It took 50 years for midwives to administer nitrous oxide to patients in labour. I look forward to the day when they are trained to provide epidural analgesia.

A B W TAYLOR

Bowthorpe Maternity Hospital,
Wisbech, Cambs

SIR,—I have read the article by Mr A B W Taylor and others (6 August, p 370) with interest. However, I firmly believe that at the present time an obstetric epidural service should be provided by anaesthetists and should be available only when there is a resident anaesthetist on call in the hospital.

The reason for this uncompromising attitude is that the greatest among the risks inherent in the procedure is the possibility of local anaesthetic being introduced into the cerebrospinal fluid. The resultant "total spinal anaesthesia" is a situation requiring resuscitation skills of the highest order, and when these are present a fatal outcome can be prevented. In this situation I think that anaesthetists, by virtue of their training and constant practice in this field, are much more likely to be successful than obstetricians. It is one thing for the latter to be taught to pass an endotracheal tube in the anaesthetic room under ideal conditions but a totally different one for them to cope with this and other aspects of management in the acute state of chaos that would be precipitated by a "total spinal" occurring in a labour ward.

I was relieved to see that Mr Taylor's team did take steps to minimise the possibility of high spinal anaesthesia. Unfortunately there is no way of ensuring it will not happen, nor is it always a complication apparent with the first dose of local anaesthetic. Obstetric epidurals have been a momentous step forward, but until we have accurate data on the incidence of complications and a realistic assessment of the procedures I do not think it is justified to relax the standards that have so far been applied to its use in Britain.

J M ANDERTON

Royal Infirmary,
Manchester

Metronidazole neuropathy

SIR,—Further to the account of recovery in metronidazole neuropathy by Professor W G Bradley and others (3 September, p 610) we wish to report a case in which complete subjective recovery has not apparently resulted two years after discontinuing the drug.

The patient is a 20-year-old girl with extensive Crohn's disease and episodes of bacteraemia and abscess formation. Prior to surgery she was treated with oral metronidazole 800 mg twice daily for seven weeks before developing numbness of the hands and feet. Examination then demonstrated diminished ankle jerks and reduced sensibility to pinprick and cotton wool below mid-calf. There was also apparent left calf muscle wasting, but weakness was not present. Fasting blood sugar, folate, and vitamin B₁₂ levels were normal; other drugs currently taken were prednisone, folic acid, and ferrous sulphate.

The metronidazole was discontinued and the hand numbness resolved over four weeks. Fourteen weeks after withdrawal of the drug the tendon reflexes were brisk and sensory testing revealed distal hyperaesthesia in the legs, more marked on the left. However, two years later she continues to be distressed by numbness in the feet, although objective signs are questionable. The patient has not consented to nerve conduction studies.

In all previously reported cases,¹⁻⁴ as in that reported by Professor Bradley and his colleagues, full symptomatic recovery was generally apparent within four months of metronidazole withdrawal. This case is exceptional for the duration of symptoms in the lower limbs. The total amount of metronidazole in reported cases of neuropathy has ranged between 30 and 314 g. In this instance it was 78 g, implying that duration and severity of symptoms in metronidazole neuropathy may not be strictly dose-related.

I J KARLSSON
ADRIAN N HAMLYN

Royal Victoria Infirmary,
Newcastle upon Tyne

¹ Ingham, H R, *et al*, *Journal of Antimicrobial Chemotherapy*, 1975, **1**, 355.

² Ursing, B, and Kamme, C, *Lancet*, 1975, **1**, 775.

³ Ramsay, I D, *British Medical Journal*, 1968, **4**, 706.

⁴ Coxon, A, and Pallis, C A, *Journal of Neurology, Neurosurgery and Psychiatry*, 1976, **39**, 403.

