

Mortality Related to Anesthesia and Sleep Deprivation in Medical Doctors

To the Editor:—The article of Lienhart *et al.*¹ must be applauded. Because of the authors' careful and high-quality methodology, this work—begun in 1996 and covering an entire nation—was able to estimate anesthesia-related mortality in France. This work confirms the critical role of cardiac disease, aging, emergency care and hemorrhage in mortality related to anesthesia.

Perhaps more importantly, the authors illuminate the importance of human error. In at least 98% of cases, one episode of substandard practice was identified, and in more than half of the cases, four deviations from accepted practice were recorded. The authors acknowledge that their analysis probably underestimated the true incidence of such system error.

Production pressure was a factor in 20% of the events, but despite their belief that errors may occur more frequently during urgent and stressful care, the authors do not mention the role, if any, of a night shift or call, the night of or the 48 h before the accident. Several recent articles have linked fatigue in anesthesia with medical errors,^{2,3} but because anesthesia deaths are rare events (according to the survey, occurring in less than 1 of 100,000 cases) and despite a rigorous methodology, analysis were unable to demonstrate a link between night shifts and anesthetic or intensive care mortality.⁴

Because of the authors' sample size and their careful analysis, the study could have provided a unique opportunity to analyze the role of

sleep deprivation⁵ in the genesis of anesthesia accidents. It would have been of interest to know whether this could have played a role in mortality related to anesthesia.

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In Reply:—We thank Dr. Georges Mion and Soledad Ricouard for their comments regarding our article¹ and their positive assessment of the work of the French Society of Anesthesia and Intensive Care and the Epidemiology Center on Medical Causes of Death at the National Institutes of Health and Medical Research. Their question regards fatigue as a factor facilitating human errors, and especially the role of sleep deprivation as a result of nighttime work. This question is indeed important enough that a government regulation was issued to mandate in France a period of "safety rest" after a night call.² This regulation did not exist at the time the study was performed and, as a matter of fact, in 9 cases of the 419 analyzed deaths (2%), the anesthesiologist having to perform an anesthetic after a night call has been noted as having possibly contributed to the fatal outcome. More broadly, circumstances having impaired the attention or the vigilance of the anesthesiologist, such as performance pressure, stress, or emergency, were found in 43% of cases. However, although the study methodology allows us to be confident regarding the number of deaths, even more so as analysis of files of insurance companies performed since then

supports this point of view, the retrospective and belated nature of the analysis of factors facilitating medical errors implies a risk of underestimating these phenomena.

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Similarity of Operation Times for Common General Surgical Procedures in the United Kingdom and the United States

To the Editor:—We recently published estimates of total surgical and anesthetic times for 31 common general surgical and urologic procedures from our center in the United Kingdom,¹ and suggested that

others in different centers should also publish similar estimates to assess whether our times were unit specific or were more widely applicable. We are pleased that Silber *et al.*² have extended this work

Table 1. Summary of Procedure Times from the Two Studies

| Procedure | Estimates of Times from Study, min | |
|------------------------------------|------------------------------------|-------------------------------|
| | Silber <i>et al.</i> ² | Pandit and Carey ¹ |
| Anterior resection | 240 | 229 |
| Hemicolectomy | 158* | 141 |
| Sigmoidectomy | 155 | 149 |
| Reversal stoma (reversal Hartmann) | 125 | 138 |
| Laparoscopic cholecystectomy | 105 | 86 |
| Incisional hernia | 103† | 85 |
| Mastectomy | 120 | 101 |
| Wide excision breast lesion | 75 | 66 |

The data of Silber *et al.*² are from their cases where no other procedure was billed. The data of Silber *et al.*² are medians of their data set; the data of Pandit and Carey¹ are means of their data set.

* Average of left and right hemicolectomy times. † Average time of incisional hernia repair with and without graft.

and published relevant time estimates. Theirs is an impressively larger database and includes data from 183 hospitals in Pennsylvania; our data were derived from a more modest combination of estimates provided by professional staff and our own operating room database. Despite such self-evident differences, comparing their estimates with ours shows that for procedures examined by both studies, any differences are very small (table 1).

