Is the Parker Flex-Tip Tube Really Superior to the Standard Tube for Fiberoptic Orotracheal Intubation?

To the Editor—Dr. Kristensen1 has concluded that during intubation with the use of a flexible fiberscope, the use of the Parker Flex-Tube results in a significantly lower rate of repositioning and repeated attempts at passing the tube into the trachea, compared to a standard endotracheal tube. We believe it would be more appropriate to conclude that the Parker tube is better only when the standard tube is improperly oriented during passage. Dr. Kristensen reported that once the standard tube was rotated counterclockwise by 90 degrees, its improper orientation during passage. Dr. Kristensen reported that once the standard tube was rotated counterclockwise by 90 degrees, its success rate improved to 26 out of 38 attempts.1 This was essentially the same as the success rate (27 out of 38 attempts) of the Parker tube1 and is consistent with our experience with the standard tube. Why not simply start with the standard tube rotated counterclockwise by 90 degrees? The Parker tube requires a higher cuff pressure,1 which, in our opinion, makes it less desirable.

The simple technique of rotating the standard tube counterclockwise by 90 degrees during the first attempt along a fiberoptic bronchoscope has been our standard practice for years, thanks to a suggestion by Katsnelson et al. in 1992.2 Eighteen years ago, Cossham3 proposed rotating a standard tube counterclockwise by 90 degrees to facilitate passage along a gum-elastic bougie, and in 1990, Dogra et al. demonstrated convincingly the usefulness of this technique.授予, this technique may not be widely appreciated, perhaps because the gum-elastic bougie is not used in some parts of the world and the use of fiberoptic bronchoscopy is infrequent.1 As such, Dr. Kristensen's study1 should help to popularize this important “trick.”


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To the Editor—The article by Dr. Kristensen highlights a problem often faced by practitioners when using a fiberoptic scope to intubate the trachea, i.e., resistance to passage of the endotracheal tube.1 This is usually attributed to the endotracheal tube being caught on structures of the supraglottic airway.2–4 We are curious to know how the bevel of the endotracheal tube was oriented as it was passed over the scope into the trachea. Dr. Kristensen states, “the tube was mounted onto the fiberscope with the concavity of the curvature facing the side of the maneuver lever.” We are unsure how this translates to the orientation of the bevel and the Murphy tip of the endotracheal tube, relative to the tip of the fiberoptic scope, and whether this relationship was maintained during passage of the endotracheal tube. In our experience, this orientation of the leading edge bevel is the most important determinant of successful passage of the endotracheal tube. The author’s figure 1 does little to clarify what the initial orientation of the bevel was and whether or not it was the same for both types of endotracheal tubes that he studied. The 90-degree counterclockwise rotation of the endotracheal tube described by Dr. Kristensen as the first maneuver to improve passage after initial failure has been advocated by others.2,5 This was originally proposed to change the “usual” orientation of the endotracheal tube, i.e., the bevel facing left, to an orientation in which the bevel is facing down2 (fig. 1). This bevel down orientation appears to improve the success of oral fiberoptic tracheal intubation by allowing the endotracheal tube to slide past the potentially obstructing right arytenoid cartilage.3 If nasal intubation is used, the endotracheal tube should be turned 90 degrees clockwise from its usual orientation so that the bevel is facing up, thus avoiding the epiglottis.3 In our practice, we begin with the endotracheal tube oriented bevel-down for oral fiberoptic intubations and bevel-up for nasal fiberoptic intubations. These simple maneuvers reduce the first-attempt failure rate for passage of the endotracheal tube into the trachea. Close reading of one of the first investigations into improving the passage of the endotracheal tube during fiberoptic intubation reveals that successful oral intubation was achieved in 9 of 11 patients after initial failure when the bevel orientation was changed as we describe.3 We have found this technique of beginning with the bevel in the optimal orientation to be useful for standard polyvinyl chloride endotracheal tubes, straight and preformed Ring Adair Elwyn, i.e., RAE.
(nasal and oral). Thus, special endotracheal tubes, which have an increased cost and may not be available in small sizes, may not be needed to improve the success rate of fiberoptic intubation.

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References


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Fig. 1. The tubes were initially introduced oriented in the “natural” way, meaning with the concavity facing downward toward the lower teeth, tongue, and epiglottis. In this position, the bevel of the standard tube (S) is facing the left side of the patient. The Parker Flex-Tip (PFT) tube has a symmetrical bevel that faces toward the convex side of the tube.

PosterioR Approach to the Sciatic Nerve: Can “Common Sense” Replace Science and Logic?

To the Editor—We read with interest Dr. Franco’s recently published article concerning his new approach to sciatic nerve block.1 We believe there are important omissions in the methodology and interpretation of the results in both stages of the study that substantially limit the applicability of the findings.

