiny of the hygienic standard in the departments where PDPM has occurred? The polymerase chain reaction is no longer exclusive and expensive, and clarifying the
We should be grateful for the significant contribution offered by Dr. Baer.

Vibeke Moen, M.D.,* Nils Dahlgren, M.D., Ph.D., Lars Irestedt, M.D., Ph.D.*County Hospital, Kalmar, Sweden. vibekem@ltkalmar.se

References
1. Ruppen W, Derry S, McQuay H, Moore RA: Incidence of epidural hemato-
toma, infection, and neurologic injury in obstetric patients with epidural analge-
 sia/anesthesia. ANESTHESIOLOGY 2006; 105:394–9
105:381–93
3. Lee LA, Posner KL, Domino KB, Caplan RA, Cherry FW: Injuries associated 
with regional anesthesia in the 1980s and 1990s: A closed claims analysis. 
ANESTHESIOLOGY 2004; 101:143–52
4. Doblar DD, Schumacher SD: Spontaneous acute thoracic epidural hema-
toma causing paraplegia in a patient with severe preeclampsia in early labor. Int 
5. Yuen TS, Kua JS, Tan IK: Spinal haematoma following epidural anaesthesia 
7. Abramovitz S, Belin V, Thrombocytopenia, low molecular weight heparin, 
8. Paech MJ, Godkin R, Webster S: Complications of obstetric epidural anal-
gesia and anaesthesia: A prospective analysis of 10 995 cases. Int J Obstet Anesth 
1998; 7:5–11
9. Moen V, Dahlgren N, Irestedt L: Severe neurological complications after 
central neuraxial blockades in Sweden 1990–1999. ANESTHESIOLOGY 2004; 101: 
950–9
10. Hipner D: Gloved and masked—will gowns be next? The role of asepsis 
during neuraxial instrumentation. ANESTHESIOLOGY 2006; 105:241–3
Anesth 2005; 14:185–8
12. Cornforth BM, Dalgleish DJ, Bromilow J, Wee M: Use of an information 
leaflet to improve general practitioners’ knowledge of post dural puncture 

(accepted for publication December 8, 2006)

In Reply—I thank Drs. Edsall, Dr. Lambert, and Drs. Moen, Dahl-
gren, and Irestedt for their interest in this controversial topic, and for 
their insightful comments and questions. Their remarks clearly show 
the hurdles to overcome in developing evidence-based guidelines for 
strict aseptic technique during neuraxial instrumentation in anesthesia 
practice.

Dr. Edsall raises a question that is often debated and quite contro-
versial: Is the loss of resistance to saline superior to that to air? 
Although many complications, including pneumocephalus, nerve root 
compression, subcutaneous emphysema, venous air embolism, incom-
plete analgesia, and paresthesias, have been attributed to the loss of 
resistance to air,¹ there is no mention in the anesthesia literature 
regarding air as a source of contamination in the epidural space. I 
would argue that there is a significantly higher chance of contamina-
tion from the large volume of air over the tray than the small amount 
injected with the syringe. The only way to prevent the epidural tray 
from being exposed to air is to do the procedure in a vacuum. 
Furthermore, epidural abscess has only been demonstrated to occur as 
a result of skin bacteria passing through a needle track, contaminated 
syringes, or local anesthetics, or hematogenous spread from another 
source.²

Dr. Edsall correctly points out that there are many reports of spotty or 
incorporate blocks when using air instead of saline.³,⁴ Belin et al.⁴ 
demonstrated that more parturients in the air group had incomplete 
analgesia and higher visual analog pain scores requiring additional local 
anesthetic when compared with those in the saline group. A survey in 
the United Kingdom demonstrated that use of loss of resistance to 
saline has progressively increased among obstetric anesthesiologists 
from just over 50% in 1998 to 74% in 2003.⁵ Interestingly, another 
United Kingdom survey demonstrated that although almost 60% of 
respondents first learned to identify the epidural space with loss of 
resistance to air, nearly a quarter have changed to loss of resistance to 
saline.⁶ I must admit that I, trained using intermittent loss of resistance 
to air, have been reluctant to change to saline for fear of having 
increased complications. However, given the abundance of data sup-
porting the use of the loss of resistance to saline and the lack of 
complications when switching from one technique to the other,¹ I am 
strongly considering a change in my practice.

