tetrabenazine was no different from that achieved with diazepam. In one patient the movements remained unchanged.

In all patients the dyskinesia returned within days after cessation of the trial. One patient who showed a good response to tetrabenazine has developed a more generalized choreo-athetosis since the trial. Side effects were few, though one patient with a previous history of stroke had a syncopal attack towards the end of her trial period on tetrabenazine. Most of the patients showed some drowsiness on tetrabenazine but this was no greater than the sedative effect of the diazepam control.

Discussion

The treatment of persistent facial dyskinesia is unsatisfactory. The movements can usually be improved only by inducing a Parkinsonian syndrome with large doses of the phenothiazines or butyrophenones responsible for the disorder, and such treatment is only rarely considered justifiable. Any medication that reduces the movements in a proportion of these cases would therefore seem to be useful. Our results suggest that some cases of persistent phenothiazine dyskinesia might respond well to tetrabenazine. We are continuing our investigation of its value in a larger series of patients with abnormal movement disorders.

Our results also suggest that the facial dyskinesia caused by

phenothiazines may be analogous to the dyskinesia in Parkinsonian patients treated with levodopa. In Parkinsonism levodopa is probably converted to dopamine in the brain, and the abnormal movements caused by levodopa may be related to the concentration of dopamine in the brain. Pind and Faurbye (1970) showed that patients with persistent phenothiazine dyskinesia have normal amounts of the dopamine metabolite homovanillic acid in the cerebrospinal fluid. Thus there is no evidence of excessive endogenous cerebral dopamine metabolism in these patients. However, if they develop an induced hypersensitivity to normal dopamine formation the improvement shown by some of our patients on tetrabenazine might have been due to partial dopamine suppression.

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MEDICAL MEMORANDA

Diagnosis of Extrahepatic Jaundice in Hodgkin's Disease

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Clinical jaundice in Hodgkin's disease may be due to excess haemolysis, hepatic infiltration, intrahepatic cholestasis, or involvement of the major bile ducts by Hodgkin's deposits at the porta hepatis. The latter is reported as a rare finding (Levitan *et al.*, 1961). Intrahepatic cholestasis occurs as a terminal event of unknown cause in Hodgkin's disease, histology showing only canalicular stasis (Juniper, 1963). Deep jaundice in Hodgkin's disease is, according to Sherlock (1968), due to intrahepatic deposits, and it is believed to be terminal (Bouroncle *et al.*, 1962). A survey of the literature in the English language showed no previous reports of the use of percutaneous transhepatic cholangiography in the diagnosis of jaundice in Hodgkin's disease.

Case Report

A 50-year-old man known to have Hodgkin's disease was admitted to hospital with a five-week history of anorexia, vomiting, pruritus, and increasing jaundice. Clinical examination showed deep jaundice and enlargement of the cervical, axillary, and inguinal

lymph nodes. The liver edge was palpable 5 cm below the costal margin. There was no other abnormality.

Three years before the present admission the diagnosis of Hodgkin's disease was confirmed by lymph node biopsy. Lymphangiography at that time showed retroperitoneal lymph node involvement. He had received several courses of radiotherapy combined with prednisone and chlorambucil.

Present investigations were: serum bilirubin 18.0 mg/100 ml; serum alkaline phosphatase 49 King-Armstrong units; erythrocyte sedimentation rate 37 mm in 1 hour; serum proteins normal; white cell count 4,100/mm³; haemoglobin 14.2 g/100 ml. Other liver function tests were normal.

FIG. 1—Percutaneous transhepatic cholangiogram showing dilated intrahepatic ducts and complete obstruction of the common hepatic duct at the porta hepatis.

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Because of the increasing jaundice and the doubt about its cause a percutaneous transhepatic cholangiogram was performed using the technique described by Young (1965). Puncture of a bile duct in the left lobe of the liver produced clear bile under considerable pressure. Injection of contrast medium into the biliary system showed dilated intrahepatic ducts and a complete obstruction of the common hepatic duct at the porta hepatis (Fig. 1).

At operation a large mass was palpable posterior to the common hepatic duct causing complete duct obstruction at the porta hepatis. The gall bladder was collapsed and there was involvement of the cystic duct. A choledochojunostomy with T-tube drainage of the bile ducts, a gastroenterostomy, and a Roux-en-Y anastomosis were performed. Postoperative recovery was uneventful. Biopsy of the gland mass in the porta hepatis showed typical Hodgkin's tissue. After a course of radiotherapy to the porta hepatis T-tube cholangiography showed free flow of the contrast medium into the intestine via the common bile duct and the choledochojunostomy (Fig. 2).

FIG. 2—Postoperative T-tube cholangiogram showing the choledochojunostomy with free drainage into the intestine.

One month after operation the patient was discharged from hospital and returned to work. The serum bilirubin was 1.4 mg/100 ml and the serum alkaline phosphatase 12.0 King-Armstrong units. He remained well for the next 18 months, but despite further treatment with radiotherapy, steroids, and immunosup-

pressive drugs he died 20 months after operation from systemic manifestations of Hodgkin's disease.

Comment

This patient showed features of both hepatic and posthepatic jaundice. Because of the possibility of extrahepatic obstruction and the progressive jaundice percutaneous transhepatic cholangiography was performed to exclude large duct obstruction. Since failure to catheterize a bile duct after four attempts at percutaneous cholangiography is reliable evidence of absence of duct dilatation in over 90% of cases it was considered a justifiable procedure in the present patient, as liver biopsy had failed to show Hodgkin's tissue.

Demonstration of the common hepatic duct obstruction preoperatively by percutaneous cholangiography enabled the surgeon to plan the operative approach. Because of the rarity of reports of glandular obstruction at the porta hepatis in Hodgkin's disease the appearances were thought to be due to a primary hepatic duct neoplasm. The differential diagnosis of complete common hepatic duct obstruction includes primary carcinoma of the hepatic duct, sarcoma, secondary deposits in the portal lymph nodes, and biliary calculi. In the absence of previous biliary surgery benign strictures are rarely found at the level of the common hepatic duct. Hepatic duct calculi are rare in the absence of gall stones and present a different appearance at percutaneous cholangiography. Since the common bile duct and gall bladder are normal in high hepatic duct obstruction, if percutaneous transhepatic cholangiography or adequate operative cholangiography are not performed the lesion will be overlooked at laparotomy. Because of the difficulty of obtaining films of adequate quality at operation preoperative cholangiography is preferable (Clemett, 1965).

The demonstration of the site of the obstruction in the biliary tract and its operative palliation enabled the patient to return to an active life free from jaundice and pruritus for a period of 18 months. It is suggested that other patients with Hodgkin's disease who develop jaundice may benefit from percutaneous transhepatic cholangiography and palliative surgery.

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