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CHRONIC RECURRENT PANCREATITIS

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Chronic recurrent pancreatitis, also known as chronic relapsing pancreatitis, has always been a problem of some magnitude for the surgeon. In writing this article we have made an attempt to give a brief summary, from a practical point of view, of the anatomical and physiological background of the problem; to describe some of the many and varied approaches to the problem; and to formulate a new approach, which, we hope, may lead to further discussion and research.

Anatomy

The pancreas consists of a head and neck, with the uncinate process lying in front of the second lumbar vertebra, and the body and tail extending to the hilum of the spleen.

The following arteries contribute to the blood supply of the pancreas:

(*a*) The superior pancreatic artery—a branch of the coeliac axis, or the hepatic artery, or the splenic artery.

(*b*) The inferior pancreatic artery—a branch of the superior pancreatic artery or the superior mesenteric artery.

(*c*) The anterior superior branch of the pancreaticoduodenal artery.

(*d*) The posterior superior pancreaticoduodenal artery. Both (*c*) and (*d*) are branches of the gastroduodenal artery.

(*e*) The supraduodenal artery which is also a branch of the gastroduodenal artery.

The principal venous drainage of the pancreas is into the superior mesenteric vein *via* the termination of the inferior mesenteric veins; through the terminations of the left gastric vein, the splenic vein, the right gastro-epiploic vein, the anterior superior pancreaticoduodenal vein, the anterior inferior pancreaticoduodenal vein, the inferior pancreatic vein, the pancreatic cervical vein, and through several tributaries directly into the splenic vein. It is noteworthy that no vein enters the anterior surface of the portal vein. The arterial supply to, and venous drainage from, the head and neck are entirely separate from those of the body and tail, thus constituting two separate entities.

The pancreatic duct system is composed of minor tributaries which enter the main duct at a right angle. This duct drains into the ampulla of Vater, and from it an accessory duct, the duct of Santorini, enters the duodenum on its own.

There are 4 principal variations in the mode of entry of the duct into the duodenum: (1) In 29% of cases two separate channels are present—one for the pancreatic duct and one for the bile duct at a common opening, (2) in 37% both ducts have a common opening which is short, (3) in 30% both ducts have a common opening which is long, and (4) in 2% there is a single opening for the common duct and the pancreatic duct draining into the common duct.

The diameter of the orifice of the ampulla of Vater varies from 1·5 to 4·5 mm., and the average width is 3 mm. The length of the ampulla from the duodenal surface to the tip of the papilla or the summit of the septum varies from 1 to 14 mm. This fact is of importance in regard to the reflux biliary theory as a factor in the causation of pancreatitis.

The musculature of the sphincter itself consists of circular fibres passing round each duct and some passing round both ducts, and Doubilet and Mulholland[1] demonstrated a common channel in 48 out of 49 operated cases; in these cases closure of the sphincter would close both ducts.

Histology

The pancreas is divided into lobes and lobules by septa consisting of connective tissue. It is covered on its anterior aspect by the posterior parietal peritoneum, but the gland itself posesses no true capsule. Neural elements arising from the sympathetic and parasympathetic systems traverse the interlobular septa. The functional components of the pancreas consist of two types of cellular elements. One of these is concerned with external secretion, (the function of the larger portion of the pancreas) and is arranged structurally in the form of acini consisting of a layer of pyramidal or cuboidal cells surrounding a central lumen which opens into a tributary of the ductal system. The lesser portion of the pancreas is taken up with internal secretion in which the cellular elements are arranged in insular fashion and known as the islands of Langerhans, numbering from 200,000 to 2,000,000 in the human gland. These islands are scattered irregularly throughout the gland and are more numerous in the tail than elsewhere. They are composed of several types of cells mostly containing granules in the cytoplasm. The alpha cells are acidophilic, while the beta series are basophilic. There is also a group of cells in the islands that contains no granules. Islet cells are arranged in chains, separated by a generous network of capillaries. There is some speculation whether the mature acinar cell can, under adequate stimulation, differentiate into an islet cell, thus permitting the number of islet cells to vary in proportion to the demand.