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First Stage, Anatomy Laboratory: 1. The author stated that his anatomical data resulted from dissection of 24 sciatic nerves. However, this information came from only 12 cadavers. Because there is no indication that individuals lack bilateral symmetry of their sciatic nerves, in reality, only 12 specimens were evaluated. No presentation
of the inter- or intraindividual variability of these values was provided. The variation reported in the linear distance from the intergluteal sulcus and its variability could be substantially underestimated by combining both inter- and intraindividual data. This error may in part account for the substantial number of subjects (60%) who required multiple attempts to locate the nerve, as well as the significant block failure rate (10%).

2. The author stated that “variation in the hip width reflects soft tissue, not bony differences between the sexes [p 724],” implying an inherent inconsistency of surface landmarks. Further, he stated that gender was not a factor in determining his calculation of the surface anatomical relationship. However, he never indicated how many of his sciatic dissections were from male and female cadavers. The lack of association between gender and surface anatomical characteristics cannot be assumed from the data he presented. Although we concur with his observations with respect to the consistent relationship between the sciatic nerve and bony structures, it is difficult to accept the extrapolation of this spatial information to the use of a surface anatomical approach to the nerve block that relies on a predetermined distance (10.1 ± 0.2 cm), primarily because variations in the contour of the buttocks are likely to alter these surface characteristics.

### Second Stage, Clinical Stage (doesn’t the author mean Clinical Study?):

The clinical study has substantial methodologic limitations. The limited clinical evaluation (20 cases) fell far short of convincing us of the validity of the author’s stage 1 hypotheses.

1. The endpoint stated by the author as appropriate for local anesthetic injection was “sciatic nerve response” at 0.6 mA. To ensure the highest degree of success with peripheral nerve blocks using neurostimulation technology, the endpoint for injection is an appropriate evoked motor response at < 0.5 mA.2,3 In addition, the latency and success of a complete sciatic nerve block depends on the type of motor response obtained via neurostimulation.2,4 The author used the very vague, nonspecific term, “sciatic nerve response.” No attempt was made to categorize whether inversion, plantar flexion, eversion, or dorsiflexion were elicited, nor how the elicited response influenced his latency and success rate.

2. The success of a peripheral nerve block, in terms of sensory anesthesia, is represented by both analgesia and anesthesia, and with regard to motor block, by paresis and paralysis. The latency of block onset is judged by the time taken from the injection of local anesthetic until these parameters are reached. We do not believe that the “time to incision” (patient’s response to a surgeon’s knife) is an appropriate measure of block latency and success. Obviously, according to this definition, a surgeon who scrubs and drapes the surgical field quickly will have a different “latency of onset” for a given patient than one who scrubs and dapes more slowly. The use of this parameter to judge the efficacy of a block in response to surgical incision is not only questionable ethically, but it also lacks scientific validity because it offers little indication as to whether both components of the sciatic nerve (tibial and common peroneal) were actually blocked.

3. The author stated, “Most of the patients were given 1 mg of midazolam plus 50 μg of fentanyl in the OR; some patients received less [p 724].” There is no validity to this vague statement, but it implies that the amount of sedation administered to individual patients was not carefully monitored.

4. The author states, “…a handheld timer (Casio, Japan) was … left running continuously. The time periods were later calculated by subtracting the previous elapsed time from the actual reading [pp 724-5].” It is difficult to imagine that five parameters (time to skin mark; time to sciatic nerve response; time to injection; time to incision; total accumulated time) could have been accurately calculated from a continuously running timing device with the described precision. It would appear that this approach might have involved a great deal of “estimating.” It is not clear if these values represent the total time of all attempts or only the time of the successful attempt. This is not a minor consideration, because in 60% of the cases more than one attempt was required. In addition, no mention was made of what adjustments were made to locate the nerve when the first attempt was not successful. Also, the actual distance from the intergluteal sulcus to the needle entry point at the successful attempt was not reported.

5. The author presented durations of anesthesia and analgesia as 2-4 h after injection in all patients, “but a strong analgesia persisted in some patients up to 24 h later [p 726].” How was this anesthesia and analgesia monitored? How is “strong” analgesia defined? How many patients required rescue analgesics, and in what quantities, during this 24 h? Furthermore, analgesic requirements will vary widely depending on the type of surgery (bunion vs. below-the-knee amputations). Was “strong” analgesia observed in all patients across the board?

6. The author stated, “… the technique was successfully used in adult patients of different gender, height, body habits [habitus?], and ethnic backgrounds, including a 147-cm tall Hispanic woman and a 196-cm tall Caucasian male … [pp 726].” We are not convinced from the present work (or any other) that ethnicity affects sciatic nerve anatomy, particularly with respect to the significant diversity encompassing the descriptors Hispanic and Caucasian. Furthermore, “We could not reach one Hispanic male patient…” (for follow-up evaluation). We fail to see why the patient’s ethnicity needed to be stated with regard to follow-up data collection.

We believe the author’s speculation as to the applicability of his work could have easily been resolved by addressing the issues raised above.

