Can Epidural Fentanyl Induce Selective Spinal Hyperalgesia?

David W. Cooper, M.B.B.S., F.R.C.A., Consultant Anaesthetist, Department of Anaesthesia, South Cleveland Hospital, Middlesbrough, Cleveland, United Kingdom
dr_david_cooper@hotmail.com

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In Reply.—Dr. Cooper’s letter suggests the possibility that opioid-induced hyperalgesia may explain those studies that have not found evidence of selective spinal analgesia after epidural administration of some opioids (particularly fentanyl). The suggestion is interesting, and, as Dr. Cooper points out, data indirectly support his supposition. However, I think this may be an example of showcasing data that support an argument, while, at the same time, ignore inconsistent data. For example, Dr. Cooper mentions that Eisenach cited two studies1,2 in his editorial3 that support his position; however, Dr. Cooper ignored two other human studies in the same editorial that are antithetical to his position.4,5 In addition, Dr. Cooper states “Bernards and Sorkin6 have shown that, in pigs, ‘epidural fentanyl moves rapidly from the epidural space to the spinal cord.’” This misses the point that it is the bioavailability of fentanyl at opioid receptors in the spinal cord gray matter that determines its spinal effectiveness. In this regard, a recent study by Ummenhofer et al.7 and a classic study by Schubert et al.8 showed that the bioavailability of fentanyl in spinal cord gray matter is poor. Also, it is by no means clear that fentanyl concentrations at spinal cord opioid receptors would “be expected to be higher after epidural than after systemic administration,” even though the dose needed for analgesia and the resultant plasma concentration have been shown by multiple (though not all) studies to be equivalent by both routes of administration.9–15 This is likely because systemic administration delivers fentanyl to within a few microns of its target site. In contrast, epidural administration delivers the drug several centimeters away from the spinal target site and necessitates that the drug traverse multiple barriers and negotiate several lipidophilic environments (e.g., epidural fat, white matter myelin) into which it can be sequestered and rendered unavailable at opioid receptors. Therefore, administration of a drug in the epidural or intrathecal space does not ensure that it will reach its target site in the spinal cord in high concentration. This is exactly what Ummenhofer et al.7 and Schubert et al.8 demonstrated. Last, Dr. Cooper has offered no explanation for why other opioids (e.g., morphine) clearly have a long-lasting selective spinal site of action after epidural administration, which is inconsistent with the idea that opioid binding to spinal opioid receptors produces acute hyperalgesia.

Cooper’s suggestion that epidural fentanyl might induce an acute hyperalgesic state is interesting and provocative. There are data that would seem to support it; however, there is also a significant amount

To the Editor.—In his editorial, Eisenach1 highlighted an interesting paradox: while attempting to produce profound analgesia with high doses of potent opioids, it is possible to produce a “preemptive hyperalgesic” effect. In the two human studies referenced,6,7 high doses of systemic remifentanil and fentanyl produced acute hyperalgesia.

In a previous human study, we found evidence that intrathecal fentanyl administration can produce acute spinal hyperalgesia.4 Administration of 25 μg intrathecal fentanyl during Cesarean section increased postoperative intravenous morphine requirements by 63% between 6 and 23 h postdelivery.

In his editorial, Bernard5 mentions his concern that “alfentanil and sufentanil (and to some extent fentanyl) are used in the epidural space, ... despite mounting evidence that these opioids do not produce analgesia by a selective spinal mechanism.” However, there is evidence that epidural fentanyl, when it is administered in the minimal effective dose, has a selective spinal action.6–10

In humans,11 lumbar cerebrospinal fluid levels of fentanyl increase rapidly after epidural fentanyl administration, and Bernards and Sorkin12 have shown that, in pigs, “epidural fentanyl moves rapidly from the epidural space to the spinal cord.” Prolonged postoperative epidural fentanyl administration can produce plasma levels similar to those of systemic administration.13 However, spinal cord levels of fentanyl still would be expected to be higher after epidural than after systemic administration. It is therefore surprising that the analgesic effectiveness of epidural and systemic fentanyl often are reported to be comparable, even if plasma levels are similar. This is especially so if, as suggested by Bernards,9 there is synergy between spinal and supraspinal opioid analgesia in humans.

