Anesthesiology 2001; 94:712

Cardiac Surgery Increases Surgical Complexity

To the Editor:—Being from an institution that has experience in port-access heart surgery, I would like to make a few comments regarding a recent article in ANESTHESIOLOGY.[1]

Dr. Chaney *et al.*[1] are to be congratulated for their efforts to provide controlled data regarding the advantages and disadvantages of port-access cardiac surgery. Most of the Discussion section of their article dealt with the longer procedure times, greater technical complexity, uncertain benefits, and potential new morbidity related to port-access *versus* standard operation.

As a matter of perspective, the article of Chaney *et al.*[1] must be interpreted with caution. The results presented are clearly an early experience with port-access at one institution. Recent data from 738 patients undergoing port-access valve surgery at 27 institutions showed that operative times improve with experience even after 200 cases.[2] As the authors admit, their reported initial experience with 46 patients was characterized by the lack of comfort of surgeons, anesthesiologists, and perfusionists that normally would accompany the adoption of new technology. After 400 port-access cases at Duke University, port-access procedures have become sufficiently streamlined that all anesthesia staff, cardiac operating room nursing staff, and perfusionists are quite comfortable with port-access techniques. More complex technologies of fluoroscopy, percutaneous pulmonary artery venting, percutaneous coronary sinus catheters, and centrifugal venous assist have been minimized.

It may be useful to remember the early days of laparoscopic cholecystectomy when similar concerns of increased operating room time, increased complexity, and uncertain outcome caused resistance among physicians for several years. Ultimately, streamlined technology and overwhelming patient demand have made laparoscopic cholecystectomy, but not necessarily other laparoscopic surgeries, dominant procedures today.

The ultimate role for port access will be decided not only by future controlled studies examining more mature technologies, but also by the patients.

Donald D. Glower, M.D.,* **Mark Stafford-Smith, M.D., Fiona M. Clements, M.D.** *Duke University Medical Center, Durham, North Carolina. glowe001@mc.duke.edu

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In Reply:—The comments of Dr. Glower *et al.* are much appreciated. All would agree that operative personnel (surgeons, anesthesiologists, perfusionists, nurses) become more comfortable and efficient with procedures involving new technology with increased exposure and experience. Although normalizing operative time is important from an economic standpoint, I believe that normalizing cardiopulmonary bypass time and demonstrating equivalent intermediate and long-term morbidity and mortality (when compared with conventional cardiac surgery) are the most important issues regarding port-access cardiac surgery. We, as have others, revealed that port-access cardiac surgery significantly increases cardiopulmonary bypass time when compared with similar procedures performed conventionally, which likely increases perioperative morbidity (neurologic, pulmonary, renal, hematologic). How port-access cardiac surgery compares with conventional cardiac surgery in terms of intermediate and long-term morbidity and mortality remains to be determined. I am always uncomfortable with the often-stated comparison of the development of port-access cardiac surgery to the development of laparoscopic cholecystectomy. I do not believe it is a fair comparison because the technical challenges that confront the cardiac surgeon (coronary anastomoses, valve repair and replacement, and others) are far greater than the surgeon's performing a cholecystectomy.

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Patient Outcomes and "Directed" Anesthesia Care

To the Editor:—In "Anesthesiologist Direction and Patient Outcomes," Silber *et al.*[1] described a reduction in risk-adjusted mortality associated with larger hospitals, higher nurse-to-bed ratios, and direction of anesthesia care by an anesthesiologist. Focusing on the effects of "directed" care, they observed that after adjustment for a variety of patient and institutional characteristics, patients for whom anesthesiologists submitted bills for performing or directing anesthesia care (the "directed" group) had fewer deaths than patients receiving "undirected" care, 61 percent of whom were not billed for anesthesia. This study has attracted considerable attention among physician and nurse anesthetists, public officials, and the public. However, this study neither compares the safety of physician and nurse anesthetists nor provides data that distinguish anesthesia safety from surgical outcomes.

The published study reported 7,665 deaths (3.5%) within 30 days after 217,440 operations in Medicare beneficiaries. This rate is nearly 9,000 times the often-cited anesthesia-related mortality rate of 1:250,000.[2] The study also reported 91,024 complications (41.9%), including psychosis, internal organ damage, gastrointestinal or internal bleeding, sepsis, deep wound infection, gangrene, gastrointestinal obstruction, and return to surgery. Clearly, these extraordinarily high

mortality and complication rates are functions of a "wide net" cast, which includes perioperative events, many of which temporally and clinically are unrelated to anesthesia care.