Dr. Lambert seems to have misinterpreted my comments and, fur-
thermore, feels that a 1:10,000 risk of post–dural puncture meningitis 
is acceptable. As noted in my editorial⁷ and the comments of Drs. 
Moen, Dahlgren, and Irestedt, Baer’s statistics suggest that many cases of 
post–dural puncture meningitis are unreported and unrecognized, 
suggesting that the actual US rate is higher than reported.⁸ In addition, 
as discussed by Dr. Moen in his letter and eloquently stated by Dr. 
Reynolds,⁹ risk varies with both clinical practice and patient popu-
lation. Causes of complications are multifactorial and include the per-
f ormance of neuraxial techniques by trainees who may not be able to
There are two points that, although not related to any of these letters, I would like to raise. The first one has to do with a typographical error in my editorial related to the American Society of Anesthesiologists Task Force on Infection Control. I inadvertently wrote “maximal sterile barrier precautions during central venous catheter infection” rather than “central venous catheter insertion.” The second point has to do with the recently published guidelines for aseptic techniques during regional anesthesia. I wrote that although a chlorhexidine solution has a faster and stronger bactericidal effect than povidone iodine, the consensus stopped short of recommending an alcohol-based chlorhexidine antiseptic solution for skin disinfection before neuraxial techniques (electronic personal communication, Joseph M. Neal, M.D., Staff Anesthesiologist, Virginia Mason Medical Center, Seattle, Washington, and Editor-in-Chief, Regional Anesthesia and Pain Medicine, April 2006). However, it seems that in the final revision of the guidelines, the decision was made to encourage the use of an alcohol-based chlorhexidine solution as the antiseptic of choice before regional techniques. The expert panel felt strongly that although the US Food and Drug Administration has not approved chlorhexidine before lumbar puncture, it has a significant advantage over povidone iodine because of its onset, efficacy, and potency (verbal personal communication, James R. Hebl, M.D., Assistant Professor, Department of Anesthesiology, Mayo Clinic, Rochester, Minnesota, November 2006). Upon contacting the Food and Drug Administration, the panel found that the lack of approval was not because of toxicity but because of lack of scientific data. Interestingly, povidone iodine is also not approved for lumbar puncture. Finally, it is important to mention that the guidelines conclude that there are insufficient data regarding the routine use of surgical gowns before performing a regional technique.

David L. Hepner, M.D., Harvard Medical School, Brigham and Women’s Hospital, Boston, Massachusetts. dhepner@partners.org

References


(Accepted for publication December 8, 2006.)
In Reply:—I welcome the letters in response to two articles1,2 and the editorial3 in the August 2006 issue of ANESTHESIOLOGY regarding aspects of infectious complications of neuraxial instrumentation.

van de Beek and de Gans discuss important information related to the treatment of post–dural puncture meningitis (PDPM). They recommend that ceftazidime or cefepime be substituted for the empiric third-generation cephalosporin recommendation in my review.1 The former drugs have been found to have greater in vitro activity against Pseudomonas aeruginosa. (However, their reference 4 does not mention either P. aeruginosa or cefepime.)

Their admonition against adjunctive steroids in PDPM needs further clarification. Kornik et al.5 (van de Beek and de Gans’ reference 3) studied 6,243 consecutive craniotomies to evaluate the effect of preoperative antibiotic prophylaxis on the incidence of postoperative meningitis. They found 50% reduction in incision infections (skin, bone) but no reduction in meningitis for the group that received prophylaxis. van de Beek and de Gans give as the reason for withholding adjunctive dexamethasone in PDPM, that PDPM resembles postcraniotomy meningitis more than it resembles community-acquired meningitis. Postcraniotomy meningitis is clearly different from community-acquired meningitis. Adjunctive dexamethasone would be inappropriate in the presence of a wound infection. But PDPM may have more in common with the community-acquired type than it has with the neurosurgical type. Should not PDPM be considered as a distinguished subset? Each of the three types of meningitis has a different spectrum of causative organisms, but the high incidence of viridans streptococci in the PDPM group (a primarily upper respiratory, mouth, and skin commensal) makes it more like the community-acquired type (Streptococcus pneumoniae, N. meningitidis) than the neurosurgical type (staphylococci [skin commensals] and enterococci). In addition, the complication of the concomitant surgical wound is absent in PDPM. Therefore, would not the desirable antiinflammatory effect of dexamethasone be the same in postpuncture meningitis as in community-acquired meningitis? (other things being equal, e.g., no other infected foci)?

van de Beek and de Gans’ reference 7,6 covering meningitis due to viridans streptococci, reports seven cases of meningitis due to viridans streptococci—three of which are termed iatrogenic. Two patients had undergone thermoagulation of the gasserian ganglion, and one had endoscopic sclerotherapy for esophageal varices. These three cases suggest meningitis of the post–dural puncture type (albeit inadvertent). It is not clear whether van de Beek’s unpublished cases of meningitis due to viridans streptococci are community-acquired, neurosurgical, or post–dural puncture in type. Two other cases described in van de Beek and de Gans’ reference 7 died of S. milleri meningitis. The underlying cause for these cases of meningitis is not given, but the mechanism of death in each case6 is given as brain herniation accompanied by cerebral edema. Whether adjunctive dexamethasone is appropriate for treatment in puncture-type meningitis needs further study.

