cardiac measurements, which are vitually irrelevant to the case they are making. Thirdly, from table III it seems that no patient received more than three injections of morphine. As this was over several months I wonder at the need for an implantable system, with all the associated risks of contamination and infection. Intermittent injection might have been just as effective.

Unfortunately this interesting, but anecdotal, account of the use of a new idea does not really help me in trying to treat patients with chronic anginal pain, as I cannot assess the value of the technique from the data provided.

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AUTHORS' REPLY—All of the patients in our group had been receiving intramuscular opiates once or more daily in doses that caused unacceptable side effects. Dr W G Notcutt's confusion about the extent of pain relief is understandable, and our use of the word "lessened" was incorrect. We would like to emphasise that all of the patients were free of anginal pain while receiving daily treatment with epidural opiates.

Though anginal pain is not always caused by ischaemia, we believe that our cardiological data are relevant as they illustrate the haemodynamic and angiographic state of our patients, whose cardiac condition was such that they experienced several attacks of angina daily. With regard to Dr Notcutt's last point, we would like to point out that the numbers of injections of morphine in table III were daily totals.

Before treatment our patients were severely incapacitated, but they have now resumed their previous physical activities.

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The Liverpool urban obstetric flying squad

SIR,—We would like to reply to some of the points raised by Dr G P R Browne, Dr C C Callander, and Drs A D G Roberts and C A J Macafee (14 February, p 442) in response to our article about the Liverpool urban obstetric flying squad.

All the correspondents referred to mixed urban and rural areas and outlying maternity units. We do not have such units in our solely urban practice, and our choice is therefore limited to transfer to the main unit or a domiciliary anaesthetic. We agree, however, that in remote areas the best solution would be to provide senior help (by which we mean that of consultants or senior registrars experienced in obstetric anaesthesia) to an outlying unit, where prior arrangements for emergencies have to be planned in the cold light of day. The prospect of domiciliary anaesthesia should be daunting to all anaesthetists. Equipment can be demonstrated and practised with, but it is impossible to devise any training that can prepare adequately for the circumstances in which anaesthesia has to be administered on the kitchen table.

Dr Browne's remark that he finds the term "occasional anaesthetist" puzzling seems difficult to understand as in his own area only 16 general anaesthetics have been administered over the past six years in outlying maternity units.

We all seem to agree that retained placenta remains the overriding reason for giving an anaesthetic. In our opinion, however, the transfer of such patients, with the placenta in situ, to a maternity unit has not been shown conclusively to cause any increase in morbidity or mortality. Indeed, if patients can be transferred to a peripheral unit where epidural anaesthesia can be performed, as stated by Drs Roberts and Macafee, then the degree of urgency must be considered to be low.

We would repeat that inexperience among junior staff, alternative cover arrangements, and the mixture of urban and rural practice are all factors that must be taken into account when each area or department decides on the provision of an obstetric flying squad. We agree with Dr Callander that no blanket policy is correct but would maintain that in purely urban areas the anaesthetist's presence is now an unnecessary luxury.

Though we recognise that for geographical reasons there may be a few areas of the United Kingdom where small outlying units are the best provision possible, we wonder whether adequate anaesthetic cover, and presumably also skilled obstetric cover, can be provided by the obstetric flying squad.

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Treating postural hypotension

SIR,—Dr R D S Watson (14 February, p 390) correctly emphasises the use of physical methods as initial treatment for postural hypotension, followed by fludrocortisone or a non-steroidal anti-inflammatory drug. He omitted to mention, however, two other important treatments that have been discussed[1 2]: the combination of fludrocortisone with a non-steroidal anti-inflammatory drug, which may be successful in postural hypotension refractory to other drugs, and atrial tachypacing, which has proved useful when other methods have failed.

