myopathy and the lack of a similar myopathic effect so far on restarting the drug suggests a dose-related effect, which was obviously not the mechanism in the two cases of motor neuropathy. Millions of people must by now have been treated with cimetidine and it is possible that other isolated cases of toxic neuromyopathy have gone unrecognised because of the apparent rarity of the association. We wonder if our case is unique or whether others have also seen unexplained weakness in patients on long-term cimetidine?

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¹ Burland WL. In: Creutzfeldt W, ed. *Proceedings of the third international symposium on histamine H₂-receptor antagonists*. Amsterdam: Excerpta Medica 1978:238-58.

Fulminant hepatic failure in childhood

SIR,—Your leading article on fulminant hepatic failure in childhood (27 September, p 823) is certainly a tribute to the efforts made at King's College Hospital over the last decade to improve the outlook in this desperate condition. Essentially patients die of encephalopathy, which is due to the effects of accumulating toxins on the integrity of the blood-brain barrier; and if appropriate care is taken renal failure, haemorrhage, hypoglycaemia, infection, and plasma volume depletion should now be only terminal and less usual events.

The mainstay of current support is charcoal perfusion,^{1,2} with prostacyclin infusion to prevent platelet destruction and if necessary haemodialysis (again preferably with a charcoal membrane). Both of these techniques can now be done by experts in regional centres. Why then should sick patients ever be transported over long distances? Such patients do not stand the journey well and even to consider it shows a lack of concern for relatives, who also have to travel and then "camp around" in London.

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¹ Engle WD, Jacobs JF, Swartz RD. *J Pediatrics* 1978;**93**:972-4.

² Gimson A, Hughes RD, Canalese J. *Lancet* 1980;i:173-5.

Test reduction

SIR,—The need to find optimal (or at least second-best) groupings of "indicants" as a (partial) means of more rational cost control in the medical sector is no less pressing for the United States than it is for the British National Health Service. The papers by Professor W I Card and Dr P A Emerson (23 August, p 543 and 6 September, p 656), and Dr C C Spicer (30 August, p 592) on test reduction were commendable for their brevity and clarity in addressing these issues, and one hopes that they will find a wide audience on both sides of the Atlantic.

One might take exception to two points in their presentations. The first relates to what seemed to be an implicit assumption—namely, that the physician is the only relevant decision maker. Needless to say, the patient has a non-trivial stake in the choice of therapy or the decision to use or to forego one more diagnostic procedure. In the face of mounting

sentiment for greater patient autonomy, the patient's beliefs or preferences in the matter should be taken more formally into account. (If the objection is that the patient is uninformed, the answer may be that it is incumbent on the physician to provide the information, not to be paternalistic and make the decision in the patient's "best interest.") Secondly, patients and physicians differ among and between themselves in the degree to which they are "risk takers." Some, for example, might prefer surgery for the gastric ulcer regardless of the probability that it is benign (or malignant), because they view the "risk" of cancer as being so much greater than any possible "risk" of the operative procedure itself; others might take a quite opposite view. One trusts that the authors will, in coming years, present us an equally lucid description of more refined statistical methods for calculating expected utilities and cost effectiveness that take some of these factors into account.

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Recurrent urinary infection with microaerophilic and anaerobic bacteria

SIR,—I was interested in the article by Professor W Brumfitt and colleagues (4 October, p 910) on a case of recurrent microaerophilic and anaerobic infection. They were certainly successful in treating the infection, but was it justified since they say that the patient was always symptom free? The fact that she had had a cystoplasty would have accounted for the "sterile pyuria" and this in itself would hardly warrant further investigation. In some hospital laboratories it is normal practice not to report on the presence, or absence, of pus cells or red cells; and since the standard cultures would in this case be reported as being sterile the matter would not be taken further.

More important than treating esoteric, asymptomatic infection in patients with a cystoplasty is to make sure that they are able to empty the bladder to completion. This may require urethral dilatation, sphincterotomy, or a bladder neck procedure.

