

Can Epidural Fentanyl Induce Selective Spinal Hyperalgesia?

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,^{2,3} high doses of systemic remifentanyl 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, Bernards⁵ 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,⁵ 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.

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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, ignoring inconsistent data. For example, Dr. Cooper mentions that Eisenach cited two studies^{1,2} 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.^{9–13} 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 lipophilic 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

of data that does not support it. We are left with a question in need of interesting, well-designed, human studies to decide the issue.

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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. Rimensberg *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 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,⁸ and it is used in many critical care units.

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In Reply:—The letter from Tusman *et al.* emphasizes the main message of our editorial about mechanical ventilation for acute respiratory failure; that is, the importance of recruiting collapsed alveoli and limiting further lung damage.¹ Their letter also states that the best method of implementing these concepts is not yet agreed on.

Tusman *et al.* suggest that Dr. Amato's "open-lung approach"² does not reach sufficient alveolar pressure and applies excessive levels of positive end-expiratory pressure. We are not so certain. We do not know what the ideal level of airway pressure should be at the beginning and at the end of a mechanical breath for a patient with adult respiratory distress syndrome. Because of the regional heterogeneity present in the lungs of many patients with adult respiratory distress syndrome, "ideal" ventilating pressures probably vary considerably among patients and over the course of the disease.

They suggest that Dr. Pelosi's strategy of repeated sighs is a "resuscitation of an abandoned method proven to be dangerous to the lungs." We disagree. Sighs never have been shown to be dangerous. Although a sustained alveolar pressure of 60 cm H₂O may injure the lung, it is unclear whether such pressure is injurious when applied for a very brief period of time, as is done during a recruitment maneuver. In one physiologic study, the "repeated sighs" strategy was reported to be effective and safe³ and therefore worthy of further consideration.

Tusman *et al.* propose their prescription for mechanical ventilation. We offer the following comments:

1. Limiting peak inspiratory pressure appears to be no better than limiting end-inspiratory volume. Pressure-control ventilation does not appear to be inherently superior to volume-control ventilation.⁴ Ventilator-induced alveolar damage is caused by excessive shear forces that result from pressure and volume.
2. The "critical alveolar pressure" of the diseased lung is not 55 cm H₂O. This value is not known and is likely to vary among different areas of the lung and during different phases of the evolution of acute respiratory failure.

3. We know of no evidence that supports the suggestion that positive end-expiratory pressure increments should be parallel to peak inspiratory pressure increments and that 10 ml/kg tidal volumes are safe. Based on accumulating evidence, they may be injurious.⁴

The intent of our editorial was to communicate to interested clinicians that the way we deliver mechanical ventilation to patients with acute respiratory failure has changed considerably during the past 16 yr. We purposefully did not attempt to dictate specific values of pressure or volume to be used or to be avoided because these values are not known. Only the rigorous testing of physiologically sound hypotheses will improve our ability to treat patients who have acute respiratory failure.

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Pulmonary Aspiration of a Milk-Cream Mixture

To the Editor:—I read with interest the report by Brodsky *et al.*¹ regarding pulmonary aspiration of a milk-cream mixture in an adult. I concur with the authors that a delay would have been appropriate, had the anesthesiologist been aware of the recent ingestion of the mixture.

In reference to pediatric practice, the authors cite the recommendation of Litman² that at least 3 h elapse between breast feeding and surgery. More recently, Ferrari³ reported the results of a survey of hospitals listed in the second edition of the *Directory of Pediatric Anesthesia Fellowship Positions*. This survey showed that most of the institutions have a 4-h restriction for breast milk and a 6-h restriction for nonhuman formula before surgery. The same guidelines are reflected in the recently published *American Society of Anesthesiologists Practice Guidelines for Preoperative Fasting*.⁴

There is evidence that nonhuman milk is cleared more slowly from the stomach than is breast milk.^{5,6} From the anesthesia perspective,

therefore, it would seem prudent to allow at least a 6-h interval before induction of anesthesia in patients who are fed a milk-cream mixture. Because of the high fat content of cream and the compromised nature of patients with a chylothorax, it also would be advisable to perform a rapid-sequence induction in this situation.

The only drawback to waiting 6 h before induction of anesthesia is that the flow of chyle may be past its peak by the time the surgeons expose the thoracic duct. This must be weighed against the risk of pulmonary aspiration, which may, as Brodsky *et al.*¹ reported, be life-threatening.

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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 same 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.

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In Reply:—We appreciate the letters by Cox and Harle *et al.* Because pulmonary aspiration of a milk-cream mixture is rare, our report was intended to describe the clinical course of this complication.¹

Although the safe time interval for delaying anesthesia after oral ingestion of milk or cream is unknown in adult patients, Dr. Cox's suggestion of at least 6 h seems reasonable.

As we stated in our original report, we, similar to Harle *et al.*, believe that administration of this mixture through a jejunostomy tube is preferable because this would reduce the risk of pulmonary aspiration. Dr. Orringer has extensive experience with these patients, so it is reassuring to learn that he and his coauthors are unaware of significant cases of pulmonary aspiration in their patients after application of cricoid pressure. Based on their suggestions, we will continue to apply cricoid pressure to these patients, recognizing that even in healthy patients, this maneuver is not always effective.²

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Venous Air Embolism in Craniostomy 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 craniostomy. 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 craniectomy in infants. Harris *et al.*² used echocardiography for detection of VAE in infants undergoing craniectomy, 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 craniostomy.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 craniostomy repair. However, a possible conclusion drawn from this article could be that only 18% of the children undergoing craniostomy 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.

