Acute Haemorrhagic Pancreatitis

A stout, middle aged man

is suddenly seized with excruciating

pain in the upper part of the

abdomen, to the right of the middle

dline. He vomits frequently a quantity

of dark "bilious" fluid, and almost

immediately falls into a state of the

most profound collapse. He has a

history of previous slighter attacks

of pain in the same position, each

attack being followed by slight

jaundice. He has been constipated

for several days. His face is

eyanosed, but does not present the

Hippocratic facets. His pulse is

between 90 and 100 per minute and

almost imperceptible at the wrist. His

temperature is 99.5°. His urine is

concentrated but has no abnormal

constituents. The liver

delusiveness is not observed, but there is

slight and increasing delusiveness in
both flanks, his abdomen is much distended above the umbilicus and tender over the region of the gall bladder. The abdomen moves freely with respiration.

The next day he is slightly delirious, and his bowels move naturally. He passes flatus freely. His pulse rate rises to 140 to 160 per minute, and his temperature to about 102°. He gradually sinks and dies within forty-eight hours from the onset of the illness.

Such is the clinical picture presented by a typical case of Acute Haemorrhagic Pancreatitis.

Still within quite recent years our knowledge of this rare disease was of the slightest, and it was looked on as a condition only to be diagnosed by an exploratory operation or in the first mortem room. Indeed the number of cases that have been diagnosed before death, can even now be
counted on the fingers of one hand, having, however, collected twenty one cases from the recent literature, and having made a careful analysis of the signs and symptoms presented by these cases, I hope to show that an ante mortem diagnosis is quite possible.

The recent researches of Mayo Robson and Magnison in pancreatic disease, have brought this subject prominently before the medical profession in this country, and consequently the reports of two cases of Acute Haemorrhagic Pancreatitis, which I have recently met with may be of considerable interest. While it is true that in neither of these cases are there any features of very special import which have not been before recorded, yet the number of published cases of this disease is small, and even two cases may add a little to our knowledge of the etiology, pathology and diagnosis of the condition.
It was first pointed out by Spiess in 1866 and by Zinker in 1874 (Oster, Principles and Practice of Medicine, p. 589) that in several cases of sudden death, the pancreas was found to be distended with blood, but our knowledge of Acute Haemorrhagic Pancreatitis was in a very nebulous condition until within the last decade. Yet his classical monograph on the subject, published in 1889 (New York Medical Record, Feb 23rd 1889) was the first systematic description of pancreatitis and since then the literature of the subject has been slowly increasing, owing chiefly to the efforts of Prince, Köte, Ofie and Robson. Nearly all of the reported cases of Acute Haemorrhagic Pancreatitis, have not been diagnosed during life, on account of the general resemblance of the symptoms to those of other abdominal disease, but several points of distinction
have recently been raised, which, in most cases at any rate, should lead one to a correct and early diagnosis. The importance of such an early diagnosis can hardly be overestimated, as the surgery of the pancreas has made such rapid advances, that not only is chronic inflammation of the gland amenable to operative treatment, but several cases of the acute hemorrhagic condition have now been treated surgically, with at least hopeful results.

The pancreas is a racemose tubular gland, closely resembling the parotid in structure. The connective tissue is looser, the acini are more tubular, and the whole gland is more vascular than the salivary glands, and among the inter-alveolar connective tissue are scattered groups of epithelium-like cells - the islets of Langerhans. The secreting cells are filled in their inner two thirds with granules
while the outer third is clear.
After secretion has been going
on for some time, the clear
part becomes wider owing to
the discharge of some of the
small granules into the lumen
of the tubule.

Extrusion of the pancreas
in animals leads to glycosuria,
and one would expect, in an
acute destructive process like
Hemorhagic Pancreatitis, to find
a diabetic condition supernumer.
Such a condition, however, is only
very rarely found.

Each lobule of the gland
has a special duct, which joins
the main pancreatic duct—the
canal of Wirsung. This duct
finally ends by opening into the
duodenum, nearly always in
conjunction with the common bile
duct. It is of the utmost
importance to remember
this fact in considering the
pathology of the inflammatory
condition, as it largely explains the connection which undoubtedly exists between Acute Haemorrhage Pancreatitis and cholelithiasis. It also explains the ease with which a micro-organismal invasion of the gland could occur.