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The anaesthetist and the pain clinic: dextromoramide analgesia

SIR,—I was interested to read the comment by Dr Lesley A M Wills (20 September, p 809) that it is a myth that tolerance and habituation to opiate analgesics occur if pain is properly managed. In the same letter Dr Wills perpetuated a similar myth—namely, that dextromoramide is a very short-acting, strongly habituating drug with little or no place in the management of terminal pain. Although Dr Wills provided a bibliography to support the comments made on the first myth, similar support was noticeably absent with regard to the second. Comments similar to those of Dr Wills on dextromoramide are published with regularity, the latest originating from an area health authority.¹ Such publications are also similar in another respect—namely, the complete lack of clinical evidence to substantiate the opinions they contain.

This lack of reference to published (or unpublished) clinical evaluations of dextromoramide is surprising as there are many reports on the use of the drug for the relief of both acute and chronic severe pain. It is known from controlled clinical studies^{2,3} that dextromoramide (Palfium) provides a relief of severe acute and chronic pain equivalent in quality to that of morphine and other opiate drugs. Dextromoramide was recently independently recommended as an alternative to oral morphine, particularly for the ambulant, working patient.⁴ When pain is properly managed with dextromoramide there appears to be little tendency for habituation to occur. Kolodny⁵ in a study of 102 patients found no evidence of tolerance, habituation, or primary addiction; while Calesnick⁶ treated patients for up to 14 months with similar results. One case of habituation, although without withdrawal symptoms when the treatment was stopped, was supported by Flavell-Matts⁷ during a study of 60 patients. Den Otter⁸ found no evidence of habituation in more than 350 patients.

With regard to the duration of dextromoramide analgesia, data in a large number of published reports indicate a range from 2 to 12 hours, with a median value of the order of four to six hours, independent of the type of patient studied and independent of the route of administration of the drug. For example, in postoperative pain relief, Swerdlow² demonstrated that dextromoramide provided analgesia with a duration equivalent to that of morphine, oxymorphone, and phenazocine. In pain relief associated with various medical conditions, including carcinoma, the mean duration of dextromoramide analgesia typically is reported to be from four to six hours.⁶⁻⁷ Objective pharmacokinetic studies by Caddy⁹ (also personal communication) indicated that the mean plasma half life of dextromoramide is approximately 45 hours (range 13.3-86.6 hours), which is inconsistent with a short duration of analgesia in the average patient.

"Let us dispel all myths about these drugs" was the exhortation by Dr Wills. I believe that this letter will help to do exactly that.

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¹ Leeds Area Health Authority. *Drug Information Bulletin*. Leeds AHA, March 1980.

² Swerdlow M, Murray A, Daw RM. *Acta Anaesth Scand* 1963;**7**:1-19.

³ Bauer RO, Free SM, Bowen EH. *J Pharmacol Exper Therap* 1961;**131**:373-80.

⁴ Anonymous. *Drug and Ther Bull* 1980;**18**:69-72.

⁵ Kolodny AL. *Antibiot Med Clin Ther* 1960;**7**:695-701.

⁶ Calesnick B. *J Chron Dis* 1959;**10**:58-66.

⁷ Flavell-Matts SG. *Practitioner* 1962;**188**:524-6.

⁸ Den Otter G. *Ned Tijdschr Geneesk* 1958;**102**:1637-41.

⁹ Caddy B, Idowu R. *Analyst* 1979;**104**:328-33.

Bayes's theorem and the evaluation of tests

SIR,—In his article on Bayes's theorem (30 August, p 592), Dr C C Spicer is correct to say that the theorem is named after the Rev Thomas Bayes, but not that Bayes discovered it in about 1760. Bayes's fame is due to his having invented a particular method of inductive argument that happened to involve a manipulation of probabilities, which may be written (following Dr Spicer) as: $P(D|S) = P(D) \times P(S|D) \div (PS)$, where D is the disease, S is the pattern of symptoms and signs observed, and the upright bar (|) is read as