Physiology

The physiology of the pancreas is manifested by two secretions—an external secretion derived from the acinar cells and an internal secretion derived from the islet cells. Dragstedt has imputed to the function of this gland the manufacture of lipocaic, a hormone concerned with fat metabolism.

Normal pancreatic juice is colourless, turbid, and alkaline, and is secreted as a response to ingested food. The normal output of pancreatic juice is 1,200 - 2,000 c.c. per 24 hours, and the enzymes contained in it are trypsin, chymotrypsin, lipase, amylase, renin and maltase. The pH is between 8·3 - 8·6, the specific gravity is 1015 and the juice contains 8 g. of protein per 100 c.c. The chloride content is between 35 - 97 mEq./l., the bicarbonate 30 - 74 mEq./l., the sodium 134 - 142 mEq./l., the potassium 4·7 - 5·4 mEq./l. and the calcium 0·4 - 4·7 mg. per 100 c.c.

The proteolytic enzyme of the pancreatic juice enters the duodenum as trypsinogen which is inert, but is activated into trypsin by enterokinase elaborated by the duodenal mucosa; it also enters as chymotrypsin. The tryptic activity is that of breaking down the peptide linkage of proteose, peptones, and higher polypeptides of the broken down protein elements that have been digested by pepsin in the stomach.

The amylolytic enzyme of the pancreas acts in a neutral medium. It is suggested that secretin is present in the duodenal mucosa in an active form. It has also been stated that secretin is present in the inert form of pro-secretin, which becomes activated by the presence of bile. Secretin is formed as a result of the stimulus of ingestion of food, and in turn stimulates the flow of bile and succus entericus.

The synthesis of protein in the pancreas is elaborate. It is not necessary for our purposes to give a detailed description of how the granules are transformed (by chemical reaction) by the action of erepsin. The lypolytic enzyme is called steapsin, which digests fats into glycerine and fatty acids. The amylase in the pancreatic juice converts carbohydrates into dextrines. The hormone secretin from the duodenal mucosa is the prime factor in promoting pancreatic secretion. Stimulation of the vagus nerve causes the flow of pancreatic juices but never that of secretin. The sympathetic fibres play only a minor role in the regulation of pancreatic secretion.

The action of various drugs on the pancreas is well known, and we do not propose to elaborate further on this, except to state that alcohol diminishes the volume of secretin. One litre of 5% glucose in water intravenously increases the flow by 50%, and cortisone has no effect on the pancreatic secretion. Antibiotics are not excreted in the pancreas, with the exception of sulphadiazine where the excretion is high, but penicillin and streptomycin are

not excreted. With regard to the internal secretion no further mention need be made of the well-known work of Banting and Best. The question of lipocaic is still under investigation, and we are not prepared at this stage to express a dogmatic view on this subject.

Aetiology

Chronic pancreatitis has been recognized for many years, but Comfort and his associates[2,3] first described it as a clinical entity in 1946. They described a syndrome characterized by attacks of upper abdominal pain with a variable degree of acinar and islet dysfunction followed by certain sequelae. It is now recognized as a separate clinical entity.

Chronic relapsing pancreatitis must be considered as a continuation or progression of acute pancreatitis, either in a mild or severe form—the recurrent attacks ultimately leading to persistent chronic symptoms and permanent physiological disturbance.

Sex. The ratio of male to female patients is 2 : 1, which rather minimizes gall-bladder disease as a causative factor.

Age. The ages of the 27 cases described by Comfort *et al.*[2] vary between 10 and 75 years. In a group of 29 cases observed in the Lahey Clinic[3] the ages vary from 20 to 64. We have had a child of 9 with recurrent pancreatitis and a man of 87 with the same disease.

Obesity, whilst common in acute pancreatitis, is not the rule in the chronic disease, since patients with the chronic disease tend to lose weight rapidly.

Alcohol. It is common to find that chronic alcoholism antedates the history of pancreatitis, and it would be fair to state that chronic alcoholism is a concomitant in between 40 - 50% of cases in this disease.

