

ACUTE PANCREATITIS
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ACUTE pancreatitis is the most terrible of all the calamities that occur in connection with the abdominal viscera. The suddenness of its onset, the illimitable agony which accompanies it, and the mortality attendant upon it, all render it the most formidable of catastrophies. The disease (in accordance with the classification suggested by Fitz, of Boston, one of the greatest of physicians, who first described it in the year 1889) is generally said to be of three types, hemorrhagic, gangrenous and suppurative. The three types differ only in degree. In the most acute form of all, which is rapidly fatal unless an early operation is performed, hemorrhage is found throughout the whole gland. In a less acute form the patient may survive to discharge sloughs of the pancreas, and of the necrosed fat around the pancreas, for many weeks after operation; in a still more chronic form, an abscess in and around the gland may form so slowly that there is time for it to point in front, or in the costovertebral angle. The essential nature of the disease is the same in all three forms. Of the cause of the disease we are at present ignorant insofar as the great majority of the cases are concerned.

Symptoms.—It is generally stated, even by the most competent authorities, that acute pancreatitis is a disease difficult to recognize; that the diagnosis will often be made only when the abdomen is opened, and a blood-stained effusion escapes, or areas of fat necrosis in the parietes, or in the omentum, are discovered. I cannot agree with such opinions. The clinical picture presented by a case of acute pancreatitis is quite unmistakable. It is because the surgeon omits to think of this disease that its presence escapes him. As Deaver says: "Unless a surgeon has seen previously two or three cases of acute pancreatitis, or unless he keeps the condition constantly in mind, it is seldom that a correct diagnosis is made before opening the abdomen." So many forms of abdominal disaster are seen, and among them so few involve the pancreas, that thought of this organ slips from the memory. I have found in not a few instances, that the moment the diagnosis is suggested, it meets with eager acquiescence. The first and chiefest symptom is pain; and of all the pains that the human body can suffer, this is by far the worst. Even the agony caused by the perforation of a gastric or duodenal ulcer is less than that in acute pancreatitis. The pain, too, is remarkable in that it comes so frequently after a good meal, and for the area of its distribution; it is of fiercest intensity in the epigastrium, but it is felt also in the back and often in both loins. Pain in the back is rare after the perforation of an ulcer, even of an ulcer eroding the pancreas, and perforation of such an ulcer is of far rarer occurrence than acute inflammation of the pancreas. The pain is so intense that it causes profound collapse, in which the pulse rises rapidly and

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loses volume, and the blood-pressure falls. Almost every writer upon the subject of perforation of ulcers of the stomach or duodenum, speaks of "shock." It is true that the patients look profoundly ill, having white and anxious faces; but the pulse in these cases remains almost unchanged in frequency and in volume, and the blood-pressure is little altered. It cannot, therefore, be quite accurate to speak of "shock" as a symptom of perforation, for the conditions inseparable from shock are in this case absent. The collapse of the patient is evident enough, and such a state exists as to render any operation of a far greater severity than normally attaches to it. Of the existence of "shock" in acute pancreatitis there is never the slightest doubt. The patient is prostrate, faint and pallid, and suffers indescribable anguish; the pulse may be hardly perceptible, the limbs and face are cold, and death itself seems imminent. No such state is seen in any other form of casualty. There is all the collapse that even the greatest hemorrhage could cause, and more than the agony of a visceral rupture. Corroboration of a diagnosis made upon these evidences alone is hardly necessary; but if it were it is never lacking. Vomiting is almost invariably present, and it occurs early. There are cases in which it is repeated with great frequency and severity, so that the resemblance to a case of high intestinal obstruction, in respect of this one symptom only, is very close. The matters ejected are, however, never in the least like those seen when the jejunum is obstructed in its upper part; they are of gastric, or of duodenal origin, never foul-smelling, and never copious. Nausea and retching, with hiccough, are more frequent here than in cases where the intestine is blocked. The patient sometimes presents a very curious, and I believe, a quite characteristic appearance, to which Halsted was the first to call attention. The face is livid, and patches of a slate-blue color may be distributed irregularly over the surface of the abdomen, or even of the limbs. This cyanosis is never found in other forms of acute abdominal catastrophe, so far as I know; it is not always present in acute pancreatitis, but if it is found, it is, I believe, an undeniable evidence of acute pancreatic disease. Grey Turner has recorded (*Brit. Journ. Surg.*, 1919, vol. vii, p. 394) two cases in which large patches of discoloration of the skin were attributed to direct action of the pancreatic juice which by infiltration had reached; in the one case the umbilicus, and in the other the costo-vertebral angle. I have seen a faint tinge of jaundice in five cases only.

