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:

1. 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.

2. 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?

3. 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?

4. 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 echocardiographic findings.

5. 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?

6. 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.

7. Were causes other than pulmonary embolism excluded as being responsible for the observed cardiorespiratory impairment?

8. 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?

9. 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.

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References


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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. 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 reproducibility of the evidence provided by transesophageal echocardiography in the operating room offer advantages over the standard technetium-99m lung scans and angio graphic diagnostic techniques. Furthermore, transesophageal echocardiography is useful for the rapid diagnosis of paradoxical embolism.

References


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Limitation of Supervision

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 attributed 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 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.

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Reference


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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 1615).” The term anesthesia providers includes attending anesthesiologists, resident anesthesiologists, and nurse anesthetists at our institution. Therefore, cases involving the system error limitation of supervision are counted among the anesthesia-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

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References


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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.

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Reference


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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/kg in divided doses). 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 μg/kg in divided doses).

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Reference


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Air-locked Epidural Filter

To the Editor:—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-μm 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, 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 amount 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, and to manufacturing defects in the catheter, the filter, and the connection screw-cap.

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References

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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 cardioprotective 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 anesthesiologist 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 heart-lung bypass and midazolam and sufentanil 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 with a reduction in the dose of midazolam and sufentanil used, but with the administration of sevoflurane (0.5–2.0%) 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; Elecsys 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 \( L \cdot \text{min}^{-1} \cdot \text{m}^2 \)) 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 previously3 in a specific setting of intermittent clamping with the use of the nucleoside transport inhibitor lidoflazine, were also present with a different surgical tech-
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.

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Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>PERIOD A</th>
<th>PERIOD B</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female/male</td>
<td>31/76</td>
<td>23/68</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>64 ± 11</td>
<td>64 ± 10</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>55 ± 14</td>
<td>57 ± 14</td>
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<tr>
<td>Body mass index (kg/m²)</td>
<td>27.8 ± 4.3</td>
<td>28.9 ± 4.6</td>
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<tr>
<td>No. of bypasses</td>
<td>4 (range, 2–6)</td>
<td>4 (range, 2–6)</td>
</tr>
<tr>
<td>Aortic cross-clamp time (min)</td>
<td>88 ± 19</td>
<td>95 ± 24</td>
</tr>
<tr>
<td>Cardiopulmonary bypass time (min)</td>
<td>110 ± 23</td>
<td>116 ± 28</td>
</tr>
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<td>Length of stay, ICU (days)</td>
<td>5 ± 10</td>
<td>3 ± 3</td>
</tr>
<tr>
<td>Length of stay, hospital (days)</td>
<td>12 ± 10</td>
<td>12 ± 5</td>
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</table>

ICU = intensive care unit.

References


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