To the Editor.—I read with considerable interest the article by Benavides et al.1 describing experiments of air bubble growth in water during exposure to 100% nitrous oxide, 100% xenon, or 50% xenon–50% oxygen. Although the experiments were nicely conducted, they explore a physics of gas flux in an unconstrained bubble permitted to grow spherically. Importantly, this geometry has limited biologic relevance for bubbles occluding vessels in the size range they have studied. The authors have referenced our previous work on xenon transport,2 but they have mistakenly interpreted the findings presented therein to indicate the growth of bubbles as spheres. Rather, that study presents some simulations for bubbles that are initially spherical and just fill the vessel lumen. Such bubbles cannot grow radially because they are constrained by the vessel wall and therefore elongate during growth while maintaining a fixed curvature on the interface. This results in a much different force balance across the gas-liquid interface and, hence, a different pressure condition on the interior of the bubble from that which occurs in the case of a time-variant interfacial shape, which the authors have studied. We have described these differences in our previous theoretical and experimental studies of intravascular gas embolism.2–5

In addition, the initial internal gas content they have studied includes nitrogen, equilibrated with test solution A, but test solution B is nitrogen free. Hence, there are large gradients for nitrogen flux when the solutions are switched. We purposely avoided nitrogen as a component of either the bubble or perfusate in our predictions and considered only oxygen and xenon as the transportable species.2 Whereas other studies have studied growth of similarly unconstrained air bubbles during cardiopulmonary bypass,6 our work has not provided any data for direct comparisons such as the authors have made, based on the different gas constituents and the governing physics dictated by the shape constraint.

I find it fascinating, however, that they haveouched their results in terms of bubble diameter growth. When transferred to the volume domain, one readily sees that the spherical bubbles exposed to 100% xenon or 100% nitrous oxide had grown to more than twice their initial volume in 25 min (figs. 2 and 3) and continued growing when the solutions were switched (downward arrow). The time required for this is surprisingly similar to the volume doubling times we reported for many of the cases we explored, despite the differences in our model and these experiments.

The curve fitting by a double exponential suggests that there will be continuous exponential growth of bubble diameter. So although the physics and gas transport are different from what we studied, the indication of the studies are the same.

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References
(Accepted for publication June 19, 2006.)

In Reply.—Our article1 on the expansion of gas bubbles by xenon and nitrous oxide investigated how air bubbles of various dimensions in aqueous solution would expand when suddenly exposed to solutions containing certain gas mixtures (particularly mixtures containing xenon). The motivation behind this work was simple: Would air bubbles that were entrained while on cardiopulmonary bypass during cardiac surgery expand to a worrying extent if xenon were used during cardiopulmonary bypass?6 However, if entrained gas bubbles expanded greatly, xenon may do more harm than good.

Indeed, Dr. Eckmann and his colleagues have suggested exactly that,4 based on theoretical calculations that concluded that small gas bubbles would expand rapidly and indefinitely if they were trapped in fine blood vessels. (We fully understand that the model assumes that the bubbles are constrained by the size of the capillaries. For example, their calculations suggest that a 50-nl bubble of oxygen exposed to 70% xenon–30% oxygen would grow to 250 nl in approximately 20 min with an ever-increasing rate of growth. Because we thought that these predictions were implausible, and because there were a large number of variables that had to be estimated, we conducted our experiments, which were designed to measure bubble growth directly under a well-defined set of conditions. We studied the expansion of both air and oxygen bubbles, and the results were similar; our data show bubble expansions of the order of 10% in diameter and 30% in volume under conditions likely to be encountered during cardiopulmonary bypass. We concluded that this is unlikely to represent a significant clinical problem.

We disagree with Dr. Eckmann’s claim that his calculations predict similar expansions to those we observed. Apart from the extent of the volume expansions that were predicted,4 their most striking aspect was the ever-increasing rates of expansion that seemed to predict unlimited bubble growth. In contrast, we observed limited bubble growth with volumes tending toward finite equilibrium values. Even making allowances for the differences between the model and the gas compositions, we believe our experimental observations probably better reflects reality than the theoretical calculations that Dr. Eckmann has published. Furthermore, in our recently published feasibility and tolerability clinical study involving exposure of cardiac surgical patients to xenon while on cardiopulmonary bypass, there was no

