ACUTE PANCREATITIS*

A CLINICAL EVALUATION AND REVIEW OF 154 CASES

LOUIS J. MORSE, M.D. AND
SAMUEL ACHS, M.D.

BROOKLYN, N. Y.

FROM THE SURGICAL SERVICES OF THE JEWISH HOSPITAL OF BROOKLYN

TWENTY YEARS AGO, one of us had the opportunity of reviewing 88 cases of acute pancreatitis with Dr. William Linder.[35] The present analysis includes an additional 66 cases, a total of 154 cases operated upon between 1913 and 1948. In our paper of 1929, we stressed the importance of diagnosis before abdominal section. The former concept, that acute pancreatitis can only be diagnosed at the operating table, is no longer true. Fitz indicated that this abdominal catastrophe should be suspected whenever violent epigastric pain is followed by vomiting and collapse. This occurs in fulminating cases. Little can be learned or done about this type, but acute pancreatitis, if borne in mind whenever an acute abdominal mishap presents itself, offers no greater difficulty in diagnosis than any other surgical condition.

The pathogenesis of acute pancreatitis, its differential diagnosis and treatment, will be discussed in some detail. The accompanying tables are meant to convey the pertinent statistical findings in the 154 cases analyzed.

ETIOLOGY

Acute pancreatic disease may be infectious or noninfectious. The proponents of the infectious theory point out the possibility of extension of infection along the lymphatics; by pyemic involvement via the blood stream; by direct contiguity; by extension along the pancreatic ducts from the duodenum or bile ducts; by activation by bacteria in the normal pancreas; or finally, by bacterial permeation from adjacent altered viscera.

In this connection, it may be of some interest to note that neither Truhart,[53] in 43 cases, nor Jones[30] found bacteria present in the pancreas. When we realize how infrequently acute pancreatitis is encountered in conjunction with perforation of duodenal or gastric ulcers into the pancreas, the rarity of the incidence of infection by contiguity becomes apparent.

Infection may be a potent factor indirectly. In late cases, the bacteria most frequently isolated are *E. Coli,* pneumococcus and staphylococcus. Brocq[8] cultured anaerobes from the gangrenous variety, but the necrotic type was sterile. Experimentally, Carnot,[10] and Korte[32] produced acute pancreatitis by injecting colon and pyocyaneus bacilli, and other bacteria into the pancreatic duct. Archibald[2] produced fatal pancreatitis by colon bacillus injection. Kemp[31] stated that certain bacteria, especially of the typhoid and colon groups, are capable of activating proteolytic pancreatic enzymes.

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Lymphogenous infection from the gallbladder, liver and appendix has been emphasized in both acute and chronic pancreatitis by Maugeret,[37] Arnsperger,[4] Deaver,[15] Graham[26] and others; but whereas most observers agree that this may be the modus operandi in the chronic type, the acute process has never been produced experimentally in this manner.

Jones[30] believes chronic pancreatitis to be the result of interstitial tissue inflammation produced by lymphatic extension, whereas acute pancreatitis involves the parenchyma via the pancreatic ducts.

Gastro-intestinal infection may reach the liver through the portal system and thence extend to the pancreatic gland.

Hematogenous pancreatitis through bacterial metastasis is rare, but pancreatic inflammation has been reported in conjunction with epidemic parotitis and infectious thrombophlebitic processes.

Rosenow,[47] experimentally, produced cholecystitis and pancreatitis by injecting intravenously streptococci obtained from the tonsils of patients with gallbladder symptoms.

The non-infectious theory presupposes first, mechanical stasis, and second, the chemical activation of inactive ferments resulting from autolysis of bile, duodenal contents or degenerated duct contents. Although associated biliary disease is a concomitant finding in the majority of cases, those without apparent cholecystitis must be explained. In these, toxic products may develop within the pancreas or may be regurgitated from the biliary tract or duodenum into the pancreatic ducts.

