the best of our knowledge been reported previously.

Two cases occurred at laparoscopic sterilisation. In one the broken metallic bit was retrieved by emergency laparotomy, while in the second case the broken fragment could not be located at laparotomy but was removed with the aid of the x-ray image intensifier five days later, when a repeat laparotomy was performed. There was no obvious peritoneal or omental reaction seen at operation. The third case occurred in mid-December 1978, during application of silastic rings to the round ligaments for ventrosuspension. After unsuccessful attempts at removal with the Palmer forceps, the foreign body was left behind. To date, no adverse effect has been noted.

Since the forceps prongs of the ring applicator are made of surgical grade stainless steel, which should not cause a toxic reaction,[1] removal of the broken metallic fragment by laparotomy is probably unnecessary if laparoscopic retrieval fails. However, the possibility of its migration is an unsettled question.

The metallic prongs are constantly subjected to stress and it would be advisable each time to check their condition prior to use. They should be replaced if the tips are malaligned or are bent as either would indicate excessive strain.[2]

T H GOH

Department of Obstetrics and Gynaecology,
University Hospital,
Kuala Lumpur, Malaysia

[1] Schofield, S R, personal communication.
[2] KLI Inc, Technical Bulletin 000685-2. Ivyland, PA, USA, KLI Inc, 1976.

## Primary screening for visual disorders in children

SIR,—Mrs A V MacLellan and Dr P Harker reported a highly successful method of screening preschool children in a rural area for visual disorders (14 April, p 994). I should like to reinforce their recommendations by relating that a very similar service has been given in the urban London borough of Barnet, with great relief to ophthalmologists in the hospital and to mothers of small children, who for obvious reasons preferred an immediate appointment nearer their homes. A succession of orthoptists over approximately 10 years have had no difficulty in arranging sessions and have proved that "case finding" at an earlier age is more thorough than the orthodox hit and miss.

Mrs MacLellan quotes three very apt histories of children likely to be missed except by such a system as hers and ours. I would draw particular attention to case 1, where near vision was reduced, which is seldom tested in young children, and to case 3, which shows the importance of testing siblings.

The main difficulty in arranging such a service appears to be lack of liaison and interest on the part of the community and hospital administrators and ophthalmologists.

P A GARDINER

London W1N 3FA

## Continuous narcotic infusions for relief of postoperative pain

SIR,—I was delighted to read the paper on relief of postoperative pain by Dr Jeremy J Church (14 April, p 977). His technique will be a most useful contribution to the problem. But there are two points that I wish to raise.

Firstly, if Dr Church could find the time to titrate his loading doses[1] he would not have to adjust the infusion rates so often. And his results would be even better.[2]

Secondly, his technique is cheap, effective, and simple; however, it needs at least intensive nursing care. I use a less demanding technique when patients have to return to general surgical wards on operation days. I give buprenorphine (Temgesic), 0·1 mg/20 kg body weight less 0·1 mg for each decade of age over 70 years, intravenously during induction of general anaesthesia. If no more than 0·5% halothane is used and it is stopped 30 minutes before the end of the operation respiration is not depressed and recovery is quick. I give papaveretum, 20 mg intramuscularly, when the patient's reflexes have begun to return but before pain is felt. Neither the level of consciousness, the respiratory system nor the cardiovascular system is depressed. This is due to the antagonist action of buprenorphine. This regimen ensures that patients feel no pain for 2-12 hours after operation, the majority lying within the range 6-8 hours. There is ample time for further injections of analgesics, usually buprenorphine, since pain develops gradually. It is my experience that if pain is prevented during the first 24 postoperative hours by this or any other technique pain on the second and subsequent days is much reduced.

E N S FRY

Department of Anaesthesia,
North Tees General Hospital,
Stockton-on-Tees,
Cleveland TS19 8PE

[1] Fry, E N S, and Desphande, S, *British Medical Journal*, 1977, **2**, 870.
[2] Fry, E N S, *Annals of the Royal College of Surgeons of England*, in press.

## Oligoclonal immunoglobulins and multiple sclerosis

SIR,—We read with interest the article by Dr E J Thompson and others (6 January, p 16) entitled "Oligoclonal immunoglobulins and plasma cells in spinal fluid of patients with multiple sclerosis." We would like to draw attention to one aspect of interpretation when looking for the presence of oligoclonal bands in the cerebrospinal fluid (CSF).