Trauma is an uncommon but nevertheless definite cause of the disease, and has been reported in 3 out of 38 cases of a series by Cattell and Warren.[4]

Disease of the biliary tract. After a detailed analysis of their clinical material Comfort *et al.*[2] concluded:

1. Inflammatory disease of the gall bladder was not a requisite of the development of chronic relapsing pancreatitis, and

2. When the pathological processes occurred simultaneously or in association, the preponderance of evidence suggested that the disease of the biliary tract is secondary to the pancreatic disease.

Common channel. Doubilet and Mulholland[5-7] more recently stated that the reflux of bile through a common pancreaticobiliary channel is primarily responsible for chronic relapsing pancreatitis. Archibald[8] popularized the theory that spasm of the sphincter of Oddi caused reflux of bile into the pancreas and in this way generated the acute disease. Doubilet and Mulholland[5] have found a common channel in most instances, and have also found that spasm of the ampulla of Vater can be induced by the application of dilute hydrochloric acid to the duodenal papilla.

Infection. There is no definite evidence that infection plays a part in the cause of this disease.

Types of Chronic Pancreatitis

There are 4 main types of chronic pancreatitis:

1. The diffuse type in which the entire gland is involved. This is often found on exploration of the abdomen.

2. Chronic pancreatitis with multilocular cystic changes.

3. Chronic pancreatitis with pancreatic lithiasis or calcinosis.

4. Localized chronic pancreatitis secondary to trauma. Pancreatic lithiasis represents the final stage in the progression of chronic relapsing pancreatitis.

The pathogenesis of pancreatic lithiasis is unknown, but the precipitating factors appear to be: (*a*) Obstruction, (*b*) stagnation, (*c*) infection (questionable), and (*d*) fibrosis and autodigestion with fat necrosis.

Pathology

Early in the disease the gland is enlarged, firm, and somewhat pale in appearance. The limits of the gland are indistinct and peripancreatic oedema is present. As the disease progresses the pancreas becomes larger and more indurated, and the body and tail lose their prismatic cross-section to become more rounded. The gland becomes fixed to surrounding structures and loses what limited mobility it had. Adjacent structures may become adherent. Pressure on the portal and superior mesenteric veins may cause portal hypertension, and the veins in the region of the stomach especially are engorged.

The head of the gland is, as a rule, disproportionately large and solitary or multiple cysts may be seen or palpated. The duct of Wirsung is frequently enlarged, and the cut surface of the gland is fibrotic and gritty, and does not bleed as readily as the normal gland.

Areas of calcification may be found, and stones varying in size from a fraction of a mm. to 3 cm. in diameter may be present; they are firm, white, round and smooth, conforming to the contour of the duct. They are laminated and range in number from 1 to 300.

SYMPTOMS AND SIGNS

Chronic pancreatitis is a difficult diagnosis to make, but if the condition is borne in mind there is no reason why an accurate diagnosis should not be established. The symptoms are as follows:

1. *Pain*

(i) Abdominal pain, which is colicky in nature, is present. As a rule the pain is severe in the acute attacks, and less severe in the recurrent attacks.

(ii) The attacks are episodic, with intervals of freedom.

(iii) With chronicity constant pain is a feature, so much so that these patients often become chronic alcoholics as a result of pain, if they are not such already, or they become morphine addicts. Of Cattell's series[4] 58% were addicted to narcotics. The pain is epigastric and is referred to the left hypochondrium and through to the back. Pylorospasm is a frequent feature. Attacks are worse at night, and the patient sits doubled up with his hands folded across his abdomen.

2. *Nausea and Vomiting*

(i) This usually appears before the onset of the pain.

(ii) Persistent vomiting is due to duodenal obstruction when it occurs.

3. *Diarrhoea, Steatorrhoea, Creatorrhoea*

These signs are found in 50% of cases, are related to the attacks and may become so severe as to produce a negative nitrogen balance.

4. *Constipation*

This is due, when it occurs to: (*a*) Diminished food intake, (*b*) reduction in fat in the bowel, and (*c*) narcotics.

5. *Weight Loss*

Weight loss is frequent, and is due to anorexia and failure of fat absorption.

