respond to the first course of treatment or the presence of urological abnormalities. Short courses of antibiotics are significantly less effective in maintaining freedom from bacteriuria for the duration of pregnancy than is continuous antimicrobial prophylaxis for the remainder of gestation.³ The rates of subsequent pyelonephritis do not differ provided that short course treatment is followed by close surveillance to detect recurrent bacteriuria.^{3,4} This does not seem to have been the policy at Northwick Park, patients being followed up only until they gave their first clear urine sample two to four weeks after treatment.

Dr Campbell-Brown and coworkers regard an outlay of £6000 as excessive to prevent just six cases out of an expected total of 30. (It is not clear why the actual number of cases among abacteriuric women could not be stated; instead a figure of 19 was extrapolated from one case in 226 controls.) If the research techniques employed are excluded I estimate that less than £3000 was needed for dipslide screening, confirmation of bacteriuria in a second urine sample, and treatment. Longitudinal studies have shown that prevention of pyelonephritis may be achieved by paying attention to follow up without elaborate microbiological back up.^{5,6} Even the need for a confirmatory urine culture has been questioned on clinical grounds as well as because of expense.⁶

The credit side of the cost equation was largely ignored. Women presenting with severe abdominal pain and fever late in pregnancy often require admission to and investigation in hospital as well as intravenous treatment with antibiotics. The expense of looking after a premature infant also needs to be considered, quite apart from the human costs that Dr Campbell-Brown and colleagues themselves alluded to. The negative attitude to screening remains to be justified, even when the prevalence of bacteriuria is low and even if unavoidable failures of treatment occur. Finally, I wonder if screening has in fact been abandoned at Northwick Park?

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- 1 Kincaid-Smith P, Bullen M. Bacteriuria in pregnancy. *Lancet* 1965;ii:395-9.
- 2 Grüneberg RN, Leigh DA, Brumfitt W. Relationship of bacteriuria in pregnancy to acute pyelonephritis, prematurity, and fetal mortality. *Lancet* 1969;ii:1-8.
- 3 Whalley PJ, Cunningham FG. Short term versus continuous antimicrobial therapy for asymptomatic bacteriuria in pregnancy. *Obstet Gynecol* 1977;49:262-5.
- 4 Lenke RR, Van Dorsten JP, Schiffrin BS. Pyelonephritis in pregnancy: a prospective randomised trial to prevent recurrent disease evaluating suppressive therapy with nitrofurantoin and close surveillance. *Am J Obstet Gynecol* 1983;146:953-7.
- 5 Harris RE. The significance of eradication of bacteriuria during pregnancy. *Obstet Gynecol* 1979;53:71-3.
- 6 Whalley PJ. Management of urinary tract infection during pregnancy. In: Schröder FH, ed. *Recent advances in the treatment of urinary tract infections*. London: Royal Society of Medicine, 1985:37-43.

AUTHORS' REPLY.—We apologise for the fact that our methods section did not describe the follow up of the patients with proved bacteriuria who were treated. The first 51 produced a fresh midstream urine specimen at each visit to the antenatal clinic after they were found to be free of infection; the mean number of these specimens examined was 8.3. To save clinic time and work midstream urine samples were thereafter examined at 20, 28, and 34 weeks of pregnancy or when there was proteinuria or another indication for culture of the urine; the mean number of these specimens was 5.7. If any specimen contained more than 10⁷ colony forming units/l a specimen obtained by suprapubic aspiration or catheter was examined.

We are also sorry that the diagram was not more

informative. The five mothers who developed pyelonephritis had all been examined by suprapubic aspiration and found to have truly infected urine. That the incidence of pyelonephritis in our treated patients was greater than that in other series is readily explained. In the other series the diagnosis was made from a midstream urine sample. As Dr Smith notes, such samples have an appreciable false positive rate, and thus a lower rate of pyelonephritis would be expected as many of those treated would never have been infected. Dr Smith also inquires about the four patients who were treated but later developed overt urinary tract infection. Two of these were cleared by the initial treatment but were again infected by *Escherichia coli* four and 16 weeks later; the two others were also infected by *E. coli*, but this was not cleared from the urine by antibiotics. The suggestion that urological abnormalities might have been investigated is reasonable, but we did not find this to be valuable, even in patients with recurrent infections, in a previous investigation.¹ The expected total number of cases of pyelonephritis was extrapolated from the 226 control (culture negative) patients because their obstetric notes were scrutinised by two of us and we were certain that 225 had not developed pyelonephritis.

We were interested to read that Dr M E Foley and coworkers (25 July, p 270) found seven cases of pyelonephritis in 100 treated mothers and eight in 120 patients with bacteriuria who had not been treated initially. This rate of 6.7% is even lower than our own of 12.5%, which was similar to that found in Aberdeen, where 11.8% of untreated patients with bacteriuria, shown by midstream urine samples, developed pyelonephritis.² All three centres found a lower incidence than the 33% expected,¹ but, while the clinical course may not have changed, the criteria for diagnosing pyelonephritis may be now more stringent. There might also be an ethnic effect, although it seems just as likely that the condition is related to social class.

