the estimation was performed. Jarnum and Jensen (1966) demonstrated protein-losing enteropathy in their patient.

Five of the reported cases have had a fatal outcome (Cronkhite and Canada, 1955; Martini and Dölle, 1961; Johnston, Vosburgh, Wiens, and Walsh, 1962; Jarnum and Jensen, 1966), usually within 18 months of the onset of the diarrhoea. Spontaneous clinical and biochemical remissions occurred in one patient (Kennedy and Hirson, 1961). Two were improved by removal of the polypi—one by a gastrectomy (Manousus and Webster, 1966) and the other by a hemicolectomy (Zdansky and Riederer, 1963).

Jejunal diverticulosis and pancreatic disease have not been previously reported in this condition and it is possible that these may be part of the syndrome.

Jejunal diverticulosis is a well-recognized cause of intestinal malabsorption. Booth, Babouris, Hanna, and MacIntyre (1963) drew attention to the not uncommon finding of hypomagnesaemia, which was present in 15 out of 42 cases of intestinal malabsorption of various causes reported in their series. An interesting feature in the present case is the presentation with paraesthesiae and convulsions due to hypocalcaemia and hypomagnesaemia, and the later development of more generalized malabsorption despite symptomatic improvement and weight

gain after treatment with oral supplements and broad-spectrum antibiotics.

We wish to thank Professor G. Smart, under whose care the patient was admitted, and Professor Sam Shuster for their advice and constructive criticism.

W. J. CUNLIFFE, M.B., B.Sc., M.R.C.P.,
Registrar in Dermatology.

J. ANDERSON, M.B., M.R.C.P.,

First Assistant to the Department of Medicine

University of Newcastle upon Tyne and the
Royal Victoria Infirmary, Newcastle upon Tyne.

REFERENCES

- Booth, C. C., Babouris, N., Hanna, S., and MacIntyre, I. (1963). *Brit. med. J.*, **2**, 141.
Cronkhite, L. W., and Canada, W. J. (1955). *New Engl. J. Med.*, **252**, 1011.
Jarnum, S., and Jensen, H. (1966). *Gastroenterology*, **50**, 107.
Johnston, M. M., Vosburgh, J. W., Wiens, A. T., and Walsh, G. C. (1962). *Ann. intern. Med.*, **56**, 935.
Kennedy, J. A., and Hirson, C. (1961). *Proc. roy. Soc. Med.*, **54**, 234.
Martini, G. A., and Dölle, W. (1961). *Dtsch. med. Wschr.*, **86**, 2524.
Manousus, O., and Webster, C. U. (1966). *Gut*, **7**, 375.
Samman, P. D. (1961). *Arch. Derm.*, **84**, 1030.
Shuster, S. (1967). In Proceedings of Consultant Conference, Royal College of Physicians, London. In press.
Zdansky, E., and Riederer, J. (1963). *Radiol. clin. (Basel)*, **32**, 254.

Haemoperitoneum Associated with a Ruptured Gall Bladder

Brit. med. J., 1967, **4**, 602

Leared (1859) was the first to recognize that the gall bladder could be the source of catastrophic intra-abdominal haemorrhage. He described finding a ruptured gall bladder and 2 quarts (2.3 litres) of blood in the abdominal cavity of a man of 22 at a post-mortem examination. Since then the condition has been reported infrequently. Raycroft and Mastrangelo (1960) were able to collect only 18 cases from the world literature.

CASE REPORT

A man of 55 was admitted to hospital on 26 January 1966. His admission was precipitated by 48 hours of severe generalized abdominal pain accompanied by abdominal distension and constipation. He gave a long history of dyspepsia—30 years previously he had had a haematemesis and since then had been subject to periodic bouts of epigastric pain relieved by food. During the year before admission he had had attacks of pain in the right lower chest and for two months had also had central abdominal colicky pains and increasing distension. A barium-meal examination shortly before admission had failed to show a cause for his dyspepsia, and six days before his admission his haemoglobin was 106%.

Examination showed an obviously ill man. He was pale and clammy and had a pulse rate of 120. The abdomen was distended and though tender all over it was particularly so in the right hypochondrium. Tenderness was also elicited on rectal examination. On auscultation bowel sounds were present. Radiographs of the abdomen showed gaseous distension of both the small and large bowel. The serum amylase was 89 Somogyi units and the haemoglobin 66%.

After preliminary intravenous fluid replacement a laparotomy was performed. One and a half litres of blood and blood clot were found in the abdomen. This clearly arose from the gall bladder, which had a 2-cm. tear across Hartmann's pouch. The cystic artery was severed and blood was continuing to spurt from the proximal end. The gall bladder contained blood clot, but both it and the common bile duct appeared to be normal otherwise. No evidence of peptic ulceration was found. Cholecystectomy was performed

and the blood was removed from the abdominal cavity. Histological examination of the gall bladder showed evidence of chronic cholecystitis. The patient's postoperative progress was entirely uneventful.

COMMENT

Severe bleeding from the biliary tract is uncommon. During recent years, however, it has been shown that in a small proportion of patients with gastrointestinal haemorrhage the biliary tract is the source of the bleeding. The diagnosis has usually been made only after repeated haemorrhages have occurred and after previous gastric surgery has been undertaken (Fitzpatrick, 1961). Haemobilia has been recorded from very varied pathological causes. Minor bleeding is not uncommon in calculous cholecystitis, especially when accompanied by stones in the common bile duct, but cholecystitis appears to be the least common cause of severe bleeding. Mailer (1939), commenting on a fatal case of spontaneous rupture of the gall bladder with intraperitoneal haemorrhage, stressed the lack of inflammation in the gall bladder. Though the gall bladder in the present case showed histological evidence of chronic cholecystitis it was not the site of acute inflammation or of ischaemic necrosis. It is therefore by no means clear why the gall bladder should have ruptured, but it does seem likely that the cause was essentially a mechanical one. If the bleeding was caused by rupture of the gall bladder, the cause of the rupture remains unexplained. It is therefore thought more likely that bleeding occurred into the gall bladder first. Blood clot may have obstructed the neck of the gall bladder, leading to a rise of tension sufficient to bring about its rupture.

I am grateful to Mr. I. W. Matheson for permission to publish this case.

PAUL ATKINS, M.B., B.S., F.R.C.S.,
Senior Registrar in Surgery, King's College Hospital,
London S.E.5, and South-east Metropolitan
Regional Hospital Board.

REFERENCES

- Fitzpatrick, T. J. (1961). *Ann. intern. Med.*, **55**, 1008.
Leared, A. (1859). *Trans. path. Soc. Lond.*, **10**, 177.
Mailer, R. (1939). *Brit. J. Surg.*, **27**, 91.
Raycroft, J. F., and Mastrangelo, M. (1960). *Amer. J. Surg.*, **99**, 361.