

Significance of a Tourniquet to Prevent Thromboembolism during Total Knee Arthroplasty as Assessed by Transesophageal Echocardiography

To the Editor:—In their article, Kato *et al.*¹ report about intraoperative transesophageal echocardiographic findings in 46 patients who underwent total knee arthroplasty, randomly assigned with or without a tourniquet around the thigh. The authors looked for abnormal echogenic findings in the right atrium during the tourniquet inflation phase, and they sought to identify the nature and composition of the echogenic material. During femoral reaming and insertion of the prosthesis, echogenic signals were detected in the right atrium of 27% patients with and 54% patients without a tourniquet. No blood samples, aspirated from the right atrium, contained nonphysiologic material in either the tourniquet or the nontourniquet group.

Although we agree with the conclusions that an inflated tourniquet does not completely prevent pulmonary emboli, we have concerns regarding the following points in the article:

- What is the morphologic substrate of the abnormal echogenic findings? We know from previous studies²⁻⁴ that air, bone cement, cold blood, fat, fresh venous thrombus, and “bone dust” have been aspirated from the right atria of patients with abnormal echogenic findings.
- How do the authors explain the discrepancy between the abnormal transesophageal echocardiographic findings and the impossibility of aspirating nonphysiologic material from the right atrium? Was it only the diameter of the catheter? Were artifacts excluded?
- Did the authors find differences between patients who did and did not show echogenic findings regarding the clinical characteristics or duration of operation or tourniquet inflation?
- Why did the authors look only for pulmonary embolism and not for venous thrombosis? It has been shown that tissue thromboplastin from bone marrow leads to systemic activation of the clotting cascade, lesions of the venous endothelium, and thrombogenesis.⁵ A recent study⁶ in patients after hip arthroplasty found that the incidence of deep-vein thrombosis was associated with abnormal intraoperative echogenic findings.
- When looking for pulmonary embolism, why did the authors rely on perfusion scans, which are known to have a low specificity? Why did they not measure pulmonary artery pressure or use helical computed tomography?
- In how many patients did the authors observe a crossing of echogenic abnormalities from the right into the left atrium? Because a patent foramen ovale is assumed to have a prevalence of 27%, such a transition must be expected, at least in some patients.

- Were causes other than pulmonary embolism excluded as being responsible for the observed cardiorespiratory impairment?
- Did the authors consider that an activation of the clotting cascade could simply derive from the compression of muscle, fibrous tissue, and fat by the tourniquet?
- What are the clinical relevance and practical implications of the study? Is it necessary to provide a more intense prophylaxis of venous thromboembolism? Should we continue the use of tourniquet-inflation during total knee arthroplasty?

In conclusion, we agree with the authors that we should be aware of acute pulmonary embolism during total knee arthroplasty. The important questions, however—which measures should be undertaken to prevent it and how transesophageal echocardiography can be useful in this situation—remain unanswered.

Claudia Stöllberger, M.D.* Josef Finsterer, M.D. *Krankenanstalt Rudolfstiftung, Wien, Austria. claudia.stoellberger@chello.at

References

1. Kato N, Nakanishi K, Yoshino S, Ogawa R: Abnormal echogenic findings detected by transesophageal echocardiography and cardiorespiratory impairment during total knee arthroplasty with tourniquet. *ANESTHESIOLOGY* 2002; 97: 1123-8
2. Pell AC, Christie J, Keating JF, Sutherland GR: The detection of fat embolism by transesophageal echocardiography during reamed intramedullary nailing: A study of 24 patients with femoral and tibial fractures. *J Bone Joint Surg (Br)* 1993; 75:921-5
3. Berman AT, Parmet JL, Harding SP, Israelits CL, Chandrasekaran K, Horrow JC, Singer R, Rosenberg H: Emboli observed with use of transesophageal echocardiography immediately after tourniquet release during total knee arthroplasty with cement. *J Bone Joint Surg (Am)* 1998; 80:389-96
4. Hayakawa M, Fujioka Y, Morimoto Y, Okamura A, Kimmotsu O: Pathological evaluation of venous emboli during total hip arthroplasty. *Anaesthesia* 2001; 56:571-5
5. Koessler MJ, Pitto RP: Fat and bone marrow embolism in total hip arthroplasty. *Acta Orthop Belg* 2001; 67:97-109
6. Pitto RP, Hamer H, Fabiani R, Radespiel-Troeger M, Koessler M: Prophylaxis against fat and bone-marrow embolism during total hip arthroplasty reduces the incidence of postoperative deep-vein thrombosis. *J Bone Joint Surg (Am)* 2002; 84:39-48

(Accepted for publication March 19, 2003.)

