... contrary to popular belief

EDITOR,—P C Goldsmith and colleagues point out that schistosomiasis mimicking vulval warts may present in genitourinary medicine clinics.[1] We report a similar case.

A 32 year old woman presented last April with a seven week history of an itchy vulval lump. On examination she had a nodular lesion with a papillomatous surface about 1 cm in diameter on the right labium minus. This did not whiten when 5% acetic acid was applied. Histological examination of a biopsy specimen showed an acanthotic squamous epithelium with an active chronic inflammatory infiltrate including many eosinophils. Schistosomes were present, some having recognisable terminal spines characteristic of *Schistosma haematobium*. On direct questioning she said that she had travelled extensively in Africa in early 1992 and had swum in Lake Malawi in April and July 1992. Further investigations showed a normal full blood count, and examination of the urine and faeces for ova yielded negative results. Excision biopsy of the remainder of the lesion was carried out, and she was given praziquantel 40 mg/kg orally as a single dose. Follow up one month after treatment showed that the lesion had healed.

Local advice to tourists in Malawi is that Lake Malawi, where two of Goldsmith and colleagues' patients had also swum, is free of schistosomes. We contacted the Malawi High Commission and the tourism officer, who confirmed their belief that Lake Malawi is the only lake in Africa that is free of schistosomes. These cases suggest that this is not correct.

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1 Goldsmith PC, Leslie TA, Sams V, Brycesan ADB, Allason-Jones E, Dowd PM. Lesions of schistosomiasis mimicking warts on the vulva. *BMJ* 1993;307:556-7. (28 August.)

Epilepsy and pregnancy

Emphasise the importance of extra folate

EDITOR,—M D O'Brien and S Gilmour-White give a physician's view of epilepsy in pregnancy, but there are too many important obstetric inaccuracies and omissions.[1] Their advice on contraception is misleading. The use of an oral contraceptive containing 50 μg ethinyloestradiol is important in women taking enzyme inducing antiepileptic drugs, but one Mercilon pill (20 μg) plus one Marvelon pill (30 μg) is more appropriate than Ovran because of the more lipid favourable progestogen (desogestrel) that Mercilon and Marvelon contain; this also allows greater flexibility in dose. Some years ago the recommendations on when to start the contraceptive pill (other than after emergency contraception) were changed from day 5 of the cycle to day 1 to ensure immediate contraception and to improve compliance. It is therefore misleading to refer to start times other than day 1.

The advice to take a small folate supplement twice a week or a diet rich in folate is insufficient. All women planning a pregnancy should take a diet rich in folate and folate supplements of 400 μg for three months before conception until 12 weeks' gestation.[2] This is particularly important for women taking enzyme inducing antiepileptics, whose fetuses are at increased risk of neural tube defect, and there is a strong argument for the women to take the 5 mg supplement recommended for women with a previously affected child. This should be explained to all epileptic women yet is overlooked in the article.

Although serum α fetoprotein concentration was extensively used in the past as a marker for neural tube defect (and at 16 rather than 18 weeks), it has been almost totally replaced by ultrasound scanning. These women should have detailed scans by a sonographer aware of the situation, in early second trimester to exclude anencephaly and at 20 weeks to exclude spina bifida. The second scan also allows cleft lip and palate to be identified.

Most first fits in pregnancy beyond 20 weeks' gestation, particularly those occurring in the absence of any localising features or obvious precipitating factors, will be eclamptic, and the urgent opinion of an obstetrician is as valuable as that of a neurologist. We have seen fits clearly attributable to pre-eclampsia which have preceded the appearance of proteinuria: the diagnosis is not excluded by the absence of proteinuria. Other markers such as raised liver enzyme activities and creatinine concentration, hyperuricaemia, abnormal clotting, or intrauterine growth retardation should be sought.

These points emphasise the importance of combined medical and obstetric care for women with epilepsy.

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1 O'Brien MD, Gilmour-White S. Epilepsy and pregnancy. *BMJ* 1993;307:492-5. (21 August.)
2 Department of Health. *Folic acid and the prevention of neural tube defects.* London: DoH, 1992.

