tion, toxins, fluid and electrolyte depletion, drug toxicity and immune complexes may play a role.[1,2]

The initial diagnosis of renal impairment relies on the history, observation of urine output, and plasma biochemistry. Hospitals vary in the range of biochemical tests offered. We present the following cases seen over a 4-month period, to illustrate some of the problems encountered in diagnosing renal impairment in acute, or acute-on-chronic hepatic failure.

A 33 year old man was transferred to St. James's University Hospital with paracetamol-induced acute liver failure. Investigations prior to transfer revealed markedly deranged liver function tests, normal plasma sodium and potassium levels and a mildly elevated plasma urea at 8.3 mmol/l (normal range 2.2–7.7). Plasma creatinine was not measured.

On transfer, he was jaundiced and encephalopathic. Investigations confirmed a mildly raised plasma urea at 12.3 mmol, but with a grossly raised plasma creatinine at 620 μmol/l (normal range 45–120). The patient was anuric, and required haemofiltration and haemodialysis.

A 60 year old woman with primary biliary cirrhosis of 5 years' duration was admitted with increasing ascites and oedema. Serial investigations demonstrated increasing plasma creatinine (223 μmol/l) without any substantial rise in plasma urea (11.5 mmol/l). With treatment of infected ascites her biochemical abnormalities resolved.

A 35 year old woman was admitted following a paracetamol overdose. On admission she had no jaundice or encephalopathy, but was noted to be oliguric. Initial investigations demonstrated a normal urea of 5.2 mmol/l with an elevated creatinine of 353 μmol/l. Over the following 24 hours she became encephalopathic and despite full supportive therapy died.

A 29 year old woman was transferred with fulminant hepatic failure secondary to halothane anaesthesia. Investigations prior to transfer revealed a normal plasma urea concentration. A plasma creatinine was not available. Following transfer she was unconscious with a plasma urea of 10.8 mmol/l, a plasma creatinine of 496 μmol/l and grossly abnormal liver function tests.

The early identification of renal impairment in patients with severe liver disease is clearly important. Impairment of hepatocyte function leads to a decreased rate of urea synthesis, and with renal impairment, the rise in plasma urea will be reduced. In contrast, plasma creatinine is largely independent of liver function, and is therefore a better marker for renal impairment in patients with liver disease.[3]

The above cases, seen over a short period, involved patients with acute, or acute-on-chronic, liver failure. One of the above cases was transferred from a hospital where estimations of plasma creatinine were not carried out acutely, and hence it was not realized that the patient was in established renal failure. In another case the discrepancy between the plasma urea and creatinine, and its implication, was not noted until transfer.

In patients with hepatic failure, an accurate assessment of fluid balance is mandatory, but is often neglected, or carried out inaccurately. The use of plasma urea as a marker of renal function is unreliable in patients with liver failure, and may lead to delay or failure to diagnose clinically important renal failure. Estimations of plasma creatinine should always be carried out in addition to urea and electrolytes.

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Dermatitis artefacta complicated by a cerebral abscess

Sir,

Dermatitis artefacta is characterized by a variety of cutaneous lesions. Here we describe a case complicated by a cerebral abscess.

A 46 year old educationally subnormal woman was admitted with a 9-day history of dysphasia following a *grand mal* fit. On examination she was febrile and disoriented. A large ulcerating scalp lesion was present over the left parietal area. Indeed this lesion had been present for 20 years and previously diagnosed as dermatitis artefacta. A biopsy of the ulcer the previous year had shown no evidence of infection or malignant disease.

A skull radiograph showed a 4 cm, well defined lytic area in the left parietal bone. Computed tomography of the head demonstrated a low attenuation mass lesion in the left parietal cortex with a smooth enhancing rim. The appearances were consistent with a cerebral abscess and this was confirmed at craniotomy. Culture of the abscess grew *Staphylococcus aureus.* Of interest was the identification, on microscopy, of dicotyledenous vegetable matter in the centre of the abscess (Figure 1). Biopsy of the adjacent bone and skin revealed no evidence of malignancy.