SIR,—Dr W G Bradley and his colleagues (3 September, p 610) state that metronidazole neuropathy is mild and recovery appears to be complete. We strongly disagree. Although the patients described by Ursing and Kamme¹ indeed fared well, those described by Ramsay² and by Coxon and Pallis³ developed a severe neuropathy which certainly did not regress completely on stopping the drug. We have a further patient who developed a severe sensory neuropathy with intolerable dysaesthesiae four months after starting metronidazole for Crohn's disease. Seven months after stopping the drug there has been no improvement, either clinically or in nerve conduction studies.

In our view patients starting long-term therapy with metronidazole should be specifically warned about this complication and should be told to stop the drug and report immediately if any neurological symptoms occur.

S HISHON
JOHN PILLING

Norfolk and Norwich Hospital,
Norwich

¹ Ursing, B, and Kamme, C, *Lancet*, 1975, **1**, 775.

² Ramsay, I D, *British Medical Journal*, 1968, **4**, 706.

³ Coxon, A, and Pallis, C A, *Journal of Neurology, Neurosurgery and Psychiatry*, 1976, **39**, 403.

Shortage of anaesthetists

SIR,—Mr A W Fowler (27 August, p 576) makes a plea for the increasing use of local anaesthesia for surgical procedures and advocates a combination of diazepam and Cyclimorph. May I offer him an alternative technique which is enjoying popularity in dental circles and which he might find of value?

Dentists are, for obvious reasons, very keen to avoid nauseating drugs. The combination of cyclizine and morphine in Cyclimorph seems designed to produce narcosis without nausea but, as Mr Fowler admits, this may only postpone the nausea so that it embarrasses the nursing staff rather than the surgeon. An alternative which has been found virtually non-nauseating is to use pentazocine with the diazepam. A standard dosage of 30 mg pentazocine intravenously will produce analgesia comparable with that produced by 10 mg morphine but without the nausea. This is then supplemented by diazepam given intravenously at the rate of 2.5 mg/30 s until sedation is evident. Both drugs take some

minutes to reach their full effect and so the administration of diazepam is stopped short of the desired point in the knowledge that sedation will deepen. The stage of ptosis where the upper eyelid droops to bisect the pupil has been found to be a reliable sign of adequate sedation.

Using this method it is possible to perform a single dental extraction without further anaesthesia, and together with effective local anaesthesia the most complicated surgical extractions may be performed without disturbing the patient and without leaving any memory of the procedures. This would seem to equate with Mr Fowler's reduction of fractures and orthopaedic operations.

It is also surprising how little diazepam is actually needed to eliminate distress and produce anterograde amnesia, and in dental operations I have never found any need for more than 20 mg of diazepam in combination with pentazocine. Readers will no doubt assess for themselves the degree of fear which is inspired by dental procedures as distinct from those on any other part of the body.

Finally, although long-term medication with benzodiazepines may well lessen the effect of diazepam, in practice this does not so far seem to be much of a problem. Dosage may be increased, but since one is working with an "approach dose" technique the result is still effective. It would be interesting to know whether Mr Fowler has come across any grossly increased dosage—or, indeed, any failures of sedation—which might be attributed to benzodiazepine habituation.

P SYKES

Amphill, Beds

Strawberry pickers' foot drop

SIR,—In reply to recent letters concerning strawberry pickers' foot drop I write to inform you that this is a relatively common disorder in the Fenland area during the strawberry season.

I had been completely unaware of this condition until I arrived here and was most alarmed at the first case that I saw but was quickly reassured by one of my senior partners. Between us in this group practice of six we have seen five cases of this disorder this year and we feel that the majority of them tend to be in casual labourers who are less aware of the consequences of continued crouching. It is a condition very rarely seen in the fenman.

K E WARREN

Wisbech, Cambs

* * * This correspondence is now closed.—ED, *BMJ*.