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1. A recently quoted 61% of asymmetry in brachial plexus4 supports the potential for any nerve asymmetry. Although 1 did find no differences in nerve location, I did find asymmetry of the nerve itself (two nerves in one sheath, one on the contralateral side).

2. Studying 12 cadavers (eight female, four male) might not be enough, as the study on 10 cadavers by Sukhani and Candido shows.4

3. The range of measurements was narrow (9.7–10.3 cm); thus “inter- or intraindividual variability” was not a factor.

4. Claiming 60% of “multiple attempts” is misleading. The fact is that
85% of the patients (17 out of 20) needed three attempts or fewer, and all were completed within six attempts, using a single skin puncture in each case. The population was 35% obese (BMI ≥ 30) and 80% overweight (BMI ≥ 25).

5. My technique makes finding the nerve easier, but it does not influence success one way or the other, after the nerve is identified. A 90% success rate is better appreciated after reviewing the ‘failed’ blocks: (i) Obese woman (BMI, 33.5) in whom only an intermittent response could be elicited (needle short for her size). This case could have been thrown out on that basis. An early incision at 16 min proved that the block was still incomplete. What is remarkable about this case is that it ‘failed’ but that the nerve was found at 10 cm in a large buttock. (ii) Young, nervous patient whose block took one attempt. Her ‘discomfort’ 10 min after the incision could have been managed with additional narcotics. However, a successful block was limited to no more than 100 μg of fentanyl.

6. The statement ‘variations in the hip width reflects soft tissue, not bony, differences between the sexes’ is paramount to understand why a block can be performed at 10 cm in both sexes. The contour of the buttocks would indeed alter this measurement, which is why it must be done in a straight line from the midline, disregarding the curvature of the buttocks.

7. The authors would have preferred my ‘Clinical Stage’ to be called ‘Clinical Study;’ but they liked my ‘Anatomy Stage.’ This objection exceeds the intended purpose of the study.

8. The results obtained in 20 patients confirmed my anatomy findings. Currently, with more than 100 blocks performed, my conviction is even stronger. On a few occasions, I have even managed to make more than six attempts to find the sciatic nerve (hersesy!). However, invariably it has been found in close proximity to the original insertion point and no skin reinsertion has been necessary.

9. The type of response and the output at which it is elicited (0.5 mA significantly different from 0.6 mA?) are indeed very important factors but are unrelated to whether the sciatic nerve is located at 10 cm from the midline.

10. I chose the incision (all within 29 min) as the main test for success because it is highly objective, but I understand that others would choose differently. However, calling it ‘questionable ethically’ is not worth a response and makes me wonder whether the authors are truly bringing these objections with a scientific purpose in mind.

11. Those who received successful blocks were not given more than the stated amounts of sedation. Inferring from this that ‘the amount of sedation administered to individual patients was not carefully monitored’ is simply wrong.

12. When the operator was ready, the timer was turned on and was never stopped until the incision. When reaching a preestablished goal (e.g., first sciatic response), the designated person would simply enter the actual reading (e.g., 1:06) on the protocol sheet and continue observing until the next goal was met. The time intervals were clear (or so I thought). ‘First sciatic’ for instance, measured the time to elicit the first sciatic response, obviously accounting for any necessary repositional(s). Later, outside the operating room, the time intervals were easily calculated. The authors’ claim that ‘this approach might have involved a great deal of estimating’ is bad ‘estimation’ on their part.

13. When repositional procedure was necessary, ‘the actual distance from the intersulcus to the needle entry point at the successful attempt was not reported.’ That is right. Because every block was started and completed through a single skin puncture, there was nothing to report.

14. Postoperative analgesia, although important, had nothing to do with my study goal.

15. When I said that successful blocks were performed on a 147-cm tall Hispanic woman and a 196-cm tall Caucasian male, I did not intend to start a discussion on race. I am confident that most of the readers understood that my technique applies to a large and diverse population.

16. Calling this new approach ‘speculation’ is refuted by the now overwhelming clinical evidence and the many anesthesiologists who have tried it with success (communications on file).

In summary, it is reassuring to know that even those who are reluctant to believe that a sciatic block in adults could be performed at 10 cm from the midline, sans geometry, offer no evidence to the contrary other than their disbelief. I would encourage them to try it, understanding that it could become addictive.

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To the Editor—We read with great interest the review article by Park.1 We appreciate the importance the author attributes to the necessity for the anesthesiologist and the cardiologist to work as a team and to the guidelines.

However, we disagree with the author about the risks of percutaneous transluminal coronary angioplasty during the acute phase of myocardial infarction (AMI). When feasible, percutaneous transluminal coronary angioplasty remains the treatment of choice during AMI, resulting in an approximately 85% success rate.2 During AMI, it appears that it is always useful to ‘open’ the occluded coronary artery, even when the patient is in cardiogenic shock. In this setting, coronary angioplasty seems to be the best treatment.3,4 The trend is to perform intravenous thrombolysis as soon as possible, and then coronary angioplasty when the patient can be managed in a center where this technique is available.

