completely healthy, without neurological symptoms or Kayser-Fleischer ring, and liver test results were normal. Serum caeruloplasmin¹ was slightly decreased at 95 mg/l (normal value in our laboratory 130-270 mg/l), and urine copper excretion was raised at 1.27 $\mu\text{mol}/24\text{h}$ (81 $\mu\text{g}/24\text{h}$) (normal value 0.78 $\mu\text{mol}/24\text{h}$ (50 $\mu\text{g}/24\text{h}$)). After a diagnostic test with ⁶⁴Cu² a preclinical stage of Wilson's disease was diagnosed.

He was given D-penicillamine 1 g/day. No side effects were observed during the first 13 months. In May 1973 he developed weakness of the right ocular muscles; in particular, ptosis was more pronounced at the end of the day. During the next 10 days the weakness became worse and left ptosis developed; no other muscles showed weakness. After edrophonium chloride 10 mg intravenously rapid opening of both eyes was noted. Electromyography and immunological investigations were not performed. Penicillamine was withdrawn and he was given neostigmine. After six weeks he showed no symptoms of myasthenia.

Eight months later he still had no neurological symptoms. Laboratory investigations confirmed the diagnosis of Wilson's disease. An electromyogram and a laminogram of the mediastinum were normal. Though weak antibodies to smooth muscles were detected in the serum by indirect immunofluorescence no antinuclear antibodies were found.

The patient was again given D-penicillamine 0.75 g/day. During the next 10 months he had no side effects. Antibodies to smooth muscles were detected in May and June 1974 but not in November 1974.

The myasthenic syndrome which developed in this patient was probably induced by penicillamine, just as other autoimmune syndromes have occurred after long-term treatment with this drug.^{3,4}

¹ Ravin, H. A., *Journal of Laboratory and Clinical Medicine*, 1961, 58, 161.

² Czlonkowska, A., et al., *Acta Universitatis Carolinae*, 1973, 56/57, 175.

³ Boudin, G., Pepin, B., and Maqueneau, M., *Nouvelle Presse Médicale*, 1974, 3, 171.

⁴ Walshe, J. M., *Postgraduate Medical Journal*, 1968, 44, Suppl. p. 6.

Department of Genetics, Psychoneurological Institute, Warsaw, Poland

ANNA CZŁONKOWSKA, M.D., Associate Professor

Duodenal Obstruction due to Abdominal Aortic Aneurysm

Patients frequently present with symptoms of upper gastrointestinal tract disease, and these sometimes suggest an obstructing lesion. Such symptoms are commonly associated with either scarring secondary to chronic peptic ulcer or hold-up due to gastric antral carcinoma. A rare cause of gastroduodenal obstruction is extrinsic pressure from abdominal aortic aneurysm.¹ We describe a case and discuss previous reports.

Case History

An 80-year-old man was admitted to hospital with a four-week history of bilious vomiting, epigastric swelling, and vague upper abdominal discomfort. The vomiting had occurred only at night. A duodenal ulcer had been treated medically. He was thin and dehydrated with no clinical signs of anaemia or jaundice. The blood pressure was 140/90. A 12 x 6 cm smooth, slightly tender, pulsatile mass was palpable in the epigastric region of the abdomen. Investigations showed haemoglobin 17.2 g/dl, blood urea 31.5 mmol/l (190 mg/100 ml), serum sodium 133 mmol/l, serum potassium 3.1 mmol/l, serum bicarbonate 39 mmol/l. A plain x-ray film of the abdomen showed a soft tissue mass in the upper abdomen and a calcified aortic aneurysm on the lateral view. A gastrograffin meal showed that the body of the stomach was raised over a large aneurysm. The proximal duodenum was dilated as far as the distal second part. On turning the patient on to his side pressure became apparent mainly at the duodenojejunal flexure.

Intravenous fluids were started, and his electrolytes reverted to normal, the blood urea falling to 5.8 mmol/l (35 mg/100 ml). At laparotomy the presence of an aortic aneurysm, 6 cm wide, was confirmed. In view of his age and relatively frail condition radical resection was rejected in favour of anterior gastrojejunostomy. Postoperative recovery was uneventful with complete relief of symptoms.

