better and slightly prolonged but again over 24 hours each bolus became progressively less effective. The dose of diamorphine was increased to 20 mg two hourly and the next day to 30 mg two hourly. At this dose the patient slept for 30 to 60 minutes after each injection and awoke in severe pain. She was referred for advice about pain control.

The intravenous diamorphine was discontinued and she was given 120 mg morphine per rectum (by suppository) every four hours (she was still vomiting but the rectum was empty). The dose of morphine was conservative: the equianalgesic dose for 60 mg intramuscular or subcutaneous diamorphine would be about 180 mg, and intravenous diamorphine is probably more potent in terms of its peak effect. Within 24 hours the patient was more or less pain free, alert, and beginning to mobilise. She remained on rectal morphine (100-120 mg four hourly), her pain was controlled, and she was up and about until her death 17 days later.

This patient appeared to have developed acute tolerance to intermittent boluses of diamorphine, and we have had experience of other patients with cancer pain running into similar problems with this method of administration. Occasionally enormous dose levels are reached. Our approach is to administer the opioid by another route—orally if possible or rectally or by subcutaneous infusion. It is invariably possible to achieve pain control in this way, often with a reduced equivalent dose of opioid—as in this case. This suggests that part of the explanation for the development of acute tolerance is pharmacokinetic. Changing the route of administration so that there is less fluctuation in drug concentration seems not merely to influence the duration of analgesia, but also the depth of analgesia produced by a given dose.

Bolus intravenous injections of diamorphine produce good analgesia, which, however, may be very short lived. In a patient with unremitting cancer pain intermittent bolus injections may result in intermittent analgesia with progressively longer periods of intervening inadequate pain relief. This cycle appears to encourage the development of tolerance, and we suspect that the crucial factor is the period of inadequate pain relief, which necessitates a larger dose of analgesic next time. The study of Dr Marshall and colleagues supports this contention. They used an inadequate fixed dose infusion of morphine postoperatively and appear thereby to have induced acute tolerance. There is extensive experience of the use of postoperative intravenous infusions of morphine and other opioids where no such problems have been reported when adequate analgesia has been achieved, as many of your correspondents showed.[1,2]

The great majority of patients with chronic cancer pain are well controlled with oral opioids. If the oral route is not available the rectal or subcutaneous routes are the best alternatives. Intravenous opioids may be indicated in the treatment of acute or postoperative pain but this route is rarely a good choice for patients with chronic cancer pain. The possible exceptions are children[3] and other patients with haematological disorders in whom subcutaneous or intramuscular administration may be contraindicated. A continuous infusion is preferable to intermittent bolus injections in such cases.

The incidence of acute tolerance to intravenous opioids in chronic cancer pain is low, but when it does develop it is a distressing and sometimes extremely difficult clinical problem to deal with. It can usually be avoided.

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1 Church JJ. Continuous narcotic infusions for relief of postoperative pain. *Br Med J* 1979;i:977-9.
2 Nayman J. Measurement and control of postoperative pain. *Ann R Coll Surg Engl* 1979;61:419-6.
3 Miser AW, Miser JS, Clark BS. Continuous intravenous infusion of morphine sulfate for control of severe pain in children with terminal malignancy. *J Pediatr* 1980;96:930-2.

New programme of antenatal care in general practice

SIR,—Dr G N Marsh (7 September, p 646) is to be complimented on his interpretation of the work of Dr Marion Hall and her colleagues and the implementation of her precepts into his own practice. I agree wholeheartedly that a doctor needs to see low risk mothers at perhaps only five key points in pregnancy—at booking, at 16 weeks, and at 28, 34, and 36 weeks[1]—and the remainder of the routine visits can be delegated with great advantage to the practice midwife unless or until the patient goes significantly past term.

However, the time honoured programme of regular visits to doctor or midwife not only has the advantage of simplicity but also establishes in the patient's mind that continuing interest is being taken in her condition and progress. For this reason I would criticise Dr Marsh for seeing his patient only once before the third trimester but, thence onward, would question whether it is necessary for her to be seen on almost every occasion by both doctor and midwife? This seems to be an unnecessary duplication of effort and rather profligate deployment of man/woman power within the primary health care team.

