"poststerilisation syndrome,"⁹ which I believe does exist. A combined approach by gynaecologists and general practitioners is likely to be most fruitful.

The analysis of consulting patterns and referrals for gynaecological complaints in sterilised women, both before and after operation, and in controls is currently being explored in this practice.

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Female sterilisation—no more tubal coagulation

SIR,—Your leading article "Female sterilisation—no more tubal coagulation" (12 April, p 1037) contained the provocative message that the gynaecologist should be careful to ensure that his operation is reversible. If his patient has been properly counselled his main concern should be to ensure that his operation is effective.

Gynaecologists who have used laparoscopic tubal diathermy safely and effectively for many years are likely to be disappointed with the high pregnancy rate with clip sterilisation and with the risk of tearing the tube and broad ligament with ring sterilisation. Despite all the effort and inventiveness, there is still no practical alternative to diathermy, preferably bipolar, for laparoscopic sterilisation.

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Setting up a drip

SIR,—Dr John Aspinall is incorrect in his assumption (3 May, p 1146) that we recommend intramuscular morphine sulphate alone for sedation and analgesia in distressed children prior to venepuncture. Our experience has been that it is of value when used in combination with oral trimeprazine for tranqualgesia as described (29 March, p 942). A suitable alternative to oral trimeprazine would be oral diazepam 0.15 mg/kg, while anaesthetists will be familiar with the use of rectal thiopentane or ketamine by the intramuscular or intravenous routes in refractory cases. Perhaps a solution to this problem for the future may lie in the use of an epicutaneous pad (Ane-Pad, Astra Lakemedel AB) containing the local anaesthetic ketocaine applied to the proposed venepuncture site. While this technique was described¹ for cutting split skin grafts in adults an extension of its use for minor superficial procedures in children deserves investigation.

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Stimulation of dorsal column in multiple sclerosis

SIR,—We apologise for not having cited some of Dr L S Illis's work in relation to dorsal column stimulation (24 May, p 1273). In fairness, however, only one of the six articles quoted were available at the time of final submission of our article (29 March, p 889). Three articles appeared subsequently earlier this year and could not be included. The proceedings of the Belgrade Symposium on External Control of Human Extremities presented difficulty. This was not available through interlibrary loan and we felt it wrong to quote them. Our own group's findings in fact appeared in these proceedings and we deliberately do not refer to them. This leaves us with one reference¹ which we could have included but did not, possibly because only two cases were evaluated.

We made no claim for a "first description" of any major response in dorsal column stimulation. Full credit for most of this goes to Professor A W Cook of Brooklyn, New York. Dr Illis and Dr E M Sedgwick are incorrect in assuming we have simply confirmed their group's findings. We agree broadly that bladder function is benefited but do not agree about voluntary motor function. In their recent article² mention is made of improved motor function in "27.7% of 18 patients," which was felt not to be due to placebo effect, etc. We also found change in gait but thought this was a placebo effect. Furthermore, mention is made² of improved sensation—"27.7% of 18 patients." One hears of descending sensory levels in non-anatomical pattern following dorsal column stimulation. The careful studies on sensation reported in our paper—some in conjunction with Dr J Scadding of Professor P Wall's unit, University College, London—suggested that no change had occurred. Sometimes a patient claimed benefit which could not be substantiated and this again is likely to be a spontaneous fluctuation or placebo effect.

Certain recent publications suggest that dorsal column stimulation has no real effect at all in multiple sclerosis.^{3,4} Perhaps we should devote more time to validating our results than disputing priorities and references to the literature.

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Beta-blockers in immediate treatment of myocardial infarction

SIR,—We are anxious to comment on the trial of beta-blockers given early after a myocardial infarction that appeared recently (29 March, p 885). It was refreshing to read such a clear and open account of the trial findings. Despite their experience with withdrawals, Dr R G Wilcox and his colleagues emphasise that to minimise potential bias it is essential to include in the main analysis all patients randomised, regardless of their course during the study. We agree wholeheartedly. With the same purpose in mind, we too would advocate the use of total mortality as the primary response variable. It is based on a

simple count with no reliance on definition or interpretation.

We must, however, take issue with the conclusion of the authors. The trial showed that at six weeks there was a 25-33% reduction in total mortality in the atenolol and propranolol groups compared with the placebo group but, because this does not reach statistical significance, it is stated that there is no place for beta-blockers in the acute management of patients with suspected myocardial infarction. We support the plea of Professor Geoffrey Rose (19 April, p 1988) that in any clinical trial with equivocal results either confidence limits or the power—that is, the likelihood of detecting a real difference—should always be given. The trial of the Nottingham group was too small to have adequate power to test the hypothesis that beta-blockers may be of benefit if started immediately after myocardial infarction. With approximately 130 patients in each of the three treatment groups and an $\alpha=0.05$, the chance of detecting a true benefit was less than 30% even when total mortality was reduced by as much as one-third. Should any intervention be shown to have an effect of this degree its impact on public health would indeed be considerable. Unfortunately in small trials a verdict of "not proven" is all too common. The question of whether or not beta-blockers prolong life in heart attack victims can be answered only by a well-designed clinical trial of sufficient size.

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SIR,—Dr R G Wilcox and others (29 March, p 885) have admirably shown how serious bias can be created in small trials if patients are withdrawn after randomisation when they fail to adhere to treatment (a point not always appreciated). We believe that their paper too suffers from some defects in design, so that one cannot conclude, as they do, that beta-blockade is of no practical use in acute myocardial infarction.

Firstly, after oral treatment significant beta-blockade is achieved too late to have any large effect on serious arrhythmias or on infarct size, which are the main causes of mortality and morbidity. Additionally, in the atenolol-treated group a proportion of the patients would have received a placebo tablet initially (midday), thereby causing a further delay of at least eight hours before the patients received any beta-blocker. This therefore reduced the chance of testing the hypothesis that early beta-blockade may reduce mortality by reduction of infarct size or serious arrhythmias.

We too started a similar trial of oral beta-blockade three years ago and abandoned this when we observed that significant bradycardia was not seen for three to four hours after the dose—that is, about six to 12 hours after the onset of infarction. This is perhaps too late to influence infarct size (and hence mortality and morbidity). We therefore embarked on a morbidity (infarct size) study with intravenous beta-blockade. Our statistical advice was that we would need very much larger numbers than our projected trial size of 200-400 to detect any influence on mortality (see also Professor Geoffrey Rose's letter in the *BMJ* of 19 April, p 1080).