Inactive pancreatic juice is harmless. Experimental ligation of the pancreatic duct produces no necrosis. If, however, ligation be effected at the height of digestion, pancreatic necrosis ensues. Section of the duct, with escape of inactive juice into the peritoneal cavity, produces neither pancreatic involvement nor clinical symptoms. Simple pancreatic injury fails to produce necrosis, even though activating substance may be liberated.

Levin[33] crushed the pancreas when associated blood vessel occlusion existed and produced characteristic hemorrhagic necrosis. Clinically, trauma has frequently been followed by acute pancreatitis.

Bile for many years has been recognized as a factor in the production of acute pancreatitis. Bradley and Taylor[7] found that bile did not activate the enzymes. Others believe that bile merely accelerates the action of the pancreatic ferments. When bile was brought into contact with the pancreas, acute hemorrhagic pancreatitis resulted. It has been demonstrated that injection of small amounts of bile into the duct of Wirsung produces acute pancreatic disease, if done at the height of digestion. Opie[41] found that considerable amounts of bile were necessary. Nordmann[40] produced very little reaction with sterile bile, but infected bile was more potent in the production of pancreatic necrosis. Sodium taurocholate appears to be the offending substance. It is found in abundance in infected bile. Focal liver necrosis is noted early in acute pancreatitis.

According to the regurgitation theory, three factors are involved in the production of acute pancreatitis. First, a change in bile composition; second, undue resistance, such as is produced by stone or spasm; and third, elevation of gallbladder pressure or bile duct pressure sufficient to produce reflux of bile into the pancreas.

Clinically, bile may enter pancreatic ducts following obstruction of the common outlet in cases where bile and pancreatic ducts join, or by regurgitation of duodenal contents.

Here a few anatomical observations may not be amiss. Mann and Giardano[36] found that in only 3.5 per cent of subjects did the anatomic arrangement of bile and pancreatic ducts permit of common channel production. Cameron and Noble[9] concluded that in 75 per cent of individuals a calculus impacted at the papilla could produce a common passageway. It should be recalled that stones were found in the ampulla in but 5.4 per cent of 1287 cases, one of these the celebrated case of Opie, operated upon successfully by Halstead. It served, however, as an impetus to the study of pancreatitis.

Zuckerkandl[61] found that the accessory and main pancreatic ducts usually communicate by a wide opening, close to the duodenal end. Opie[41] found this situation in 90 of 100 dissections. Baldwin[5] found that the common duct opened separately into the duodenum in 25.8 per cent and in a common ampulla in 74.2 per cent.

Obstruction may be due to stone, spasm, inflammatory swelling, mucus, parasites, and even venous congestion due to cardiac decompensation. Spasm can be induced by dilute hydrochloric acid in the duodenum. It is also increased by gastroduodenitis and dietary indiscretion.

Duodenal contents in the absence of infection may produce acute pancreatitis. Polya[42] accomplished this by injecting small amounts of duodenal contents into the pancreatic duct. In support of the duodenal-origin hypothesis is a large number of cases where biliary disease is not apparent. Regurgitation may occur in acute or chronic dilatation, or aided by antiperistalsis, in the presence of a relaxed or dilated sphincter of Oddi.

Brocq[8] completely obstructed the duodenum following gastroenterostomy and produced pancreatic necrosis. Seidel[50] accomplished the same by causing stasis in the duodenum. Opie suggested the presence of delicate valves in the diverticulum of Vater, that normally prevent regurgitation.

Brocq suggested that predominance of the hemorrhagic feature in acute pancreatitis is the result of the activation of trypsin by regurgitated duodenal contents, while preponderant fat necrosis is due to bile regurgitation.

Substances that have been utilized to produce acute pancreatitis experimentally are diphtheria toxin, acid gastric juice, alkalies, calcium chloride, formaldehyde and leucocytes with bacteria.

Rich and Duff[45] have described a peculiar hyaline necrosis of the walls of the pancreatic arteries and veins in acute pancreatitis. They believed necrosis to be the initial lesion, and to be due to the extravasation of active trypsin.

