

Is the Operating Room of the Future a Viable Economic Reality?

To the Editor:—In the August 2005 issue of ANESTHESIOLOGY, Sandberg *et al.*¹ describe an Operating Room of the Future (ORF) that includes extensive physical and workflow redesigns for “optimal support of advanced minimally invasive surgery.” The ORF enhancements incorporate increased capital costs for advanced equipment and increased personnel costs as compared with their standard operating room. These costs are justified by their findings that they are more than offset by increased revenues resulting from increased efficiency in the ORF.

Several points should be carefully considered before any institution attempts to replicate such a model.

Insurance mix: Revenues are strongly affected by the “insurance mix” of the patient population. In California, Medi-Cal (Medicaid) covers only approximately 46% of fully allocated hospital costs, whereas some preferred provider organization plans cover as much as 120% of those same costs. Sandberg *et al.* do not reveal the insurance mix of their study population. Given that capital and personnel costs are entirely independent of fluctuations in insurance mix, a hospital with an unfavorable insurance mix could easily fail to offset the increased costs of the ORF model with increased revenue.

Operating Room of the Future utilization: Sandberg *et al.* use a model for utilization that places only one surgeon working within the ORF each day. In work at our institution, we found that, using single surgeon utilization, similar daily enhancements in operating room throughput could be achieved with extensive workflow redesign. However, when multiple surgeons were scheduled within a single operating room on a given day, all time savings gained through enhanced efficiency were lost awaiting next surgeon arrival. Seventy-four percent of all delay codes during a study period were under the heading of “awaiting surgeon arrival,” whereas 83% of total delay minutes were under that same heading (R. A. Dritz, M.D., Berkeley, California, unpublished data, 1997; data gathered by assigned observational nurse in the operating room). At our institution, only 8–12% of

all operating rooms are scheduled with only a single surgeon on any given day. Given that Sandberg *et al.* further note that, even when only using a single surgeon, not all surgeons and surgical case mixes benefit from the inclusion in the ORF (see Sandberg *et al.*, table 2); one can see that extreme care must be taken in managing operating room utilization patterns if one is to achieve the financial enhancements they describe.

Capturing productivity gains: It is important that time savings resulting from increased efficiency be filled with other productive activities and not lost to downtime. For example, if a surgeon required a full 8-h shift to perform a surgical caseload in a standard operating room setting but successfully completed those same cases in 6 h in the ORF, it is essential that the 2 h saved not be squandered either awaiting another surgeon’s arrival or enhancing coffee break times. Because of the increased costs per hour in the ORF, time savings lost to nonproductive activities in the ORF could make it a net loss when compared with the standard operating room.

In summary, Sandberg *et al.* present an intriguing model to enhance operating room efficiency. However, extreme care must be exercised before choosing to replicate such a model in another hospital setting. Given the current realities of hospital economics, the Operating Room of the Future may not be economically viable in the present.

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1. Sandberg WS, Daily B, Egan M, Stahl JE, Goldman JM, Wiklund RA, Rattner D: Deliberate perioperative systems design improves operating room throughput. ANESTHESIOLOGY 2005; 103:406–18

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The Elephant in the Operating Room

To the Editor:—The three articles^{1–3} addressing improved operating room (OR) efficiency plus the accompanying editorial⁴ skirt an important determinant of OR efficiency. At many institutions, different surgeons performing the same procedure in the same ORs on the same acuity of patient vary more than threefold in times to perform operations.

Anesthesiologists, perioperative nurses, OR schedulers, and hospital architects can improve OR times by minutes per case. Surgeons may potentially improve times by hours per case. Clearly, the savings are in surgical techniques and behaviors.

Hospital administrators are reluctant to embrace this approach. In my former hospital, the anesthesia service met several times with the hospital chief executive officer to discuss means of shaving minutes off turnover and induction times. The chief executive officer had no conversations with surgeons about sharing surgical techniques that might save hours of time.

Administrators, services chiefs, and clinicians avoid the elephant in the operating room: The biggest determinant of OR efficiency is the facility of the surgeons who work there.

Hospitals might consider rewarding surgeons who can, for example, perform a routine laparoscopic cholecystectomy in 45 min and retraining surgeons taking 3 h for the same procedure. This simple alteration

in OR scheduling, giving preference to faster surgeons, carries far more likelihood of allowing one or two extra procedures per OR per day than intubating patients in an induction room.

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2. Torkki PM, Marjamaa RA, Torkki MI, Kallio PE, Kirvela OA: Use of anesthesia induction rooms can increase the number of urgent orthopedic cases completed within 7 hours. ANESTHESIOLOGY 2005; 103:401–5
3. Sandberg WS, Daily BM, Egan M, Stahl JE, Goldman JM, Wiklund RA, Rattner D: Deliberate perioperative systems design improves operating room throughput. ANESTHESIOLOGY 2005; 103: 406–18
4. Dexter F: Deciding whether your hospital can apply clinical trial results of strategies to increase productivity by reducing anesthesia and turnover times. ANESTHESIOLOGY 2005; 103:225–8

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In Reply:—We welcome the comments of Drs. Dritz and Metz on our recent report¹ in ANESTHESIOLOGY. All of the points they raise are valid and must be considered by any institution considering the future of its operating rooms (ORs). Dr. Dritz correctly points out that payer mix influences the cost–revenue balance of any perioperative system redesign that increases capital or operating costs. If a hospital is barely breaking even on its current case and payer mix, increasing costs so that more cases can be performed is a poor decision. However, we would not recommend abandoning the redesign of perioperative processes as a means to improve OR throughput. As we mentioned in our Discussion,¹ the Operating Room of the Future (ORF) is a single-OR research space with many purposes, one of which was to assess the financial impact of extensive physical plant reconfiguration to support parallel processing of perioperative tasks. Therefore, the ORF gains no advantages from the economies of scale that would accrue from even a two-room arrangement. We are aware of several parallel processing perioperative system design initiatives that involve no physical plant modifications or capital equipment purchases, and some of these are staff-level neutral.² Even in such instances, the payer mix strongly influences the results—it is still a poor decision to lose money faster by doing more cases per day if the payer mix is unfavorable. However, when the payer mix and revenue profile are favorable, perioperative system redesigns to achieve parallel processing and higher throughput at modest expense are clearly advantageous.

Dr. Dritz points out the value of block booking (*i.e.*, the practice of allocating a single OR to the same surgeon for a complete day with a full schedule) to enhance OR productivity. His experience of waiting for surgeons when switching surgeons between cases suggests that the surgical staff do not sufficiently appreciate the value of OR time or were not well informed about the OR's impending readiness for their cases. This experience should prompt the hospital to seek and correct the root cause of delays that involve waiting for surgeons, because the cost of unused OR time is prohibitive. As much as 50% of this cost is a variable cost that can be controlled by efficient use of OR time.³ Failure to control such costs degrades the hospital's bottom line but has little impact on the surgeon. Therefore, with incentives misaligned, one worries that it might not be possible to improve performance. However, as we will describe in our response to Dr. Metz below, ORF initiatives may offer a solution.

Dr. Dritz further comments that one must be careful not to allow the productivity gains from parallel processing to be lost as downtime. Acknowledging this concern, it can be seen from table 2 of our work¹ that we were careful to use all of the 9-h workday in both the ORF and standard ORs, thus fully capturing the benefit of the increased capacity. Hospitals considering expending resources on perioperative system redesigns should carefully analyze both their case mix and their case volume before expending resources to enhance perioperative system capacity. The additional capacity should reduce staffing costs (by eliminating overtime or allowing a shorter work shift), allow complete additional cases to be performed, or both. Individual hospitals should apply their own financial frameworks for costs and revenues to the contemplated workflow changes before initiating a perioperative system redesign effort.

To address the concern that our institution-specific analysis is difficult to translate to other settings, we are reanalyzing the cost effectiveness of the ORF using national cost data. In this new analysis, the

ORF is cost effective relative to standard ORs at our institution. In particular, the incremental cost of an additional case in the ORF is quite small—much smaller than the typical net margin for a simple general surgery case.⁴ Therefore, we would challenge Dr. Dritz' final comment that the OR of the Future may not be economically viable in the present. If the incremental cost of an additional case performed in a high-throughput environment is smaller than the cost of a case that must be performed on a different day because the typical OR cannot accommodate it during regular work hours, the ORF is advantageous regardless of the payer mix.