The remarkable similarity of the operative times is significant. First, it confirms our conclusion—and that of others³—that experienced surgeons and anesthesiologists can predict operation times very well. Second, because the times reported by the two studies were from very different centers located in different countries, it suggests that these estimates that can be more widely applied. If this is the case, it follows

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In Reply:—We thank Dr. Pandit for his letter. It is interesting that Medicare anesthesia claims, chart abstraction in the United States,¹ and computerized records from operating rooms in the United Kingdom all produce similar procedure durations, consistent with the validity of all three measures.

Dr. Pandit observes that procedure times at his center are similar to the median times for 183 hospitals in Pennsylvania—this one United Kingdom center would fit near the center of the distribution in Pennsylvania. Although this observation is interesting, it is not logically inconsistent with variation in procedure time between hospitals in both Pennsylvania and the United Kingdom. To see variation between hospitals, one must study several hospitals.

Dr. Pandit asks whether differences between Pennsylvania hospital procedure times are different for different procedures. Indeed they are. We looked at the five most common procedures in our data base (total knee replacement, open reduction of fractured femur with internal fixation, laparoscopic cholecystectomy, total hip replacement, and partial hip replacement) for the 10 largest hospitals in Pennsylvania, adding interaction effects to the model in our article.² Of the $5 \times 10 = 50$ possible interactions, 15 were significant using the Bonferroni correction ($P < 0.05/50 = 0.001$), so that at least for common procedures at large hospitals, procedure duration differs in different ways at different hospitals. This is consistent with the notion that procedure duration at a hospital is a function of the individual physicians, nurses, and care team members practicing within hospitals and possibly performing different procedures with different skills. For example, future

that these estimates can be reasonably applied to the booking of operating lists. This should reduce the incidence of overrunning, which,⁴ as we reported in the United Kingdom, can affect approximately 50% of operating lists and result in approximately 14% of patients being cancelled.^{1,5}

We note that Silber *et al.* also reported that procedure time varies with hospital (a conclusion which is at odds with the similarity of their data with our United Kingdom data, because we might expect wider disparity between the two countries than between different hospitals in the same country). However, it is unclear from their article whether the statistical influence of hospital affected all procedures or just some specific procedures, or whether the between-hospital variability for any (or all) procedures was different from the within-hospital variability.

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research exploring physician characteristics and style of practice inside and across hospitals may help to explain the differences in procedure times we observed by the race and income of the Medicare patient.²

Using our algorithm, Medicare claims can now provide investigators with an opportunity to study a vast number of questions involving patients, surgeons, anesthesiologists, hospitals, and even health systems. Although procedure times have generally been reported from single-institution studies, having times now available from literally 40 million procedures yearly, dating back to 1994 and extending to the present, should allow many new investigations that will greatly benefit our understanding of surgical care and healthcare delivery.

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Disruption of Water Trap from Leak of Isoflurane during the Vaporizer Filling Process in the Dräger Apollo

To the Editor:—We write to inform your readers about our experience with the configuration of the Dräger Apollo anesthesia machine (Dräger Medical, Inc., Telford, PA). We have had several instances where end-tidal carbon dioxide readings were skewed or undetected by the gas analyzer after successful tracheal intubation. Upon inspection of the source of the problem, it was found that the water trap (positioned just below the isoflurane vaporizer; fig. 1) was severely cracked and disfigured (fig. 2). This compromise of the water trap led to the inability to accurately detect and monitor end-tidal carbon dioxide. Upon replacement of the water trap, accurate carbon dioxide monitoring was once again possible.

It was discovered that during the filling process of the isoflurane vaporizer, a small amount of volatile anesthetic had dripped down on the water trap, leading to compromise of the integrity of the plastic outer shell of the trap and loss of the ability to accurately detect and monitor end-tidal carbon dioxide. These conditions were easily reproducible and always led to the same water trap disruption.

