tenderness. The serum amylase was 4,500 Somogyi units and the blood urea was 36 mg/100 ml. She was treated with intravenous fluids and antibiotics. Three days after admission her urine output began to fall. Her serum calcium fell to 7.6 mg/100 ml. After 10 days her blood urea was 346 mg/100 ml, and intermittent haemodialysis was begun. Laparotomy next day confirmed the diagnosis of pancreatitis. Intermittent haemodialysis was maintained for the next two weeks, when a diuresis occurred. She then made a rapid recovery and was discharged from hospital seven weeks after admission. She was readmitted four months later with a mild attack of pancreatitis, and eight months after her first illness she was readmitted for a second time when a laparotomy and cholecystectomy were performed. The gall bladder contained multiple faceted stones. She remained well one year later.

Case 3

A 60-year-old training officer was admitted with central abdominal pain and vomiting. There was abdominal tenderness and guarding. The serum amylase was 7,100 Somogyi units and the blood urea was 45 mg/100 ml. He was treated with analgesics, antispasmodics, antibiotics, and intravenous fluids. He remained well until two days after admission, when he became confused. His blood urea was 101 mg/100 ml, amylase 4,500 Somogyi units, and serum calcium 5.3 mg/100 ml. He was treated with additional intravenous calcium. Four days after admission he was very ill and confused, with bilateral pleural effusions, a soft silent abdomen, and a strongly positive Grey-Turner's sign in both flanks. The blood urea was 193 mg/100 ml, calcium 6.0 mg/100 ml, amylase 1,300 Somogyi units, and the white cell count was persistently high. Peritoneal dialysis was begun and continued for the next 10 days. He was also treated with intravenous glucagon (Knight *et al.*, 1971). Despite dialysis his blood urea rose to 365 mg/100 ml on the next day, and he was comatose and unrousable. He was passing altered blood per rectum and fresh blood was aspirated from the nasogastric tube. He was treated with blood transfusion. The blood sugar rose to 800 mg/100 ml and small doses of insulin were given. He developed an *Escherichia coli* bronchopneumonia, and was given ampicillin, kanamycin, and oxygen.

He remained in this grave condition for the next three days and then began to show slow improvement. Two weeks later he managed to take a little food. One month after admission he developed a swinging pyrexia, and an abdominal mass was felt. Barium-meal examination showed a swelling 12 cm in diameter in the head of the pancreas. At laparotomy the mass was found to be a pseudocyst which was drained into the stomach. After the operation he made a slow but steady recovery. He was discharged after more than two months in hospital. He remained well nine months later.

Discussion

The aetiology of acute renal failure in acute pancreatitis is uncertain. It is often held to be due to a fall in blood pressure and in circulating blood volume due to loss of fluid into the retroperitoneum. In dogs up to 30% of the blood volume may be lost into the retroperitoneum in an experimental acute pancreatitis (Keith and Watman, 1954). Careful scrutiny of notes and blood pressure charts, however, has failed to show any appreciable changes in blood pressure preceding most of the cases of acute renal failure that we have seen. Possibly toxic products of pancreatic autodigestion are responsible for renal damage. Vasoactive substances are found in the pancreas (Hollenberg *et al.*, 1962) and these might disturb the regulatory mechanisms of the renal circulation.

The present cases show that vigorous treatment of acute renal failure is needed when it occurs in acute pancreatitis. Haemodialysis (Cases 1 and 2) is more efficient and comfortable for the patient but there are risks of disequilibrium in such patients with severe electrolyte imbalance, and heparin must be used very cautiously in patients with acute intra-abdominal inflammation. Peritoneal dialysis has the advantage of relative safety, and the action of peritoneal lavage may be beneficial to the pancreatic inflammatory process. Peritoneal dialysis has been advocated as a treatment for uncomplicated acute pancreatitis (Wall, 1965; Bolooki and Gliedman, 1968).

Prevention of acute renal failure in pancreatitis must rely on scrupulous attention to fluid balance and hydration throughout the course of the disease. Further elucidation of the cause of the renal lesion is necessary for more effective prevention.

We would like to thank Mr. W. A. B. Smellie for permission to report Case 2. We would also like to thank Professor M. D. Milne and Mr. Rodney Smith for their help in the care of Case 1, and Dr. D. B. Evans for his help in the care of cases 2 and 3.

References

- Bolooki, H., and Gliedman, R. L. (1968). *Surgery*, 64, 466.
- Hollenberg, R. M., Kobold, E. E., Pruett, R., and Thal, A. P. (1962). *Surgical Forum. Proceedings of the Forum Sessions, Clinical Congress of the American College of Surgeons*, 13, 302.
- Howard, J. M., and Jordan, G. L. (1960). *Surgical Diseases of the Pancreas*. Philadelphia, Lippincott.
- Keith, L. M., and Watman, R. N. (1954). *Surgical Forum, Proceedings of the Forum Sessions, Clinical Congress of the American College of Surgeons*, 5, 380.
- Knight, E. J., Condon, J. R., and Smith, R. (1971). *British Medical Journal*, 2, 440.
- Trappnell, J. E. (1971). *Annals of the Royal College of Surgeons of England*, 49, 361.
- Wall, A. J. (1965). *Medical Journal of Australia*, 2, 281.

MEDICAL MEMORANDA

Transient Ischaemic Attacks and Stroke due to Extracranial Aneurysm of Internal Carotid Artery

H. G. BODDIE

British Medical Journal, 1972, 3, 802-803

Aneurysms of the extracranial segment of the internal carotid artery are rare and usually present to the otorhinolaryngologist or vascular surgeon as masses in the peritonsillar or cervical regions respectively.

