receiving oral lorazepam and term babies of mothers with no drug therapy except epidural bupivacaine. This finding may still be consistent with our own finding if the maternal doses were different. In our study the babies with delayed feeding had mothers who had received the relatively high mean lorazepam dose of 5·5 mg a day for a mean of 22 days. I suspect that the dosage used in Manchester was lower or shorter in duration. Our cord blood analyses suggested that sedation of the infant was related to plasma lorazepam level and to dosage.

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Painful iceberg

SIR,—In a recent study[1] we asked multiracial groups of apparently healthy doctors attending postgraduate courses to make a self-diagnosis of the irritable bowel syndrome. Although this method of diagnosing irritable bowel syndrome was not ideal, we found that about 13% of white doctors thought that they had the syndrome—a rate not dissimilar from that found in the Bristol study[2] mentioned in your leading article (2 May, p 1414).

We also found that over 18% of non-white doctors thought that they had irritable bowel syndrome. In male non-white doctors aged 25-49 years the prevalence of cows' milk intolerance in those who had irritable bowel syndrome (20 out of 29 doctors) was much higher than in their counterparts who did not have irritable bowel syndrome (29 out of 115 doctors) ($p < 0.0001$). These results suggest, as Pena and Truelove have already indicated,[3] that the syndrome may in some cases be a manifestation of lactase deficiency. In the white doctors milk intolerance occurred much less frequently. It is highly likely that the prevalence of irritable bowel syndrome varies greatly between different ethnic populations and is influenced by many cultural and dietary variables as well as genetic factors.

The considerable iceberg of irritable bowel syndrome in the community and the common occurrence of the syndrome in general practice may indicate that those patients reaching hospital are a highly selected group whose disease might differ quite markedly from that of those who are never seen in a gastrointestinal clinic. Virtually all the research into the management of irritable bowel syndrome has been carried out on hospital patients. Clearly the results may not be applicable to the syndrome as it occurs in the community. More research in the general practice setting would be welcome. If, on the other hand, patients reaching hospital have a spectrum of disease similar to that occurring in the community, invasive and costly investigations may not be warranted.

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[1] Fowkes FGR, Ferguson A. *Scott Med J* 1981;**26**:41-4.
[2] Thompson WG, Heaton KW. *Gastroenterology* 1980; **79**:283-8.
[3] Pena AS, Truelove SC. *Scand J Gastroenterol* 1972; 7:433-8.

Treatment of ulcerative colitis

SIR,—In your leading article (18 April, p 1255) the advice is given that patients with severe attacks of ulcerative colitis "should be monitored daily with abdominal radiographs." This is lamentable advice, which I hope is not taken seriously.

What kind of radiography is contemplated? What is likely to be the quality of the films in a severely ill patient unfit to stand? For how many days should the performance be continued? What are the short-term and long-term hazards? No mention is made of abdominal findings as an indication for such radiology.

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**An acute severe episode of ulcerative colitis rarely lasts more than four days, but silent perforation in patients having high-dose steroids and toxic megacolon onset are not uncommon occurrences. They may occur without appreciable change on clinical examination even by the most experienced alert physician. Supine and erect abdominal radiographs can be performed at the bedside with little extra distress to the ill patient. The quality of the radiographs is sufficient for reaching a diagnosis. Prompt surgical intervention is required if a perforation occurs, and within 48 hours if toxic megacolon does not respond to medical management.—ED, *BMJ*.

Virus particles in oysters

SIR,—We have recently discussed virus infection as a possible cause of foodborne gastroenteritis of unknown aetiology (30 May, p 1801). We report here our first observation of virus particles in shellfish responsible for an outbreak of non-bacterial gastroenteritis.

During December 1980 an outbreak of gastroenteritis occurred among a group of people who attended a buffet meal at which oysters were served. The incubation period was 36-56 hours and symptoms included both vomiting and diarrhoea. Bacteriological examination of faecal specimens from five patients and samples of the remaining oysters failed to reveal the presence of campylobacters, salmonellas, shigellas, *Staphylococcus aureus*, *Bacillus cereus*, *Clostridium perfringens*, or the vibrio group. The oysters gave total viable counts per gram of $4·0 \times 10^3$ at 37°C and $8·9 \times 10^5$ at 22°C after 24 hours' incubation. *Escherichia coli* was detected but the level was <1 per gram.

Faecal specimens from four patients were also examined by electron microscopy. Small, round, featureless viruses were observed in all four specimens. Similar particles were detected in the intestinal tracts dissected from some of the oysters. The particles were morphologically similar to parvovirus-like particles seen in faecal specimens from several other outbreaks associated with the consumption of shellfish, as reported in our recent article and by Appleton and Pereira[1]; but this is the first time that such particles have been seen in shellfish in this country.

In a large outbreak of gastroenteritis in Australia in 1978 parvovirus-like particles were also observed in both faecal specimens and in one batch of oysters.[2] In the same outbreak several other viruses were detected in the faecal specimens by both culture and electron microscopy, but in the outbreak we studied, small, round, parvovirus-like particles were the only viruses found in the patients and the oysters.