Dr. Metz in his letter correctly identifies differences in practices and performance between surgeons as a major, and frequently the largest, single contributor to differences in OR throughput for a given list of cases. Although we agree that different surgeons have drastically different operative times for the same case type performed in the same patient population, we made a conscious decision to sidestep this issue. Dr. Metz endorses rewarding surgeons who meet benchmarks for operative time. However, structuring such rewards can be problematic. For example, simple financial incentives purely for speed may create conflicts related to quality and patient safety. On the other hand, the ORF project described in our article offers several incentives for superior operative time performance: a small, dedicated team, rapid turnovers, and brief nonoperative times that translate into on-time completion of workdays and extra throughput. By focusing on nonoperative time and by reducing the nonoperative time by restructuring workflow rather than pressuring OR staff to hurry, the ORF creates an environment in which patient contact time and safety are preserved while productivity is enhanced. Because the enhancement in productivity comes almost exclusively from better nonoperative performance, cases with shorter operative times capture the most benefit from the ORF. This logical and inescapable conclusion dictates that block time in high-throughput environments be given preferentially to the most efficient surgeons. Therefore, an OR suite with a few high-capacity ORs such as our ORF gives administrators a tool to reward desirable performance, while creating incentives for other surgeons to improve operative times, all the while preserving the safety and quality profile for the hospital's patients.

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In Reply:—Dr. Metz explains that differences in surgical time among individuals and institutions can be very large, markedly affecting anesthesia group revenue per operating room (OR). This point is so much not under debate that anesthesiologists are paid based on anesthetic time, unlike surgeons and other physicians. German hospitals are describing encouraging success with using transfer pricing so that reimbursement is based just on the surgical time.^{1,2}

Depending on the vagaries of reimbursement, sometimes payment based on anesthesia time is insufficient to compensate an anesthesia group fully for slower surgeons (*e.g.*, revenue per hour is less than costs per hour).³ This is precisely why Amr Abouleish *et al.*³ developed the methodology that affected anesthesia groups can apply or have applied for quantification of these differences (*e.g.*, to explain to stakeholders why group profits are less than expected). For many anesthesia groups, though, the larger financial problem in having variability in OR times among surgeons is the resulting empty but staffed OR time. Statistically developed staffing plans perform well at reducing such variation, thereby increasing anesthesia group productivity and profits. The methods can also be used to calculate an appropriate stipend for the anesthesia group based on the empty but staffed OR time.⁴

Dr. Metz suggests that “Hospitals might consider rewarding surgeons who can, for example, perform a routine laparoscopic cholecystectomy in 45 min and retraining surgeons taking 3 h for the same procedure.” Dr. Metz addresses a concept that I too⁵ thought was logical. However, scientific research found this argument to be economically irrational.

First, rewards of additional resources cannot and should not relate to individual patients, but rather a surgeon’s overall impact on a hospital.⁶ The majority of hospital costs are fixed.⁷ Therefore, contribution margin (*i.e.*, revenue minus variable costs) invariably^{6–8} averages at least \$1,600 per OR hour for a cholecystectomy. Regardless of whether the general surgeon works fast or slowly, on average the hospital increases profit by doing his or her cases. This is important, because hospitals need excess of revenue to costs (*i.e.*, profit) to buy information systems (*e.g.*, anesthesia information management systems), to buy equipment (*e.g.*, anesthesia machines), and to provide financial support to physicians (*e.g.*, anesthesiologists available in-house for obstetrics and trauma).

Second, if facilities were to select surgeons to be rewarded with more resources based on production, speed in performing cases would likely have little influence. Because of differences in fixed costs (*e.g.*, perfusion), reimbursement (*e.g.*, many patients without insurance), and/or implant costs (*e.g.*, cochlear implant), contribution margins per OR hour consistently vary among subspecialties by more than 1,500%.

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In Reply:—We thank Dr. Metz for his comment on our study.¹ We agree with the drawn conclusions. In fact, surgical performance is a key point of operating room efficiency.

The different studies demonstrated measures to successfully reduce anesthesia-related time intervals.^{1,2} Measures such as overlapping induction of anesthesia, implementation of anesthesia induction rooms, and introduction of a deliberate perioperative system improved anesthesia workflow with increase of anesthesia efficiency. These improvements may be deteriorated by inefficient surgical performance.

Anesthesiologists have been investigating operating room time flow

The surgeon’s subspecialty is the key issue. This is like a comparison of stocks for rebalancing one’s portfolio⁹—economic return often depends more on a company’s industry and market than on how well the company executes. The decision of whether to provide more resources to one general surgeon performing cholecystectomies *versus* another is of small financial importance as compared with the comparison of a general surgeon to a cardiac surgeon. The financial argument is even stronger when considered for facilities without incremental reimbursement for each patient (*e.g.*, Canada).¹⁰ Regardless of whether a breast surgeon is fast or slow, the money spent in a day of OR time is insignificant relative to a spinal surgeon. If preferred, these tactical analyses can also be considered in terms of value to society by using cost utility (*e.g.*, cataract replacement *vs.* bariatric surgery).

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for a long time. This should exert pressure on our surgical colleagues to challenge their procedures as well. Nevertheless, one must keep in mind that the fastest surgeon may not be the best one in terms of the patient’s safety, intensive care unit stay, total hospital stay, and rate of complications. Therefore, quality of an operation is a complex, multi-factorial task, and surgical case duration is only one (nevertheless important) part of it.

We hope that there will be several studies in the near future published in surgical journals as well, demonstrating measures to improve surgical work flow and efficiency showing an increase of surgical quality.

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In Reply:—On behalf of my coauthors, I would like to thank Dr. Metz for his concern regarding the role of a surgeon in operating room efficiency. It is true that longer-than-average case times cause inefficiency and can lead to increased staffing costs as well as increased fixed costs.^{1,2}

Our study, however, was merely a process-oriented approach in which the focus was not the value-adding time, be it anesthesia time or surgery time. Instead, the goal was to decrease nonoperative time. In fact, before implementing the induction room model, the average nonoperative time in our orthopaedic case mix exceeded the average surgery time. Because the percentage seems to be substantial in many other surgical services as well,³ decreasing nonoperative time seems like a logical starting point in improving operating room efficiency.

Fortunately, not all surgeons are slow. Lengthy nonoperative times, in turn, tend to be an everyday phenomenon, occurring between every case and easily adding up to at least one case length per day.⁴ Surely, after the nonoperative time has been decreased to minimum, attention should be turned to value-adding time.

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Carnitine Deficiency, Mitochondrial Metabolism, and Abnormal Response to Anesthetics

To the Editor:—We would like to commend Dr. Uezono *et al.*¹ for their insightful letter describing the repeated occurrence of a clinical picture resembling propofol infusion syndrome in a patient with carnitine deficiency during fat emulsion therapy. The inference is that carnitine deficiency sensitizes patients to challenges that either overwhelm (fat infusion) or inhibit (propofol) β -oxidation or mitochondrial function in general. Our report several years ago of a patient with systemic carnitine deficiency who exhibited severe arrhythmias after a small subcutaneous dose of bupivacaine is entirely consistent with this observation.^{2,3} Propofol, in addition to the well-described occurrence of metabolic acidosis of the infusion syndrome, can also induce severe bradycardia and hypotension with acute administration of a standard induction dose. This apparently idiosyncratic reaction might result from underlying carnitine deficiency or another asymptomatic or unrecognized abnormality in mitochondrial function. The further implications are that patients with known mitochondrial disease should not

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In Reply:—I appreciate Drs. Weinberg and Baughman for their interest in our Letter to the Editor.¹ They call attention to an underlying carnitine deficiency associated with an increased risk of unexpected metabolic acidosis and cardiovascular collapse triggered by drugs that may potentially induce mitochondrial dysfunction. Acquired carnitine

2. Torkki PM, Marjamaa RA, Torkki MI, Kallio PE, Kirvela OA: Use of anesthesia induction rooms can increase the number of urgent orthopedic cases completed within 7 hours. *ANESTHESIOLOGY* 2005; 103:401-5

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receive propofol and that patients presenting with unexpected acidosis or cardiac dysfunction after a usual dose of propofol should be screened for metabolic abnormalities, including carnitine deficiency.

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deficiency may be caused by cirrhosis, chronic renal failure, malabsorption syndrome, renal tubular acidosis, and certain drugs that are conjugated carnitine (*e.g.*, valproate, zidovudine), which are not uncommon in the intensive care unit setting.² Of particular note, patients receiving valproate may develop low concentrations of free carnitine

in plasma, thus rendering them at risk for development of mitochondrial β -oxidation defects when propofol is used for sedation. This may partly explain why many case reports of so-called propofol infusion syndrome have been reported from patients in the neurosurgical intensive care unit but not the general surgical intensive care unit.³ If this is the case, I suspect that L-carnitine therapy may reverse clinical manifestations of propofol infusion syndrome.