I thank Dr. Eads for drawing attention to the practice, by some, of injection of air into the epidural space as a possible source of infection.

Dr. Lambert’s letter is puzzling. Science is the wedding of logic and observation. Dr. Lambert’s readiness to jettison logic is perverse. He is encouraged that statistics show “... that the odds that I will have an infection are exceedingly low.” However, the data reflect incidence expected when the anesthesiologist washes his or her hands. Lambert admits that he has “... not routinely done so...” but he wants credit for sterile gloves and cap. No mention of a mask. May we conclude that he does not “routinely” wear one? He states, “If the neuroaxial anesthesia infection rates were the same as for [central venous catheter] insertions, no one would argue against the use of neuroaxial full-barrier precautions.” (Isn’t it logical to surmise that if all anesthesiologists were as cavalier about sterile technique, the infection incidence might approach that of inserting a central venous catheter?) He then implies that because the incidence rate of central venous catheter infection is much higher than that for dural puncture, that makes applying the central venous catheter precautions “illogical” because a 1:10,000 infection rate is a “nonproblem.” (It is a serious problem for that unlucky 1 in 10,000.) He asks, “... what risk is acceptable?” The answer is that (in the case of prevention of PDPM) the acceptable risk is the lowest possible under conditions of meticulous attention to sterile technique. The idea that there must be a cost–benefit analysis for every intervention, no matter how trivial, is impractical. Why does Dr. Lambert wear a cap and gloves while invading a patient’s nervous system? Where are the data, and where is the logic?

Drs. Moen, Dahlgren, and Irestedt point out an error in the article text,1 stating that in their series, 28 cases (instead of 11 cases) of PDPM were culture positive for a-hemolytic streptococci. (Table 1 and Table 4 have the correct value.) They ask, “Why do physicians have such an aversion to facemasks?” (Perhaps the situation is analogous to the reaction to automobile seat belts and bike helmets when they were first introduced.) They opine that the unnecessary deaths could reflect, among other things, “ ... lower diagnostic preparedness.” Not only does this serious complication often go unrecognized, but in several of the case reports, the existence of PDPM is denied and attributed to other causes.

The absence of inclusion of PDPM in the US Practice Guidelines7 for the management of bacterial meningitis is inexplicable, as is the Centers for Disease Control and Prevention’s exclusion of PDPM as a nosocomial disease. Perhaps forums such as this one will reduce the incidence, as well as heighten awareness, of this preventable disease.

Estelle Traurig Baer, M.D., retired. Previous affiliation: Department of Medicine, Kaiser/Permanente Medical Center, Richmond, California. etbaer@comcast.net

References

2. Ruppen W, Derry S, McQuay DM, Moore A: Incidence of epidural hema
toma, infection, and neurologic injury in patients with epidural analge
sia/anesthesia. ANESTHESIOLOGY 2006; 105:394–9

David C. Warltier, M.D., Ph.D., served as Handling Editor for this exchange. Dr. Ruppen was asked to provide a reply to the two letters regarding his Review Article but did not feel that a response was required.
To the Editor—The article by Heller et al.1 was informative and adds precision to the effect of temperature on the baricity of local anesthetics. They provide interesting new data on the temperature at which local anesthetics used for spinal anesthesia are isobaric (the “isobaric temperature”). Clearly, the next question is, what is the clinical relevance of this added precision? The authors themselves state, “Whether this concept in fact improves patient safety in terms of hemodynamic stability or even allows dose reductions of local anesthetics must be confirmed in further clinical studies.”

In 1989, Beardsworth and I published a simple study comparing the injection of 5 ml plain 0.5% bupivacaine at room temperature to an identical solution adjusted to 37°C (very close to but not precisely within the limits [34.3°–35.8°C] of the so-called isobaric temperature).2 The injection was performed with the patients in the lateral decubitus position, and they were then immediately turned to the supine horizontal position. For the same reasons indicated by Heller et al., we hypothesized that increasing the temperature of the bupivacaine would make it more isobaric and limit its spread. We found no difference in the extent of pinprick analgesia. However, the 37°C solution produced a more prolonged block, which we suggested was due to a decrease in pKa associated with the increased temperature.

