Sir,—I was interested to read the article by Dr Grundy and his colleagues (1978) on extradural analgesia, but I find some of their data and conclusions surprising. The authors used bupivacaine 0.75% in volumes of 10–20 ml in a series of 344 patients. No other analgesic drugs were tested and the subjects' ages were in the range 16–86 yr.

In no instance did the authors observe any cephalad spread of analgesia beyond dermatome T2. I find this astonishing in such a large series where large doses were used, and this apparent lack of upward spread is not in accordance with my own experience or that of others. Reviewing a relatively small series of extradural blocks performed at L2/3 with bupivacaine 0.75% given in the sitting position, I found that two patients of 68 experienced spread of analgesia beyond T2. One 44-yr-old patient received 0.75% bupivacaine 18 ml and analgesia spread to T1; another aged 43 yr received 20 ml and analgesia spread to include C8. Dr Nigel Sharrock (1977), who is quoted by the authors, gave 40 patients only 10 ml of 0.75% bupivacaine and found a spread to T1 in one patient. Dr Grundy and his colleagues acknowledge the help given by their residents-in-training in collecting their data, and without being in any way critical, I wonder if this may explain the nature of the data that has been presented, and the limited upward spread of analgesia that the authors seem to have observed in their elderly patients.

Anyone who has attempted to assemble accurate statistics on the dermatome spread of sensory anaesthesia knows the pitfalls. It takes a long period of training and observation and a compulsive degree of attention to detail to obtain precisely identical findings among a small group of observers, and the chances of between-observer error increases greatly as the group enlarges. Could it be that the observer group was enlarged to a point where such an error became significant?

In attempting to explain the apparent freedom from spread beyond T2, the authors postulated an anatomical barrier in the cervico-thoracic region of the extradural space. I know of no clinical, radiological or anatomical evidence to support the contention that fluid spreads with difficulty above the T2 extradural space in the living subject. Certainly, fluids pass relatively easily from the intervertebral foramina in the cervico-thoracic region, but that does not dictate that the cause is a block to axial flow. Segmental mid- and upper-thoracic blocks frequently spread to involve the lower cervical segments unless care is taken to limit the dose injected. In dogs, also, radiological experiments with metrizamide have shown unimpeded flow between the thoracic and cervical regions of the extradural space (Bromage et al., 1978).

The authors looked for, but did not find, an increased segmental spread in the arteriosclerotic subjects, and they quote Dr Sharrock's article of 1977 to support their own findings. Unfortunately, neither their own data nor that of Dr Sharrock show evidence that their observations were carried on long enough to detect complete spread of analgesia. One of the features of the spread of lignocaine in arteriosclerotic subjects is unusual slowness (Bromage, 1962a). The observer may have to wait for 30 min or more before spread is complete. Sharrock's measurements were completed in only "20 to 30 minutes" and so may well have missed some extremes of spread. Grundy and his colleagues did not comment on the duration of observations and it is possible that between-observer error and dermatome onset profiles of less than 30 min may have led to an underestimate of the true degree of segmental spread.

With regard to their linear regression data: in several of the figures, linear regression lines of spread and age are extrapolated well beyond the limits of the data and this is misleading and statistically unacceptable.

Finally, the authors claim to have refuted an earlier schema of mine (Bromage, 1962b) expressing a direct volume-spread relationship in extradural blockade. That schema is admittedly an approximation (Bromage, 1978), and since our isotope work of 1963 (Bromage, Joyal and Binney, 1963) it has not been assumed to represent an homogeneous uptake of local anaesthetic within the spinal canal. However, it has been a useful model if only to make anaesthetists aware of the potential danger of using excessively large doses of drugs. The authors do seem to appreciate this lesson, as shown by the uneven selection of their dose groups. It is commendable that they injected a volume of 0.75% bupivacaine 20 ml in only one patient older than 70 yr, whereas they did not hesitate to use smaller volumes in 32 patients aged 70–86 yr—a difference that is considerably greater than can be attributed to chance.

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REFERENCES


Sir,—We hope we can reassure Dr Bromage. Our residents performed many of the extradural blocks, but the blocks were performed under close supervision and the levels of anaesthesia achieved were checked, mostly by one of the three principal authors. The timing of this check was usually 20–40 min after injection, and those patients studied during operation and into the recovery period showed no late progression of anaesthesia. Initially, we also were surprised by the consistency of perineal anaesthesia and the relatively small spread of values for the upper level of anaesthesia. We sought actively evidence of anaesthesia in the arms wherever there was a likelihood of a high level of anaesthesia and latterly we used 10-ml injections in young