design features. Case matching is a more reliable means of eliminating errors than comparing the study group with a control group of a different size as in Hinton's survey.

The question of the season in which our data were collected is largely irrelevant but raises other issues. Patients attending the clinics in Cheltenham and Gloucester with suspected otitis media with effusion had waited four or five months for their first outpatient appointment and before their referral had had symptoms for several months. This time added to three months' follow up in an outpatient clinic and time on the waiting list for surgery brings the period of symptoms to roughly 12 months.

Some children have a seasonal deafness, which is usually worse in the winter or early spring. As these children's middle ear effusions resolve spontaneously the indication for surgery—or even for diagnosing otitis media with effusion—is less substantial.

Therein lies our criticism of the Edinburgh study of 892 schoolchildren aged 7, which found an association between salivary cotinine concentrations from passive smoking and tympanometric abnormalities.⁴ Tympanometry alone is insufficient for diagnosing otitis media with effusion; otoscopy and audiometry are required to increase the sensitivity and specificity for diagnosing the cause of childhood deafness. Furthermore, the Edinburgh children were examined only once. As over 80% of children have a middle ear effusion at some stage it is difficult to know whether these children were normal or had a pathological process.

There is no firm evidence to suggest that tobacco smoke, whether inhaled directly or by passive smoking, causes otitis media with effusion. If smoking does cause the condition an increase in otitis media with effusion in teenage girls might be expected as this group has recently increased its tobacco consumption. As most patients in our survey were from lower socioeconomic groups smoking would be more prevalent among their parents. To date the only firm evidence linking otitis media with effusion with passive smoking has been associative.

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- 1 Hinton AE. Passive smoking and otitis media with effusion. *BMJ* 1992;304:53. (4 January.)
- 2 Barr GS, Coatesworth AP. Passive smoking and otitis media with effusion. *BMJ* 1991;303:1032-3. (26 October.)
- 3 Hinton AE. Surgery for otitis media with effusion in children and its relationship to parental smoking. *J Laryngol Otol* 1989;103:559-61.
- 4 Strachan DP, Jarvis MJ, Feyerabend C. Passive smoking, salivary cotinine concentrations, and middle ear effusions in 7 year old children. *BMJ* 1989;298:1549-52.

Drug treatment for acute upper gastrointestinal bleeding

SIR,—In their editorial on drug treatment for acute upper gastrointestinal bleeding Colin Brown and W D W Rees make light of the evidence for benefit from tranexamic acid¹ on the grounds that in the largest controlled trial, in which 256 patients were randomised to receive tranexamic acid and 260 to receive placebo, the treatment groups were poorly matched in terms of important prognostic factors.² In fact, adjustment for these baseline differences made negligible difference to the overall outcome as mortality was consistently lower among patients receiving tranexamic acid within all the high risk subgroups.² In any case, dismissing the results of such a study on the basis of mismatching is usually based on a logical fallacy.

In a properly blinded randomised controlled trial baseline differences between treatment groups occur only by chance. Thus the *p* value obtained for the difference in outcome (<0.01 for the

fatality rates in this particular trial) must reflect the probability that the result was simply due to mismatching.

The most surprising feature of the trial of tranexamic acid, done in Nottingham, was the failure to detect any differences in rates of rebleeding and operation to account for the reduction in deaths.² This contrasts with the large trial of omeprazole reported by T K Daneshmend and colleagues, in which endoscopic signs of bleeding were reduced in patients receiving active treatment yet there was no reduction in death rates.³ In both trials roughly three quarters of deaths were associated with continued or recurrent bleeding.

Postoperative mortality in the trial of tranexamic acid was strikingly different between the two groups (four deaths out of 44 operations compared with 10 out of 35 in the placebo group), yet there was no difference in the timing of surgery. This suggests that fibrinolytic inhibition may have conferred benefit independent of any haemostatic effect. Recent analysis of the data from the large trials of fibrinolytic treatment for myocardial infarction (R Collins, Cardiovascular Symposium presentation) showed an early excess of deaths in patients receiving active treatment, mainly due to cardiac rupture. Thus it seems possible that fibrinolysis impairs (and thus tranexamic acid might improve) tissue healing.