This report emphasises that a satisfactory bacteriological result obtained during routine monitoring of shellfish does not guarantee the absence of potentially pathogenic viruses.

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[1] Appleton H, Pereira MS. *Lancet* 1977;i:780-1.
[2] Murphy AM, Grohman GS, Christopher PJ, Lopez WA, Davey GR, Millsom RH. *Med J Aust* 1979;ii: 329-33.

Spinal meningioma presenting as focal epilepsy

SIR,—I was most interested to read the report (16 May, p 1584) of a spinal meningioma causing focal attacks in the right lower limb. However, I would question the authors' conclusion that there has been "no other published description of spinal epilepsy with such a clear focal motor event occurring with a spinal lesion," unless it is their intention to imply neoplastic lesions only.

I think it possible that these attacks are related in some way to the tonic seizures which are well described by Matthews in multiple sclerosis.[1] In Matthews's cases involvement of the lower limbs in these seizures was less common than the upper limb, but when involved the limb was extended and the foot plantar flexed, which is very similar to the attacks in the present case. The duration was also similar, in all cases being less than 90 seconds and in most less than 30 seconds. The seizures described in multiple sclerosis were in contrast often painful and often induced by movement, but neither of these features occurred in all the cases.

Presumably, and as I think the authors imply, the similarity could be extended to an anatomical level. The spasms in the patient described may well have originated in the lateral corticospinal tract and not in grey matter (as in Hughlings Jackson's quoted definition of epilepsy). The tonic seizures in multiple sclerosis have been attributed to lateral spread of excitation between demyelinated axons in the lateral columns of the spinal cord.[2]

It may also be relevant that in discussions of the neuropathology of spinal cord compression it has often been suggested that myelin is particularly vulnerable to mechanical pressure and to minor degrees of anoxia. McAlhony and Netsky,[3] in their study of the neuropathology of cord compression, observed that the greatest area of demyelination often lay either immediately under a tumour or on the opposite side of the cord.

If the abnormal activity were occurring in grey matter at the segmental level of the observed movements, then the compressive lesion must have been exerting its influence by an alteration in supraspinal influences. If this were the case, then I think one must say that the phenomenon has more in common with the spasms frequently observed in cord compression, which are thought to be due to reflex activity.

Nathanson[4] has described a patient, shown to have a meningioma causing cord compression at the level of the first thoracic vertebra, who presented with similar tonic spasms affecting the left leg. In the patient the

attacks were painful, but in other respects were comparable.

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[1] Matthews WB. *J Neurol Neurosurg Psychiatry* 1975; **38**:617-23.
[2] Ekbom KA, Westerbery CE, Osterman PO. *Lancet* 1968;i:67.
[3] McAlhony HS, Netsky MG. *J Neuropathol Exp Neurol* 1955;**14**:276-87.
[4] Nathanson M. *Journal of the Mount Sinai Hospital* 1962;**29**:147-51.

Ethnic factors in disease

SIR,—As a practising gynaecologist in the West Midlands, I read your leading article (9 May, p 1496) on ethnic factors in disease with particular interest. Surely it is right to study the different incidence of various diseases in the migrant population and the different presentations of disease, as well as the individual approach of those of Asian and Caribbean origin to their illnesses. This knowledge can help the clinician. For example, carcinoma of the ovary and the uterine corpus are virtually unknown in our Asian patients while they are remarkably rare in West Indians. Invasive cervical carcinoma certainly occurs in Indian women and carcinoma in situ is increasingly common. These facts may reflect the relative youth of the Asian women.

It is helpful to remember that in practice postmenopausal bleeding and discharge in a 60-year-old Asian is more likely to be due to endometrial tuberculosis than carcinoma. Pelvic inflammatory disease seems to affect all ethnic groups but is less common in Asians, particularly of the first generation, who adhere to a strict moral code.

There are great demands for sterilisation from all groups but the Asians seem to want two sons and at least three children before considering the operation. The commonest indication for hysterectomy in Asians is, unfortunately, poststerilisation menorrhagia, the next commonest being carcinoma in situ. In contrast, our West Indians will consent to part with their fibroid uterus only when it is causing considerable menorrhagia or pain. Endometriosis is rare in black patients and, as yet, uncommon in Asians but common enough in Caucasians. The tolerance of spasmodic dysmenorrhoea, infertility, and menopausal symptoms varies greatly, reflecting different educational and social backgrounds. The young Asian teenager wishes to obey her parents and yet to behave as her Caucasian friends.

Obstetric differences would seem to be more easily measured. You mention perinatal morbidity but not maternal mortality. In our unit for example, 14 women have undergone emergency hysterectomy in the delivery suite in the past decade, when we delivered over 40 000 women. There were three deaths, all in Asians, who seem unable to withstand severe haemorrhage and emergency surgery as well as their Caucasian sisters.

The differences in the incidence and nature of disease between the various ethnic groups are fascinating in practice and may give some guide to the aetiology of such appalling diseases as carcinoma of the ovary and carcinoma of the breast, both much less common for the moment in Asian and West Indian women.

