tension during epidural anesthesia in conscious dogs. ANESTHESIOLOGY 73:694–702, 1990

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In Reply—We appreciate Dr. Stevens’s interest in our recent papers and the comments made. In our opinion, the statement that “sympathetic effe rrents were largely, if not completely eliminated by high epidural anesthesia” in these experiments appears to be entirely appropriate.

Certainly, none of the mentioned criteria (e.g., plasma catecholamines and paralysis of the nictitating membrane) represents a direct quantitative measure of efferent sympathetic drive. The only suitable method and gold standard in this regard is a direct recording of spike traffic from multiple sympathetic efferents, the neurophysiologic correlate of sympathetic tone. For obvious reasons, these measurements cannot and could not be performed during the same experiments in these conscious unsecdated dogs.

However, there is further, albeit indirect evidence for sympathetic blockade in our dogs, be it partial or complete. In a previous study, we measured an increase in both fore- and hindlimb skin temperatures with epidural anesthesia in these dogs using similar dosages of bupivacaine 0.5%. In addition, we noted that the usual reflex blood pressure and heart rate increase induced by bilateral clamping of the carotid loop arteries is either completely abolished or markedly (>80%) attenuated during epidural anesthesia. This indeed argues for a partial, if not complete sympathetic block.

Finally, whether sympathetic blockade was complete or only partial does not appear to be particularly relevant with regard to our conclusions. In fact, if sympathetic blockade were only partial, our results would even underestimate the impact of the experimental findings.

Accordingly, our main conclusions, i.e., that blood pressure is supported by endogenous vasopressin during epidural anesthesia and that sympathetic blockade by epidural anesthesia blunts the cardiovascular response to hypoxemia, appear well supported by the presented data, and stand.

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REFERENCES

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Inadvertent Intraarterial Placement of a Sheath Introducer while Using the Raulerson Syringe

To the Editor—Percutaneous central venous placement of a sheath introducer has always been a task requiring a considerable degree of skill. The recently introduced Raulerson syringe by Arrow International (Reading, PA) in which the spring wire guide is threaded directly through the syringe and needle as a one-step modification of the Selinger technique, promises to facilitate this procedure, with less risk of contamination, trauma, wire guide misplacement, and air embolism.

We report here a case of inadvertent intraarterial placement of the sheath introducer with use of the Raulerson syringe.

A 65-year-old woman was brought to the operating room for urgent coronary artery bypass grafting. Because the patient was very anxious despite intravenous sedation (morphine 0.15 mg/kg and scopolamine 0.3 mg), general anesthesia was induced prior to insertion of a pulmonary artery catheter. After satisfactory induction and uneventful tracheal intubation, we attempted right internal jugular percutaneous placement of a sheath introducer using the technique of English et al., with the Raulerson syringe. Blood was aspirated and did not appear to be arterial. The wire guide was advanced and the introducer was inserted. Blood then was noted to be flowing through the side port in a pulsatile manner, and when transduced, demonstrated an arterial waveform. The sheath introducer was immediately withdrawn, and despite application of direct pressure, an expanding hematoma was noted.

In light of both left main coronary artery stenosis and unstable angina in this already anesthetized patient, it was decided to surgically explore the neck. Exploration of the right neck was performed via an anterior incision, which was an extension of the median sternotomy. The hematoma was found to originate from the thyrocervical trunk. There