Inhumanity to man

SIR,—The title of your leading article (3 September, p 591) makes your position clear, and many should agree with you that the increasing failure of psychiatric hospitals to admit patients who have committed offences is inhumane. Yet as the problem becomes increasingly acute, swelling the prison population (at a cost of over £4000 a year per inmate), there seems to be no effort to solve it or at least ease it. The regional secure units, in the planning stage, will cope with only a minute proportion of patients requiring hospital care, and none is yet established, but by

recommending them the authorities have given the opportunity to psychiatrists and nurses to refuse disturbed offenders with the excuse that their hospital does not provide security or that these patients will disturb others. Many of the patients now refused are not patients likely to be accepted by the secure units when they exist. They are individuals often in the category you describe who should be cared for in psychiatric hospitals now (as they always were in the past) and will still require accommodation in them in the future.

Psychiatric hospitals have a responsibility to provide a comprehensive psychiatric service short of maximum security accommodation and psychiatrists have never agreed a policy with the Department of Health and Social Security which would relieve them from that obligation. Despite this it is a selective policy that has slowly been adopted and apparently accepted (by default) by the Department without any alternative service being proposed by anyone. The DHSS says that in the end it cannot insist that a psychiatrist cares for a particular patient. The doctors say the Department fails to provide alternative care. So that in practice there is an impasse. The Department would also make the point that it has distributed some £5m or more annually in 1976 and 1977 to develop "interim facilities" before regional secure units are established, but figures recently published indicate that most hospital regions find that very little of this money is being taken up and spent as intended.

All this is a serious problem which is unique in not being primarily a financial one. It is to be hoped therefore that a more urgent effort will be made by all concerned to solve it. The prisons, after all, cannot refuse people sentenced by the courts as hospitals can. Perhaps the courts should be more selective in deciding who really requires custody. More remands on bail will help.

R S BLUGLASS

Midlands Centre for Forensic
Psychiatry,
All Saints Hospital,
Birmingham

The neonatal electrocardiogram and unexpected death in infancy

SIR,—The excellent studies reported by Dr D P Southall and others (3 September, p 597) and by Dr B R Keeton and others (p 600) of cardiac arrhythmias found on routine screening in infants and the conduction disorders in babies found in a collapsed state call for a short comment in view of their recommendations of clinical screening related to cot deaths.

Unexpected death in infancy is a clinical syndrome only. It has varying aetiologies and different causes of death. However, among these there is both indirect evidence, as from papers such as those cited above, and also direct evidence from necropsy studies of hearts that indicate that a group of these deaths have their chief cause within the heart. The suggestion of neonatal electrocardiogram screening for such lesions is an obvious one and when in 1973 we set up a prospective study in Sheffield with a view to identifying features in babies at increased risk of unexpected death we included a neonatal ECG in the study of all high-risk infants. In a cohort of 11 424 babies, we made a special study of 627 who were thought to be at increased risk of unexpected death. In the four babies of this cohort that subsequently died

no abnormalities were noted in the neonatal ECG. One of these deaths was probably of cardiac origin.

From a present logistic viewpoint, if we assume that one in 10 cot deaths has a cardiac cause, which is my present estimate, we would need to score at least 5000 general births to hit one case and if the figures published by Dr Southall and his colleagues are general we would, in this group, pick up perhaps 100 children with arrhythmias with, at present, no evidence that such ECG changes predispose to unexpected death.

The situation at the present time would appear to call for further, more detailed study and refinement in our tools of assessing the heart in infants rather than widespread screening.

JOHN L EMERY

Department of Histopathology,
Children's Hospital,
Sheffield

SI units and acidity

SIR,—One of the by-products of the introduction of SI (Système Internationale) units has been to cast doubt on what are the normal mean values for hydrogen ion concentration of the various biological fluids. For arterial blood the customarily accepted mean of normality has been pH 7.40 or 40 nanomoles of hydrogen ion per litre. This mean has been obtained in the past by averaging observed pH measurements from large numbers of samples.

