view of the great variation in management,[3] the results of such a trial would help women with bleeding in early pregnancy, and their general practitioners, decide what is best.

Women are dissatisfied with the care that they receive when they miscarry,[4] and the lack of effective treatment for threatened miscarriage probably contributes to their dissatisfaction. Perhaps, it is the medicalisation of miscarriage—a sad but common physiological event—that has greatly contributed to women's dissatisfaction. If so, we as doctors have only ourselves to blame.

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Better quality data for Down's syndrome register

EDITOR,—We agree with Kevin Spencer[1] and M R Gaudoin[2] that the data on the national Down's syndrome register[3] depend on the quality of information provided to the cytogenetic laboratory by the referring clinicians. In about half of the cases on the register we obtain additional information from the clinicians that was not supplied to the laboratories, and we would of course welcome more. Perhaps the most pressing need in evaluating the current genetic service is data on the proportion of mothers who are offered different types of screening and the numbers of amniocenteses that follow. Some but not all of this information is held by local laboratories, and there is a need to aggregate these data. We also agree that it is not always easy to know whether ultrasound scanning for malformations preceded or followed serum testing, and there is certainly a problem with the terminology used to describe the tests performed.

We have, however, perceived a considerable and continuing improvement in the quality of data we have received over the four years that the register has been functioning, and the consistent nature of the trends we have reported is some evidence of their validity. Preliminary analysis of data entered on the register by February this year shows that referrals after positive results of serum screening rose from 7·8% of all diagnoses in 1991 to 11·7% for the first half of 1992. In the same period, cases in which ultrasound findings were reported as the prime indication for fetal karyotyping rose from 7·7% to 9·3%, and cytogenetic referrals said to be for raised maternal age fell from 20% to 16·5%.

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Coding of clinical diagnoses

Clerical and medical errors contribute to inaccuracy

EDITOR,—C Yeoh and H Davies found that the accuracy of inpatient clinical coding improved when responsibility was transferred from clerical to medical staff.[1] Reasons for miscoding, however, are complex. We recently used data on inflammatory bowel disease of juvenile onset held in Scottish Hospital In-Patient Statistics to examine this subject.[2] We derived a geographically based sample of 255 patients aged 1-20 who had been coded in the statistics as having either Crohn's disease or ulcerative colitis,[3] and we examined the relevant hospital case records. We found that the coded diagnosis was incorrect in 47 (18·4%) instances.

In only 16 cases was the error clerical; in each of these cases the clinical records clearly showed that the doctors had made some other diagnosis but the summary form prepared by a coding clerk had the code number for Crohn's disease or ulcerative colitis. Most of these clerical errors were for patients with conditions with names similar to synonyms for Crohn's disease or ulcerative colitis.

In 24 cases the doctors' clinical diagnosis was subsequently shown to be wrong, although in most of these cases the available clinical information was compatible with the diagnosis made at the time. In 13 of these cases the diagnosis was revised when more clinical and laboratory information became available; in the 11 others symptoms settled and the patient was discharged from follow up without any firm alternative diagnosis being made. In seven other patients there was merely a misclassification within irritable bowel disease, in six because the clinical features in the early stages of disease did not allow definitive diagnosis and in one because of a clerk's miscoding.

In view of the implications of the diagnosis of incurable chronic but treatable illnesses such as irritable bowel disease we suggest that the degree of confidence in the clinical diagnosis (possible, probable, definite), or the absence of any firm diagnosis, might reasonably be incorporated in the coding system. A way of doing this should be considered when new decisions on coding policies are being made.

Finally, although prompt dictation and typing of a discharge letter may seem ideal, in some cases it may be sensible to delay final documentation until critical radiological or pathological reports are available. An early but incomplete discharge document may be administratively tidy but is clinically and epidemiologically meaningless.

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Persevere with Körner system

EDITOR,—Given the low quality of captured data on diagnosis reported by previous studies,[1] it is refreshing to read that C Yeoh and H Davies resolved this problem by purchasing a new information technology system and transferring the entire responsibility of clinical coding to medical staff.[2]

But given the already considerable workload of medical staff, which is likely to get even worse with the gradual implementation of the junior doctors' new deal on working hours,[3] it is doubtful whether doctors in most other hospitals could take on the burden of coding clinical material and entering the codes into the computer. New systems implemented by individual departments or hospitals incur additional costs and they are not uniform, thus making linkage and comparison with other units and hospitals more difficult. The Körner information system is superior in this regard because it was standardised across all regions in the country.[4] It would be preferable for hospitals to identify and rectify the reasons for inaccuracy of their existing Körner information system. Furthermore, there is provision within the system to expand data capture in order to include items of local interest or needs.

