Sir,—Thank you for the opportunity of replying to Drs Bethune and Latimer. First, we must take issue with their statement that our paper is the "key reference to the test method used in HEI 166". This is not the case. As we pointed out in HEI 1, the method used to produce the results in that publication was closely modelled on that of Weeks and Ramsey [1], although we chose to use a humidity sensor which we had described elsewhere [2]. We constructed a new, somewhat simpler test rig to confirm a suspicion we had formed during our DHSS work that leaks between heat and moisture exchangers (HME) and our ISO test lung might be influencing the humidity delivered at the patient connection port of the system.

In our account of the method, we have stated clearly that water was introduced into our relatively crude lung simulator, the Lung Ventilator Function Analyser, to produce saturation of its contained gas by water vapour and that saturation was confirmed before and after each test. We think the sentence at the opening of our discussion may have been misinterpreted as a description of our method. What we wished to imply was that the generation of saturation at the point of production of humidified gas in such test rigs does not guarantee saturation under all conditions in adjacent parts of a dynamic system.

We did consult the literature, including the classic work of Déry and co-workers [3], as this has long been a natural starting point for any serious study of respiratory humidity, together with other sources confirming Déry’s original observations. We agree that alveolar gas is almost certainly saturated with water vapour at the prevailing temperature, but that is not to say that, when gas is introduced into the large airways from the exterior and mixes with gas leaving the alveoli, this condition still obtains. The graph included in a communication from Drs Bethune and Shelly [4] confirms this, in that they showed the "humidity range in the upper trachea during inspiration with nasal breathing" to be in the range 56–78% R.H. at 37°C, attributing this to Déry. We suspect that the hygrometer to which Drs Bethune and Latimer refer is insufficiently rapid in response to reflect the changing conditions and must be used outside the respiratory tract, albeit at a site in communication with it. Of course, any significant decrease in temperature will ensure saturation down-stream and we do not dispute that the gases reaching an HME are likely to be saturated, as we believe would have been the case in our study. Nevertheless, the water content at the lower temperature would be lower than that of gas saturated with water vapour at body temperature.

The reference [6] (DHSS, 1986) in connection with the “possible and actual drawbacks” of HME is correct. These appear on page 36 of HEI No. 166. We could also have added the DHSS’s “Hazard” Health Notice HN(HAZARD) (857), referring to a problem with the proprietary device which Dr Bethune favours, but, instead, mentioned this also on page 36 of HEI No. 166. We think the title of Buckley’s paper indicates its content and the reason for it having been cited. Moreover, he refers to the dangers of linking HMEs with heated humidifiers, a practice which has been indulged in by those believing that the output of HME might need to be supplemented.

The tidal volumes were representative values in the clinical range produced by the ventilator settings and have no individual significance. Although we knew that the ICOR HME for neonates was to be released, it was not on sale at the time of the study. Therefore, we used an adult HME which we knew to be one of the most efficient. It may be that the compressible volume which Drs Bethune and Latimer describe as “deadspace” could have had a detrimental effect on the results, especially in the case of the smallest delivered volume. Conversely, it might be argued that failure of some gas to pass out of the HME and re-enter it might have had the opposite effect. All we set out to show was that the presence of a leak detracted from the efficiency of an HME and that this might be in some way related to the magnitude of that leak. We have not purported to have discovered the reasons for the deterioration, other than the obvious one of gas lost from the system, but we tried to stimulate further interest by outlining the possibilities from a thermodynamic viewpoint. Further, all models thus far, including our own, are the crudest imitations of the lung itself and we believe we are right to say that work in human subjects is the only way to confirm the relevance of the findings for patients. In the meantime, we hoped that our work might alert practitioners humidifying patients with leaks around tracheal tubes to the possibility that values of humidity lower than those normally returned by their HME might apply. These circumstances are common in neonatal and small paediatric practice and the leak becomes more severe when compliance decreases or airways resistance increases.

The study by Gedeon, Mebius and Palmer [5] was not available to us when we submitted our paper for consideration, but we did refer to the new HME from ICOR, the subject of Gedeon’s study, believing that it might show an improvement on its adult counterparts when specially designed for the application. We were, of course, delighted to learn that these workers had noted the same deterioration in performance when leaks were present and had investigated it independently, with conclusions similar to our own, but that the device had performed well under stringent test conditions.

We noted that HME are being used extensively and increasingly and that clinical experience in neonates in a centre of known excellence had been satisfactory [6].

We acknowledge the value of HME in many circumstances, but we do not accept that they are the only or necessarily the best answer to all humidification problems.

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REFERENCES

368 BRITISH JOURNAL OF ANAESTHESIA

**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 Justins. 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.

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 single 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 discoursing 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 Justins 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**