Drs. Franks and Maze have a financial interest in an Imperial College spin-out company (Protecon Ltd., London, United Kingdom) that is interested in developing clinical applications for medical gases, particularly xenon, and both are paid consultants for this activity. Drs. Franks and Maze also sit on a Strategic Advisory Board that advises Air Products, Allentown, Pennsylvania, on possible medical applications for gases, including anesthetic gases. The funding for the current study was provided by Carburos Metálicos, a wholly owned subsidiary of Air Products.
increase in the number of observed emboli, which would have oc-
curred had the bubble size of gas emboli increased significantly.5

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and xenon. ANESTHESIOLOGY 2006; 104:299–302

To the Editor.—I read with interest the report by Campos et al.1 This 
article reported 39% and 36% incidences of failure of proper placement 
of lung isolation devices by faculty and senior residents, respectively. 
All participating anesthesiologists did not regularly practice thoracic 
anesthesia. The authors stated that one-lung ventilation is primarily 
provided by either a double-lumen tube, the Univent® torque control 
blocker (VitaId Ltd., Lewiston, NY), or the Arndt® blocker (Cook 
Critical Care, Bloomington, IN). However, an additional blocker that 
was not mentioned is the Cohen blocker (Cook Critical Care). This 
commonly used blocker has a wheel at the operator end that, when 
turned, flexes the tip and gives the anesthesiologist the ability to direct 
the blocker to the appropriate bronchus.

This is an important article that highlights the difficulties for anes-
thesiologists who need to provide one-lung ventilation on an occa-
sional basis. There is a learning curve with all of these techniques. I 
agree with the authors that the successful practice of thoracic anes-
thesia requires familiarity with the devices, skill in fiberoptic bronchos-
copy, and knowledge of the tracheobronchial anatomy. Residents 
learning thoracic anesthesia should learn all three of these types of 
skills before graduation, and these should be specific goals in residency 
programs.

The authors stated that the preoperative instruction included the 
fiberoptic views that constituted correct positioning of the device. 
When checking the position of the lung isolating device, it is also 
important to identify the right upper lobe, the characteristic D-shaped 
opening to the middle lobe, and the basilar and superior segments of 
the lower lobe. The left mainstem bronchus is identified, as well as the 
left upper and lower lobes. The views of just the double-lumen tube 
or blocker balloon are not adequate for establishing correct 
positioning, which could account for some of the malpositions re-
ported in the study. If a simplified instruction is given, it is most 
important to identify the right upper lobe. It would be helpful to know 
which views were included in the tutorial.

It would be interesting to repeat the study, with more training, to 
avoid the bubble size of gas emboli increased significantly.5

One-lung Ventilation Provided by Anesthesiologists Having Minimal 
Experience with Thoracic Anesthesia

To the Editor.—I read with interest the report by Campos et al.1 This 
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It would be interesting to repeat the study, with more training, to 
avoid the bubble size of gas emboli increased significantly.5

Results of the Study Comparing Devices Used for Lung 
Isolation Are Rather Concerning

To the Editor.—The study by Campos et al.1 presented some interesting 
findings. It was surprising that the lung isolation device used to achieve 
one-lung isolation did not provide an advantage to the anesthesiologist 
with limited experience. There was no difference in frequency of failure or time to 
achieve a higher success rate. Anesthesiologists who may have to 
provide one-lung ventilation on occasion should become familiar with 
the devices, learn the tracheobronchial anatomy, and practice fiberopt-
ic bronchoscopy. There are hands-on workshops that are given at the 
annual meetings of the American Society of Anesthesiologists and the 
International Anesthesia Research Society and at the PostGraduate 
Assembly in Anesthesiology.

When choosing a lung isolation technique, there are other issues to 
consider besides the initial placement. Blockers, especially when used 
on the right side, are more easily dislodged than double-lumen tubes. It is 
also easier to suction and apply oxygen via a double-lumen tube, 
compared with a blocker. The main advantage of the blocker is in the 
patient with the difficult airway, which was excluded in the study. It is 
essential to be knowledgeable of possible complications. For example, 
a blocker may become dislocated into the trachea and block ventilation 
completely. Techniques for prevention and treatment of hypoxemia 
must be understood.

There is more to practicing thoracic anesthesia than just the initial 
placement of the lung isolation device.

The most important teaching message from this study is that prac-
titioners who may have to provide one-lung ventilation on an occa-
sional basis need to do continuing medical education. Development of 
a tutorial and/or attending a hands-on workshop might be beneficial.

