failed. In the present series of 39 patients 8 had received conservative treatment for periods up to two years, and in spite of this their condition had worsened; five of these derived benefit from the operation. The results of operation are set out in Table III.

TABLE III.—*Results of Operation in* 39 *Patients*

Post-operative death	1
Slowly worsened (3 died of intercurrent disease)	8
Remained stationary (1 died of intercurrent disease)	8
Shown slight improvement (1 died of intercurrent disease)	9
Improved considerably	13

Of those who have derived benefit from the operation none has become entirely normal, though a few have very little disability. Digital dexterity is perhaps the function which recovers least and most slowly. It may be felt that the results are disappointing, and certainly they do not compare favourably—so far as recovery of function is concerned—with the usual experience of the relief of spinal-cord compression due to tumour. But the patients are in no way comparable, for the operation is essentially a palliative one. In many cases, perhaps in all, the damage to the spinal cord is to a greater or less extent ischaemic and the degree of compression unmeasurable. This is perhaps borne out by the cases in which the nervous condition slowly worsens, as happened in eight patients, three of whom died of intercurrent disease. We also do not yet know whether improvement following operation will continue to be maintained: one of the patients who nine years ago derived some benefit from a restricted operation has recently shown deterioration, and a more extensive laminectomy has been carried out. Emphasis must be laid on another aspect: the age of many of the patients is such that reparative powers have already waned. It is probably significant that in the group of 13 patients who improved considerably, only 3 are over the age of 50; thus it includes the bulk of the younger patients.

Summary

Cervical spondylosis may lead to changes in the spinal cord conveniently termed myelopathy, in which compression of the cord by intraspinal osteophytes and ischaemia by interference with its blood supply both play a part to an unknown degree. The results of 39 cases of spondylotic myelopathy treated by operation are reviewed. The duration of the symptoms varied between 6 weeks and 20 years, and the ages of the patients ranged from 30 to 70 years.

Disturbance of function of the cervical spine was insignificant. Weakness or stiffness of one or both legs was the commonest initial symptom—present in 15 cases.

In the upper limbs, weakness, wasting, flaccidity or spasticity, and increase or decrease of tendon reflexes occurred in all but 2 cases. In the lower limbs there was moderate to marked spastic weakness in 27 cases. Sensory disturbances of all varieties occurred in the majority of cases; detection of these, however, may need care.

Disturbances of micturition, rarely severe, occurred in only a minority.

Radiography, including myelography, is essential for diagnosis. Osteophytes may be present at only one level or may be widely distributed, in the latter event producing an extensive cord lesion.

Operation comprised laminectomy, the arch above and below the disk being removed. The ligamenta denticulata have also been divided, and where necessary the intervertebral foramina have been enlarged. In three cases spinal fusion was carried out.

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AETIOLOGICAL FACTORS IN THE PANCREATITIS SYNDROME

BY

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The major obstacle to effective diagnosis and treatment of diseases of the pancreas is the paucity of knowledge of the aetiological factors in pancreatitis. Since the single case reported by Halsted (1901) and Opie (1901), the attention of both clinicians and pathologists has been focused upon the possibility that pancreatitis is the result of impaction of gall-stones in the ampulla of Vater with retrograde flow of bile up the main pancreatic duct. However, many cases of pancreatitis occurring in the absence of cholelithiasis have been reported, and many patients with stones in the common bile duct do not develop pancreatitis. There is, in addition, accumulating evidence of the role of infective, traumatic, and metabolic agents in the genesis of pancreatitis, while the contention of Whipple (1907) that no fundamental distinction can be drawn between acute and chronic pancreatitis has been strongly supported by more recent work (Comfort *et al.*, 1946). It is now certain that "pancreatitis" represents not a disease entity but a syndrome due to pathological processes of differing aetiology affecting the pancreas, relatively few of which are initially inflammatory in nature (Saint and Weiden, 1953).

