

goal in every case. Despite best intentions on the part of designers, equipment interactions can be unpredictable. Users must exercise caution when combining equipment from different manufacturers.

This case serves as a reminder of the benefits of equipment standardization among hospital departments. Although ideal, equipment standardization may not be practical in individual institutions. A convenient alternative for a hospital is to standardize on sensors. Towards this end, Nellcor licenses its patented sensor calibration technology or supplies its pulse oximetry modules to selected companies meeting our quality and safety standards. Nellcor sensors may be used with pulse oximeters from Hewlett-Packard, Medical Data Electronics, Mennen, Ivy, Drager, and Siemens that have been *specifically designed* to meet Nellcor sensor standards. Nellcor sensors should be used *only* with Nellcor monitors or oximetry modules, or with instruments licensed to use Nellcor sensors—a warning that is clearly stated in all Nellcor operator's manuals and sensor package inserts. As companies design

new equipment meeting our sensor standards, those instruments can be added to the list of compatible equipment.

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REFERENCE

1. Murphy KG, Secunda JA, Rockoff MA: Severe burns from a pulse oximeter. *ANESTHESIOLOGY* 73: - , 1990

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Regional Anesthesia and Aspirin

To the Editor:—The case report by Camann *et al.*¹ describing the use of epidural anesthesia for cesarean delivery in a patient with a transplanted heart highlights what some might consider a therapeutic dilemma—that is, the use of epidural anesthesia in patients taking low-dose aspirin. The question that needs to be answered is: Does the benefit of epidural anesthesia outweigh the perhaps theoretical risk of epidural hematoma should a vessel be punctured? Low-dose aspirin (60–80 mg/day) is now commonly used in many patients who are likely to benefit from epidural anesthesia—for example, women with pregnancy-induced hypertension (PIH) and patients requiring vascular surgery. Epidural hematomas are rare, but many of those described have occurred in association with anticoagulant or antiplatelet therapy.^{2,3} Conversely, Rao and El-Etr reported no problems with patients given intraoperative heparin after the performance of the spinal or epidural block.⁴ Low-dose aspirin inhibits platelet aggregation, and since effective therapy should prolong the bleeding time, this simple test should be carried out in all patients in whom epidural or spinal anesthesia is to be performed. If the bleeding time is significantly prolonged (> 12 min or > 15 min?) or if there are other factors that might predispose to bleeding, such as in PIH, with a decreasing platelet count or prior administration of heparin, then the block should not be performed without a careful risk–benefit analysis.

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REFERENCES

1. Camann WR, Goldman GA, Johnson MD, Moore J, Greene M: Cesarean delivery in a patient with a transplanted heart. *ANESTHESIOLOGY* 71:618–620, 1989
2. Ruff RL, Dougherty JH: Complications of lumbar puncture followed by anticoagulation. *Stroke* 12:879–881, 1981
3. Maguire T, Doli S: Spinal subarachnoid haematoma after lumbar puncture in a patient receiving antiplatelet therapy. *Anesth Analg* 62:777–779, 1983
4. Rao ILK, El-Etr AA: Anticoagulation following placement of epidural and subarachnoid catheters: An evaluation of neurological sequelae. *ANESTHESIOLOGY* 55:618–620, 1981

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In Reply:—The letter by O'Sullivan raises, once again, the controversial issue of regional anesthesia in patients receiving aspirin therapy. Although aspirin is a known inhibitor of platelet aggregation and is known to be associated with a prolonged bleeding time, regional anesthesia is commonly performed in these patients. For example, large numbers of orthopedic patients, in whom aspirin and other nonsteroidal antiinflammatory agents are commonly used, routinely undergo lower extremity joint arthroplasty with spinal or epidural anesthesia. No case reports have yet appeared describing epidural hematomas in this patient

population. The report by Mayumi, to which O'Sullivan refers, concerns a patient who was receiving ticlopidine, a new antiplatelet drug, and not aspirin.¹ Furthermore: 1) the coagulation profile (including platelet count and bleeding time) was normal in that patient; 2) the patient had a preexisting compression fracture of the tenth thoracic vertebrae; and 3) multiple attempts were required during the spinal anesthetic.

The report by Rao and El-Etr, as well as others concerning regional anesthesia for vascular surgery, all seem to confirm the safety of this type of anesthesia when heparin is administered *after* the anesthetic is

performed.² These reports, although encouraging, are not directly relevant to the issue of antiplatelet therapy at the time of placement of the block. In contrast, Benzon reports on 239 epidural and 7 spinal anesthetics in patients receiving aspirin; no neurologic complications were noted.* Furthermore, Horlocker recently reported 805 patients given epidural or spinal anesthesia while receiving antiplatelet therapy; none developed hemorrhagic complications or postoperative neurologic sequelae.³

Certainly, risk-benefit analysis is an important aspect of any medical intervention. In our patient, we believed that epidural anesthesia clearly was the technique of choice for reasons of hemodynamic stability and our patient's desire to be awake during the delivery of her infant.