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Investing Layer of the Cervical Fascia of the Neck May Not Exist

To the Editor:—Nash *et al.*¹ provide some fascinating anatomical data, based largely on plastination techniques and confocal microscopy, to suggest that the investing layer of cervical fascia may not exist. We write first to correct some of their assumptions related to our previous anesthetic work and second to crystallize a general hypothesis that stems from their conclusion.

Nash *et al.*¹ state in their opening paragraph that the previous work of Pandit *et al.*² concluded that the “superficial cervical plexus block” injection should be placed superficial to the investing layer. In fact, the study of Pandit *et al.*² (in preserved cadavers) concluded that only an injection deep to the putative investing layer would enable the injectate to spread beyond the prevertebral fascia. Pandit *et al.*² observed that a strictly superficial injection did not spread beyond the subcutaneous layers. The implication was that a purely superficial or subcutaneous injection would be clinically ineffective. It was this that led to the suggestion that an injection just deep to the so-called investing fascia should be properly termed an *intermediate* cervical plexus block,³ whereas an injection deep to the prevertebral fascia should be termed a *deep* block.⁴

The conclusion of Nash *et al.*¹ (which we find anatomically persuasive) that the investing fascia does not exist not only raises further problems for proper nomenclature of the various anesthetic blocks, but also leads to a specific hypothesis.

If the result of Nash *et al.*¹ is correct and the investing fascia does not, in fact, exist, the clinical efficacy of a subcutaneous injection should be as effective as an intermediate injection below the putative investing fascia. If, however, the result of Pandit *et al.*² is correct, the intermediate injection should be more effective clinically

than the subcutaneous injection. We are currently investigating this hypothesis in a clinical study and hope to report our results soon.

Although it might be supposed (as a matter of prejudice) that we hope our own results are correct and that “intermediate” injections prove to be more effective than simple subcutaneous ones, it would actually be desirable for overall patient care if the more superficial injections were found to be equally effective. As we have observed elsewhere, safety is increased by more superficial, as opposed to deep, injections.^{5,6}

In summary, Nash *et al.*¹ have offered some truly exciting anatomical data on which to formulate an important clinical question.

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In Reply:—We thank Drs. Pandit, Dorje, and Satya-Krishna for their encouraging comments on our article.¹ From the anatomical angle, we support the suggestion regarding proper nomenclature of the various anesthetic blocks,² but we are concerned about what anatomical landmark could be used to demarcate a “superficial” and an “intermediate” cervical plexus block. We understand that a superficial block in clinical practice involves making an injection in the subcutaneous layer, whereas an intermediate injection is intended to be placed just deep to the sternocleidomastoid muscle.² From an anatomical viewpoint, if the investing fascia does not exist, we suggest that the location of the intermediate injection may be imprecise.

The pattern or configuration of connective tissue is much more complex than our previously held view.^{1,3} In addition to the investing layer of deep cervical fascia, two anatomical points should also be considered when testing the hypothesis raised in the letter of Pandit *et al.*

One is the muscular and aponeurotic fibers of platysma. As shown in figure 5 of Nash *et al.*,¹ these fibers cover the anterior and lateral cervical regions, are layered, and often mimic the investing layer of deep cervical fascia (also see fig. 4C of Nash *et al.*¹). So it may be possible to achieve the functional result predicted by Pandit *et al.* in their letter, despite the anatomical absence of the investing fascia. Zhang and Lee³ also revealed that there is no aggregation of fibrous

connective tissue connecting the sternocleidomastoid and trapezius muscles, but skin ligaments are visualized between the muscles (fig. 2b of Zhang and Lee). The structure, arrangement, and density of the skin ligaments vary greatly through the body⁴ and could mimic the behavior of a fascia. Therefore, a number of clinical and anatomical questions must be further investigated.

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Academic Highway Buzzing, but Clinicians in Crisis

To the Editor:—Professor Ikeda justly brings to light the immense contributions of Michinosuke Amano, M.D. (1916–; Professor Emeritus, Department of Anesthesiology, Keio University, Tokyo, Japan) and the little known Government Account for Relief in Occupied Area program to the progress of anesthesiology in Japan.¹ Because of the efforts of pioneers such as Dr. Amano and Hideo Yamamura, M.D. (1920–; Professor, Department of Anesthesiology, University of Tokyo, Tokyo, Japan), Japanese academic anesthesiology has attained remarkable levels as witnessed by the numerous scientific publications originating from these institutions. The state of clinical anesthesiology in Japan, however, is not as rosy. The specialty suffers from a chronic workforce shortage. The majority of practitioners are salaried hospital employees, forced to work long hours for relatively poor compensation—a clear reason the specialty has trouble attracting personnel. One of the fundamental problems is the inability of anesthesiologists to directly bill the social health insurance system for their services and become independent private practitioners. The Japanese Society of Anesthesiologists; academic centers; the Ministry of Health, Labor and Welfare; and other interested organizations, while acknowledging this problem, have thus far been unwilling or unable to implement the necessary changes. For unclear reasons, what would usually be considered significant bargaining power has not been used to improve the

predicament of clinical anesthesiologists. The result is what can only be described as a crisis, with no relief in sight. Calls are mounting from the surgical (and even within the anesthesia) community for introduction of alternative anesthesia providers—a move that will further devalue the specialty. It is unclear what it will take to force change, because repeated reports of mishaps during surgeon-administered anesthesia are apparently not reason enough.

In Japan, although academic anesthesia flourishes, things have not changed much in the operating room since 1955, being “understaffed, and (with many) anesthetics . . . still given by junior surgeons.”¹ Someone must step up to the plate soon, at the very least to honor the efforts of Dr. Amano and the pioneers, if not for the patients.

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In Reply:—I thank Dr. Kurosu for his interest in my article.¹ Although I am unable to make any specific comments on the current state of clinical anesthesia in Japan, I believe that Dr. Kurosu's concerns about nonacademic anesthesia practice in Japan have parallels in the history of American anesthesiology. The American Society of Anesthesiologists is now celebrating its centennial. In the past century, the American Society of Anesthesiologists and American anesthesiologists have dealt with many problems, which include dealings with government regulation, fair professional compensation, attracting talents to the specialty, and many more similar to the current Japanese situation.²

American anesthesiologists have a century of experience; Japanese anesthesiologists have only had approximately half that time to seek solutions to these problems. Dr. Eugene Sinclair, American Society of Anesthesiologists President from 2004 to 2005, in assessing progress of American anesthesia in a century, observed that the dedication and commitment of pioneers and past leaders laid the foundation of professionalism in our specialty that commands a respectful stature among our peers in medicine and in the public. He believes that our current generation will continue to build on past achieve-

ments and predicts that future anesthesiologists will regard prospective improvements in patient care with equal admiration.²

Current obstacles for Japanese anesthesiology may have their traditional roots indigenous to Japanese society. Pioneers in Japan with great visions, such as Michinosuke Amano, M.D. (Professor Emeritus, Department of Anesthesiology, Keio University, Tokyo, Japan), and Hideo Yamamura, M.D. (Professor, Department of Anesthesiology, University of Tokyo, Tokyo, Japan), established the specialty with true professionalism half a century ago.¹ Given time, the new generations of Japanese anesthesiologists will confidently face any challenges, adapting to a new practice environment ably, and will prevail in the new century. During his 2005 Rovenstine lecture, Mark Warner, M.D. (Professor and Chair, Department of Anesthesiology, Mayo Clinic, Rochester, Minnesota), advised that modern anesthesiologists should dedicate themselves to honor those who have passed before them by making the difficult transitions necessary to thrive in the future.³ Current leaders in anesthesiology, either Japanese or American, should take up the challenges to further the vision and goals set by our pioneers. Examining the history of our profession will help prepare us to encounter these trials and prevail. I hope my article will continue to generate thoughtful and healthy debates on the anesthesiology practice in Japan.

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Critical Role of Intraoperative Transesophageal Echocardiography for Detection of Extrapulmonary Thromboemboli during Surgical Pulmonary Embolectomy

To the Editor:—We read with great interest the excellent case report by Espeel *et al.*¹ that describes two patients experiencing pulmonary embolism with additional extrapulmonary thrombi requiring surgical intervention. The positive outcome of both patients corroborates the favorable experience in patients from our institution undergoing surgical pulmonary embolectomy.² We agree with the authors that intraoperative transesophageal echocardiography is a relatively safe and noninvasive diagnostic modality that allows early detection of intracardiac thrombi. However, we were surprised that the importance of transesophageal echocardiography for the guidance of surgical extraction was not emphasized in this case report. We recently demonstrated that extrapulmonary thromboemboli can be present in the right heart and the vena cava in up to 26% of all patients with massive pulmonary embolism undergoing pulmonary embolectomy.³ Such extrapulmonary thromboemboli may have a significant impact on the surgical procedure, because they may influence cannulation placement and surgical technique during the operation. For example, it may become necessary to perform circulatory arrest in order to evacuate thrombi from the inferior vena cava. Moreover, extrapulmonary thromboemboli that remain unrecognized and are not surgically removed can become the source of recurrent pulmonary embolism.⁴ Therefore, we believe that intraoperative transesophageal echocardiography is not only an excellent tool for hemodynamic monitoring⁵ and management of acute right heart failure⁶ during surgical pulmonary embolectomy, but should also be considered an important diagnostic tool to detect

concurrent extrapulmonary thrombi and should guide their surgical extraction.