6. *Jaundice*

Jaundice was observed in 18 out of 65 cases collected by Haggard and Kirtley[9] and in 26% of cases at the Lahey Clinic. Most of these cases are due to stones in the common duct, but pancreatic calcification and fibrosis is often associated with hepatocellular disease, which may account for the jaundice, or compression of the common bile duct in the pancreas.

7. *Diabetes Mellitus*

This is a late complication, and is often latent, but diabetes associated with upper abdominal pain should suggest the diagnosis of pancreatitis.

8. *Physical Signs*

Physical signs are totally absent, except for cyst formation.

9. *Laboratory Investigations*

Undue stress has been placed on these investigations, but they may be of some assistance, e.g. (*a*) serum amylase is raised only in the acute episodes; (*b*) the diminution of pancreatic secretion in response to intravenous secretin as determined by analysis of the duodenal contents (the secretin test), although revealing in some instances, is a tedious and

unnecessary procedure; (*c*) alterations in the glucose tolerance occur in approximately 1/3rd of the cases; and (*d*) fat and meat fibres are found in the stool. This is a most inconstant finding.

10. *X-ray Findings*

Pancreatic calculi may be demonstrated radiologically, but the principal features are distortions or displacements of adjacent viscera. The duodenal C is widened if the head is enlarged, or large cysts cause pressure deformities of the stomach. The most helpful sign is the presence of calcification in the pancreas, and it must be remembered in the radiological technique to scrutinize the control film carefully for a solitary stone in the region of the ampulla.

COMPLICATIONS

The following complications occur in chronic pancreatitis: (1) Diabetes mellitus, (2) pancreatic cysts, (3) pancreatic abscesses, (4) pancreatic haemorrhage, (5) portal hypertension, (6) biliary cirrhosis, and (7) carcinoma of the pancreas, which supervenes in a small percentage of cases.

TREATMENT

The wide variety of therapeutic measures at present advocated for this disease is an indication of the inadequacy of the treatment of the condition. The programme that has been adopted can be summarized as follows:

1. The Elimination of Causative Factors

The elimination of all possible causative factors, such as alcoholism, biliary-tract disease, diabetic errors, etc.

2. A Medical Regime

A careful medical regime, consisting of: (i) A bland diet free of fruit and vegetables, with a reduction, but no elimination, of fats; (ii) the prohibition of alcohol; (iii) the elimination of morphia, which is a cause of spasm of the sphincter of Oddi; and (iv) the administration of 6-8 g. of pancreatin per day, of insulin where necessary, of bile salts and of dehydrocholine, 1 tablet 3 times daily before meals.

3. Surgical Manoeuvres

Despite careful medical treatment, the majority of cases will require surgery, and the surgical manoeuvres can be listed as follows:

1. *Indirect methods of treatment*

These include:

(*a*) *Biliary tract procedures*, i.e. (1) Cholecystectomy-choledochostomy, (2) biliary-intestinal anastomosis, and (3) sphincterotomy.

(*b*) *Gastro-intestinal diversion*, i.e. (1) gastro-enterostomy, (2) pyloric exclusion, and (3) gastrectomy.

(*c*) *Nerve interruption*, i.e. (1) Sympathectomy (thoracolumbar and splanchnicectomy) and (2) vagotomy.

2. *Direct methods of treatment*

These include: (*a*) Drainage of cysts, (*b*) lithotomy, (*c*) anastomosis (continuity and diversion), and (*d*) resection (distal pancreatectomy, pancreatoduodenectomy, and total pancreatectomy).

Comment

Cholecystectomy and choledochostomy are only indicated in the presence of biliary disease, and this, as stated earlier, is not a frequent accompaniment of pancreatitis. Biliary intestinal anastomosis may be necessary as a result of the pancreatic disease. It is, however, not of much value in the treatment of the disease.

Sphincterotomy is advised by Doubilet and Mulholland;[5] it is also the procedure adopted by Rodney Smith.[10] These authors report good results, but in our experience only 40% of patients obtain relief by this procedure, which should always be carried out by the transduodenal route. Cattell[11] inserts the long limb of the T-tube into the duodenum, but we have had 2 cases where this produced acute pancreatitis.

