line was inserted and an arteriovenous shunt created in the left leg. After haemodialysis for eight hours the patient passed urine for the first time in 48 hours. A diuresis occurred over the next two days and the blood chemistry reverted to normal. On the fifth day after admission a left deep vein thrombosis developed, which was treated with intermittent subcutaneous heparin 5000 units eight-hourly. The maternal and fetal conditions remained satisfactory and urinary and plasma oestriol concentrations were within normal limits. After 36 weeks' amenorrhoea spontaneous labour occurred and a healthy male infant weighing 3·6 kg was delivered. The puerperium was uncomplicated. Anticoagulation was continued for six weeks. Urine analysis and blood pressure were normal. Two weeks after delivery an intravenous pyelogram and renal scan showed normal postnatal appearances with slightly dilated collecting systems. At the postnatal visit mother and baby were well.

### Comment

The absence of infection, shock, or toxaemia and the complete reversibility of the condition suggests that pressure from the gravid uterus may well have been a contributory factor in this case. The short duration and favourable outcome seem to rule out a serious lesion. In the absence of gross hydronephrosis and with the almost universal availability of dialysis units haemodialysis seems to offer a more rational approach to these unusual and mystifying cases.

I thank Dr C S Ogg and Mr T M Coltart for permission to publish this case.

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# Anergy—a prognostic indicator in early breast cancer

The type IV delayed hypersensitivity skin response to recall antigens may be reduced in early breast cancer.[1 2] The degree of anergy apparently increases as the disease progresses.[2] We report a three-year follow-up of 100 patients with early breast cancer who were skin tested preoperatively with three recall antigens.

### Patients, methods, and results

The patients were part of a unicentric randomised prospective clinical trial comparing simple mastectomy alone with simple mastectomy and radical radiotherapy as treatments for early breast cancer (T1 and T2, N0 and N1, M0). Axillary lymph node biopsy for more accurate staging and a skeletal survey were done preoperatively in all patients but bone scanning was not routine. Ten units purified protein derivative (PPD), 0·02 ml 0·5 % *Candida albicans* (Bencard), and 10 units Varidase (streptokinase/streptodornase) were injected intradermally into the forearm opposite the affected breast and the diameter of induration at 72 hours measured: 5 mm or over was called positive.

Seventeen patients failed to react to any of the recall antigens. Ten (59%) of these 17 anergic patients had died or had distant metastases by three years, whereas by then only 23 (28 %) of the 83 reactive patients had died or developed metastases. This difference is highly significant ($\chi^2$ with Yates's correction = 6·96, P = < 0·01). Treatment did not affect the clinical outcome,[3] and five of the 10 anergic patients had had additional radical radiotherapy as a primary treatment. Six (86 %) out of seven anergic patients who also had histological evidence of axillary lymph node involvement had died or developed metastases by three years. The mean (±SD) age of the anergic patients (62·8±5·9 years) was significantly greater that of the reactive patients (53·7±8·6 years) ($t = 5·26$, P = < 0·01).

### Comment

Eilber and Morton[4] reported that over 90% of unselected cancer patients presenting for surgery who failed to react to dinitrochlorbenzene (DNCB) were either inoperable or had early recurrence. Our study shows that anergy is also a prognostic indicator in early breast cancer, since over half (59%) of the patients who failed to react to any of three recall antigens had died or had distant metastases by three years. Perhaps anergy to a selection of recall antigens could be used in conjunction with other high risk factors, such as lymph node involvement, in selecting patients for adjuvant therapy. Whether anergic individuals are more prone to develop cancer or cancer induces a state of anergy has yet to be determined. Anergy does become more marked with advancing disease,[2] however, and failure to react may well be related to this. Our patients who had died or developed distant metastases by three years must have had occult metastases when they were skin tested, which may have affected their responsiveness. The results also confirm the reported[5] correlation between anergy and advancing age.

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# Stercoral perforation: case of drug-induced impaction

Stercoral perforation of the colon, a rare complication of faecal impaction, often results in life-threatening peritonitis. This case report illustrates a potential hazard in giving drugs with anticholinergic side effects to patients with refractory constipation.