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In Reply:—I thank Dr. Nowak *et al.* for their interest in my case report¹ and appreciate the opportunity to reply. There is currently no recommendation for the treatment of acute pulmonary embolism (APE) complicated by embolized type A thrombus in the right cardiac cavities.² However, Rose *et al.*³ suggested that it was reasonable to advocate thrombolysis as frontline treatment after careful screening for contraindications and to reserve surgical thrombectomy for patients with contraindications to thrombolysis or in cases of failure of thrombolysis. For me, the interest in this case report was double. My first interest was to underline the utility of transthoracic echocardiography and transesophageal echocardiography not only for hemodynamic monitoring and management of acute heart failure, but also for systematic research of embolized thrombi in cardiac cavities. Their presence, 3 to 26% in the literature, directly influences the prognosis, with a mortality significantly higher (26-50%) with regard to 8 to 10% for "isolated" pulmonary embolisms.^{3,4} Therefore, it seems essential to search and locate these thrombi precisely, not in the intraoperative period, but immediately after the diagnosis of massive APE, because

this minimally invasive and fast examination will directly affect therapeutic decisions.⁵ With regard to the diagnosis of inferior vena cava thrombosis, I think that when APE is diagnosed on helicoidal computed tomography pulmonary angiography, the addition of venous phase imaging of the abdomen and pelvis is useful and more powerful than transesophageal echocardiography because it allows complete exploration of the femoro-ilio-caval venous network.⁶

My second interest was to report the experience of our team in the management of a particular APE complicated by the presence of a type A thrombus, floating in both right and left cardiac cavities through the oval foramen. We chose the surgical thrombectomy as treatment of choice because of the high risk of systemic embolism, in particular cerebral, making thrombolysis dangerous. In our practice, we choose surgical thrombectomy first in these clinical situations or in the presence of contraindications to thrombolysis and second if thrombolysis is ineffective. We do not consider thrombolysis as an absolute weapon but as one of the therapeutic alternatives available in the management of serious APE as well as surgical thrombectomy. Caval or pelvic

venous thrombus does not seem to me to constitute a real contraindication to thrombolysis because, even though the fragmentation of the thrombus with pulmonary embolization is often feared by the physicians, it could never be shown clinically.^{7,8} Thrombolytic therapy can be given quickly; is available at all centers; and results in the simultaneous thrombolysis of venous, cardiac, and pulmonary clots. In addition, I think that surgical thrombectomy should not be reserved for desperate cases of refractory cardiogenic shock or cardiac arrest, where mortality is close to 100%. A well-designed, prospective, randomized, multicenter trial is needed to determine which treatment has the best cost-effectiveness/safety ratio.

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Is Low-current Search a Risk Factor in Peripheral Nerve Localization?

To the Editor:—I read with interest the article by Capdevila *et al.*¹ In this article, the authors presented a multicenter prospective analysis of the quality of postoperative analgesia and complications after continuous peripheral nerve blocks. After reading this analysis, it occurred to me that some points may be added to the discussion. Capdevila *et al.*¹ reported an overall incidence of different neurologic events of 6.6% and an incidence of severe neurologic deficit of 0.2%, a value quite near that reported in other studies.^{2,3} Although I agree with the authors about their risk factors, I believe that the use of low current output of less than 0.5 mA might present an additional risk factor for nerve injury. The intensity of the electrical current delivered is related to the distance between the needle and the stimulated nerve.⁴ Different authors^{4,5} have shown that, with an intensity of 0.1 mA, the needle must be in contact with the nerve to elicit a motor response, whereas at 2.5 cm, the current required to give a motor response is 2.5 mA. This presumes that electrical stimulators must offer sufficient precision while using low current to locate nerves.⁴ Lack of this precision may lead to the release of currents of less intensity than the rating actually selected, with a higher risk of nerve injury.⁴ Hadzic *et al.*⁵ evaluated the characteristics of 15 stimulators used for peripheral nerve blocks in clinical practice and reported that the median error increased from 2.4% at 0.5 mA to 10.4% at 0.1 mA, and 4 of their tested stimulators varied by more than 30% when set to deliver a current of 0.3 mA. In contrast, they suggested that it would seem more prudent to use a current of 0.5 mA or greater. Accordingly, with a low current intensity of less than 0.5 mA, the stimulator may deliver a lower current than what the operator had selected, leading him or her to continue to advance the needle toward the target nerve when, in fact, it is very close to the target nerve as in paresthesia techniques. Moreover, Karaca *et al.*⁶ reported that painful sensory paresthesia is not frequent during low-intensity stimulation, and others⁷ suggested that a degree of contact might exist between the needle and the nerve even in the absence of motor response. Some authors⁴ consider that a current between 0.5 and 1 mA is sufficient to ensure efficient block. Capdevila *et al.*¹ performed their blocks using a current of less than 0.5 mA (frequency of 1 Hz and impulse duration of 100 μ s). The incidence of their nerve complications corroborate with the study of Horlocker *et al.*,³ where five of seven nerve injuries were related to paresthesia search of a target nerve.

Although severe neurologic damage after peripheral nerve blocks is rare, it is devastating for the patient and for the medical staff. The most common recurrent theme in peripheral nerve block claims is nerve injury.⁸ Accordingly, we can presume that a high percentage of severe nerve injuries after peripheral nerve blocks might lead to claims. However, temporary minor complications that are encountered in clinical practice, such as several days or weeks of paresthesia, do not lead to claims but might be disabling for the patient. Furthermore, such minor complications might also lead to a delay in patients' rehabilitation and return to normal activity. After regional anesthesia techniques, the event presumed to be most damaging is needle trauma and local anesthetic toxicity.⁸ Surprisingly, medical experts never evoke the lack of precision of stimulators as a possible factor for damage in claims.

In conclusion, despite that severe nerve injury after peripheral nerve blocks is rare, it may lead to claims. However, I believe that low-current search of less than 0.5 mA could present an additional risk factor for nerve injury. A current less than 0.5 mA provides almost an equal success rate as currents of 0.5-0.6 mA. Accordingly, I believe that low-current search should not go less than 0.5 mA, which is an acceptable limit for a good success rate and safety.

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In Reply:—We thank Dr. Al-Nasser for the attentive reading of our article regarding the use of continuous peripheral nerve blocks after orthopedic surgery.¹ We understand and accept some remarks regarding the possible risk of neuropathy for intensities lower than 0.5 mA during the nerve stimulation procedure. Dr. Al-Nasser's concerns, which have already been evoked by Auroy *et al.*,² are supported by recent articles reporting that for low-intensity and short-duration nerve stimulation (< 0.5 mA, 0.1 ms), needle-nerve contact can be obtained without any muscle movement³ or pain.⁴ However, some points must be clarified: Research of a minimal intensity during nerve stimulation was not a part of our study design; all of the studies reported by Al-Nasser were related to single-shot blocks and not continuous peripheral nerve blocks; the authors do not decide, regardless of whether it seems important, that one element or another is a risk factor—rather, the multivariate analysis by logistic regression concludes that; the authors^{5,6} who reported the vicinity of nerve and needle tip for values less than 0.5 mA used theoretical biophysics data but did not check their data in clinical practice (ultrasound studies) or in animals; and it was recently reported that signs of nerve inflammation after a peripheral nerve block appeared only after a minimal low-intensity threshold value of 0.2 mA.⁷

The stimulating current at which a needle is sufficiently close for a successful block but still at a safe distance from the nerve to avoid injury is unknown.⁸ In our study, the placement of the needle was considered successful when a specific muscle contraction was obtained at a current output of less than 0.5 mA (1 Hz and impulse duration of 0.1 ms). The current was then gradually decreased until the muscle twitch stopped between 0.4 and 0.2 mA. Nerve stimulation below 0.2 mA was never sought. Intensity of less than 0.5 mA did not seem to be a risk factor. Several elements might explain that: All continuous peripheral nerve blocks were performed by highly trained anesthesiologists following standardized insertion techniques; the nerve stimulators, which delivered the dialed current, were regulated to deliver the actual current; the catheters were inserted for values between 0.2 and 0.5 mA; and there was no motor response for intensity of less than 0.2 mA.

