not a correct interpretation of their own data. In patients Nos 1 and 3 the consumption of pethidine was the same or less in the morphine groups 6 h after administration. Morphine-6-glucuronide (M6G) was of greater analgesic potency only in patient No. 2 at this time. There is marked irregularity in the cumulative dose in all patients, implying that perhaps other aspects were involved. What about diurnal rhythms in these individuals? Why is there delayed analgesic potency in the M6G groups—is it perhaps because M6G is deglucuronated to morphine to be analgesic?

Finally, under what circumstances were the patients selected to have their analgesia discontinued and replaced by a less potent opioid and then to have temporary intrathecal catheters inserted? Was it to improve the analgesia for these patients or solely for the purpose of a trial?

P. A. HARDY
Cheltenham


Sir,—We are grateful for the opportunity to reply to Dr Hardy’s letter.

We were intrigued that he regards intrathecal morphine 0.5 mg as a sub-analgesic dose, and would be interested in any controlled scientific evidence he has to support an intrathecal: oral potency ratio of 1:25 in chronic cancer patients. Other clinicians experienced in this field have found that the institution of intrathecal morphine at only 0.5–1 % of the oral dose results in improved analgesia [1], and previous studies of cancer patients have used intrathecal morphine 0.5 mg to good effect [2, 3]. The total consumption of pethidine during the morphine limb of our study ranged from 140 to 580 mg day⁻¹, which can not be regarded as "significant".

It is our experience that the oral: intrathecal morphine potency ratio varies considerably between patients, and it is not uncommon to see patients who, despite large oral doses of morphine and high serum concentrations have poorly controlled pain, and even undetectable, CSF concentrations of morphine, morphine-6-glucuronide (M6G), or both. They often respond well to very small intrathecal doses of morphine. An example of the variation in response is seen in our paper in that the patient receiving the highest oral morphine dose required the least pethidine during the morphine limb.

We were aware, of course, that more patients are needed in order to establish the exact potency and CSF kinetics of intrathecal M6G, and of the many factors which may affect analgesic requirements. We have investigated the pharmacokinetics of intrathecal morphine and M6G in a further three patients and the results obtained support our published data. The free use of a PCA system allows the patient to titrate analgesia to their desired activities, and produces marked variation in dose requirements throughout the day, as seen here. The use of a crossover design, standardized dosing times and assessment by cumulative PCA requirements up to 24 h for each drug minimizes these factors.

We doubt if deglucuronidation of M6G is responsible for delayed onset of analgesia, as administration of intrathecal M6G did not result in the production of detectable CSF concentrations of morphine. It could simply be explained by the physicochemical properties of M6G (the onset being related to lipophilicity of the drug).

All three patients who took part in this study had residual pain combined with unacceptable side effects, a situation in which we consider spinal opioids or use of a PCA system, or both, clinically appropriate (not “solely for the purpose of the trial”). Indeed, under these circumstances we would consider it unethical not to offer the patient spinal analgesia if facilities for this are available. Entry was considered only following informed consent and independent patient counselling, as is our usual practice. At all times during the study the patients scored pain as nil or mild, and the limits set on the PCA systems were never reached (a considerable improvement in pain control from the patient’s point of view!).

M. H. HANNA
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REFERENCES

SPINAL ANAESTHESIA WITH HYPOBARIC BUPIVACAINE

Sir,—I read with surprise the article by Drs Taivainen, Tuominen and Rosenberg [1] in which a relatively large volume of markedly hypobaric bupivacaine was given to patients in the sitting position. Perhaps not surprisingly, the study was abandoned because of an unacceptably high incidence of dangerously high blocks. In past times when hypobaric solutions were commonly in use, the standard procedure was to place the patient in a head-down position and gain height of anaesthesia by use of increasing volume. This article flies in the face of this conventional teaching.

I was surprised that the authors made no reference to the traditional teaching, or explained their reasons for ignoring it, even though it was shown ultimately to be justified.

R. H. JAMES
Leicester

REFERENCE
1. Taivainen T, Tuominen M, Rosenberg PH. Spinal anaesthesia with hypobaric 0.19 % or plain 0.5 % bupivacaine. British Journal of Anaesthesia 1990; 65: 234–236.

Sir,—Thank you for the opportunity to respond to Dr James’s comments on our paper.

The cephalad spread of spinal anaesthesia with plain (slightly hypobaric) 0.5 % bupivacaine is characterized by a low interindividual variation of the block [1]. Several attempts have been made to achieve better prediction of the high levels of anaesthesia which would be suitable especially for abdominal surgery. These include keeping the patient in a