Wrong advice on postpartum contraception

EDITOR,—In their article on epilepsy in pregnancy, M D O'Brien and S Gilmour-White state that if a woman is not breast feeding either the combined pill or the progestogen only pill can be used from four weeks postpartum and that contraception before this is unnecessary.[1] Most patients who do not breast feed have a period at about six weeks. This means that they ovulated two weeks before, and if no contraception is used pregnancy may ensue. Some patients, however, have a period before six weeks. I hope that the authors are the only people giving this wrong advice.

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1 O'Brien MD, Gilmour-White S. Epilepsy and pregnancy. *BMJ* 1993;307:492-5. (21 August.)

Microalbuminuria in chronic obstructive lung disease

Consider coexistent disease

EDITOR,—R Wilkinson and colleagues claim that microalbuminuria is a feature of chronic obstructive lung disease,[1] but their data do not exclude the possibility that it is a feature of coexistent disease. The patients' blood pressure is not given, although high blood pressure is known to be correlated with microalbuminuria in elderly patients[2] and those with essential hypertension.[3] Microalbuminuria is also a marker of cardiovascular disease[4] and, in particular, has been shown in patients with intermittent claudication, in whom it increases after exercise.[5]

A group of patients with chronic obstructive lung disease (age range 57-85) would probably include several with a history of smoking and thus an increased likelihood of cardiovascular disease. These factors should be taken into consideration before microalbuminuria is concluded to be a consequence of chronic obstructive lung disease.

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3 Parving H-H, Jensen H, Morgensen CE, Evrin PE. Increased urinary albumin excretion rate in benign essential hypertension. *Lancet* 1974;i:1190-2.
4 Yudkin JS, Forrest RD, Jackson CA. Microalbuminuria as a predictor of vascular disease in non-diabetic subjects. Islington diabetes survey. *Lancet* 1988;ii:530-3.
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Multiple urine samples should be timed

EDITOR,—R Wilkinson and colleagues describe a heterogeneous group of patients with chronic obstructive lung disease who were found to have microalbuminuria.[1] We question the choice of urine sample used and the authors' suggestion that their observations are consistent only with renal glomerular disease.

The issue of the subclinical increase in urinary albumin excretion and its prognostic importance is confused because the literature contains conflicting nomenclature. What has become clear from several studies,[2] however, is that the variable nature of urinary protein excretion (coefficient of variation 30-40% daily for albumin) demands that at least two and preferably three samples of urine are tested before firm conclusions can be drawn about the true extent of any proposed abnormal protein handling by the kidney. In addition, the term microalbuminuria refers to a timed overnight urinary albumin excretion of >30 μg/min, not a value expressed per mmol creatinine. Wilkinson and colleagues evaluated only one, untimed urine collection and do not mention whether this was a specimen of first pass morning urine or simply a random daytime specimen; random daytime specimens give rise to a particularly high variation, essentially as a function of exercise.[3]

The fact that urinary *N*-acetyl-β-D-glucosaminidase excretion was raised as well as the albumin concentration in the patients with chronic lung disease was mentioned in passing, and no comment was made on this enzyme's association with proximal renal tubular disease. The lysosomes of the renal tubules contain a particularly high activity of *N*-acetyl-β-D-glucosaminidase, and therefore excretion of this enzyme may be considered to be a marker of the integrity of tubular cells. This is reaffirmed by the fact that *N*-acetyl-β-D-glucosaminidase (which has a molecular weight of 150 000 Da, twice that of albumin) has a low intrinsic glomerular penetration. In addition, Mogensen and Solling reported urinary albumin excretion of 500-600 mg/day in normal subjects in whom complete inhibition of tubular reabsorption of albumin was induced by infusion of lysine.[4] This suggests that proximal tubular reabsorption plays an important part in the retention of not only low molecular weight proteins but also albumin.

In their comment Wilkinson and colleagues suggest that the enzymuria identified in this study may reflect tissue hypoxia, renal hypertrophy, or smoking history. Though we do not have any information regarding the first two hypotheses, a study has indicated that smoking habit is unrelated to the incidence of microalbuminuria in both diabetic and healthy control subjects.[5]

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