The 58 excess deaths observed in the "undirected" group have several plausible etiologies, many of which were acknowledged by the authors. Among them are the following: (1) misassignment of unbilled "directed" cases to the "undirected" group on the assumption that financial incentives always result in anesthesiologists' billing for direction; (2) the decision to categorize a case as "undirected" if any "undirected" anesthesia was administered during the hospital stay, even if "directed" anesthesia was administered during the primary operation that resulted in complications and subsequent death; (3) high mortality in cases performed by undirected residents; (4) coding and billing errors; (5) clinical information insufficient for complete risk adjustment; (6) unrecognized differences in institutional support; (7) the effect of patient care unrelated to anesthesia administration; and (8) multicollinearity within the logistic models.[3] Cognizant of the limits of this study, the authors themselves observed the need for an "in-depth, large-scale medical chart review of surgical cases" to "assist in determining the etiology of differences in outcomes."

The study published in ANESTHESIOLOGY contains some interesting speculation about data and analyses that ultimately may prove fatally flawed or that may lead to future productive investigation. However, it remains a work hampered by the amorphous nature of the "undirected" group, the methodologic issues noted, and the enumeration of surgical outcomes unrelated to anesthesia administration.

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Medical Direction during Anesthesia: What or Who is the Problem?

To the Editor:—The first casualty of any war is truth. After many months and hearing through secondary sources and vested interests what the Silber *et al.*[1] study did or did not find, it is published in full, and anesthesiologists are free to distinguish truth from fiction for themselves. Silber *et al.*[1] conclude that with medical direction of certified registered nurse anesthetists (CRNAs) by anesthesiologists *versus* nonanesthesiologist direction of CRNAs, there are 2.5 excess deaths per 1,000 patients and 6.9 excess failures to rescue (deaths) per 1,000 patients. However, by the nature of its design, this study could not and does not address the key issue: can CRNAs practice independently? In fact, the negative outcomes in this retrospective study may be related to the medical direction of nonanesthesiologists and may not be related in any way to the practice of CRNAs *per se*. This is not as far fetched as one at first may think. Many surgeons, after many years of long, arduous, highly specialized training, are far removed from physiologic concepts central to anesthetic and supportive care in general. Who amongst us has not heard a surgical colleague express a forceful opinion regarding anesthesia practice that clearly violates the standards of contemporary anesthetic care? I also suspect that when supervising a CRNA, many surgeons still believe in the concept of "surgeon as captain of the ship." These factors could lead to injudicious directives on the part of the surgeon to the CRNA during critical events. Therefore, the negative outcomes as reported by Silber *et al.*[1] might be attributed wholly to the supervisor (surgeon) rather than the person being supervised (CRNA). The hypothesis that positive outcomes (or at least neutral outcomes) might result from the independent practice of CRNAs is in no way ruled out. Silber *et al.*[1] freely admit as much when they state, "Future work will be needed to determine whether the mortality differences in this report were caused by differences in the quality of direction amongst providers, the *presence or absences of direction itself* [italics added], or a combination of these effects."[1] That is why the proposed national study of comparative anesthesia outcomes is critically important.

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Measuring the Influence of Anesthesiologists' Medical Direction

To the Editor:—Despite an Herculean analysis, Silber *et al.*[1] are challenged when exploring the influence of the anesthesiologist's medical direction on clinical outcome of surgical anesthesia. After adjusting for patient and hospital characteristics, they found "statistically significant" relations between absence of medical direction and both death and failure to rescue, but significance testing and related *P* values convey nothing about the magnitude of an intervention's effect or clinical importance. Indeed, small differences of little clinical importance can be found to be statistically significant with very large study populations because *P* values are sensitive to sample size. More indicative of the importance of a factor in such a logistic regression analysis are the odds ratios. However, with odds ratios of 1.08 and 1.10, respectively, the influence of absence of medical direction seems very small, at least for an unselected patient population. By comparison, the odds ratios for relations between customary health risks and specific outcomes (*e.g.*, cigarette smoking and lung cancer, asbestos exposure and mesothelioma, chronic alcoholism and hepatic cirrhosis) are in the range of 5 to 20, making inferences about the importance of these risk factors relatively easy.

