The average number of treatments required in the satisfactory cases was 4.5.

Forty cases of monilial infection in pregnancy treated with the same preparation were all regarded as cured.

We thank Messrs. Camden Chemical Co. for liberal supplies of this compound, which is marketed under the name of "pruvagol."

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RADICAL DUODENOPANCREATECTOMY FOR PAPILLOMA OF THE PANCREATIC DUCT

BY

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The case described below is presented as a pathological rarity. F. M. Owens, in 1948, mentioned a similar case, details of which are appended below, but no other case can be traced in the literature. Willis (1948) makes no mention of the condition, nor does Brunschwig (1942) or Boyd (1947). The recent literature on the operative removal of the pancreas confines itself to (1) tumours of the ampullary region; (2) tumours of the pancreas itself, usually designated as adenocarcinoma; (3) tumours or enlargements of islet tissue; and (4) chronic pancreatitis.

Case History

A storekeeper aged 50 first came under observation in October, 1950, when he had already had pain in the right upper abdomen for one year. It occasionally radiated to the back and then was referred to an area over the angles of the left tenth, eleventh, and twelfth ribs. The pain was occasionally severe, but usually simply a dull ache. It seemingly had no relation to food, nor had it impaired his appetite or digestion. The bowel action remained regular, and there were no symptoms referable to any other system. His general health was good, and at no time had he been jaundiced. He had had diphtheria 40 years previously and haemorrhoidectomy 11 years ago.

A complete general examination revealed no abnormality, and the only positive abdominal sign was slight tenderness over the fundus of the gall-bladder on deep inspiration. A cholecystogram showed a gall-bladder functioning well and no radiological evidence of gallstones. A clinical diagnosis of probable cholecystitis was made and a fat-poor diet, with the addition of cholagogues, was prescribed for a trial period. I saw him again in February, 1951, but there was no change in the syndrome of fairly constant pain of the same distribution, with momentarily severe attacks. There was still no upset in digestion, no jaundice, or any deterioration in general health. Examination again revealed nothing wrong except a tender gall-bladder on deep inspiration. Laparotomy was now advised and accepted.

Operation.—On March 2, under general anaesthesia (thiopentone, gas and oxygen, and intermittent intravenous curare), a right upper paramedian incision was made. The gall-bladder was not distended, but was slightly thickened and opaque. The whole pancreas, including the uncinate process, was thickened and nodular, but no definite separate tumour could be felt in the head. The gland was thought to be the site of an obstructive lesion in its main duct

system, most probably a carcinoma. On this account it was decided that an attempt at radical cure should be undertaken; but, whatever the nature of the lesion, it was obvious that pancreatectomy alone would abolish the severe continual pain.

Technique.—Radical duodenopancreatectomy was performed, closing the fourth part of the duodenum and dividing the pancreas so as to leave only a small piece of the body and the tail. The following reconstruction was undertaken: The first loop of the jejunum was drawn up in front of the transverse colon and into its apex was implanted the common bile duct, with the help of a small rubber tube. The stomach was implanted end to side into the efferent limb. The raw pancreatic area was oversewn with interrupted sutures of catgut and linen thread, after ligation of the pancreatic duct. Drainage was provided for the biliary anastomosis and the pancreatic stump. The whole procedure took four and three-quarter hours, and during it the patient was transfused with 4 pints (2.2 litres) of blood to replace blood lost from a damaged portal vein behind the pancreatic neck. This was finally dealt with by lateral suture.

Post-operative Progress

The patient's condition was remarkably good on return to the ward, and he had a systolic blood pressure of 110. Bed-blocking by the foot and an oxygen tent prevented any post-operative shock in the first 24 hours. He began taking nourishing fluids on the third day and thereafter a light diet, progressing to a fuller diet poor in fats. There was no drainage from either of the drainage-tubes, and these were removed on the fifth day. He got up for a short time on the second post-operative day, and thereafter for increasing periods until the eleventh day, when he developed a left pleural rub associated with a thrombosis in the left calf. This cleared satisfactorily with rest in bed and a few days' heparin therapy. By this time the patient was having three to four loose stools a day, but had no other complaint. Traces of reducing substance were occasionally noted in the urine.

Post-operative Investigations.—Faecal analyses (see Table I); sugar-tolerance tests (see Table II); serum amylase, 3 units; urinary diastase, 7 units.

TABLE I.—Faecal Examination

Date	Undigested Meat Fibres	Total Fat	Neutral Fat	Free Fatty Acids	Soaps
19/4/51	Present	44%	12%	21%	11%
22/10/51	"	28%	1.8%	10.6%	15.6%

TABLE II.—Sugar-tolerance Tests

	Fasting	½ hr.	1 hr.	1½ hr.	2 hr.
19/4/51 mg./100 ml.	102	108	152	184	152
2/10/51 mg./100 ml.	90	140	162	106	82

Altered Physiology.—First of all there was a not unexpected trypsin deficiency, as evidenced by undigested meat fibres in both faecal analyses. On the other hand, there seemed to be enough lipase left to split fat adequately, but there was a pronounced deficiency in the absorption of fatty acids. I can only suggest that the intestinal lipase was not sufficient to split the fat before it had left the absorptive area of the gut, but was sufficient to split the fat before final excretion per rectum. The sugar-tolerance test on May 19 showed a mild diminution in tolerance with a high blood sugar at the end of two hours. This suggests that there had been left just enough islet tissue to deal with the insulin needs of the body. It seems likely that work hypertrophy of islets had taken place, as the sugar-tolerance test on October 22 showed a normal series of figures.