Dr Watson warned of the dangers of hypertension with the use of sympathomimetics but did not mention that hypertension can be overcome by simultaneous use of a β antagonist.[1 2] In individual patients with postural hypotension several mechanisms may be at work to varying degrees, and it is important to tailor treatment to the individual requirements. Attention should thus also have been drawn to other drugs that may be successful in some cases of postural hypotension—namely, metoclopramide, yohimbine, propranolol, midodrine (a newer α antagonist), the combination of an α antagonist with fludrocortisone,[1 2] and the use of clonidine's partial α agonist activity.[3]

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1 Anonymous. Management of orthostatic hypotension. *Lancet* 1981;i:963-4.
2 Davies IB. Chronic hypotension. *J R Soc Med* 1982;75:577-80.
3 Bannister R. Treatment of progressive autonomic failure. In: Bannister R, ed. *Autonomic failure. A textbook of clinical disorders of the autonomic nervous system.* Oxford: Oxford University Press, 1983:316-34.

AUTHOR'S REPLY—Dr I B Davies's letter raises several important issues. Firstly, what is the function of a leading article? It is clearly impossible to review exhaustively in the space of 600 words all the forms of treatment, in various combinations, that have been used in patients with postural hypotension. My purpose was to describe, briefly for a general readership, the pathophysiology of this uncommon condition and the principles of treatment, emphasising some of the hazards, and to highlight some new treatments that may prove beneficial.

Secondly, we have a responsibility to ensure that there is a sound basis for any treatment that we recommend. I attempted to draw readers' attention to some of the important points—for example, few, if any, studies have investigated the benefit of drugs in addition to night time head up tilt. The combination of a non-steroidal anti-inflammatory drug with fludrocortisone may be beneficial, but in the paper cited in Dr Davies's review only two of the five patients studied showed sustained improvement.[1] Atrial tachypacing has been reported to be helpful in a patient described by Moss *et al*,[2] who presented very limited evidence to indicate that sympathetic failure was the underlying cause (the patient may have had sick sinus syndrome). Dr Davies cites two additional papers in his review to support the value of this treatment: one letter concerned interpretation of plasma catecholamine concentrations in the paper by Moss *et al* and did not discuss therapeutic effects[3] and the other described a patient who failed to respond to this form of treatment.[4] Dr Davies commended yohimbine, but the reference quoted concerned patients with postural hypotension caused by the antidepressant clomipramine rather than those with autonomic failure.[5] Finally, Dr Davies refers to midodrine as a α antagonist; it is in fact an α agonist, which has been associated with supine hypertension.[6]

There is great potential for causing harmful side effects with drugs in patients with postural hypotension. I would maintain that recommendations should be based on carefully performed studies in which the patients and their responses to treatment are clearly documented.

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2 Moss AJ, Glaser W, Topol E. Atrial tachypacing in the treatment of a patient with primary orthostatic hypotension. *N Engl J Med* 1980;302:1456-7.
3 Hiltbold P. Atrial tachypacing for primary orthostatic hypotension. *N Engl J Med* 1980;303:885.
4 Goldberg MR, Robertson RM, Robertson D. Atrial tachypacing for primary orthostatic hypotension. *N Engl J Med* 1980;303:885-6.
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6 Schirger A, Sheps SG, Thomas JE, Fealey RD. Midodrine. A new agent in the management of idiopathic orthostatic hypotension and Shy-Drager syndrome. *Mayo Clin Proc* 1981;56:429-33.

Macrocytic anaemia in patients treated with sulphasalazine for rheumatoid arthritis

SIR,—Dr M Greaves and colleagues (7 February, p 373) suggest that folate deficiency with sulphasalazine treatment of rheumatoid arthritis is an uncommon complication; this is not our experience. Since our report, which suggested that this problem may be more common in patients with rheumatoid arthritis than inflammatory bowel disease, we have seen a further five cases of macrocytosis associated with low serum folate concentrations in patients with rheumatoid arthritis treated with sulphasalazine. None of these patients had other possible causes of folate deficiency.

We acknowledged in the report that some of the patients had other medical conditions, but we considered these to be non-contributory. The bacterial overgrowth (case 1) caused by jejunal