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isolation used by anesthesiologists with limited thoracic experience: Comparison 
of double-lumen endotracheal tube, Univent® torque control blocker, and Arndt 
wire-guided endobronchial blocker. ANESTHESIOLOGY 2006; 104:261–6

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ally require longer time for placement compared with the left-sided DLT.\textsuperscript{3,4} The results of the study\textsuperscript{1} are rather concerning, because training and exposure for residents largely consist of the use of the DLT, which is used more often and has been in clinical practice for a longer period of time. Because a combination of unfamiliarity with tracheobronchial anatomy and skill in fiberoptic bronchoscopy was responsible for most of the malpositioning,\textsuperscript{1} perhaps training for residents should concentrate on building a stronger foundation in basic knowledge and skill. Personal experience with the routine use of video-bronchoscopy for confirmation of DLT position has shown that it greatly facilitates the learning process. Anatomy can be demonstrated clearly, and due to simultaneous viewing by the attending and resident, there is better appreciation of the steps involved in the identification and correction of any malposition problems. The use of a virtual bronchoscopy simulator may also be useful in skill acquisition in flexible bronchoscopy.\textsuperscript{5}

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Anesthesiology 2006; 105:1061–2

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number of malpositions was quite low for both tube types and did not differ between the groups. Therefore, the choice of device for lung isolation in the right mainstem bronchus does not matter when a cardiothoracic anesthesiologist places these devices. This concept might not apply to anesthesiologists with limited experience in thoracic anesthesia, but to our knowledge, this has not been tested scientifically.

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Ambesh Maneuver during Subclavian Vein Catheterization Successfully Prevents and Detects Misplacement of the Catheter into Ipsilateral Internal Jugular Vein

To the Editor—During subclavian vein catheterization, one of the most common malplacements of the catheter is into the ipsilateral internal jugular vein (IJV). Chest radiography is performed to identify the exact location of the catheter and procedure-related complications. Misplaced catheters have increased risks of thrombophlebitis in addition to impairment of the central venous pressure (CVP) measurement. Recently, Ambesh et al.1,2 showed that manual occlusion of the ipsilateral IJV in the supraclavicular fossa during and after insertion of subclavian vein catheter is successful in preventing and diagnosing the misplacement of the subclavian vein catheter into the IJV, respectively. Since Ambesh et al. developed this maneuver and reported excellent results, no other study has validated these results. Therefore, in a randomized and controlled study, we tested whether the Ambesh maneuver is successful in preventing and diagnosing the misplacement of a subclavian vein catheter into the IJV.

After approval by the Ethics Committee (King George’s Medical University, Lucknow, India), 300 adult patients of either sex scheduled to undergo central venous cannulation through the subclavian approach were randomly allocated into two groups of 150 each. Informed verbal consent was obtained from all patients before the procedure. In a patient lying supine with a 15°–20° Trendelenburg position and the head turned to the left, the junction of the medial one third and lateral two thirds of the clavicle in the right infraclavicular area was chosen as the puncture point. An 18-gauge introducer needle was inserted at this point and directed toward the sternoclavicular joint. After free flow of venous blood, the J-tip guide wire was threaded through the cannula into the subclavian vein. In the Ambesh maneuver group of patients, the ipsilateral IJV was occluded, as described by Ambesh et al.,3 during threading of the J guide wire, whereas in control group, no such maneuver was performed. The subclavian vein catheter was then railroaded over the guide wire. The catheter was then connected with a transducer, and the CVP value and waveform pattern were observed. Next, the Ambesh maneuver4 was reapplied for approximately 10 s, and changes in CVP value and waveform pattern were noted. If there was an increase in CVP value by more than 3 cm H2O along with flattening of the waveform, it was presumed that the catheter tip was misplaced into the ipsilateral IJV. At the end of the procedure, chest radiography was performed, and position of the catheter was identified in all patients. The characteristics of the patients were analyzed using the Student t test and Fisher exact test. P values were two-tailed, and P > 0.05 was considered significant.

The age, sex, and body weight of the patients were comparable in two groups. Five patients in the control group and 7 patients in the Ambesh maneuver group could not be cannulated; therefore, 145 patients in the control group and 143 patients in the Ambesh maneuver group were analyzed. Chest x-ray films showed that in control group, there were 10 (6.5%) misplaced catheters, 9 (6.2%) in ipsilateral IJV and 1 (0.7%) in opposite subclavian vein, whereas in the Ambesh maneuver group, there were 2 (1.4%) (95% confidence interval, 1.4–6.9%; P < 0.05) misplaced catheters, both in the opposite subclavian vein and none in the IJV. The operator experienced difficulty in inserting the guide wire in 3 patients of control group and 9 patients of Ambesh maneuver group. The withdrawal and reinsertion of the guide wire and catheter were easy.