Discussion

Osler² first described duodenal obstruction as a feature of abdominal aortic aneurysm. There have been seven similar cases.³ These patients invariably complained of nausea, vomiting, and abdominal pain. Five of the patients admitted to weight loss and several were emaciated. The duration of symptoms was variable, ranging from days or weeks to 11 years. An abdominal mass was palpable. Duodenal obstruction

was confirmed in all but two cases by contrast radiology. The site of obstruction was the third part of the duodenum except for one patient where there was hold-up at the pylorus. Two patients had symptoms severe enough to cause electrolyte imbalance due to vomiting. In another a phytobezoar was found at necropsy in the obstructed duodenum.⁴ Four patients were treated by simple bypass: posterior gastrojejunostomy in two, anterior gastrojejunostomy in one, and isoperistaltic duodenojejunostomy in the fourth case. One patient underwent resection of his aneurysm but died ten hours later.⁵ A further patient died before surgery could be performed.⁴ Two survived bypass procedures and their symptoms were relieved.

Our patient conformed to the usual pattern. Interestingly, vomiting occurred only at night but was sufficiently severe to cause dehydration, alkalosis, and hypokalaemia. He was treated by anterior gastrojejunostomy and this was very successful. It had also proved effective in two previous cases. The only patient to undergo radical surgery died immediately afterwards. A palliative bypass procedure thus appears to be satisfactory and the surgeon should not be tempted to perform major surgery if the patient's fitness is doubtful.

I should like to thank Mr. J. Bla'ne and Dr. R. Courtenay-Evans for permission to report this case and for their advice in preparation of the manuscript.

¹ Eastcott, H. H. G., *Arterial Surgery*, 2nd edn. London, Pitman Medical, 1973.

² Osler, W., *Lancet*, 1905, 2, 1089.

³ Sondheimer, F. K., and Steinberg, C. L., *American Journal of Roentgenology*, 1964, 92, 1110.

⁴ Panoro, V. A., and Melzer, M. J., *New York Journal of Medicine*, 1970, 70, 673.

⁵ Desiderio, V. C., Kownacki, V., and Wilson, W. L., *Gastroenterology*, 1959, 37, 766.

Mayday Hospital, Mayday Road, Thornton Heath, Surrey, CR4 7YE
H. M. ADAIR, F.R.C.S., Surgical Registrar

Side Effects due to Treatment of Hypertension with Prazosin

Prazosin is a new antihypertensive agent which has recently become available. Its antihypertensive action is said to be due to phosphodiesterase inhibition in arteriolar smooth muscle. Early reports¹ indicated that postural hypotension should not be a major problem, and only a few patients had had other side effects. This apparent freedom from side effects would make it very useful, particularly in elderly patients. We have used this drug in a total of eight patients, two (nos 1 and 2) in hospital, and six in general practice. Both hospital cases and five others developed severe side effects after starting Prazosin.

Case Histories

All the patients had normal renal function on the basis of blood urea concentrations. The table summarises the relevant details for each patient. Severe hypotension occurred in patients 1 and 7, and symptoms of postural hypotension in patients 2 and 6. Severe recorded tachycardia occurred in patients 3 and 7, and patient 5 experienced palpitations. Other symptoms were weakness, light headedness, dizziness, and diarrhoea.

Discussion

We believe, from our experience with relatively few patients, that side effects due to Prazosin may be more common and on occasions much more severe than previously reported. Other reports of side effects have led to the Committee on Safety of Medicines to issue an Adverse Reaction Notice.² In view of the combination of hypotension and appreciable vasodilatation in patient 1 we postulate that hypotension is due to rapid onset of alpha-receptor blockade. It is now recommended that the first dose of Prazosin should be given just before the patient goes to bed at night. We think that the severity of hypotension can be so great that even this method of starting therapy could be