Trying to extract greater meaning from their analyses, Silber *et al.*[1] note that absence of medical direction corresponds to 2.5 excess deaths per 1,000 anesthetic procedures. Given that there are an infinite

Table 1. The Benefits of Selected Medical Interventions

Intervention	Events to Be Prevented	Number Needed to Treat
Coronary bypass graft for left main coronary artery occlusion[3]	Death	6
Magnesium sulfate (*vs.* diazepam) for eclampsia[4]	Recurrent seizure	7
Antihypertensive medication for diastolic BP 115–129 mmHg[5]	Myocardial infarction, stroke, death	9
Antenatal corticosteroids for preterm babies[6]	Respiratory distress syndrome	11
Aspirin for transient ischemic attacks[7]	Stroke, death	14
Isoniazid for inactive tuberculosis[8]	Active tuberculosis	96
Antihypertensive medication for diastolic BP 90–109 mmHg[9]	Myocardial infarction, stroke, death	128
Medical direction by anesthesiologist[1]	**Anesthesia-related death**	**400**
Breast exam plus mammography in women aged 50–69 yr[10]	Death from breast cancer	1,075

BP = blood pressure.

number of "excess deaths" that might be avoided generally in health care with different interventions, one might wonder how the benefit of medical direction compares with those of other health care interventions. A handy way to compare interventions involves estimating the number of patients needed to be treated to prevent one negative outcome,[2] which in the Silber example would be 400 (*i.e.*, 1,000 anesthetic procedures per 2.5 deaths). Compared with many other interventions (table 1), medical direction is much less effective but still within the spectrum of interventions that are widely thought to be beneficial.

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In Reply:—Dr. Obst argues that the death rate noted in our study[1] is far higher than 1:250,000 or the 0.0004% rate[2] for "anesthesia mortality" and therefore suggests a flaw in our analysis. Dr. Obst compares the overall 3.5% 30-day mortality rate after general and orthopedic surgery reported in our study (and other studies[3]) with the quoted anesthesia mortality rate to suggest our results are inconsistent by an astounding 9,000-fold ratio. However, results from our model attribute the lack of direction from an anesthesiologist to be associated with an excess of 2.5 deaths per thousand admissions (0.25%), not 35 deaths per thousand (3.5%). Using the 3.5% figure would imply that all deaths after surgery are caused by anesthesia practice. Although we believe anesthesia is critical to patient outcome, we have never contended it was solely responsible for all deaths after surgery. Using 2.5 excess deaths per 1,000 (0.25%) and the Eichhorn[4] anesthesia mortality estimate of 1:151,400 would suggest a 378-fold increase, as we discussed in our paper. Nevertheless, the question remains: is our study consistent with anesthesia mortality as reported in other studies? We believe it is consistent for five reasons. First, comparing anesthesia mortality to excess deaths associated with lack of direction as reported in our study represents a comparison of different quantities. The anesthesia mortality rate is a figure used to track and compare immediate and clearcut anesthesia-related deaths only. The measure was intended to be highly specific for anesthesia events but makes no claims regarding sensitivity. In contrast, 30-day mortality is intended to reflect the full impact of differences in anesthesia practice. This distinction is obvious to any clinician providing anesthesia care to an elderly patient—a risk of only 1 in 150,000 is certainly negligible, but no clinician would dismiss the risks of anesthesia in the elderly as negligible. Clearly, counting only clearcut anesthesia-related deaths underestimates the full risks of anesthesia. If better anesthetic care can reduce deaths, it is valuable, even if when using claims data, patient by patient, it is not always possible to say this one action caused that one death. Smoking causes large numbers of deaths from cardiac disease, but, among smokers, we cannot tell which particular deaths were caused by smoking and which were caused by something else. Nonetheless, we advise people to quit smoking, confident that quitting will reduce their risk of death from cardiac disease. In the same way, our study reports the significant association between lack of direction by the anesthesiologist and death, suggesting that anesthesiologist direction reduces the risk of death. Second, anesthesia mortality as used by Dr. Obst is not a risk-adjusted statistic, unlike the results of our report. Rates of mortality may be orders of magnitude lower for young patients with easier case mix than older patients with more difficult case mix. Third, anesthesia mortality is an inferior outcome statistic because it is susceptible to bias related to the ability of caregivers to temporarily prevent "deaths on the table" that are followed by subsequent death in the hospital or even after discharge from the hospital. It is precisely to correct for the classic "discharged quicker and sicker" bias[5] that the 30-days-from-admission figure has been used by health services researchers. Counting only "deaths on the table" or clearcut immediate anesthesia mishaps would drastically undercount anesthesia-related deaths, reducing sensitivity, and would be highly susceptible to bias across caregiver

abilities to temporarily prolong life for hours or days—an undesirable feature of any outcome measure. Thirty-day mortality, for the reasons noted, is the gold standard measure of quality used in almost all studies of provider quality. Had we not used 30-day mortality, we would have been criticized for using an insensitive measure susceptible to bias. After adequate adjustment for relevant patient covariates and hospital characteristics, differences noted in the 30-day mortality between directed and undirected cases suggest differences in the quality of care provided by anesthesia providers. Fourth, anesthesia mortality reflects practice that generally involves direction by an anesthesiologist, so that low estimates are in part a result of medical direction. Fifth, as we discussed in our report, there are many newer studies that suggest that anesthesia practice influences patient outcomes far beyond the immediate perioperative period, again suggesting that anesthesia mortality rates as cited by Obst underestimate anesthesia mortality.