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In Reply:—As noted in our article, the 82.6% incidence of venous air embolism (VAE) detected by precordial Doppler monitor during craniectomy is higher than previously reported.¹ However, it is not dramatically different from the incidence of 66% reported by Harris *et al.*,² who used a similarly sensitive monitor. One would expect the incidence of VAE to be higher when using a precordial Doppler monitor as compared with only end-tidal carbon dioxide (ETCO₂) monitoring. In the current article, 51.6% of VAE episodes were associated with decreases in ETCO₂ (36% with ETCO₂ decrease only, and 15.6% with hypotension and decreased ETCO₂). However, I do not believe that in the absence of Doppler monitoring, these decreases in ETCO₂ would be diagnosed as episodes of VAE. I would expect that the majority of these episodes of decreased ETCO₂ not associated with changes in hemodynamic status would not be diagnosed and that a portion of those episodes with hypotension would also not be attributed to VAE. Therefore, I do not agree with the observation of Meyer *et al.* that extrapolation of our data would result in a 40% incidence of VAE if monitored with ETCO₂ only.

Our data agree with the suggestion of Meyer *et al.* that precordial Doppler monitoring detects transient episodes of small-volume VAE.

Even without detection, many of these episodes are likely to be self-limited because the surgeons wax bone edges or proceed in other ways to obliterate sources of air entry as a routine part of the procedure. However, use of the Doppler monitor allows the surgeon and the anesthesiologist to evaluate the success of these maneuvers. It is not surprising that, in the study by Meyer *et al.*,³ the incidence of hypotension during VAE is greater. The monitor used to detect VAE is less sensitive than precordial Doppler monitor, which results in the undetected transient, small VAE episodes and causes other episodes of VAE to progress to larger volumes of air entrained than before the diagnosis. In the study by Cucchiara *et al.*,⁴ the incidence of hypotension with VAE was 69% in children, rather than 36%. It should be noted that these children were also in the sitting position. Precordial Doppler monitoring is a noninvasive monitoring tool that adds to the anesthesiologist's understanding of intraoperative events. Children undergoing craniostomy repair are subject to significant hemodynamic changes, including VAE and significant blood loss. The major advantage of precordial Doppler monitoring is that it allows the diagnosis of potentially dangerous VAE before it becomes clinically significant.

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In Vitro Model for Cardiac Function under Xenon Anesthesia

To the Editor:—It is with interest that we read the study by *Stowe et al.*¹ 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."¹ 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-Krebs-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.² Replacing the carbogen with 20% O₂ and 80% N₂ for equilibration of the crystalloid buffer yields an established model for hypoxia in isolated hearts.³ 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.^{4,5} 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 systolic 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-Krebs-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.

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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 prepara-

tions 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 vivo* 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

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.

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Combination of Two Standard Pneumatic Calf Compression Devices to Fit the Morbidly Obese

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.¹ 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.¹ Compression therapy augments peak venous velocity in the deep venous system by 87–302%, reduces stasis, and stimulates intrinsic thrombolytic activity.^{2,3} 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 31.4 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.

Support was provided solely from institutional and/or departmental sources.

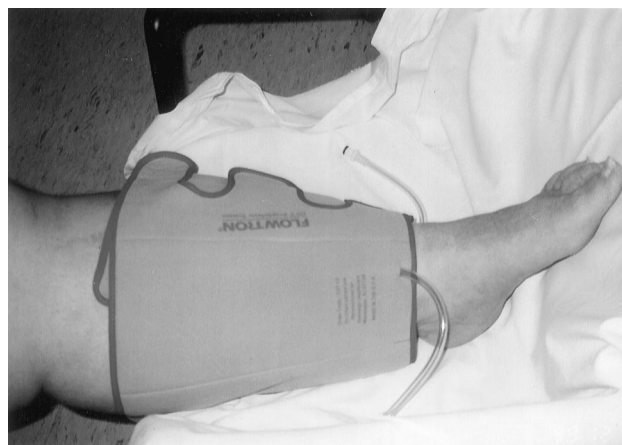


Fig. 1. Combination of two standard-size calf Flowtron single-chamber cuffs using Velcro closures.

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(Accepted for publication May 18, 2000.)

Henry Isaiah Dorr Was the First Person to Hold the Title Professor of Anaesthesia

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 anesthesia,¹ we recognized the startling finding that Dorr appears to have been the first person to hold the title Professor of Anaesthetics and Anesthesia.

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 Anesthesia 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 Tem-

ple University, which the Philadelphia Dental School joined in 1907 to create the Temple University School of Dentistry, elected Dorr Emeritus Professor of Anesthesia and Anesthetics. 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.

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Philadelphia Dental College

... AND ...

Hospital of Oral Surgery.

Cherry St. above Seventeenth.

Philadelphia, March 14th 1896

Fig. 1. Letterhead that documents Dorr's academic titles and his position as treasurer. During the special faculty meeting, March 14, 1896, Dorr expressed his intention to resign because of his precarious health. He lived for 31 yr longer and accumulated funds to endow professorships in anaesthetics at Harvard University, Boston, Massachusetts, and in dentistry at Temple University, Philadelphia, Pennsylvania.

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Anesthetic Implications of a New Implantable Device: The Loop Recorder System

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 modes 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.

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A Modest Proposal

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.

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Fig. 1. Frontal view of electrocardiographic contact placement.



Fig. 2. Lateral view of unencumbered field.

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