Beardsworth’s study compared but one dose of bupivacaine and one position after its injection. Other doses and patient positions will likely produce different results.

Heller’s and Beardsworth’s studies beg the question as to whether it is possible (with the exception of using a very hyperbaric solution for saddle or thoracic levels of block) to precisely control the level of spinal anesthesia. Although the temperature effect on the baricity of local anesthetics used for spinal anesthesia reported by Heller has achieved this pinnacle of precision, this effect will have to overcome the manifold factors that control the level of spinal anesthesia3 to significantly impact clinical practice. Whether this can be accomplished will only be determined through clinical trials that will undoubtedly derive from Heller’s publication. However, after 25 yr of studying, practicing, and watching spinal anesthesia, I suspect that the precise control of the level of spinal anesthesia will require more than simply adjusting the temperature of the injected local anesthetic.

Donald H. Lambert, Ph.D., M.D., Boston University Medical School, Boston, Massachusetts. donlam@fastmail.us or donald.lambert@bmc.org

References
2. Beardsworth D, Lambert DH. Warming 0.5% bupivacaine to 37°C increases duration of spinal anesthesia. Reg Anesth 1989; 14:199–202
(Accepted for publication December 19, 2006.)
injected at 37°C⁰ are comparatively effective for 2 min and may account for earlier onset of blockade but not, however, for the prolonged LA effects more than 2 h later.

Taken together, the discussed effects carry uncertainty for daily practice but may, besides others, explain the high interindividual ranges in maximum level of sensory blockade reported in many studies using “isobaric” solutions. In vitro studies and modeling as performed in our work⁴ always observe and depict a limited part of reality. They never allow conclusions on the reality itself; rather, they may be hypothesis generating or may improve existing hypotheses, which then must be verified (or falsified) in reality. The problems associated with the complex physiology of subarachnoid block may not be solved with simple physics. The intention of our study was to identify isobaric temperatures and, thus, make the course of LA within the subarachnoid space more predictable to improve the nonprecise anesthetic.

Axel R. Heller, M.D.,* Thomas Roessels, M.D., Rainer J. Litz, M.D. *University Hospital Carl Gustav Carus, Dresden, Germany. axel.heller@uniklinikum-dresden.de

References


Use of Vasopressin Bolus and Infusion to Treat Catecholamine-resistant Hypotension during Pheochromocytoma Resection

To the Editor—In a recent review of vasopressin, it was stated that there were only two reported cases using bolus vasopressin (10–20 U) to restore blood pressure after pheochromocytoma resection.⁵ This letter documents another rare case, using a lower dose of bolus vasopressin, for treatment of catecholamine-resistant hypotension after pheochromocytoma resection.

A 54-yr-old man (height, 180 cm; weight, 84 kg) underwent laparoscopic right adrenalectomy for treatment of pheochromocytoma. Preoperative medications included phenoxybenzamine, metoprolol, ramipril, and atorvastatin. Preinduction blood pressure was 129/74 mmHg, and heart rate was 57 beats/min. During manipulation of the adrenal gland, the patient developed hypertension, which was treated with sodium nitroprusside (up to 10 ml/h of 200 µg/ml) and esmolol (up to 5 ml/h of 10 mg/ml) infusions. After resection and discontinuation of the nitroprusside and esmolol, the patient developed hypotension. A norepinephrine infusion of 24 µg/min was only able to increase the patient’s systolic blood pressure to the low to mid 80s. Two 0.4-U vasopressin boluses were administered, which increased the systolic blood pressure to 120 mmHg. The patient was started on a 4 µg/h infusion of vasopressin resulting in maintenance of a systolic blood pressure at 110 mmHg while permitting a decrease in the norepinephrine infusion rate. Both infusions were continued throughout the rest of the surgery and were weaned several hours postoperatively. The patient did well and was discharged home on postoperative day 2.