They observed a remarkable metaplasia of the pancreatic duct epithelium in 13 of 24 cases examined, and since reflux of bile, infection and obstruction account for but two-thirds of the cases, they suggested that duct obstruction caused by this proliferation might be responsible for escape of trypsin, and consequently of hemorrhagic pancreatitis by thrombus and hemorrhagic production.

Inspissated secretion in the pancreatic ducts appears to be another cause. Acute pancreatitis may be encountered concomitant with degenerative pancreatic processes such as tumors, vascular degeneration, toxic changes in systemic disease, and trauma.

Clinically, acute pancreatitis is not a primary disease, but rather a complication or sequela. Whether we subscribe to one or another of the hypotheses, the realization that biliary disease is so frequently the etiologic precursor must be evident. Egdahl's[19] incidence of biliary calculi in 50 per cent and cholecystitis in 75 per cent is not mere coincidence. Where no biliary disease is apparent, it is well to recall Hess'[29] work. He produced acute pancreatic necrosis by ligating the pancreatic duct at the height of digestion. Pratt[43] did the same in fasting animals with no resultant necrosis. Notwithstanding all the possible methods of, and etiologic factors in the production of acute pancreatitis, we may assume the great majority to be due to biliary disease. Certainly these latter cases are those with which we are most frequently confronted.

Fat necrosis has been taken as the diagnostic criterion of the 154 cases herein analyzed. That acute pancreatitis does exist without fat necrosis has been proved clinically, but it was deemed advisable to include only those cases in which fat necrosis was demonstrable to prevent the possible inclusion of doubtful cases.

CLASSIFICATION

There is ample justification for considering acute edematous or interstitial pancreatitis as a milder grade of acute pancreatic necrosis, which may be predominantly hemorrhagic, necrotic or suppurative. Quick[44] operated upon a patient with edematous pancreatitis only to find two days later that she succumbed to extensive pancreatic necrosis as demonstrated at autopsy. Cole's[12] work offered corroboration by the finding, in six cases of acute pancreatic edema, of a sharp rise in blood amylase, a very significant observation in all forms of acute pancreatitis.

INCIDENCE

Seventy-six per cent of our cases involved females. The female-male ratio was the same in acute pancreatitis as in our cases of gallbladder disease, though Riese's[46] and Korte's[32] observations were to the contrary. The former observed males predominating, 79 to 42, the latter 30 to 14. If we regard biliary disease as the important etiologic factor, males and females are alike affected. Forty-five cases herein included occurred in a period during which

2049 cases of biliary disease were submitted to operation, an incidence of approximately 2 per cent.

Fifty per cent of our cases occurred in the fourth and fifth decades. The youngest patient was 19, the oldest 73.

Previous gastro-intestinal disorders are of considerable significance. All but 19 gave a history of some form of "dyspepsia" and of these, six had had repeated alimentary disturbances for three to four weeks prior to hospital admission. Sixty-three per cent gave a definite history of biliary disease.

Alcoholism, in the literature, constitutes one of the cogent factors in acute pancreatitis. When we realize that no case is permitted to enter our institution in which alcoholism is a predominating symptom, it becomes apparent why this series contains no case in which alcoholic diathesis plays any role.

PATHOGENESIS

Pancreatic symptomatology in its acute manifestations depends on the activation of trypsinogen into trypsin. Physiologically this activation occurs in the duodenum through stimulation by succus entericus. Protein digestion by this liberated enzyme may affect blood vessels, lymph structures or pancreatic cells. Activation is furthered by the products of cytolysis and by the calcium of extravasated blood serum. The liberated trypsin destroys protein molecules which have been combined with bile salts, thus setting the latter free to produce further cell damage. The resultant edema and sanguination may be extensive and severe in a very short time. If the process terminates at this stage, complete restitution may ensue. Progression may result in local or diffuse necrosis of the entire parenchyma.