In Reply:—We appreciate the interest of Drs. Stöllberger and Finsterer regarding our article, and we thank the Editor for the opportunity to reply to their remarks. Small echogenic findings were not stained with Sudan or Giemsa, so we can only speculate that they represented air or cold blood. The large echogenic findings were strongly suspected of being thrombi, on the basis of their inhomogeneous internal structure. Because echogenic materials were observed in all patients subjected to basically the same operational procedure, they were not considered artifacts. We considered small materials, 2.33 mm (inner diameter of the central catheter) or less, to be air or cold blood. In the tourniquet group, both two patients who had previously undergone total hip arthroplasty in ipsilateral leg showed no echogenic findings during tourniquet inflation. It is impossible to avoid generation of the echogenic materials observed after each manipula-

tion, because they could directly flow from the drainage vein to the inferior vena cava. In the recovery room, cardiopulmonary function returned to its preoperative state in all patients, so we simply observed patients. But we relied on perfusion lung scans in all patients (n = 4) with a postoperative decrease in PaO₂. Hagen *et al.* reported a patent foramen or atrial septal defect in approximately 27% of individuals, but no atrial septal defects was detected by color flow Doppler in any patient in our study.¹ The considerable increase in right atrial and pulmonary artery pressure favored right-to-left shunt flow *via* atrial septal defect, but in the one patient in whom large, grade 3 echogenic materials were detected and atrial septum bulged toward the left atrium, we found no echogenic materials in the left atrium. Considering reports that emboli reduce the cross-sectional area of the pulmonary arterial bed by at least 40% to produce hemodynamic changes, we

agree with the hypothesis that these phenomena may have been caused not only by mechanical obstruction but also by vasoconstriction of the pulmonary vasculature due to the release of neurohumoral substances, such as serotonin released from platelets adhering to the embolus.^{2,3} We consider that other factors (e.g., inflow of bone marrow components and cement monomer) to the systemic circulation and limited mobility in the ward can also lead to activation of the clotting cascade. We think that venous thromboembolism formation can be easily avoided without tourniquet inflation. A high risk of blood transfusion is the only drawback to this method. The most important preventive measure is to be aware of the possibility of this complication, so the real-time investigation, safety, portability, and reproductivity of the evidence provided by transesophageal echocardiography in the operating room offer advantages over the standard technetium-99m lung scans and angiography diagnostic techniques. Furthermore, transesophageal echocardiography is useful for the rapid diagnosis of paradoxical embolism.

Anesthesiology 2003; 99:513

To the Editor:—Lagasse found an anesthesia-related mortality rate of 1 per 13,000 among 184,472 anesthetics administered to patients with American Society of Anesthesiologists Physical Status 1–5 across two hospitals.¹ According to the peer review process by which the cause of each perioperative death was classified, deaths attributable to “limitation of supervision” were not counted as anesthesia-related. For example, if an “attending anesthesiologist is unable to prevent a resident anesthesiologist from committing a human error because of multiple supervisory responsibilities,” the resulting death was classified as a “System Error” and thus not related to anesthesia. However, it would not be unreasonable for patients to view any death caused by a physician administering anesthesia to be anesthesia-related, whether a

David C. Wartier, M.D., Ph.D., handled this exchange as Editor of the Review Article.

