definite spontaneous activity but poor relaxation and severe contraction fasciculation. Twitching was associated with a polyphasic unit of long duration firing about five times a second. Units were prolonged and polyphasic up to 3 mV. The interference pattern was reduced. We concluded that the quadriceps weakness was neurogenic and that the pattern of electrophysiological abnormality was consistent with motor neurone disease.

Left radical pneumonectomy was performed with good clearance of lymph nodes from the mediastinum. The neoplasm was a large-cell carcinoma with a suspicion of adenocarcinomatous tubules in places. All fasciculation immediately resolved and pain in the knees disappeared.

Six months later there was much less wasting of quadriceps and no fasciculation. Right quadriceps and hamstrings were slightly weak but the left leg was normal. Reflexes in the legs were normal and plantar responses flexor. Clubbing was reduced, and the gynaecomastia had gone. Electromyography of the right vastus medialis and rectus femoris showed no fibrillation or fasciculation and, though the interference pattern was mildly reduced, the motor units looked normal. Some polyphasic units were seen in the right tibialis anterior but they were not prolonged or increased in amplitude. There was no evidence of a lower motor neurone lesion.

### Comment

Carcinomatous neuromyopathies occur in 16% of men with bronchial carcinoma.[3] Brain *et al* reported on 11 patients with a syndrome indistinguishable from motor neurone disease—though running a more benign course—associated with malignancy.[1] Five had bronchial carcinoma, one of whom partially improved after resection. They concluded that motor neurone disease may be a manifestation of carcinomatous neuromyopathy. Our patient achieved complete remission, which we believe suggests a causal relation between the carcinoma and the neurological syndrome.

We thank Dr C J Earl and Dr F H Scadding for their help and advice in preparing this case report.

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[2] Jokelainen, M, *Journal of the Neurological Sciences*, 1976, **29,** 55.
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# Acute tubular necrosis after exposure to diesel oil

Exposure to diesel oil has been implicated in two previous reports of acute oliguric renal failure.[1 2] Our case represents the first such report from Britain and provides further evidence that diesel oil should be considered a nephrotoxin.

### Case report

A 47-year-old man presented with a month's history of epigastric and loin pains, thirst, nocturia, nausea, anorexia, and severe exhaustion, and a week's history of scrotal swelling. This unusual presentation prompted an inquiry into possible toxic exposure, and he admitted to cleaning his hands and arms with diesel oil over several weeks while building a home extension. On examination he looked pale and ill and had bilateral loin tenderness and scrotal oedema. Initial investigations showed: haemoglobin 11·9 g/dl, white cell count 6·7 $\cdot 10^9$/l, erythrocyte sedimentation rate (ESR) 76 mm in the first hour, blood urea 6·5 mmol/l (39 mg/100 ml); plasma sodium 141 mmol(mEq)/l, plasma potassium 4·1 mmol(mEq)/l, plasma bicarbonate 26 mmol(mEq)/l, total protein 77 g/l, and serum albumin 43 g/l; urine analysis showed no protein, cells, casts, or growth.

Two weeks later he developed pitting ankle oedema and further investigation showed: haemoglobin 9·4 g/dl, reticulocytes 0·2%; blood urea 17·0 mmol/l (102 mg/100 ml), plasma sodium 139 mmol/l, plasma potassium 5·0 mmol/l, total protein 66 g/l, serum albumin 35 g/l, serum creatinine 910 μmol/l (10·3 mg/100 ml); urine analysis normal, 24-hour urine protein 0·19 g, and creatinine clearance 14·2 ml/min. Intravenous urography showed a faint nephrogram, normal-size kidneys, and no evidence of obstruction.

His urine output fell to 100 ml in 24 hours but he responded to frusemide with diuresis of 1500-2500 ml per 24 hours with disappearance of his oedema. His blood urea peaked at 28 mmol/l (169 mg/100 ml) during the diuretic phase. Renal biopsy showed the classic findings of nephrotoxic acute tubular necrosis—namely, patchy degeneration and necrosis of proximal and distal tubular epithelium with preservation of basement membranes.[3] There were 14 normal glomeruli and some interstitial oedema. He subsequently made a good recovery with a haemoglobin concentration of 12·6 g/dl, ESR 27 mm in the first hour, blood urea 6·5 mmol/l (39 mg/100 ml), and serum creatinine only slightly raised at 140 μmol/l (1·6 mg/100 ml) three months later.

### Comment

The intermittent but frequent use of diesel oil before the onset of this patient's illness may explain the subacute development of renal failure. Nevertheless, the patient had histologically proved acute tubular necrosis. Detailed questioning revealed no history of exposure to any other nephrotoxin. Reidenberg *et al*[1] described a patient with acute oliguric renal failure following 10 days' exposure to diesel oil vapour in the cab of a lorry. Abdominal cramps and epigastric and loin tenderness, and oedema were prominent features, as in our patient. Strong evidence for absorption of diesel oil through the skin was offered by the patient described by Barrientos *et al*.[2] He developed acute oliguric failure on the same day as washing his hair with diesel oil, but renal biopsy—performed much earlier than in our patient, on the second oliguric day—showed only tubular dilatation with casts and a "light proliferation" of mesangial cells in the glomeruli. Considering the widespread use of diesel oil, toxic effects are extremely rare, although contact dermatitis is well recognised.[4] Nevertheless, diesel oil by topical exposure or vapour inhalation should be accepted as a rare cause of acute renal failure.

Requests for reprints should be sent to Dr B I Hoffbrand.

[1] Reidenberg, M M, *et al*, *American Journal of the Medical Sciences*, 1964, **247,** 25.
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[3] Jones, D B, in *Pathology*, ed W A D Anderson and J M Kissane, p 959. Philadelphia, Mosby, 1977.
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# Insulin oedema

Oedema occurring soon after starting insulin treatment is occasionally mentioned in textbooks on diabetes mellitus,[1] but we have found few descriptions of cases. We here report one such case.

### Case report

A 31-year-old man was admitted as an emergency with a 5-day history of vomiting, diarrhoea, and increasing confusion. He had been fit, and there was no family history of diabetes. He was disorientated, dehydrated, and smelled of acetone. Urine tests showed large quantities of sugar and ketones, and his blood glucose concentration was 52·8 mmol/l (951 mg/100 ml). He was given intramuscular injections of 5 units of soluble insulin every hour and appropriate intravenous fluids. He responded well, and after eight hours his blood glucose concentration had fallen to 13 mmol/l (234 mg/160 ml). He was eventually stabilised on 50 units of Monotard insulin daily.

After seven days he developed ankle oedema, which became generalised over the next 24 hours and was particularly noticeable on the face; he resembled a patient with the nephrotic syndrome. His weight increased from 63 kg on the second day, when he had already been rehydrated, to 67 kg on the eighth day. His blood pressure and electrocardiograph were normal, and he felt well. Twenty-four-hour urinary protein output was 2·43 g/l and no erythrocytes or casts were seen. Serum total protein concentration was 60