

beclomethasone components of her aerosols but on the fluorinated hydrocarbons used as the propellant. Addiction to fluorocarbons, which are used in domestic aerosols, results in euphoria and intoxication.<sup>1</sup> Fluorocarbons may cause cardiac arrhythmias and electroencephalographic changes, and many deaths have been described.<sup>1</sup> The serum concentrations of fluorocarbons achieved when patients abuse their inhalers are not known. Not all fluorocarbons can be readily measured, different manufacturers use different propellants, and few studies have been performed. In one study inhalation of three 25 µl doses (115 mg of propellant) produced arterial blood concentrations of 1.7 mg of one of the fluorocarbon components.<sup>1,5</sup> Commonly used aerosol canisters deliver 63 µl doses containing about 85 mg of fluorocarbon, which would give higher serum concentrations. Our patient inhaled up to 400 doses of drug and fluorocarbon (34 g) at one time, and considerably raised blood concentrations must have occurred.

Less severe forms of abuse of inhalers may possibly be common, especially in younger age groups. If excessive use of aerosol treatment is noted dependence should be suspected, especially when the asthma is mild. Psychiatric intervention and the use of inhalation capsules may be helpful in preventing morbidity and mortality.

We thank Dr B Woods for referring this patient to us.

<sup>1</sup> Gluckman L. Ventolin psychosis. *NZ Med J* 1974;**80**:411.

<sup>2</sup> Edwards JG, Holgate ST. Dependency on salbutamol inhalers. *Br J Psychiatry* 1979;**134**:624-6.

<sup>3</sup> Pratt HF. Abuse of salbutamol inhalers in young people. *Clin Allergy* 1982;**12**:203-9.

<sup>4</sup> Bass M. Sudden sniffing death. *JAMA* 1970;**212**:2075-9.

<sup>5</sup> Dollery CT, Draffan GH, Davies DS, Williams FM. Blood concentrations in man of fluorinated hydrocarbons after inhalations of pressurised aerosols. *Lancet* 1970;ii:1164-6.

(Accepted 4 August 1983)

**Professorial Unit, Department of Medicine, Cardiothoracic Institute, London SW3 6HP**

P J THOMPSON, FRACP, clinical lecturer

**Brompton Hospital, London SW3 6HP**

P DHILLON, MD, MRCP, registrar

**Host Defence Unit, Department of Medicine, Cardiothoracic Institute, and Brompton Hospital, London SW3 6HP**

P COLE, BSC, FRCP, senior lecturer and honorary consultant physician

Correspondence to: Dr P J Thompson, Professorial Unit, Department of Medicine, Cardiothoracic Institute, Fulham Road, Brompton, London SW3 6HP.

## Does metabolic bone disease follow truncal vagotomy and gastrojejunostomy?

Up to 20% of patients who have undergone partial gastrectomy have been found to have metabolic bone disease 10 years later. Alleged causal factors are reduction of appetite, reduced intake of dairy food due to dumping, intolerance of milk, a feeling of fullness, malabsorption, raised concentrations of faecal fat, and diarrhoea. Postoperative reduction of acid decreases absorption of calcium. These effects may all be encountered after truncal vagotomy and gastrojejunostomy. Total bypass of the duodenum, where calcium and vitamin D are maximally absorbed, is not essential as metabolic bone disease has been described after partial gastrectomy with gastroduodenal anastomosis.<sup>1</sup> Theoretically, therefore, metabolic bone disease might be expected after truncal vagotomy and gastrojejunostomy. We studied a group of patients for metabolic bone disease eight to 15 years after they had undergone truncal vagotomy and gastrojejunostomy for chronic duodenal ulceration.

### Patients, methods, and results

In the course of a postoperative review<sup>2</sup> we studied 69 patients (54 men and 15 women; mean age 55.3 years) eight to 15 (mean 11.2) years after

truncal vagotomy and posterior gastrojejunostomy for chronic duodenal ulcer to assess the incidence of metabolic bone disease.