Dr. Obst asserts that our definition of direction was flawed, without providing any evidence of bias. We defined direction to determine whether the presence of an anesthesiologist benefited the patient. There can be little doubt that using our definition of direction, undirected cases had vastly greater odds of having an undirected anesthetist involved with patient care than did cases defined as directed. This was a very large study, based on claims records of 194,430 directed and 23,010 undirected cases. Although we did acknowledge in our report that the potential for occasional billing misclassification is present in a study of this size, and we agree that a chart review study is the next logical step in this research, we also provided evidence that our estimates were not biased. For example, results were unchanged when restricted only to those cases with bills. Furthermore, to the extent that there was misclassification, such an effect may blur the distinction between provider groups, tending to underestimate the difference in mortality between the directed and undirected groups.

Could undirected resident cases have accounted for our findings? The evidence we reported points to the contrary. First, as was reported, the vast majority of resident cases were counted in the directed group, with at most only 5.6% of undirected cases possibly involving a resident— under the strong assumption that all "no-bill" cases at programs with residents were resident cases. Clearly, the actual percent of resident cases in the undirected group was far smaller than 5.6%. Second, as we stated in our report, when we estimated our results using only the cases with anesthesia bills (which had no resident cases in the undirected group but did have resident cases in the directed group), our results were unchanged. If there were any bias in this study resulting from resident performance, it would be that the difference between directed and undirected cases was underestimated.

Could the difference in results be caused by institutional differences or differences in postoperative care, not differences in directed or undirected anesthetic care? As we reported in numerous analyses, when we adjusted for each hospital individually in the modeling, we found our results to be unchanged. If differences in postoperative care were the cause of our observed differences in outcome, one must hypothesize that undirected patients somehow were sent to different recovery rooms, intensive care units, and surgical floors inside the same hospital than were their directed counterparts. Because these were all Medicare patients, this hypothesis seems implausible. The evidence of stable results after adjustment for individual hospitals supports the conclusion that the differences observed in our study were not caused by differences across institutional postoperative care.

Was inadequate risk adjustment the cause for the observed differences? Much of our paper was related to that question. Extensive risk adjustments, using Medicare data, were performed first. We then appended a well-recognized and validated physiologic-based admission severity score[6] available by law for Pennsylvania hospital admissions and found our results to be unchanged. Furthermore, we saw that failure to rescue, a measure less influenced by errors in severity assessment,[7,8] revealed equally concerning results. There was no evidence that inadequate risk adjustment was responsible for these results.

Were our results the result of multicollinearity within the logistic regression models? Dr. Obst is confused about multicollinearity. Multicollinearity among observed variables may explain why an important coefficient failed to reach statistical significance. It does not explain away a statistically significant coefficient, such as the one in our study.

Dr. Kleinman suggests that the results of our study "may not be related in any way to the practice of CRNAs *per se*" and that the results may be due to medical direction by nonanesthesiologists. Our study clearly suggests that practice situations that include a directing anesthesiologist have lower mortality than situations that lack direction by an anesthesiologist. Undirected cases lacked evidence of direction and only rarely were labeled undirected because a nonanesthesiologist, such as a pathologist or an internist, directed their care. Dr. Kleinman apparently believes that when anesthesiologists are not present, ill-informed surgeons force anesthetists into making bad decisions. We cannot determine this from our study. What we do know is that the presence of an anesthesiologist was associated with lower mortality.

Dr. Orkin suggests that 1 excess death in 400, the difference between undirected and directed care found in our study, is important when compared with other medical interventions. We agree, but we believe Dr. Orkin's analysis understates the importance of our findings by failing to account for the large numbers of potential cases affected by each intervention. Anesthesia practice influences the care of millions of patients annually. Hence, the odds ratio of 1.08 is very important when the potential number of exposed patients is so large.