Further investigation revealed that inspired and end-tidal volatile anesthetic levels may be skewed as well. In a clean anesthesia circuit with gas flow consisting of 100% oxygen in the presence of a water trap disrupted by spillage of isoflurane, high levels of inspired and expired isoflurane were detected despite the vaporizer having never been turned to the "on" position.

Most anesthetic machines have a configuration that places the water trap above the vaporizers. The Dräger Apollo has a unique configuration that places the water trap directly below the vaporizers and at risk from spillage of volatile anesthetic agents during the filling process. Since these episodes, we have repositioned the isoflurane vaporizer into the position to the far left side of the machine, away from the water trap. So far, this change seems to have eliminated the issue. Incidentally, sevoflurane and desflurane seem to cause little to no disruption of the water trap based on our limited trials of direct exposure. Perhaps chronic exposure to these agents may lead to similar disruptions, but certainly not to the same degree as isoflurane. The filling system on the desflurane vaporizer also seems to be less prone to spillage, thereby making it a seemingly more appropriate choice for placement in the position above the water trap. Other possible solutions might include removal of the vaporizer before filling it and avoiding overfilling of the vaporizer to minimize dripping during the filling process.

Potential problems that can arise may include incorrect assessment of correct tracheal placement of the endotracheal tube, skewed or absent capnograms, and elevated inspired and end-tidal volatile anesthetic readings. It is hoped that this information will help practitioners have early recognition of these water trap disruptions to avoid misinterpretation of clinical situations. Practitioners should also consider avoiding the placement of the isoflurane vaporizer directly above the water trap and should have replacement water traps readily available so that patient care is not interrupted.

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Fig. 1. Dräger Apollo anesthesia machine with water trap located directly below the isoflurane vaporizer.



Fig. 2. Water trap that was exposed to isoflurane (left) compared with a normal water trap (right).

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Tega-dermabrasion

To the Editor:—Injury to soft tissues including eye injury and blindness is a cause for concern, especially during prone positioning during anesthesia. We report a case of periorbital skin injury in a healthy 19-yr-old man after the use of a transparent medical dressing (TMD) (Tegaderm; 3M Health Care, St. Paul, MN) for eye closure during general anesthesia.

At our institution, TMD (Tegaderm) is routinely applied for eye closure to prevent injury, especially for prone positioning during general anesthesia. In our case, a healthy 19-yr-old man underwent general anesthesia in the prone position for radiofrequency ablation of a coccygeal osteoma. After an uneventful induction of general anesthesia, two 2 3/8" × 2 3/8"-inch pieces of TMD (Tegaderm) were applied for eye protection. The patient's head was supported with a standard foam headrest pillow (Gentle Touch 7-inch Head Rest Pillow with Right Intubation Slot; OSI, Union City, CA). The patient had no history of sensitivity to tape or latex. The procedure lasted for 90 min, with no blood loss.

At the conclusion of the case, the TMD was carefully removed. The periorbital skin appeared warm, erythematous, hemorrhagic, and mildly edematous. The inflamed areas were rectangular and conformed to the shape of the TMD. The patient reported periorbital burning and tingling pain.

There was an immediate concern of permanent disfiguring periorbital scarring from the hemorrhagic wound. Further inquiry revealed that the patient had been prescribed a topical acne treatment wash, Benzac (5% benzoyl peroxide), and had been using it regularly. The dermatologist was consulted, and options for reducing scarring were discussed. Desonide 0.05% cream, a topical glucocorticoid, was applied to the affected areas to minimize reactive scarring and discoloration. Over a period of 6 h, the erythema improved; the patient was discharged home (fig. 1). Over the next week, the inflammation subsided, with no residual scarring or discoloration.

Most corneal abrasions during general anesthesia are thought to be secondary to lagophthalmos, an incomplete closure of the eyelids.¹ Adhesive tape or TMD (Tegaderm) are routinely used to approximate the eyelids. We are reporting this case because it is unique for a young, otherwise healthy adult to sustain a skin stripping injury from an adhesive used for this purpose.