Anesthesiology 2003; 99:513

In Reply:—The suggestion to include “limitation of supervision” in the classification of human errors would not change the previously reported anesthesia-related mortality rate of approximately 1 per 13,000 anesthetics.¹ Consider an anesthetic involving a perioperative death judged by peer review to be due to a limitation of supervision on the part of the attending anesthesiologist. That means, by definition, that the attending anesthesiologist was unable to prevent a resident anesthesiologist or nurse anesthetist from committing a human error because of multiple supervisory responsibilities being performed as expected. The human error committed by the resident anesthesiologist or nurse anesthetist would also be recorded as a result of our peer-review process. As noted in the Discussion, “Anesthesia-related mortality was defined as a perioperative death to which human error on the part of the anesthesia provider, as defined by our peer review process, had contributed (p 1613).” The term *anesthesia provider* includes attending anesthesiologists, resident anesthesiologists, and nurse anesthetists at our institution. Therefore, cases involving the system error *limitation of supervision* are counted among the anes-

Nobuya Kato, M.D., Ph.D.*, Kazuhiro Nakanishi, M.D., Ph.D., Shinhiro Takeda, M.D., Ph.D., Zen'ichiro Wajima, M.D., Ph.D., Tetsuo Inoue, M.D., Ph.D., Shinichi Yoshino, M.D., Ph.D., Ryo Ogawa, M.D., Ph.D. *Chiba Hokusoh Hospital, Nippon Medical School, Chiba, Japan. n-kato@mva.biglobe.ne.jp

References

1. Hagen PT, Scholz DG, Edwards WD: Incidence and size of patent foramen ovale during the first 10 decades of life: An autopsy study of 965 normal hearts. *Mayo Clin Proc* 1984; 59:17–20
2. McIntyre KM, Sasahara AA: The hemodynamic response to pulmonary embolism in patients without prior cardiopulmonary disease. *Am J Cardiol* 1971; 28:288–94
3. Malik AB: Pulmonary microembolism. *Physiol Rev* 1983; 63:1114–207

(Accepted for publication March 19, 2003.)

© 2003 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

Limitation of Supervision

fatal error was caused by a resident anesthesiologist or an attending anesthesiologist.

Because the perspective of patients may differ from the opinion of anesthesiologists in this regard, and because Lagasse's analyses are otherwise compelling, it would be useful to know the death rate calculated from his data set with perioperative mortality attributable to “limitation of supervision” classified as anesthesia-related.

John Hartung, Ph.D.* James E. Cottrell, M.D. *State University of New York, Downstate Medical Center, Brooklyn, New York. jhartung@downstate.edu

Reference

1. Lagasse RS: Anesthesia safety: Model or myth? A review of the published literature and analysis of current original data. *ANESTHESIOLOGY* 2002; 97:1609–17

(Accepted for publication April 11, 2003.)

© 2003 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

thia-related deaths because they also involve a human error by a supervised anesthesia provider. Interested parties may find more detailed descriptions of this peer-review model in previous issues of *ANESTHESIOLOGY*.^{2,3}

Robert S. Lagasse, M.D. Weiler Division of Montefiore Medical Center, Bronx, New York. BobLagasse@yahoo.com

References

1. Lagasse RS: Anesthesia safety: Model or myth? A review of the published literature and analysis of current original data. *ANESTHESIOLOGY* 2002; 97:1609–17
2. Lagasse RS, Steinberg ES, Katz RI, Saubermann AJ: Defining quality of perioperative care by statistical process control of adverse outcomes. *ANESTHESIOLOGY* 1995; 82:1181–8
3. Levine RD, Sugarman M, Schiller W, Weinsel S, Lehning EJ, Lagasse RS: The effect of group discussion on interrater reliability of structured peer review. *ANESTHESIOLOGY* 1998; 89:507–15

(Accepted for publication April 11, 2003.)