Our analysis raises concerns about anesthesia care that lacks direction by an anesthesiologist. Future research, through the use of more detailed chart review studies, should explore why this difference in outcomes exists. Clearly, decisions based on evidence and data, rather than opinion and speculation, would serve the community well when making future staffing decisions.

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Abnormal Serum Sodium and Chloride Concentrations May Contribute to Cognitive Dysfunction during Severe Isovolemic Anemia

To the Editor:—I read the article by Dr. Weiskopf *et al.*[1] with great interest. Their published data lead one to the conclusion that severe anemia adversely affects cognitive function. However, I propose that concurrent changes in serum sodium or chloride concentrations possibly could have contributed to this change in cognitive function.

During a volunteer study of serum osmolality,[2] we made the unexpected observation of vague subjective decreases of cognitive function after infusions of 0.9% sodium chloride but not lactated Ringer's solution. We did not quantify the cognitive dysfunction because it only became apparent after we reviewed the comments of the volunteers who were blinded to the nature of the intravenous infusion.

In the study of Dr. Weiskopf *et al.*,[1] 5% human albumin (which is dissolved in 0.9% sodium chloride) was administered in addition to the subject's plasma to maintain isovolemia. Therefore, the subjects could have had increased serum chloride or sodium concentrations when they were severely anemic and demonstrating cognitive dysfunction. It would be interesting to know the values of these serum electrolyte concentrations during their study and whether there was a correlation with cognitive function change.

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In Reply:—We thank Dr. Williams for her interest in our report of subtle, reversible cognitive function and memory changes at hemoglobin concentrations of 6 and 5 g/dl.[1] Dr. Williams *et al.*[2] reported anecdotally reported perceived difficulty in abstract thinking in 13 of 18 volunteers to whom 50 ml/kg NaCl, 0.9%, was administered and in none to whom a similar volume of lactated Ringer's solution was administered. Dr. Williams suggests that their subjective observation might have been caused by a change in sodium or chloride concentrations. However, those values were not outside the normal range, and they did not change significantly in either group, although the difference before and after infusion for sodium concentration differed between the two groups (NaCl, 1 ± 2 mEq/l *vs.* lactated Ringer's solution, −1 ± 2 mEq/l).[2]

We did not measure serum sodium or chloride concentrations in the subjects in whom we studied cognitive function.[1] However, we did measure sodium concentrations in a group of 28 similar subjects in whom we acutely decreased hemoglobin concentration using similar methodology. When hemoglobin concentration was decreased from 12.7 ± 1.0 g/dl to 7.1 ± 0.3 g/dl, sodium concentration significantly increased ($P < 0.0001$) from 137.6 ± 1.6 to 139.7 ± 1.9 mEq/l. However, we found no changes of cognitive function in our volunteers at a hemoglobin concentration of 7 g/dl. In contrast, with further decrease of the hemoglobin concentration to 5.7 ± 0.3 g/dl, a value within the range in which we detected changes in cognitive function, sodium concentration did not significantly change further (140.2 ± 1.6 g/dl; $P = 0.1$).

Thus, our data does not seem to support the thought that some or all of the cognitive function changes we noted at hemoglobin concentrations of 6 and 5 g/dl but not at 7 g/dl were related to alterations of serum sodium concentration.

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The Effect of Lumbar Plexus Block on Blood Loss and Postoperative Pain

To the Editor:—We read the article of Stevens *et al.*[1] with interest and would like to congratulate the authors on their effort in investigating some advantages of regional anesthesia. However, we have some concerns regarding the methodology of the study.

One important outcome investigated by Stevens *et al.*[1] in this trial is to evaluate whether a lumbar plexus block could reduce preoperative and postoperative blood loss. Because this question is crucial and may have important clinical implications, all means to evaluate blood loss as precisely as possible have to be used. Unfortunately, in our opinion, these conditions were not fulfilled in the current trial. We agree that intraoperative estimation of blood loss is difficult. Indeed, we are surprised that the authors only make a rough estimation of 40 ml blood per dressing. First, it is well-known that the amount of blood absorbed by operative swabs is variable, usually from 20 to 150 ml. The minimal condition to ascertain the validity of these results would have been to weigh the swabs before and after their use. Second, in addition to

blood collected in suction canisters and swabs, there is also a considerable portion in the operative surroundings. Unfortunately, this was neither mentioned nor evaluated by the authors.

We also have some concerns regarding the statistical analysis. Looking at the results in detail, great interindividual variation in Visual Analogue Scale scores is evident, which may imply a non-Gaussian distribution. Therefore, we believe that the use of a nonparametric test would have been more accurate for the analysis of Visual Analogue Scale scores. Furthermore, a *post hoc* test for repeated measurements would have been necessary but does not seem to have been performed.