### Case report

A 74-year-old woman was admitted four hours after sudden onset of severe abdominal pain maximal in the left iliac fossa. Before the onset of pain she had passed a small, hard motion with some bright blood after prolonged straining. She was an active woman prone to severe constipation since childhood. She also had bouts of depression, for which she had taken amitriptyline 10 mg twice daily for four months. During this period her constipation had worsened. Her only other medication was benorylate suspension, which she took occasionally for backache. She looked ill, her facies were ashen, and extremities cyanosed. Her blood pressure was 200/100 mm Hg and pulse rate 100/min. Signs of general peritonitis were present, and rectal examination yielded blood-stained faeces with marked tenderness anterosuperiorly. Investigations did not contribute to the diagnosis, which was thought to be a perforated diverticulum.

At laparotomy generalised faecal peritonitis was found associated with a 2-cm circular perforation with bruised ragged edges on the antimesenteric border of the rectosigmoid. There were several scybala in the pelvis and extensive faecal staining of the peritoneum. The entire colon and terminal 90 cm of ileum were solidly impacted with faeces. No other abnormality of the bowel was seen. After scrupulous peritoneal lavage Hartmann's procedure was performed. Intravenous antibiotics and metronidazole were started intraoperatively. Postoperative recovery was complicated by wound infection and deep venous thrombosis which developed after 11 days. She progressed satisfactorily but required regular colostomy enemas for persisting constipation.

### Comment

Fewer than 40 cases of stercoral perforation have been reported. Mortality figures are not readily available but faecal peritonitis from all types of colonic perforation treated surgically carries a mortality of about 50%.[1] Perforation occurs most often in the recto- and mid-

sigmoid regions and occasionally in the caecum. It results from pressure necrosis of the colonic wall from impacted faeces. Constipation is the pre-eminent symptom in all cases and straining at stool the precipitating event. Drug-induced faecal impaction is becoming more prevalent and is not uncommon in opiate addicts. Both faecal impaction and perforation have been recently reported in renal transplant patients in whom aluminium-based antacids have been implicated.[2] In the present case constipation worsened during antidepressant therapy. Ayd[3] reported that 60% of patients taking tricyclic antidepressants suffer from constipation, and cases of adynamic ileus have been described in patients taking these drugs.[4] Giving drugs that strongly inhibit colonic motility to patients with refractory constipation is not without hazard.

Adequate resuscitation, antibiotic cover, and early operation form the basis of treatment. In generalised peritonitis exteriorisation[5] with limited resection of the perforation and thorough peritoneal lavage remains the operation of choice. In contrast to similar situations in diverticular disease, mobilisation of the colon presents few problems. Advocates of resection and primary anastomosis must carefully consider the dangers of gross contamination and unprepared bowel in an often moribund patient. In localised infection or in right-sided perforations, however, this procedure becomes more feasible. Some surgeons delay primary closure of the skin incision in view of the unavoidable wound contamination. Antibiotic cover should be directed against Gram-negative and anaerobic organisms. Later, persisting constipation merits careful attention.

I thank Mr J C Hammonds, consultant surgeon, Plymouth General Hospital, for permission to publish this report.

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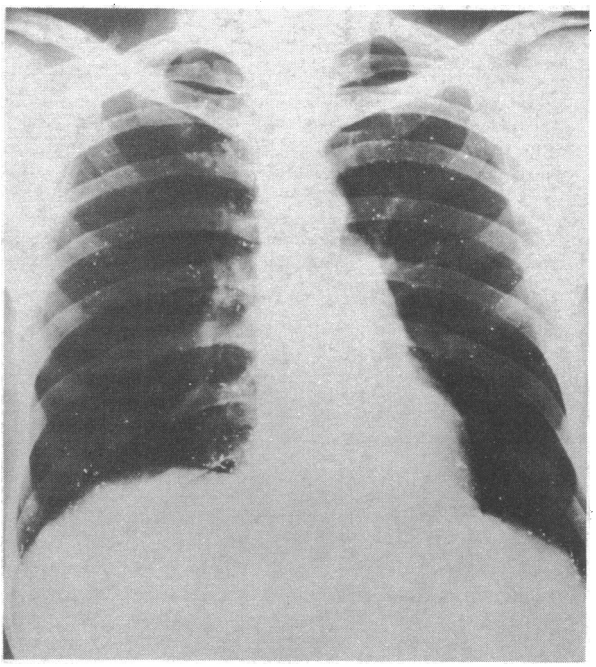
# Self-administration of metallic mercury by intravenous injection