Anesthesiology 2003; 99:514

© 2003 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

Preoperative Epidural Placement in Elderly Patients with Hip Fractures: A Request for Essential Information

To the Editor:—I read with interest the recent article by Matot *et al.*¹ The reduction in adverse preoperative cardiac events was significant enough to warrant study termination following the interim analysis. However, for other healthcare providers to provide these benefits to their patients, the authors' technique must be strictly adhered to. Missing from the article's Methods section is any mention of premedication before epidural placement and patient positioning for catheter placement. Although these may, at first, seem to be trivial issues, I would suggest otherwise. As the authors specifically noted, pain is increased with even a slight movement of the fractured extremity (see Results and fig. 2 of the article). Because the study hypothesis was "that the use of epidural analgesia during the stressful *presurgical* period would decrease the incidence of adverse cardiac events," it is essential to know if and how the authors decreased the often-excruciating pain during patient positioning for catheter placement. If others attempt to reproduce the favorable outcomes found in this investigation but

dramatically increase patients' pain during catheter placement, they may inadvertently cause an increased stress response and decrease (or negate) the effectiveness of this important preoperative intervention. Therefore, I would like to request that the authors clarify their technique for minimizing pain and the resulting stress response during catheter placement.

Brian M. Ilfeld, M.D. University of Florida College of Medicine, Gainesville, Florida. bilfeld@ufl.edu

Reference

1. Matot I, Oppenheim-Eden A, Ratrot R, Baranova J, Davidson E, Eylon S, Peyser A, Liebergall M: Preoperative cardiac events in elderly patients with hip fracture randomized to epidural or conventional analgesia. *ANESTHESIOLOGY* 2003; 98:156-63

(Accepted for publication April 18, 2003.)

Anesthesiology 2003; 99:514

© 2003 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

In Reply:—I take this opportunity to thank Dr. Ilfeld for his comment and showing keen interest in my recent article.¹ As he suggested, positioning the patient for catheter placement often causes excruciating pain, which in the present study was relieved with the intravenous administration of the short-acting opioid, alfentanil (maximal dose of 10 $\mu\text{g}/\text{kg}$ in divided doses).

Idit Matot, M.D. Hadassah Hebrew University Medical Center, Jerusalem, Israel. matoth@cc.huji.ac.il

Reference

1. Matot I, Oppenheim-Eden A, Ratrot R, Baranova J, Davidson E, Eylon S, Peyser A, Liebergall M: Preoperative cardiac events in elderly patients with hip fracture randomized to epidural or conventional analgesia. *ANESTHESIOLOGY* 2003; 98:156-63

(Accepted for publication April 18, 2003.)

Anesthesiology 2003; 99:514-5

© 2003 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

Endotracheal Tube Foreign Body after Intubation with a Vital Signs, Inc., Lightwand

To the Editor:—Many reports document the potential complications of lightwands in clinical practice.¹⁻⁶ A complication we have encountered is the creation of a foreign body after intubating with the Vital Signs lightwand (Vital Signs, Inc., Totowa, NJ).

A 32-yr-old, 81-kg woman with diabetes mellitus, asthma, and hypertension was scheduled to undergo elective laparoscopy for lysis of intraperitoneal adhesions. After preoxygenation in the operating room, a routine induction was administered (fentanyl 50 μg , lidocaine 40 mg, propofol 200 mg, mivacurium 20 mg). The patient was ventilated without difficulty with bag-mask ventilation. After appropriate muscle relaxation was verified with a peripheral nerve stimulator, airway manipulation and intubation was completed. The technique for intubation was a Vital Signs lightwand. The nonlubricated lightwand was threaded through a 6.5-mm endotracheal tube with the tip of the lightwand proximal to the Murphy eye of the endotracheal tube. The lightwand was bent at a 75-degree angle about 3 cm proximal to the tip. After adequate transillumination of the glottis, the endotracheal tube was advanced into the trachea without difficulty. On withdrawal of the lightwand, however, increased resistance was experienced. The

lightwand was removed with visible damage to its distal tip (fig. 1). Visual inspection confirmed the presence of a white foreign body from the damaged lightwand in the upper part of the endotracheal tube. The foreign body was removed easily from the endotracheal tube without extubating the patient. The remainder of the anesthetic administration proceeded uneventfully.

