

## CORRESPONDENCE

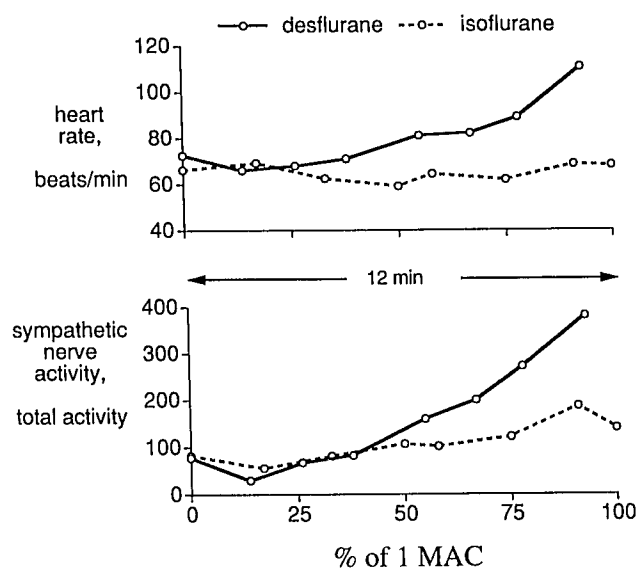


Fig. 1. Heart rate and sympathetic nerve activity responses of two healthy young volunteers receiving isoflurane ( $n = 1$ ) or desflurane ( $n = 1$ ) by mask. No premedication was given and, in each, anesthesia was induced with 2.0 mg/kg propofol. The inhaled anesthetic was added to the inspired oxygen and titrated so that the rate of rise of the end-tidal anesthetic concentration over a 12-min period to a desired 1 MAC was identical in each volunteer. Total activity is calculated as the frequency of sympathetic bursts per 100 cardiac cycles times the mean burst amplitude.

propofol). In our second volunteer, we matched the end-tidal rate of rise to 1 MAC with isoflurane by giving high inspired isoflurane concentrations (3–4%) at a high fresh gas flow rate (8 l/min, 100%  $O_2$ ). We cannot be certain that the anesthetic uptake in the central nervous system was similar, and admittedly, two subjects do not constitute a study, but despite a matched (equipotent) rise in alveolar concentration, we noted profound sympatho-excitation and tachycardia with desflurane (fig. 1).

The second concern expressed by Eger and Weiskopf was that subject randomization was poor and that the same subjects did not participate in both limbs of the study. The isoflurane group was added for comparison to the desflurane study as dictated by the expert reviewers that critiqued the original manuscript submission. The concern that subjects were chosen from different volunteer pools is unwarranted because all subjects were from the same population, *i.e.*, young, healthy students. Appropriately, unpaired statistical comparisons were employed.

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## Factors Affecting Outcome in Patients Undergoing Peripheral Vascular Surgery: I

*To the Editor:*—I applaud the efforts of Rosenfeld *et al.*<sup>1</sup> and Christopherson *et al.*<sup>2</sup> for addressing the complex and controversial issue of regional *versus* general anesthesia for patients undergoing peripheral vascular surgery. Unfortunately, these studies disregard the role of the surgeon in the overall rate of early graft failure in patients undergoing vascular surgery. For example, their overall incidence of early graft failure (13%) is very high when compared to results from other major medical centers. Bandyk *et al.*,<sup>3</sup> in a consecutive series of 353 patients undergoing lower extremity revascularization procedures, reported an early graft failure rate of 5%. The major causes of early graft failure in this series were related to inadequate outflow, poor-quality vein, and technical errors on the part of the surgeon. In another series, Bandyk *et al.*<sup>4</sup> found that 79% of early graft revisions were necessary to correct specific anatomic

problems with the graft and that relatively few (<7%) were secondary to unexplained thrombosis. Unfortunately, Rosenfeld *et al.* and Christopherson *et al.* did not report on the type of graft used (synthetic *vs.* natural vein) or the quality of the anastomosis as demonstrated by intraoperative angiography or angiography. Lacking such crucial information, it is difficult to conclude that general anesthesia alone was the major contributory factor to their observed high early graft failure rate.