There are now three previous reports of bolus vasopressin being used to treat hypotension after adrenal resection for pheochromocytoma.⁵-⁷ Repeated bolus doses of 10–20 U followed by an infusion were required to treat hypotension after pheochromocytoma resection.⁵ In another adult patient, an infusion of vasopressin required 20 min to achieve improvement in blood pressure during pheochromocytoma resection complicated by a large blood loss.⁷ In an 11-yr-old patient, a 5-U bolus followed by an infusion was successful in treating postresection hypotension.⁸

Although vasopressin infusions have been used in a variety of other situations, there are limited data to guide bolus dosing. Others report lower doses of vasopressin bolus. A 2-U bolus dose was used to treat anaphylactic shock.⁹ As in this case, two 0.4-U boluses successfully treated hypotension secondary to both bowel retraction in patients having abdominal aortic resection repair and postreperfusion syndrome during liver transplantation.⁶-⁷ Terlipressin (a vasopressin precursor) in doses of 1 or 2 mg successfully treated hypotension secondary to induction of anesthesia in patients chronically treated with renin-angiotensin system inhibitors.⁹ Additional well-controlled studies must be conducted to establish the indications, safety, and efficacy of bolus vasopressin for rapid correction of hypotension, particularly catecholamine-resistant hypotension.

This patient adds to the small number of reported cases that suggest vasopressin can be safely and effectively used to treat postadrenalectomy catecholamine-resistant hypotension in patients with pheochromocytoma. Although vasopressin is a vasoconstrictor and it is not surprising that it can treat hypotension, there may be a specific need for vasopressin in some pheochromocytoma patients. First, vasopressin is effective in the presence of residual α-adrenergic blockade and down-regulation of those receptors. Second, in some patients with pheochromocytoma, there is an oversecretion of vasopressin, which may contribute to the induced hypotension.⁸,¹⁰ It is possible that chronic oversecretion of vasopressin leads to a down-regulation of vasopressin receptors, thus contributing to the postresection hypotension. Whether preoperative determination of vasopressin over secretion will affect management is an open question. Catecholamines are also known to inhibit vasopressin release.¹¹ It has been proposed that chronic inhibition by catecholamines may down-regulate neurophysiological vasopressin synthesis, thereby preventing the acute high release of vasopressin during a hypotensive episode. Last, the use of
vasopressin can reduce the catecholamine dose, thus allowing one to avoid their undesirable side effects such as increased myocardial oxygen consumption and ventricular arrhythmias.

Jonathan V. Roth, M.D., Albert Einstein Medical Center, Philadelphia, Pennsylvania. rothj@einstein.edu

References

7. Roth JV: The use of vasopressin bolus to treat refractory hypotension secondary to reperfusion during orthotopic liver transplantation. Anesth Analg 2006; 103:261

(Accepted for publication December 19, 2006.)

Impact of Terlipressin on Hepatosplanchnic Perfusion: “Only the Dose Makes a Thing Not a Poison” (Paracelsus)

To the Editor—With great interest, we read the comprehensive and well-written review article of Drs. Treschan and Peters1 providing a thorough overview of the physiology and therapeutic indications of arginine vasopressin and its synthetic analogs. Nevertheless, we believe that the impact of the long-acting vasopressin analog terlipressin on hepatosplanchnic perfusion in the treatment of sepsis-related arterial hypotension has not been discussed appropriately. The authors conclude that “terlipressin is a potent intestinal vasoconstrictor, and evidence suggests decreased intestinal perfusion with terlipressin infusion.”1 First, we wish to rectify that the cited study of Westphal et al.2 reported that the effects of arginine vasopressin on gut mucosal microcirculation in septic rats, and not of terlipressin (as wrongly cited by Drs. Treschan and Peters)2. Second, the effects of terlipressin on splanchnic perfusion are dependent on two important aspects, which the authors of the current review article did not refer to: (1) the role of aggressive fluid resuscitation and (2) the dose itself. In this context, Asfar et al.3 reported that terlipressin even improved ileal microcirculation in fluid-challenged endotoxic rats. In contrast, in non-fluid-challenged rats, terlipressin infusion contributed to detrimental effects within the intestinal microcirculation and microcirculation. In addition, the impact of terlipressin on intestinal perfusion seems to be dependent on the drug dosage and application form. Again, Asfar et al.4 demonstrated that a goal-directed continuous low-dose infusion of terlipressin not only reversed the hypotensive–hyperdynamic circulation in porcine endotoxemia but also decreased global systemic oxygen consumption without compromising splanchnic metabolism and organ function. In summary, the current literature, also limited in extent, supports the view that low-dose terlipressin, in conjunction with aggressive fluid challenge, is a promising adjunct in our therapeutic repertoire for the treatment of systemic arterial hypotension resulting from distributive shock.5

Matthias Lange, M.D.,* Hugo Van Aken, M.D., Ph.D., Martin Westphal, M.D., Ph.D. *University of Muenster, Muenster, Germany. lange-m@anit.uni-muenster.de

References


(Accepted for publication December 19, 2006.)