The complication may have been due to a structural flaw in the lightwand device, by not lubricating the lightwand before insertion into the endotracheal tube, or by a combination of both. Previous to the current case, we had performed over 200 lightwand intubations using 6.5-mm endotracheal tubes and nonlubricated Vital Signs lightwands with no complications. Subsequent to this case, however, we encountered two identical de-sheathings. We have queried Vital Signs to determine if any recent manufacturing changes had occurred in the construction of the lightwand (*e.g.*, change in plastic quality or assembly). A written reply received in June 2002 from Gail D. Rice, Compliance Engineer at Vital Signs stated that no changes had occurred and that this complication would not occur with a lubricated stylet. A randomized study might be useful in assessing the potential risks and benefits of lubricating *versus* not lubricating stylets. It also might be prudent for Vital Signs to consider a change in the design of their lightwand. If possible, elimination of the elevated ridges would lead to a decreased likelihood of friction, snagging, and plastic degloving.

Funding was provided solely within the Department of Anesthesiology, Indiana University Medical Center, Indianapolis, Indiana.

Anesthesiology, V 99, No 2, Aug 2003

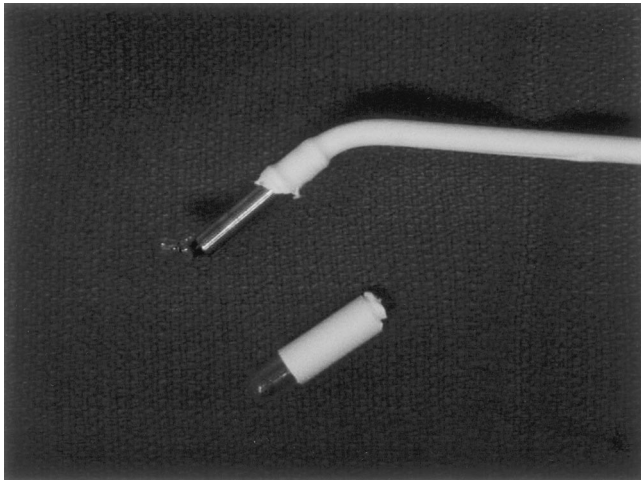


Fig. 1. Lightwand with the de-sheathed plastic tip exposing the lightbulb.

Bradley A. Stalter, M.D.* David S. Currier, M.D. *Indiana University School of Medicine, Indianapolis, Indiana. b.stalter@anesthesia.iupui.edu

References

1. Debo RF, Colonna D, Dewerd G, Gonzalez C: Cricoidytenoid subluxation: complication of blind intubation with a lighted stylet. *Ear Nose Throat J* 1989; 68:517-520
2. Stone DJ, Stirt JA, Kaplan MJ, McLean WC: A complication of lightwand nasotracheal intubation. *ANESTHESIOLOGY* 1984; 61:780-781
3. Cohn AI, Joshi S: Lighted stylet intubation: greasing your way to success. *Anesth Analg* 1994; 78:1200-1207
4. Dowson S, Greenwald KM: A potential complication of lightwand-guided intubation. *Anesth Analg* 1992; 74:165-172
5. Moukabary K, Peterson CJ, Kingsley CP: A potential complication with the lightwand. [Letter] *ANESTHESIOLOGY* 1994; 81:523-524
6. Szigeti CL, Baeuerle JL, Mongan PD: Arytenoid dislocation with lighted stylet intubation: case report and retrospective review. *Anesth Analg* 1994; 78:185-186

(Received for publication January 7, 2003.)

Anesthesiology 2003; 99:515

© 2003 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

Air-locked Epidural Filter

To the Editors:—A 20-g epidural catheter was uneventfully placed 4 cm into the epidural space at L3-4 in a patient in active labor (Perifix Continuous Anesthesia kit; B. Braun Medical, Bethlehem, PA). After the initial dose, local anesthetic was continuously infused using a syringe pump, through a 0.2- μ m epidural filter.