Gastro-enterostomy is only indicated in obstruction. This procedure has been considered of value in some quarters because of the diversion of the food and gastric contents. Gastrectomy has been advocated in order to obtain a similar result, but we do not believe that this procedure should be attempted except where there is existing disease of the stomach or duodenum.

Sympathectomy and splanchnicectomy have been advised for relief of pain, and some favourable results have been reported in employing these procedures, but recourse to them should be the last resort since they are only symptomatic methods of treatment.

Vagotomy, in our opinion, is not a worthwhile undertaking. Cysts will require either internal or external drainage. Cattell[11] is still of the opinion that external drainage is the most satisfactory procedure, but we have found that internal drainage, preferably to the stomach, has met with success.

Pancreatic lithotomy is indicated where stones are observed. Some workers have advised ligations of the pancreatic ducts to produce atrophy of the acinar portion of the pancreas, but if this is successful it will produce a chronic state of nutritional deficiency.

Anastomosis of the duct of Wirsung has been advised by Cattell.[12] This procedure does not appear to have any appreciable applicability for the duodenal end of the duct, but is valuable for the distal end where there is obstruction anywhere between the neck and the tail; the procedure is then accomplished by distal pancreatectomy with anastomosis of the remaining portion of the pancreas to the jejunostomy by the Roux-en-Y technique.

Pancreatoduodenectomy may be indicated on rare occasions —usually in association with carcinoma or multiple calculi.

The same applies to total pancreatectomy, but the control of diabetes after this operation is difficult, because of the patient's extreme sensitivity to insulin.

RECENT WORK

Having reviewed the position in regard to pancreatitis up to the date when we undertook the project which we are about to describe, we will now deal with the recent work in relation to this disease. This project was commenced in 1951 when we began doing cholangiography. We were impressed with the fact that on many occasions the pancreatic duct was visualized during this procedure. We felt that it was reasonable that if the pancreatic duct could be totally visualized there might be a better understanding of the depressing disease of chronic recurrent pancreatitis.

The first problem was that we had no established normal. At that time (1951) no such procedure had been undertaken and operative pancreatography was totally unknown. The problem was how to approach the pancreatic duct, and it was only with the advent of sphincterotomy in 1953 that we realized that this was the mode of access.

Cases were few and far between and, in 1956 when only 3 cases had been dealt with, an article was published by Doubilet and Mulholland[13,14] describing a project similar to

Fig. 1. Visualization of the pancreatic ducts during operative cholangiography.

ours. Within a short while they published an evaluation of this work in March 1957.[15]

Our theory was that chronic relapsing pancreatitis was due, in the main, to causes within the pancreatic duct system, and all our investigations were related to this factor.

We felt that the following information could be obtained by pancreatography:

1. Obstruction at the entrance of the duct at the sphincter of Oddi.
2. Stricture of the pancreatic duct anywhere in its course.
3. The discovery and accurate pin-pointing of pancreatic calculi.
4. The demonstration of ductal ectasia.
5. Cyst formation, either large, small, single, or multiple.
6. The presence of an abnormal duct system.

It was also hoped that this procedure would be of value in demonstrating small tumours of the pancreas, and in demonstrating extrinsic pressure on the pancreatic duct system from outside sources. Furthermore, it was felt that the operation of distal pancreatico-jejunostomy or partial resection of the pancreas, as first described by Aird and Buckwalter[16] in 1955, would be indicated after the demonstration of pathology in the duct system as shown in pancreatography.

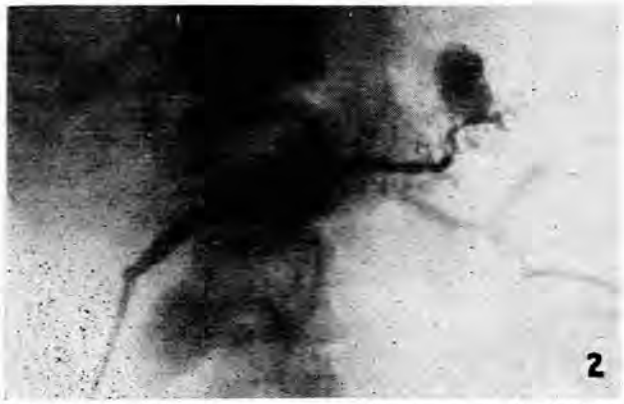

Fig. 2. Operative pancreatogram showing a grossly dilated duct with a terminal cyst.