The respirations are quickened in all cases, and are faint and shallow in proportion to the degree of collapse, which is in turn dependent upon the degree of swelling in, and around, the pancreas. An examination of the abdomen makes the diagnosis still more certain. There is a degree of rigidity in the whole abdomen, and the epigastric region is certainly a little firmer than the rest. But the fixity and hardness are not to be compared with the conditions present when a hollow viscus has burst. Then the rigidity is obdurate and unyielding, and immobility of all the abdominal muscles, including the diaphragm, is complete. The lightest handling is then resented. In acute inflammation of the pancreas, however, the rigidity, and a degree of

tenderness, are confined to the parts above the umbilicus, and even in early hours a degree of fulness may be observed here. The whole abdomen is tender, but the tenderness is more acute above the umbilicus than below, and often is far more exquisite to the left of the middle line than to the right, a point not without significance. If the patient survives a few days, as will happen in the less acute cases, the contrast between the upper protruding parts of the abdomen, and the emptiness, or even retraction of the lower parts may be very striking. This is the condition to which Fitz gave the name "epigastric peritonitis."

The symptoms I have now described occur only in the gravest form of pancreatic necrosis. If they are less acute, they indicate a degree of inflammation in which the necrosis and the hemorrhage are less extensive, in which perhaps, a rupture of the pancreas into the lesser sac (the "perforation of the pancreas" of W. J. Mayo) does not occur. It may be that in some of these cases the invasion of the gland occurs through the duct of Santorini, and that only a part, and that perhaps a small part of the gland is attacked: the fact that some patients give a history of earlier attacks makes this suggestion not unlikely. All the manifestations of the disease are in this case subdued, and the patient may gradually improve for three or four days. Then the temperature begins to rise at night to 102° or 103° , falling in the morning almost to the normal; vomiting becomes distressing and exhausting, wasting is very evident, and the epigastric fulness gradually increases. A swelling may appear behind, between the last rib and the iliac crest, and additional evidences of pancreatic disease may then be elicited. Of these "Löwe's test" is of no little value. Two or three drops of a $1/1000$ solution of adrenalin are allowed to run slowly on to the conjunctiva of one eye; after a few minutes this dose is repeated. Within 15 to 30 minutes the pupil of this eye becomes dilated in cases of pancreatic disease, and remains unchanged in cases where disease is absent. Urinary changes may also be noticed. Glycosuria is not infrequent, but its absence means nothing. Diastase, normally present in small quantities only, is increased tenfold, or even more. Cammidge's reaction, in the hands of its inventor, has proved useful, but other observers have not found cause to rely upon it.

The differential diagnosis in cases of acute pancreatitis should present little difficulty. The more acute the case the easier is the recognition. No other catastrophe within the abdomen produces at once such unendurable agony, and so profound a collapse. Intraperitoneal hemorrhage, resulting from a ruptured tubal gestation, causes intense prostration, blanching, loss of volume in the pulse with increase in its rate, but relatively, the pain is almost negligible, and the site of it is different. The abdomen in these cases is fuller below than above. The history of a missed period and perhaps of a little vaginal hemorrhage, at the time of the onset of pain, are incidents suggestive of a catastrophe of this kind.

