A 58-year-old Caucasian man was admitted for evaluation of a nephrotic syndrome. He had a past history of mild hypertension which had been treated with a combination of hydrochlorothiazide and amiloride. Blood pressure was 130/70 mmHg; there was no oedema. Serum electrolytes were normal, creatinine 112 μmol/l, albumin 29 g/l, gammaglobulin 5.3 g/l, total cholesterol 10.2 mmol/l; 24-h urinary protein ranged from 3 to 5 g; urinary sediment was normal, and urine culture sterile. GFR was 86 ml/min per 1.73 m^2, as assesssed by ^{125}I Iothalamate clearance. Light microscopy and immunofluorescence studies of the specimens obtained by percutaneous renal biopsy revealed a typical pattern of stage 2 membranous nephropathy.

Further evaluation showed normal blood cell count, serum complement, alphafetoprotein, and carcinoembryonic antigen; there was absence of anti-DNA antibodies, hepatitis B virus markers, antithyroglobulin and antimicrosomal antibodies, cryoglobulinaemia, and mercury in urine. VDRL and FTA tests were negative. There was no evidence of neoplasia, sarcoidosis, or multisystemic or infectious diseases.

Drug inquiry revealed that the patient had been taking fenoprofen 600–1500 mg daily for the preceding 12 months, because of loin pain following a vertebral trauma. Fenoprofen was withdrawn while antihypertensive treatment was maintained, and complete remission of proteinuria occurred within 6 months, without steroid or immunosuppressive therapy, and was persisting 2 years later.

Nephrotic syndrome induced by NSAID has been related to minimal-change glomerulopathy and has been most often associated with acute renal failure and severe intersitial nephritis [1]. NSAID-associated membranous glomerulonephritis is extremely unusual. We are aware of only four reported cases, three with diclofenac [2–4] and one with ketoprofen, a propionic-acid derivative like fenoprofen [5]. All patients presented with the nephrotic syndrome, and complete remission occurred in all within 12 months of stopping the presumed causative drug.

Thus, a search for a prior NSAID treatment should be included in the drug inquiry conducted in every patient with membranous glomerulonephritis.

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Systemic and Disseminated Candidiasis Complicating Renal Failure

Sir,—

We read with great interest the paper 'Systemic and disseminated candidiasis complicating acute renal failure' by Stevens and co-workers [1].

It was rather disconcerting to note that none of their patients were on prophylactic oral antifungal treatment. In a controlled trial Ledingham and co-workers [2] using a Selective Parenteral and Enteral Antisepsis Regimen (SPEAR) which included systemic cefotaxime, oral polymyxin E, tobramycin and amphotericin B were able to demonstrate a substantial reduction in the incidence of acquired infection (10% vs 24%). The isolation of yeasts from stomach, throat and trachea declined appreciably during days 2–4 in the test group, but changed little with time in the control group.

It has been our time-honoured practice to give nystatin 100 000 units 6-hourly to all patients admitted to our Regional Renal Unit, irrespective of the type of renal failure, thereby obtaining gratifying results. However, we were recently involved in the management of a patient with acute myeloid leukaemia who had not received oral nystatin and had developed hepatic candidial abscesses and septicaemia; despite treatment with amphotericin B and flucytosine, the patient died with renal failure.

One of us (P.McC) recently investigated 27 patients who required ventilation and dialysis for a period of more than 5 days. Cultures (1641) were taken from tracheal aspirate, urine, wound and blood. Of 1011 cultures in 15 patients using SPEAR therapy, 103 were positive for yeasts (10.4%). Of the 630 cultures taken in the remaining 12 control patients, 137 were positive for yeasts (21.7%). Our study is in agreement with the findings reported by Ledingham and co-workers [2]. We cannot but emphasise the importance of oral antifungal therapy, which is not only a simple, but also a cheap method of preventing candidiasis.

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More on an Easily Obtained Accurate Dialysis Index

Sir,—

The search for quantification of dialysis treatment as stimulated by the NCDS study resulted in different approaches to collecting data in order to calculate $K \times t/V$ (K = clearance; t = treatment time; V = distribution volume). K was most often taken from in vitro data provided by the manufacturers of filters, whereas the distribution volume was either set at 0.58 × bodyweight, or was calculated from urea extraction divided by change of blood urea concentration before and after treatment.

We measured urea extraction by collecting dialysate, and obtained measurements for blood urea before and after treatment. Results for $K \times t/V$ obtained from these 'true data' were compared with those obtained from both calculated and in vitro data. Furthermore, a formula is derived which permits the estimation of the 'true' dialysis index from blood urea measurements before and after treatment.