TECHNIQUE

The abdomen is opened by a Kocher's incision. The gall-bladder and biliary tract are explored, and a cholangiogram is performed on the table. If any disease of the biliary tree is found it is dealt with as required, and the common duct is in any event opened. Thereafter the duodenum is opened and a sphincterotomy is performed, whereby only the musculature of the sphincter is cut through at 12 o'clock, and the musculature of the duodenum is not disturbed.

At this stage the opening of the main pancreatic duct can usually be visualized in its position at 5 o'clock opening at the sphincter of Oddi. If the duct opening is not found in this position, it usually opens ½ inch above this into the common duct itself, and can be demonstrated in that position. The duct having been opened, a fine ureteric catheter is passed into it, and 3 c.c. of urografin 60% is introduced into the catheter. X-ray pictures are then taken. It is important at this stage to realize, as we have found from experience, that the maximum capacity of the pancreatic duct system is never more than 3 c.c. If any resistance is therefore met with when the injection is performed, it should be stopped immediately, otherwise the duct will be blown—usually at the terminal end, with the possible establishment of a localized area of pancreatitis.

If a stricture is discovered in the proximal portion of the duct, it is dilated with ureteric dilators, and at the completion of the dilatation a further pancreatogram is done. If the stricture is found in the distal end of the duct, then this together with the distal portion of the gland is resected. If the stricture is more towards the head and neck, then the distal portion of the duct is anastomosed to the jejunum by the Roux-en-Y technique. If calculi are discovered they are dealt with either by removal or by resection, and similarly, any local pathology in the gland itself can be handled according to what is discovered on X-ray.

If no pathology is discovered then the sphincterotomy has acted as a therapeutic procedure for the treatment of the pancreatitis.

The following case reports are of interest in this connection:

Case 1

Mr. L., aged 48, was seen on 17 April 1957. His history was that during the war, whilst on active service, he had had several attacks of pain in the abdomen, and an ulcer was diagnosed. He was X-rayed and no ulcer was found, but he was subsequently discharged from the army with the diagnosis of 'gastric trouble'. On the night that we were asked to see him he was a desperately ill man, with a history that 48 hours before this date he had an attack of pain in the left loin and the back, radiating across the upper abdomen. He had vomited incessantly for 36 hours, the pain was constant, and had become progressively worse. His blood pressure was 90/60 mm. Hg, his temperature was 100°F, his pulse rate was 68, his upper abdomen was distended, and the whole abdomen was completely rigid and silent. A diagnosis of acute pancreatitis was made, and he was admitted to the nursing home. At that stage, 48 hours after the commencement of his illness, his serum amylase was 640 Somogyi units, and his white-blood count was 21,000 per c.mm. He was treated conservatively with intravenous fluids and antibiotics, and he gradually improved and was discharged from the nursing home 9 days later. Sixteen days after his discharge from the nursing home he had a recurrence of the same type of pain, and was readmitted to the nursing home. At this stage his serum amylase was 1,600 Somogyi units, and his white-blood count was 9,000 per c.mm. He was treated conservatively again and was discharged from the nursing home approximately 10 days later. At this stage he was not entirely free of symptoms and, because of the persistence of his pain, he was readmitted a week later and operated on. The pancreas was large, swollen

and oedematous. The common bile duct was opened, and no pathology was found. The gall bladder appeared to be entirely normal. The duodenum was opened and a sphincterotomy was performed. The pancreatic duct opening was visualized, and a ureteric catheter inserted and the dye injected. At a distance of 1½ inches from the opening a stricture was found. This was dilated and the patient was re-X-rayed, when it was found that the pancreatic duct was now normal. His progress thereafter was satisfactory, and within 14 days he was discharged from the nursing home. Subsequent follow-up reveals that this patient, almost 2 years later, has had no further trouble, and is doing a full normal day's work on a normal full diet.

