

**POSTOPERATIVE INTERCOSTAL BLOCK**

Sir,—I read with interest the account by Baxter and colleagues [1] of their use of continuous intercostal nerve blockade after cardiac surgery. Their expressed intention was to study the effect of intercostal nerve blockade on pain relief and pulmonary function after cardiac surgery and to document the occurrence of any adverse effects they encountered. Whereas the excellent conduct of their trial allowed them to document their information accurately, I feel they have fallen prey to a very common fault of deriving incorrect conclusions based on the data they have collected.

It is well documented that patients undergoing major surgery suffer a reduction in respiratory capacity and that this may persist for quite some time after surgery [2–6]. To infer, as Baxter and colleagues did [1], that there is little to recommend intercostal analgesia over opioid analgesia merely because pulmonary function is not improved is quite erroneous. Based on their data, they may claim that intercostal nerve blockade provided significantly better pain relief than opioid analgesia and that, by their criteria, no improvement in pulmonary function was attended by this improved quality of analgesia. They may, therefore, conclude that there are factors other than improvement in pain relief which affect postoperative pulmonary function. This is already well documented. If they had looked at pulmonary function in the postoperative period before and after intercostal nerve block “top-ups” were required, these authors would have been able to document the precise effect of the nerve blockade on respiratory function, as this would have been the only variable.

In my first paper on continuous intercostal nerve blockade [7], I measured respiratory peak flow in the postoperative period after operation. In view of the controversy regarding the spread of local anaesthetic within the chest wall [6,7], could the spread of local anaesthetic within the chest wall [6,7], could intrapleural spread of local anaesthetic be the mechanism of action of this technique? In any case, it does not appear to matter whether the catheter is in the “extrapleural space” or intrapleural to produce analgesia, but pneumothorax has been reported after operation. In view of the controversy regarding the spread of local anaesthetic within the chest wall [6,7], could intrapleural spread of local anaesthetic be the mechanism of action of this technique? In any case, it does not appear to matter whether the catheter is in the “extrapleural space” or intrapleural to produce analgesia, but pneumothorax has been reported.

I measured respiratory peak flow in the postoperative period after each “top-up” as Murphy found in his unblinded study (in a different patient population) which he quotes if, overall, this confers no significant improvement in the pattern of recovery, incidence of complications, and so on. Indeed, Murphy suggested in his paper that more studies are required to assess whether the technique actually reduces pulmonary complications; in this patient population it would appear that it does not.

We agree with Murphy that individual intercostal blocks are usually performed at the angle of the rib, and we changed our technique only for the purpose of convenience for this study. This did not appear to affect the efficacy of the blocks and, indeed, satisfactory analgesia was obtained. However, several reports have shown that good analgesia is produced with an intrapleural catheter [3–5]. Indeed, we have seen a few of our catheters within the pleural cavity and good analgesia was obtained after operation. In view of the controversy regarding the spread of local anaesthetic within the chest wall [6,7], could intrapleural spread of local anaesthetic be the mechanism of action of this technique? In any case, it does not appear to matter whether the catheter is in the “extrapleural space” or intrapleural to produce analgesia, but pneumothorax has been documented with a catheter in both sites.

Similarly, we do not advocate performance of these blocks in patients with a coagulopathy from any cause. All blocks in our study were performed after the coagulation profile had improved by a mean of 37%. This is in direct conflict with the results of Murphy’s own [2], have not been controlled or double-blind and are therefore subject to problems of bias, etc.

Pulmonary dysfunction after major surgery is already well documented, and we studied the effects of 36 h of continuous intercostal analgesia on the recovery of postoperative pulmonary function after cardiac surgery. From this point of view, it is irrelevant if there is a 37% improvement in peak flow after each “top-up” as Murphy found in his unblinded study (in a different patient population) which he quotes if, overall, this confers no significant improvement in the pattern of recovery, incidence of complications, and so on. Indeed, Murphy suggested in his paper that more studies are required to assess whether the technique actually reduces pulmonary complications; in this patient population it would appear that it does not.

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D. F. Murphy
Perth

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returned to normal, and in the patient with the complication to which Murphy refers, coagulation studies were normal before institution of the intercostal block. We know of no studies documenting the safety or otherwise of intercostal blocks with or without catheters after prior administration of, for example, low-dose heparin for DVT prophylaxis, although a conservative approach to this has been advocated by Bromage for extradural blocks.

The conclusion of our study was that, with intercostal catheters the patients may be more comfortable, but the recovery of pulmonary function in this patient population was no better than with i.v. narcotics, and we consider that, if the latter regimen had been optimized, it may have been as effective and a less invasive way of achieving the same degree of comfort.

A. D. Baxter
F. O. Jennings
J. F. Flynn
Ottawa

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USE OF NALOXONE IN OPIOID-INDUCED ANAPHYLACTOID REACTION

Sir,—Mast cells have been shown to possess opioid receptors, and subcutaneous injection of endorphins, morphine, codeine and pethidine has been shown to cause weal reactions with mast cell degranulation [1]. We report a case of an anaphylactoid reaction to dihydrocodeine (DF 118) apparently responding to i.v. naloxone.

A 20-yr-old female with a family history of asthma was admitted 2 h after an overdose of an unspecified number of dihydrocodeine tablets. She was drowsy but co-operative. Pupil size was pinpoint, there was no sign of bronchospasm, and her heart rate was 90 beat min⁻¹ with a systemic arterial pressure of 110/60 mm Hg. There was moderate facial oedema with a widespread urticarial rash, and a number of scratch marks were present. Naloxone 0.8 mg was given i.v. There was an immediate reversal of the drowsiness accompanied by pupillary dilatation. Within 15-20 min there was a reduction in vasomotor flushing and the patient reported a marked improvement in pruritus. Facial oedema improved over the next few hours. Peripheral blood eosinophil and C-1 esterase inhibitor concentrations were normal. Salicylate and paracetamol were not detected in blood.

Many clinical phenomena appear to involve mast cell degranulation without a clear relationship to allergy. These include cold, cholinergic and solar urticaria [2]. I.v. opioids induce increases in plasma histamine concentration in humans, confirming the capacity of these agents to cause mast cell degranulation [3, 4]. Naloxone by subcutaneous injection has been shown to inhibit morphine-induced weals, in a dose-dependent manner [1]. The clinical response to i.v. naloxone seen in our patient is consistent with the above observations. We suggest that the use of i.v. naloxone in anaphylactoid type reactions produced by opioids may be beneficial, possibly by blocking further mast cell degranulation caused by circulating opioids.

M. Z. Panos
S. Burnett
B. G. Gazzard
London

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