The perforation of a gastric and of a duodenal ulcer occurs, in the great majority of instances, in patients who have suffered from dyspepsia for

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years, and have undergone one or more periods of medical treatment for its relief. The pain is very severe, but it does not reach the fierce intensity of that experienced in acute pancreatitis; and "shock" as measured by pulse-rate and volume, and fall of blood-pressure, does not occur. For some years I have given special attention to the appearance and condition of a patient in the early hours after perforation of an ulcer has occurred. The pulse, it is always a surprise to find, is very little changed from the normal. In pancreatitis the pulse is so poor, and so rapid, that it can hardly be counted. The abdominal rigidity in perforation is far more inflexible and widespread, and vomiting does not occur with such frequency. There is no lividity of the face, but rather a pallor with sweating of the brow and temples.

A patient who suffers from hepatic colic is restless, moaning and twisting, changing his position every moment, searching for relief which does not come. He folds his arms across the abdomen and doubles himself over them; he presses the abdomen against the back of a chair, or on the edge of the bed: rests for a few moments and walks about, groaning in agony. A patient who has suffered the perforation of an ulcer is motionless; and in acute pancreatitis is almost so. The history of former attacks of the like kind, of flatulence causing great distress, and possibly of jaundice following upon the pain, may help to make the diagnosis clear.

Acute intestinal obstruction may cause the closest mimicry of the symptoms of pancreatitis. Fitz, in his original paper, spoke of epigastric peritonitis with the symptoms of high intestinal obstruction, as being significant of this disease. The onset of the symptoms, when the intestine is caught and closed, may be acute, but there is nothing of the sudden and overwhelming seizure that is seen in acute pancreatitis; nor is there collapse. Vomiting is projectile, and the ejecta are progressively more and more offensive. General abdominal distention with perhaps a local inflation of an obstructed coil, recognized after a long scrutiny and the gentlest palpation, will be distinguished without difficulty from the appearances of distention above and flatness below, which are so frequent when the pancreas is involved. In acute pancreatitis a local distention of the transverse colon may sometimes be noticed.

I do not know of any case in which the victim of an attack of acute pancreatitis has been regarded as suffering from poisoning. Leriche and Arnaud (*Rev. de Gynéc. et de Chir. Abd.*, 1909, vols. iv and v) have suggested that in the fulminating cases of pancreatic apoplexy, when the onset is sudden, the collapse profound, the vomiting severe, and a fatal ending not long delayed, the likeness to the symptoms caused by an acute irritant poisoning may well excite uneasy suspicions in the mind of the medical attendant.

When all the cases that I have seen are reviewed, the truth remains, that it is chiefly because the suspicion of this comparatively infrequent disease does not enter the mind that an accurate diagnosis is not more often made. The symptoms and the signs are clear enough.

The cause of acute pancreatitis cannot always be determined. The close association of the common bile duct with the head of the pancreas, the rela-

tionship of this duct with the canal of Wirsung at their termination in the duodenum; the presence of the sphincter of Oddi at the ampulla of Vater, have all helped to foster a belief in the dependence of pancreatic inflammation upon causes arising in the biliary passages. And further, the frequent association of cholelithiasis with pancreatitis, both acute and chronic, has done much to strengthen this belief.

The common duct is completely embedded in the head of the pancreas in approximately three cases in four; in the remaining case, there is a deep groove in the posterior surface of the head of the gland, to receive the duct on its way to the duodenum. A degree of inflammation sufficient to attack all the walls of the duct will, therefore, inevitably spread to the head of the pancreas; the more acute the inflammation and the longer it continues, the greater will be the change produced in the gland. The termination of the common duct in the duodenum is in relation with the canal of Wirsung. Four different modes of association are found. (1) The two ducts end in a cavity which discharges into the duodenum by a small orifice. (2) The common duct is joined about one-third inch above the duodenum by the duct of the pancreas. (3) The two ducts open separately but close together on the surface of a depression in the duodenum. (4) The two ducts open similarly but on the surface of a papilla in the duodenum. In the first two forms any block below the point where the two ducts come together will convert them into a single channel. In the last two any blockage of one duct has no effect upon the other. A further intimacy between the biliary system and the pancreas exists through the agency of the lymphatic system. Attention was first called to this by Maugeret (*Thèse de Paris*, 1898). It is suggested that in cases of gall-bladder infection, a lymphangitis spreads downwards to the common duct, and thence to the pancreas, causing on its way an enlargement of the cystic gland, and of the glands which lie along the duct. The recent work of Braithwaite (*Brit. Journ. of Surgery*, 1923, vol. xi, p. 7) has shown, that when one lymph channel is blocked, a retrograde current is set up, and the flow occurs through other channels. A pancreatic lymphangitis may be set up by infective material coming from the gall-bladder and turned aside from its main direction of flow by a block in the enlarged glands.