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*Benzon HT, Brunner EA, Vaisrub N: Bleeding time and nerve blocks after aspirin. *Regional Anesthesia* 9:86-89, 1983

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Questioning the Benefits of Epidural Anesthesia in Hemorrhagic Shock

To the Editor:—The recent paper by Shibata *et al.*¹ presents important data concerning hemorrhagic shock and the "survival benefit" of "upper-level" epidural anesthesia. Although we acknowledge the difficulties inherent in this study, we do have some questions as to the interpretation of the data.

Hemorrhagic hypotension elicits a complex array of hormonal and vascular changes which are altered by ablating sympathetic neuronal function. Brunner *et al.*² and other investigators have shown that in response to hemorrhagic hypotension, "capacitance vessels" (splanchnic organs) vasoconstrict. This leads to the expulsion of "reservoir blood" to the systemic circulation and shunts blood away from splanchnic organs. Hypotension also results in the release of catecholamines and neuropeptides from the adrenal,³ secretion of renin by the kidney, etc. Despite the existence of these well-known peripheral sympathetic responses, the authors lead the reader to believe the unsubstantiated claim that the "survival benefit" of "upper epidural" is secondary to decreased catecholamine release.

First, the catecholamine response to shock is not "overactivity," but compensatory. Its magnitude is established by the need for blood pressure and volume support. Since the adrenal response to hypotension cannot be singularly ablated by epidural or spinal anesthesia, plasma catecholamine concentration can be viewed only as a marker for peripheral sympathetic tone, and not as the cause of this tone.

Second, the authors state that alterations in blood flow are responsible for the "survival benefit" of "upper-level" epidural; however, no measurements of regional blood flows were obtained. While it may be appealing to assume that "upper-level" epidural increases survival by altering the normal compensatory vascular effects of the other abdominal organs innervated by the peripheral sympathetic system, the current data demonstrate simply that a slower heart rate during hemorrhagic shock increases myocardial survival. Preservation of the myocardium during systemic hypotension by slowing the heart rate is well known.⁴ This is an important point ignored by the current study. Although the authors conclude that "reductions of MAP and of CI following induced hypovolemia were similar in all groups," no other determinants of myocardial dysfunction were measured, and their data are skewed by animal nonsurvival. Thus, it seems to us more likely that ablation of the cardiac accelerators in the "upper-level" epidural group provides

the protection to myocardial damage not afforded in the "lower-level" epidural or control groups.

Third, the authors state that the "maximal bleeding volume" did not differ among the groups of animals. However, they did not establish that a comparable shock stimulus was induced in each animal group, nor did they establish that the time needed to obtain a blood pressure of 40 mmHg and "maximal" bleeding volume was similar. Since the reader may be comparing apples to oranges, these data are important. For example, 20 min after blood loss, the mean arterial blood pressure of the "upper-level" epidural group was decreased from 84 to 40 mmHg and pulmonary capillary wedge pressure (PCWP) decreased from 9 to 7 mmHg. In contrast, the mean arterial blood pressure of the control group was decreased from 116 to 40 mmHg and PCWP from 11 to 7 mmHg. Systemic vascular resistance (SVR) was unchanged in both groups. It seems unlikely that similar volumes of blood were extracted from both groups of animals at this time point. If, however, the extracted blood volumes were similar, for some unknown reason, then the time course to arterial pressure decompensation must have been shorter in the epidural group. Further, if both the blood volume and the time course to decompensation were similar, then it can be said that splanchnic vascular, adrenal, and cardiac mechanisms do not compensate hypotension, or that shock was not truly induced in all three animal groups by the first 20 min.

Finally, the authors do not explain why baseline values of heart rate, epinephrine, and norepinephrine are so much higher than those of previous studies. Indeed, the level of "background" anesthesia (0.5% halothane, 50% N₂O, and pancuronium) may provide "light" anesthesia and high baseline sympathetic tone. This is evidenced by the 64% decrease in mean plasma epinephrine concentration after epidural anesthesia. High sympathetic tone and the selected agents for the "minimal" background anesthetic may have affected the results of the current study. These effects include the effects of halothane on the baroreceptor response to hypotension.⁵ In addition, during "upper-level" epidural and hemorrhagic shock, plasma norepinephrine concentration did not rise. Since norepinephrine production is not confined to the adrenal medulla or abdominal paragangli, an increase in circulating levels is expected. Thus, the authors' findings are inconsistent with what is now known about peripheral norepinephrine release.⁶

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REFERENCES

1. Mayumi T, Dohi S: Spinal subarachnoid hematoma after lumbar puncture in a patient receiving antiplatelet therapy. *Anesth Analg* 62:777-779, 1983
2. Rao ILK, El-Etr AA: Anticoagulation following placement of epidural and subarachnoid catheters: An evaluation of neurological sequelae. *ANESTHESIOLOGY* 55:618-620, 1981
3. Horlocker TT, Wedel DJ, Offord KP: Does preoperative antiplatelet therapy increase the risk of hemorrhagic complications associated with regional anesthesia? *Anesth Analg* 70:631-634, 1990

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