We believe that over a 20 year period continuous self-mutilation, perhaps with superadded infection, led to the destructive lesion of the skull vault. At some stage the vegetable matter was implanted, presumably during an episode of self injury. The foreign matter later served as the nidus of infection.

In dermatitis artefacta a variety of cutaneous lesions occur. Lacerations, ulcers, blisters or burns are often present. The diagnosis is often suggested by the bizarre appearance and distribution of the lesions.[1] It is usually a disease of women[2] and is vehemently denied, as in our patient.

This case serves to illustrate two main points. First, the

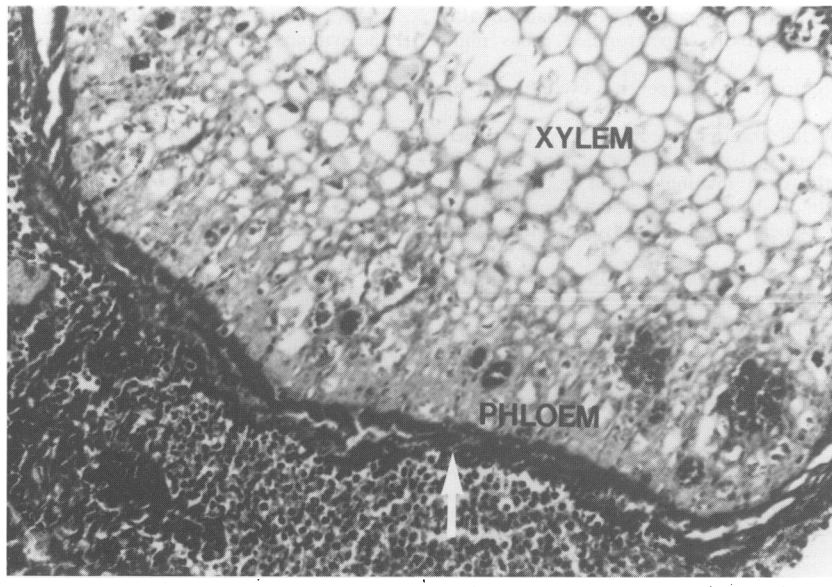

Figure 1 High power photomicrograph in which the plant wall is clearly seen (arrow). Xylem and phloem elements are present. Neutrophils and macrophages surround the plant. Magnification ×100. Haematoxylin and eosin.

association of a scalp lesion and neurological signs should prompt urgent neuroradiological investigations, no matter how superficial the former may look. Second, in dermatitis artefacta one should always remember that 'anything is possible'.[3]

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Bilateral pelvic masses in a longdistance cyclist

Sir,

A 37 year old man presented with a two week history of bilateral pitting oedema of the legs. He had previously given up longdistance running due to 'jogger's trots' and had taken up competitive cycling. Just prior to presentation he had competed in a 150 km cycling race.

Physical examination showed a very fit man with mild ankle oedema. Blood count, ESR, urinalysis, serum biochemistry and chest radiograph were normal. Computed tomographic (CT) scan of the abdomen was reported as showing bilateral massive pelvic lymphadenopathy (Figure 1).

Because of the patient's clinical history and wellbeing and the symmetrical nature of the 'pelvic lymphadenopathy', a lymphangiogram was performed; this showed no evidence of lymphadenopathy. On review of the CT scans,

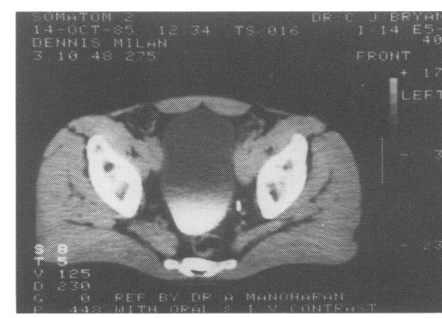

Figure 1 Computed tomographic scan with oral and I.V. contrast showing bilateral, symmetrical pelvic 'masses' impinging on the urinary bladder.