If a series of pH values are taken which average 7.40 and then the average is taken of the SI equivalents the results must differ—an example is given in the accompanying table. This is because averaging logarithmic numbers is a root function (square root, cube root, N^{th} root, depending on how many values there are). In blood gas homeostasis the differences are of academic rather than clinical interest, as the range of pH values is so narrow. When considering other biological fluids such as gastric secretion or urine acidity significant errors can arise. In the example in the table the real mean hydrogen ion concentration of gastric secretion is 30 times more than that obtained by converting the average of the pH values. The implications are that our older textbooks and published articles which give mean pH values are wrong, since these mean pH values are not the true mean hydrogen ion concentrations even if the latter are expressed in pH notation.

The problem is further compounded where standard deviations are concerned. The growing laboratory practice of indicating the degree of abnormality of a particular bio-

pH values and SI equivalents (mol/l) of blood and gastric juice

pH	SI equivalent
<i>Blood</i>	
7.2	63×10^{-9}
7.4	40×10^{-9}
7.6	25×10^{-9}
Average 7.4	42.6×10^{-9} **
<i>Gastric juice</i>	
7.0	10×10^{-8}
5.0	10×10^{-4}
3.0	10×10^{-11}
Average 5.0	3.3×10^{-14} †

*but 42.6×10^{-9} mol/l = pH 7.37.

†but 3.3×10^{-14} mol/l = pH 3.52.

chemical measurement by the number of standard deviations by which that measurement differs from the normal mean imposes a heavy reliance on the validity of that mean. Further, if the standard deviation has been calculated in pH units and subsequently converted, then any connection between this and the true standard deviation is likely to be coincidental.

Fundamental to this is the underlying rationale of reporting biochemical data. Ideally to the clinician any departure from normality should be related to the extent to which that departure is a threat to health. If this threat is either alinear or asymmetrical about the mean, then reporting in terms of standard deviations either in SI mode or logarithmic (pH) mode is a cumbersome irrelevance. If the distribution of normal values for hydrogen ion concentration does not follow a Gaussian curve, then the simple linear scale proposed by the SI system offers no advantage to the clinician.

Could our biochemical colleagues give us guidance? The question is not so academic as it seems—how is excessive acid production to be identified or how is one to judge whether antacids or H^2 receptor blockers are better at controlling intragastric acidity?

P J TOMLIN

University Department of
Anaesthetics,
Queen Elizabeth Hospital,
Birmingham

Miscarriages as a cause of spina bifida and anencephaly

SIR,—The data presented by Professor K M Laurence and Dr C J Roberts (6 August, p 361) confirm much of those of Clarke *et al*¹ and would seem to indicate that the reproductive performance of mothers of anencephaly spina bifida (ASB) fetuses improves progressively if it is accepted that a miscarriage represents a greater disturbance than the survival of an ASB fetus beyond 28 weeks' gestation. The behaviour of the control group is predictable, since except at the extremes of the reproductive life span the maternal reproductive capacity remains relatively stable and at its maximum efficiency.²

However, the data from (ASB) families are, in my opinion, open to an alternative interpretation which, like that of Professor Laurence and Dr Roberts, does not lend support to the hypothesis that early miscarriages are the cause of malformations in subsequent pregnancies.¹ Indeed, the latter concept is extremely difficult to accept on teleological grounds since it would seem very unlikely that the major mechanism by which abnormal conceptuses are eliminated should in itself be the cause of the most common of human major malformations.

Postconceptional subfertility must by definition result from the loss of fertilised ova. The greater part of this loss is due to inherent defects within the conceptus and occurs before the first missed period, a relatively small proportion occurring as clinically manifest spontaneous abortions.³ A very small proportion of these defects result in late abortions and premature births. The evidence that the interval between miscarriage and the next pregnancy in both the ASB and control series is similar indicates that the very early losses must be comparable in both groups. Thus the differences in reproductive behaviour would appear to lie in the number of clinically mani-