Details of Patients and their Treatment

Patients No.	Age	Sex	B.P. (mm Hg)	Previous Treatment of Hypertension	Dose of Prazosin	Side Effects
1	79	F	200/120 L&S	No Treatment	2 mg (only 1 dose taken)	Collapsed 45 min after first dose, B.P. 80/mm Hg. Severe peripheral vasodilatation with cherry-red skin. Given I.V. Hydrocortisone and oxygen, recovered over 1 hour. No evidence of myocardial infarction.
2	77	M	220/120 L&S	No Treatment	2 mg (only 1 dose taken)	30 min after first tablet, could not stand up without feeling faint. No evidence of myocardial infarction. No further Prazosin given.
3	65	F	210/115 L 180/110 S	Methyldopa	Increasing dose regime—0.5 mg a.m., 1 mg midday, 2 mg evening, then 2 mg t.d.s.	30 min after first 2 mg dose had severe palpitations, which recurred with each subsequent dose. Also lack of energy, headaches and muzziness. After 5 days no change in B.P., tachycardia 124/min regular. Prazosin stopped—all symptoms rapidly disappeared.
4	51	M	180/120 L&S	No Treatment	2 mg t.d.s.	Rapid onset of lassitude, weakness, and diarrhoea. Became bed-ridden. Persisted for nine days until Prazosin discontinued. No recurrence since then.
5	43	F	180/120 L&S	No Treatment	2 mg t.d.s.	2 months after starting B.P. 180/115 mm Hg. lying and standing—dose increased to 5 mg t.d.s.—became sleepy, unable to drive, had palpitations. Dose decreased to 2.5 mg t.d.s. and Nephrol one daily added—still feels tired on this dose.
6	57	F	190/120 L&S	Methyldopa Bethandine Propranolol	2 mg t.d.s. (added to Propranolol 40 mg q.d.s.)	Within few hours of starting drug experienced light-headedness, feelings of unreality and dizziness. She stopped of her own accord. B.P. now controlled with clonidine alone.
7	48	M	190/110 L&S	Methyldopa	2 mg (only 2 doses taken 2 weeks apart).	30 min after first tablet—palpitations, pulse 180/per min. 2 weeks later took 1 more tablet—tachycardia 150/per min B. P. 60/0 mm Hg. Controlled now on clonidine.

L = lying blood pressure. S = standing blood pressure.

dangerous—particularly in elderly patients who may have coexisting cerebral arteriosclerosis. In some patients successive doses of the drug produce the same side effects as the initial dose and tolerance does not develop.

We conclude that the recommended starting dose of 2 mg is too high and that further investigations are needed to establish a safe starting dose and to discover which patients are most likely to suffer side effects.

¹ Prazosin—Evaluation of a New Anti-hypertensive Agent, Proceedings of a symposium held at the Interprofessional Centre, Geneva, 8 March 1974. Amsterdam, Excerpta Medica, 1974.

² Prazosin and Loss of Consciousness—Adverse Reaction, Series No. 12. London, Committee on Safety of Medicines, 1975.

Department of Geriatric Medicine, St. Mary's Hospital, Colchester, Essex

M. J. BENDALL, M.B., M.R.C.P., Consultant Physician

K. H. BALOCH, M.B., B.S., Clinical Assistant

P. R. WILSON, B.M., M.R.C.P., Consultant Physician

Unusual Foreign Body in the Ear

The wearing of a sprung wire collar or "choker" with hooked ends acting as a fastener (see fig.) has become fashionable in adolescents. Three patients have been seen with a hooked end caught in the ear. The three chokers were machine-cut and the ends were sharp with protruding slivers of metal, which increased the liability of the hook to penetrate the meatal skin. The danger could be greatly reduced if these ends were smooth and, preferably, bulbous.

Case Reports

Case 1.—This patient, aged 15, while putting on his choker accidentally let it slip into his left external auditory canal. He failed to remove it because of pain and bleeding. The choker was hanging from his left ear, and after microsuction the end was seen penetrating the skin of the superficial meatus, preventing withdrawal. It was pushed further into the canal and withdrawn by pressing it firmly against the opposite canal wall to prevent reimpaction. The meatal wall was excoriated but the tympanic membrane was intact and the hearing normal.

Case 2.—A 15-year-old boy was on an aircraft when his choker slipped and one end sprang into his right ear. Attempted removal caused pain and bleeding but he stopped trying only when a severe persistent pain developed in the ear. Four hours later he reached a local casualty department, where an x-ray picture showed that the choker was deep in the external canal at the level of the tympanic membrane. Pressure on this had probably caused the persistent pain. The hook was impacted under the skin and was removed under general anaesthesia. The canal was badly excoriated but the tympanic membrane was intact and the hearing normal.

A metal "choker" with hooked ends for fastening.

Case 3.—This patient, a girl aged 12, got the hook of the choker stuck in her external auditory canal when pretending it was a stethoscope. It could not be removed because of pain. Her ear was full of blood and she would not tolerate microsuction. An x-ray picture showed the position of the hook, which was then removed by manipulation. The canal skin was lacerated but hearing and the tympanic membrane were normal.

Conclusion

These patients suffered extreme discomfort due to the presence of the choker in the external auditory canal. Fortunately none incurred damage to the tympanic membrane or ossicles, though these structures are at risk with any foreign body in the ear.

I thank Mr. D. Kilby for permission to report details of his patients.

University Department of Otolaryngology, Royal Victoria Infirmary, Newcastle upon Tyne NE1 4LP

N. S. LONGRIDGE, F.R.C.S., M.R.C.P., Registrar