Most importantly, the risk of nerve lesion increases when a physician uses an old nerve stimulator that reports only the theoretical current and not the current actually delivered, which can be lower. If anesthesiologists use this standard of nerve stimulator, they should not set their threshold at 0.5 mA, but invest in a new nerve stimulator to limit the risk of nerve injury.

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The Treatment Should Not Be Worse Than the Disease

To the Editor:—I read with interest the study by Ngan Kee *et al.*¹ They found that a combination of high-dose phenylephrine infusion and rapid crystalloid cohydration virtually eliminated hypotension in women undergoing cesarean delivery during spinal anesthesia. Preventing or treating hypotension in the parturient after spinal anesthesia for cesarean delivery has been the subject of numerous studies and, as the authors noted, has been referred to as the “Holy Grail” of obstetric anesthesia.² However, the incidence of major complications from hypotension, such as myocardial infarction or stroke to the mother, or neonatal acidosis or low Apgar scores in the baby is almost nonexistent.^{2,3} The most common complications from hypotension are nausea and vomiting, which

may be disturbing but are not dangerous.⁴ Furthermore, treating hypotension when it does occur is straightforward; it almost always responds to relatively small boluses of either ephedrine or phenylephrine.

I contend that using a phenylephrine infusion to prevent hypotension during routine cesarean delivery is too aggressive and not safe, as the authors suggest.¹ A phenylephrine infusion is not benign. Phenylephrine is a potent vasoconstrictor that can cause reactive hypertension and reflex bradycardia. Indeed, close to 50% of the patients in this study developed hypertension from the phenylephrine. Furthermore, to safely use a phenylephrine infusion, especially in high doses as used in this study, the patient

should have an indwelling arterial line for continuous blood pressure monitoring. This monitor would be otherwise unnecessary in a healthy parturient. Assessing blood pressure even every minute by an automated blood pressure cuff is simply not sufficient and impractical. Studies to prevent hypotension in parturients are important, but this regimen seems to have risks that outweigh its benefits. The treatment should not be worse than the disease.

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In Reply:—Dr. Beilin does not consider the use of phenylephrine infusions to be justified given the “minor” consequences of hypotension during spinal anesthesia for cesarean delivery. We disagree. One should not underestimate the importance of hypotension and its prevention. Dr. Beilin contends that serious adverse effects from hypotension are “almost nonexistent.” However, history warns us that major complications can indeed occur when hypotension is inadequately managed.¹ Dr. Beilin cited several articles^{2–4} to support his contention, but careful reading of these reveals somewhat different messages. Macarthur² reported that “several reviews of maternal anesthetic deaths identified inadequately treated maternal hypotension as the major source of spinal anesthesia’s morbidity and mortality.” Desalu and Kushimo³ attributed low Apgar scores in some neonates in their study to relatively long durations of hypotension and stated that hypotension should be avoided in pregnant patients. Juhani and Hannele⁴ emphasized the high incidence of minor complications and recommended that “hypotensive periods should be prevented.” Surprisingly, Dr. Beilin suggests that hypotension is not associated with neonatal acidosis or low Apgar scores. Datta *et al.*⁵ showed just such an association more than 20 yr ago. More recently, concern has been expressed that spinal anesthesia depresses fetal pH and base excess.⁶ We believe the most important cause of this is hypotension and the way that it is treated.⁷

Dr. Beilin trivializes the seriousness of nausea and vomiting. Nausea and vomiting can cause significant distress to the patient and can interfere with surgery.⁸ We regard its prevention as an important clinical indicator of quality of care. Examination of closed claims has emphasized the prominence of “minor” injuries including emotional distress in obstetric anesthesia cases,⁹ and thus, there may also be medicolegal implications. Pulmonary aspiration has occurred;⁹ that’s pretty dangerous.

Dr. Beilin describes treatment of hypotension as “straightforward.” The large volume of research dedicated to this subject argues otherwise. There remain major controversies over the choice, dose, timing, and methods of administration of vasopressors and fluids. Dr. Beilin implies that it is sufficient to wait for hypotension to occur and then treat it with small boluses of ephedrine. It was the inadequacy of this very approach that several years ago stimulated us to direct research toward finding a better way.¹⁰

We make no excuses for our aggressive approach to the prevention of hypotension. Our work indicates that this provides the best outcomes for mother and baby.¹¹ Although, as stated in our article, we do advocate some caution with phenylephrine infusions because of the potential for blood pressure to transiently increase above baseline, we disagree that use of direct intraarterial blood pressure monitoring is necessary when using this technique in healthy patients. From our experience^{11–15} of many years of using infusions of α agonists in many hundreds of patients, we have found measurement of noninvasive blood pressure every minute until delivery together with continuous monitoring of heart rate to be quite sufficient and, contrary to Dr. Beilin’s opinion, quite practical. We have not found small transient increases in blood pressure and relative slowing of maternal heart rate to be harmful. Arguably, this is a safer

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physiologic state than profound vasodilation with marked tachycardia, the likely alternative. Furthermore, in clinical practice, with continuous heart rate monitoring and the freedom to titrate the phenylephrine infusion without the strict constraints of a study protocol, hypertension is less of a problem.

No treatment is perfect. But make not the mistake of underestimating the disease.

Warwick D. Ngan Kee, M.B.Ch.B., M.D., F.A.N.Z.C.A., F.H.K.A.M.,* Kim S. Khaw, M.B.B.S., F.R.C.A., F.H.K.A.M., Floria F. Ng, R.N., B.A.Sc. *The Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, Hong Kong, China. warwick@cuhk.edu.hk

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Better Methods for Meta-analysis Available

To the Editor:—Recently, Wu *et al.*¹ published a systematic review comparing the treatment of postoperative pain by intravenous patient-controlled analgesia (PCA) *versus* epidural analgesia—either patient-controlled epidural analgesia (PCEA) or continuous infusion epidural analgesia (CEI), usually with a local anesthetic-opioid mixture. The literature search discovered 50 articles. Using visual analog scale measurements of pain as the primary outcome, the meta-analysis used a fixed effect analysis of variance (ANOVA) to compare the treatment groups—PCA *versus* PCEA and CEI. The average visual analog scale values for all data (mean \pm SD) were 3.2 ± 1.6 *versus* 2.1 ± 1.3 ; this difference—without good reason—of publications in languages other than English.² Empirical research has shown that under some circumstances, trials not published in English demonstrated statistically significant results less often³; other research on language bias in systematic reviews concluded that exclusion of non-English language trials had shown unpredictable consequences on the summary statistics of a meta-analysis.⁴ The authors report no reasons for limiting their literature search to the English language. They should reconsider their exclusion of trials in other languages.

The authors specially limited their literature search to the English language, but some non-English research reports otherwise qualifying for inclusion were incidentally identified. The authors report that inclusion of five such studies would not have changed the meta-analytic results. Current recommendations for performance of systematic reviews specifically discourage exclusion—without good reason—of publications in languages other than English.² Empirical research has shown that under some circumstances, trials not published in English demonstrated statistically significant results less often³; other research on language bias in systematic reviews concluded that exclusion of non-English language trials had shown unpredictable consequences on the summary statistics of a meta-analysis.⁴ The authors report no reasons for limiting their literature search to the English language. They should reconsider their exclusion of trials in other languages.

The numbers of patients reported in the 50 trials were 1,625 (PCEA and CEI) and 1,583 (PCA), whereas the numbers of observations included in the ANOVA of overall data were 7,744 (PCEA and CEI) and 7,666 (PCA). This difference in the number of observations *versus* the number of patients is the consequence of including visual analog scale scores obtained at multiple times in each patient. These multiple observations in each patient are not considered independent variables. The inclusion of multiple observations has been denoted as a “unit of analysis” error.⁵ The likely consequence of a unit of analysis error is a spurious precision in the calculation of SD. The authors should restrict their meta-analysis to the observations obtained independently; this can be done simply by dropping all analyses using “overall” and “all” data in table 2.¹

The authors chose ANOVA as the statistical method for comparing PCEA and CEI *versus* PCA. ANOVA is a method for hypothesis testing. The main emphasis in a systematic review is the effect measure. The effect measure is a single number that contrasts the treatments; statistically, this is parameter estimation, not hypothesis testing. Because the visual analog scale score may be considered approximately a continuous variable, the relevant effect measure is the difference in mean

values—also known as the weighted mean difference.⁶ By contrast, the ANOVA results presented show the mean values for each treatment group. The meta-analysis should be redone using the weighted mean difference effect measure. The calculation of a summary effect measure is accompanied by statistical tests of heterogeneity. The identification of heterogeneity may encourage the use of a different statistical model, the random effects model. The fixed effect ANOVA reported in this study does not allow identification of heterogeneity among the 50 studies. Finally, with the use of an effect measure, the results should be presented in a Forest plot.⁷ This allows the inspection of the effect measure for each individual trial as well as the summary value of the effect measure; heterogeneity may become more easily recognizable. The Forest plot also makes clear the statistical significance of both the individual studies and the summary effect measure. For a weighted mean difference effect measure, if the lower and upper boundaries of the 95% confidence interval do not bound the zero value, the estimate of the effect measure is declared to be statistically significant.