In Reply.—We appreciate the comments of Dr. Roth regarding the use of vasopressin to restore blood pressure after pheochromocytoma resection. This case certainly adds an interesting experience to the few published cases discussed in our recent review.1

The use of a very small vasopressin bolus dose (0.4 U) is a reasonable approach to evaluate the patient’s reaction toward the drug. Dr. Roth used a vasopressin infusion (4 U/h ~ 0.07 U/min) for blood pressure maintenance. This dose is consistent with the recommendations for continuous vasopressin infusion when used as an adjunct vasopressor in septic shock (1–4 U/h ~ 0.01–0.07 U/min).2

Use of vasopressin during pheochromocytoma resection has been described in a few patients with very different preoperative conditions (well-controlled blood pressure vs. hypertensive spells3–4), treated with different anesthetic regimens (intraoperative use of thoracic epidural vs. general anesthesia only5–4), and intraoperative complications (e.g., severe blood loss6). Therefore, data are not comparable, and it is much too early to provide treatment recommendations. Undoubtedly, the

David C. Warltier, M.D., Ph.D., served as Handling Editor for this exchange.

Anesthesiology 2007; 106:884–5

Copyright © 2007, the American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

Anesthesiology 2007; 106:884

Copyright © 2007, the American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

Copyright © 2007, the American Society of Anesthesiologists. Unauthorized reproduction of this article is prohibited.
role of the vasopressin system is of greatest interest in patients with pheochromocytoma and stresses the importance of the vasopressin system as an important backup system for blood pressure control.1 We agree with Dr. Roth that some patients may have a specific need for treatment with exogenous vasopressin, due to down-regulation and/or preoperative pharmacologic blockade of adrenoceptors, down-regulation of vasopressin receptors, and/or “inadequately low” vasopressin release during acute hypotension. Further studies are needed to address these issues.

We thank Drs. Lange, van Aken, and Westphal for their comments on the impact of the long-acting vasopressin analog terlipressin on hepato-splanchnic perfusion. Westphal et al.8 themselves summarized similar results as “data suggest that, in sepsis, vasopressin and terlipressin infusion may decrease gastrointestinal mucosal blood flow.” In our recent review,1 we concluded that “terlipressin is a potent intestinal vasoconstrictor, and evidence suggests decreased intestinal perfusion with terlipressin infusion.”

We agree with Dr. Lange et al. that this conclusion can be discussed further by taking into account also the role of fluid management and the dose itself. Asfar et al.9 used studies in animals to investigate the influence of terlipressin on splanchnic perfusion, and results obtained in pigs show that a continuous low-dose infusion of terlipressin (5–15 μg·kg⁻¹·h⁻¹) does not have detrimental effects on hepatosplanchnic perfusion, oxygen exchange, and metabolism. However, to our knowledge, this regimen has not been used in clinical trials in humans. Rather, terlipressin is usually administered in intravenous boluses of 1–2 mg. Bolus doses have been used by Asfar et al.10 in their study on endotrophic rats. The bolus of 6 μg/kg used in this study, however, is only 25–50% of the dose reported to be used in humans, and even with this low bolus, Asfar et al. found a high mortality in endotrophic animals treated with terlipressin alone. Only when terlipressin was administered after adequate fluid resuscitation, ileal microcirculation improved, as expected. Therefore, these latter results support the importance of early and aggressive fluid resuscitation in sepsis11 and also warrant further research on dosing of terlipressin in sepsis. However, until these results are available, terlipressin can not be recommended for routine use in septic patients because of its potentially detrimental intestinal vasoconstrictor activities. That is, what dose it takes to make this drug not a poison is unsettled.

Tanja A. Treschan, M.D.,+ Jürgen Peters, M.D. *Universitätsklinikum Essen, Essen, Germany. tanja.astrid.treschan@uk-essen.de

References


(Accepted for publication December 19, 2006.)

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

Anesthesiology 2007; 106:885

Copyright © 2007, the American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

GlideScope® Intubation Assisted by Fiberoptic Scope

To the Editor—The difficult airway continues to challenge anesthesiologists. Recently, the development of laryngoscopes that have video cameras built in has led to some improvement in visualization of airway anatomy. One such device is the GlideScope® (Saturn Biomedical Systems, Burnaby, British Columbia, Canada). It is equipped with a patient antifogging system that, together with a design that tends to keep the camera free of blood and secretions, has made visualization of airway structures better. However, despite better glottic visualization, on some occasions the endotracheal tube may still be difficult to pass into the larynx.

