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In Reply:—We agree that intravenous fluid administration and low doses of intravenous catecholamine agents are effective and clinically proven treatments for hypotension following spinal injection of local anesthetics and clonidine. However, the rationale for our study examining intrathecal neostigmine was not to propose its use as a "pressor" to supplant these therapies. Rather, we are examining, in this study and in ongoing research, two hypotheses: (1) analgesia from spinal α_2 -adrenergic agonists is mediated *via* acetylcholine (ACh release); and (2) ACh stimulates, whereas α_2 -adrenergic agonists inhibit, preganglionic sympathetic neuron activity.

It follows from these hypotheses that addition of neostigmine to clonidine for intrathecal administration would enhance clonidine's analgesia while counteracting its sympatholytic effect. Should this be the case, a combination injection would reduce clonidine's major

side effects: sedation (which is dose-related) and hypotension. Clearly, we do not need a spinal "pressor," nor has adequate pre-clinical toxicity assessment been presented warranting intrathecal neostigmine use in humans. However, this line of investigation likely will yield better understanding of spinal pharmacology of analgesia and sympathetic nervous system control and may be directly clinically applicable.

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Should Epidural Clonidine Be Used for Reflex Sympathetic Dystrophy?

To the Editor:—We commend the work of Rauck *et al.*,¹ which describes the effects of epidural clonidine for the treatment of reflex sympathetic dystrophy (RSD). We also commend their statement that "the role for such invasive therapy in symptomatic treatment and functional recovery in RSD remains to be assessed." Their study raises several questions that should be addressed at this time:

1. Is there sufficient data to support their conclusion that transdermal clonidine produces analgesia only in its area of application, whereas epidural clonidine produces more "extensive" analgesia? Contrary to Davis *et al.*,² we have found that the effects of transdermal clonidine are not confined to the borders of the patch.³⁻⁵ Given the relatively high rate of serious complications (25% infections) and the cost associated with the use of epidural catheters in their study, should patients first fail a trial with a safer and less

expensive treatment (transdermal clonidine) before a test with epidural clonidine is considered?

2. Is the "analgesic" effect of epidural clonidine a conditioned response to the sedative effect of clonidine experienced by the patients in the study, or might it be the result of the sedation/relaxation produced by the clonidine?

To substantiate the potential therapeutic benefits of epidural clonidine, the authors refer to a book that allegedly supports their position that chronic opioid administration is *not* "recommended" in the treatment of RSD. However, the assertion in the chapter they cite is not supported by reference to clinical data. That is, it represents merely an opinion. On the other hand, published clinical data⁶ and our clinical experience support the position that oral opioids should be considered a viable treatment option in select patients with chronic

CORRESPONDENCE

neuropathic pain, including RSD. Clearly, in the era of healthcare reform, our strength as a specialty will depend more and more on our willingness to explore safer and more cost-effective therapeutic options to anesthetic procedures.

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In Reply:—Our patients already had received a multitude of conservative and aggressive therapies (table 2) with unsuccessful results. We agree that conservative therapies should be employed when a reasonable chance for relief exists. Many of these patients had received systemic clonidine at some point in the course of their disease. Unlike the experience cited by Kirkpatrick and Miller, we have not seen good results in patients with advanced disease who receive systemic clonidine. We would add that the three references cited by Kirkpatrick and Miller in support of transdermal fentanyl include one letter and two abstracts. No peer-reviewed, placebo-controlled trials have demonstrated its efficacy. We currently are involved in a double-blind study comparing systemic clonidine with epidural clonidine in this population of patients.

We feel certain that the epidural effect was analgesic in nature and not sedative. Figures 1 and 3 demonstrate that the analgesic effect of epidural clonidine was not dose-dependent, whereas the sedative effect increased with dose, providing strong evidence that patients could report the difference between analgesia and sedation.

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Cricoid Pressure for Preventing Gastric Insufflation in Infants and Children

To the Editor:—In a recent study of the effectiveness of cricoid pressure for preventing gastric inflation,¹ the single, nonblinded investigator relied on breath sounds during cricoid pressure to determine the adequacy of ventilation and on detection of a "gurgle" by

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The use of opioids in chronic nonmalignant pain has become a controversial issue in recent years. Whether one supports or discounts their use in this patient population, we would agree with Kirkpatrick and Miller that "our strength as a specialty will depend more and more on our willingness to explore safer and more cost-effective therapeutic options." The use of clonidine, epidurally and systemically, represents an effort to expand our therapeutic horizons.

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auscultation of the upper abdomen to indicate gastric insufflation. Because the investigators did not measure exhaled volumes or volumes of gases in the stomach and there is no mention of end-tidal CO₂ or CO₂ waveform, one cannot be certain that cricoid pressure