To evaluate the quality of two different postoperative analgesia techniques, Visual Analogue Scale scores have to be compared at certain points during the course of the study. In our opinion, these time points have to be exactly defined and, for this reason, a reference point, the time point zero (t = 0) is necessary. The lack of a time point (t = o) may introduce bias in the interpretation of the results.

There is little information given on exclusion criteria. Were patients with diabetes mellitus, peripheral neuropathy, or steroid intake included? In part, these pathologies may influence the results of the study.

Finally, we were surprised to read that the lumbar plexus block was performed after induction of general anesthesia. At our institution, we perform all nerve blocks with the patient awake or, if necessary, with light sedation. The safety of performing nerve blocks in patients during general anesthesia or heavy sedation has been questioned.[2] The counter argument is that by excluding verbal contact with the patient, the most useful warning of impending nerve contact is lost and that no potential benefit to the patient is worth the risk of serious nerve damage.

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In Reply:—We appreciate the interest expressed by Ekatodramis *et al.* regarding our recent article.[1] The principal objective of our study was to determine whether postoperative pain and analgesic use in the setting of prosthetic hip replacement were influenced significantly by lumbar plexus block. We also assessed a number of secondary endpoints, such as blood loss. At our institution, evaluation of operative blood loss related to orthopedic surgery is a standardized procedure and involves determining the quantity of blood in suction canisters and counting the number of surgical dressings. The volume of blood per dressing depends on the size and type of dressing and on the quantity of blood it has absorbed. With the dressings used for this type of surgery at our hospital, we have observed reproducibly a quantity of blood approximating 40 ml. Using this method of evaluation, we have obtained an excellent correlation with blood losses calculated using preoperative and postoperative hematocrits.

The question regarding the statistical tests used for comparing Visual Analogue Scale scores between groups is legitimate. We assumed that the distribution of results was Gaussian and therefore used the Student *t* and chi-square tests where relevant. However, to test the suggestion of Ekatodramis *et al.*, we reanalyzed the data assuming a nonnormal distribution and performing the pertinent nonparametric tests (Wilcoxon rank sum test or Mann-Whitney U test) and obtained nearly identical *P* values, which confirms our initial assumption of a normal distribution.

We can only agree that it is important to define time points clearly in studies of postoperative outcome. In our article, parameters related to analgesia (Visual Analogue Scale scores and morphine consumption) are expressed consistently as a function of the time elapsed after randomization. In one figure (3A) representing pain scores in the postanesthesia care unit, the time origin was modified deliberately to enhance clarity. This modification is explicit, and we do not believe it introduces significant bias.

The exclusion criteria given in the text summarize contraindications that are known and accepted by most providers of regional anesthesia and therefore were not reported in full. They include major coagulation abnormalities, sepsis at the site of injection, and hypersensitivity to local anesthetics. We did not exclude patients with diabetes mellitus or those with stable peripheral neuropathy.

Finally, the issue of administering nerve blocks to patients undergoing general anesthesia has already been addressed in a previous exchange of letters.[2,3] Although we concur that performing blocks in individuals who are awake is an intuitively sound anesthetic practice, several large-scale studies have not shown increased risk of nerve injury when the procedures are performed in anesthetized patients.[4,5]

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Rapid Extraction of Middle-latency Auditory-evoked Potentials

To the Editor:—In a recent article, Iselin-Chaves *et al.*[1] compared the prediction probability of two methods, *i.e.*, bispectral analysis of the electroencephalograph (BIS®) and peak amplitudes and latencies of middle-latency auditory-evoked potential (AEP). The authors list four inconveniences, which, in their opinion, may limit the use of AEP in clinical practice.

1. AEPs need considerable time to produce a response.
2. Difficulties exist in interpreting the waveforms and on-line measurement of Pa and Nb latencies.
3. Difficulties exist in obtaining and recording AEP.
4. An AEP needs auditory stimuli and, therefore, is applicable only to patients with preserved hearing.

