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.

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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.

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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.

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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.

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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