For these reasons the present paper reports a study of 90 patients with pancreatitis in which special attention has been paid to probable aetiological factors, and presents a tentative aetiological classification of pancreatitis based upon this study. No attempt has been made to discuss fully the clinical features of these patients, for several reviews of the clinical picture of pancreatitis have been published recently, and the present findings agree with these (Comfort *et al.*, 1946; Fallis, 1951; Edmondson *et al.*, 1952; Dolan and Hopkirk, 1953).

In all cases the patients were assessed clinically. Laboratory estimations included repeated estimations of urinary diastase, blood examination, liver-function tests, estimations of serum γ-globulin and total fasting serum lipids (Kunkel *et al.*, 1948). In some patients a glucose-tolerance test, histamine test meal, and gastric biopsy (Wood *et al.*, 1949) were also performed. The biliary tree was examined radiologically by oral cholecystography and later by intravenous choledochography (Hare, 1955), whilst operative cholangiograms were obtained during laparotomy in some instances (Hughes and Kernutt, 1954a; Hughes, 1955). Laparotomy was undertaken in 51, and post-mortem examination was performed in 10 of the 90 patients. The intravenous secretin test was used in some of the first patients studied, but later was discarded as it provided little relevant information not obtainable by other methods.

Diagnostic Criteria

The diagnosis of pancreatitis was considered to be definitely established if one or more of the following criteria were satisfied: (1) Histological evidence of acute or chronic

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pancreatitis in a specimen obtained at either operation or post-mortem examination (24 cases). (2) Radiological demonstration of diffuse calcification in the pancreas (3 cases). (3) Pancreatic insufficiency, shown by the intravenous secretin test (13 cases). (4) The presence of fat necrosis at either operation or post-mortem examination (11 cases). (5) Urinary diastase level of over 200 units in the presence of clinical manifestations of pancreatitis and in the absence of other known causes of elevation of the urinary diastase (23 cases). (6) Operative diagnosis of chronic pancreatitis but without histological confirmation (43 cases).

By one or more of these criteria, a definitive diagnosis of pancreatitis was established in 70 of the 90 patients. In addition a further 20 patients have been included in whom the diagnosis was highly probable but not finally proved. These were mainly patients with a clinical picture of chronic relapsing pancreatitis who did not come to either operation or post-mortem examination, and in whom no diagnostic rise of urinary diastase was demonstrated: the majority showed increases of the urinary diastase coincident with their attacks of pain but not reaching diagnostic levels.

Results

In 68 of the 90 cases there was clinical or laboratory evidence of some factor which bore a probable aetiological relationship to the pancreatitis (Table I). In some patients more than one such factor was present. These groups, and others described in the literature, are considered in greater detail.

TABLE I

Probable Aetiological Basis of Pancreatitis	No. of Cases
Post-operative pancreatitis	2
Mumps	2
Viruses of viral hepatitis	8
Pancreatico-duodenitis	1
Hydatid disease of pancreas	1
Hyperlipaemia	6
Alcoholism	17
Associated with pregnancy	11
Haemochromatosis	1
Sprue syndrome	1
Ulcerative colitis	1
Systemic vascular lesions	17
Unknown aetiology	22
Total	90

Congenital Pancreatitis

Since the initial description of fibrocystic disease of the pancreas (mucoviscidosis) by Andersen (1938), this disease has become a well-recognized clinical and pathological entity. Other congenital lesions of the pancreas, such as anatomical variations and lipomatous pseudohypertrophy (Høyer, 1949), are not inflammatory in nature. None of the present patients showed evidence of any of these conditions.

Pancreatitis and Trauma

The occasional association of trauma and pancreatitis is well known.

External trauma, especially to the upper abdomen, may cause pancreatitis, either acutely or delayed, when the patient may present with a pseudopancreatic cyst. None of the present cases gave any history of external trauma related to the onset of pancreatitis.

Post-operative Pancreatitis.—Operative trauma may also result in pancreatitis, and post-operative pancreatitis is more frequent after biliary tract surgery, gastrectomy, and splenectomy (Warren, 1951; Dunphy *et al.*, 1953; Boles, 1954). However, the pathogenesis of post-operative pancreatitis is still in dispute. Millbourn (1949) regards injury to the duct of Santorini as an important precipitating factor, whilst Dunphy *et al.* (1953) consider many cases to be due to interference with the blood supply of the pancreas, especially the posterior pancreatico-duodenal artery. Two of the present patients developed pancreatitis post-operatively.

