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.

Majed Odeh

Arie Oliven

Harry Bassan

Department of Internal Medicine 'B',

Bnai Zion – Medical Center,

and Technion Faculty of Medicine,

Israel Institute of Technology,

Haifa, Israel.

Correspondence to: Majed Odeh, M.D.,

P.O. Box 6477, Haifa 31063, Israel.

References

1. Duthie, J.R. & Nimmo, W.S. Adverse effects of opioid analgesic drugs. *Br J Anaesth* 1987, **59**: 61–77.
2. Jaffe, J.H. & Martin, W.R. Opioid analgesics and antagonists. In: Gilman, A.G., Goodman, L.S. & Gilman, A. (eds) *The Pharmacological Basis of Therapeutics*, 6th ed. Macmillan Publishing Co., Inc., New York, 1980, pp. 494–534.
3. White, I.D., Hokin, P.J., Hanks, G.W. & Bliss, J.M. Morphine and dryness of the mouth. *Br Med J* 1989, **298**: 1222–1223.
4. Di Flore, M.S.H. *Atlas of Human Histology*, 4th ed. Lea & Febiger, Philadelphia, 1978, pp. 106–107.

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.

M. Drah

R.R. Ghose

Singleton Hospital,

Sketty, Swansea SA2 8QA, UK.

References

1. Mellinkoff, S.M. & Tumulty, P.A. Hepatic hypoglycaemia. Its occurrence in congestive heart failure. *N Engl J Med* 1952, **247**: 745–750.
2. Benzing, G., Schubert, W., Hug, W. & Kaplan, S. Simultaneous hypoglycaemia and acute congestive heart failure. *Circulation* 1969, **XI**: 209–216.
3. Fischer, K.F., Lees, J.A. & Newman, J.H. Hypoglycaemia in hospitalized patients. Causes and outcomes. *N Engl J Med* 1986, **315**: 1245–1249.