Although this systematic review was not created for the Cochrane Collaboration, the methods for systematic reviews and meta-analysis presented in their publications provide a rigorous guide for this research.⁸ The authors should reconsider several of their experimental methods that may have produced biased inferences. It is possible and to be desired that the conclusions of a revised systematic review will be unchanged from the current version.

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David C. Warltier, M.D., Ph.D., acted as Handling Editor for this correspondence. The above letter was sent to the authors of the referenced article. The authors did not feel that a response was required. —David C. Warltier, Section Editor

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Another Cause for Ventilator Failure

To the Editor:—Recently, a 44-yr-old woman came to the operating room for the resection of a liver mass. After induction of general anesthesia, her trachea was intubated, and the patient was placed on mechanical ventilation using a previously checked Draeger Fabius GS anesthesia machine (Draeger Medical Inc., Telford, PA). There were no problems with mechanical ventilation. Approximately 10 min later, the mechanical ventilator stopped working, and the anesthesia machine monitor display reported a ventilator failure. We continued to ventilate the patient using manual ventilation.

In looking for the cause of the ventilator failure, we found a plastic cap lodged under the lower rim of the mechanical ventilator piston (fig. 1). Although it is possible for this cap to have entered the ventilator housing before the start of the case, we hypothesize that the cap rolled under the lower rim sometime after the institution of mechanical ventilation.

The Draeger Fabius GS anesthesia machine mechanical ventilator is housed within a see-through compartment that can be opened by simply swinging it out. This creates an entry route for objects to fall into the ventilator compartment. The ventilator operates using a piston driven by a motor and ball-screw arrangement. A light-activated position sensor on the ventilator signals the control board when the piston has reached its lower limit. When this does not occur, the zeroing position is invalid, and the ventilator will not work. This is known as error code V002 in the Draeger Fabius GS reporting nomenclature.

There is no mechanism to lock the ventilator compartment in the closed position. Furthermore, the auxiliary oxygen source is mounted under a swing-out door. Therefore, the compartment is easily opened under a variety of circumstances, such as pulling on oxygen tubing connected to the auxiliary source.

Small objects, particularly plastic caps, are ubiquitous in the operating room. The upper shelf edge in the Draeger Fabius GS lies just above



Fig. 1. Plastic cap under the mechanical ventilator piston.

the opening created when the ventilator compartment is swung out. One can easily envision how small objects can find their way into this chamber. After discussing the case with our biomedical engineers, they reported that they have previously retrieved a few small objects from these ventilator compartments.

Considering the importance of mechanical ventilation in an anesthesia machine, equipment manufacturers must find a way to prevent these incidents. It is ironic that such an expensive and vital piece of equipment can be totally disabled by a simple plastic cap.

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Shoulder Restraints as a Potential Cause for Stretch Neuropathies: Biomechanical Support for the Impact of Shoulder Girdle Depression and Arm Abduction on Nerve Strain

To the Editor:—Stretch-induced neuropathies of the brachial plexus and median nerve are the second most prevalent perioperative neuropathies. In 2002, we demonstrated that movements that elongate the nerve bedding, such as shoulder girdle depression and wrist extension, significantly reduce the available range of elbow extension in healthy subjects in a brachial plexus tension test position.¹ Although an increase in strain in the brachial plexus and median nerve was the most plausible explanation, we could only speculate that this was the underlying mechanism for the reduced range of motion.

Because shoulder girdle depression and abduction of the arm greater than 90° have been associated with stretch-induced perioperative neuropathies, we measured strain in the median nerve in three em-

balmed undisturbed male cadavers in four different arm positions: arm by the side, without shoulder girdle depression (1, reference position) and with shoulder girdle depression (2), and in 90° arm abduction without depression (3) and with depression of the shoulder girdle (4). Because insertion of strain gauges in the brachial plexus requires excision of several structures that may alter nerve biomechanics, we decided to insert miniature linear displacement transducers (Differential variable reluctance transducers; Microstrain, Burlington, VT) into the median nerve at the level of the humerus and also just proximal to the carpal tunnel where the nerve runs relatively superficially. Mean values representing the change in strain relative to the strain in the reference position are reported. Because there is at least mild tension in a peripheral nerve in most positions, the strain gauges were inserted with the nerve under some tension. Therefore, it was impossible to calculate absolute strain values. Electrogoniometers (Biometrics, Black-

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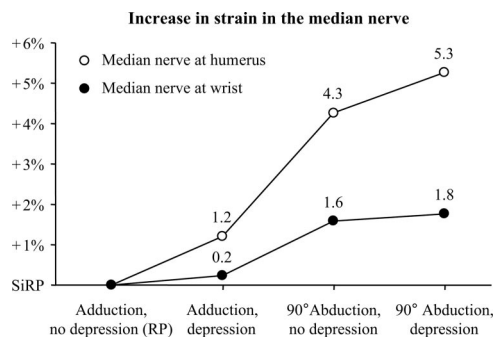


Fig. 1. Increase in strain in the median nerve at the level of the humerus (○) and wrist (●) in four different arm positions: with and without abduction of the arm, and with and without depression of the shoulder girdle. In all four positions, the elbow was maintained in extension (170°). RP = reference position; SiRP = strain in the median nerve in the reference position (anatomical position).

wood, Gwent, United Kingdom) were used to guarantee that the position of the elbow was maintained at 170° extension throughout the experiment and the wrist in a neutral position (180°). The mean available depression, measured at the acromion, equaled 17 mm when the arm was by the side and 10 mm with the arm abducted. Excursion of the median nerve in relation to its surrounding structures was measured at both sites during arm abduction. A digital caliper was used. The Anatomy Ethics Committee of the School of Biomedical Sciences of The University of Queensland, Brisbane, Australia, approved the study.

Figure 1 demonstrates the increase in strain in the different positions. As anticipated, strain in the proximal part of the median nerve increased with shoulder girdle depression, both with the arm by the side (+1.2%) and with the arm abducted (+1.0%). In addition, an increase in strain in the median nerve at the level of the wrist could be observed in one cadaver (+0.6%). In all cadavers, arm abduction resulted in a large increase in nerve strain at the level of the humerus (+3.1%) and a relatively large increase at the level of the wrist (+1.4%). This substantial increase was to our surprise as we allowed the physiologically coupled movement of shoulder girdle elevation to occur during arm abduction. It also demonstrated that strain is transmitted well beyond the site at which the nerve bed is elongated. Abduction of the arm resulted in a proximal excursion of the median nerve of 11.8 mm at the humerus. The nerve movement at the wrist was too small to quantify.

It is well documented that strain may impair several processes, such as microcirculation, axonal transport, and nerve conduction. Animal models have demonstrated that 5–10% elongation results in impaired blood flow.² At 11% strain, axonal transport is inhibited,³ and the amplitude of the action potential is reduced by 70% after a 1-h sustained elongation of 6%.⁴ There is only indirect evidence, but it is to be expected that even lower strain values can affect these processes when blood pressures are reduced. Hypotension occurs frequently during anesthesia and surgery.

Although there is evidence that the magnitude of nerve strain obtained in cadaveric experiments is meaningful,⁵ extrapolation of these values should be made with caution. Our findings may be an underestimation of the true strain increase because the available range of shoulder girdle depression was limited as a result of embalment. In addition, the increase in tension in the brachial plexus with shoulder girdle depression is probably larger than the increase we recorded at the level of the humerus. There is ample evidence that the largest increase in strain occurs nearest the site of nerve bed elongation.⁶ Finally, it is important to realize that the reported strain measures are increases in strain, not absolute values. Because the strain in the reference position to which values were normalized was not zero, absolute strain values are higher than the reported increases. Therefore, the cumulative strain recorded in this experiment may be of a magnitude that impairs microcirculation, axonal transport, and nerve conduction.