We evaluated our data on the efficacy of screening for bacteriuria in pregnancy as well as the cost of routine clinics and bacteriology and have stopped routine screening at Northwick Park. Though symptomatic urinary tract infection is four to six times more common in patients with bacteriuria in Dublin, Aberdeen, and Harrow than in those without bacteriuria, screening and antibiotic treatment to prevent a few cases (four per year in our population) needs to be justified. When more cases of symptomatic infection occur overall in women who yield negative results on screening at booking than in those who yield positive results such a policy is unnecessary unless there is a high local incidence of confirmed bacteriuria or of its complications.

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- 1 McFadyen IR, Eykin SJ, Gardener NHN, et al. Bacteriuria in pregnancy. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1973;80:385-405.
- 2 Chng PK, Hall MH. Antenatal prediction of urinary tract infection. *Br J Obstet Gynaecol* 1982;89:8-11.

Influence of non-steroidal anti-inflammatory drugs in faecal occult blood tests

SIR.—Mr G Pye and colleagues suggests that a finding of occult faecal blood cannot be attributed to upper gastrointestinal tract bleeding caused by non-steroidal anti-inflammatory drugs (13 June, p 1510). Their finding contradicted what is commonly believed—that these drugs can produce occult blood in the absence of gastrointestinal disease.¹⁻³

We have just finished a study which confirms the conventional view, in contrast to that of Mr Pye and others and indeed our own previous opinion.⁴⁻⁸ In a colorectal cancer screening programme in upper Galilee, north Israel, 1797 participants aged over 40 performed the Haemoccult II test for faecal occult blood. Those screened were supposed to stop taking non-steroidal anti-inflammatory drugs while performing the test for occult blood, but when directly investigated 26 admitted not stopping the medication. The prevalence of drug treatment was 8.9 times higher among participants with a positive Haemoccult result than among those with a negative test result ($Z=6.108$, $p<0.0001$). Seven of the 26 who admitted taking non-steroidal anti-inflammatory drugs (27%) had positive results compared with 64 out of 1771 people who did not take these drugs (4%) ($Z=6.297$, $p<0.0001$).

Our results imply that we should follow Simon's advice¹ that those who give positive results on a test for occult faecal blood and are taking non-steroidal anti-inflammatory drugs should have the test repeated before colonic investigations are undertaken.

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- 1 Simon JB. Occult blood screening for colorectal carcinoma: a critical review. *Gastroenterology* 1985;88:820-37.
- 2 Rees WD, Turney LA. Reappraisal of the effect of aspirin on the stomach. *Lancet* 1980;ii:410-3.
- 3 Grossman MI, Matsumoto KK, Lichter RI. Faecal blood loss produced by oral and intravenous administration of various salicylates. *Gastroenterology* 1961;40:383-8.
- 4 Doran J, Hardcastle JD. Bleeding patterns in colorectal cancer: the effect of aspirin and the implication for faecal occult blood testing. *Br J Surg* 1982;69:711-3.
- 5 Doubilet P, Donowitz M, Panker SG. Evaluation for colon cancer in patients with occult faecal blood loss while taking aspirin: a Bayesian viewpoint. *Medical Decision Making* 1982;2:147-60.
- 6 Bahrt KM, Korman LY, Nashel DJ. Significance of a positive test for occult blood in stool of patients taking anti-inflammatory drugs. *Arch Intern Med* 1984;144:2165-6.
- 7 Norfleet RG. 1300 mg of aspirin daily does not cause positive faecal Hemoccult tests. *J Clin Gastroenterol* 1983;5:123-5.
- 8 Niv Y. Occult blood in colorectal cancer. *Gastroenterology* 1985;89:705-6.

Right atrial thrombus: a complication of total parenteral nutrition

SIR.—Like the patient described by Dr H Chamsi-Pasha and Professor Miles Irving (1 August, p 308), one of my patients also developed a right atrial thrombus as a complication of parenteral nutrition.

A 19 year old man with Crohn's disease was referred for home parenteral nutrition in 1983 because of intestinal failure and growth retardation. Treatment progressed uneventfully for four years, during which he gained 17 cm in height and was able to take up employment. Six months ago he was admitted with subacute intestinal obstruction, when he was noted to have developed a pansystolic tricuspid murmur. There was no evidence of infection or pulmonary embolism. A chest radiograph showed calcification in the region of the right atrium, and an echocardiogram showed a right atrial mass that was prolapsing into the ventricle. A thrombus was subsequently removed.

Thrombosis is probably the most important complication of prolonged central parenteral nutrition. In our experience, central vein thrombosis is associated with the use of high concentration dextrose solutions¹ (rather than three in one lipid mixes, which tend to block feeding catheters²) and occurs when the catheter tip lies in the superior vena cava rather than in the right atrium.³ The recently described development of atrial thrombi emphasises the need for research into thrombus