	Calculation mode	K	V	K × t/V
1	K: in vitro data V: 0.58 × BW	225.36 ± 25.15	34.34 ± 4.56	1.05 ± 0.15
2	K: in vivo data V: 0.58 × BW	173.00 ± 23.69*	34.34 ± 4.56	0.80 ± 0.09*
3	K: } in vivo dialysate V: } and blood urea	173.00 ± 23.96*	39.12 ± 10.95	0.74 ± 0.17*

M ± SD; $n = 14$; *Significance: 1 vs 2, 1 vs 3: $P < 0.001$

Results (Table) indicate that the 'true' dialysis index according to formula F3 is 25% lower than the calculated value using in vitro clearance data and 0.58 × BW distribution volume (F1). Replacing the estimated clearance data by measured values (F2) adapts the index value partially to the true finding. The calculation of the 'true' dialysis index from extracted urea in dialysate and concentration pre- and post-treatment included

$$\text{clearance (K)} = \frac{\text{urea extracted/treatment time}}{(\text{conc. pre} + \text{conc. post})/2}$$

$$\text{urea distribution volume (V)} = \frac{\text{urea extracted}}{\text{conc. pre} - \text{conc. post}}$$

Combining these formulae for calculation of

$$I = \frac{K \times t}{V} \text{ results in}$$

$$I = \frac{\text{conc. pre} - \text{conc. post}}{(\text{conc. pre} + \text{conc. post})/2}$$

In conclusion, the 'true' dialysis index, taking into account in vivo filter clearance and individually calculated distribution volume, is easily obtained from routine pre- and post-dialysis blood urea measurements.

Note added in proof: This formula was recently described by Barth and Berlyne in abstract form (*Kidney International* 1988; 33, 216)

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Treatment of *Pseudomonas aeruginosa* Peritonitis in a CAPD Patient with Ciprofloxacin

Sir,—

CAPD-associated peritonitis caused by *Pseudomonas aeruginosa* is a very serious complication of CAPD treatment, often resulting in catheter removal. Ciprofloxacin, one of the new quinolones, seems to have promising properties for the treatment of serious *Pseudomonas aeruginosa* infections [1,2].

A 58-year-old woman treated by CAPD for 10 months suffered five episodes of *Staphylococcus aureus* peritonitis. She developed a further episode of peritonitis and *Pseudomonas aeruginosa* was cultured from the effluent fluid. She was treated with intraperitoneal gentamicin and ticarcillin for 8 days and after 4 days of treatment the cultures became negative. Therapy was continued, but after another 4 days *Pseudomonas aeruginosa* was again cultured.

Ciprofloxacin was started with an initial intravenous dose of 400 mg, followed by intraperitoneal administration of 50 mg q.d.s. Serum levels were between 0.2 and 0.6 mg/l and peritoneal dialysate levels varied between 1.4 and 2.2 mg/l. The minimum inhibitory concentration (MIC) of ciprofloxacin for the *Pseudomonas aeruginosa* strain was 0.05 mg/l. After 2 days, dialysate cultures became negative. Because of persistent high leucocyte counts in the peritoneal fluid, gentamicin was added to the regimen after 8 days of ciprofloxacin therapy. The cultures remained negative and leucocyte counts returned to normal after another 3 days. No side-effects were observed.

Ciprofloxacin has a bactericidal action against Gram-negative bacteria including *Pseudomonas aeruginosa* [3]. The quinolones have a low incidence of side-effects [4]. Ciprofloxacin was very well tolerated by our patient. Intraperitoneal doses of 50 mg q.d.s. resulted in adequate ciprofloxacin levels in the peritoneal dialysate, which were 30 to 40 times the MIC value of the infecting organism. Ciprofloxacin seems to be a very promising drug in the treatment of CAPD-associated peritonitis caused by *Pseudomonas aeruginosa*.

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A Modification to the 'Shaldon Technique' when Inguinal Fibrosis is Present

Sir,—

Shaldon's introduction of the Seldinger technique to catheterise the femoral vein [1] was a milestone in the management of the uraemic patient. Since then thousands of patients with acute renal failure and chronic renal insufficiency have benefited from this procedure. However, its repeated use has occasionally resulted in a zone of fibrous tissue in the inguinal region due to either consecutive localised haematomas and/or the effect of the local anaesthesia. In these cases its use is then not viable, as even if the vessel can be localised and the guidewire inserted, the catheter cannot be advanced because of the underlying fibrosis.

Although the development of different vascular access techniques [2], the use of unipuncture systems for undertaking haemodialysis sessions [3], or the permanence of catheters in the femoral vein for long periods of time [4] have decreased the incidence of this problem, it is still necessary on occasions to resort to the use of the classic Shaldon technique for the introduction of a vascular catheter.

We suggest a modification in the technique described by Shaldon to introduce a femoral venous catheter through an inguinal region when fibrosis makes its use impracticable. The femoral vein is localised in the traditional way, using a needle of Cournand through which a guidewire is passed. The needle is