This hypothesis is clearly speculative but deserves investigation. In particular, further large trials of tranexamic acid or similar drugs, possibly combined in a factorial design with other new treatments for acute upper gastrointestinal bleeding, are urgently needed. The only published statistical overview of such trials indicates a consistent reduction in mortality with tranexamic acid of around 40% (95% confidence interval 11 to 60%), an apparent benefit that is hard to ignore.⁴ Brown and Rees suggest that this meta-analysis was biased by the results of the Nottingham trial. Such statistical reviews are much more likely to suffer from the omission of unpublished studies (probably with negative results) than from the inclusion of large published trials. It seems that the narrative approach to reviews of published work is more prone to bias than the statistical.

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- 1 Brown C, Rees WDW. Drug treatment for acute upper gastrointestinal bleeding. *BMJ* 1992;304:135-6. (18 January.)
- 2 Barer D, Ogilvie A, Henry D, Cronfield M, Coggon D, French S, *et al.* Cimetidine and tranexamic acid in the treatment of acute upper gastrointestinal tract bleeding. *N Engl J Med* 1983;308:1571-5.
- 3 Daneshmend TK, Hawkey CJ, Langman M, Logan R, Long RG, Walt RP. Omeprazole versus placebo for acute upper gastrointestinal bleeding: randomised double blind controlled trial. *BMJ* 1992;304:143-7. (18 January.)
- 4 Henry DA, O'Connell DL. Effects of fibrinolytic inhibitors on mortality from upper gastrointestinal haemorrhage. *BMJ* 1989;298:1142-6.

Omeprazole for acute upper gastrointestinal bleeding

SIR,—T K Daneshmend and colleagues are to be congratulated on their remarkable feat of performing over three years the largest controlled trial ever of treatment for gastrointestinal haemorrhage and concluding that their "data do not justify the routine use of acid inhibiting drugs in the management of haematemesis and melaena."¹ Unfortunately, even this trial does not answer the question at issue—Would abolition of gastric acid improve mortality from gastrointestinal haemorrhage?—because neither the effects nor the side effects of a therapeutic intervention can be attributed to a drug alone: they depend on the

dose, defined as the amount needed to produce the required effect.

The gastric acid outputs or acidities of the authors' patients with bleeding were not measured, and it was assumed that these patients would behave in response to omeprazole as did their patients with inactive duodenal ulcers tested with their intravenous injection regimen,² which they claim produced "rapid and profound inhibition of acid secretion"—transmogrified in Colin Brown and W D W Rees's editorial to "virtually to abolish acid secretion."³ In their acidity studies, however,² although the pH rose above 4 in 90% of samples with this intravenous injection regimen of 80 mg, then three doses of 40 mg at eight hourly intervals, it was below 7 in almost every sample.

In our studies in healthy doctors the same eight hourly intravenous injection regimen gave a mean 24 hour pH of 7.05 but half the samples had a pH below 7.⁴ Even with our novel continuous 24 hour intravenous infusions of omeprazole, which resulted in a mean 24 hour pH significantly higher at 7.33, a quarter of the samples had a pH less than 7.

I still look forward to a trial of the abolition of gastric acid (pH above 7) in gastrointestinal bleeding, but that must wait until we have available a regimen (drug, route, frequency, and dose) that has been shown to produce sustained acidity in such patients.

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- 2 Walt RP, Reynolds JR, Langman MJS, Smart HL, Kitchingman G, Somerville KW, *et al.* Intravenous omeprazole rapidly raises intragastric pH. *Gut* 1985;26:902-6.
- 3 Brown C, Rees WDW. Drug treatment for acute upper gastrointestinal bleeding. *BMJ* 1992;304:135-6. (18 January.)
- 4 Sackier JM, Halliday K, Sprott P, Coelho LGV, Balten JJ, Li SK, *et al.* The effects of intravenous omeprazole on 24 h gastric secretion in healthy subjects. Comparisons of continuous and intermittent infusions: a pilot study. *European Journal of Gastroenterology and Hepatology* 1991;3:679-84.

Audit of gastrointestinal bleeding in a district general hospital

SIR,—T K Daneshmend and colleagues report their study of omeprazole in patients presenting with acute upper gastrointestinal bleeding.¹ In 1990 we established a protocol for managing patients admitted with gastrointestinal bleeding. We have now completed a retrospective audit of such patients over the six months from 1 January to 31 June 1991. We have evaluated how closely the protocol was adhered to, identified shortcomings in the management and protocol, and revised the protocol in the light of these observations.