Circulation of foreign protein in the form of incomplete protein digestion products may in part serve to explain the severe toxic picture. Some believe the significant product of proteolysis to be histamine, produced in the necrotic tissue or in the peritoneal fluid. Dragstedt[17] showed that intraperitoneal fluid and products of pancreatic autolysis, when injected, were not toxic. However, when these were infected, the characteristic symptoms developed. Experimental intravenous injection of peritoneal fluid from cases of acute pancreatitis by Cooke and Whipple,[13] was without toxic effect.

Blood vessel digestion contributes the hemorrhagic features. Peritonitis is due to toxic products circulating in the subserous lymphatics. Thus, the entire syndrome depicts a definite clinical entity that can be recognized.

Fat necrosis in acute pancreatitis may involve the fat of the abdominal wall, pericardium or pleura. The dissemination of this necrosis depends on the circulation of lipase in the blood. Wood[60] suggests that the activation of steapsinogen in the pancreas is by some constituent of the bile, and Flexner[24] demonstrated a fat splitting enzyme in necrotic foci. Splitting of a fat into soluble glycerin and fatty acid results in deposition of the latter as crystals, which later, with calcium, form globular masses surrounded by zones of round cell infiltration. Wells[57] found that a moderate amount of these necrotic areas might be absorbed in a week.

Digestion of pancreatic cells liberates their contained trypsin and lipase, and consequently not only these two enzymes, but the products of their complete and incomplete digestion as well, are circulated through the lymph channels. Therefore, symptoms of acute pancreatitis are due first to local pancreatic irritation, and second to the circulation of toxic products.

SYMPTOMS AND SIGNS

Pancreatic inflammatory reaction results in edema and swelling, with resultant stretching of the pancreatic capsule. This causes pain and obstructs diaphragmatic excursion. Bile drainage becomes impeded by common duct obstruction. Pressure on the abdominal brain, the celiac plexus, must be considered in the explanation of acute pancreatic symptomatology at least in so far as local manifestations are concerned.

Of all the symptoms, constant epigastric pain is most frequently encountered. This pain is splitting in character, "as though something within were boring its way through." The localization of pain in this series is shown in the accompanying Table I. Pain in acute pancreatitis is of intense severity. Morphine in repeated doses fails to relieve it. It is interesting to note that patients volunteer the information that a hypodermic injection has failed to ease their discomfort, whereas during previous attacks, relief had been immediate from one dose of narcotic.

Vomiting is a troublesome, persistent complaint. However, it is never progressive and never fecal. One gastric lavage usually suffices for relief, a differential feature from high intestinal obstruction. Coffee ground or bloody vomitus may occur, and is easily explicable when one considers the proximity of the stomach to the hemorrhagic pancreas.

Protein split products introduced into the circulation call forth a peculiar train of symptoms designated allergic or anaphylactic. At times these symptoms predominate. Shock, collapse, cyanosis, dyspnea, and in some instances, dermatologic reactions (urticaria) occur. The significance to the clinical picture of pressure on the celiac plexus can merely be mentioned, but Whipple[58] pointed out a peculiar asthenia characteristic of pancreatic disease. Cyanosis may in part be due to diaphragmatic embarrassment resulting from local pancreatic swelling.

Temperature plays little, if any, role in the diagnosis. Jaundice may be due to local pancreatic pressure on the ductus choledochus. It is never severe. Concomitant liver necrosis may influence the degree of jaundice, as will the presence of stones in the common duct. Acute yellow atrophy has been found as a complication of acute pancreatitis.

Peritonitis, whether bacterial or chemical in origin, produces the same symptoms, local or diffuse. Local peritonitis due to mechanical stretching of the peritoneum over the pancreas adds the local objective signs to the diffuse ones caused by the chemical irritant.

Constipation is not uncommon. Diarrhea, when it occurs, is usually a late manifestation indicating considerable pancreatic destruction. Constipation is

not complete, a fact of value in differentiating low intestinal obstruction. The silent paretic abdomen of acute pancreatitis offers a marked contrast to the abdominal findings in mechanical obstruction. Turner[54] described a bluish-brown discoloration of the skin overlying the left flank and back. This has never been seen by us. He also mentioned the diagnostic value of the para-umbilical skin discoloration, which was similarly lacking in our series (Cullen's sign).

