four patients with liver cirrhosis, in which a renal impairment is often present. After administration of osmotic agents (mannitol, dextran)⁷ or radiological contrast media⁸ the increase of urinary enzyme and protein excretion is more evident in patients with chronic renal diseases than in subjects with normal renal function.

We conclude that the urinary excretion of α -glucosidase and probably of other renal glucosidases is not influenced in normal subjects by the induction of a diuresis and may be considered a reliable and sensitive index of tubular damage after drug administration. In patients with underlying liver or renal diseases, however, the possible administration of diuretic agents must be taken into account before drawing any conclusion. One must be cautious in normal subjects too when determining enzyme or protein indexes of tubular proteinuria such as lysozyme and β_2 -microglobulin. Conversely, the determination of tubular enzyme excretion after induction of a diuresis (chiefly after an osmotic load) might be used as a sensitive test of underlying renal diseases, as suggested also by Burchardt *et al.*⁷

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⁶ Guarnieri, G F, *et al*, *Enzymes in Health and Disease*, p 193. Basel, Karger, 1978.

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Vaginal microbial flora in normal young women

SIR,—I was interested to read the careful study of vaginal microbial flora in normal young women in Edinburgh (2 June, p 450) but feel that it may not fully reflect the situation in all parts of Britain.

In particular, I feel that *Trichomonas vaginalis* is, at least in Cleveland, a more frequent pathogen than the Edinburgh study suggests. Analysis of the findings in 1000 cervical smears received consecutively by this laboratory in January and February this year from family planning clinics shows that *Trichomonas vaginalis* was positively identified (by at least two observers) in 70 (7%). In addition, cytological features suggesting trichomonas infestation were present in a further 61 in which the parasite could not be identified with certainty. Inflammatory changes were present in all but three of those 131 smears and vaginal discharge was recorded on the accompanying request form in 107 (82%). By contrast, *Candida* organisms were identified in only 24 of the 1000 smears, though cytological features suggestive of candidiasis were seen in a further 12. Comparison with a similar study made in 1969 showed that the findings are relatively constant in this area.

While I admit that a cervical smear is an inferior technique to culture in demonstrating vaginal candidiasis, there is no doubt that examination of a smear is an effective way to demonstrate trichomonas infestation and that

this pathogen is a major cause of vaginal discharge. Though there clearly may be differences in selecting the patients studied here and in Edinburgh, it is difficult to account for the different frequency of *Trichomonas vaginalis* in cervical smears in Cleveland (at least 7.0%) and Edinburgh (1.2%) except by postulating a difference in prevalence. It would be of interest to know the position in other parts of Britain.

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Uterine rupture after intra-amniotic injection of prostaglandin E₂

SIR,—We wish to report a case of uterine rupture following intra-amniotic injection of prostaglandin E₂ and hypertonic saline in order to induce abortion.

The patient, aged 39 years, had had three previous vaginal deliveries and requested abortion at the 20th week of pregnancy. This was induced by an eventful transabdominal amniotic injection of 5 mg prostaglandin E₂ and 100 ml of 30% hypertonic saline following the removal of 150 ml of clear liquor. Contractions commenced after four hours but after 11 hours the patient developed continuous lower abdominal pain and a pulse rate of 120 beats per minute. Abdominal examination revealed a tender suprapubic mass, equivalent to a 16-week gestation and a separate firm mass, 8 cm in diameter, in the right hypochondrium. A diagnosis of ruptured uterus was made and at laparotomy there was a rupture at the junction of the upper and lower segments involving the anterior wall and both lateral walls of the uterus. The intact gestation sac was bulging through the deficit. Total abdominal hysterectomy was performed and the postoperative course was uneventful. The cervix was normal.

This is the first reported case of uterine rupture following mid-trimester termination of pregnancy with prostaglandin E₂ and hypertonic saline, although one case has been reported following intra-amniotic injection of prostaglandin F₂ and hypertonic saline.¹ Cervicovaginal fistula, however, has been reported following 1% of terminations induced using intra-amniotic prostaglandin.² We report this case in order to draw attention to an unusual complication which must be taken into account when choosing a method of terminating a pregnancy in the mid-trimester.

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An open-access morbid pathology facility for GPs?

SIR,—Family doctors have often received a rap over the knuckles for inaccurate certification of death. We are as interested as are any of our colleagues in knowing why patients die, but we are often prevented from finding out.

An elderly patient dies at home after an illness in which investigation by open-access pathology requests, consultant domiciliary consultation, or a period of inpatients or out-patient surveillance has produced a variety of possible pathological diagnoses. Age, frailty, and humane considerations have precluded

further probing. The obvious need, then, is for a postmortem examination. The only way that this can be procured is by reference to the coroner and then only at his discretion. This cumbersome procedure is quite inappropriate where there is no question of foul play, where the final mode of death (but not the antecedent condition) is obvious, and where a visit from a police officer, however kindly, is an ordeal for the relatives. How many doctors can honestly say that they have not certified deaths as being due to, for example, bronchopneumonia, while knowing that a deeper cause for that has not been uncovered?

There is a clear need for an open-access morbid pathology facility for general practitioners. We should all be humbled, but enlightened, by this; and our patients would stand to gain.

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Enterotoxigenic *Escherichia coli* and travellers' diarrhoea

SIR,—Mr R J Gross and others (2 June, p 1463) reported that they isolated enterotoxigenic *Escherichia coli* (ETEC) from the faeces of 6/55 (11%) patients who developed diarrhoea during or shortly after travel abroad, and also from 1/50 (2%) patients with diarrhoea but no history of recent travel outside the UK. We have been conducting similar studies in Manchester and wish to report our confirmatory results.

We tested the toxigenicity of five colonies of *Escherichia coli* isolated from the faeces of each of 13 cases of diarrhoea in travellers returning to the Manchester area from abroad, and also in a control group of 13 patients who developed diarrhoea at about the same time but gave no history of recent travel. A group of 13 babies with diarrhoea admitted to Wythenshawe Hospital, South Manchester, were also included in this survey. In an earlier study we examined 210 strains of "specific" *E coli* isolated from cases of infantile diarrhoea by various hospital laboratories in the north-west. Heat-labile enterotoxin was detected by the Chinese hamster ovary cell assay¹ and heat-stable enterotoxin by the infant mouse gut test.² All toxigenic strains isolated were tested serologically using *E coli* O-agglutinating antisera (Wellcome) and then sent to Dr B Rowe of the Central Public Health Laboratory, Colindale, London, for further serotyping.

Our results show that 6/13 (46%) of travellers with diarrhoea were infected with ETEC, each of a different serotype. None of the control group yielded ETEC. All 13 infants from Wythenshawe Hospital were clear of ETEC. Furthermore, all the strains (210) of specific *E coli* from cases of infantile diarrhoea in the north-west were non-toxicogenic.

While confirming the conclusions drawn by Mr Gross and his colleagues, our results show a significantly higher isolation rate of ETEC from travellers with diarrhoea than their results. We note, however, that, whereas they examined only one or sometimes two "cultures" (*sic*) from each patient, we have been routinely screening five colonies of *E coli* from each patient. We feel that this may account for our higher isolation rate. We also find that in the north-west of England ETEC do not appear to be a significant cause of sporadic diarrhoea in infants or adults; this is in agreement with the results of Gross *et al* and other workers in the United States³ and Sweden.⁴

Finally, it is necessary to report that not all the toxigenic strains of *E coli* we isolated from