Any anesthesia monitor requires a certain time to process its measured data. Therefore, a number presented on the screen will never correspond to the most recent data acquired. We define the difference between data acquisition and data presentation as *total update delay*. The total update delay is composed by the time needed to acquire the data and the time needed by the algorithm to calculate the number that should appear on the screen. The total update delay should not be confused with the *update time*, which is the period between the appearance of two subsequent numbers on the screen. The total update delay of an AEP is considerable when using the classic extraction technique, *i.e.*, the moving time average. However, the literature describes methods other than the moving time average, which can extract the AEP within a few sweeps.[2,3] The method implemented in a monitor by our group is the autoregressive model with exogenous input, which facilitates extraction of the AEP within 15 sweeps, thus producing a total update delay of the AEP of 1.7 s.[4,5] Iselin-Chaves *et al.*[1] state that the method used by Davies *et al.*,[6] the moving time average, has a total update delay of the AEP index within 3 s. This is not correct—the method used by Davies *et al.*[6] displays a new index value every 3 s (the update time), but the total update delay is 36.9 s. The update time is independent of the total update delay; thus it is important that a method with a short update time is not interpreted mistakenly as a method with a short total update delay. Iselin-Chaves *et al.*[1] state that the A-2000® monitor (Aspect Medical, Natick, MA) has a continuous display of the Bispectral Index; however, the total update delay is approximately 30 s.

According to the second issue, difficulties in the interpretation of the latencies of AEP peaks exist. However, to overcome these difficulties and simplify the interpretations, different research groups have defined indices that automatically quantify the peak changes into a single number. The objective of an AEP index is to substitute manual interpretation.[7–9]

The third issue (setup time > 5 min) is solved by modern monitors, such as the A-line® AEP monitor (Danmeter, Odense, Denmark). The only difference between this monitor, and, for example, the A-2000 BIS® monitor is that besides electrodes, a set of headphones are needed.

The fourth issue is deafness. An AEP cannot be created in a patient who has 100% hearing loss. However, if a patient has only partial hearing capability, an AEP could be evoked by a click stimulus. Changes in hearing level in the range 20–30 dB do not have significant influence on AEP peak amplitudes and latencies.[10] The percentage of people with total hearing impairment is low—0.074% of the total population.[11]

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Tracheostomy Tube Replacement: Role of the Airway Exchange Catheter

To the Editor:—We read with interest the case report by Dr. Baraka[1] and Dr. Benumof's accompanying editorial[2] regarding difficulties associated with the use of an airway exchange catheter. The following case shows a potential pitfall of using the catheter to exchange tracheostomy tubes, a previously unreported use of the device.

A 67-yr-old man who had a fresh tracheostomy for prolonged ventilatory support required fiberoptic bronchoscopy for evaluation of hemoptysis. Because of concerns that bronchoscopy would be difficult *via* the tracheostomy tube (Shiley-6; Mallinckrodt Inc., Critical Care Division, St. Louis, MO), a plan was formulated to change to the next larger sized (Shiley-8) tube using an airway exchange catheter (JEM Instrumentation Industries, Bethel Park, PA). The catheter was in-

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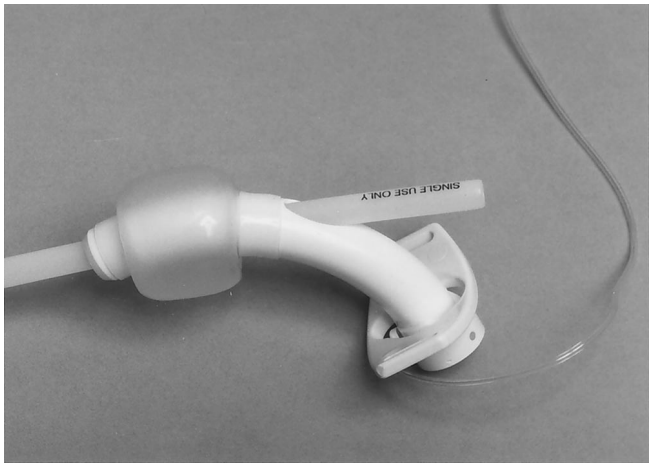

Fig. 1. Demonstration of the attempted tracheostomy tube change with an airway exchange catheter. In attempting to pass the tracheostomy tube over the airway exchange catheter, the catheter tends to protrude through the fenestration rather than traversing the curved tracheostomy tube.

serted, and the old Shiley-6 tube was removed easily. However, difficulties occurred when attempting to position the new Shiley-8 tube, which was fenestrated. With its inner cannula in place, the tracheostomy tube would not slide over the airway exchange catheter, even with liberal lubrication. With the inner cannula removed, the catheter tended to protrude through the fenestration instead of traversing the curvature of the tracheostomy (fig. 1). After several unsuccessful attempts of guiding the exchange catheter through the tracheostomy tube channel, we abandoned the technique and subsequently secured the airway with conventional laryngoscopy and intubation. When the bronchoscopy was completed, we reinserted a tracheostomy tube under fiberoptic guidance. Afterward, we learned *via* an *ex vivo* trial that the airway exchange catheter would bypass the fenestration and pass through the tracheostomy tube if the end of the catheter first were bent into a curve, mimicking the curvature of the tracheostomy tube.

