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Anesthesiology

In Reply—We appreciate Dr. Steven’s interest in our recent
papers1,2 and the comments made. In our opinion, the statement that
“sympathetic efferents were largely, if not completely eliminated by
high epidural anesthesia” in these experiments appear to be entirely
appropriate.

Certainly, none of the mentioned criteria (e.g., plasma catecholamines
and paralysis of the nictitating membrane) represents a direct quan-
titative 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 sedated dogs.

However, there is further, albeit indirect evidence for sympathetic
blockade in our dogs, be it partial or complete. In a previous study,3
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 blood pressure and
heart rate increase induced by bilateral clamping of the carotid
loop arteries is either completely abolished or markedly (>80%) at-
tenuated 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 conclu-
sions. In fact, if sympathetic blockade were only partial, our results
would even underestimate the impact of the experimental findings.

Accordingly, our main conclusions,1,2 i.e., that blood pressure is sup-
ported 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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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 Sel-
dinger 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 pul-
monary 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.2
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 he-
matoma was found to originate from the thyrocervical trunk. There