We recently provided general anesthesia to an obese female patient, aged 32 yr, weighing 142 kg, with a Mallampati class 4 airway. The patient had a short neck with a hyomental space of three finger-breadths. We had a control laryngoscope for difficult airways: A manikin study. Eur J Anaesthesiol 2006; 23:1056–61


Support was provided solely from institutional and/or departmental sources.
Optimizing GlideScope® Laryngoscopy: An In Vitro Study on an Airway Model

To the Editor—The GlideScope® Videolaryngoscope1* (GVL; Diagnostic Ultrasound Corporation, Bothell, WA, and Saturn Biomedical Systems Inc., Burnaby, British Columbia, Canada) has a video camera incorporated in the undersurface of its curved plastic blade, providing a detailed airway image on an integrated monitor. The shape of the GVL partially resembles the shape of the standard Macintosh laryngoscope (fig. 1A). However, a 60° upward angulation of the distal half of the blade allows for an easy visualization of the larynx, which is often better than that of a rigid laryngoscopy.2 Despite better laryngoscopic view with the GVL, intubation using a Macintosh laryngoscope was significantly faster.2 In addition, in one series of 728 patients, the intubation failure rate of the GVL was 3.7% despite the ability to obtain grade 1 or 2 views most of the time.3–5 The paradox that the GVL provided better glottic visualization but not easy intubation may have been caused by patient-related factors, insufficient skills of intubators, and/or limitations of the device. Intubation with the GVL is limited by a sole reliance on the video image, because a line-of-sight view is nonexistent. We wondered whether this special design of the GVL might actually hinder accomplishment of intubation as a central purpose of the device.

Three steps are necessary for a successful intubation with the GVL: laryngoscope insertion and glottic visualization, delivery of the stylet-oxid endotracheal tube (s-ETT) in front of the GVL camera, and guidance of the disengaged endotracheal tube (d-ETT) through the glottis and into the trachea. As described in the operator’s manual, the GVL is inserted down the midline of the tongue and can be used as either a curved blade (Macintosh style) or a straight blade (Miller style). We noticed that some novice intubators actually lever the laryngoscope and halt its advancement as soon as they obtain a glottic view, which can ensue before the tip of the laryngoscope reaches the vallecula. Others insert the GVL too quickly and too deeply and obtain either a close glottic view or an esophageal view. Retracting the GVL from the esophagus provides a close glottic view. The operators’ manual states, “maximum laryngeal exposure may not facilitate intubation.”1 This is contrary to usual attempts made by intubators to obtain the best laryngoscopic view possible. We postulated that such attempts might actually render the passage of the ETT more difficult or impossible, and we explored these observations in an airway model.

We used a Laerdal airway anatomical model 252 500 on which to simulate GlideScope® laryngoscopy and tracheal intubation (figs. 1B and C). The GVL was slowly advanced down the midline of the model’s tongue. The advancement was ceased at three locations: At location 1, the blade tip was at the tip of the epiglottis; at location 2, the blade tip was within the vallecula; and at location 3, the laryngoscope was behind (posterior to) the epiglottis (figs. 2A–C, main panels). The initial position of the laryngoscope at each location was adjusted to give a view of only the arytenoids, grade 2b laryngoscopic view (figs. 2A–C, accessory panels in the upper right corner). After performance of tracheal intubation with this initial laryngoscopic view, the GVL position was modified twice at each location. The laryngoscope was lifted first, and the trachea was intubated. The laryngoscope was then levered, with the distal end pointing up, and the trachea was intubated.

Fig. 1. (A) A standard GlideScope® Videolaryngoscope (Diagnostic Ultrasound Corporation, Bothell, WA, and Saturn Biomedical Systems Inc., Burnaby, British Columbia, Canada) with a visible bulge of the camera that is embedded in the distal half of the GlideScope® Videolaryngoscope blade. The tip of the GlideScope® Videolaryngoscope blade extends above the camera axis. (B) View of the oropharynx and the glottis of a plastic Laerdal airway anatomical model 252 500 (Laerdal Medical LTD, Orpington, Kent, United Kingdom). (C) Side view of the larynx and the trachea of the same model.

Support was provided solely from institutional and/or departmental sources. The authors have no financial relation to the manufacturer of the GlideScope® Videolaryngoscope (Diagnostic Ultrasound Corporation, Bothell, Washington). Presented as a poster at the 80th Scientific Congress of the International Anesthesia Research Society, San Francisco, California, March 21–24, 2006, and at the 10th Annual Scientific Meeting of the Society for Airway Management, San Diego, California, September 15–17, 2006.