The very close connection between the pancreas and the solar plexus and semilunar ganglia should also be noted, as it has been pointed out by Zemke (Osser, page 590) that to this connection is due the profound shock and collapse which accompanies the onset of the disease. In the cases I record, the intense shock from which the patient suffered was one of the most prominent features of the condition.

Pancreatitis has been divided clinically by Mayo Robson into three divisions—acute, subacute and chronic (Lancet, July 28th, 1900). According to Robson, chronic pancreatitis is a relatively common disease, but
is very frequently mistaken for gall stones, owing to the jaundice which so frequently accompanies it. He reports seven cases treated by operation, of which six were cured and one relieved.

Sub-acute pancreatitis presents nearly all the appearances of acute pancreatitis, but none of the symptoms are so marked, and shock is slight or altogether absent. It frequently passes on to a condition of chronic intestinal pancreatitis.

Acute Pancreatitis: is the most rare form of the disease, and may pathologically be divided into three classes — haemorrhagic, suppurative, and gangrenous (T. J. New York Medical Record, Feb. 23, 1889).

Between these three classes there is no clear distinction, as in most cases there is a combination of two of them, in some of all three.

One of my cases combined the haemorrhagic with the supplicative, the other, the haemorrhagic with the gangrenous...
Of Acute Haemorrhagic Pancreatitis, Köte has collected 41 cases, of which only 4 were in women. From the literature of the last ten years I have collected 21 more, of these only two were in women. The notes I have on my two cases are as follows:

Case I,

A stout man, age 62, was admitted to the General Hospital, Northampton, on March 11th. The history showed that on March 9th he was suddenly seized with intense pain in the upper part of the abdomen. He was very ill and faint, and next morning was markedly jaundiced. Once, three months before admission, he had had a slighter attack of similar pain, followed by jaundice. On both occasions he vomited freely after the attack.

On admission, the patient was in an extremely collapsed condition. The pulse was almost imperceptible, running 135 per minute; the temperature...
was subnormal, and the respiration
31. He frequently vomited considerable
quantities of dark coloured "bilious" fluid
His bowels had not been moved for
three days, and a simple enema
gave very little result. The
abdomen was much distended, especially
so above the umbilicus, and very
tender. There was no area of
dullness over the distended part, and
the liver dullness was not diminished,
the lower edge being at the costal
margin. Puffy fomentations
were applied to the abdomen and
gave great relief. Stimulant
stimulent enemata were administered
four hourly. Under this treatment
the pulse improved somewhat, and on
March 12th, as no urine had been
passed since admission, 4 oz. were
drawn off by catheter. There was
no sugar in the urine. The
vomiting continued and the
temperature remained subnormal
thill evening, when it rose to 100.2°.
During the night his pulse became
weaker, he became delirious, and
died on the morning of March 13th, 1904.

Post mortem.

On opening the
abdomen, the stomach and intestines
were found to be greatly distended,
but there was no obstruction anywhere.
The heart weighed 8 7/8 lbs and was healthy.
The lungs were deeply congested at
their bases, but were otherwise normal.
The liver weighed 3 1/2 lbs and
showed considerable fatty degeneration.
The bile capillaries were somewhat
distended.
The gall bladder contained a large
oval stone, 1 inch in length. The
bile duct appeared to be healthy.
The kidneys beyond showing one
or two cysts on the surface, were
healthy. The kidney fat was
spotted with small, dead white areas
of "fat necrosis" varying in size from
a pin's head to a pea.
The stomach was filled with dark
bile stained fluid.

On lifting up the great omentum,
which showed numerous spots of "fat necrosis." the pancreas bulged forwards, forming a large prominent swelling. It was surrounded by fat showing numerous patches of necrosis. On clearing away this fat, the surface of the pancreas was seen in some places to be of a deep, claret red, in others of a pale opaque colour, and again haits were blackish, almost gangrenous in appearance. The whole organ weighed 1 lb 5 oz.

In making sections of the pancreas, the duct was found to be widely dilated, and contained a considerable quantity of turbid fluid. The lobules were clearly defined by dilated blood vessels in the interlobular connective tissues. Scattered throughout, there were many gangrenous patches, and also a good many patches of dead white colour. Some of these white patches were circular, others were irregular, as if several areas had
coalesced. The greatest incidence of the disease had evidently fallen on the body and tail, although there was a good deal of change in the head also. Scattered throughout the peritoneal fat, more especially round the root of the mesentery, there were numerous minute patches of "fat necrosis" similar to those described in the perinephritic fat.