The important issue of the patient with AMI who needs noncardiac surgery must be addressed. In any case, AMI should be managed first. Especially when the patient with AMI needs urgent noncardiac surgery, establishing the best strategy will rely on an emergency coronary assessment in a center experienced with primary angioplasty.

If the occluded artery is a large vessel with a large myocardial territory involved, angioplasty and stenting must not be delayed, and any urgent surgery must be done under antplatelet therapy while considering the increased risk of bleeding. If the occluded artery is a small vessel with few myocardial territories at risk, angioplasty is not necessary, avoiding the need for antplatelet treatment and the risk of subsequent bleeding. Close collaboration between the anesthesiologist and cardiologist is warranted during the perioperative period.

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To the Editor:—Dr. Park does us all a service in his review of preoperative cardiology consultation by clarifying the state of the art of preoperative cardiologic interventions and their associated outcomes. At the same time, however, Park’s review perpetuates a common myth: that anesthesiologists need to request, or consider, preoperative cardiology consultation in the many patients who present for elective noncardiac surgery who also have moderate or severe concomitant heart disease. Park’s very own analysis of the perioperative interventional outcome data in fact proves what an exercise in wasted time, money, and effort most preoperative cardiology consultations are. Specifically, he clearly points out the lack of definitive data demonstrating the efficacy of preoperative cardiologic interventions (percutaneous transluminal coronary angioplasty, coronary artery bypass graft surgery) in improving outcomes for patients undergoing noncardiac surgery who have concomitant ischemic heart disease.1 Indeed, he presents data which strongly suggest that some preoperative interventions, such as percutaneous transluminal coronary angioplasty with stent deployment, can be harmful.1 In the case of valvular heart disease, Park presents convincing data to suggest that patients with aortic stenosis may undergo elective surgery safely.1

Fourteen years ago, we found that the major preoperative problem generating a cardiology consultation was ischemic heart disease.2 I suspect that is still true at present. We also suggested that the major reason for obtaining a consultation could only be that the consultant possesses a singular level of expertise over and beyond that of the requesting physician (in most cases, the anesthesiologist).2 In the case of our cardiology colleagues, that level of expertise is related to the immediate treatment of acute coronary syndromes in which the intervention required clearly is outside the expertise and skills of an anesthesiologist. Furthermore, the possible medical treatment of unstable coronary syndromes—potent antiplatelet agents—in and of themselves automatically precludes most elective noncardiac procedures. But let’s be clear, in terms of quantifying perioperative risk and managing that risk, anesthesiologists do not need a cardiologist. Perioperative risk assessment and management is not just within the purview or expertise of our cardiology colleagues; rather, it has a long and honored history within our discipline. Historically, anesthesiologists were one of the first groups of physicians to study perioperative risk and outcomes.3-6

How exactly, then, is the singular expertise of a cardiologist obtained during a preoperative cardiology consultation supposed to “make things better” for the patient or the anesthesiologist? What hidden pearls of wisdom does the cardiologist possess that the anesthesiologist does not? And what pearls of wisdom are going to unequivocally improve perioperative outcome? In the one generally accepted efficacious intervention—the use of perioperative β-blockade—clearly, an anesthesiologist does not need the expertise of a cardiologist to initiate this therapy. After all, the idea that perioperative β-blockade might be efficacious was originally proposed by anesthesiologists.7,8 Anesthesiologists now claim to be perioperative physicians, but being a perioperative physician demands more than just a name change. It demands a behavioral change as well.

In my opinion, what Park’s review has clearly shown is that in terms of perioperative management—other than the management of perioperative acute coronary syndromes—the cardiologist has little to add. Given what we know about altering perioperative outcomes, there are very few instances when a preoperative cardiology consultant will prove useful to the anesthesia-surgical care team. If the overriding principle of preoperative cardiology consultation is “in general, indications for further cardiac testing and treatments [in the perioperative setting] are the same as those in the nonoperative setting,”1+7 then might I suggest that most cardiology consultations are best obtained after the patient has had elective surgery. It is in that setting where our patient’s long-term diagnostic and therapeutic needs are best addressed. As the situation stands currently, there is still much confusion. I am left scratching my head in disbelief at the number of times the response from the cardiology consultant is “Patient cleared for surgery, high-risk, use Swan-Ganz catheter!” Indeed, the emperor has no clothes!