It may be that, by producing relatively high spinal compared with systemic levels of fentanyl, epidural fentanyl administration can induce acute selective spinal hyperalgesia. The greater the magnitude of selective spinal hyperalgesia induced, the smaller the difference in analgesic effectiveness of epidural and systemic fentanyl would be. This could help to explain why several studies have not found a difference between epidural and systemic fentanyl analgesia. Administration of epidural fentanyl in the minimal effective dose may limit the development of spinal hyperalgesia, thereby facilitating selective spinal analgesia.

In Reply.—Dr. Cooper’s letter suggests the possibility that opioid-induced hyperalgesia may explain those studies that have not found evidence of selective spinal analgesia after epidural administration of some opioids (particularly fentanyl). The suggestion is interesting, and, as Dr. Cooper points out, data indirectly support his supposition. However, I think this may be an example of showcasing data that support an argument, while, at the same time, ignoring inconsistent data. For example, Dr. Cooper mentions that Eisenach cited two studies1,2 in his editorial3 that support his position; however, Dr. Cooper ignored two other human studies in the same editorial that are antithetical to his position.4,5 In addition, Dr. Cooper states “Bernards and Sorkin6 have shown that, in pigs, ‘epidural fentanyl moves rapidly from the epidural space to the spinal cord.’” This misses the point that it is the bioavailability of fentanyl at opioid receptors in the spinal cord gray matter that determines its spinal effectiveness. In this regard, a recent study by Ummenhofer et al.7 and a classic study by Schubert et al.8 showed that the bioavailability of fentanyl in spinal cord gray matter is poor. Also, it is by no means clear that fentanyl concentrations at spinal cord opioid receptors would “be expected to be higher after epidural than after systemic administration,” even though the dose needed for analgesia and the resultant plasma concentration have been shown by multiple (though not all) studies to be equivalent by both routes of administration.9–15 This is likely because systemic administration delivers fentanyl to within a few microns of its target site. In contrast, epidural administration delivers the drug several centimeters away from the spinal target site and necessitates that the drug traverse multiple barriers and negotiate several lipidophilic environments (e.g., epidural fat, white matter myelin) into which it can be sequestered and rendered unavailable at opioid receptors. Therefore, administration of a drug in the epidural or intrathecal space does not ensure that it will reach its target site in the spinal cord in high concentration. This is exactly what Ummenhofer et al.7 and Schubert et al.8 demonstrated. Last, Dr. Cooper has offered no explanation for why other opioids (e.g., morphine) clearly have a long-lasting selective spinal site of action after epidural administration, which is inconsistent with the idea that opioid binding to spinal opioid receptors produces acute hyperalgesia.

Cooper’s suggestion that epidural fentanyl might induce an acute hyperalgesic state is interesting and provocative. There are data that would seem to support it; however, there is also a significant amount
How to Open the Lung? The Unsolved Question

To the Editor—We read with interest the editorial by Bigatello et al.,1 which described “protective” ventilatory techniques as part of an integral approach to the treatment of adult respiratory distress syndrome.

Some points, however, need to be addressed. Although it is reasonable to use tidal volumes less than 8 ml/kg and to keep the plateau pressure less than 30 cm H2O, it is more important to avoid shear forces “opening-collapsed” that are repetitively generated in the small airway during the respiratory cycle. The use of different maneuvers to open a collapsed lung and to keep the lung open was postulated years ago.2 The optimal method of alveolar recruitment, however, is a subject of controversy.

Bigatello et al. discussed two possible methods to obtain alveolar recruitment. The first was the “open-lung approach” of Dr. Amato.3 We believe that this strategy does not obtain alveolar-opening pressures. A peak airway pressure of 40 cm H2O is enough to recruit completely a healthy lung; but higher pressures are necessary to open the lung of a patient with adult respiratory distress syndrome. In addition, to avoid lung collapse, the open-lung approach applies a positive end-expiratory pressure (PEEP) of 2 cm H2O above the lower inflexion point, as found on the inspiration curve of the volume–pressure loop. Ri-mensberg et al.,4 while studying the expiration part of the volume-pressure curve, clearly demonstrate that the level of PEEP needed to avoid collapse was lower than the lower inflexion point. The open lung approach, therefore, overestimates the level of PEEP necessary to avoid collapse, which increases the peak airway pressure and limits the appropriate carbon dioxide clearance.