The microscopical appearance of the pancreas was practically identical with that described in Case II except that the necrotic process was further advanced, and in many of the lobules, no appearance of lobules or secering cells could be found. No cultures were made at the time, either from the pancreas itself or from the peritoneal fluid.

Case II

A woman, age 35, married, was admitted to the General Hospital, Northampton on Jan 12th 1903.
in a condition of extreme collapse. The history obtainable was as follows:

For nine years she had been subject to recurrent attacks of pain in the right side of the epigastrium. This had been diagnosed as biliary colic, and under treatment improved. At the beginning of November these attacks had become more frequent and severe, recurring every few days. The intense pain would last for an hour or two and then disappear. There was no jaundice. This condition persisted up till a week before admission, when after a severe attack of pain, she was deeply jaundiced. On January 11th she was seized with the worst pain she had yet experienced, and was ordered into the hospital by her doctor.

On admission her face was cyanosed and shrunken. The radial pulse was imperceptible, the temperature
subnormal, and the respirations 31 per minute. She had no pain but was slightly jaundiced. The abdomen was distended, tender on pressure over the epigastrium, but there was no marked rigidity of the abdominal muscles, and the abdomen moved freely with the respirations. The lower border of the liver was about 3/4 inch higher than usual. She was frequently sick, the vomited matter being dark and “bilious”. Her bowels had not been moved for three days, and a simple enema gave no result. A provisional diagnosis of Acute Pancreatitis was made, but she was considered too weak to stand an operation. Under stimulant treatment, however, her pulse improved somewhat, and accordingly on January 13th the abdomen was opened to the right of the middle line, through the rectus muscle. There were no adhesions and as soon as the peritoneum was opened, a large
quantity of bile stained fluid escaped from the wound. This was at first thought to be bile, but the gall bladder was full and on pressure was found to empty itself into the duodenum. It contained numerous gall stones. The omentum and mesentery were found to be thickly studded with small white shot, varying in size from a millimeter in diameter to smaller than a pin point. This, of course, pointed to the diagnosis of Acute Pancreatitis being correct, but the condition of the patient was so bad that a permanent measure was rapidly made and a drainage tube passed up to the pancreas. The abdominal wound was also drained.

When she was got back to bed the pulse was imperceptible, but by evening had improved. was 122, temperature normal and respirations 36. The vomiting continued and no flatus was passed.

On the following day, she
seemed rather better, and her bowels moved naturally in the evening. The vomiting persisted and there was a large amount of bile-stained discharge from the drainage tubes. She retained nutrient enemata well. On the 15th she seemed worse, and she still vomited dark blackish fluid, but the bowels moved twice with an apparently normal motion. Towards evening her temperature fell to 96.4°, the pulse was 160 and very weak, and the respiration 45. She gradually sank and died in the early morning of January 16th. Throughout the course of the illness there was no sugar or other abnormal constituent in the urine.

**Post mortem**

Only a limited post mortem examination was allowed, and this was done through the abdominal wound on January 17th.

The whole omentum was shredded with small white areas of
“fat necrosis.” The gall bladder was half full and on pressure emptied itself normally into the duodenum. In the gall bladder itself there were half a dozen small stones. There was also a small stone in the right hepatic duct. The common bile duct was greatly distended, and in the diverticulum of Vater there was a large collection of gall-stones which distended it to about the size of one’s thumb. The liver was small, but beyond a small amount of fatty degeneration was apparently normal. At the back of the upper part of the abdomen there was a large mass, which proved to be a greatly enlarged pancreas. With the exception of a small part of the head, the whole organ was friable and haemorrhagic. On section it showed numerous pale necrotic areas.

Cultures made from the biliary fluid gave negative results.
but those made from the pancreas itself gave typical cultures of the 
Bacillus coli communis.

On microscopic examination of the pancreas most of the lobules
were found to show complete disintegration of the outlines of the cells could hardly
be distinguished, and the nuclei did not take the stain. Some of
the lobules appeared moderately
healthy, but contained blood capillaries
and were infiltrated by a good many
leucocytes. The interlobular
connective tissue was distended
with blood capillaries — indeed in
many sections very little but blood
capillaries could be seen. In a
few places there were tufts of
crystals — evidently fatty.

These two cases may
be taken as fairly typical of
the course usually taken by
Acute Haemorrhagic Pancreatitis:

Etiology

The etiology of Acute
Haemorrhagic Pancreatitis is at present rather obscure.