A woman aged 76 underwent cholecystjejunostomy for obstructive jaundice due to carcinoma of the head of the pancreas with hepatic metastases, and a man aged 62 was submitted to exploration of the common bile duct for obstructive jaundice due to stones in the common bile duct. Both died within a few days of operation, and necropsy revealed an unsuspected acute haemorrhagic pancreatitis.

Pancreatitis and Cholelithiasis

There is considerable disagreement concerning the role of cholelithiasis in the aetiology of pancreatitis. Halsted (1901) reported a fatal case of acute pancreatitis in which the post-mortem examination revealed a gall-stone impacted in the ampulla of Vater, and following this Opie (1901), as a result of this case and of experimental work in dogs, suggested that the "frequent association of cholelithiasis with haemorrhagic and gangrenous pancreatitis is the result of impaction of gall-stones at the orifice of the diverticulum of Vater and penetration of bile into the pancreas." Other investigators, including Claude Bernard, also stated that the flow of bile up the pancreatic duct produced acute pancreatitis. This hypothesis was accepted without question for many years, especially as there was little knowledge of other possible factors in the aetiology of pancreatitis. The cases in which gall-stone impaction did not produce pancreatitis were thought to be due to local anatomical variations in the duct systems. The cases of pancreatitis in the absence of cholelithiasis remained without explanation.

Recent work has thrown considerable doubt upon Opie's hypothesis. Many cases of pancreatitis without cholelithiasis have been reported, and it has become evident that, even in the presence of cholelithiasis, pancreatitis due to gall-stone impaction is a great rarity. Study of the original experimental work shows that pancreatitis may be produced in animals by the injection of bile up the main pancreatic duct only if this procedure is combined with some other procedure such as duct ligation, or the addition of some irritant chemical to the bile (Palmer, 1951; Elman and Wheat, 1954).

Of the present 90 cases, gall-stones were absent in 42, present in 26, and had been present previously in 14, whilst in 8 no definite information was available. In only 2 of the 26 cases in which cholelithiasis was present were stones detected within the common bile duct, and in neither were they impacted at the ampulla of Vater. In 14 cases pancreatitis persisted after removal of gall-stones. These figures provide no support for the theory that gall-stones bear an aetiological relation to pancreatitis.

The relationship between pancreatitis and regurgitation of bile up the duct of Wirsung has also been clarified in recent years. Anatomical studies by Sterling (1954) and by Hughes and Kernutt (1954b) have shown that a common functioning channel for bile and pancreatic fluids exists in less than 15% of cases, and that the so-called "fibrosis" of the sphincter of Oddi is a normal and not a pathological finding. In addition, functional studies by Hicken and McAllister (1952) and by Hughes and Kernutt (1954a), and increasing experience with cholangiography (Hare, 1955; Hughes, 1955) show that in man some degree of reflux up the duct of Wirsung is a frequent and normal state, unrelated to the presence or absence of pancreatic disease.

There is, however, a definite clinical and pathological association of pancreatitis and cholelithiasis, and this may be due to two separate mechanisms. Obstruction of the common bile duct, either partial or complete, is a not infrequent complication of pancreatitis, being observed in 23 of the present 90 cases. This obstruction may well result in cholelithiasis secondary to the pancreatitis. Secondly, there is considerable evidence that pancreatitis may result from hyperlipaemia (see below), and it is possible that this hyperlipaemia may bear an aetiological relation to both cholelithiasis and pancreatitis. It is of interest that five of six patients with pancreatitis due to hyperlipaemia had chole-

lithiasis, but 11 of 17 with alcoholic pancreatitis had no cholelithiasis. The relation between cholelithiasis and pregnancy, which is also related to pancreatitis, has been discussed elsewhere (Gross, 1929; Joske *et al.*, 1954).