Using Thompson's modification of polyacrylamide gel electrophoresis[1] and staining with Coomassie Blue, we have examined the CSF of 131 patients whose provisional diagnoses on admission included multiple sclerosis, and 33 control CSF samples from patients who clinically had no neurological disease. Of the 33 control samples, 10 showed one or more oligoclonal bands in the $\gamma$ region following electrophoresis. In five of these 10 a serum sample (100 $\mu$g protein) was run with the CSF sample (100 $\mu$g protein) and the bands present in the CSF were found to correlate in position with bands in the serum sample. In eight other control samples where serum and CSF samples were run simultaneously, oligoclonal bands present in the serum were not found in the CSF, which showed only a diffuse pattern. The remaining 15 control CSF samples showed only a diffuse pattern. Over one-third of our 131 patients CSF samples have shown bands which correlated with bands in their serum. These patients, with two exceptions, have subsequently not been diagnosed clinically as having multiple sclerosis. We therefore assume that in these cases the discrete bands found in the CSF are due to diffusion into the CSF of immunoglobulins or other proteins present in the plasma.

As a consequence of these findings, we routinely perform electrophoresis on CSF and serum from each patient and disregard (for diagnostic purposes) those oligoclonal bands in the $\gamma$ region of the CSF electrophoretogram which correlate in position with those of serum. It has also been found that CSF samples left more than 24 hours at 4°C are not suitable for electrophoretic analysis, as the bands become indistinct.

In summary, to avoid false positive results, it is necessary to run a fresh serum sample simultaneously with each fresh, red-cell-free CSF sample. Only those bands in the CSF which do not have a corresponding band in the serum should be called abnormal oligoclonal immunoglobulins.

B R CASEY
A J MASON

Wellington Hospital,
New Zealand

## Beta-blockers and renal function

SIR,—The letter from Dr R Wilkinson (3 March, p 617) commenting on the paper by Dr A D Wright and others (20 January, p 159) regarding reduction in renal function with beta-blockers aligns with observations that propanolol caused a reduction in renal plasma flow from 213 to 184 ml/min/100 g in 12 patients with essential hypertension.[1] However, such studies and the controversy over the relative effects of cardioselective and non-cardioselective agents only tend to highlight the fact that all beta-blockers studied to date have been shown to *reduce* renal function.

Recent studies with nadolol,[2] using a xenon-133 washout technique have shown maximal *increases* in renal blood flow compared with control ranging from 16% to 26%. Eight volunteers on a low-sodium diet were studied, three normotensive and five hypertensive. Dosage ranged from 0·3 to 10 $\mu$g/kg. Mean renal blood flow increased from $270 \pm 19$ ml/100 g/min in the control state by a mean of $46·9 \pm 9$ ml/100 g/min at 1·0 $\mu$g/kg, by $72 \pm 4$ ml/100 g/min at 3 $\mu$g/kg, and $70 \pm 5$ ml/100 g/min at 10 $\mu$g/kg—an increase of 26%. In addition, a dose-ranging clinical study of the treatment of hypertension with nadolol[3] showed an increase in 24-hour sodium excretion from a pretreatment average of 203 mmol(mEq) to 258 mmol after 14 weeks' treatment.

As Dr Wilkinson points out, such an effect on renal function may well be of importance in patients with renal disease on treatment with beta-blockers.

D A JACKSON

E R Squibb and Sons Ltd,
Twickenham, Middx TW1 3QT

[1] Sullivan, J M, Adams, D F, and Hollenberg, N K, *Clinical Research*, 1976, **39**, 532.
[2] Hollenberg, N K, *et al*, *British Journal of Clinical Pharmacology*, 1979, **7**, 219.
[3] Frithz, G, *Current Medical Research and Opinion*, 1978, **5**, 383.

## Treatment of exophthalmos and pretibial myxoedema with plasmapheresis

SIR,—The observations of Dr Dandona and others (10 February, p 374) on treatment of exophthalmos and pretibial myxoedema by plasmapheresis are certainly interesting, but the emphasis on attributing the beneficial result to a drop in the concentration of thyroid-stimulating immunoglobulins (or ophthalmogenic IgG or dermatogenic IgG) is not justified by the data.

Plasmapheresis obviously could lower the concentration of many different proteins present in plasma—for example, thyroglobulin