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To the Editor.—I applaud Dr. Park’s conclusion that preoperative cardiology consultations are often unnecessary if the patient is amenable to perioperative β-blockade; however, if β-blockade is contraindicated, I disagree with obtaining a consultation when the American College of Cardiology/American Heart Association algorithm recommends stress testing. Anesthesiologists should be sufficiently knowledgeable to determine the indicated type of stress test. Frequently, the test may be ordered after discussion with the patient’s primary care physician, as the test has long-term patient-care issues. If the test is negative for ischemia at a significant workload (e.g., achievement of 85% of maximum predicted heart rate for exercise or dobutamine echocardiography), then no consultation is indicated. If a stress test is positive for significant ischemia, the cardiologist should be asked whether more invasive testing (coronary angiography) with possible treatment (percutaneous coronary intervention or coronary artery bypass graft) is indicated.

The author also concludes that cardiac consultation is not indicated for patients with severe aortic stenosis (AS) because studies demonstrate no increased risk of surgery if the AS is recognized. However, the studies were small and did not have the power to determine increased risk (Okeefe et al. studied only 23 patients with general anesthesia or spinal anesthesia, and Raymer and Yang’s sample size was adequate to detect only a fourfold increase in risk). Patients with severe AS and any of the triad (angina, syncope, or dyspnea) should be referred for prompt valve replacement. In addition, even if patients with severe AS are asymptomatic, some authors believe that certain patients with stress testing-induced symptoms will benefit from aortic valve replacement. Every patient with a murmur consistent with AS should be sent for echocardiography; if the aortic valve area is less than 1.0 cm², a cardiology consultation should be strongly considered prior to elective surgery to determine if preoperative valve replacement is indicated.

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In Reply.—The comments by Lena et al., Kleinman, and Lustik in response to my review article are appreciated, as they attest to the importance and clinical relevance of the subject of preoperative cardiology consultation. An acute, evolving myocardial infarction (AMI) that Lena et al. refer to would be considered a major clinical predictor in the American College of Cardiology/American Heart Association guidelines. Unless the proposed surgery is an emergency operation for a life-threatening indication, the presence of such a major indicator should lead to a cardiology consultation, as delineated in the review (step 2 in fig. 1 of reference 1). The American College of Cardiology/American Heart Association has published the recommended role of percutaneous transluminal coronary angioplasty (PTCA) and thrombolysis in the setting of an AMI. Class 1 indications for PTCA are as an alternative to thrombolytic therapy in (a) patients with AMI and ST-segment elevation or new or presumed new left bundle branch block who can undergo PTCA of the infarct-related artery within 12 h of symptom onset, or even beyond 12 h with symptom persistence; or (b) patients who are within 36 h of an AMI with ST-segment elevation or new left bundle branch block who develop cardiogenic shock, are younger than 75 yr, and in whom revascularization can be performed within 18 h of onset of shock. I do not disagree with Lena et al. that a patient with an AMI needs immediate attention, in the absence of an overriding consideration of emergency surgery (such as repair of a ruptured abdominal aortic aneurysm), and that PTCA as well as thrombolytic therapy has an important role in the management of such a patient. The relative priority of PTCA and thrombolysis in such a setting is still being worked out.

On the other hand, an emergency operation for a life-threatening indication should take priority even over the management of an AMI. Most true surgical emergencies (e.g., such as a ruptured abdominal aortic aneurysm, perforated viscus, or major trauma) do not permit more than a cursory cardiac evaluation. In such a situation, the anesthesiologist is faced with the challenge of resuscitating the patient and attempting to achieve hemodynamic stability in a way that optimizes coronary perfusion and minimizes myocardial stress. Should the patient survive such an operation, any further necessary coronary intervention may take place postoperatively.

If the proposed surgery is not an emergency but is nevertheless urgent, and if the patient has an AMI, the guideline calls for preoperative management of the major predictor (AMI) by a cardiology consultant. In such a situation, the management strategy chosen for the AMI should consider (1) the increased risk of myocardial infarction and death in patients having noncardiac surgery within 2–4 weeks of coronary stenting or within 3–4 weeks of coronary artery bypass surgery; and (2) how long the urgent surgery may be safely delayed. Close collaboration between the anesthesiologist, cardiologist, and surgeon would be essential.

Although Kleinman’s candid comments about the value (or lack thereof) of preoperative cardiology consultations are appreciated, I do not believe that the data are available yet to unequivocally state that the consultations are “an exercise in wasted time, money, and effort.” I agree that as anesthesiologists, we should have the tools and data to assess the perioperative risk on the basis of the patient’s comorbidities and functional status and the risk of the proposed surgery. In fact, the review advocates that we should not seek a consultation to “determine whether cardiology consultation is indicated, and if so, to take the necessary diagnostic and therapeutic measures.” The need for consultation exists (a) for the management of major clinical predictors such as acute coronary syndromes (as Kleinman agrees); and (b) for additional diagnostic workup that is under the purview of the cardiologists, such as stress testing and cardiac catheterization, when such is deemed indicated based on our initial assessment of comorbidities and functional status and the risk of proposed surgery. Note that in the case of the second indication of consultations, the review advocates periprocedural β-adrenergic blockade, whenever possible, followed by postoperative cardiology follow-up—as Kleinman agrees. Interestingly, the criteria for ordering additional preoperative cardiac testing are being questioned as being too cost-ineffective and leading...
to few additional interventions: Morgan et al.,9 for example, found in a retrospective chart review that when dobutamine stress echocardiography is performed in accordance with the American College of Cardiology/American Heart Association guidelines, only 4.7% of the patients had a positive result. Even when they have a positive stress test, they can have a relatively low perioperative complication rates with the use of β-adrenergic blockade,10–13 further questioning the utility of preoperative testing. The day may yet come when the only indication for preoperative cardiac consultations may be for the management of acute coronary syndromes, as Kleinman suggests, but we must gather more prospective data to support such a practice.

