

performed.<sup>2</sup> 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.<sup>3</sup>

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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## Questioning the Benefits of Epidural Anesthesia in Hemorrhagic Shock

*To the Editor:*—The recent paper by Shibata *et al.*<sup>1</sup> 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.*<sup>2</sup> 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,<sup>3</sup> 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.<sup>4</sup> 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% N<sub>2</sub>O, 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.<sup>5</sup> 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 paraganglia, an increase in circulating levels is expected. Thus, the authors' findings are inconsistent with what is now known about peripheral norepinephrine release.<sup>6</sup>

Clearly, there is much work to be done. We do agree with the authors that the current study does not "preclude" the dangers of epidural anesthesia, especially in the presence of uncorrected hypovolemia. We concur also that the study of splanchnic blood flow and concentrations of other mediators in addition to plasma catecholamine are required.

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*In Reply:*—We appreciate the opportunity to respond to the letter from Dr. Jordan and Dr. Dickstein. We agree that the first two points identify weaknesses in our study and that it may well be the slower heart rate rather than decreased catecholamine concentrations that were of principal importance.

In response to the third point: The advantages and disadvantages of various experimental models of hemorrhagic shock have been discussed.<sup>1</sup> An ideal experimental model should fulfill several requirements: reproducibility, predictable outcome, economic feasibility, and reasonable similarity to clinical reality. It is obvious that few, if any, of the currently known models fulfill all of these requirements. We are now using a single blood-withdrawal method (30 ml/kg) in order to confirm whether or not the survival benefits of UEA are reproducible in other experimental models.

In response to the last point: There is no question that general anesthesia has an effect on the development of experimental shock and results in a different baseline level of functional activity. However, since many reports from several countries have spoken of the benefits of the combined use of light general anesthesia and epidural anesthesia, we do not believe that the situation we present is excessively artificial or difficult to extrapolate to clinical circumstances.

Moreover, we do not agree that our findings are inconsistent with current knowledge. Circulating norepinephrine is released from sympathetic nerve terminals, while the adrenal glands release a mixture of epinephrine and norepinephrine into the blood stream.<sup>2</sup> Harrison *et al.*<sup>3</sup> reported the release of norepinephrine from the nerve endings to be significantly diminished in hemorrhagic shock. Therefore, our findings for circulating catecholamine are quite consistent with what is known about peripheral norepinephrine release. Indeed, Stanek *et al.*<sup>4</sup> also found that in dogs receiving epidural anesthesia, the plasma norepinephrine concentration was not increased by hypovolemia.

In conclusion, the survival benefit as described was applicable only to a particular situation, *i.e.*, "in dogs lightly anesthetized with halothane

and nitrous oxide and in experimental hemorrhagic shock when UEA is performed before hemorrhage and when the mean arterial blood pressure is constant (40 mmHg)." We do not claim that this study has universal validity in the relationship between hemorrhagic shock and UEA.

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