COMMENT

Thompson *et al.* (1952), in reporting a case of *Kl. pneumoniae* meningitis with bacteraemia and pneumonia, made a careful review of the literature and analysed 118 cases, 31 with recovery. According to their Table 3, in the pre-sulphonamide era, of 60 cases only 4 recovered, but with sulphonamides alone, of 20 cases 9 recovered. With a combination of sulphonamides and penicillin, of 15 cases 9 recovered; and with sulphonamides, penicillin, and streptomycin, of 13 cases 7 recovered. It is noteworthy that with penicillin and streptomycin, alone or in combination, none recovered. On the other hand, one case (Martin and Sureau, cited by Bombardier, 1950) recovered with chloramphenicol treatment after failure of sulphonamides, penicillin, and streptomycin.

On theoretical grounds it would appear that the drug of choice in the treatment of *Bacterium friedländeri* meningitis is chloramphenicol, because of the ready attainment of therapeutic levels in the cerebrospinal fluid (Ross *et al.*, 1949; Hewitt and Williams, 1950). There is some evidence that oxytetracycline ("terramycin") also crosses the blood-brain barrier (Werner *et al.*, 1950), but aureomycin is known to be a poor drug for systemic administration in meningitic disease.

The case here reported did extremely well on sulpha-dimidine alone, and this shows that clinical judgment must remain, in the last analysis, the supreme arbiter, even in questions of treatment. Except when confronted with fulminating disease, I would renew a plea, made by Banks and others, for withholding intrathecal treatment with antibiotics in purulent meningitis until the bacteriological diagnosis and the clinical course of the disease show it to be imperative. Sulphonamides by the oral or parenteral route, when they do not effect a cure, always give time for deliberation and for completely informed final treatment.

My thanks are due to Dr. Martin Hynes for the bacteriological reports.

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Perforation of a Duodenal Ulcer into the Common Hepatic Duct: With Relief of Obstructive Jaundice

Gastro-intestinal biliary fistulae are not uncommon as a complication of cholecystitis and gall-stones. Kehr (quoted by Hicken and Coray, 1946) found them in 5% of 2,000 patients having an operation on the biliary tract. Perforation of a duodenal ulcer into the biliary tract, however, is very rare. Lønnerblad (1932) described a case and collected 11 others from the literature. Barclay (1936), Hicken and Coray (1946), Nivière (1947), and Vergoz and Lebond (1948) described and collected further cases from the literature, making 30 in all. The evidence in many of the reported cases is only radiological—that is, filling of the biliary tract by a barium meal—but in some the diagnosis was confirmed at operation. In most of the cases reported no special symptoms, apart from those due to the duodenal ulcer, could be attributed to the fistula, but sometimes, particularly where the perforation was into the gall-bladder, the symptoms were those of an acute upper abdominal emergency.

CASE REPORT

A company secretary aged 65 developed a few small painless red nodules on his left leg. Biopsy showed these to be due to reticulosarcoma. The nodules cleared up

after radiotherapy, but a large ulcer developed at the back of his left knee which never healed. Examination of the peripheral blood showed no abnormality.

Two years later, on November 9, 1952, he was admitted to hospital with marked cachexia, flatulent dyspepsia, and a four-weeks history of painless obstructive jaundice. His faeces were pale and his urine contained bile. His serum bilirubin was 4 mg. per 100 ml. and the direct van den Bergh reaction was positive. Although his general condition deteriorated the jaundice improved dramatically on December 5. He died in coma on December 17, which was almost certainly uraemic.

At necropsy there was no trace of the obstructive jaundice. The pancreas was diffusely permeated by reticulosarcoma, giving the appearance usually associated with a carcinoma of the head of the pancreas (see Fig.). The intrahepatic

and extrahepatic biliary tract above the pancreas was extremely distended. The gall-bladder was distended but contained no stones. In the head of the pancreas the duct cavity was compressed so that the lumen had almost disappeared, and contained no bile. The common hepatic duct communicated with the duodenum through the crater of a large chronic ulcer on the posterior duodenal wall 4 cm. from the pylorus. On histological examination the liver cells contained some bile pigment but there was no dilatation of the peribiliary bile ducts or any sign of inflammation around them. Both lobes of the thyroid gland showed nodules of reticulosarcoma, and both kidneys were almost completely replaced by tumour tissue.

COMMENT

In spite of the frequency of duodenal ulcers and their close anatomical proximity to the biliary tract, only very few cases of choledochoduodenal fistula due to perforation of a duodenal ulcer have been found in the literature. Many of these reports are based on radiological evidence only. A case is described in which at necropsy obstructive jaundice was shown to have been relieved by perforation of a duodenal ulcer into the common hepatic duct, thus producing a spontaneous choledochoduodenostomy. No other symptoms could be attributed to the duodenal ulcer or to the perforation. No similar case has been found among those described in the literature.

I am grateful to Professor T. F. Hewer for his help and advice and to Dr. C. D. Evans for the clinical details.

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