footplate when the canvas heel loops were torn away and the protective plastic caps lost. Two wheelchairs had a brake mechanism missing on one wheel, and on two others the footplates had been tied with string because they were loose.

Faults found in 123 hospital wheelchairs

Fault	No (%) of wheelchairs	Fault	No (%) of wheelchairs
Defective pneumatic tyres	56 (46)*	Defective footrest plates	39 (32)
Soft	37	Missing	27
Punctured	16	Spikes	6
Leaking valve	2	Loose or stiff	6
Worn	1	Defective arms	14 (11)
Defective brakes	75 (61)	Dangerous	10
Torn seat or backrest	10 (8)	Missing	4
Defective steering	2 (2)		

*57% of the 98 wheelchairs with pneumatic tyres.

During the two months 11 accidents related to wheelchairs were reported. In five of these a defective wheelchair was partly or completely responsible for the patient's injury. One patient received a laceration to her hand from an exposed bolt on the armrest, and another sustained a large shin laceration when a loose footrest plate dropped down during transfer. Three patients fell while getting up from wheelchairs with defective brakes.

Comment

The hospital wheelchairs inspected in this study were in an unsatisfactory state. Many were unsafe, and some resulted in injury to the patient. Only 28 (23%) were safe and in good working order. The most common problems—defective brakes, soft or punctured tyres, and absent or defective footrest plates—were simple faults that could easily have been corrected. Although the danger of exposed spindles on the footrest plate was specifically highlighted in a circular from the Department of Health and Social Security (safety information bulletin No 14, 1984), six wheelchairs had exposed spindles.

The results of this study support the suggestion of a recent report that hospitals should organise regular inspection and maintenance of wheelchairs.² Most hospital staff have no special knowledge of problems connected with wheelchairs, and better instruction and information about wheelchairs and their use are required.

At present many hospital wheelchairs are in a poor state of repair; this neglect should not continue.

We thank Miss D Wood for help in preparing the manuscript.

1 Able EW. Survey of attendant-propelled mobile chairs used in hospitals. *Health Bull (Edinb)* 1983;41:275-7.

2 Working Group of the Prosthetics and Orthotics Committee. *The wheelchair service in Scotland*. Edinburgh: HMSO, 1983.

(Accepted 13 August 1985)

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Macroamylasaemia: how common is it?

In macroamylasaemia a macromolecular complex circulates in the plasma and usually causes benign hyperamylasaemia. Possibly these findings may be interpreted incorrectly as due to acute pancreatitis. We therefore studied the extent of unrecognised problems arising from macroamylasaemia in a British teaching hospital.

Present series

Serum specimens from all 74 patients with hyperamylasaemia seen over three months were assayed by a recently described simple polyethylene glycol

precipitation test.¹ Two patients were found to have macroamylasaemia. This was confirmed by gel filtration chromatography, which distinguishes the macromolecular complex from the usual molecular mass amylase; urinary amylase activity assay, because low activity in the presence of high serum activity suggests low renal clearance if glomerular function is not impaired; and serum lipase activity assay, because a high serum amylase activity without raised lipase activity suggests that the former is not due to pancreatic damage.

Case 1—A 62 year old man with severe abdominal pain and jaundice was admitted and diagnosed clinically as having acute cholecystitis. Serum bilirubin concentration was 55 $\mu\text{mol/l}$ (3.2 mg/100 ml; reference range 0-17 $\mu\text{mol/l}$; 0-10 mg/100 ml), alkaline phosphatase activity 190 U/l (reference range 20-120 U/l), and γ -glutamyltransferase activity 145 U/l (reference range 7-42 U/l). Investigations showed a serum amylase activity of 5772 U/l (reference range 70-300 U/l) and acute pancreatitis was therefore added to the diagnosis. Pancreatic ultrasonography was performed twice but the pancreas was poorly visualised; there were no abnormalities. Intravenous cholangiography showed a small stone in the common bile duct. At laparotomy a perforated gall bladder was found and cholecystectomy performed. Serum amylase activity fell to within the reference range after massive blood loss and transfusion but later rose again to between 3920 and (on discharge) 7590 U/l. This was considered to be worthy of suspicion but no further biochemical investigations were initiated. The results suggested a pancreatic pseudocyst but two further ultrasound studies failed to confirm this. A serum specimen was submitted for assessment of amylase activity during our study when the patient was seen three months after discharge; macroamylasaemia was found and confirmed by gel filtration, urinary amylase activities of 114 and 228 U/l (reference range 800-2000 U/l), and serum lipase activity of 75 U/l (reference range up to 190 U/l). Renal impairment was not evident—serum urea concentration 5.7 mmol/l (34.3 mg/100 ml; reference range 3.3-6.6 mmol/l; 19.9-39.8 mg/100 ml), creatinine 110 $\mu\text{mol/l}$ (1.2 mg/100 ml; reference range 44-150 $\mu\text{mol/l}$; 0.5-1.7 mg/100 ml).

Case 2—A 65 year old woman presented with a three day history of persistent, severe abdominal pain radiating across the abdomen, urinary and faecal incontinence, nausea, and haematemesis. She had had angina pectoris since 1970 and two episodes of myocardial infarction, the most recent in 1981, complicated by axillary vein thrombosis. She also had congestive cardiac failure and chronic respiratory disease. Serum amylase activity assay was requested and was 564 U/l. A clinical diagnosis of ischaemic colitis or mesenteric infarction was made. At laparotomy extensive small bowel infarction due to a superior mesenteric artery embolus was discovered and partial bowel resection undertaken. Her stormy postoperative course in the intensive care unit was complicated by aspiration pneumonia. Macroamylasaemia was found by us during her stay in the unit and confirmed by gel filtration and, early during hospitalisation, urinary amylase activities of 58 and 89 U/l and a serum lipase activity of 17 U/l; serum urea concentration was 18.5 mmol/l (111.4 mg/100 ml) and creatinine 196 $\mu\text{mol/l}$ (2.2 mg/100 ml). Serum amylase activity remained high and was 800 U/l on discharge after 13 days.

Comment

Review of case 1 showed that the abdominal pain was due to acute cholecystitis and obstruction of the common bile duct which culminated in gall bladder perforation. Initial treatment may have been delayed unnecessarily, since the clinical diagnosis appeared to us to have been modified after finding a raised serum amylase activity. Macroamylasaemia did not cause confusion in case 2, since the symptoms led to a clinical diagnosis. Serum amylase activity was, however, determined to exclude pancreatitis; this was certainly included in the differential diagnosis.

It is well known but not always remembered that hyperamylasaemia is not diagnostic of pancreatitis.² Macroamylasaemia, in particular, is an important potential source of diagnostic confusion.

In the short term we suggest that the polyethylene glycol precipitation test for macroamylase should be introduced by hospital service laboratories. We agree with recent reports that efforts should be made to replace amylase activity assays with alternative methods such as assay of lipase activity.³ Macroamylasaemia would thus be eliminated as a cause of diagnostic confusion.

We thank Mr M G Walker and Mr W F Walker for permission to study their patients.

1 Levitt MD, Ellis C. A rapid and simple assay to determine if macroamylase is the cause of hyperamylasaemia. *Gastroenterology* 1982;83:378-82.

2 Salt WB, Schenker S. Amylase—its clinical significance: a review of the literature. *Medicine (Baltimore)* 1976;55:269-89.

3 Eckfeldt JH, Kolars JC, Elson MK, Shafer RB, Levitt MD. Serum tests for pancreatitis in patients with abdominal pain. *Arch Pathol Lab Med* 1985;109:316-9.

(Accepted 19 August 1985)

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