In acute pancreatitis, the immediate cause of the gland necrosis is the activation of the pancreatic juice by some agent or other, within the substance of the gland. In normal conditions the trypsinogen of this secretion is converted into trypsin by an enterokinase in the duodenum. A reflux of the activated juice into the gland is physically impossible. An increase of the intraduodenal pressure to 1000 mm. of water does not overcome the resistance of the ducts. It is impossible by experiment to force back the duodenal contents into the ducts (Archibald, *Surg., Gyn. and Obst.*, 1919, vol. i, p. 531). The problem, therefore, is to discover what agency is competent to cause the activation of the pancreatic juice before it leaves the gland.

The first demonstration of the influence of the bile, in this respect, was

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given by Claude Bernard in 1856. He produced acute necrosis of the pancreas by the injection of bile mixed with sweet oil, into the duct of the pancreas. For many years after this an association between cholelithiasis and acute pancreatitis had occasionally been noticed, but attention was especially directed to this subject in 1901 by Opie, in his work on "Diseases of the Pancreas." On examining the body of a patient of Halsted's, who had died from acute pancreatitis, Opie found that the common duct and the canal of Wirsung ended in a common cavity, a diverticulum, the outlet of which into the duodenum was small: this outlet was completely blocked by a calculus which did not fill the diverticulum. In consequence the ducts became converted into a single channel, and a retrojection of bile into the canal of Wirsung, stained a deep green color, had occurred. This led Opie, Flexner, and others to study the effect of the experimental injection of bile, bile salts, and chemical irritants of several kinds, into the pancreatic duct. Pancreatitis, strictly comparable to the condition met with in man, was found. In all these experiments the injections were made by a syringe, with a greater pressure than could be produced by any agency during life. Nordmann's experiments (*Archiv f. klin. Chir.*, 1913, vol. cii, p. 66) appeared to show that two factors were necessary to the production of acute hemorrhagic pancreatitis in the dog, (a) stasis of pancreatic juice within the ducts of the gland, (b) the access of organisms with, or without, bile to the duct. Polya's experiments (*Mitt. a. d. Grenzgeb.*, 1912, vol. xxiv, p. 1) demonstrated that typical pancreatic necrosis with hemorrhage, and fat necrosis, causing death rapidly, was most readily produced by the intraductal injection of a strongly active trypsin solution, less constantly by the injection of duodenal contents, or by bile mixed with bacteria, and rarely by the injection of bacteria alone or of the discharges from intestinal fistulæ.

Mann, of the Mayo Clinic, has conducted a series of experiments upon goats (*Mayo Clinic Papers*, 1921, vol. xiii, p. 185). In these animals, the pancreatic duct enters directly into the common duct a few centimetres above the duodenum. A ligature applied to the duct below the entrance of the canal of Wirsung, converts the two ducts into one channel. After such an operation the animals lived sometimes three or four weeks. The pancreas was then deeply stained with bile, and all the ducts of the gland were filled with bile. Acute pancreatitis did not develop. If, however, the common bile duct was ligatured above the entrance of the pancreatic duct, and through a cannula placed in the lower part of the duct, bile drawn from the goat's gall-bladder was injected into the pancreatic duct, acute hemorrhagic pancreatitis developed and caused death. The pressure of bile in the common duct, he found, was due to three causes: the secretory pressure of the bile, the contractile pressure of the gall-bladder, and the pressure of the abdominal muscles. The secretory pressure of bile was assessed at 350 mm. of bile; the contractile pressure of the gall-bladder at 300 mm. as a maximum, though usually it seemed "of little consequence." The mechanical effect of the abdominal muscles and of the diaphragm produced the most marked changes upon the pressures within