Statistical resumés help but little in the diagnosis of acute pancreatitis. However, when a patient with a history of gallbladder disease presents a somewhat altered symptom complex, if morphine fails to alleviate the pain,

TABLE I.—*Pain in Acute Pancreatitis (154 Cases)*

Location	Percentage
Epigastric	87%
Right hypochondriac	61
Left hypochondriac	28
Left lumbar	54
General abdominal	30

which now radiates from epigastrium transversely to the left, if cyanosis and slight icterus, together with a peculiar asthenia and dyspnea occur, acute pancreatitis must be considered. Tenderness over the left hypochondrium and left costo-vertebral area should strengthen the clinical impression.

Tables I and II depict the location of pain and tenderness in our present series. In Table III, the more pertinent signs and symptoms, other than pain, are indicated.

TABLE II.—*Tenderness in Acute Pancreatitis (154 Cases)*

Location	Percentage
Epigastric	58%
Right hypochondriac	59
Left hypochondriac	30
Left costovertebral	37
General abdominal	26

DIFFERENTIAL DIAGNOSIS

Symptomatology must be clearly analyzed, for acute pancreatitis may simulate other acute abdominal entities. Acute empyematous cholecystitis may be troublesome in the differential diagnosis. This is especially true when impending perforation and local peritonitis exist. The presence of a palpable mass which attaches itself to the lower border of the liver aids greatly. Pear-shaped or globular, this mass denotes a distended gallbladder due to cystic duct block. Irregularity or bogginess of the mass may be caused by adherent omentum protecting an impending blow-out, or an already perforated gallbladder. Because of its superficial position, light palpation performed by placing the

palm of the hand on the abdomen with the patient breathing slowly and deeply, easily discloses the mass. It is significant that only 7 per cent of our cases of acute pancreatitis had acute cholecystitis at the same time.

Perforation of a diseased gallbladder into the general peritoneal cavity produces shock simulating that of acute pancreatitis, but the differential diagnosis may be made by the history of a previously distended, acutely inflamed gallbladder, where medical treatment failed to relieve the cystic duct block. Gallbladders do perforate, but if the sudden onset of acute pancreatitis is borne in mind, and the local findings enumerated above are absent, a correct diagnosis can be made.

Acute intestinal obstruction, especially the high variety, may offer some difficulty. Although the shock produced by the sudden snaring of a loop of gut by a band or internal hernia may be great, the early visible peristalsis, the progressive vomiting, the painless abdomen, the absence of epigastric and left costovertebral tenderness and the absent malar flush, all assist in making a correct diagnosis.

TABLE III.—*Other Symptomatology (154 Cases)*

	Percentage
Cyanosis	45%
Shock	17
Emesis	88
Abdominal distention	56
Jaundice	27
Urticaria	3

Left renal colic, particularly when attended by extreme pain and shock, offers some difficulty. If radiation of pain to the groin is absent, acute pancreatitis must be considered. Pyuria or the presence of erythrocytes in the urine certainly help. Sudden torsion of a ptotic kidney with renal pedicle strangulation simulates pancreatitis. The history of ptosis in a patient with considerable weight loss and the picture of Dietl's crisis following sudden exertion supports the diagnosis. The kidney may be readily felt, and found enlarged and tender, if it is palpated in the lumbar region. If it is displaced to the midline its mobility can be demonstrated, and it may be reposited in the lumbar fossa.

Tenderness in the left inguinal region, a definite Head zone, is not infrequent in acute pancreatitis, particularly when considerable bloody fluid is present in the peritoneal cavity.

