POSTOPERATIVE PARAVERTEBRAL BLOCKS FOR THORACIC SURGERY

Sir,—We were particularly interested in the findings of Drs Conacher and Kokin’s radiological study of paravertebral block [1], but feel that this is potentially misleading. Our observations from a continuing series of 45 cases of paravertebral block contrast significantly with the findings in their small study. Using a modification of Moore’s technique we have injected 0.75% bupivacaine 5 ml, diluted with Conray 420, into the paravertebral space under x-ray control and observed the spread. We demonstrated a widespread distribution of contrast on A-P and lateral x-ray, as well as on CT scan, and this was confined to the paravertebral space in only 20% of cases. We have concluded that extradural spread is of more than “the occasional relevance” implied by the authors since this occurred in 70% of the patients in our series. Furthermore, it contributed significantly to the sensory blockade, which was more widespread in those with extradural spread than in those in whom paravertebral distribution occurred alone. Bilateral radiological spread of contrast was usually associated with ipsilateral analgesia.

We cannot agree with the authors’ implication that disabling hypotension does not occur with paravertebral block. Profound hypotension, secondary to sympathetic blockade, occurred in 9% of our patients and was not always associated with demonstrable extradural spread.

We agree that the risk of intrapleural injection is substantial. However, none of the 7% with intrapleural spread in our series developed a pneumothorax. Pain relief in this latter group was very poor.

It may be that the different spread of contrast that we have noted is related to the different patient population rather than minor variations in technique. Our patients were not postoperative thoracotomy cases with potentially distorted tissue planes, but Pain Relief Clinic patients with undisturbed paravertebral anatomy. This would not support the authors’ contention that extradural spread occurs only in the presence of pathology (e.g. fractured ribs).

We confirm the authors’ finding that a single paravertebral injection provides excellent analgesia over several dermatomes, but the extent of the block is unpredictable and the risk of significant complications exists. We would also question the wisdom of paravertebral injection of neurolytic solutions—a technique sometimes advocated for terminal pain relief.

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REFERENCE

Sir,—Thank you for showing me the informative and informed comments of Drs Purcell-Jones and Justin. The correspondents have perhaps been a bit journalistically destructive by not quoting the sentence about relevance in full. It was not our intention to deny the possibility that extradural spread may occur in the absence of identifiable factors such as local trauma, but merely to point out that the presence of anatomical dislocation may make the extradural spread a preferential route on occasion. My impression from various studies and, in particular, from my own observations of resin injection in cadavers would confirm their view that extradural spread from paravertebral injection is more common than has been demonstrated radiologically heretofore.

Radiological contrast studies are often limited because spread of marker is only demonstrated in two dimensions and its actual site can only be presumed. My own resin studies confirm the cadaver ones of Moore [1] that significant spread occurs alongside and across the vertebral bodies where it may, as demonstrated in vivo [2], cross the midline. In this context, and I am hazarding guesses, it may be difficult to distinguish, from x-ray plates, whether spread is actually extradural or just confined to the sides or the fronts of vertebral bodies. The CT scans mentioned by the correspondents would therefore be very informative in defining whether extradural spread in some cases is actual or just apparent. I wonder if this may be one explanation for the clinical disparity between extradural spread of local anaesthetic being demonstrated radiologically and the finding that analgesia and sensory deficit are ipsilateral.

Turning to the issue of hypotension as a result of local anaesthetic paravertebral blockade that the correspondents have raised: we did not dispute that it occurs and that it may require treatment. The sentence in our study in which this fact is discussed does not contain a denial that significant hypotension occurs: it contains a comparison as rider, that the degree of hypotension is not as severe as that which was seen in a study made in thoracotomy patients who had extradural blocks. An emphasis on the potential for the development of hypotension is contained in the penultimate sentence of our study. Without discussing on the mechanisms of this hypotension, it is my experience when performing paravertebral blocks in awake patients, that some develop hypotension as part of a vaso-vagal episode.

Of course, there is nothing new under the sun and we are all highlighting problems with paravertebral techniques that were apparent 40 and more years ago [3]. Clearly, the correspondents’ experience, like our own, is that the thoracic paravertebral space, despite the idiosyncrasies of spread, is useful, if not the best, repository for drugs in some cases that come within the ambit of anaesthetists. It would seem that it is the common experience that injected substances frequently go beyond the bounds of the neat, definitive (and, for that matter, desired) anatomical locations adjacent to the thoracic spine, so that here at least Drs Purcell-Jones and Justin and I can concur, and question the wisdom of injecting drugs with irreversible, tissue destructive properties into thoracic paravertebral spaces.

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REFERENCES


**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 when further analgesia was requested and 30–40 min after the “top-up” was given. In these patients the peak flow improved by a mean of 37 %. This is in direct conflict with the results of Baxter and colleagues, but, unlike these authors, I can conclude that this effect must be a direct result of continuous intercostal nerve blockade.

With regard to complications of intercostal nerve blockade, I feel that it should be stressed again that, based on the studies of Nunn and Slavin [8], it is clear that intercostal nerve blockade should be performed posteriorly at the site where the potential for pneumothorax is least, that is, where the rib is at its thickest. One can not recommend the injection in the mid axillary line as used by Baxter and colleagues.

Finally, the acceptability of performing intercostal nerve blockade in a patient in whom extradural analgesia is considered unsuitable because of prior administration of heparin must seriously be questioned, particularly in the light of the complication described by Baxter himself [9].

D. F. MURPHY

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**REFERENCES**


*Sir,*—Thank you for the opportunity to reply to Dr Murphy’s comments regarding our study on Continuous Intercostal Blockade after Cardiac Surgery [1]. Our intention was to assess this new technique objectively, as previous reports, including 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.

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