The sex of these attached is important. Of the 21 cases I have collected, only 2 were in women, this being practically identical with the proportion mentioned by Körner 4 out 41 (Oder Prüfungen and Practice of medicine p. 589).

A large proportion of the cases are in adult males. One, reported by McPhedran, and verified post mortem was in a male infant 9 months old (Canadian Practitioner, September 1896). The age incidence of the cases I have analyzed, is shown in the following table.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Number of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Under 1 year</td>
<td>1 case</td>
</tr>
<tr>
<td>Between 1 and 20</td>
<td>0 cases</td>
</tr>
<tr>
<td>20 - 30</td>
<td>3 cases</td>
</tr>
<tr>
<td>30 - 40</td>
<td>5 cases</td>
</tr>
<tr>
<td>40 - 50</td>
<td>3 cases</td>
</tr>
<tr>
<td>50 - 60</td>
<td>3 cases</td>
</tr>
<tr>
<td>60 - 70</td>
<td>2 cases</td>
</tr>
<tr>
<td>70 - 80</td>
<td>2 cases</td>
</tr>
</tbody>
</table>
4 females

Between 30 and 40  1 case

... 60 ... 10  1 case

Thus 30 per cent were between
the ages of 30 and 40.

The majority of the cases
occurred in fat people

Acute Haemorrhagic Pancreatitis
has been stated to follow injury, to
occur in those addicted to alcohol, and
in those who have previously
suffered from attacks of abdominal
pain.

In none of these
21 cases has there been any
suggestion of a preceding trauma;
in these only has it been possible to
obtain a definite history of alcoholism;
while in 10 there was a history of
attacks of pain in the abdomen,
occurring at varying intervals before
the onset of the acute pancreatic
affection. This last fact is of
great importance as in seven of
these cases, gall-stones were found
at the post-mortem examination.
In eight cases no previous history was reported, and in these and three more, only a partial necropsy was made, and the condition of the bile passages was not investigated. Out of 21 cases, then, 10 only were submitted to a complete and thorough examination after death, and in seven of these a condition of cholelithiasis was found. In addition, none case reported by Nash (Lancet, Vol. ii. 1902, p. 1192) and operated on by him, a large gall stone was removed, and the patient recovered. In 80 per cent., then, of the cases examined, gall-stones were found to be associated with Acute Haemorrhagic Pancreatitis.

These facts point very strongly to there being more than a casual relationship between cholelithiasis and Acute Haemorrhagic Pancreatitis. In the majority of cases the duct of Wirsung joins the common bile duct about five millimeters before
the combined duct enters the duodenum. As there is a slight projection at the point of entry into the bowel— at the duverterculum of Vater—this is the place where a gall-stone passing down the common duct would most probably become impacted. The outlet into the duodenum being thus closed, the bile duct and the pancreatic duct become for all practical purposes a continuous tube. In this condition of affairs the bile would naturally regurgitate into the pancreatic duct. That this does occur is shown by a case reported by Halsted (John Hopkins Hospital Bulletin 1901, no. 121, 122 and 123) This was a typical case of Acute Haemorrhagic Pancreatitis, in which, at the post mortem examination, a small biliary calculus was found impacted in the duct at its outlet into the intestine. Both the bile duct and the pancreatic duct
were found dilated, and the lumen of the latter was stained with bile. As a result of this case, O. he conducted some experiments in the laboratories of the John Hopkins Hospital, the results of which he reported in the American Journal of the Medical Sciences (January 1901, p. 24).

He took seven dogs; in five of these he opened the abdomen and injected bile into the common duct; in the two remaining he opened the pancreatic duct itself, injected bile, and ligatured the duct. In all seven cases haemorrhage pancreatits with fat necrosis developed. The condition was verified in all cases post mortem.

Hailed in his papers in the John Hopkins Hospital Bulletin, 1901 no. 121, 122, 123, laid down the following conditions as the most likely to favour a retrojection of bile into the pancreas.

1. The stone must be too small to
exclude the pancreatic duct.

2. The stone must be too large to pass the constriction at the outlet of the ampulla of Vater.

3. One calculus is more likely to lead to pancreatitis than several, as more than one would weaken the face of the bile duct, which drives the one small calculus into the narrow outlet, like a ball valve.