For these reasons it is considered that cholelithiasis and biliary reflux bear little if any aetiological relation to pancreatitis, and this hypothesis has not been invoked in any of the present cases.

Infective Pancreatitis

Virus Pancreatitis.—There is considerable evidence that some cases of pancreatitis are due to direct viral infection of the pancreas. The association of mumps and pancreatitis is well established, and Hausmann (1954) found the urinary diastase raised in 55 of 65 children with mumps. Myhre and Nesbitt (1949) reported pancreatitis complicating infectious mononucleosis. Lisney (1944) first described pancreatitis following virus hepatitis, and this has been confirmed by Sarles (1950) and Arias Vallejo (1951). Two of the present cases developed chronic pancreatitis directly following an attack of mumps: both were females in the third decade. In eight cases pancreatitis was associated with viral hepatitis. These will be reported fully elsewhere. Three of these patients died of fulminating acute viral hepatitis, and acute pancreatitis was also present at post-mortem examination. Three with less severe acute hepatitis showed concomitant elevation of the urinary diastase, one having a typical attack of acute pancreatitis. Two had chronic relapsing pancreatitis associated with viral hepatitis. It is also possible that other agents, such as the Coxsackie and "orphan" viruses, may occasionally cause pancreatitis in man (Pappenheimer *et al.*, 1950; Steigman *et al.*, 1953).

Bacterial Pancreatitis.—The evidence for primary bacterial pancreatitis is much less strong, although the pancreas may be involved by direct spread of infection from an adjacent focus such as a penetrating peptic ulcer or an overlying peritonitis. Secondary bacterial infection of pancreatitis due to other causes is also frequent, playing a large part in the mortality of pancreatitis before the advent of antibiotic therapy (Schweinburg *et al.*, 1953). There is, however, no evidence that primary bacterial pancreatitis occurs in man. Bacteriological studies on the present cases were not made, because most were treated conservatively and received antibiotic therapy.

Pancreatico-duodenitis.—Bove (1953) has suggested that the primary lesion in some cases is an inflammatory process of the choledocho-pancreatico-duodenal junction, which may produce jaundice, cholelithiasis, and pancreatitis. One of the present cases conforms to his description.

A man aged 44 presented with recent obstructive jaundice. At operation the pancreas was enlarged and inflamed and the ampulla of Vater enlarged, reddened, and projecting into the duodenum. Biopsies showed acute-on-chronic pancreatitis and acute diffuse inflammation of the ampulla. No gall-stones were present. His symptoms were relieved by cholecystenterostomy. A later gastric biopsy showed atrophic gastritis. No infective agent was isolated in this case.

There is thus little doubt that pancreatico-duodenitis is an entity, but its aetiology and significance remain to be assessed.

Parasitic Infestations.—Of the various parasitic infestations, several cases of pancreatitis due to ascariasis have been reported (Ochsner *et al.*, 1949). One case of hydatid infestation of the pancreas is included in the present series.

A man aged 44 first presented with obstructive jaundice, and at operation was found to have a hydatid cyst of the head of the pancreas pressing upon the common bile duct. Despite removal of the cyst he developed pancreatic insufficiency and diffuse calcification of the pancreas some years later.

Chemical Pancreatitis

Several chemical agents are capable of causing pancreatitis (Apolant, 1913). These include boric acid (Fisher, 1951), carbon tetrachloride (Speckmann, 1953), methyl alcohol (Bennett *et al.*, 1952), and methionine (Goldberg and Chaikoff, 1951). No such agents could be incriminated in the present cases. The relation between alcoholism and pancreatitis is discussed below.

"Metabolic" Pancreatitis

There is abundant evidence that acute and chronic pancreatitis may result from general disorders of metabolism. These include essential hyperlipaemia, pregnancy, alcoholism, and malnutrition.