North Staffordshire Hospital Centre, Stoke-on-Trent, Staffs

H. G. BODDIE, M.R.C.P., Registrar, Department of Neurology (Present address: Muscular Dystrophy Research Laboratory, Newcastle General Hospital, Newcastle upon Tyne)

The following case is unusual in that not only were transient ischaemic attacks culminating in a stroke the mode of presentation, but they occurred in a young man.

Case Report

A 24-year-old man was admitted to hospital on 2 September 1970 after the sudden onset of a left hemiparesis. For four months from August 1969 he had experienced several transient ischaemic attacks of weakness and paraesthesiae affecting the left upper limb. These were similar, being sudden in onset, associated with right-sided temporal headaches, and followed by complete recovery within an hour. In December 1969 he was admitted to another hospital for investigation of a left hemiparesis of sudden onset. Examination showed a left visual field defect, left facial weakness, and a left hemiparesis and sensory impairment. Investigations, which were repeated on this admission and are described below, suggested that his symptoms and signs were due to recurrent emboli from the right internal carotid artery. He refused surgery

and was treated with anticoagulants. He made a rapid recovery and when seen as an outpatient in March 1970 no abnormal neurological signs were present. Anticoagulant therapy was discontinued. He remained well until the present admission apart from a similar attack in May, which, however, lasted four hours before recovery.

General examination showed nothing abnormal. An indistinct mass was palpable on the right side of the neck, but both carotid arteries were palpable and pulsatile and no bruits were heard. In the central nervous system the abnormal physical signs were similar to those found in December 1969 except that no visual field defect was present.

Investigations.—Full blood count, urea, electrolytes, cholesterol, plasma proteins, W.R. and Kahn test, and urine analysis showed

Right common carotid arteriogram showing distortion and aneurysmal dilatation of internal carotid artery with atheromatous changes proximally.

nothing abnormal. X-ray pictures of chest and skull were normal, but the cervical spine showed a calcified mass on the right side of the neck opposite C1 and C2. Bilateral common carotid arteriography showed the left side to be normal but on the right side (see photograph) there was irregular dilatation of the origin of the internal carotid artery with a prominent posterior kink. Atheromatous changes were present in the proximal portion. The artery was displaced medially and ventrally by the calcified mass, which appeared to adhere to the artery in its lower part. A 99-Tc brain scan (2 September 1970) was normal but when repeated four days later showed a peripheral lesion in the right frontal area. Serial electroencephalography showed bilateral abnormality but was consistent with extensive ischaemia of the left hemisphere.

A clinical diagnosis of recurrent cerebral emboli secondary to aneurysmal dilatation of the internal carotid artery was made. On 15 September Mr. J. Lawson explored the neck and removed a large, fusiform aneurysm of the right internal carotid artery; an end-to-end anastomosis was performed using a vein graft.

The aneurysm started 1 cm above the bifurcation and extended almost to the base of the skull. The operation was carried out under hypercapnia and total occlusion time was 64 minutes. Recovery was uneventful. Histologically the aneurysm was atheromatous. When seen as an outpatient six weeks later the patient's neurological deficit persisted.

Comment

Alexander *et al.* (1966) reviewing the 155 cases of extra-cranial aneurysms of the internal carotid artery reported to that date, found only eight cases which presented with neurological deficit secondary to cerebral ischaemia. Since then a further seven cases have been reported (Ambler *et al.*, 1967; Kianouri, 1967; McEntyre *et al.*, 1967; Webb and Barker, 1969; Gros *et al.*, 1970; Cifarelli and Sadiq, 1971).

Including the present case, six of the 16 presented with transient ischaemic attacks, in three of which a stroke occurred with persistent neurological deficit despite anticoagulant therapy. In two others the symptoms were sensory only, and in the remaining case there was recurrent visual impairment due to retinal emboli. Neurological symptoms and signs in the other cases were hemiparesis, coma, dizzy spells, monoplegia, Horner's syndrome, and blurred vision. A palpable mass in the neck was present in five cases and an audible bruit in only three.

Carotid arteriography showed the aneurysm in all 15 cases. Twelve patients underwent surgery, either excision of the aneurysm with an end-to-end anastomosis (eight cases) or proximal ligation of the internal carotid artery (four cases).

Before the introduction of antibiotics the underlying aetiology was usually syphilis or contiguous infection. Today most cases are due to atherosclerosis or trauma.

In two cases the aneurysms were bilateral and the underlying aetiology was thought to be congenital (Alexander *et al.*, 1966; Cifarelli and Sadiq, 1971). An unusual feature about the present case was that typical histological features of atherosclerosis were seen though the patient was aged 24 and had been previously healthy. The cause of the transient ischaemic attacks in most cases was thought to be due to emboli from atheromatous plaques in the internal carotid artery adjacent to the aneurysmal dilatation, and the emboli were seen in one case (McEntyre *et al.*, 1967).

Although rare, early recognition before permanent neurological deficit intervenes is desirable since surgery is curative.

I wish to thank Dr. E. C. Hutchinson for his helpful advice in the preparation of this report.

References

- Alexander, E., jun., Wigser, S. M., and Davis, C. H. (1966). *Journal of Neurosurgery*, 25, 437.
- Ambler, J., Ulc, M., and Ledinsky, Q. (1967). *Neurochirurgia*, 10, 169.
- Cifarelli, F., and Sadiq, S. (1971). *Archives of Surgery*, 102, 74.
- Gros, C., *et al.* (1970). *Neurochirurgie*, 16, 367.
- Kianouri, M. (1967). *Annals of Surgery*, 165, 152.
- McEntyre, J. H., Keates, E. U., and Whiteley, W. H. (1967). *Archives of Ophthalmology*, 77, 317.
- Webb, R. C., jun., and Barker, W. F. (1969). *Archives of Surgery*, 99, 501.