Bode *et al.*<sup>5</sup> recently presented the results of 307 patients random-

\* Bode RH, Lewis DP: Graft occlusion after peripheral vascular surgery vs. regional anesthesia, *Manual of the Society of Cardiovascular Anesthesiologists*. 1993, pp 244–245.

ized to general, spinal, and epidural anesthesia for lower extremity revascularization procedures and found that the overall incidence of acute graft failure was very low (1.6%) and did not significantly vary among the anesthesia techniques. Furthermore, in another study of 423 patients randomized to general, spinal, and epidural anesthesia, Bode *et al.*<sup>5</sup> found no evidence that regional anesthesia reduced the incidence of major cardiovascular events in a high-risk patient population (86% diabetic patients). One cannot ignore the intensity of monitoring used in their studies by both the anesthesia team (pulmonary and systemic arterial pressure monitoring) and the surgical team (vascular angioscopy and hourly Doppler monitoring), which most likely contributed to their low complication rate, be it in the incidence of acute graft failure or major cardiac complications. One can make a case about the value of intensive hemodynamic monitoring as noted also by Berlauk *et al.*<sup>5</sup> in these high-risk patients. The studies by Rosenfeld *et al.* and Christopherson *et al.*, despite the great effort by the co-investigators, will remain equivocal at best if they ignore the role of the other key player, namely, the surgeon.

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*In Reply:*—The role of the surgeon in the overall rate of graft failure in our study was not disregarded, as suggested by Kupeli. Christopherson *et al.* reported that randomization to epidural or general anesthesia was stratified by surgeon, thus eliminating the possibility that experience or referral pattern would be maldistributed between anesthesia groups.<sup>1</sup> Kupeli also overlooked our report of the surgery performed and graft material used. Patients were stratified into low risk (femoral bypass graft and femoral aneurysm repair) and high risk (grafts to the popliteal artery and other distal sites) for occlusion. This resulted in similar numbers of high-risk patients in each group; furthermore, the same number in each high-risk group received synthetic grafts (Christopherson *et al.*, page 429).

Although it may be possible to find lower rates of revascularization in the early postoperative period than reported in our paper, it is also possible to find higher rates. As we stated,<sup>1</sup> our rate of in-hospital reoperation of 13% is within the range reported in the literature.<sup>2-5</sup> Bode *et al.* should be commended for the very low rate of acute graft failure that they found in the study of 307 patients randomized to epidural spinal or general anesthesia.<sup>6</sup> However, as we explained, (Christopherson *et al.*, page 432) when an outcome occurs rarely, it is usually not possible to find a significant difference between treatment groups even when such a difference exists. With a low rate of graft failure such as reported by Bode *et al.*,<sup>6</sup> it is improbable that

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they had an adequate sample size to have power to find a significant difference if there was a difference related to type of anesthesia.

We agree with Kupeli that intensive hemodynamic monitoring, such as that reported by both Berlauk *et al.*<sup>6</sup> and Bode *et al.*<sup>6</sup> might greatly lower the rate of vascular graft occlusion because of improved flow through the graft. We mentioned this in our discussion (Christopherson *et al.*, page 431). However, invasive hemodynamic monitoring has never been shown to alter patients' tendencies for thrombosis. We showed such an alteration related to anesthesia.<sup>7</sup> We agree with Kupeli that postoperative vascular graft occlusion is a complex phenomenon that may be caused by the difficulty of the procedure, the skill of the surgeon, the use of synthetic rather than natural vein grafts, and blood flow through the graft. Despite these contributions to graft occlusion, the type of anesthesia and its attendant effects on hemostatic function also appear to be very important determinants, as shown by us<sup>7</sup> and Tuman *et al.*<sup>2</sup>

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