Hyperlipaemia and Pancreatitis.—The occurrence of hyperlipaemia in acute pancreatitis was first reported by Speck in 1846. Thannhauser (1940) and Collett and Kennedy (1948) also described chronic pancreatitis with hyperlipaemia. However, it was not until 1950 that Poulsen (1950) and later Klatskin and Gordon (1952) clearly showed that in some instances pancreatitis was a result of the genetically determined disease "essential hyperlipaemia." The literature has been reviewed by Klatskin and Gordon (1952) and Joske (1955a). Of the present 90 cases, six were considered to be suffering from pancreatitis secondary to essential hyperlipaemia, although the fasting serum lipids were estimated in only 57 of the 90 cases. The six patients ranged in age from 30 to 58 years; two were males and four females; four had cholelithiasis; four had significant atherosclerosis; three had xanthelasma; their fasting serum lipids ranged from 870 to over 2,000 mg. per 100 ml., the upper limit of normal in this laboratory being 650 mg. per 100 ml. It is of interest that one of these patients presented with "acute pancreatitis" two months after parturition: a similar case has been recorded by Comfort and Steinberg (1952).

Pregnancy and Pancreatitis.—The relation between pregnancy and pancreatitis has been fully reviewed by Langmade and Edmondson (1951) and Joske (1955b), and it has been suggested that in women suffering from pancreatitis the onset is frequently related to pregnancy, symptoms commencing either late in pregnancy or in the puerperium. This syndrome has been termed "post-partum pancreatitis." In 11 of the present 90 cases the onset of symptoms was clearly related to pregnancy. Six of these cases have been reported in full in a previous communication (Joske, 1955b). Of the 11 patients, 8 were young women between 19 and 25 years of age, whilst 3 were older, being aged 58, 59, and 61 years. These latter had chronic relapsing pancreatitis for periods of some 30 years. Only one of them had cholelithiasis; none had hyperlipaemia, vascular disease, or diabetes mellitus, although one had a mild diabetic glucose-tolerance curve. In addition, a further patient with hyperlipaemia, mentioned above, first developed symptoms shortly after pregnancy. The mechanism of this type of pancreatitis is still obscure. Oliver and Boyd (1955) have investigated the lipaemia of pregnancy and shown that the serum electrophoretic pattern in late pregnancy resembles that seen in coronary sclerosis. FitzGerald (1955) has emphasized the frequency of minor degrees of pancreatic dysfunction during pregnancy and suggested that post-partum pancreatitis might be a rebound phenomenon. It is of interest, therefore, that MacFarlane and Norman (1954) have shown the serum antithrombin titre to be decreased during pregnancy, for it is greatly increased in acute pancreatitis (Innerfield *et al.*, 1952).

Alcoholism and Pancreatitis.—The association between alcoholism and pancreatitis has been established for many years (Opie, 1902; Egdahl, 1907), and alcoholism was considered responsible for pancreatitis in 17 of the present 90 patients. All were males. Gall-stones were absent in 13, present in 2, and undetermined in 2 of the 17 patients. The pancreatitis was chronic in 13 and acute in 4, being fatal in 2 of the latter. The mechanism whereby alcoholism results in pancreatitis is uncertain. It is improbable that malnutrition alone is responsible, for in some cases (2 of the present 17) ingestion of only small amounts of alcohol results in severe pancreatic pain. This is a similar "trigger"

mechanism to that seen in Hodgkin's disease (Bichel and Bastrup-Madsen, 1953; de Winter, 1953). Several authors have reported elevations of serum amylase in acute alcoholism (Culotta and Howard, 1954). In patients showing this "trigger" effect it appears that some factor besides alcoholism is necessary to produce pancreatitis. Of the two such cases in the present series, one had a minor degree of hyperlipaemia (fasting serum lipids 880 mg. per 100 ml.), whilst the other had a strong family history of biliary disease in both his parents and in his siblings, though without hyperlipaemia. The remaining 15 patients were chronic alcoholics, and no such "trigger" mechanism was apparent in these cases. However, it may be noted that 6 of the 15 were hypertensive.

Other Metabolic Causes of Pancreatitis.—One patient with chronic pancreatitis due to haemochromatosis is included in this series. Althausen *et al.* (1951) showed that impaired pancreatic function is usual in haemochromatosis. Transfusion siderosis may also involve the pancreas (Stewart, 1953). Acute pancreatitis complicating acute porphyria has recently been described (Saint *et al.*, 1954).