Acute carbunculosis of the left kidney calls for discriminatory analysis. Here, the antecedent history of some focal infection, coupled with sudden onset of chill, rise in temperature, tenderness and spasticity in either lumbar region, occurring in an otherwise healthy individual, even in the absence of pertinent urinary findings, are differential characteristics. If the anterior surface of the nephros is the site of adhesions to the parietal peritoneum, there may be added

confusion with acute pancreatitis. However, the toxemia, the shock, the flush, the paretic abdomen and the characteristic epigastric tenderness are missing.

Perforated peptic ulcer with its antecedent ulcer history, board-like rigidity and scaphoid abdomen, may well be distinguished from the pancreatic paretic abdomen. The absent mid-epigastric tenderness, the early signs of spreading peritonitis, the obliteration of liver dullness, with roentgen ray evidence of air under the diaphragm, the expiratory grunt and the absent malar flush exclude pancreatitis. The ulcer patient usually presents a characteristic facies, the long, thin, drawn, dyspeptic countenance portraying chronic pain.

Coronary and cardiac disease demand consideration. Careful precordial examination is essential, for the manifestations of heart disease are protean, and they may well simulate intra-abdominal catastrophes. A pre-existing cardiac history, with liver tenderness due to right coronary closure, upper abdominal rigidity, and an extremely hypersensitive abdominal wall, will lead to the correct diagnosis.

Acute appendicitis should give no difficulty, even when spreading peritonitis is present.

LABORATORY AIDS

What are the laboratory aids that may be utilized? The total and differential leucocyte counts of the blood are of little value. In fact, it may be stated that the importance of the white cell count in acute abdominal conditions is probably over-emphasized. Infection, hemorrhage, shock, and dehydration influence it. It should be used merely as corroborative evidence. Acute abdominal pain produces leucocytosis.

The erythrocyte count is usually high, over 5 million. Hemoglobin content, too, is elevated because of the dehydration. Increased hemoglobin may be utilized as an index of the severity of shock, and should considerably influence the surgeon as to the advisability of immediate operative intervention. Serial determinations should be made frequently in order to determine the necessary fluid requirements for the restoration and maintenance of normal blood volume.

Blood enzyme determinations should be performed early, and frequently repeated. The serum amylase test is of great value (especially early in the disease). Its determination can be carried out rapidly, and elevation of amylase levels occurs early. The lipase determination has greatest value after the first 48 hours of the illness, and its concentration remains elevated for a longer time than that of the amylase, since the latter enzyme is excreted from the body quite rapidly. Determinations for both these enzymes when acute pancreatitis is suspected should be urgently requested. There is a definite relationship between amylase findings and clinical progress. Normal determinations may re-appear within a few days in a mild attack. Normal values indicate little or no pancreatic destruction. Following cholecystostomy, the blood amylase values in pancreatic disease return to normal rather abruptly, because of relief of pancreatic pressure. Similar subsidence may occur when

pancreatic necrosis is progressive. Here, destruction of parenchyma which is the source of the hyperenzymemia, prevents manufacture and liberation of the pancreatic enzymes. Hyper-cholesterolemia has been frequently reported.

The urine may manifest the intoxication by the presence of albumin and casts. Glycosuria need not be, and usually is not present. Any severe peritonitis may be accompanied by glycosuria. Urinary diastase may rise from the normal of 30 units to 200–1000.

Recently, Edmondson and Berne[18] have drawn attention to the marked mobilization of calcium in and around the pancreas resulting from the formation of calcium soaps which are clinically recognized as the areas of "fat necrosis." This results in an absolute hypocalcemia which is most apparent between the fourth and tenth days of the disease. Fatal prognostic significance has been attached by them to serum calcium levels below 7 mg. per 100 cc. Early serum calcium determination has little value in the diagnosis of acute pancreatitis; or where fat necrosis is very minimal.

A scout film of the abdomen may show free air under the diaphragm or fluid levels in intestinal loops, indicating perforated ulcer or acute intestinal obstruction respectively. Later, if the patient's condition permits, a barium meal may disclose a wide duodenal sweep, an irregular greater curvature of the stomach, pyloric obstruction, or a depressed transverse colon, all signs of an enlarged pancreas.