Secondly, in such a forward looking and innovative practice, I was surprised that Dr Marsh made no mention of routine screening for neural tube defects in the fetus. While grosser defects may perhaps be detected by ultrasonography at 16 weeks, surely the value of α fetoprotein estimation in the maternal serum is accepted as worth while by most health authorities in the United Kingdom? Certainly in Oxford general practitioners have taken part in such a scheme for several years with considerable success.

Finally, in describing his new style antenatal care programme for low risk nulliparous women, Dr Marsh fails to define his criteria for selecting them. While low risk multiparas can be identified from their obstetric history with reasonable reliability, in nulliparas the judgment is essentially a retrospective one. It is my experience that up to half of all nulliparas will develop deviations from normality of some degree in either pregnancy or labour. Therefore, if one holds the view that in fact there is no such thing as a low risk nullipara might not the implication of a dual standard of care for this category be regarded as somewhat rash?

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1 Bull MJV. A problem orientated approach to antenatal care. *Medicine in Practice* 1984;2:9-23.

**Dr Marsh replies below.—ED, *BMJ*.

SIR,—I am heartened that Dr Bull agrees with the reduction in attendances to the doctor for antenatal care and I would not quarrel too fiercely over which particular consultations he feels he himself should continue. But to suggest that the remainder of the "time honoured" visits should be "delegated" (his words, and certainly not mine) to the midwife on the grounds of simplicity, and "to establish in the patient's mind that continuing interest is being taken in her condition and progress" seems anachronistic. It certainly demeans the role of the midwife—she is a fellow professional who like him expects her work to be clinically valuable—and continuing interest can still be maintained despite fewer attendances. It also undervalues the intelligence of women in the 1980s. If the women of Stockton on Tees can accept the logic of these reductions surely Oxford women can do the same. I suggest that they are given the opportunity.

As far as routine screening for neural tube defects by α fetoprotein estimation in maternal serum is concerned, it is accepted that it is of very doubtful value especially in regions where the incidence is relatively low. It may well be superseded by more sophisticated ultrasonography.

If Dr Bull rereads the first paragraph of Methods and Results in my paper he will see that the risk factor for nulliparous patients is determined at the beginning of their pregnancies. I would agree that with hindsight a goodly proportion of nulliparous women have developed deviations from the norm in their pregnancy and their labour, but many of these prove to be of little concern in terms of the outcome for mother and baby, and in this practice their care is left to the primary health care team.

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GP obstetrics: safe but endangered

SIR,—I was interested to read Dr David Jewell's essay on GP obstetrics (14 September, p 711). In Brackley, Northamptonshire, where I practise, we face the prospect of the closure of our small GP obstetric unit at the Cottage Hospital and the transfer of our cases to Banbury (10 miles) or Oxford (23 miles). The labour ward suite was paid for and equipped largely by the enthusiasm and voluntary funds of the Friends of the Cottage Hospital in the 1970s. The prospect of its demise has provoked a large protest petition from the local population. They see their town growing rapidly with the advent of many young people and this facility being removed.

To eliminate the element of maternity choice for purely short term economic reasons and to centralise the service is not acceptable in the context of increasing community care. If the future lies with increasing local involvement and enthusiasm in all aspects of health care, then surely local facilities in rural areas such as ours should be preserved. Dr Jewell makes a very valid point when he emphasises the advantage of familiar faces at delivery. It is also important to ensure that the obstetric skills acquired by GPs do not wither by disuse atrophy.

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Carbamazepine induced systemic lupus erythematosus

SIR,—Dr D E Bateman (7 September, p 632) describes a case of systemic lupus erythematosus in a young woman, but I cannot find any evidence in his report that suggests that the condition was induced by carbamazepine. Furthermore, when the drug was withdrawn prednisolone 30 mg daily was also added. The patient improved and prednisolone was stopped after six months but one year later she still had a positive antinuclear factor titre of 1/60, although DNA binding had returned to normal. There is nothing in this history that is inconsistent with a spontaneous case of systemic lupus erythematosus.

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**Dr Bateman replies below.—ED, *BMJ*.

SIR,—Absolute proof is, of course, lacking. The only way to have obtained this would have been