Malnutrition and Pancreatitis.—It appears probable that malnutrition may also result in pancreatitis, for pancreatitis has been reported in undernourished natives in the West Indies (Waterlow, 1948; Davies, 1948), in ulcerative colitis, and in rats fed a diet in which protein was low or absent (Lindsay *et al.*, 1948; Wachstein and Meisel, 1954). Linder *et al.* (1953) found pancreatic atrophy *post mortem* in a patient who died of malnutrition three and a half years after a massive resection of the small intestine. Malnutrition appeared to be the basis of pancreatitis in two of the present cases—a woman aged 26, with chronic ulcerative colitis and chronic pancreatitis producing obstructive jaundice, and a man aged 59 with the sprue syndrome due to abdominal reticulosis.

Pancreatitis and Vascular Disease

The relation between vascular disorders and pancreatitis has not yet been clarified. It has been suggested that ischaemia of the pancreas may be the final common pathway whereby other primary causes produce pancreatitis. This has been postulated for post-operative pancreatitis by Millbourn (1949), and for pancreatitis complicating acute porphyria (Saint *et al.*, 1954). Hyperlipaemia also results in ischaemic vascular disease (Malmros *et al.*, 1954; Lever *et al.*, 1954). Thal and Brackney (1954) produced acute haemorrhagic pancreatitis in animals by a local Shwartzman reaction, using *Bact. coli* endotoxin as antibody. Harvey *et al.* (1954) reported acute pancreatitis in systemic lupus erythematosus.

The blood vessels supplying the pancreas may also be involved in generalized vascular disease. Heinz (1952) has stressed the association of hypertension and pancreatitis. Baggenstoss (1948) noted a high incidence of pancreatic parenchymal lesions in nephrosclerosis, and in malignant hypertension (Hranilovich and Baggenstoss, 1953). Burn (1951) found a close association between acute coronary thrombosis and acute pancreatitis, and this has been confirmed by Saint (1954) and by Bauerlein and Stobbe (1954).

In 17 of the present 90 cases it is considered that pancreatitis was probably due to primary vascular disease, and these patients showed a very characteristic clinical picture.

The 17 patients comprised 5 males and 12 females, their ages ranging from 48 to 81 years (average 63 years). Hypertension (resting blood pressure greater than 160/90 mm. Hg) was present in 12, and significant atherosclerosis in 15, three having gross cerebral arterial disease. Emphysema was also noted in the majority. None provided a family history of cholelithiasis or diabetes mellitus. Cholelithiasis (past or present) was found in all except 4 of the 17. Gastric function was investigated in 14, and achlorhydria or atrophic gastritis was demonstrated in 11 cases. Six had diabetes mellitus. In addition, one of these patients had a large aneurysm of the splenic artery at laparotomy (Hughes and Joske, 1955). Hyperlipaemia was not present in any of these cases.

This group of lesions—atherosclerosis with or without hypertension, emphysema, cholelithiasis, atrophic gastritis, and chronic pancreatitis—appears to form a definite syndrome which is capable of clinical recognition. This type of pancreatitis is provisionally termed "degenerative pancreatitis," and is thought to be a result of chronic arterial disease producing pancreatic ischaemia, which is slowly progressive. It is also possible that this disease complex is related to an anomaly of fat metabolism the nature of which is unknown. This group of patients is largely responsible for the high incidence of gastritis observed in patients with pancreatitis (Joske *et al.*, 1955).

Pancreatitis of Unknown Aetiology

In 22 of the 90 instances it was not possible to ascribe pancreatitis to any particular aetiological factor. In some cases this may have been due to incomplete clinical or laboratory investigation, in that a history of alcoholism or a relation to pregnancy was not elicited, or no estimation of fasting serum lipids was possible.

