showed dense consolidation in the left upper lobe. Sputum samples showed numerous acid-fast rods on smear. He was started on rifampicin, isoniazid, pyrazinamide and streptomycin. Seven days later his chest X-ray showed a left upper lobe cavity 3 cm in diameter containing a dense central opacity. This enlarged to a maximum size of 4 cm at 10 days and subsequently resolved over 3 months. Because of the rapid appearance of cavitation, a bronchoscopy was performed which showed purulent secretions draining from the left upper lobe with underlying erythematous bronchial mucosa. Bronchoalveolar lavage showed numerous acid-fast bacilli, no *P. carinii*, fungi or neoplastic cells. Cultures grew no pathogens other than fully sensitive *M. tuberculosis*.

The radiological pattern of pulmonary tuberculosis is often atypical in AIDS patients. Hilar or mediastinal lymphadenopathy and localized pulmonary infiltrates in the middle or lower lung fields are the most common features,² while pulmonary cavitation is infrequent.^{3,4} Cavitation may occur due to *P. carinii* and can mimic TB with the rapid appearance of single or multiple cavities.⁵ Although *Klebsiella* sp., *Nocardia*, *Cryptococcus neoformans*, and *Mycobacterium avium-intracellulare* can also cause pulmonary cavitation in HIV patients, *M. tuberculosis* alone can cause very rapidly progressive pulmonary cavitation in patients with HIV infection.

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Pseudogout associated with thyrotoxicosis

Sir,

Pseudogout is a frequent cause of arthritis in the elderly and is characterized by calcium pyrophosphate dihydrate (CPPD) crystals in the joint fluid and chondrocalcinosis.

We describe a case of acute pseudogout associated with thyrotoxicosis.

An 89 year old woman was admitted with abdominal pain and vomiting of 4 days duration. She had lost 7 kg in weight over 1 year and was in atrial fibrillation. There were no other clinical features of thyrotoxicosis. The abdomen was distended and the rectum loaded with faeces. A diagnosis of faecal impaction was made. She responded to treatment with enemas and laxatives, but 3 days later she developed a swollen tender hot right knee and a temperature of 37.8°C. A total of 60 ml of cloudy fluid was aspirated and she responded to treatment with non-steroidal drugs. The synovial fluid aspirate contained large numbers of calcium pyrophosphate crystals. An X-ray of the right knee showed chondrocalcinosis. The serum calcium level was normal. Thyrotoxicosis was confirmed biochemically and she was then referred on for radioactive iodine treatment.

The association of pseudogout with hypothyroidism is well recognized. Alexander *et al.* found 10% of their CPPD study patients to be hypothyroid.¹ However, there is no recorded case of pseudogout associated with thyrotoxicosis. Hypothyroid patients with previously asymptomatic CPPD crystal deposition often have a flare-up of arthritis when thyroid medication is started.² Thyroxine thus probably has some role in precipitating acute crystal deposition, the mechanism being unknown. Hence the association of thyrotoxicosis in our patient with pseudogout may be more than casual.

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Morphine and severe dryness of the lips

Sir,

Dryness of the mouth is not generally recognized as a side effect of morphine,^{1,2} but clinical experience suggests that it is a common complaint when used in patients with cancer.³ The mechanism for this effect is unclear. We report a case of dryness of the lips accompanied by severe pain after intravenous administration of morphine, and suggest possible pathophysiological mechanisms.

A 60 year old man presented with acute myocardial infarction with no complications. The patient received 5 mg morphine intravenously for the chest pain. No other drugs were given. A few minutes later, gradual relief of his chest pain began accompanied by a complaint of progressive dryness of the lips which shortly became very dry and

stiff; their colour changed from red to very pale with local severe pain. These manifestations gradually resolved spontaneously within 2 hours. Two months later, the patient presented with unstable angina pectoris and received 5 mg morphine intravenously followed by the same phenomena as before.

These observations indicate that morphine was responsible for the patient's manifestations. The mechanisms by which morphine induces dryness of the mouth and lips are unclear, but two can be proposed. First, it is well known that secretions of some exocrine glands such as the pancreas, are diminished by morphine,^{1,2} and we suppose that the same may occur to salivary and mucous glands, causing dryness of the mouth and lips. Second, the underlying papillae of the lips and oral mucosa are abundantly supplied with capillaries and the colour of the blood showing through the overlying tissues, results in the characteristic red appearance.⁴ The change of colour to very pale shortly after morphine administration indicates a local vasoconstriction of the capillaries or the supplying small arterioles.

The above phenomena in the lips as a side effect of morphine have not been reported previously. This observation and the results of another recent study³ merit detailed investigation of the underlying mechanisms.

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Hypoglycaemia and heart failure

Sir,

Hypoglycaemia has been reported with congestive cardiac failure,^{1,2} but scant attention has been paid to the subject.

It is frequently overlooked as an explanation for convulsions or coma, and symptoms such as palpitations, sweating or bizarre behaviour may be dismissed as manifestations of heart failure. We wish to report a 79 year old man with a long-standing history of heart failure due to ischaemic heart disease with atrial fibrillation, who was admitted with coma. He was peripherally cyanosed, with heart rate 120 beats/min in atrial fibrillation and

blood pressure 60/40 mmHg. Investigations included serum sodium 136 mmol/l, potassium 6 mmol/l, creatinine 260 mmol/l, alkaline phosphatase 260 u/l, aspartate transaminase 407 u/l, gamma glutamyl transaminase 216 u/l, bilirubin 52 µmol/l, albumin 26 g/l, calcium 2.12 mmol/l and blood glucose was 0.2 mmol/l. Treatment with 50 ml 50% dextrose intravenously caused the patient to recover and wake up. He was then commenced on a continuous infusion of 10% dextrose, together with frusemide 50 mg twice daily, but blood glucose kept falling below 2.0 mmol/l and he needed additional bolus injections of 50% dextrose. In spite of this he died 4 days later with intractable hypoglycaemia. Post-mortem examination confirmed the presence of an enlarged heart with widespread coronary atheroma, the liver showed severe congestion and central necrosis. The pancreas brain and pituitary gland were normal.

The commonest causes of hypoglycaemia observed in hospital occur in diabetes and the use of insulin and other hypoglycaemic drugs, chronic renal failure, various tumours, different forms of liver disease, adrenal insufficiency, or hypopituitarism.³ None of these causes was present in this patient. Persistent and intractable hypoglycaemia is unlikely to be due to functional hyperinsulinism. Furthermore an insulin-secreting tumour of β-cells of the pancreas was not found at post mortem.

Mellinkoff and Tumulty⁴ described 5 patients like ours who had hypoglycaemia during the course of congestive cardiac failure, in which chronic passive congestion of the liver was shown as post mortem. They considered that hepatic glucose output was reduced by poor diet, poor glucose absorption and impaired hepatic glycogenolysis and gluconeogenesis.

In 27 paediatric patients with congestive cardiac failure, and hypoglycaemia, liver biopsies were performed in 4 patients, and hepatic glycogen content was found to be extremely low.² It was observed that improvement in cardiac failure did not occur until hypoglycaemia was corrected.

Hepatic glycogen is an important endogenous source of blood glucose. Glycogen depletion appears to be the mechanism for the observed hypoglycaemia in the paediatric patients. Low liver glycogen may have resulted from poor dietary intake preceding the development of hypoglycaemia. In a 79 year old man with chronic angina the difficulties of maintaining adequate food intake are self-evident, but when hypoglycaemia supervenes the prognosis of cardiac failure may deteriorate, and correction with intravenous glucose should be an essential component of cardiac failure treatment.

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