CORRESPONDENCE

The funding of in vitro fertilisation in NHS hospitals J Parsons, MRCOG, and Virginia Bolton; A F Riddle, MRCOG, and others.....	1064	Self referral to consultants R Cocks, FRCSed; J Dunn, MRCPsych.....	1067	Physical exercise and mental health R Ramsay, MB, and R Farmer, MRCPsych	1069
A blot on the profession Frances Lefford, MB, and Vivienne Van Someren, MRCP.....	1065	Postviral fatigue syndrome A V M Hughson, MRCPsych; G A Jamal, MD, and S Hansen, PhD; B D Calder, MRCP, and P J Warnock, MRCP; J V Dadswell, FRCPATH.....	1067	Peaks and troughs in demands on hospitals W Kirkup, MFCM	1070
The pathogenesis of acute pancreatitis A J Goodman, FRCS, and others; E W Benbow, MRCPATH; R E Barry, FRCP.....	1065	Percutaneous cholecystolithotomy A G Johnson, FRCS, and others; J E A Wickham, FRCS, and others	1068	Prenatal screening G F Rowland, MRCOG	1070
Should the pill be stopped preoperatively? R J Farrell, FRCS, and Jennifer Lamb, PhD; H M Sue-Ling, FRCS, and L E Hughes, FRCS	1066	Nifedipine for epilepsy? P Wen, MRCP.....	1069	Drug points Erythromycin and nightmares (R J Black and T A J Dawson); Nifedipine for epilepsy? (J W A S Sander and S D Shorvon); Neuroleptic malignant syndrome (R D Mann); Ventricular arrhythmia precipitated by flumazenil (T G Short and others)	1070
Effect of stanozolol on itching in primary biliary cirrhosis Carol A Seymour, MRCP, and C B Summer-ton, MRCS; R P Walt, MRCP.....	1066	Hypnotic drug use among the elderly M K Thompson, FRCP.....	1069	Psychiatric symptoms associated with carbamazepine (G Mathew); Benzylamine oral rinse and rash (Margaret Turner and R Laith); Severe toxic erythema caused by diltiazem (R A Wakeel and others)	1071
		Procedures in dermatology G Ashall, FRCS, and A L H Moss, FRACS	1069		

- All letters must be typed with double spacing and signed by all authors.
- No letter should be more than 400 words.
- For letters on scientific subjects we normally reserve our correspondence columns for those relating to issues discussed recently (within six weeks) in the *BMJ*.
- We do not routinely acknowledge letters. Please send a stamped addressed envelope if you would like an acknowledgment.
- Because we receive many more letters than we can publish we may shorten those we do print, particularly when we receive several on the same subject.

The funding of in vitro fertilisation in NHS hospitals

SIR,—The title of the paper by Dr Peter Bromwich and others on in vitro fertilisation (12 March, p 759) is misleading. The paper describes *private* in vitro fertilisation in a small unit in the NHS, and to omit the word "private" from the title is to invite misinterpretation.

Payment for treatment, whether £1400 or £400, whether in a private hospital or in a "small NHS unit," whether for profit or merely to cover costs, means it cannot be deemed to be anything but private treatment. For the Oxford team to accept this fact without comment is a dangerous step, condoning policies of the present government that undermine the ethos of the National Health Service. Rather than accept that treatment such as assisted conception should be available only to those who can afford it, it is essential to campaign for more funding for infertility therapy.

Statistics from in vitro fertilisation units throughout the world show that fewer than one in 10 in vitro fertilisation cycles are successful, with many couples having multiple attempts before succeeding. Thus even for patients treated at Oxford, where the charge is less than half that of any private clinic, successful treatment may remain out of reach.

At King's College Hospital, where the assisted conception unit is also housed in an NHS building, self funding is achieved in a different manner. Twenty couples are treated each week, five of them privately. Private patients are those patients who either choose not to wait for one to two years on the

non-paying waiting list or are not eligible for the list because they have been referred for in vitro fertilisation from outside King's College Hospital and do not live within Camberwell Health District. They pay £1000 per completed cycle, and the revenue covers staff salaries. The non-paying patients are encouraged to join the King's IVF support group, which is run entirely by patients (both paying and non-paying). The support group, which is a registered charity, raises enough money to cover all the additional costs of the assisted conception unit (about £50 000 a year). Not all patients join the support group or raise funds for it if they do, and we have no way of knowing which of our patients are members.

After admission as a non-paying patient treatment is continued for as long as necessary. The only limitations to continued treatment are physiological.

Raising money from our patients is an unfortunate necessity. We do not support the economic philosophy under which we are forced to operate and feel that it is the duty of doctors in our position to fight against perpetuation of the current situation, rather than to report without criticism on the introduction of private medicine into NHS hospitals.

JOHN PARSONS
VIRGINIA BOLTON

Department of Obstetrics and Gynaecology,
King's College School of Medicine and Dentistry,
London SE5 9PJ

SIR,—Mr David Barlow and his colleagues report a significantly better outcome for in vitro fertilisation using transvaginal compared with transvesical ultrasound for oocyte recovery (12 March, p 751). However, they base this conclusion on a consecutive series of 100 patients—50 transvesical procedures followed by 50 transvaginal collections. We question the validity of this comparison, where allowance has not been made for operator experience.