In others, some of whom were obese, the pancreatitis appeared to be secondary to some undiagnosed metabolic upset related to fat metabolism. Thus an obese woman aged 50 presented with the post-cholecystectomy syndrome and was shown to have chronic relapsing pancreatitis: her mother and five sisters all had cholelithiasis. A man aged 23 developed relapsing pancreatitis: his mother had diabetes mellitus and his two sisters had undergone cholecystectomy. A woman aged 55 developed the post-cholecystectomy syndrome due to chronic pancreatitis: she also had a spastic paraplegia, xanthomatosis, and later developed diabetes mellitus, while her mother suffered from angina pectoris. In these and four other cases pancreatitis is probably a result of some inborn metabolic derangement.

Nevertheless these 22 patients still present an unsolved clinical problem upon which further work is necessary. In particular the aetiology of chronic relapsing pancreatitis in some younger patients is still completely unknown.

Discussion

There are four stages in the production of the clinical picture of pancreatitis. Some initiating factor must be present and cause changes in the pancreas; these changes must then result in tissue destruction and liberation of pancreatic enzymes, while in chronic disease pancreatic insufficiency is added to this chemical and/or bacterial inflammation. The present study is concerned only with the first of these problems—what are the factors that may initiate the group of changes, both acute and chronic, collectively termed "pancreatitis"? The relative roles of ischaemia, the functional changes within the duct system stressed by Archibald (1929), direct necrosis of the exocrine cells, added bacterial infection, enzymatic changes due to lipase and trypsin, and pancreatic insufficiency in the production of clinical pancreatitis constitute a second problem which is not within the province of the present paper.

It is apparent that pancreatitis is not a disease entity but a syndrome due to different causes in different cases, many of which are not initially inflammatory, and should be termed "pancreatopathy" rather than "pancreatitis." It is probable, but not yet proved, that these differing aetiological factors are reflected by differences in the local pathological changes within the pancreas and in the natural history of the clinical disease produced. For this reason it is unjustifiable to treat all patients with "pancreatitis" alike or to assess the prognosis in an individual patient without regard to the probable aetiology of the disease in that case.

Of the present 90 cases, some reasonable aetiological factor was identified in 68; the remaining 22 were classed

as "pancreatitis of unknown aetiology." From this experience an aetiological classification of the pancreatitis syndrome has been constructed, and this is shown in Table II.

TABLE II.—*An Aetiological Classification of the Pancreatitis Syndrome*

1. Congenital pancreatitis:
   - Fibrocystic disease of the pancreas
2. Traumatic pancreatitis:
   - External trauma
   - Post-operative pancreatitis
   - ? Pancreatitis due to cholelithiasis
3. Infective pancreatitis:
   - Viral infections:
     - Mumps
     - Infectious mononucleosis
     - Hepatitis viruses
   - ? Bacterial infections:
     - "Pancreatico-duodenitis"
   - Parasitic infections:
     - Ascariasis
     - Hydatid disease
4. Pancreatitis due to extrinsic chemical agents
5. Metabolic pancreatitis:
   - Hyperlipaemia
   - Pregnancy group
   - Alcoholism
   - Haemochromatosis haemosiderosis
   - Acute porphyria
   - Malnutrition
6. Pancreatitis secondary to systemic vascular disease
7. Pancreatitis of unknown aetiology

It must be emphasized that this classification is still tentative, and will undoubtedly require modification and expansion or contraction as further study is made of the pancreatitis syndrome. Nevertheless it provides a pragmatic basis for the clinical and laboratory assessment and the management of patients with pancreatitis, and for further investigation of the pancreatitis syndrome itself.

Summary

Ninety patients with pancreatitis have been studied with particular reference to aetiology.

Some aetiological factor was identified in 68 of the 90 cases. These included alcoholism (17 cases), vascular lesions (17 cases), pregnancy (11 cases), virus infection (10 cases), hyperlipaemia (6 cases), post-operative pancreatitis (2 cases), and one case each of pancreaticoduodenitis, hydatid disease, haemochromatosis, sprue syndrome, and ulcerative colitis. In 22 cases the aetiology was unknown.

Analysis of the present cases and a survey of the literature suggest strongly that gall-stone impaction and biliary reflux are unimportant in the aetiology of pancreatitis in man.

An aetiological classification of the pancreatitis syndrome is proposed.

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