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 radiolocal 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 (L_{2-3}) using lidocaine 2% 18 ml, general anaesthesia was maintained with enflurane- N_2O -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.

results suggest that the preanaesthetic use of oral clonidine does not reduce the clearance and/or metabolism of lidocaine in the liver.

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