

pulmonary resection.⁶ The safety of this method used for five days is demonstrated in our experience of over 800 patients.

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REPLY

We thank Drs. Eng and Sabanathan for their comments on our paper. The technique of continuous intercostal nerve block described in our study was based on the findings that an injected bolus of local anaesthetic in large volume into a single intercostal space can spread to and achieve anaesthesia in multiple adjacent intercostal levels.¹⁻³ We assume that our technique would have a similar target site of anaesthetic activity as the conventional intercostal nerve block approach since the landmark for needle and subsequent catheter insertion, i.e., 7-8 cm from the posterior midline, was similar. The major difference from the conventional method is that by inserting two indwelling catheters, repeated local anaesthetic injections may continue to block peripheral intercostal nerves without the unpleasant experience of multiple needle punctures.

As clearly demonstrated, the technique of continuous extrapleural "intercostal" nerve block used by Drs. Eng and Sabanathan is quite different from ours in that the indwelling catheter is placed in the paravertebral space under direct vision.⁴⁻⁵ Local anaesthetic deposited in this region will block not only intercos-

tal nerves, but also posterior primary rami. In our opinion, this method is more appropriately named continuous extrapleural "paravertebral" nerve block since the site and mechanism of action would be similar to that of conventional paravertebral nerve block. With this direct approach to the paravertebral space, it is likely that potential risks of dural puncture, postural headache and inadvertent subarachnoid injection would be minimized.

Despite major differences in the mechanisms of action, there are similarities between the technique of Eng and Sabanathan and that of ours. In both cases, the common goal is to achieve the desired block with a direct approach, not blindly. Under direct vision, catheters are more likely inserted successfully with the least amount of trauma and a higher degree of accuracy.

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Tumour invasion of the brachial plexus: management of pain with intrapleural analgesia

To the Editor:

I would like to present a case that illustrates a different use of intrapleural analgesia in a cancer pain patient. This elderly man presented to our department with intractable pain caused by metastatic invasion of his left brachial plexus, from an adenocarcinoma of the palate. He had not responded to radiotherapy, and was experiencing severe pain (VAS 7), poorly controlled with MS-Contin, MOS, and carbamazepine. He was also having side effects from this regimen.

It was decided to treat the patient with sympathetic blockade of the cervical and upper extremity area to rule out any sympathetically maintained pain. Because of metastasis in the anterior cervical triangle, a stellate ganglion block was deemed contraindicated. We elected to

use intrapleural injection of local anaesthetics to produce blockade of the upper thoracic sympathetic chain, as described by Reiestad *et al.*¹ A catheter was inserted in the intrapleural space as described earlier,²⁻⁴ directing the tip towards the left apex. After radiological confirmation of the position, the catheter was injected daily with bupivacaine 0.375%, 30 ml. The injections were performed in the right lateral position, with 30° Trendelenburg tilt, to insure distribution of the LA at the apex. This position was maintained for 30 min, and the blood pressure, heart rate, respiratory rate and oxygen saturation were monitored.

Following each injection, there was evidence of sympathetic blockade (Horner and increased temperature), and decreased level of pain. The patient received a total of 11 daily injections, until no more benefit was gained. Pain was easily controlled with a reduced amount of narcotics, and no side effects were noted. The patient remained comfortable until his death, seven weeks after his discharge from hospital.

This report represents another example of the efficacy of intrapleural analgesia (and the sympathetic block associated with the technique) in the treatment of chronic pain.

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Oral clonidine preanaesthetic medication does not alter plasma lidocaine elimination during epidural anaesthesia in lightly anaesthetized patients

To the Editor:

Although clonidine has been utilized with local anaesthetics,^{1,2} clonidine could inhibit hepatic blood flow (HBF)-limited elimination of these drugs.^{2,3} Thus, during epidural anaesthesia, clonidine preanaesthetic medication may affect the hepatic elimination of lidocaine which is sensitive to HBF changes.

Fourteen female patients (32-52 yr) were assigned randomly to receive either oral clonidine $4.89 \pm 0.14 \mu\text{g} \cdot \text{kg}^{-1}$ (mean \pm SE) ($n = 7$), or control no medication. After epidural anaesthesia (L₂₋₃) using lidocaine 2% 18 ml, general anaesthesia was maintained with enflurane-N₂O-oxygen. The plasma lidocaine concentration was measured at 5-60 min after epidural injection of lidocaine by homogenous enzyme immunoassay. During the early distribution phase, plasma lidocaine concentrations in the clonidine group showed a trend to be slightly higher than in the control group (Figure), but did not reach statistical significance. There was no difference between the two groups regarding plasma lidocaine concentrations during the elimination phase (Figure).

The trend toward higher plasma lidocaine concentrations during the distribution phase in the clonidine group is suggestive of enhanced systemic absorption of lidocaine by clonidine from the epidural space. However, the effect of oral clonidine of $5 \mu\text{g} \cdot \text{kg}^{-1}$ upon cardiac output, and thus upon HBF, seems to be minimal because of similar plasma lidocaine concentrations in both groups. These

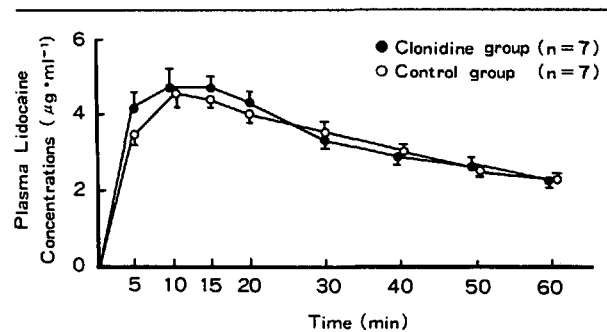


FIGURE Plasma concentration of lidocaine after epidural injection of 2% lidocaine 18 ml in clonidine group receiving oral clonidine $5 \mu\text{g} \cdot \text{kg}^{-1}$ and control group receiving no medication.