In a recent study at Leicester General Hospital (presented at the meeting of the Medical Research Society, April 1993) we found that completeness of Körner coding (performed by trained clerical staff) was virtually 100%, and recorded codes were correct in 75%, partially correct in 19%, and incorrect in only 6% of cases. These findings are more encouraging than in previous reports, but clearly there is room for improvement. The main source of error was insufficient information given by junior doctors in the document from which coding clerks derived the diagnoses. One way to improve doctors' contribution to clinical coding is to ensure that the appropriate diagnoses are entered on the coding document during the consultant and registrar ward rounds each time a patient discharge is arranged. As patients' details and diagnoses are usually reviewed by the more senior staff just before their discharge, correct entry of clinical data for coding can be achieved quickly and with minimal added effort. Coding clerks could then allocate the appropriate codes to the right diagnoses. Our study of 117 patients found that experienced coding clerks allocated an incorrect code despite thorough and clearly presented diagnoses in only two cases.

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Hepatitis C from immunoglobulin infusions

EDITOR,—In the Hammersmith staff round on chronic liver disease due to hepatitis C the discussion group thought it unlikely that the patient, who had common varied immunodeficiency, could have acquired hepatitis C after immunoglobulin infusions: Levi states that "immunoglobulin preparations . . . are treated to render viruses inactive."[1] Immunoglobulin preparations in present use are treated to inactivate viruses, and screening of blood donors for hepatitis C makes the chance of infection less likely. In the early 1980s, however, several commercial immunoglobulin preparations caused outbreaks of non-A, non-B hepatitis, most of which have since been confirmed as having been hepatitis C.[2]

In one of the best documented studies use of intravenous immunoglobulin prepared by the British Blood Products Laboratory with alcohol fractionation led to the development of non-A, non-B hepatitis in 12 patients with agamma-

globulinaemia.[3] The consequence of this infection in these patients has been devastating: four of nine with common varied immunodeficiency and hepatitis C have died from liver disease, one after liver transplantation. Four of the remaining patients with common varied immunodeficiency have died from other complications, which may have been precipitated or exacerbated by chronic hepatitis C. Other studies have also documented the poor prognosis of this disease in patients with hypogammaglobulinaemia.[4]

The patient under discussion received intravenous immunoglobulin infusions in the early 1980s. Without further information as to the source of the immunoglobulin preparation it is not possible to ascertain whether she acquired the hepatitis C from immunoglobulin or from one of the many other blood products she received. It should be noted, however, that transmission of hepatitis C by immunoglobulin infusions is well documented and has potentially disastrous consequences.

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Intraocular foreign body missed by computed tomography

EDITOR,—Andrena M McElvanney and Alistair R Fielder emphasise the importance of thorough clinical examination when an intraocular foreign body is suspected, and the unreliability of plain radiography.[1] Bray and Griffiths have also ranked radiography second in importance to careful clinical examination.[2] We have examples of intraocular foreign bodies missed by radiography and inappropriate use of radiography after injuries with a negligible risk of penetration, but we report here a case in which even computed tomography failed to show an abnormality.

A 25 year old man working abroad was using a sledge hammer to repair a vehicle. He felt something hit his eye but continued working. Later he became aware of floaters and sought help. A metallic intraocular foreign body was diagnosed and removed with a magnet via the pars plana, and he was transferred back to Britain. Further examination showed two metallic fragments remaining within the mid vitreous gel and two minuscule fragments, possibly metallic, near the ora serrata. The visual acuity was normal. Computed tomography was arranged mainly to ascertain the number of metallic fragments and was done with 2 mm axial cuts. Surprisingly, no abnormality was apparent. Vitrectomy was performed and three intraocular foreign bodies removed. The largest measured about $1 \times 0{\cdot}5$ mm and proved to be ferromagnetic.

Possibly, the use of thin cuts on computed tomography and eye movement during scanning allowed the intraocular foreign bodies to go undetected. The conclusion, however, is that *x* ray examination and computed tomography do not always show a metallic foreign body. The lesson implied but not spelt out by McElvanney and Fielder is that accident and emergency staff suspecting an intraocular foreign body should refer patients for examination by an experienced ophthalmologist and must not reassure on the basis of radiography alone.