Gottesman and his associates,[25] and more recently, Bockus and Raffensperger[6] have described significant electrocardiographic changes in acute pancreatitis. Improvement in the patient's clinical status is concomitant with the disappearance of these abnormal tracings. Hypopotassemia occurring in association with vomiting and/or dehydration has been ascribed as the cause for these changes by Bockus, quoting Bellet.

TREATMENT

During the past decade, therapy of acute pancreatitis has tended toward conservatism. Surgery is usually either delayed or withheld entirely. Immediate operation is justified only when the diagnosis is uncertain, when peritonitis is marked, when ileus persists without improvement, or when abscess is present. Subsequent operative intervention may be contemplated following the acute phase, especially when cholecystitis is the precursor (as it has been in practically all our cases).

If we have, with a fair degree of certainty, established the diagnosis of acute pancreatitis clinically, immediate surgical intervention avails us naught, and the added trauma may act to the detriment of the patient. The therapy should be directed to stopping the activation of trypsin within the pancreas, for, regardless of whether we believe that the mortality in acute pancreatitis results from shock, anaphylaxis, or toxemia, the basic mechanism in the evolution of the pathologic and the clinical picture is parenteral tryptic digestion. If perforated ulcer and intestinal obstruction can be definitely excluded, conservatism directed towards halting trypsin digestion is indicated.

MEDICAL REGIME

The principles of treatment which are generally employed include: starvation, which markedly inhibits the secretion of tryptic ferments; continuous gastric suction, to further rest the pancreas; the relief of pain by means of opiates and by procaine injection of the splanchnic nerves; the restoration of normal blood concentration and volume, utilizing whole blood, plasma and glucose (the latter to be given with sufficient insulin "coverage" to prevent pancreatic stimulation); the prevention of distention by Miller-Abbott intubation; the inhibition of nervous stimulation of the pancreas, using atropine and ephedrine; and the recognition and elimination of hypocalcemia, utilizing calcium gluconate parenterally. Recently roentgen ray therapy has been used successfully by Chisholm and Seibel[11] to decrease the serum amylase in acute pancreatitis in experimental animals, and also in a small series of clinical cases.

SURGICAL TREATMENT

Surgically, five procedures have been developed as follows:

1. *Pancreatic drainage,* by splitting the peritoneal covering of the pancreas or by section of the gland itself, to encourage external discharge of the necrotic material. The process in the pancreas is not ameliorated by such manipulation and pancreatic incision may enhance the danger of hemorrhage. A fatal result was reported by Walzel[55] due to trauma to the splenic vein. Besides, the anatomic arrangement of the pancreas into lobules precludes complete drainage unless each lobule be split. Operative handling of the pancreas serves no good purpose, but on the contrary, may destroy natural protective barriers.

2. *Lesser sac drainage* to prevent toxic product accumulation and forestall pseudocyst formation. Smead[51] has demonstrated that the dilution and neutralization of these substances by blood and exudate is so complete that they are not harmful and removal is unnecessary.

3. *Cholecystostomy* to decompress the entire biliary tract. This procedure is less hazardous than common duct drainage with or without gallbladder excision. However, pancreatitis does recur after gallbladder drainage, as does the pre-existent cholecystitis. At times, emergency cholecystostomy must be done.

4. *Choledochostomy,* although more efficacious, can have no rational indication, except in the presence of jaundice due to stone, or dilated ductus choledochus.

5. *Cholecystectomy,* although it can accomplish the removal of the underlying cause, may add too much trauma to an already shocked patient. Too frequently have renewed attacks of acute pancreatitis followed immediately after this procedure, and too often have pancreatic pseudocysts resulted. Cholecystectomy is followed either by dilatation of the common duct with a continent sphincter of Oddi, or the sphincter too may dilate, with resultant incontinence. These events require time, and before such sphincter relaxation ensues, spasm may lead to pancreatic necrosis. Disturbed innervation results

in common duct and sphincter relaxation. Unless this sphincteric atony occurs, increased biliary pressure results, and continues until either the ducts distend or the sphincter dilates. This primary increase in pressure predisposes to acute pancreatitis. Rost[48] reported a fatality in acute pancreatitis following instrumental dilatation of the sphincter at operation.

