source of uncertainty and even scepticism about the applicability of the result of such a trial to a population in general and to a single patient in particular.

We suggest that participating clinicians should be prepared to enter *all* their patients into the selection process and, with a minimum of exclusion clauses, that the *whole* population be documented. The results should then be reported in two parts—those for patients who have been randomised and those for patients who have been excluded from the trial. This comprehensive approach would allow would-be followers of new methods to have greater confidence in deciding whether an individual might benefit from the results of a reported investigation.

Although this would increase the task, perhaps it is a necessary bridge between the scientist's wish to study populations and the clinician's drive to identify the best treatment for the individual patient.

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Localisation of vipoma

SIR,—I was interested to read the short report on the localisation of a vipoma by Dr J G C Kingham and others (16 December, p 1682). In this paper the authors state that the vasoactive intestinal peptide (VIP) concentrations in the portal and systemic veins around the head of the pancreas fell dramatically 10 minutes after removal of the small tumour which was presumed to be the site of VIP secretion. Presumably this fall in concentration was not known to the surgeon until several days later, when the radioimmunoassay results were known.

Can the authors advise us on the use of any preoperative techniques which may aid the surgeon in localising a vipoma or the presence of micrometastases? Could they also comment on how they would have managed this case had the VIP concentrations not been reduced by the resection of this tumour—this fact only being known several days after laparotomy?

It seems to me that there is still the need for a rapid assay, which perhaps could be performed in an operating theatre side room in order to provide the surgeon with information regarding the adequacy of resection of a hormone-secreting tumour. Perhaps the further development of cytochemical bioassays will have a part to play in this context.

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* *We sent a copy of this letter to the authors, whose reply is printed below.—Ed, *BMJ*.

SIR,—In reply to Mr Hoile's comments, he presumes correctly the surgeon was unaware of the rapid fall of VIP levels after tumour excision. The VIP radioimmunoassay used in the management of this case¹ takes four days and therefore is not of value as a theatre side room assay in its present form. It is possible that a highly modified VIP radioimmunoassay, as has been described for insulin,² may be of use preoperatively. Such an assay has not yet been

developed, but our VIP assay could be performed in two hours at the expense of considerable loss of sensitivity. Most bioassays for VIP are not sufficiently sensitive to detect physiological levels of VIP or even levels in the vipoma range. However, a bioassay has been reported recently that is sensitive to 10 pmol/l VIP,³ though its complexities would make it unsuitable as a side room technique.

If tumour excision had cured this man's symptoms but not reduced his VIP levels to normal, then further treatment would not have been warranted. If excision had neither cured the symptoms nor reduced VIP levels despite the tumour's being a histologically proved vipoma, then metastases would have been presumed responsible and we would have considered a trial of streptozocin rather than continued high-dose steroids.

We know of no currently available technique for localising tumour or micrometastases preoperatively and agree that there is need for one.

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¹ Mitchell, S J, and Bloom, S R, *Gut*, 1978, **19**, 1043.
² Turner, R C, *et al*, *Lancet*, 1976, **1**, 515.
³ Waldman, D B, *et al*, *Gastroenterology*, 1977, **73**, 518.

Iodine and acetone-containing plastic spray dressing

SIR,—We read with interest the letter from Dr J C Morgan-Hughes and Mr R A Bray (26 August, p 639) on the suggested hazard of using an acetone-containing spray (Op-Site) in conjunction with tincture of iodine, and would like to offer some information which may help to interpret their observations.

It is known that iodine can react with acetone under appropriate experimental conditions,¹ as Dr E Powell pointed out (25 November, p 1500); and it has been postulated that iodoacetone may be formed in an aqueous acetone solution of iodine.² In our opinion it is unlikely that such a reaction could occur to a significant extent under the conditions of an acetone-containing spray dressing being applied to iodine-treated skin, and no evidence of such a reaction has been put forward in the current correspondence.

Clinically, it is well known that solutions of iodine applied to the skin should not be covered with an occlusive dressing,³ a view supported by Dr Morgan-Hughes's observations¹ that "erythema occurred in all areas painted with tincture of iodine and covered by Sleek" (an occlusive strapping). The "more severe" reaction demonstrated when iodine, Op-Site spray, and Sleek were used suggests an exaggeration of the effects of occluding iodine on the skin. The combination of Op-Site and Sleek failed to produce skin reactions, and it is unfortunate that the effect of Op-Site spray alone on iodine-prepared skin was not investigated by Dr Morgan-Hughes, since the film formed by the Op-Site spray is highly permeable and not occlusive.

In a recent communication Dr P R W Lanham (30 September, p 961) has indicated that, in his experience, Op-Site spray on iodine-prepared skin does not cause a reaction since a gauze swab is used to protect the iodine- and Op-Site-prepared skin from the occlusive Sleek strapping. We have demonstrated that Op-Site spray itself, when applied directly to iodine-treated skin, does not give rise to any complications.⁴ Thus we conclude that the essential factor in the skin reactions reported by Dr Morgan-Hughes was occlusion of the iodine-prepared skin by an occlusive strapping. Op-Site spray alone over iodine-prepared skin does not constitute a hazard.

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¹ Benesi, H A, and Hildebrand, J H, *Journal of the American Chemical Society*, 1949, **71**, 2703.

² Dawson, H M, and Leslie, M S, *Journal of the Chemical Society*, 1909, **95**, 1860.

³ Wade, E (editor), *Martindale: The Extra Pharmacopoeia*, 27th edn, p 826. London, Pharmaceutical Press, 1977.

⁴ Smith and Nephew Research Ltd, internal report, October 1978.

Long-term parenteral nutrition

SIR,—With reference to the paper by Drs Karin Ladefoged and Stig Jarnum (22 July, p 262) I would like to comment on table IV, "Complications unrelated to long-term parenteral nutrition."

The authors mention two patients with paresis of the peroneal nerve without giving any explanation of this. I too observed in a patient undergoing long-term total parenteral nutrition the onset of a bilateral paresis of the peroneal nerve after seven to eight months of completely intravenous nutrition. Paralysis was associated with "burning" feelings and sensations of heat, which affected mainly the soles of the feet and less frequently the dorsal aspect of the feet as well. Sometimes the pain took the form of a dull, constant ache in the feet or sometimes in the entire leg; it was often sharp and lancinating, even if momentary in duration. Neurological examination revealed a severe bilateral motor, reflex, and sensory loss confined to the legs and feet. No cardiac abnormality was found on the electrocardiogram except sinus tachycardia unresponsive to digoxin.

Since the patient had been maintained on a daily dosage of 3.2 mg thiamine for seven months and balance studies¹ indicate a minimum daily requirement of approximately 1 mg, a thiamine deficiency was not initially suspected and high dose of pantothenic acid was administered. Subsequently, owing to the persistent symptoms, I suspected that this patient, who had been receiving parenterally about 600-750 g dextrose a day, might have a high consumption of thiamine since it is an important cofactor in carbohydrate metabolism: thiamine in fact is required for the oxidative decarboxylation of both pyruvate and α -ketoglutarate and also participates in the transketolase reaction of the pentose shunt. I administered, therefore, 100 mg thiamine a day, and the patient experienced a prompt and progressive resolution of his pain and an improvement of the sensory function, though only a slight recovery of the motor activity.