In a fairly new in vitro fertilisation unit with their stated experience of just 250 treatment cycles one would expect success rates to improve over the next 100 cases and five months of their study. In our practice (now totalling over 3400 ultrasound directed oocyte collections) we observed such an effect of experience¹ and see a similar trend as each new member of staff is trained.

With a well designed ultrasound probe and biopsy guide vaginal oocyte recovery is a technique more easily learnt than transabdominal collection. Nevertheless, it is advantageous for operators to master all techniques of ultrasound directed oocyte recovery and to be adaptable in assessing the best approach for the individual patient. We reviewed 739 oocyte collections in an unselected series of patients treated between April 1987 and January 1988 (table). The three groups of patients were comparable in age, diagnosis, drug regimen, and response to stimulation. We found no difference in outcome between transvesical and transvaginal procedures.

Results of transvesical and transvaginal recovery of oocytes in 739 patients

	Trans- vesical	Trans- vaginal	Combined
No of oocyte retrievals	601	54	84
Mean No of oocytes retrieved	5.9	7.1	6.8
No of embryo transfers	493	48	71
Mean No of embryos transferred	2.8	2.9	3.1
Clinical pregnancies	119	12	19
Rate per embryo transfer (%)	24	25	27

Technically, some ovaries will be accessible only by the vaginal route, but the converse also applies. Other factors may also be important: some patients experience extreme discomfort from a full bladder and prefer vaginal collection; others may have psychological or cultural objections to this method. We believe that flexibility in approach to oocyte recovery offers the best outcome.

A F RIDDLE
M C DAVIES
J S PAMPIGLIONE
B A MASON

Hallam Medical Centre,
London W1N 5LR

1 Riddle AF, Sharma V, Mason BA, *et al*. Two years' experience of ultrasound-directed oocyte retrieval. *Fertil Steril* 1987;48: 454-7.

A blot on the profession

SIR,—We congratulate Drs Stella Lowry and Gordon Macpherson on their recent leading article (5 March, p 657) challenging the social and cultural attitudes of doctors which lead to the sexist and racist behaviour so frequently observed among colleagues. We endorse the statement that it is necessary for discrimination to be "seen to be swept out" of the profession but looked in vain for proposals on the means to that end.

We agree that identifying discriminatory practices alone is not enough, but it is a vital first stage in tackling the problem. The difficulty lies in obtaining the unequivocal evidence with which to invoke the threat of legal action. It is unlikely that any institution in future will commit its prejudices to open scrutiny by incorporating them in a computer program. The Commission for Racial Equality had, however, been alerted to the issue of unfair student selection in the London medical schools before this by research reports which analysed the student population by gender and race.

The reasons for discriminatory practices in medicine are obvious enough; they will increase with increased competition for jobs which provide training and good career prospects. If the editorial views of the *BMJ* reflect those of the British Medical Association then we feel the association should act to remedy the situation it deplors. To begin with, we suggest the BMA should ask the Department of Health and Social Security to arrange the routine collection of the number and gender of all applicants for NHS appointments at each stage of the selection procedure, including the original applications. Monitoring appointments in this way, as recommended by the Commission for Racial Equality,¹ would substantiate—or negate—the claim of many women doctors that gender discrimination blocks their appointment to senior posts, particularly in hospital medicine. Such information would pave the way for remedial action. In this way the BMA would prove its credibility to women, who form a significant proportion of the 70% of practising doctors it represents.

Events at St George's show that the unease felt

by knowledgeable individuals within the institution does not lead to it changing its discriminatory practices. The intervention of an outside authority was necessary to force the institution to take remedial action. The BMA would gain the respect of its membership and the larger community—our patients and the many non-medical women and ethnic minority workers in the NHS—if it is seen to take the initiative in sweeping the profession clean of unfair discrimination. If the medico-political will is there it will happen; and solutions will be found to the underlying problems, inherent in the career structure, which are evoked to justify discrimination.

FRANCES LEFFORD
VIVIENNE VAN SOMEREN

London Association of the Medical
Women's Federation,
London NW5 1UE

1 Commission for Racial Equality. *Medical school admissions: report of a formal investigation into St George's Hospital Medical School*. London: Commission for Racial Equality, 1988.

The pathogenesis of acute pancreatitis

SIR,—We were disappointed by Dr R E Barry's leading article (27 February, p 589) and wish to take issue with a number of points he raised, in particular his critique of the infrequent role of gall stone impaction as a causative agent in acute pancreatitis.

It has long been known that a common biliary-pancreatic channel exists in 80% of normal subjects^{1,2} and in a similar proportion of patients with gall stone associated acute pancreatitis.³ These patients with pancreatitis have smaller more numerous gall stones, wider cystic ducts, longer common channels, and more frequent pancreatic duct reflux during cholangiography.^{4,6} These features predispose to acute pancreatitis by encouraging the passage of gall stones with transient ampullary obstruction. The author quotes Shader, implying that ampullary stones are rarely associated with acute pancreatitis; however, this study was a postmortem review conducted after the acute attack. Acosta *et al*⁷ and Stone *et al*⁸ showed impacted ampullary gall stones in 63-75% of patients with biliary pancreatitis when the bile ducts were inspected during the attack.