Halstead,[28] in 1890, successfully operated on acute pancreatitis, merely establishing the diagnosis at celiotomy. He closed the abdomen without drainage of the peritoneal cavity. Halstead, again in 1901, unsuccessfully operated

TABLE IV.—*Operative Findings in 154 Cases*

	Percentage
Fat necrosis	100%
Gall bladder disease	81
Calculi	54
Beef broth fluid	55
Granular omentum	29
Acute cholecystitis	23

upon Opie's case of stone impacted at the ampulla of Vater. Von Haberer,[27] in 1909, advocated routine cholecystostomy to provide a drainage vent, but he presupposed ampullar block in all cases. It thus becomes fairly well established that splitting the capsule and pancreatic drainage do not influence the pancreatic process for recovery and certainly in the severer cases, cholecystectomy and choledochostomy will not be tolerated.

TABLE V.—*Operative Procedures in 154 Cases With Mortality Rates*

	Cases	Deaths	Percentage
Cholecystectomy	30	2	6.6%
Cholecystostomy	83	20	24.0
Choledochostomy	2	0	0.0
Choledochostomy with cholecystectomy	10	0	0.0
Pancreatic drainage	23	14	60.8
Exploratory laparotomy	4	1	25.0
Incision and drainage of lesser sac	1	0	0.0
Multiple operations in the acute phase			
A—Pancreatic drainage followed by B—Cholecystostomy	1	0	0.0
Total	154	37	24.0

Korte,[32] Abell,[1] Eggers,[20] Wolfer,[59] McWhorter[39] and Jones[30] have advocated immediate intervention. Wangensteen,[56] Smead,[51] Lewis,[34] Mikkelsen,[38] De Takats and MacKenzie[52] indicated a preference for early conservatism followed by subsequent biliary tract operation.

Eliason and North,[21] although advocating early operation, conclude that emergency operation may not be best. McWhorter,[39] who believes early operation the procedure of choice, observed the lowest mortality rates in the deferred operative group.

Mortality rates of 50 per cent to 70 per cent are reported by many observers. Mikkelsen[38] by delaying operative intervention from one to three weeks reduced his mortality to 7.5 per cent in 39 cases, 20 of whom were very ill. Demel,[16] from 1926 to 1934, operated upon 95 per cent of his 22 cases immediately, lost 78.3 per cent. In the next 34 cases, but 50 per cent were operated upon immediately, with 26.4 per cent succumbing.

During the past ten years, cholecystectomy has been our operation of choice, if performed one week to ten days after subsidence of acute symptomatology. Ligation of the cystic duct, however, has been purposely omitted. A soft rubber tube is placed over the cystic duct stump, thereby establishing a vent for biliary drainage should sphincteric spasm with possible recurrent acute pancreatitis ensue. In effect, choledochostomy without its additional trauma and shock is accomplished, with decompression of the biliary system and concomitant removal of the focus of infection.

Placing a drainage tube within the cystic duct would accomplish the same result, but with this difference: if subsequent spasm did not occur, convalescence would be unduly delayed by persistent biliary drainage. By placing the tube over the cystic duct, closure of this duct progresses without interference. The danger of biliary peritonitis need give us no concern. We have practiced this mode of treatment for the past ten years with happy results.

Further analysis of the 154 cases will be found in the accompanying tables.

SUMMARY

The pathogenesis of acute pancreatitis has been presented.

Differential diagnosis has been discussed with especial emphasis on acute intestinal obstruction and perforated ulcer.

The immediate operative and conservative forms of therapy have been compared.

Our present mode of delayed operation has been described.

There remains but one further thought, and that is to urge the necessity for removing the principal offending organ before the complication of recurrent acute pancreatitis presents itself.

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