In most practices, performance and interpretation of the stress test is currently under the purview of the cardiologists, unlike what Lustik may be implying. Therefore, after we determine a need for such a test, a cardiology consultant should then be involved in the testing. Aronson et al. have demonstrated the feasibility of intraoperative dobutamine echocardiography14 and, as our collective expertise in intraoperative echocardiography increases, performance of preoperative/intraoperative stress echocardiography may possibly come under the purview of our specialty in the future.

The review notes that the studies by O’Keefe et al.15 and by Raymer and Yang16 suggest that severe aortic stenosis may not be a major clinical predictor which necessitates postponement of all nonemergent noncardiac surgery, so the condition may be worked up and treated first. Indeed, a similar conclusion is reached in the review article by Carabello,15 which Lustik quotes. Admittedly, the studies by O’Keefe et al.15 and by Raymer and Yang16 had small n’s and were retrospective in nature; however, there are no retrospective or prospective studies with results to the contrary (i.e., no studies with data indicating that correction of severe aortic stenosis is needed prior to noncardiac surgery). Certainly, severe aortic stenosis is not a condition to be taken lightly, and patients with severe aortic stenosis should be managed intraoperatively with knowledge of the implications of the pathophysiologic changes associated with aortic stenosis. Moreover, there should be an appropriate postoperative follow-up, so that any indicated intervention may be performed, especially if the patient is symptomatic of angina, syncope, and/or dyspnea.

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To the Editor.—We read with great interest the article by Connolly et al. describing in sheep that isoflurane, rather than mechanical ventilation, caused decreased urinary excretion and increased interstitial fluid volume.1 The authors final comment states “Confirmation of the clinical relevance of these findings requires an evaluation in humans [p 681].”

Several years ago, in a prospective observational study of 466 patients who underwent cardiac surgery, we evaluated the incidence and risk factors of postcardiopulmonary bypass hypoxemia.2 Three anesthetic techniques were used: intravenous midazolam with fentanyl, intravenous midazolam and fentanyl combined with either enflurane or isoflurane. One of the findings of this study was that patients anesthetized with isoflurane had a lower 
\[P_A/O_2\] ratio at 1 and 6 h after postcardiopulmonary bypass—hence the conclusion that the use of isoflurane as an anesthetic agent is a risk factor for postcardiopulmonary bypass hypoxemia. Postcardiopulmonary bypass hypoxemia may be a result of atelectasis or interstitial and alveolar pulmonary edema. At the time, we had no explanation for the different effects of these anesthetics on the resulting hypoxemia and, indeed, did not discuss it in our article. Therefore, we find the report by Connolly et al. enlightening, as it provides a possible explanation for our previous findings.

To prove the effect of the different anesthetics on pulmonary edema formation in humans, there is a need for future studies evaluating extravascular lung water in patients undergoing surgical procedures with a high risk of postoperative lung injury. However, we would also be interested in any information regarding respiratory function that the authors might have gathered.

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Isoflurane Promotes Extravascular Fluid Accumulation in Humans

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To the Editor.—We read with great interest the article by Connolly et al. describing in sheep that isoflurane, rather than mechanical ventilation, caused decreased urinary excretion and increased interstitial fluid volume. The authors final comment states “Confirmation of the clinical relevance of these findings requires an evaluation in humans [p 681].”

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In Reply——We would like to thank Drs. Weiss and Pizov for their comments regarding our recent report.1 The main finding of our study was that isoflurane anesthesia increases extravascular accumulation after a bolus infusion of 0.9% NaCl in normovolemic sheep, and the response was associated with an antidiuresis. In our anesthetized and ventilated experiments, a fractional inspired oxygen tension of 50% was administered with a tidal volume of 10–15 ml/kg and respiratory rates between 10 and 15 per min. In the ventilated protocols, a mean end-tidal carbon dioxide of 36 mmHg was achieved. Arterial blood gases were not routinely measured in these experiments, but mixed venous oxygen saturation measured with a fiberoptic pulmonary artery catheter was not lower than that in the conscious groups. We believe that there was no hypoxemia, but we cannot provide direct evidence for or against pulmonary edema.