The findings of this study provide experimental support for the experientially based recommendations regarding positioning in anesthesia and surgery. It supports the guidelines to minimize the use of shoulder restraints and to monitor the position of the shoulder girdle rigorously if restraints must be used. However, because we observed a substantial increase in strain with 90° abduction, the recommendation to not exceed 90° abduction may even have to be adjusted to a more conservative guideline to further limit the occurrence of stretched-induced perioperative neuropathies. However, the substantial increase in strain associated with movements that are well within physiologic ranges and the fact that strain is transmitted well beyond the site of nerve bed elongation strengthen our previous statement that even when the positioning of all upper limb joints is carefully considered, complete prevention of perioperative neuropathy seems almost inconceivable.¹

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Lidocaine Lollipop for Awake Fiberoptic Bronchoscopy

To the Editor:—Numerous techniques and maneuvers have been described to anesthetize the upper airway in preparation for awake

tracheal intubation, notably the nerve block techniques to the superior laryngeal or the glossopharyngeal nerves as well as the topical application of a local anesthetic, in the form of a gel, spray, or inhaler.¹ The current report describes the efficacy of a lollipop containing 150 mg lidocaine HCl for providing upper airway analgesia for patients under-

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going awake fiberoptic bronchoscope (FOB) tracheal intubation or direct laryngoscopy.

After extensive search through Medline and multiple other databases about the stability of lidocaine HCl salt, a lidocaine lollipop (LL) was developed in collaboration with the pharmacy at the American University of Beirut. Fifty grams of white sugar was heated until liquefied; an equal amount of maple golden syrup was slowly added. For each lollipop, 3 ml of this mixture was poured into a small cylindrical container, to which 150 mg lidocaine HCl salt was added and stirred. As the temperature cooled down and before the mixture solidified, a small plastic stick was plunged at one end for holding the LL. The ready-to-use LL was then labeled and stored in a refrigerator.

The protocol used was approved by the internal review board, and informed consent was obtained from all participants. Exclusion criteria consisted of any history of allergic reaction to local anesthetics, diabetes, or risk for aspiration of gastric contents. All participants had noninvasive serial blood pressure measurements, pulse oximetry, and continuous electrocardiographic monitoring. A total of 45 patients aged 25–78 yr, with American Society of Anesthesiologists physical status I–III, scheduled to undergo elective surgery and requiring general anesthesia and tracheal intubation were recruited. Premedication consisted of 5 mg oral diazepam and 0.2 mg intramuscular glycopyrrolate. All patients were given the LL on arrival to the holding area. The LL was easily consumed by all patients in 8–17 min. Its taste was described as good in more than 80% of patients and acceptable in the remaining participants. The onset of analgesia as depicted by sensation of tongue numbness was reported within 1–2 min.

After finishing the whole LL, and without any additional sedatives, patients were transferred to the operating room. Thirty of the 45 patients underwent awake FOB intubation. A single anesthesiologist introduced a No. 80 Berman intubating oral airway, advanced the FOB (3.8 mm Olympus LF2; Olympus Corporation, Lake Sweeney, NY) to the level of the vocal cords, and injected 2 ml lidocaine HCl, 2%, *via* the working channel to anesthetize the vocal cords and the trachea. The FOB was then advanced into the trachea, and the endotracheal tube was slid over the insertion cord of the FOB into the trachea.

In case of inability to perform FOB-guided intubation, 1 mg midazolam and 1 µg/kg fentanyl were administered intravenously, and then the FOB intubation was reattempted.

Tracheal intubation using the FOB was easily performed in 93.4% of

patients with minimal or no discomfort, with no associated hemodynamic changes, and without the need for additional sedation. Furthermore, the incidence of gagging and discomfort that warranted additional sedation was observed in only 6.7% of patients, as compared with the reported 9.5% for topical analgesia, 10.5% for nerve block techniques,² or 8% for combined nerve block and topical anesthesia techniques.³

In the remaining 15 patients, an awake direct laryngoscopy in an attempt to visualize the vocal cords was performed. General anesthesia was then administered whether direct laryngoscopy and vocal cord visualization were successful or not. The incidence of gagging and failure of direct rigid laryngoscopy was significantly higher than that observed during FOB (46.7% *vs.* 6.7%, respectively). This may be due to the higher number of pressure receptors recruited during awake direct laryngoscopy than during the awake FOB-aided intubation.

In conclusion, the LL containing 150 mg lidocaine may provide a simple, noninvasive, hands-free, effective technique for awake FOB-aided tracheal intubation. The observed effectiveness of the LL technique could be explained by the continuous release of lidocaine from sucking the LL, in addition to swallowing of the saliva mixed with the local anesthetic. This allows for the homogenous spread of the local anesthetic, not only to the mucosa of the oropharynx, but also to the posterior third of the tongue, the area that contains the deep pressure receptors responsible for the gag reflex.¹

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(Accepted for publication January 25, 2006.)

Perinatal Diagnosis of Malignant Hyperthermia Susceptibility

To the Editor:—The presymptomatic diagnosis of malignant hyperthermia (MH) susceptibility is based on the *in vitro* contracture test in Europe and the caffeine-halothane contracture test in North America. Both tests are invasive in requiring an open muscle biopsy undertaken in a specialized center. There are 23 MH investigation centers in Europe, but testing facilities are limited to only 6 centers in the United States.^{1,2}

The locus of the ryanodine receptor of skeletal muscle on chromosomes 19q 13.1 has been shown to link to MH, and several mutations have been identified on this gene.³ Molecular genetic diagnosis of MH susceptibility in persons from MH families with identified mutations in the ryanodine receptor gene has recently been introduced.^{1,4}

A 29-yr-old woman contacted our MH investigation unit to be tested for MH susceptibility. Being pregnant, she was concerned about a possible cesarean delivery because she had a history of a clinical MH episode. She underwent adenotomy as a 6-yr-old child in 1982. The clinical and laboratory findings on the clinical grading scale for MH episodes ranked the episode in the highest category.⁵

The child was discharged without permanent sequelae on the 11th postoperative day. In 1986, MH testing became available in Switzerland, and an open muscle biopsy and *in vitro* contracture test were performed on her parents rather than on the child, because she did not consent to being tested. Although the father was MH negative, the mother was MH susceptible. As part of our research program, the frozen muscle biopsy of the mother was subsequently investigated for MH-associated mutations in the ryanodine receptor gene. Mutation G2434R, known to be causative for MH, was identified. Therefore, the patient contacting our center was tested for this mutation and found to be carrier of G2434R. She agreed to have the umbilical blood of her baby taken at labor, to have the newborn assessed for MH susceptibility. Vaginal delivery was uneventful, with the mother receiving lumbar epidural analgesia with 0.12% ropivacaine and 2 µg/ml fentanyl. Umbilical cord blood was collected in an EDTA tube and sent to the Swiss MH investigation unit. DNA was automatically extracted using the MagNA Pure DNA isolation kit I (Roche Diagnostics, Rotkreuz, Switzerland). Exon 45 of the ryanodine receptor gene was amplified by polymerase chain

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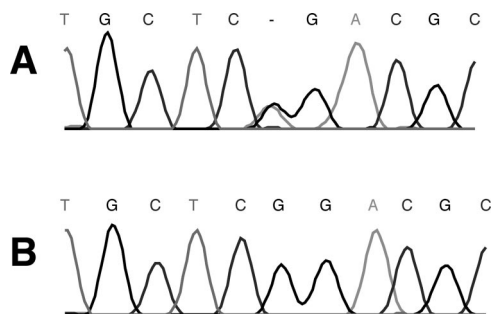


Fig. 1. The patient sample was analyzed by automated sequencing for mutation G7300A in exon 45 of the ryanodine receptor gene, leading to a substitution of glycine for arginine at position 2434. The base pair ambiguity is shown in *A* in the middle of the sequence (-), where two peaks, one for adenine and one for guanine, represent the mutated and wild-type allele. A wild-type control sample is represented in *B*.

reaction using the following primer set: forward: AGA ACG CCA ATG TGG TGG T; reverse: CTG CAT GAG GCG TTC AAA G. Presence of the mutation was proven by an automated sequencing technique (Applied Biosystems, Rotkreuz, Switzerland) and is shown in figure 1. The results were discussed in detail with the mother, and she was given a warning card about the MH status of her newborn son.

This is the first report of MH susceptibility in a newborn by molecular genetic testing of umbilical cord blood. Although knowledge of MH status may be of lesser importance during daily life, it is valuable to confirm or exclude MH susceptibility before surgery in individuals from families with known MH susceptibility. Although elective procedures can be performed during either regional anesthesia or intravenous anesthesia without triggering agents, patients are likely to be exposed to inhalation agents or succinylcholine during emergency or obstetric interventions, and volatile anesthetics are preferred in pediatric anesthesia, because venous access can be established after induction of anesthesia.

Muscle biopsy and contracture testing must be performed in specialized centers and may not be readily available. Most MH centers do not perform biopsies in infants and children because of the limited availability of skeletal muscle. Therefore, the MH status of this newborn would have remained unknown for at least the first decade of his life.

