CORRESPONDENCE

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.1 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.3 Harrison et al.3 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.4 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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