the duct. Deep respiratory movements, struggling, retching and especially vomiting, caused the pressure to reach as much as 1000 mm. of bile. If bile was injected at this pressure, directly into the pancreatic duct, acute pancreatitis did not develop, except in one case where there was fat necrosis of the gland. The difference in the results of injection of bile with a syringe and at the lower pressures seemed to be due to trauma, to the rupture of the small pancreatic ducts and to infiltration of the gland substance by bile when the syringe with its higher pressure is used.

Mann examined the condition of the common bile duct, and the pancreatic duct, in 200 consecutive bodies in the post-mortem room, and found that in 40 only was there the condition of the ampulla described in Opie's case. He gave the following table:

Group	Location of opening of pancreatic duct	Number of specimens	Percentage
1.	Separately into duodenum	62	31
2.	Two mm. from the apex of ampulla of Vater	90	45
3.	Three mm. to 10 mm. from the apex of the ampulla of Vater	40	20
4.	Duct absent or reduced to fibrous cord	8	4
	Total	200	

Archibald has suggested (*Surg., Gyn. and Obstet.*, 1919, vol. xxviii, p. 529) that the mechanism by which the two ducts are converted into one may be the sphincter of Oddi. This muscle, described by Oddi (*Arch. p. L. Sc. Torino*, 1888, vol. xii, p. 333) had escaped the notice of all surgeons until Archibald called their attention to it. The following brief account of Oddi's work is given by Archibald:

"Oddi studied this muscle both physiologically and anatomically. In brief, he found that the sphincter in dogs was able to resist a pressure of 50 millimetres mercury, which equals about 675 millimetres of water. He demonstrated in microscopical sections, that the sphincter was composed of a special bundle of circular fibres. He found that the common duct, outside its course through the bowel wall, possesses no muscular fibres. From the physiological side, he discovered that this sphincter could be put into spasm by a mechanical irritation of the duodenal mucosa, or by the application of dilute hydrochloric acid in either the duodenum or the stomach; and that even mere cutting of the bowel to expose the papilla would cause spasms lasting from twenty to thirty seconds. Stimulation of the vagus apparently provoked a very prompt and intense contraction of the sphincter. A like result was obtained by stimulating the central end of the cut sciatic, while stimulation of the splanchnics had no effect. He also observed dilatation of all the extrahepatic ducts in dogs deprived of their gall-bladder. He thought a catarrhal condition in the duodenum was a stimulus to the sphincter, and that this might explain some cases of icterus where other causes could not be found."

After a series of experiments, Archibald concluded that the lesions indicating the existence of an acute pancreatitis were brought about entirely through the action of the sphincter of Oddi, combined usually, but not always, with some increase in pressure in the biliary system behind the sphincter. The effect of the bile, or of its salts, is to cause a cytolytic effect, a direct disintegrating effect, upon the cells of the pancreas (Bradley and Taylor,

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Journ. Biol. Chem., 1917, vol. xxix, p. 281). This work of Archibald encouraged Mann to undertake a further series of investigations upon the anatomy of the sphincter in man. It is evident that if the sphincter is to be able to convert the two ducts into one channel, it must lie distal to the entrance of both the ducts into the ampulla: otherwise it would merely compress the lower ends of the two ducts. In most instances Mann found that the position of the muscular fibres was proximal to the termination of the common bile duct, and that some fibres also passed round the lowest part of the canal of Wirsung. By their contraction, the two ducts would be narrowed. In rare instances, that disposition of the sphincter was found which would close only the outlet of the ampulla and so lead to a free communication between the two ducts.