During the course of the patient's 21 h of labor, she required several syringe refills and additional boluses of local anesthetic for breakthrough pain. Approximately 1 h after the fourth bolus had been given and the third 60-ml syringe had been connected, the anesthesiologist was informed that the epidural pump was signaling an "occlusion." Although it was still possible to inject normal saline through the catheter, it was now requiring significantly more force than when the catheter had been initially placed. The force required for injection did not alter with a change in the patient's position or after the catheter had been pulled back 1 cm. However, after the epidural filter was removed from the infusion line, the force necessary to successfully inject was noted to have returned to its initial level. With the epidural filter removed, the epidural infusion was restarted. There were no further problems, and the patient completed her labor and delivery without incident.

Subsequently, the epidural filter was examined closely; no defect was grossly visible. No debris was seen inside except for what appeared to be a small amount of entrapped air. Although the small bubbles may have been insignificant individually, it is hypothesized the

accumulated trapped bubbles reached a sufficient volume to obstruct the filter and the flow of infusate.

To test this hypothesis, air was deliberately introduced into an epidural filter after it was primed, and measurements were taken of the amount of air necessary to cause obstruction at an infusion rate of 15 cc per hour. As little as 0.3 ml of air in the Braun/Periflex epidural filter was sufficient to cause the infusion pump to signal obstruction. This air-lock phenomenon was also possible in another brand of epidural filter (Arrow International, Reading, PA, 0.7 ml air to obstruction). The amounts of air needed to obstruct the filters correspond to their priming volumes (Braun/Periflex 0.35 ml; Arrow 0.75 ml).

Complete or partial obstruction to epidural injections and infusions have been attributed to catheter-related problems such as kinking, knotting, blood clotting, or stretching,¹ and to manufacturing defects in the catheter, the filter,² and the connection screw-cap.³

Christopher C. Lin, M.D. Kaiser Foundation Hospital, Bellflower, California. christopher.c.lin@kp.org

References

1. Khalouf FK, Kunkel FA, Freeman J: Stretching with obstruction of an epidural catheter (letter). *Anesth Analg* 1987; 66:1202-3
2. Clark VA, Dickson J: Blocked epidural filter (letter). *Anaesthesia* 1993; 48:537
3. Chandhok D, Vijayakumar E: An unusual case of epidural catheter obstruction (letter). *ANESTHESIOLOGY* 1999; 91:895-6

(Accepted for publication March 19, 2003.)

Anesthesiology 2003; 99:515-6

© 2003 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

Preoperative Antibiotic Prophylaxis: The Role of the Anesthesiologist

To the Editor:—Antibiotics given for prophylaxis of surgical site infections are known to be significantly more effective when administered

immediately before surgical incision.¹ Many institutions, therefore, expect the anesthesiologist to assume the responsibility for antibiotic administration.^{2,3} However, anesthesiologists receive little, if any, training in antibiotic therapy during residency or through continuing medical education.³

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

Anesthesiology, V 99, No 2, Aug 2003

Pressure from our institutional infection control group to take responsibility for antibiotic administration led to much debate in our department as to what role we were willing to assume. Although most of us were comfortable with the actual administration of the antibiotic, we did not feel qualified to make the antibiotic selection. Furthermore, we have found that although most of our surgeons have considerably more knowledge about antibiotic prophylaxis than we do, many lack confidence in drug selection, particularly in complex cases. Although several articles have advocated the training of anesthesiologists in antibiotic administration,^{2,3} to our knowledge the issue of who should select the drug has not been addressed in the literature.

After much discussion, a compromise was reached whereby the infection control committee at our institution provides a protocol for antibiotic administration for the prophylaxis of surgical site infections. This protocol specifies antibiotic choice and dose by procedure, including specific patient considerations. It also includes alternatives in the event of allergy to the first-line drug. The protocol is condensed onto laminated cards, which are attached to our anesthesia machines. The infection control committee is responsible for intermittently updating the antibiotic protocol based on new developments in antibiotic therapy and evolutions in local epidemiology. We have not attached our current protocol because of the rapid evolution of antibiotic

therapy and regional differences in epidemiology, but we would happy to share our current protocol on request.

The antibiotic protocol for surgical prophylaxis provides both anesthesiologists and surgeons with the comfort that we are giving our patients the optimal antibiotic regimen. Appropriate administration of the recommended antibiotic is a responsibility we are willing to assume to ensure timely administration of the drugs and to do our part to reduce surgical site infections.