The populations usually at risk of skin injury from adhesives are patients in extremes of age (premature infants and elderly) and patients on long-term steroid therapy. TMD (Tegaderm) material has been used in patients with thin and friable skin, including premature neonates, as a method of preventing excessive fluid loss.² However, when used on skin pretreated with benzoyl peroxide, it seems to cause skin stripping, similar to dermabrasion. Benzoyl peroxide is a powerful bleaching agent and is used in the treatment of many kinds of acne.

Cases of periorbital contact dermatitis have been reported in the literature.³ However, contact dermatitis usually results from repeated exposure after an initial sensitization.

Although benzoyl peroxide skin treatment for acne is effective, side effects include skin drying and peeling.⁴ The effect of benzoyl perox-



Fig. 1. Appearance before discharge.

ide on skin stripping has not been studied extensively. However, the location of the skin, the time of contact, and the applied pressure play a role in the extent of skin stripping.⁵ Therefore, the skin of patients using benzoyl peroxide facial wash is likely more susceptible to skin stripping with TMD (Tegaderm). This cross-reaction between benzoyl peroxide facial wash and TMD (Tegaderm) has not been reported in the literature.

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Perioperative Thrombotic Risk of Coronary Artery Stents: Possible Role for Intravenous Platelet Blockade

To the Editor:—I read with great interest the recent science advisory by Dr. Grines *et al.*¹ detailing their recommendations with respect to dual platelet therapy (aspirin and clopidogrel) after placement of a coronary stent. The advisory highlights the perioperative thrombotic risk for coronary stents. In the perioperative setting, they should be regarded as an unstable coronary syndrome.¹⁻² Where possible, elective procedures should be postponed for at least 1 month (bare-metal stent) or up to 1 yr (drug-eluting stent). Furthermore, dual antiplatelet therapy should be continued throughout the perioperative period, with only temporary cessation of clopidogrel therapy where clinically indicated, *e.g.*, excessive bleeding risk.

Even with protocol-based intensive perioperative treatment of these patients in the noncardiac surgical setting, there is still a 4.9% mortality rate and a staggering 44.7% complication rate.² The major etiology of perioperative stent thrombosis is the activated platelet, which may require multimodal blockade even in the setting of an overt bleeding risk.

What about the adjunctive role of perioperative intravenous platelet blockade with IIa/IIIb blockers such as tirofiban? This would allow preoperative withdrawal of clopidogrel with ongoing precise control of platelet blockade. This application was recently successfully illustrated in the following integrated protocol involving three patients: clopidogrel discontinued 3 days before surgery; preoperative hospital admission for infusion of unfractionated heparin and tirofiban until 6 h before surgery; loading dose of clopidogrel on the first postoperative day, followed by maintenance therapy; aspirin therapy continued throughout the perioperative period.³ This encouraging pilot experience awaits confirmation of efficacy and safety in further trials. Until

then, the safety and efficacy of this perioperative approach are not conclusively established.

It is imperative that aggressive anticoagulation, including dense platelet blockade, be maintained throughout the perioperative period to maintain coronary stent patency. This is particularly important if the stent has not endothelialized, as may be the case in emergency surgery. The coronary stent, whether bare metal or drug eluting, should be managed as a high-risk coronary lesion in the perioperative period. Intravenous IIa/IIIb platelet blockade has tremendous promise as an adjunct in the perioperative management of these iatrogenic unstable coronary artery syndromes.

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Lipid Reversal of Central Nervous System Symptoms of Bupivacaine Toxicity

To the Editor:—Having read with interest the recent reports of treatment of local anesthetic toxicity,¹⁻³ I would like to report a recent case of inadvertent intravenous bupivacaine injection leading to central nervous system toxicity, managed primarily by intravenous lipid. I believe it may lend support to the academic models suggesting the efficacy of lipid emulsion in bupivacaine-induced cardiac toxicity.⁴⁻⁶

An 18-yr-old primigravida, weighing 86 kg, presented at 38 weeks gestation for induction of labor. She had an unsatisfactory nonstress test, borderline hypertension (160/81 mmHg), and mild proteinuria. Her cervix was 1 cm dilated, and the fetal heart rate was 180 beats/min with decelerations to 110 beats/min.