Although it was determined in our study that isoflurane alone causes a significant extravascular accumulation of a crystalloid bolus, the specific organ distribution of the fluid was not determined. We were surprised when we first found that isoflurane anesthesia alone could have such a profound effect on acute fluid accumulation; we would again be surprised if this increased fluid accumulation caused significant pulmonary dysfunction. If the extravascular accumulation of 22.5 ml/kg at 3 h after bolus was evenly distributed by organ weight (assuming lung weight is 420 g, body weight is 70 kg, and normal extravascular lung water is 4.3 ml/kg body weight) the extravascular lung water would have increased to only 4.4 ml/kg, or by 3.1%. Most experimental studies have shown the lungs to be remarkably refractory to dysfunctional pulmonary edema in the absence of an inflammatory increase in capillary permeability.2

We certainly agree with Drs. Weiss and Pizov that studies measuring the effects of different anesthetics on extravascular lung water and pulmonary function are needed in humans and animals subjected to fluid therapy in a variety of clinical scenarios. The use of thermal dye dilution monitoring of extravascular lung water and blood gas analysis, along with calculations of changes in plasma volume and total extravascular volume, would be useful in this regard.3

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References

Low Volume Neurolytic Celiac Plexus Block with Computed Tomography Guidance

To the Editor—The use of neurolytic celiac plexus block in the treatment of pain arising from upper abdominal structures is widely recognized by physicians.1 The block has been performed using surface landmarks, fluoroscopy, ultrasound, and with computed tomography guidance.2–4 We present an interesting case in which the celiac plexus was only accessible from the right side because of extensive tumor infiltration. The plexus was blocked successfully from the contralateral side with a very small volume of alcohol.

A 58-yr-old man with metastatic non-small cell lung cancer was hospitalized for the management of severe pain in the left hypochondrium. He was known to have a large (9 × 7 cm) metastatic lesion in his left adrenal gland occupying the space between the adrenal gland and the aorta. Because the mass obliterated the left periarterial space at the L1 level, a right-sided single-needle approach using computed tomographic (CT) guidance was chosen. Under light sedation, a 22-gauge 5’/inch needle was placed using traditional surface landmarks, as first described by Kappis. CT-generated coordinates were then used as the needle was targeted anterolateral to the aorta, superior to takeoff of the superior mesenteric artery, and anterior to the crus of the diaphragm.5 A solution of 16 ml bupivacaine 0.75% and 4 ml Omnipaque 180 was injected. After 20 min, a thorough sensory and motor examination was performed without change from a preprocedure examination; 10 ml anhydrous alcohol was injected. The patient reported good pain relief, and his Dilaudid infusion was decreased from 40 mg/h to 15 mg/h. The patient was converted to a fentanyl infusion and discharged to home 5 days after the neurolytic block. At that time he had good pain control. He was seen in follow-up 3 weeks later with progression of his metastatic disease, pulmonary embolism, dehydration, and increased pain. He died 2 weeks later.

Because of the anatomical considerations in this case, we believe that the use of CT guidance, as opposed to fluoroscopy or blind techniques, provided the only effective approach to neurolytic blockade of the celiac plexus. With the mass occupying the periarterial space on the left, accurate placement of the single needle from the right was required to offer any chance of effective blockade. In addition, CT guidance allowed us to avoid vascular puncture. Although vascular puncture would be unlikely to lead to significant morbidity, blood may dilute local anesthetics and neurolytic agents, decreasing their effectiveness.6

The literature and textbooks of neural blockade describe a variety of neurolytic volumes. Volumes from 15–40 ml are described, with most references using 20–40 ml of total solution.7 In his important textbook, Moore describes the use of 50 ml.2 In this case, we found that later, a neurolytic procedure was performed. Using the same surface landmarks and CT guidance, the needle was replaced. A solution of 8 ml 2% lidocaine 1:200,000 epinephrine and 2 ml Omnipaque 180 was injected. After 20 min, a thorough sensory and motor examination was performed without change from a preprocedure examination; 10 ml anhydrous alcohol was injected. The patient reported good pain relief, and his Dilaudid infusion was decreased from 40 mg/h to 15 mg/h. The patient was converted to a fentanyl infusion and discharged to home 5 days after the neurolytic block. At that time he had good pain control. He was seen in follow-up 3 weeks later with progression of his metastatic disease, pulmonary embolism, dehydration, and increased pain. He died 2 weeks later.

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10 ml of alcohol was enough volume to provide an effective neurolytic block. Although we cannot prove this hypothesis, limiting the volume of neurolytic solution would intuitively seem to decrease tissue destruction and the potential for neurologic injury.