The second method, described by Pelosi et al.,5 is the sigh. The sigh is a method to increase the functional residual capacity during general anesthesia. Later, by extension, it was applied to a critically ill patient undergoing mechanical ventilation. Even when this strategy did not prove to be beneficial for patients,6 it was included as a ventilatory mode in most of the ventilators at the time. To apply this strategy, volume-control ventilation was used, which doubled (and sometimes more than doubled) the tidal volume.

We believe this is an erroneous strategy. We believe the concept of recruiting the collapsed lung by increasing inspiratory pressures, but we disagree about the use of a volume-controlled method without limitation of the maximal level of pressure, which can increase epithelial–alveolar damage.7

We believe that an alveolar recruiting maneuver should be performed in consideration of the following key points:

1. Use a pressure-controlled method or a volume-controlled mode with limited pressure. This will allow the maximal peak inspiratory pressure to be set to avoid iatrogenic lung damage.
2. Reach the critical alveolar pressure by setting the peak inspiratory pressure. The critical alveolar-opening pressure is approximately 40 cm H2O in a healthy lung8 and approximately 55 cm H2O in a diseased lung.2
3. Avoid shear-force lesions by limiting pressure and volume differences in the airway during the respiratory cycle. To do this, PEEP increments should be parallel to peak inspiratory pressure increments, and the respiratory rate should be adjusted to limit the tidal volume to 10 ml/kg.
4. Maintain ventilation with these parameters during an adequate period of time: 10–20 respiratory cycles are sufficient.
5. Decrease the peak airway pressure and return the ventilator settings to that used before maneuver; keep PEEP above the collapse pressure.

This alveolar recruitment strategy has proven to be useful in patients with healthy lungs undergoing general anesthesia,9 and it is used in many critical care units.

Gerardo Tusman, M.D., Department of Anesthesiology
Elisia Turchetto, M.D.
Alicia Rodriguez, M.D., Department of Critical Care Medicine, Hospital Privado de Comunidad, Mar del Plata, Argentina
hpc-quir@argenet.com.ar

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Pulmonary Aspiration of Milk and Cream: 
An Avoidable Complication

In Reply—We have read with interest the case report by Brodsky et al.1 We believe there are some important issues that are raised by this report. Brodsky et al.1 suggest that the usual steps recommended for reduction of aspiration risk probably are ineffective. Although the effectiveness of H2-receptor antagonists in reducing acidity in the presence of vagotomy is questionable, prokinetic agents have some theoretic potential benefit; the neutralization of any acid present with sodium citrate also would be beneficial. We also refute the notion that the Sellick maneuver is ineffective. After transhiatal esophagectomy, the cervical esophagogastric anastomosis is located endoscopically 19 or 20 cm from the upper incisors, 4 or 5 cm distal to the upper esophageal sphincter. Correctly applied cricoid pressure should be as effective in preventing passive regurgitation of intrathoracic gastric contents in a patient who underwent transhiatal esophagectomy as it is in any other patient. In our institution, we have significant experience with this procedure,2 and we frequently anesthetize patients for subsequent surgeries; it is standard practice to apply cricoid pressure during induction and intubation. We are unaware of significant cases of pulmonary aspiration in our patients and strongly recommend the use of cricoid pressure when anesthetizing patients who have undergone transhiatal esophagectomy.

We also strongly endorse the use of a jejunostomy tube for the administration of milk and cream in these patients, for the purpose of identifying the thoracic duct in cases of chyle leak. If the jejunostomy already has been removed or was not placed at the original operation, a Dobhoff feeding tube placed distal to the ligament of Treitz permits safe delivery of this mixture into the gut.

Christopher Harle, F.R.C.A. 
David Jones, F.R.C.A., Visiting Instructor, Department of Anesthesiology 
Mark B. Orringer, M.D., Professor and Head, Section of General Thoracic Surgery, University of Michigan, Ann Arbor, Michigan charle@umich.edu

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(Accepted for publication June 1, 2000.)
Venous Air Embolism in Craniosynostosis Surgery: What Do We Want to Detect?