Epidural analgesia was requested by the obstetrician after membranes were ruptured and was performed with the patient sitting, at the L1-L2 interspace with good loss of resistance to saline at 7 cm. The catheter was advanced 4 cm and had free flow and no return of blood or cerebrospinal fluid. A test dose of 4 ml lidocaine, 2%, was negative for intravenous or spinal effects after 5 min. Six milliliters isobaric bupivacaine, 0.25%, was then given over 2-3 min, with good pain relief within 10 min. Blood pressure, however, slowly increased over

the next 15 min to 172/114 mmHg, with a heart rate of 86 beats/min and more pronounced fetal heart rate decelerations. Oxygen was administered by mask at 10 l/min, the obstetrician was called to come urgently, and in anticipation of cesarean delivery, 100 µg fentanyl in 6 ml was given *via* the epidural catheter. The blood pressure continued to rise over a further 15 min to 165/123 mmHg, with a pulse of 119 beats/min, and the patient was given a 10-ml dose of bupivacaine, 0.5%, *via* the catheter after a negative aspiration check, in anticipation of the likely decision for operative delivery. Within 90 s, the patient became restless and agitated and did not obey commands. She displayed twitching of her face and limbs. A further aspiration of the epidural catheter now revealed venous blood easily withdrawn, and the patient then became unresponsive. Blood pressure was maintained at 150/110 mmHg, pulse was 120 beats/min, and fetal heart rate was 130 beats/min with decelerations to 90 beats/min. There was no electrocardiographic monitoring of the patient—it is not routine in this hospital.

The differential diagnosis included cerebral irritation secondary to either pregnancy-induced hypertension or, more likely, intravenous bolus bupivacaine. Because of the obvious concern for imminent cardiac arrest, the crash cart was brought in, and I elected to administer lipid emulsion—which our department had recently elected to

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keep on the cart—intravenously, while anticonvulsant medication was being drawn up (diazepam). Two 50-ml boluses of 20% Intralipid were given, and the remaining 400 ml was run in freely as an infusion. Within 30 s, the patient regained full consciousness, and although she was scared, she was considerably calmer than I was! Her blood pressure was 170/109 mmHg, her heart rate was 88 beats/min, and the fetal heart rate was bradycardic at 87 beats/min.

The patient was transported from the labor suite to the operating rooms (located in our hospital one floor above), where an emergency cesarean delivery was performed during general anesthesia. A 6-lb neonate was delivered with Apgar scores of 0 at 1 min, 7 at 5 min, and 10 at 10 min after intubation and suction. The neonate was extubated in the operating room, and subsequently the neonatal examination was pronounced normal.

The mother had an uneventful postoperative course (of note, there was no significant epidural block in the postanesthesia care unit), with her blood pressure settling after MgSO₄-labetalol infusions as per our hospital protocols for 24 h, and both were discharged home on the fourth postoperative day. Both remain well to date.

I believe that this patient's epidural catheter migrated into an epidural vein, and that her symptoms were due to inadvertent intravenous bupivacaine. The timing of events makes the diagnosis of eclampsia less likely. The decision to use the lipid early was in an attempt to avoid the likely catastrophic consequences to both mother and baby of bupivacaine-induced cardiac arrest. Such arrests are often refractory to conventional resuscitative techniques, and it was the opinion of this doctor that the potential benefits of the lipid outweighed its potential risks.

However, several alternative explanations for the scenario described above are possible and need to be considered. The seizures may have been self-limiting and may have resolved spontaneously without intervention. Progression to cardiotoxicity may not have occurred even without treatment. The safety of rapid bolus administration of lipid is

not certain. It is impossible to say with certainty or with the backup of hard data that the lipid emulsion had any effect whatsoever, or that the rapid recovery seconds after its administration was anything more than coincidence.

However, it is only by case reports that the merits or otherwise of such an intervention (which can never be subjected to controlled trials) can be put forward for the anesthetic community to decide whether it will become a part of accepted practice. More case information that would shed light on the role of lipid in the management of local anesthetic toxicity—both for and against—would be sincerely welcomed.

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