Assuming this blocking of the outlet of the combined duct to be the cause of Acute Haemorrhagic Pancreatitis, then I think that experience proves this last statement to be unjustifiable. In Case II reported above, the ampulla was distended by a large number of small calculi, none of which excluded completely the outflow of bile into the duodenum. This did not prevent the retrojection of bile into the pancreatic duct. In Case I there was only one single large stone in the cystic duct and gall bladder. Without doubt Opra's experiments proved conclusively
that Acute Haemorrhagic Pancreatitis might be caused by bile gaining access to the duct of Wirsung. That it is so caused there is no evidence to show, except the case of Halsted's referred to above, and even there the bile staining in the pancreas may have occurred post mortem. Hlava produced Acute Haemorrhagic Pancreatitis by injecting the Klebs-Löffler bacillus into the pancreatic duct (Hamburger fur Pathologie 1897 viii page 492). It seems more probable then that any foreign and irritative substance in the duct of Wirsung, has the power of producing acute pancreatitis and that the bile injected by Hlave acted simply like any other irritant, and had no specific effect.

It has been proved that gall-stones often arise after bacterial infection of the bile passages. Welch has demonstrated the presence of micro-organisms in the centre of gall-stones. Gilbert and Fournier
accomplished the experimental production of gall-stones by injecting micro-organisms into the gall bladder of animals (Oster, Principles and Practice of Medicine, p. 562). Owing to the very close anatomical relationship between the bile passages and the pancreatic ducts, it is most probable that in catarhal inflammation of microbial origin of the former, the latter would also become infected. That such an infection might cause Acute Haemorrhagic Pancreatitis is certain. In only seven of the 21 cases I have collected and analyzed, were cultures made from the diseased pancreas. In everyone of these, practically pure cultures of bacillus coli communis were obtained. This is very strong evidence indeed that the condition is due to invasion of the pancreas by this bacillus. The ease with which such an invasion could occur is evident, and no
doubt the irritation caused by the passage of a gallstone, might
precipitate to the entrance of bacteria
from the intestine. This theory
accounts satisfactorily for the
frequent association of Acute
Haemorrhagic Pancreatitis with
gall-stones, and also explains
how the onset of the acute
symptoms so often follows
immediately after an attack
of typical biliary colic followed
by jaundice.

Recently (Dec 16th, 1902)
at a meeting of the Pathological
Society of London, Dr Starling
read a paper on the Physiology of
the pancreas. He found that
pancreatic secretion collected from
the duct had practically no
tryptic properties, but when
added to the "succus entericus"
it became powerfully tryptic.
He suggested that if "succus entericus"
regurgitated into the pancreatic
duct, this would produce the
tryptic ferment in the pancreas and probably give rise to Acute Haemorrhagic Pancreatitis. That this would be the result there is little doubt, but why should succus entericus regurgitate into the pancreas? It is very difficult to conceive any possible combination of circumstances which would cause such a regurgitation. No experimental proof of the possibility has been offered.

The three theories of the causation of Acute Haemorrhagic Pancreatitis which thus hold the field at present are:

1. That it is caused by the regurgitation of bile into the duct of Wirsung through the blocking of the outlet of the combined duct by a biliary calculus.

2. That it is caused by a bacterial invasion from the intestine, probably brought about by the irritation caused by a biliary calculus passing through the bile duct.
3. That it is caused by regurgitation of "acrisus entericus" into the pancreas, uniting with the pancreatic secretion, and causing a typhic and inflammatory reaction.

All the evidence at present available points to the second as being most frequently the exciting cause.

**Signs and Symptoms.**

No symptom is pathognomonic of Acute Haemorrhage Pancreatitis, yet when all the signs and symptoms are taken into account, a clinical picture is exhibited which should not be mistaken for anything else. While in many points there is a considerable resemblance to acute intestinal obstruction, and to deformed gastric ulcer or ulcer of some other abdominal organ, yet there are sufficient distinctions in the majority of cases to make a
diagnosis possible.

1. **Pain**
   - This is invariably the first and most urgent symptom. The pain is of the most excruciating character, exceeding in severity even that of bilious or renal colic. It is very sudden in its onset and is felt in the upper part of the abdomen, generally to the right of the middle line. In all of the cases I have collected this was the site of the pain, and in all it was of the utmost and most agonizing severity.

2. **Vomiting**
   - In all the cases I have examined this was a prominent feature. In some cases it was more urgent than in others, but in nearly all the patient vomited within half an hour of the onset of the pain, and continued vomiting at varying intervals till death intervened. The vomited matter in all cases was dark and "bloody" in type, consisting of a greenish sour smelling fluid
In no case was there faecal vomiting. Treatment had very little effect in controlling the illness.