The correct placement of the central venous catheter is an essential prerequisite for accurate monitoring of CVP and long-term use of the catheter. Misplacement of the tip may enhance the risk of clot formation, thrombophlebitis, and catheter erosion in addition to impaired CVP measurement.5,6 Recently, Domino et al.7 reported that the proportion of malpractice claims related to central catheters and vascular access injury has increased significantly. The incidence of malpositioning of CVP catheters through the infraclavicular technique of the subclavian vein varies between 4% and 8%.5,6 Our study shows a 6.9% incidence of misplacement of subclavian vein catheter through the right infraclavicular approach, and most of the misplaced were in the ipsilateral IJV (6.2%). The operator encountered difficulty in inserting the guide wire in only 3 patients without IJV occlusion and in 9 patients with IJV occlusion. It becomes obvious that the guide wire in some of these 9 patients of the IJV occlusion group was intended to go into the ipsilateral IJV, but the IJV was occluded manually. The occlusion of ipsilateral IJV in the supraclavicular area may have prevented the cephalad insertion of the guide wire and therefore the subclavian vein catheter into the IJV.

We conclude that the Ambesh maneuver is a simple, inexpensive, and handy bedside technique that helps in preventing and diagnosing the misplacement of a subclavian vein catheter into the IJV. We strongly believe that the Ambesh maneuver should be used in all patients undergoing subclavian vein catheterization.

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To the Editor—American inventor and businessman Thomas Alva Edison is legendary for his contributions to such technologies as the lightbulb, the telephone, the phonograph, and motion pictures, among many others. In his lifetime, Edison obtained 1,093 US patents and some 1,239 patents in other countries. Little known among these efforts was his “improved anesthetic compound.”

In the summer of 1882, George F. Shrady (Founder and Editor, Medical Record 1866–1904) (1837–1907), reported that Thomas Edison invented a new anesthetic made of chloroform, ether, alcohol, and camphor and had applied for British and German patents. The witty but misinformed editor added, “Edison may wish to use it on his stockholders until electric light was in successful operation.”

In fact, the “anesthetic” actually was an analgesic liniment that Edison had prepared in early 1878. He named it Polyform and advertised it for “neurologic pain.” Polyform was a mixture of chloroform, ether, camphor gum, alcohol, chloral hydrate, morphine, and oils of peppermint and clove. Edison believed that his compound’s various analgesics would potentiate each other and that the mixture would attack pain in a “shotgun manner.”

In 1879, Edison applied for a US patent but, for unknown reasons, withdrew his application shortly thereafter. In February 1880, the British patent No. 599 was granted to his London agents for a slightly modified compound. The editor of the Medical Record was misinformed: Edison did not apply for a German patent (written personal communication, Hubert Rothe, Director, Information Department, German Patent and Trademark Office, Munich, Germany, May 2004).

Topical ether and, especially, chloroform had been widely used for musculoskeletal and neurologic pains since their discovery. At the time of Edison’s invention, not only were liniments of chloroform and of camphor used in the United States, but there also existed lotions made of chloroform, camphor, ether, alcohol, morphine, and chloral hydrate. One, Sankt Jakob’s Oil, had been popular in Germany since the mid-1870s. It was marketed in the United States during the 1880s under the name of St. Jacob’s Oil by the firms of C.A. Voegeler in Baltimore and Kroeger Ltd. of Cincinnati. Its formula is given in several US formularies.

Whether Edison knew of St. Jacob’s Oil when he invented his Polyform or whether he learned of its existence later on is unknown. The latter may explain why he did not apply for a German patent and withdrew his US application.

The authors thank Gregory J. Highby, Ph.D. (Professor and Director of the American Institute of the History of Pharmacy, Madison, Wisconsin), Michael A. Flannery, Ph.D. (Associate Professor and Associate Director for Historical Collections, University of Alabama at Birmingham, Birmingham, Alabama), and Hubert Rothe (Director, Information Department, German Patent and Trademark Office, Munich, Germany) for their valuable information.

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A Rare Iatrogenic Cause of Upper Gastrointestinal Bleeding

To the Editor—An 85-yr-old man was transferred to our institution for evacuation of a subdural hematoma. His course was complicated by deep venous thromboembolism necessitating intravenous heparin. He ultimately required intubation and was ventilated for several minutes before the recognition of an inadvertent esophageal intubation. The endotracheal tube was correctly repositioned, and a nasogastric tube was inserted with the return of approximately 500 ml of fresh blood.