R. David Wartens, M.D.,* Peter Szmuk, M.D., Evan G. Pivalizza, M.B.Ch.B., Ralf Gebhard, M.D., Tiberiu Ezri, M.D. *The University of Texas Medical School at Houston. Robert.D.Wartens@uth.tmc.edu

References

1. Matuschka PR, Cheadle WG, Burke JD, Garrison RN: A new standard of care: Administration of preoperative antibiotics in the operating room. *Am Surg* 1997; 63:500-3
2. Cheng EY, Nimphius N, Hennen CR: Antibiotic therapy and the anesthesiologist. *J Clin Anesth* 1995; 7:425-39
3. Cheng EY, Nimphius N, Hennen CR: Training in antibiotic administration. *Anesth Analg* 1992; 74:619-20

(Accepted for publication April 9, 2003.)

Anesthesiology 2003; 99:516-7

© 2003 American Society of Anesthesiologists, Inc. Lippincott Williams & Wilkins, Inc.

Cardioprotective Effects of Volatile Anesthetics in Cardiac Surgery

To the Editor:—There is increasing experimental evidence that volatile anesthetics may have a cardioprotective effect, either by decreasing the extent of the reperfusion injury or by a pharmacologic preconditioning effect. These findings may ultimately have an impact on the anesthetic practice for patients with ischemic heart disease.¹ Recent studies in coronary surgery patients have suggested that the use of volatile anesthetic agents may result in the preservation of left ventricular function and lower troponin I levels after cardiopulmonary bypass.^{2,3} However, variables such as differences in cardioplegic solutions, adequacy of myocardial protection, presence of cardiopulmonary bypass, adequacy of surgical revascularization, and many others may affect the extent of perioperative myocardial ischemia and, ultimately, the success of the intervention. This results in a complex situation in which the potential beneficial effects of volatile anesthetics may depend on these determining factors.

With respect to this issue, we would like to report some of the results of the yearly quality assessment report of one of our cardiac surgical centers. Last year was significant, because this center was rebuilt to provide for the implementation of a volatile anesthetic regimen during the cardiac surgical procedures. Previously, the anesthesia machines and cardiopulmonary bypass circuits were not equipped with vaporizers. In addition, the monitoring equipment did not include an end-tidal anesthetic concentration measurement system. From January 2 to June 26, 2002 (period A), 107 patients underwent coronary artery surgery. During this period, anesthesia consisted of a combination of midazolam (0.2–0.3 mg/kg) and high-dose sufentanil (5–8 μ g/kg) without the use of volatile anesthetics. From July 2 to December 31 (period B), 91 patients had coronary surgery. During this second period, anesthesia consisted of the same combination of drugs

Support was provided solely from institutional sources.

Editor's Note: This Letter, by Philippe J. Van der Linden *et al.*, indicates that there may be clinical evidence that volatile anesthetics protect myocardium against ischemia and reperfusion injury. The *Journal* Symposium, "Preconditioning against Ischemia and Reperfusion Injury," to be held at this year's Annual ASA Meeting on October 14, 2003, in San Francisco, California, will be devoted to an in-depth discussion of this phenomenon. For more information, see the Special Announcement that will appear in the September 2003 issue.

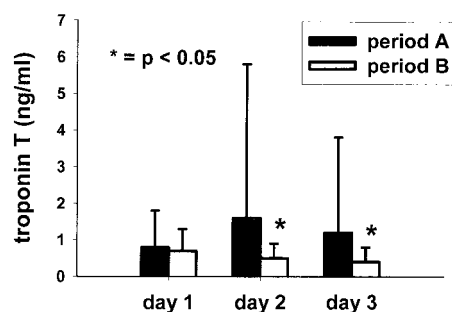


Fig. 1. Troponin T levels at day 0 (arrival at the intensive care unit), 1 (day 1), and 2 (day 2) postoperatively during both time periods. Data are mean \pm SD.