Pathology.—The main pancreatic duct widened into a considerable dilatation some 1 in. (2.5 cm.) from the duodenal

wall. This dilatation was lined with papillomatous tissue. Behind it the pancreatic gland was a picture of atrophy and fibrous replacement. There were marked fibrosis, collections of cellular infiltration, and a few acini here and there. The islets of Langerhans were prominent in the fibrous tissue. The obstructive element was suggested by the marked dilatation of the duct system. Section of a part of the papilloma showed the branching villi lined with regular columnar epithelium on an unbroken basement membrane. The pathologist thought that at this stage it was probably benign but might well develop malignant tendencies.

Progress Report.—This case has been followed up at regular intervals from March, 1951, to the time of writing, and in the main has presented a satisfactory clinical picture. The patient's body weight, which in health was 12 st. 9 lb. (80.3 kg.), has remained steady at 11 st. 8 lb. (73.5 kg.), and his general feeling of well-being has slowly increased since pancreatectomy. At first he was subject to violent attacks of diarrhoea and abdominal colic with the least dietary indiscretion, but when he remained on a strict fat-poor diet with the addition of one tablet of pancreatin before each meal, these were infrequent. He usually had two or three semi-formed stools a day, but any intake of excess fat immediately put the number up. He began light work in July, 1951, four months after operation, and after a month resumed a full day's work as a clerk in a publishers' office.

Summary of Case Reported by F. M. Owens.—A man aged 59 was operated on for suspected carcinoma of the pancreas. The pancreas was hard, except for two large cystic areas. A total resection revealed a severe chronic pancreatitis with an intraductal papilloma obstructing the main pancreatic duct near the ampulla of Vater. He died four months later from a perforated anastomotic ulcer.

Conclusion

The case reported above might well have been classified as "chronic pancreatitis" if excision had not been undertaken, and even the histology supported the diagnosis until the obstructive papilloma in the duct system was found. As the aetiology of "chronic pancreatitis" remains obscure, it is suggested that some cases may well be due to a ductal obstruction of this nature. A complete exclusion of the residual pancreas from the digestive tract is not incompatible with continued well-being, but it is obvious that, at least, in this case great care must be taken over the ingestion of fat. Pancreatin capsules are not adequate substitutes for the natural glandular secretion.

Summary

A case of obstructive pancreatitis due to papilloma of the duct of Wirsung is presented.

Its pathology, treatment, and post-operative course and follow-up are detailed.

The altered physiology is discussed.

I wish to thank Dr. P. O. Ellison, Royal Northern Hospital, for an opinion on the pathology.

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The U.S.A. is at present suffering an exceptionally severe epidemic of acute poliomyelitis, according to reports in the weekly bulletin of W.H.O. In the week ending September 13, 4,032 cases were notified, which is the highest figure ever recorded for a single week. From January 1 to September 6, 27,362 cases were notified. This total is 3.5% more than for the corresponding period in 1949, in which year the epidemic was the greatest ever recorded. The total to September 6 this year is 77% and 80% more, respectively, than the totals for the corresponding periods in 1950 and 1951.

BANTI'S DISEASE IN TWO BROTHERS WITH A BLOOD-GROUP ANALYSIS OF THE PARENTS

BY

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AND

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As is well known, Banti's syndrome comprises splenomegaly with fibrotic degeneration, in some cases associated with cirrhosis of the liver. The most important clinical manifestations are anaemia, haematemesis, and ascites. In general the disease is not regarded as hereditary, although a familial occurrence has been reported in a few instances (Gunn, 1926; Gutzeit and Wendt, 1930). Some diagnostic difficulty is often encountered, as a histological examination of the spleen is usually necessary to distinguish the syndrome from other forms of splenomegaly.

Recently we had occasion to observe Banti's disease in two brothers. Both died, and the diagnosis was confirmed by necropsy. The parents and the nine sibs showed no sign of the disease. There was no history of hepatitis in either case. The patients were not alcoholics, and the Wassermann reaction was negative in both.

Case Reports

Case 1.—Male, born 1924. At the age of 23 the patient developed anorexia and periodical epigastric pain. Admitted to the Copenhagen City Hospital. Diagnosis: splenomegaly, haematemesis, ascites. Splenectomy was performed, but the patient died after the operation from phlegmonous cholecystitis with perforation and cholangitis. At necropsy the spleen measured 25 by 17 by 8 cm., and was of a firm and hard consistency. Histological examination showed it to be slightly fibrotic. The liver measured 28 by 16 by 6 cm.; its surface was granular and the consistency firm. Histological examination revealed cirrhosis of the liver. In addition, pronounced oesophageal varices and a large thrombus in the portal vein were found.

Case 2.—Male, born 1925. At the age of 16 years splenomegaly was diagnosed. At age 23 the patient was admitted to the Copenhagen City Hospital because of haematemesis and melaena. At the age of 24 he underwent splenectomy with spleno-renal anastomosis. The spleen measured 10 by 15 by 20 cm. and was very firm. Histological examination showed it to be fibrotic. At the age of 26 he had severe haematemesis and melaena: death ensued. Necropsy: liver enlarged, tissue very firm, pronounced hepatic cirrhosis, moderate oesophageal varices, remains of widespread thrombosis in the portal vein.

Discussion

Ever since Banti first described the disease at the end of the past century its aetiology has been the subject of discussion. Several authors have emphasized the portal hypertension as the primary pathogenetic factor, whereas others believe morbid conditions in the splenic arteries to be the actual cause. This question has not yet been finally elucidated.

Quite recently Tidy (1952) advanced the theory that Banti's disease could be caused by rhesus incompatibility between mother and child. Gerrard (1952), however, examining the question, concluded that splenic enlargement could only be a rare sequel of rhesus iso-immunization.

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