This report emphasizes some significant points:

- Compared with muscle biopsy, sampling for molecular genetic investigations is much easier, and collected tissue can be transported to the center by regular mail. Sampling of umbilical blood is noninva-

sive. A possible concern might be the potential contamination of umbilical cord blood with maternal nucleated cells. However, the concentration of maternal cells was found to be 10^{-4} to 10^{-5} times lower than neonatal nucleated cells,⁶ and therefore, the identical signal intensity of both alleles in our analyses represent the neonatal MH mutation. For verification, we excluded contamination with maternal DNA by short tandem repeat profiling.

- The genetics of MH are complex, because this disease shows substantial locus and allelic heterogeneity.⁷ More than 40 mutations have been identified in the ryanodine receptor, and not all have been proven to be causative of MH. A careful selection of patients eligible for genetic testing of MH susceptibility must be made on the basis of family history and molecular genetics, as well as results of *in vitro* contracture tests, to prevent unnecessary and costly genetic investigations.
- Every pregnant woman with a self or family history of MH and an identified MH-causative mutation should be offered the option of molecular genetic investigations of umbilical cord blood.
- A positive test result confirms and hence avoids uncertainty about MH susceptibility in the newborn.
- It is important to note that because of the heterogeneity of MH and according to the guidelines,¹ individuals will still need to have a muscle biopsy to confirm their MH status in case of a negative genetic test.

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Safe, Low-technology Anesthesia System for Medical Missions to Remote Locations

To the Editor:—The most important challenge an anesthesiologist faces on a medical mission in a developing country is to provide safe and effective care. Typically at these locations, one finds an old anesthesia machine. On our recent trip to Shimla, India, we found a Boyle anesthesia machine with a Goldblat halothane vaporizer. The local anesthesiologist was very comfortable with its use. Only one team member (H.J.K.) had ever seen and used such a machine, some 40 yr

ago during his stay in the United Kingdom. The younger anesthesia team members had seen similar machines on other mission trips but declined to use them. Older anesthesiologists are a diminishing breed, whereas younger ones may fear the challenges of the past. There are no reports in the literature that have specifically addressed this problem. We decided to explore the possibilities.

In an ideal world, it would be prudent to send a scout team to the mission location beforehand to check on the availability of equipment and supplies, but our medical mission runs on a very restricted budget and cannot afford such an expense. Instead, we decided to develop a

The volunteer missions were funded by World Missions Possible, Pearland, Texas.



Fig. 1. Anesthesia circuit (oxygen tank not in the picture).

self-sufficient system with which all team members would be familiar and comfortable. Over time, our efforts have evolved into a sophisticated but low-technology approach to the problem of delivering safe anesthesia without a modern anesthesia machine.

At a first consideration, all medical facilities throughout the world where surgical procedures are performed seem to have large (size H) oxygen tanks available. This is the only item our system requires from the host country. Before our arrival, we arrange for a minimum of two H tanks for each operating room along with compatible gas tank regulators and low flow meters.

The equipment we bring is listed here: (1) a standard Compressed Gas Association oxygen regulator for the H tank along with a flow meter that delivers up to 10 l/min oxygen (usually, the host country's oxygen tanks do not have the standard threads, so we use the one provided by them); (2) standard plumber's sealing tape to obtain an airtight seal for the tank threads; (3) two pieces of standard suction tubing with universal connectors (Cardinal Health, McGaw Park, IL); (4) sevoflurane (Sevotec) and halothane (Fluotec) vaporizers together with 23-mm inlet and outlet adapters (General Anesthetic Services, Bridgeville, PA) to attach to the suction tubing (we routinely use sevoflurane, but we find it advantageous to have a halothane vaporizer because, in case of a supply problem, halothane is still readily available in developing countries, whereas sevoflurane is not); (5) portable disposable sealed carbon dioxide absorber (KAB 001; King Systems Corporation, Nobelsville, IN); (6) breathing circuits, Adult and Pediatric Ultra Flex, latex free (King Systems Corporation); and (7) an Ambu bag for use if there is any unforeseen problem.

The system can be assembled in less than 5 min. Segments of suction tubing connect the oxygen tank to the vaporizer and the vaporizer to the fresh gas inlet of the carbon dioxide absorber. The breathing circuit and the rebreathing bag are attached to the designated ports on the absorber. Anesthesia can proceed with either spontaneous or hand-controlled ventilation. The carbon dioxide absorber itself has a round bottom; hence, for ease of use, we put it into a small plywood box to keep it upright. A support stand is available from the supplier, but we find the wooden box more economical and easier to transport across the world. The absorber has an exhaust port to which we attached a standard long green corrugated plastic hose to carry the waste gases out of the operating room. Figure 1 is a picture of the circuit.

It is important that the team members familiarize themselves with assembling the system and are comfortable with its use before leaving the home base. This precaution also assures that the correct size of adapters, tubing, and hose are available.

No system, however simple, is without some drawbacks. Oxygen tanks in different countries have different color codes and the gas content must be verified before use. Our system uses freestanding vaporizers that are physically very stable and are clearly labeled in red with a notice to be kept upright when charged. The team is well aware that an accidentally tilted vaporizer will deliver an increased concentration of the anesthetic agent. Great care must also be taken when refilling the vaporizer, which may not have an indexed filling port. There is no safety measure aside from full diligence in filling each vaporizer with the proper agent. At the end of surgery each day, all vaporizers are firmly secured to prevent accidental tipping. In use of the carbon dioxide absorber, it is well recognized that the inspiratory or expiratory valve mechanism may malfunction. Monitoring of end-tidal carbon dioxide levels during anesthesia alerts the anesthesiologist if there is a developing problem.

We have used Propaq (Welch Allyn, Beaverton, OR) transport monitors, which provide the standard heart rate, blood pressure, electrocardiogram, temperature, arterial blood oxygen saturation, and concentration of end-tidal carbon dioxide. We follow the guidelines set by the American Society of Anesthesiologists in monitoring our patients. We have no plans for changing the Propaq monitors in the foreseeable future, but someone starting from the beginning may wish to consider other monitors, such as the GE Health Care Datex-Ohmeda Cardiopac/5 (Datex-Ohmeda, Madison, WI), which also monitors inspired and expired oxygen and inhalational agents. This would act as an added safety margin, because it confirms the veracity of the oxygen tank and the dialed concentration of the anesthetic agent.

On a recent mission to Shimla, India (World Missions Possible, Pearland, TX), we administered 92 anesthetics with three surgical teams over a period of 5 days. The surgeries included repair of cleft lip and palate (some children had a combined procedure) and plastic repair of scars and adhesions in severely burned children. We used three absorbers during the period. The work day was never longer than 10 h.

Most medical missions are on a limited budget. With this in mind, we found it possible to reuse the carbon dioxide absorber. There have been many reports in the literature showing that exposure of volatile anesthetics to desiccated carbon dioxide absorbents may result in exothermic reactions leading to production of toxic substances and a fire hazard in the breathing circuit. We made a 3/4-inch hole with a trephine on one side of the absorber, removed the spent absorbent granules, and replaced them with fresh Medisorb granules (Datex-Ohmeda), which are safer than the old Baralyme granules. We placed a regular wine cork in the hole, which makes a very tight fit. The carbon dioxide absorber was back in service, and we experienced no problems. Alternately, a reusable carbon dioxide absorber (KAB 002; King Systems Corporation) is available. The one-way valve mechanism and the plastic pressure release valve (the pop-off valve), the two most important parts, are the same in both carbon dioxide absorbers, and the only difference is that in the reusable variety the manufacturer makes the hole on the side and provides a rubber stopper. Therefore, we decided to use the KAB 001 absorber and make the necessary adjustments ourselves while reducing our cost basis. Figure 2 shows the two absorbers.

We found that this system has many advantages. Most importantly, it requires no sophisticated instrumentation and is low-technology, easily portable, lightweight, independent of electricity supply, comparatively inexpensive (less than \$1,000 to outfit an operating room including the vaporizer, which once acquired is not a recurring expense), and easy to assemble and use.

The system we have described here has worked very well for us, and we have experienced no anesthesia-related complications. However, under normal conditions, anesthesia morbidity and mortality are indeed very low, and we realize that our 92 successes do not support a blanket statement. Nevertheless, we feel confident that the simplicity, ease of use, and portability of our system will



Fig. 2. The disposable absorber with a corked hole made by us on the left side and a reusable absorber with a rubber stopper from the manufacturer on the right side.

prove our point. During the past two decades, multiple groups have participated in humanitarian medical missions throughout the world. It stands to reason that variations on the theme we describe must have been used by others, but there are no published reports to the effect. We very much hope that our report will be of help to a new group about to undertake a medical mission to an underdeveloped area. This system might also be useful in emergency conditions in the field or for makeshift operating rooms. We recommend this system for the administration of anesthesia in remote areas and developing countries or anywhere where a functioning anesthesia machine is not available.

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