Tracheostomy tubes, frequently placed surgically in critically ill patients who require prolonged airway access and ventilatory management,[3] occasionally need to be changed. Attempting to change a fresh tracheostomy tube before maturation of the tracheal cutaneous tract (typically 5–7 days)[3] may result in dissection between tissue planes, creating a false passage and loss of the airway.

The airway exchange catheter may be of benefit when a tracheostomy tube must be changed before maturation of the tracheal cutaneous tract. Use of the airway exchange catheter for tracheostomy tube exchange, although intuitive, has not been described previously. The preceding case was presented to highlight that potential difficulties may not be apparent readily when using an airway exchange catheter for an unconventional application, such as changing a tracheostomy tube.

Obviously, we could have avoided this problem had we used a nonfenestrated tracheostomy tube, or even rehearsed the exchange maneuver *ex vivo*. However, we proceeded with the equipment we had at hand and did not anticipate any difficulty because of our previous experience with airway exchange catheters. Clearly, such experience was insufficient to predict that the lack of flexibility of the tracheostomy tube would limit the excursion of the airway exchange catheter.

In conclusion, although use of an airway exchange catheter may be intuitive in exchanging tracheostomy tubes, we strongly encourage *ex vivo* rehearsal with the catheter and tracheostomy tube before performing the exchange to ensure a smooth, efficient procedure.

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Transient Lingual and Glossopharyngeal Nerve Injury: A Complication of Cuffed Oropharyngeal Airway

To the Editor:—Lingual nerve injury is an uncommon complication of anesthetic airway management.[1–8] We report the first case of transient bilateral lingual and glossopharyngeal nerve injury after use of a cuffed oropharyngeal airway (COPA).

A healthy 32-yr-old female with a height of 164 cm and a weight of 65 kg was anesthetized for suction and curettage after spontaneous abortion. Anesthesia was induced using 2.5 mg/kg propofol and 1 μg/kg remifentanil. A 9-cm COPA was inserted easily, and the cuff was inflated with 30 ml air, according to the recommendations of the manufacturer, and secured with ribbon. Because of an audible leak during manual ventilation, 8 ml air was added subsequently into the cuff of the device. Anesthesia was maintained using nitrous oxide–oxygen (50/50) and remifentanil (0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$). At the time of recovery, the COPA was removed, *i.e.*, 20 min after its insertion. The patient immediately reported numbness of the entire tongue. Neurologic examination showed decreases in temperature and in response to light touch and pin prick in the presulcal and postsulcal parts of the tongue, indicating lingual and glossopharyngeal nerve injury. There was neither a motor function deficit nor intraoral trauma. The symptoms resolved spontaneously within 2 h after the operation.

This uncommon complication of the lingual nerve previously described with a laryngeal mask airway is not surprising.[1,2,4,6] Anesthetic airway management with both laryngeal mask airway and COPA uses pharyngeal cuff inflation, which causes an increase of oropharyngeal soft tissue pressure. Excessive pressure exerted against the oropharyngeal mucosa could explain the injury of superficial nerves, such as the lingual nerve and the lingual branch of the glossopharyngeal nerve. Over the styloglossus muscle, these nerves are close to each other and can be injured simultaneously.[9,10] In the case we report, the 8 ml air added into the cuff of the COPA overinflated the cuff by 25% of the maximum volume recommended by the manufacturer (COPA size 9, 30 ml), and was probably the cause of excessive pressure exerted against the oropharyngeal mucosa. With an 11-cm COPA, Brimacombe *et al.*[11] recently showed that even with an inflated volume according to the recommendation of the manufacturer, pharyngeal mucosal perfusion is decreased and decreases further with additional volume. Moreover, nitrous oxide, even used during a brief period, could increase the

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cuff pressure further. A 30-min exposure to 66% N_2O in oxygen induces a 30-mmHg mean increase in laryngeal mask airway cuff pressure.[12]

In conclusion, this case shows the natural course of lingual nerve injury after misuse of the COPA, which is typical of acute compression because of its brief duration and subsequent resolution. The fact remains that the COPA inflated cuff volume recommendation must be followed. In case of air leakage, a change to a larger-sized COPA is probably better than a moderate overinflation of the cuff.

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