The same course of action was followed at locations 1, 2 (figs. 3A and B), and 3. We performed the whole process of nine intubations twice: the first time using a recommended 60° stylet angle1 and the second time using a 90° stylet angle.6 A lubricated 5.5-mm s-ETT was used to minimize adhesion between the ETT and the model structures. The ETT was loaded on a malleable stylet with the ETT bevel facing to the left for all intubations. The intubator’s hands maneuvering the laryngoscope and the s-ETT were recorded using a camcorder, while the passage of the d-ETT through the glottis was captured from the GVL video output (figs. 2 and 3, main panels and accessory panels, respectively). These two videos were synchronized and processed using video editing software (Adobe Premiere Pro 1.5; Adobe Systems Inc., San Jose, CA). Two compiled videos were then reviewed simultaneously.

Because of softness of the tongue, lifting the laryngoscope at location 1 misdirected the camera axis from the arytenoids toward the vallecula, shifting the view from grade 2b to grade 3. Gentle lifting of the GVL at locations 2 and 3 enhanced the laryngoscopic view from grade 2b to grade 2a. Levering the GVL pointed the camera axis upward as to provide visualization of the anterior portion of the glottis (fig. 3B). During this phase, the intubator purposefully avoided lifting the laryngoscope to avoid lifting the glottic structures. Extreme levering of the laryngoscope provided a grade 1 view at locations 1 and 2. The position of the camera close up to the glottis prevented visualization of the most anterior glottic portion at location 3. Insertion of the s-ETT and advancement of the d-ETT was easier and quicker at location 2 than at locations 1 and 3. Advancement of the d-ETT was easier and quicker at location 2 than at locations 1 and 3. Advancement of the d-ETT was easier and quicker at location 2 than at locations 1 and 3. Advancement of the d-ETT was easier and quicker at location 2 than at locations 1 and 3. Advancement of the d-ETT was easier and quicker at location 2 than at locations 1 and 3. Advancement of the d-ETT was easier and quicker at location 2 than at locations 1 and 3. Advancement of the d-ETT was easier and quicker at location 2 than at locations 1 and 3. Advancement of the d-ETT was easier and quicker at location 2 than at locations 1 and 3.
time to intubation was shorter using the 60° stylet angle than the 90° stylet angle.

The GVL gently lifted within the vallecula provided a grade 2a laryngeoscopic view and yielded the smoothest and quickest intubation using the 60° stylet angle. Adequate room for delivery of the s-ETT, optimal alignment of the camera axis with the laryngotracheal axis, and decreased impact of the d-ETT tip with the anterior tracheal wall are the most probable explanations (fig. 3A). The Miller-style placement of the GVL resulted in a close glottic view but reduced the space between the GVL camera and the glottis for the s-ETT insertion. We recommend the Miller-style use of the GVL only if the best laryngoscopic view with the Macintosh style is a grade 3 view. Levering the GVL shifts the proximal end of the laryngoscope down and brings more bulk of the GVL blade into the oropharynx. That will severely reduce the space available to pass the ETT, especially if intubators attempt to pass the s-ETT underneath the scope. Levering the GVL also produces upward orientation of the camera portion of the blade such that the d-ETT that is passed alongside the blade easily gets hung up on the anterior commissure or cricoid or tracheal cartilages (fig. 3B). In either case, although the view is improved, accessing the laryngeal inlet and advancing the tube may be more complicated, resulting in failed intubations.

In conclusion, several considerations support our suggestion that maneuvers necessary to achieve a grade 1 laryngoscopic view may render intubation with the GVL more difficult. We believe that a grade 2a view with the blade lifted within the vallecula is preferable for intubation with this device. It provides the adequate space in the oropharynx and in the immediate glottic area for the s-ETT insertion as well as optimal alignment of the camera axis with the laryngotracheal axis for ETT advancement.

The use of an open airway model does not, of course, completely model the challenges of tracheal intubation in patients. However, the use of the open model allowed us to visibly demonstrate both good and bad laryngoscopic maneuvers and their corresponding glottic views. We hope that this letter may bring forth more understanding to GVL users about proper laryngoscopic and intubating maneuvers and may guide them to more intubation success with the device.

Mirsad Dupanovic, M.D.,† Richard Jensen, B.S. †University of Rochester School of Medicine, Rochester, New York.
mirsad_dupanovic@urmc.rochester.edu

The authors thank Peter L. Bailey, M.D. (Professor), and Denham S. Ward, M.D., Ph.D. (Professor and Chair Emeritus), Department of Anesthesiology, University of Rochester, Rochester, New York, for reading this manuscript and providing helpful advice.

References


(Accepted for publication October 31, 2006.)