

CORRESPONDENCE

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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