Corrected serum calcium concentrations and alkaline phosphatase activities were measured by autoanalyser, as were serum bilirubin concentrations and aspartate aminotransferase activity to exclude a hepatic cause of raised alkaline phosphatase activity. Bone mineral content was measured by photon absorptiometry with a Norland-Cameron 178 bone mineral analyser.<sup>3</sup> Reduced bone mineral content usually indicated osteoporosis, but if alkaline phosphatase activities were raised in addition osteomalacia was diagnosed. Faecal fat was measured over three days. A dietary survey was carried out over four days. Corrected serum calcium concentration was below 2.2 mmol/l (8.8 mg/100 ml) in two patients, and alkaline phosphatase activity was above the laboratory normal of 91 IU/l in three patients (above 120 IU/l in one), in all of whom tests of liver function and bone mineral concentrations were normal. In one patient the bone mineral content was more than 2 SD below the mean for the patient's age and sex (estimated from 114 volunteers); serum calcium concentration and alkaline phosphatase activity and a biopsy specimen of iliac crest bone were normal in this patient.

Mean intakes of energy, protein, fat, and carbohydrate were no different in patients with bone mineral concentrations more than 1 SD below the mean. Dietary intake of calcium was below 500 mg/day in one patient whose bone mineral content was normal. Dietary intake of vitamin D was below 2.5 µg/day in 35 patients (51% of the series) and below 1.25 µg/day in 12 of these (17%). Mean intake of vitamin D was 2.4 µg/day in patients with a bone mineral content more than 1 SD below the mean and 1.9 µg/day in the others. One patient with a raised alkaline phosphatase activity had a vitamin D intake of 1.9 µg/day. Symptoms and steatorrhoea after vagotomy were not related to abnormal biochemical values or reduced bone mineral content. Stress fractures did not occur.

### Comment

This study failed to show an increased incidence of metabolic bone disease eight to 15 years after truncal vagotomy and gastrojejunostomy. Although dietary intake of vitamin D was generally low, confirming previous studies,<sup>4</sup> there were no detectable metabolic consequences over this period. Inadequate intakes of energy and calcium were not shown, although they have been commonly found after gastrectomy and in association with bone disease after gastrectomy.

Osteomalacia after gastrectomy has been diagnosed in previous series by the finding of raised alkaline phosphatase activity in 10-20% of patients. Raised activities, however, are also found in patients with liver disease (excluded by biochemical tests in this series), patients who have not fasted, patients with blood group O (who are in excess in a population with duodenal ulcer), and patients with steatorrhoea, which is a common problem after gastric surgery. When histological examination of bone is not possible osteomalacia should be diagnosed only if raised alkaline phosphatase activities are found in association with reduced bone mineral content. Radiological methods of assessing demineralisation are poor, but photon absorptiometry is simple to carry out, is reproducible, and correlates with total body calcium and bone mineral content measured by ashing.<sup>5</sup> The one patient with low bone mineral content was statistically expected, and this patient volunteered to undergo biopsy of the iliac crest, which yielded normal results.

This series of 69 patients showed no clinical evidence of osteoporosis or osteomalacia 11 years after vagotomy and gastrojejunostomy.

<sup>1</sup> Harvald B, Krogsgaard AR, Lous P. Calcium deficiency following partial gastrectomy. *Acta Med Scand* 1962;**172**:497-503.

<sup>2</sup> Smith G, Irving AD. Age at operation and the results of truncal vagotomy and gastrojejunostomy for chronic duodenal ulcer. *Surg Gynecol Obstet* 1981;**152**:153-5.

<sup>3</sup> Cameron JR, Sorenson J. Measurement of bone mineral in vivo: an improved method. *Science* 1963;**142**:230-2.

<sup>4</sup> Morgan DB, Pulvertaft CN. Effect of vagotomy on bone metabolism. In: Alexander-Williams J, Cox AG, eds. *After vagotomy*. London: Butterworths, 1969:161-71.

<sup>5</sup> Chesnut CH, Manske E, Baylink D, et al. Correlation of total body calcium (bone mass) and regional bone mass in osteoporosis. *J Nucl Med* 1973;**14**:386.

(Accepted 18 August 1983)

**Department of Surgery, Ninewells Hospital and Medical School, Dundee DD1 9SY**

A D IRVING, MRCP, FRCS, lecturer in surgery

**University of Aberdeen, Aberdeen**

G SMITH, DSC, FRCS, formerly professor of surgery

G R D CATTO, MD, FRCP, senior lecturer in medicine

Correspondence to: Mr A D Irving.