There were 104 admissions for acute gastrointestinal bleeding, but only 58 case notes were available for assessment. The average age of the patients studied was 66 years, and 15 were over 80. Thirty nine patients underwent diagnostic endoscopy, the results of which were similar to those of a study by Madden and Griffiths.² Four patients underwent surgery, none of whom died. Altogether seven patients died: three had carcinoma, two had massive bleeding from a duodenal ulcer (one) and oesophageal varices (one), and two (aged 88 and 94) had been managed conservatively.

Evaluation of the protocol as applied to these patients led to several conclusions.

Firstly, the frequency of observations to be made was often not recorded in the notes or satisfactorily communicated to the nursing staff,

resulting in inadequate monitoring of 21 patients. Secondly, urine output was not monitored in 34 patients.

Thirdly, shocked patients (blood pressure <100 mmHg, postural drop in blood pressure, thirst, tachycardia) were identified but not always monitored as closely as they should have been. Of the eight shocked patients, four did not have a urinary catheter, six received colloid in the first hour, and five received blood in the first four hours.

Fourthly, blood transfusion was judged to be late (>24 hours after admission) or insufficient owing to underestimation of blood loss in nine patients.

Fifthly, surgical assessment on admission was delayed by more than six hours in 24 patients.

Finally, abnormal clotting was not corrected in two patients.

It is salutary that the deficiencies in the management of these patients were largely due to the failure to apply simple clinical and nursing measures rather than any deficiency of high technology medicine. Careful haemodynamic monitoring and fluid and blood replacement have always been the key to managing patients with gastrointestinal bleeding successfully. This simple audit serves to re-emphasise this.

On the basis of the deficiencies identified by the audit the protocol has been modified. The revised protocol will again be given to all junior staff concerned in managing patients with gastrointestinal bleeding and will be incorporated into a handbook of protocols for common medical emergencies being compiled for junior staff.

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- 2 Madden MV, Griffiths GH. Management of upper gastrointestinal bleeding in a district general hospital. *J R Coll Phys Lond* 1986;20:212-5.
- 3 Management of gastrointestinal bleeding. *BMJ* 1981;283:456-7.

Psychological aspects of lower urinary tract infections in women

SIR,—In their article on the psychological aspects of lower urinary tract infections in women David Sumners and colleagues make the point that assessment of clinical symptoms alone was not helpful in distinguishing between true urinary tract infection and the urethral syndrome in women presenting with dysuria and frequency.¹ They also suggest that antibiotics might be prescribed more appropriately if significant bacteriuria could be predicted before the result of culture was available. We agree with both these points but suggest that this would be the case if microscopic examination of the urine specimen was undertaken in the practice straight after the sample was passed.

Several studies have shown the value of early microscopic examination in diagnosing urinary tract infection in both general practice and hospital.^{2,3} Not only can pyuria be detected in fresh, unspun urine but recent work has shown that bacteriuria can be recognised and is more specific for diagnosing urinary tract infection than pyuria alone.⁴ Uninfected samples can be recognised and discarded, which saves laboratory expense, unnecessary antibiotics, and costly investigation.

Obtaining the equipment to perform urine microscopy need not be prohibitively expensive.

We have found that a mirrored counting chamber costing under £50 (Hawksley and Sons, Lancing, Sussex) gives good definition and allows bacteria to be recognised easily even with a cheap light microscope. Phase contrast microscopy gives optimum definition, and a relatively inexpensive model is now available—namely, the updated McArthur microscope (K W Kirk and Sons, Milton, Cambridge), which has been around as a light microscope since 1974. This has the advantage of measuring only 10×9×5 cm and being very robust. Coltman comprehensively documented its usefulness in general practice.²

Our experience has been that microscopic examination of urine is not a difficult skill to acquire, and the time saved in writing out laboratory forms and chasing up results more than compensates for the short time it takes to look at the specimen, in addition to the satisfaction of making an instant diagnosis for the patient.

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- 1 Sumners D, Kelsey M, Chait I. Psychological aspects of lower urinary tract infections in women. *BMJ* 1992;304:17-9. (4 January.)
- 2 Coltman KMD. A new technique for detecting urinary tract infection. *Practitioner* 1978;221:243-6.
- 3 Ditchburn RK, Ditchburn JS. A study of microscopical and chemical tests for the rapid diagnosis of urinary tract infections in general practice. *Br J Gen Pract* 1990;40:406-8.
- 4 Robins DG, White RHR, Rogers KB, Osman MS. Urine microscopy as an aid to detection of bacteriuria. *Lancet* 1975;i:476-8.
- 5 Vickers D, Ahmad T, Coulthard MG. Diagnosis of urinary tract infection in children: fresh urine microscopy or culture? *Lancet* 1991;338:767-70.