After the heparin was discontinued, his partial thromboplastin time normalized, and the platelet count and international normalized ratio were also normal. On upper endoscopy, the esophagus and duodenum were completely normal. In the gastric fundus, there were linear mucosal tears (fig. 1). There was no surrounding inflammation, and the appearance was not consistent with trauma from the nasogastric tube. There was minimal oozing of blood and small adherent clots, but there was nothing necessitating endoscopic treatment.

Tears in the gastric mucosa from overdistension during endoscopy for percutaneous endoscopic gastrostomy tube placement in the set-
We report a case of acute upper gastrointestinal bleeding from mucosal tears resulting from gastric overdistention after inadvertent esophageal intubation. To our knowledge, this complication has not been previously described and should be included in the differential diagnosis of upper gastrointestinal bleeding in critically ill patients who have had inadvertent esophageal intubation or cardiopulmonary resuscitation. The combination of history of esophageal intubation, involvement of the lesser curvature, and similarity to the mucosal tears seen by Green and Tendler in 1981 during percutaneous endoscopic gastrostomy tube placement support this diagnosis. In addition, in cases of upper gastrointestinal bleeding when a history of inadvertent esophageal intubation is obtained, gastric perforation should be ruled out with abdominal imaging before performing upper endoscopy.

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Anesthesia in a Patient with Gitelman Syndrome

To the Editor.—We would like to report our recent experience with a patient with a rare disorder known as Gitelman syndrome.

**Gitelman syndrome**, a variant of Bartter syndrome, is a congenital autosomal recessive disorder characterized by hypokalemia, hypomagnesemia, and hypocalcineria associated with metabolic alkalosis.1,2 Unlike Bartter syndrome, which presents in the neonatal period and childhood up to 5 yr of age, Gitelman syndrome presents in early adulthood. The two syndromes may also be distinguished from each other because Gitelman syndrome presents with hypomagnesemia and hypocalcineria, whereas Bartter syndrome presents with normal serum magnesium and high urinary calcium.1,2 Patients with Gitelman syndrome usually present with cramps, fatigue, muscle weakness, and carpopedal spasms.

A 47-yr-old woman recently presented to us for repair of nasolacrimal duct stenosis during general anesthesia. Her medical history was significant for diabetes, gastrointestinal reflux disease, and thyroid disease. Her surgical history included a hysterectomy, an appendectomy, a thyroidectomy, a breast biopsy, and an exploratory laparotomy for ovarian cancer. She described her symptoms as cramps in her legs. She related that 2 yr ago during the surgery for her thyroid, she had a cardiac arrest. The patient stated that her cardiac arrest had occurred because of unrecognized hypokalemia and hypomagnesium. Her medications included potassium chloride, magnesium, spironolactone, levothyroxine, glyburide, and lensoprazole. Her vital signs included a blood pressure of 111/77 mmHg, pulse of 98 beats/min, respiration of 18 breaths/min, and temperature of 97.9°F. Her laboratory studies showed the following: white blood cells, 10.6 (4.1–11.2); hemoglobin, 15.3 g/dl (11.5–15.1); hematocrit, 45.0% (35–46); platelets, 503 × 103 (140–400); Na, 137 mEq/L (136–145); K, 4.2 mEq/L (3.5–5.1); Cl, 96 mEq/L (98–107); HCO3−, 27 (20–27); blood urea nitrogen, 20 mg/dl (6–20); creatinine, 0.8 (0.5–1.2); glucose, 68 mg/dl (65–115); Ca, 10.9 mEq/L (8.8–10.5); Mg, 1.9 mEq/L (1.3–2.1); and urine creatinine, 181 mEq/L. She was 4 ft 11 in tall and weighed 70 kg.

In the operating room after placement of an electrocardiograph, noninvasive blood pressure cuff, and pulse oximetry, general anesthesia was induced using propofol and fentanyl for muscle relaxation and for tracheal intubation. Desflurane was used...
for maintenance of anesthesia. At the end of the case, the muscle relaxant was reversed using glycopyrrolate and neostigmine. When the patient was awake and responding to commands, her trachea was extubated in the operating room without any complications.

Although the patient’s current magnesium level was only slightly decreased, we decided not to replace it. Her current potassium level was also normal. She was aware of her diagnosis of Gitelman syndrome and was taking potassium and magnesium replacement. Review of the literature reveals that ventricular tachycardia has occurred in patients with Gitelman syndrome when potassium and magnesium levels are low. This potentially fatal arrhythmia must be recognized and treated early.

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