with a reduction in the dose of midazolam and sufentanil used, but with the administration of sevoflurane (0.5–2%) throughout the procedure. When the different outcome variables were analyzed at the beginning of this year, troponin T levels (measured using monoclonal antibodies immunologic assay; Elecys 2010®, Roche, Basel, Switzerland) were found to be consistently lower in the patients operated on in period B (fig. 1). The need for inotropic support for weaning from cardiopulmonary bypass was significantly lower in period B (36% of patients in period B *vs.* 52% in period A; $P = 0.019$). In addition, the incidence of low cardiac output (defined as the need for inotropic support for a cardiac index below $2.0 \text{ l} \cdot \text{min}^{-1} \cdot \text{m}^2$) was also significantly lower in period B (19% of patients in period B *vs.* 35% in period A; $P = 0.014$). Patient characteristics, medication, and intraoperative data were similar during both time periods (table 1). The same surgical and anesthetic team performed all procedures, and the hemodynamic strategies were the same. The only difference was the introduction of sevoflurane in the anesthetic regimen, supporting a possible cardioprotective effect of this agent in the setting of coronary surgery. This observation seemed interesting because it suggested that the effects of sevoflurane observed previously,³ in a specific setting of intermittent cross-clamping with the use of the nucleoside transport inhibitor lidoflazine, were also present with a different surgical tech-

Table 1. Patient Characteristics

	PERIOD A	PERIOD B
Female/male	31/76	23/68
Age (yr)	64 ± 11	64 ± 10
Ejection fraction (%)	55 ± 14	57 ± 14
Body mass index (kg/m ²)	27.8 ± 4.3	28.9 ± 4.6
No. of bypasses	4 (range, 2–6)	4 (range, 2–6)
Aortic cross-clamp time (min)	88 ± 19	95 ± 24
Cardiopulmonary bypass time (min)	110 ± 23	116 ± 28
Length of stay, ICU (days)	5 ± 10	3 ± 3
Length of stay, hospital (days)	12 ± 10	12 ± 5

ICU = intensive care unit.

nique using continuous aortic cross-clamping and cardioprotection with cold crystalloid cardioplegia. Another interesting point was that patients undergoing valve surgery also demonstrated a difference in postoperative troponin T release between both time periods (period A [n = 18] at day 0: 1.1 ± 0.8 ng/ml, at day 1: 1.3 ± 1.0 ng/ml, at day 2: 1.1 ± 0.8 ng/ml; period B [n = 28] at day 0: 0.8 ± 0.7 ng/ml, at day 1: 0.7 ± 0.5 ng/ml, at day 2: 0.6 ± 0.3 ng/ml; difference between periods at day 1 and 2 statistically significant for $P < 0.05$)

Although these observations lack the power of a prospective randomized study, they suggest that the beneficial effects of a volatile anesthetic regimen on myocardial function represent a genuine phenomenon in the clinical setting of cardiac surgery. They provide additional circumstantial evidence for a potential cardioprotective effect of sevoflurane. Clinical investigations on a large patient population scale should now be undertaken to definitively elucidate this question.

Philippe J. Van der Linden, M.D., Ph.D., Anne Daper, M.D., Anne Trenchant, M.D., Stefan G. De Hert, M.D., Ph.D.*
*University Hospital Antwerp, Edegem, Belgium. sdehert@uia.ac.be

References

1. Wartier DC, Kersten JR, Pagel PS, Gross GJ: Anesthetic preconditioning: Serendipity and science. *ANESTHESIOLOGY* 2002; 97:1-3
2. Belhomme D, Peynet J, Louzy M, Launay JM, Kitakaze M, Menasché P: Evidence for preconditioning by isoflurane in coronary artery bypass graft surgery. *Circulation* 1999; 100(suppl II):340-4
3. De Hert SG, ten Broecke PW, Mertens E, Van Sommeren EW, De Blier IG, Stockman BA, Rodrigus IE: Sevoflurane but not propofol preserves myocardial function in coronary surgery patients. *ANESTHESIOLOGY* 2002; 97:42-9

(Accepted for publication May 21, 2003.)