Case 2

Mr. M., aged 36, was admitted to a urological ward with a history that he had been on an alcoholic spree for approximately a week before the date of admission on 30 May 1958. On the day before admission he had developed acute colicky pain in both loins, which radiated round to the front, especially to the left, where it reached the left iliac fossa. The registrar, an acute observer, decided that this case was possibly one of acute pancreatitis, and the serum amylase was 458 Street-Close units. This patient was treated conservatively without his symptoms completely resolving, and was operated upon on 8 July 1958 through a Kocher's incision. The pancreas, once again, was thick and oedematous in its entire extent. The gall bladder was normal, and the common duct was explored and also found to be normal. The duodenum was opened and a sphincterotomy was performed. A ureteric catheter was introduced into the duct of Wirsung, and a stricture was found at its opening. This was dilated up eventually to a No. 2 De Bakey, and after the dilatation there was a gush of pancreatic juice into the operative field. The patient made an uninterrupted recovery, and was discharged from hospital 22 days later. He has been followed up since, and on only one occasion did he have a mild attack of lower abdominal pain, when he was readmitted to hospital and a diagnosis of diverticulitis was established. As far as his pancreas is concerned, he has had no trouble for the past 10 months.

Case 3

Mr. I., aged 33, is a confirmed alcoholic. He had suffered from abdominal pain on and off for some years. He had been readmitted to hospital on several occasions with attacks of pancreatitis.

He was dealt with in the same way as the other two cases, and he was found to have a stricture in the middle of the pancreatic duct. This case was dealt with by an anastomosis of the distal portion of his pancreas to the jejunum by the Roux-en-Y technique.

On 15 October 1957 there was a complete resolution of symptoms, and he was discharged from hospital 11 days later. He has been readmitted on two occasions, once in June 1958 and again on 16 July 1958, on both occasions having had an acute alcoholic episode with a mild recurrence of symptoms. Despite psychiatric therapy he still continues to remain a chronic alcoholic, and we are afraid that the result will not be good in his case if this state of affairs persists.

Case 4

Mr. J. was admitted to hospital on 7 June 1958, having had a previous attack of pancreatitis. This admission was for a very acute episode, which was treated conservatively and subsided within a few days, but kept on recurring. During an exploratory examination on 24 June 1958 he was dealt with in a similar manner to the cases mentioned above. In this case ectopic pancreatic tissue was found in the duodenum in the region of the ampulla, which was markedly engorged and oedematous. The sphincter was only found with difficulty, and a sphincterotomy was performed. The pancreatic duct was dilated, but the pancreatogram was unsatisfactory because of a technical radiological difficulty.

He was returned to the ward, but developed a massive collapse of both lungs the same night. He died 3 days later. Autopsy revealed an extensive collapse of the lungs with bronchopneumonia and a complete fatty infiltration of the liver with a severe degree of intra-acinar fibrosis. Sections of the kidney showed tubular necrosis. The pancreas was still oedematous and showed evidence of chronic pancreatitis, and the duct itself was thickened and fibrosed and small foci of epithelium were embedded in the duct.

Whilst the literature on this subject is still very small, nevertheless, Pollock[17] in a recent article reported on his experiences in doing 33 pancreatograms on cadavers and 11 on live patients, and he is prepared to condemn this operation. Other writers to date feel as we do that there is a future for this procedure.

CONCLUSION

It is too early at this stage to evaluate results, and the number of cases, viz. 8, is still too small, but we are satisfied that this procedure yields valuable information, and has added fundamental knowledge to a subject about which we are very ignorant. Its use, we are sure, will throw light on what was previously a valley of darkness.

SUMMARY

1. A brief summary is given, from a practical point of view, of the anatomical and physiological background of the problem of chronic recurrent pancreatitis.

2. A summary is given of the wide variety of therapeutic measures at present advocated for this disease.

3. The technique of operative cholangiography and pancreatography is described and 4 case studies are quoted.

4. It is concluded that the procedure described has added fundamental knowledge to a subject about which little definite information is available.

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