To the Editor—We are greatly interested in the recent article by Faberowski et al.1 that analyzed the incidence of venous air embolism (VAE) in a small series of children undergoing repair of craniosynostosis. We were very impressed by the 82.6% incidence of VAE detected by precordial Doppler monitoring. As stated in their article, this percentage was much higher than any previously reported incidence of VAE during cranietomy in infants. Harris et al.2 used echocardiography for detection of VAE in infants undergoing cranietomy, some of whom were at risk of VAE because of major cranial malformations, and found what we considered to be a very high incidence of 66%. In our own experience, VAE occurred in only 3 of 130 children (2.6%) undergoing repair of craniosynostosis.3 In all cases, these patients had complex vault remodeling, heavy perioperative blood losses, and severe hypotension during VAE. None of these patients experienced postoperative consequences of VAE. Since that time, surgical prevention was enhanced, and no additional cases were noted in the previous 3 yr in more than 350 procedures.

It could be argued that the detection at our institution is only based on continuous end-tidal carbon dioxide monitoring, which is much less sensitive than Doppler monitoring. However, extrapolating from the results of Faberowski et al.,1 the incidence of VAE that is detectable by capnography could be more than 40%; 23 times greater than in our experience. Minimal venous air embolisms probably occur very frequently during vault resection before the surgeon can apply efficiently bone wax. This risk of air entry is increased in the presence of hypovolemia related to abrupt blood losses. If a very sensitive monitor is used, these minimal and short-lasting episodes of VAE will be detected. In these conditions, it is not surprising that only 30% of the children experiencing VAE had related hypotension; but the question of the clinical implications of detecting such a small amount of air entry is not answered. In the study by Cucchiara et al.,4 36% of the adult patients in the sitting position and experiencing VAE had hypotension. In a similar pediatric population, we found an 85% incidence of cardiovascular variations related to VAE,5 which is in greater accordance with the reported incidence of hypotension related to VAE in pediatric patients.

The authors are to be congratulated for pointing out the problem of VAE during craniosynostosis repair. However, a possible conclusion drawn from this article could be that only 18% of the children undergoing craniosynostosis repair could be spared perioperative episodes of VAE that increase morbidity and mortality. This probably does not reflect the clinical practice of other centers with extensive experience with this type of surgery.

Philippe G. Meyer, M.D., Staff Anesthesiologist
Dominique Renier, M.D., Professor of Neurosurgery
Gilles Orliaquet, M.D., Staff Anesthesiologist
Stephane Blanot, M.D., Staff Anesthesiologist
Pierre Carli, M.D., Professor of Anesthesiology, Hôpital des Enfants Malades, Paris, France
philippe.meyer@nck.ap-hop-paris.fr

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To the Editor.—It is with interest that we read the study by Stow et al. that evaluates the effects of xenon on isolated guinea pig hearts and cation currents in isolated cardiomyocytes. We agree fully with the authors that xenon anesthesia may be beneficial for patients with cardiac disease who cannot tolerate the depressant effects of commonly used gas anesthetics. However, we would like to express our concerns regarding two points of the methods used in this study. First, in the current work, the halothane and sevoflurane concentrations are measured, but, for xenon, the authors’ description informs us that “after vigorous shaking (the gas mixture and the bath solution) for several minutes to facilitate equilibration, oxygen partial pressure was verified to be near 150 mmHg at one atmosphere.” They seem to conclude that after determination of the oxygen partial pressure, the rest, i.e., 610 mmHg at one atmosphere, must be xenon. However, if one tries to show that xenon does not alter cardiac functions, it seems prudent to determine the xenon concentration in the chamber and in the erythrocyte–Kreb’s–Ringer’s solution. Otherwise, it cannot be excluded that, in the current study, an alteration of cardiac functions could not be shown because a full equilibration of xenon gas mixture with the perfusion medium was not reached during the experiment.

A second minor issue is the isolated heart preparation; the Langendorff heart procedure, perfused with crystalloid buffer and gassed with carbogen (i.e., 95% O₂ and 5% CO₂), is certainly a commonly accepted experimental setting. Replacing the carbogen with 20% O₂ and 80% N₂ for equilibration of the crystalloid buffer yields an established model for hypoxia in isolated hearts. In the blood-perfused Langendorff model, the isolated heart usually is perfused with blood from an anesthetized support animal, i.e., with blood at physiologic hemoglobin and hematocrit concentrations. The experimental setting used in the presented work with crystalloid buffer supplemented with erythrocytes at a concentration of 2.8 g hemoglobin/100 ml equilibrated with an oxygen fraction of 0.2 is not widely used, as far as we know. Although we estimate a sufficient oxygen supply from venous oxygen tension and calculation of the oxygen capacity, the astolic left ventricle pressure is lower than expected for a guinea pig Langendorff model. Possible reasons for this observation are a general lack of oxygen or an inhomogenous perfusion of the myocardium. The authors discuss low calcium as a cause for the low left ventricle pressure; nevertheless, hypoxia should have been excluded. Determination of venous lactate or establishment of an additional control group perfused with the erythrocyte–Kreb’s–Ringer’s solution and equilibrated with carbogen would have been easy to realize and very convincing.