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Electrocardiographic signs of pulmonary hypertension in children who snore

SIR,—The recent paper by Dr A R Wilkinson and others (16 May, p 1579) concerning the prevalence of pulmonary hypertension and right ventricular hypertrophy in children with enlarged tonsils and adenoids is clearly very important and has considerable consequences both in ENT and in general paediatric practice. However, we consider that the recommendation to perform electrocardiograms on all children who snore will flood the ECG department with many unnecessary requests.

We share the authors' concern that the prevalence of right ventricular hypertrophy and pulmonary hypertension is grossly underestimated and we have in this hospital attempted to define the problem a little more clearly. We have performed transcutaneous multiple gas analysis by mass spectrometry on over 50 selected children during sleep to assess the degree of hypoxia or hypercapnia that might be reversed by adenoidectomy and tonsillectomy. Thirty-seven of these children have undergone surgery. Thirty-two of the 37 had a history of snoring (25 frequent snoring and seven occasional snoring). Forty-one per cent of this group had normal transcutaneous oxygen and normal transcutaneous carbon dioxide levels; 45% had abnormal oxygen and carbon dioxide levels (11 children had a high carbon dioxide and normal oxygen, six children had both a high carbon dioxide and low oxygen).

Many children who snore will have normal transcutaneous gases. Indeed, in a series of children admitted to this hospital for surgery not related to ENT the incidence of snoring was 13%. Since chronic hypoxia and chronic hypercapnia are the likely causes of pulmonary hypertension in children who have chronic upper airways obstruction, it is the children with abnormal gases who are likely to be at most risk. So far we have only been able to take two of the more severely affected children from this group for further studies. Neither of them had pulmonary hypertension on ECG or echocardiography.

When we look more closely at the symptoms of children in our series with abnormal transcutaneous gases we find that the single most consistent finding was of periodic apnoea, which was either reported by the parents or observed during our studies. The simple question "Does he ever appear to stop breathing when he is asleep?" may further differentiate those children at most risk and it is these children in whom an ECG is desirable.

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Investigations on women with a single kidney

SIR,—Your expert's reply to the question about investigations in a woman with a single kidney (28 March, p 1053) is incomplete as it contains no reference to the fact that patients in whom the urinary tract has developed on only one side frequently have a similar abnormality of the genital tract, with a single Fallopian tube and a unicornuate uterus. Such patients are liable to complications in pregnancy, such as abortion, premature labour, inertia, malpresentations (especially breech), and third-stage complications. I therefore suggest that any woman with a single kidney should also have either a laparoscopy or hysterosalpingography.

Conversely, whenever a patient is found to have a unicornuate uterus an intravenous pyelogram should be carried out, which may reveal a single kidney on the same side.

B ETON

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**The association Mr Eton reports is well known to obstetricians, though not mentioned in some reference books on anatomical variations. Despite the association we doubt whether the procedures he recommends for women of childbearing age should be routine. All obstetricians observe their pregnant patients for the complications of pregnancy to which he refers. The invasive procedures of laparoscopy and hysterosalpingography, which use hospital resources and skilled staff and are not without risk, would not seem to be justified simply because a woman is known to have a single kidney. There is certainly no great merit in doing an intravenous urogram in patients found to have an unicornuate uterus, since all that can be said is "How interesting"—which does not seem to justify the procedure's cost and risks, even though these are small. If Mr Eton's suggestions were considered valid we could argue that every pregnant woman should have an intravenous urogram to detect whether she has a single kidney, and therefore perhaps a unicornuate uterus, as the abnormality is not very uncommon. When there is one congenital abnormality there may well be others, and this point is worth making. In obstetric practice any abnormality, including those, such as congenital absence of kidney, that are known to be associated with genital abnormalities, might be considered to provide stronger reasons than some others for specialist supervision during pregnancy and delivery.—ED, *BMJ*.

Drug-induced oesophageal ulceration

SIR,—The case of naftidrofuryl (Praxilene)-induced oesophageal ulceration reported by Drs Elizabeth C McCloy and Stephen Kane (23 May, p 1702) is supported by a recent personal observation.

My wife has suffered from intermittent claudication for the past five years. Four years ago she was started on naftidrofuryl 100 mg thrice daily with an additional 200 mg prior to undertaking prolonged walking. The response in relieving her symptoms was excellent and no side effects were noticed. Three years ago she suffered a massive posterior myocardial infarction, and as a result her activities were curtailed; so for a year her daily dosage of naftidrofuryl was stopped except for 200 mg prior to going for a walk. Some five weeks ago she experienced nocturnal leg cramps and took naftidrofuryl 100 mg with a sip of water immediately before going upstairs to retire. After two days she developed retrosternal pain and pain in the back of the neck radiating into the upper arms and both shoulders. At first it was thought that the effort of climbing stairs at the end of the day was producing angina of effort, but it was noticed that the pain was accompanied by marked spasm of the lower trapezeus and supraspinatus muscles, which was atypical. The discomfort or spasm was not relieved by glyceryl trinitrite 500 μg sublingually, but some relief was obtained on massage and on spraying the back of neck and shoulders with an analgesic spray. During the next two days she had persistent discomfort in her neck with increasing pain during