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Open up about stress

EDITOR,—Minerva reports the efforts of the mother of a young doctor who committed suicide to form a network of retired doctors to whom younger doctors could turn for support.[1] The pressures on doctors have been recognised for many years,[2,3] and the profession needs to turn away from the British attitude of keeping a stiff upper lip at times of stress and allow younger doctors to admit to their worries; this would be easier if senior colleagues were open about their own feelings.

Young doctors may be uncertain about approaching their own consultant because of a perceived need to be seen to be able to cope with pressure. If this is the case clinical tutors are a local source of independent advice and support for any doctors in training who need someone to talk to in confidence. The National Counselling Service for Sick Doctors (1 Park Square West, London NW1 4LJ; 071 935 5982) will also suggest the name of a senior doctor in the same discipline but from a different part of the country if any doctor wants confidential help.

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Asthma management guidelines

EDITOR,—P N Black[1] takes issue with the British Thoracic Society and others' recommendation that intravenous aminophylline should be used to treat life threatening asthma.[2] As justification for this belief Black notes that one study reported that adding intravenous aminophylline to frequently nebulised β agonists did not lead to faster improvement in objective measurements of lung function in patients with acute severe asthma. Black then speculates that although this study was not of patients with life threatening asthma, there is no reason why patients with life threatening asthma should differ from those with acute severe asthma. In fact, there is a good theoretical pathophysiological basis to suggest that patients with life threatening asthma should differ in their responsiveness to frequently nebulised β agonists and in their need for intravenous aminophylline from those with acute severe asthma.

In life threatening asthma (such as worsening respiratory failure or pre-respiratory arrest), bronchospasm and subsequent airflow limitation will be so severe as to preclude the delivery of adequate therapeutic doses of nebulised β agonist. If this was not so no patient with acute severe asthma being treated with frequently or continuously nebulised β agonist should ever develop life threatening asthma, which most physicians' experience will attest is patently not the case. In life threatening asthma, in which airflow limitation induced by bronchospasm is so severe as to prevent the inhalation of adequate doses of nebulised β agonist, the administration of an intravenous loading dose of 250 mg of aminophylline produces an almost immediate clinically detectable and objectively measurable bronchodilatation (that is, the peak flow increases from unrecordable to detectable). There is generally then enough airflow to permit the inhaled nebulised β agonist to reach the terminal airways in adequate doses to provide a therapeutic effect.

Junior medical staff who may have to manage patients with life threatening asthma should be aware of the British Thoracic Society's recommendations, adhere closely to them, and understand the likely pathophysiological processes underlying them. Failure to follow them may have grave consequences.

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Cimetidine and weight loss

EDITOR,—John Garrow's leading article puzzles over the results of two randomised controlled trials on cimetidine and weight loss in obese patients, one reporting a significant effect and the other not.[1] We should like to put forward some suggestions for this.

In the first trial Grethe Støa-Birketvedt reports that the group randomised to treatment with cimetidine lost on average 7·3 kg more than a control group receiving placebo over eight weeks.[2] As noted in the paper, the mean number of previous slimming attempts at baseline was six in the cimetidine group and 14 in the placebo group. Those who have previously made several unsuccessful attempts to lose weight may have been less likely to lose weight in a trial. Adjusting for baseline covariates such as number of previous slimming attempts and alcohol consumption in a regression model may result in a reduction in the difference in weight loss.[3] However, the difference in weight loss between the treatment and placebo group was large and so may not be entirely explained by this. The second trial, by Michael Højby Rasmussen and colleagues does not provide such data. In their study members of the placeo group and the cimetidine group lost almost 3 kg a month.

A second possible reason for the different findings is the starting weights. The average initial body mass index was about 28 kg/m^2 in the first trial compared to at least 33 kg/m^2 in the second trial. Both the placebo and treatment groups in this second trial may have been already losing appreciable amounts of weight with non-pharmacological measures alone and so may not have been helped by the addition of cimetidine.

Finally, and most importantly, the analysis in the second trial excluded four subjects in the cimetidine group and one from the placebo group because of non-compliance. In general, randomised trials should be analysed by intention to treat. Inclusion of the non-compliant subjects may have resulted in a significant difference between the placebo and treatment group if they had been losing even more weight than those remaining. This possibility is suggested by the sudden rise in weight seen between weeks 2 and 3 in the cimetidine group. If follow up data are available on