Because of the general interest in this subject, we would appreciate any further information the authors could provide.

Sylvia Schroth, M.D., Staff Anesthesiologist
Matthias Reyle-Hahn, M.D., Chief Staff Anesthesiologist
Rolf Rossaint, M.D., Professor and Chairman, Department of Anesthesia, Technical University Aachen, Aachen, Germany
sgillessen@post.klinikum.rwth-aachen.de

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In Reply.—We appreciate the interest expressed by Drs. Schroth, Reyle-Hahn, and Rossaint with regard to our recent article. First, using a Marquette Gas Analyzer (Model 1100; Marquette Gas Analysis Corp., St. Louis, MO), we verified that the oxygen and nitrogen fractions were approximately 20% and 40%, respectively, and 20% and 0%, respectively, to give estimated xenon fractions of 40% and 80%, respectively, in the gas reservoir bags. In addition, effective xenon concentrations in solutions were verified by placing the samples and xenon standards into sealed 1-ml vials and conducting a head-space analysis using the HP 5989 MS-ENGINE Mass Spectrometer (Hewlett-Packard, Palo Alto, CA).

The second concern was that the hearts might become hypoxic with 20% oxygenated perfusate solution, despite the presence of approximately 2.8 g hemoglobin/100 ml perfusate. Most Langendorff preparations that are not perfused with crystalloid solutions are perfused with washed erythrocytes obtained from other species. As the authors pointed out, the venous oxygen tension and pH do not suggest hypoxia. Oxygen consumption of Langendorff hearts is approximately 50–70% of in vitro hearts, and lactate is not produced with carbogen equilibrated in crystalloid perfusate. This is caused in part by the lack of kinetic (stroke) work and decreased potential (isometric) work. Nevertheless, in our erythrocyte-perfused hearts, we conducted a control experiment suggested by the authors; that is, erythrocyte perfusion of hearts with 95% and 5% CO₂ before switching to the reservoirs containing 20% O₂. The change from 95% to 20% O₂ produced no appreciable change in left ventricular pressure, and so we doubt that the hearts became hypoxic. However, we believe that the use of erythrocyte solution might result in a lower left


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ventricular pressure because of buffering of the calcium in the solution. A small increase in viscosity may also contribute to somewhat lower left ventricular pressure.

David F. Stowe, M.D., Ph.D., Professor
Zeljko J. Bosnjak, Ph.D., Professor, Departments of Anesthesiology and Physiology, Medical College of Wisconsin
Georg C. Rehmert, M.D., Postdoctoral Research Fellow
Wai-Meng Kwok, Ph.D., Assistant Professor

To the Editor—Morbidly obese patients undergoing procedures with general anesthesia are at increased risk for deep venous thrombosis (DVT) and subsequent pulmonary embolism. Because of the extreme lower extremity dimension in these patients, properly fitting pneumatic compression devices for the prevention of DVT may not be available. We describe how this problem can be resolved by the combination of two standard-size pneumatic compression cuffs for which inflation is regulated by a single pump.

A 50-yr-old man was scheduled for elective ventral hernia repair during general anesthesia. The patient had multiple high risk factors for the development of DVT: history of DVT, chronic lower extremity phlebitis, morbid obesity (245 kg), and anesthesia was anticipated to last longer than 30 min. Immediately before the procedure, it was noted that the standard-size Flowtron pneumatic compression cuff (Huntleigh Healthcare, Manalapan, NJ) was too small for the circumference of the patient’s calf. Because it was believed to be important to provide this regimen of prophylaxis in the patient, two standard-size Flowtron single-chamber cuffs were joined using Velcro closures (Velcro Industries B.V., Manchester, NH) and applied to each calf (Fig. 1). The two hoses from this assembly were then connected to the two hoses of a Flowtron pump. Because the Flowtron pump alternates inflation between the two hoses, this approach created a dual-chamber sequential cuff and allowed for proper fitting to the large-circumference calf. Postoperatively, the patient was administered subcutaneous heparin for DVT prophylaxis. Clinical signs of DVT or pulmonary embolism were not observed in this patient throughout his 5-day hospital stay and 2-month follow-up period.