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To the Editor—At the University of Louisville, the Human Patient Simulator (METI, Sarasota, FL) and other simulation models are used for the training of anesthesiology residents. Although the simulator can provide excellent training models in the form of case scenarios, we have discovered that when a central venous or pulmonary artery catheter is required, we cannot simulate the actual placement of the catheter. Learning to be facile in the placement of these monitors is essential to the residents’ training. That facility includes tray preparation (filling the wells with heparinized saline, arranging the syringes and needles), setting up the transducers, preparing the patient, and using the proper sequence of steps to locate and cannulate the vessel. We believe that the more physicians in training practice these procedures, the greater their skills will be at performing them. In most institutions, these skills are acquired by regularly performing the procedures on actual patients. The skill of a resident, therefore, is dependent on the opportunities he or she has to attempt central venous monitor placement. A device that allows a trainee to practice these procedures regularly in a realistic, nonthreatening environment would therefore be beneficial in the acquisition of these skills.

We have developed a device with which the residents can practice central venous catheter placement using the human patient simulator as the human interface. The device, composed of varying sizes of tubing attached to a reservoir filled with artificial blood, simulates either the internal jugular or subclavian vein (fig. 1). The device accepts a J-wire, a triple lumen catheter, an introducer, and a pulmonary artery catheter. The device is placed underneath the skin of the human patient simulator in the anatomically appropriate locations (fig. 2). Using this device, the trainee can perform essentially all of the steps, in their proper sequence, in placing a central venous monitor (the suturing of the catheter to the skin is omitted). The vein can be located using anatomic landmarks (e.g., palpat ing the carotid pulse) and punctured with a seeker needle to reveal a “flash” of artificial blood. Following this step, the Seldinger technique can be used to place either a triple lumen catheter or introducer. Proper placement of the catheter can be confirmed by the aspiration of “blood.” While the catheter is being placed, the simulator operator can simulate events such as premature ventricular contractions with J-wire placement. For the passage of a pulmonary artery catheter, the location of the balloon within the vasculature may be simulated by the resident’s notifying the simulator operator of the distance of the catheter tip from the entrance point of the introducer. As the catheter is advanced, the correlating waveforms, up to the wedge position, may be transmitted to a monitor. This provides additional training in recognizing right-sided cardiac pressure waveforms. The device may also be connected to a reservoir via a stopcock to accommodate the infusion of simulated drugs or additional material related to this article can be found on the ANESTHESIOLOGY Web site. Go to http://www.anesthesiology.org, click on Enhancements Index, and then scroll down to find the appropriate article and link. Supplementary material can also be accessed on the Web by clicking on the “ArticlePlus” link either in the Table of Contents or at the top of the Abstract or HTML version of the article.

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To the Editor—The Rapid Infusion System (RIS) (Hemonetics, Braintree, Massachusetts) is a device designed to deliver blood products and other fluids at precise flow rates up to 1.5 l/min. An important feature of the RIS is the readout of the total infused volume. We present a case in which kinking of a RIS-tube resulted in the inability to infuse fluids and led to erroneously high infusion volume readout without alarms.

A 64-yr-old man presented for thoracoabdominal aortic aneurysm repair. The initial course of surgery was uneventful, and blood loss of 16 l (as judged from cell saver reading) was substituted via the RIS. Later in the procedure, however, the patient developed marked hypovolemia, although the infusion rate setting (1.5 l/min) and corresponding infused volume readout of the RIS by far exceeded the actual rate of blood loss (about 200 ml/min). Inspection of the RIS revealed that the reservoir level did not decrease according to the infusion rate and that infusion pressure was far below the expected value. Finally, we noticed a kinking of the tube located between the heat exchanger and roller pump, which prevented fluid inflow into the roller pump (fig. 1). After removing the kink, infusion pressure correlated with infusion rate, reservoir level decreased appropriately, and hypovolemia resolved.

The case demonstrates that the infusion volume readout displays erroneous high values if an obstruction of the roller pump inflow occurs. The false measure is due to the infused volume readout being obtained by the rotation of the roller pump rather than by direct flow or volume determination. The malfunction occurred after an uneventful initial surgical phase. Most likely, warming of the tube had resulted in kinking. Two additional cases associated with the use of the RIS have been described in the literature: the connection of one of the infusion lines to the reservoir, and the incorrect positioning of the recirculation line in the respective clamp.1,2 Similar to the present case, the real infusion rate was much lower than that displayed by the infused volume readout.

The RIS is a valuable tool for the management of massive blood loss. It is, however, important to realize that the infused volume readout is a measure of roller pump rotation, which might not always be identical to the fluid volume applied to the patient. Careful observation of the infused volume readout, infusion pressure, volume added to the reservoir, and assembly of the apparatus, as well as the hemodynamic effects of infusion, is mandatory for the early detection of malfunctions.

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Fig. 1. Schema of the Rapid Infusion System (RIS) and photography showing the kinking of the tube. Inflow of fluids from the reservoir to the roller pump was interrupted; thus, no fluid was infused. The total infusion volume readout, however, steadily increased according to the chosen infusion rate because this value is obtained from the rotation of the roller pump. Although the RIS is featured with various safety devices, an occlusion of the tube proximal to the roller pump does not result in an alarm.

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