The conversion of the two ducts into one, by the closure of the outlet from the ampulla into the duodenum, by the action either of the sphincter, or of a stone, seems to be possible only in 4.5 per cent. of the total number of bodies examined (E. S. Judd, Journ. Amer. Med. Assoc., 1921, vol. lxxvii, p. 197). The conclusion has therefore been drawn, that the occurrence of acute pancreatitis as a result of the injection of bile into the duct of the pancreas, must be "exceedingly small." But may not the conclusion be drawn with greater likelihood, that it is only those patients in whom the anatomical or physiological conditions are favorable, who suffer from acute pancreatitis? Those conditions are present, it is true in only 4.5 per cent. (Judd) of the total number of bodies examined: but is it not possible, indeed probable, that they are present in a large proportion of those patients who suffer from acute pancreatitis? The incidence of this disease is very rare in comparison with many other forms of abdominal catastrophe; its rarity may well be due to the fact that the essential conditions for its development being absent, most people are safe from its attack. The examination into the exact anatomical conditions present in all the fatal cases, can alone answer these questions. The two conditions, which may be considered as almost essential in the development of acute hemorrhagic pancreatitis, appear to me to be (1) an anatomical arrangement of the parts at the termination of the two ducts, which permits of their conversion into one channel by closure of the orifice of the ampulla into the duodenum, and (2) the presence of infected bile, associated or not with cholelithiasis, in the gall-bladder or in the bile ducts.

The possibility of the duct of Santorini being the only duct or the larger of the two ducts of the pancreas, the duct of Wirsung being absent or very small, must be remembered. This condition is found in 10 per cent. of bodies examined. The duct of Santorini opens directly into the duodenum without any protective mechanism at its termination. The entrance of duodenal contents into the duct may accordingly take place under conditions which would be powerless to effect an entrance to the canal of Wirsung. A case of acute pancreatitis where the necrosis of the gland was limited to the area of the duct of Santorini was recorded by Opie in the first edition of his work. Other

cases have since been observed; the area of the necrosis of the gland being large or small, in proportion to the extent of the gland which is drained by this duct. Brocq (Compt. Rend. Soc. de Biol., 1919, vol. lxxxii, p. 371) has shown the potency of duodenal contents injected into the ducts in setting up an acute inflammatory condition, with hemorrhage, and he found also that normal bile will produce necrosis of the gland if it is injected during the progress of digestion.

An acute lymphangitis can play little part in the onset of the most acute forms of pancreatitis. The disease is too sudden in onset, its ravages too widespread and too intense for that. The essential quality of the disease is auto-digestion of the pancreas, and that can rapidly occur only through an invasion of its duct.

Treatment.—There can be no doubt that recovery from acute pancreatitis, of all grades, except the most severe, is possible without operation. In a number of cases in which I have operated for stones in the gall-bladder, or in the common duct, very extensive areas of fat necrosis have been found, and the pancreas has been large, infiltrated with blood, and œdema around it has been considerable. In one patient, a medical man, accustomed to the practice of surgery, the diagnosis of acute pancreatitis had been made by himself. He was able to detect an “immense difference” between the ordinary attacks of hepatic colic to which he had long been subject, and the last attack which was “beyond everything,” and in which, his partner said, he had nearly died. It is, however, equally certain that recovery from this disease, apart from operation, is so rare that no case should be left untreated. Not all the operations that have been practised have helped in the recovery of the patient: for a few instances are related in which the abdomen was opened, the diagnosis made, and the wound closed without anything more being done. Recovery in such circumstances would probably have occurred if the patient had been left alone.