SIR,—In their study of the psychological aspects of lower urinary tract infections in women David Sumners and colleagues attempt to differentiate between urinary tract infection and the urethral syndrome by using “case notes, clinical symptoms, or psychiatric state.”¹ Their criteria for diagnosing the urethral syndrome, however, are unclear, and they seem not to have included infection with organisms such as *Neisseria gonorrhoeae*, *Chlamydia trachomatis*, and *Trichomonas vaginalis*.²

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- 2 Brumfitt W, Hamilton-Miller JMT, Gillespie WA. The mysterious “urethral syndrome.” *BMJ* 1991;303:1-2.

SIR,—David Sumners and colleagues' paper on the psychological aspects of lower urinary tract infections in women would have been more informative if they had paid greater attention to detail.¹ In their comparison of women with and without urinary tract infections presenting to general practitioners one of the two significant differences that they were able to show between the two groups was in the prevalence of nocturia. Regrettably, they did not define nocturia. It is generally accepted that voiding once in the night is acceptable but twice or more times is regarded as abnormal. The other significant difference was in the dysuria score between the two groups. The

authors state that the difference in the means yielded a *t* statistic of 1.998, which is significant. Calculation of this statistic from their presented means and standard deviations, however, results in *t*=1.8, which is not significant.

The authors have assumed that patients presenting with dysfunction of the lower urinary tract who do not have a urinary tract infection have the urethral syndrome. This is obviously incorrect as they have ignored the possibility of various other abnormalities of the urinary tract, such as detrusor instability. The authors do not define the urethral syndrome but state that its aetiology has been the subject of considerable debate.² This is hardly surprising as the syndrome is thought to occur in young women as well as in postmenopausal women, and the treatments suggested for these two groups are completely different.³ It would be better to drop the term the urethral syndrome as it is not clearly defined and does not help clarify the therapeutic options for a problem that is already difficult to manage.

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- 2 Brumfitt W, Hamilton-Miller HMT, Gillespie WA. The mysterious “urethral syndrome.” *BMJ* 1991;303:1-2.
- 3 Versi E, Cardozo LD. Oestrogens and lower urinary tract function. In: Studd JWW, Whitehead MI, eds. *The menopause*. Oxford: Blackwell Scientific, 1988:76-84.

SIR,—Unfortunately, the data in David Sumners and colleagues' study of the psychological aspects of lower urinary tract infections in women do not support their conclusion that “patients with the urethral syndrome are no more neurotic than those with significant bacteriuria.”¹ Once again, the reason is that there are too few patients in the study groups.

Wisely, O'Dowd *et al* did not claim a significant difference between the eight out of 40 patients with the urethral syndrome who had received tranquillisers and the one out of 46 patients with urinary tract infection who had received tranquillisers in their study.² Statistical tests on groups with such small numbers can be misleading. If 10% of patients with urinary tract infection had neurosis and 20% of patients with the urethral syndrome had neurosis 307 patients in each group would be needed to reach significance (95% level with power 80%) (Epi-Info computer program).

I am at present analysing the results of a study to evaluate prospectively a method of diagnosing urinary tract infection based on a bayesian probability score which was described in 1987.³ Seven patients had received a psychoactive drug in the previous two years out of 66 patients with urinary tract infection, as compared with eight out of 60 patients with the urethral syndrome. This study is also too small to decide whether there really is a difference in prescribing of psychoactive drugs between the groups.

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- 1 Sumners D, Kelsey M, Chait I. Psychological aspects of lower urinary tract infections in women. *BMJ* 1992;304:17-9. (4 January.)
- 2 O'Dowd TC, Smail JE, West RR. Clinical judgment in the diagnosis and management of frequency and dysuria in general practice. *BMJ* 1984;288:1347-9.
- 3 Dobbs FF, Fleming DM. A simple scoring system for evaluating symptoms, history and urine dipstick testing in diagnosis of urinary tract infection. *J R Coll Gen Pract* 1987;37:100-4.

AUTHORS' REPLY,—We are pleased with the interest generated by our paper. We did not intend our study to provide diagnostic criteria but to