Pneumatic compression is a safe and cost-effective method that is equally as effective as heparin for the prevention of DVT. Compression therapy augments peak venous velocity in the deep venous system by 87–302%, reduces stasis, and stimulates intrinsic thrombolytic activity. To evaluate the effectiveness of compression therapy using the aforementioned combination of two standard-size Flowtron single-chamber cuffs, Doppler flow velocities were measured in a morbidly obese volunteer (213 kg; calf circumference, 56 cm). The positive effect of this approach was confirmed; peak venous velocity was augmented from 19.1 to 34.1 cm/s (164%) per compression in the femoral vein and from 11.0 to 30.6 cm/s (278%) per compression in the greater saphenous vein.

In conclusion, the approach we describe allows for effective pneumatic compression therapy in morbidly obese patients in whom standard-size compression cuffs are inadequate.

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Henry U. Weigt, M.D., Postdoctoral Research Fellow, Department of Anesthesiology, Medical College of Wisconsin, Milwaukee, Wisconsin
Michael Georgieff, M.D., Professor and Chairman, Department of Anesthesiology, University of Ulm, Ulm, Germany

zbosnjak@mcw.edu

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Fig. 1. Combination of two standard-size calf Flowtron single-chamber cuffs using Velcro closures.

Kimberly A. Winslow, C.R.N.A.
Maximilian W. B. Hartmannsgruber, M.D., Assistant Professor of Anesthesiology
James H. Chung, M.D., Associate Clinical Professor of Anesthesiology
Albert C. Perrino, Jr., M.D., Associate Professor of Anesthesiology
Department of Anesthesiology, VA Connecticut Healthcare System, Yale University School of Medicine, New Haven, Connecticut
maximilian.hartmannsgruber@yale.edu

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To the Editor:—While conducting research for a comprehensive biography of Henry Isaiah Dorr and the Henry Isaiah Dorr Chair of Research and Teaching in Anaesthetics and Anaesthesia at Harvard University, which is the world’s first endowed professorship in anaesthesia,1 we recognized the startling finding that Dorr appears to have been the first person to hold the title Professor of Anaesthetics and Anaesthesia.

Dorr attended courses at Harvard University from 1869 to 1870 and then embarked on a career as a dentist. From 1875 to 1876, he was a student at the Philadelphia Dental College and earned the doctor of dental surgery degree. He joined the faculty as Demonstrator and was promoted to Adjunct Professor of Dentistry in 1878. Later that same year, a new professorship of Clinical Dentistry was established, and Dorr was appointed. In 1889, his title changed to Professor of the Practice of Dentistry, Anaesthetics and Anaesthesia. This is confirmed by letterhead with the date March 14, 1896, which lists the faculty and their titles (Fig. 1).

The earliest previously known appointment of Professor of Anaesthesia was that of T. S. Buchanan at the Flower School of Medicine in New York City in 1905.2 Dorr’s appointment predated that of Buchanan by 16 yr. Little is discoverable of Dorr’s specific contributions to anesthetic science and practice, but he may have established the first systematic courses of instruction in this discipline.3

On December 13, 1926, 37 yr after his initial appointment as Professor of Anaesthesia and Anaesthetics, the Board of Trustees of Temple University, which the Philadelphia Dental School joined in 1907 to create the Temple University School of Dentistry, elected Dorr Emeritus Professor of Anaesthesia and Anaesthetics. This may represent another world first for him and for the academic anesthesia community.

Dorr proposed the establishment of an endowed Chair in Anaesthetics and Anaesthesia in a letter to President A. Lawrence Lowell of Harvard College on November 7, 1910. The president and fellows formally established the chairship on February 17, 1917. It was occupied for the first time by Henry K. Beecher, M.D., on July 1, 1941.