Dr Barry quotes White as the source of the incidence of common bile duct gall stones in acute pancreatitis (4% of 2653); however, we cannot find this in the text of the paper and assume it to be a typographical error.

Dr Barry implies that intraparenchymal activation of pancreatic proenzyme is unlikely to result in haemorrhagic pancreatitis. However, pancreatic enzymes are capable of destroying vascular structures, resulting in haemorrhagic necrosis.⁹

Dr Barry has provided us with an unbalanced insight into the possible pathogenesis of this disease. He has in the main ignored the wealth of research carried out in the past 20-30 years, choosing to concentrate his review on dated reference material and the personal and highly theoretical work of Keynes.

A J GOODMAN
N C BIRD
D D KERRIGAN
A G JOHNSON

Department of Surgery,
University of Sheffield,
Royal Hallamshire Hospital,
Sheffield S10 2JF

1 Ivy AC, Gibbs GE. Pancreatitis: a review. *Surgery* 1952;31:614-2.
2 Hand BH. Anatomical study of the choledochoduodenal area. *Br J Surg* 1963;50:486-9.
3 Kelly TR. Gallstone pancreatitis: pathophysiology. *Surgery* 1976;80:488-92.

4 McMahon MJ, Shepta JR. Physical characteristics of gallstones and the calibre of the cystic duct in patients with acute pancreatitis. *Br J Surg* 1980;67:6-9.
5 Kelly TR. Gallstone pancreatitis: local predisposing factors. *Ann Surg* 1984;200:479-85.
6 Armstrong CP, Taylor TV, Jeacock J, Lucas S. The biliary tract in patients with acute gallstone pancreatitis. *Br J Surg* 1985;72: 551-5.
7 Acosta JM, Pellegrini CA, Skinner DB. Etiology and pathogenesis of acute biliary pancreatitis. *Surgery* 1980;88:118-23.
8 Stone HH, Fabian TC, Dunlop WE. Gallstone pancreatitis. Biliary tract pathology in relation to time of operation. *Ann Surg* 1981;194:305-10.
9 Steer ML, Meldolesi J, Figarella C. Pancreatitis: the role of lysosomes. *Dig Dis Sci* 1984;29:934-8.

SIR,—A wide range of drugs have been associated with acute pancreatitis. With the exceptions of azathioprine and the thiazides, Dr R E Barry briskly dismisses the potential pathogenic importance of all of them because most of the patients who take each one do not get acute pancreatitis. This is a seductive way of thinking which, if generalised to other drugs and other complications, would eradicate most of our concerns about drugs and their side effects.

I recently found acute pancreatitis in a patient who had been taking methyldopa,¹ though, since the diagnosis was made only at necropsy, the association might have been "coincidence rather than causality," as Dr Barry puts it. Likewise, transient hyperamylasaemia occurring during treatment with methyldopa² might simply be coincidence. Nevertheless, published studies contain more convincing evidence of causality. In three cases in which acute pancreatitis followed treatment with methyldopa rechallenge with the drug promptly produced a new episode of illness, together with hyperamylasaemia and hyperlipasaemia.^{3,4} These observations, together with the rapid return to clinical and biochemical normality which occurred in each case when the drug was discontinued, strongly suggest that methyldopa should not be dismissed from the list of drugs that really cause acute pancreatitis.

E W BENBOW

Department of Pathology,
University of Manchester,
Manchester M13 9PT

1 Benbow EW. Simultaneous acute inflammation in entopic and ectopic pancreas. *J Clin Pathol* (in press).
2 Warren SE, Mitas J, Swerdlin AHR. Pancreatitis due to methyldopa: case report. *Milit Med* 1980;145:399-400.
3 Rominger JM, Gutierrez JG, Curtis D, Chey WY. Methyldopa-induced pancreatitis. *Am J Dig Dis* 1978;23:756-8.
4 Heide H van der, Haaf MA ten, Stricker BHC. Pancreatitis caused by methyldopa. *Br Med J* 1981;282:1930-1.

AUTHOR'S REPLY,—My leading article was certainly an unbalanced presentation of some aspects of the pathogenesis of pancreatitis. But my brief was to comment on the recently published ideas of Keynes,¹ who has suggested a pathogenesis for severe acute pancreatitis which is at variance with standard teaching, and to do so the non-specialist should appreciate that other data do exist which do not conform to the "party line." Unfortunately, the 500 word format did not permit (and neither does this reply) a detailed and balanced review of the published studies on a contentious issue of long standing which is overburdened with inconsistencies.

The role of gall stones and biliary tract disease in the aetiology of acute pancreatitis is surely beyond doubt. Indeed, there is increasing evidence that most attacks of acute pancreatitis are associated with gall stone migration.² But this is not the issue. Keynes's ideas concern the mechanism by which gall stones may cause pancreatitis—that is, the pathogenesis. Do the gall stones cause pancreatitis