Intravenous injection of metallic mercury is an unusual event which may have different consequences. Death may result after an acute illness characterised by increasing pulse rate, fever, muscle spasms and rigidity, respiratory failure, and an increasing blood urea concentration.[1] Large (20 ml) intravenous injections of mercury resulting in extensive pulmonary embolism due to mercury globules may cause only moderate upset of pulmonary function. This may include moderate degrees of restrictive lung disease and diminution of diffusing capacity. These usually return to normal.[2] In other cases only transient impairment of renal and hepatic function may occur,[3] or if only minute quantities of mercury have been injected no ill effects may result.[4]

## Case report

A 34-year-old man with a history of psychiatric illness was admitted allegedly having ingested two packets of Weedol (containing 1·5 g paraquat) followed by boiled hawthorn roots, which he thought might be the antidote. He was symptom-free but examination showed a chronic granuloma in the left antecubital fossa. He stated that he had injected himself six months before with liquid mercury. The exact quantity was not known.

Chest radiography (see figure) showed numerous specks of mercury throughout both lung fields, mainly at the peripheries and bases. Small specks were also seen throughout the liver, in the left kidney, and in the region of the right parotid gland, indicating that the mercury had passed through the lungs into the systemic circulation. The haemoglobin concentration was 14·5 g/dl. A midstream specimen of urine initially contained $60 \times 10^6$ white cells/l (60/mm$^3$) and moderate amounts of protein but later became normal. Blood urea was 5·5 mmol/l (33 mg/100 ml); serum creatinine 80 μmol/l (0·9 mg/100 ml). Creatinine clearance varied from 68 to 102 ml/min, and 24-hour urinary protein excretion was 56 mg. Serum total bilirubin was 12 μmol/l (0·7 mg/100 ml), and the transaminase concentrations were normal. Serial blood gas measurements and ECGs were also normal. Results of pulmonary function tests were: forced expiratory volume in 1 s 4·6 l, forced vital capacity 5·2 l, peak flow 560 l/min, and carbon monoxide transfer factor 6·7 mmol/min/kPa—predicted 11·4—(20 ml/min/mmHg—predicted 34). Urinary mercury on admission was 850 μg/l, and five days later it was 2 mg/l (normal less than 80 μg/l). Blood mercury was 123 μg/l (normally undetectable). The blood paraquat concentration was below 1 μg/l, which suggested that little or no paraquat had been ingested or absorbed.



Radiographic appearances of mercury in both lung fields. Heart is displaced by depressed sternum.

## Comment

This patient showed evidence of considerable mercury embolisation in the pulmonary and systemic circulation associated with raised blood and urinary mercury concentrations. There was a moderate decrease of diffusing capacity in the lungs and possibly a diminished creatinine clearance but no other abnormalities.

The long-term effects of metallic mercury poisoning are not fully known. Removal of mercury granulomas at injection sites should be attempted as considerable quantities may be removed.[5] Slow ionisation of metallic mercury by biological oxidation forms mercuric salts that are largely excreted via the colon, kidneys, and salivary glands.[4] Penicillamine or dimercaprol may be used to enhance the elimination of heavy metals from the body and are recommended in acute inorganic mercury poisoning, but their value in chronic mercury poisoning is not known.

No specific measures were employed in this patient, who unfortunately was lost to follow-up.

I am indebted to Lt-Col M Brown, RAMC, for permission to report this case.

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