Edward Lowenstein, M.D., Henry Isaiah Dorr Professor of Anaesthesia, Professor of Medical Ethics, Harvard Medical School, Provost, Department of Anesthesia and Critical Care, Massachusetts General Hospital
Richard J. Kitz, M.D., Henry I. Dorr Distinguished Professor, Faculty Dean for Clinical Affairs, Emeritus, Harvard Medical School, Anesthetist-in-Chief, Emeritus, Department of Anesthesia and Critical Care, Massachusetts General Hospital, Boston, Massachusetts lowenstein@etherdome.mgh.harvard.edu

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To the Editor—

I recently saw a patient for a preanesthetic evaluation who had an Insertable Loop Recorder System (ILRS) (Reveal; Medtronic, Inc., Minneapolis, MN), which is used to continuously record a single lead electrocardiogram in patients with a history of syncopal or presyncopal episodes. The patient was a 43-yr-old woman scheduled to undergo an operative laparoscopy, and the only significant medical information was the presence of dizziness, which was being evaluated at an outside hospital. The patient stated that she had a Holter monitor (GE Marquette Medical Systems, Milwaukee, WI) implanted for 24 h on three separate occasions, and, because it had not revealed abnormalities, the decision was made 6 months previously to insert a more permanent monitoring device. During consultation with her cardiologist, I learned that this device was an internal Holter monitor used to detect arrhythmias.1,2 Nothing unusual was necessary before her proposed surgery, and results of her workup, including the readings from the ILRS, were normal. The team that performed the anesthetic and surgical procedures was informed of this device, and the surgery proceeded uneventfully.

The ILRS has been introduced to circumvent patient compliance and technical limitations. It is the size of a pacemaker and contains two sensing electrodes that are 32 mm apart within its shell. It continuously records a single-lead electrocardiogram that is stored in a circular buffer capable of either one 21- or 42-min segment or three 7- or 14-min segments of recorded rhythm. Using a magnet, the patient activates the device during a syncopal or presyncopal episode, storing the preceding 20- or 40-min segment (storage mode A and B) or 6- or 12-min segment (storage mode C and D). The ILRS stores 1 or 2 min of electrocardiography after activation and one to three events, depending on the storage mode chosen. The device is implanted in the left pectoral region in the subcutaneous fat and has a battery life of 2 yr.3 There are no absolute contraindications for the implantation of this device.4 There is no wiring between the device and the heart, and no treatment is provided by the ILRS.

There are several anesthetic implications in patients with an ILRS. Although there is no need to disable the device for surgery, it is important to discuss the patient’s history and workup with the cardiologist. It may be important to interrogate the device before the proposed surgery to determine whether the ILRS has recorded recent life-threatening arrhythmias.

The shell of the device is made of titanium, and the inside contains ferromagnetic components. Medtronic states that it is safe to use the device in the presence of a magnetic force (such as during magnetic resonance imaging), but that the patient should be warned that pulling sensation may be perceived. Also, it is imperative to collect all data obtained in the ILRS beforehand because the magnetic forces may adversely affect the data collection. Electrocautery is safe, but may cause the device to reset, resulting in lost data. Lithotripsy may damage the device if it is at the focal point of the beam.

David L. Hepner, M.D., Instructor in Anesthesiology, Department of Anesthesia, Perioperative and Pain Medicine, Brigham and Women’s Hospital, Harvard Medical School, Boston, Massachusetts 02115
dhepner@zeus.bwh.harvard.edu

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To the Editor:—During placement of thoracic epidurals or paravertebral blocks when the patient’s gown is tied around his or her neck, the gown tends to slip down and expose the chest or fall into the field. Figure 1 depicts a simple technique for preservation of the patient’s modesty while allowing access to the upper thorax. This technique can be used for any procedure performed with the patient in the seated position, including lumbar epidural or spinal anesthesia (fig. 2). We affix the electrocardiographic contacts on the patient’s anterior or posterior shoulders and snap the tabs of the patient’s hospital gown to the electrocardiographic contacts. These contacts later can be used to attach the electrocardiograph leads.

J. C. Gerancher, M.D.
Sylvia Y. Dolinski, M.D., Assistant Professor of Anesthesiology, Department of Anesthesiology, Wake Forest University School of Medicine, Winston-Salem, North Carolina
dolinski@wfubmc.edu

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