The procedure I adopt and advocate consists—(a) in the opening of the abdomen by a paramedian incision above the umbilicus; (b) the gaining of access to the pancreas sometimes above the stomach, through the gastro-hepatic omentum, sometimes below the greater curvature through the gastro-colic omentum, sometimes, though rarely, through the transverse mesocolon, after the omentum has been turned upwards; (c) the isolation of the pancreas by gauze packing covered by mackintoshes; (d) the evacuation of any fluid around the pancreas, by aspiration if the quantity is large; (e) incision of the capsule of the pancreas to allow the escape of blood, of fluid, or of sloughs already detached from the pancreas, or resulting from necrosis of large areas of fat; (f) adequate drainage through the anterior abdominal wall, a drainage tube being surrounded by gauze so as to create a barrier of lymph around the area as speedily as possible; (g) posterior drainage when necessary; (h) cholecystotomy (rarely cholecystectomy) if stones are present, or the gall-bladder appears diseased.

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The diagnosis is almost invariably made before the operation commences. In the fat of the abdominal wall, areas of fat necrosis may be seen before the peritoneum is opened. As soon as the abdominal cavity is reached blood-stained fluid escapes. Whatever doubts may have been previously felt as to the nature of the disease, they are at once resolved when these two conditions, fat necrosis, and sanguineous exudate, are found. In a very large proportion of the cases a great, but local, dilatation of the transverse colon, below the pancreas is found. The colon here is not only much larger than either the ascending or the descending colon, but it is congested in appearance, sometimes very deeply congested, or even inflamed. The condition of the pancreas and of the parts around varies very much. In the most severe cases the pancreas is a phlegmon filled with blood, deep purple in color, looking ready to burst. In the less severe cases, hemorrhage into the gland is only slight and patchy, and a little turbid blood-stained fluid is found either in the lesser sac or behind its posterior layer.

The isolation of the pancreas at this stage is very important. The fluid about the pancreas is extremely toxic, and its escape into the general cavity of the peritoneum, followed by its absorption, might gravely affect the result of the operation. A dread of this result has influenced many surgeons in their refusal to incise the posterior layer of the lesser sac, and the capsule of the pancreas. It is a mistake not to give vent to this exudate; it can be evacuated without any risk, if adequate care is taken, and after all absorption is more likely to take place from the pancreas itself, or from the cellular tissue around it, if this fluid is allowed to remain under great tension. Incision of the pancreas itself need never be made. If the gland feels very œdematous and soggy, the finger may be very gently insinuated into it here and there, so as to make points of escape for retained secretions and blood. Drainage must be free. In a week or ten days after operation a copious discharge of a dirty looking, turbid, blood-stained fluid with sloughs, large and small, may occur. Displacement of the rectus to the outer side will do something to prevent the development of a hernia subsequently. If a hernia develops it can be repaired; if drainage is not adequate the patient will die.

The question as to what shall be done in respect of the gall-bladder will depend upon two considerations, the general condition of the patient, and the state of the gall-bladder itself. Whatever the condition of the patient may be, if stones are present in the gall-bladder or common duct, a tube should be placed in the gall-bladder, as many stones as possible being removed from them both. The complete emptying of the gall-bladder, or its removal may be left to another day, but a drainage tube in the gall-bladder will allow the escape of bile and prevent any great pressure within the ducts. If the patient's condition permits it, complete evacuation of the stones should be possible; or, in the most favorable circumstances, cholecystectomy may be considered safe. The margin of safety must not be overstepped. The surgeon is operating to save a life in jeopardy; not to cure his patient of cholelithiasis. If the patient is very ill, and very stout, having a gall-bladder

shrunk and fibrotic, buried in adhesions and inaccessible, it is safer to leave matters alone. Archibald, of Montreal, whose work on pancreatitis is characterized by great industry and insight and by much ingenuity in the suggestion of experiments, has advised that the duodenum should be incised, the ampulla of Vater slit open, and the sphincter of Oddi divided, in order to prevent the retention of bile, and its confirmed passage into the duct of Wirsung. I have not used this method in any of my cases, never having found a stone in the ampulla in this disease. The objection to its routine employment is, of course, that it is applying to all patients a method, difficult and time-consuming, which may only be of value in a small proportion of them.