3. **Collapse**

Fitz in his monograph (New York Medical Record, Feb 23, 1889) drew attention to the profound collapse which followed within a short time from the end of the illness. The collapse may indeed be so intense as to lead to immediate death, thus giving rise to the old term of "aphoplexia abdominalis pancreatia." (Friedrich Gimmern's Handbuch der Pathologie und Therapie Vol viii p 621). In all cases it is one of the most marked features, the radial pulse being usually almost imperceptible. When counted by the apex beat, the pulse runs generally between 80 and 90 per minute, but within a few hours becomes faster. The patient is cold and complains of feeling very faint. The collapse is usually greater than that met with in perforated gastric ulcer. In all
probability this profound collapse is caused the sudden fracture of
the swollen pancreas on the solar
plexus and semilunæ ganglia. Very
often the plexus is found, had
not been, to be embedded in
collected blood.

In only one
case have I found that the
bowels were not very obstinately
constipated (Hawkins, Lanced Aug 12th, 1893
p. 358). In all the others there was
constipation for at least three days
before the onset of the illness, and
in many cases a simple enema
gave no results. 4 laters, however,
is almost always passed freely
throughout the course of the disease.
In many cases the bowels move
naturally on the second day from
the onset of the symptoms. The
motion is usually normal and
never contains unchanged fat, as
sometimes occurs in chronic
pancreatic disease.

by Dr. Robson and
Mognihan considers this a valuable sign (Practitioner Vol II
1903 p. 275) in both of my cases it was slight and affected the
face only. It was present in a case reported by Mognihan (Practitioner
Vol I 1902-3 p. 504).

6. Jaundice. In many cases there is some jaundice, but it is
usually slight and may escape notice. It was present and reported
in 6 out of the 21 cases I have accumulated.

7. Temperature. The temperature is at first slightly raised
usually between 99° to and 100°. Later in the course of the disease
it rises, and often shortly before death reaches 102° or 103°. In one
case (Practitioner Vol II 1894 p. 914) reported by Paul it reached 104°.
The temperature may be subnormal throughout the whole course of the
illness, as in case II reported by myself.

8. Swelling. The abdomen is
always much distended, especially so above the umbilicus. The muscles are not rigid and the abdominal wall usually moves freely on respiration. There is a moderate amount of tenderness on palpation over the whole of the abdomen, but chiefly in the right hypochondrium over the region of the gall bladder. In the early stages there is no dulness on percussion, except sometimes slight in both flanks, caused by an accumulation of fluid in the pericardium. In the later stages there may be a large area of dulness, extending right across the abdomen over the region of the pancreas. Occasionally the pancreas itself can be felt as a large hard mass in the upper part of the abdomen. This, however, only very rarely happens (Bryant, Samuel Nov 19th. 1900 p. 1341).

There is often a partial suppression of urination. In one case no urine was passed for 16
hours after admission, then a catheter was passed and only four ounces could be drawn off. In only 1 out of 21 cases was sugar found in the urine (Nash, Lancet Nov 1, 1902 p. 1192). In the other 20 not a trace was discovered. In Acute Haemorrhagic Pancreatitis, therefore, the occurrence of glycosuria is of great rarity.

Diabetes: There is no change in the character of the feces, and no appearance whatever of unchanged fat or fatty crystals. In 1 case (Hawkins, Lancet Aug 12, 1893, p. 358) there was a small amount of blood in the stools.

The relative frequency with which the outstanding features of the disease show themselves is shown in the table on the following page. In the column on the right is shown the number of cases out of 21 which exhibited the particular symptom under consideration.
<table>
<thead>
<tr>
<th>Symptom or Sign</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute pain</td>
<td>21</td>
</tr>
<tr>
<td>Vomiting</td>
<td>21</td>
</tr>
<tr>
<td>Profound collapse</td>
<td>21</td>
</tr>
<tr>
<td>Constipation</td>
<td>20</td>
</tr>
<tr>
<td>Oliguria, reported in</td>
<td>3</td>
</tr>
<tr>
<td>Jaundice, reported in</td>
<td>6</td>
</tr>
<tr>
<td>Temperature raised</td>
<td>12</td>
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<tr>
<td>Temperature normal</td>
<td>2</td>
</tr>
<tr>
<td>Temperature subnormal</td>
<td>7</td>
</tr>
<tr>
<td>Swelling and tenderness of abdomen</td>
<td>21</td>
</tr>
<tr>
<td>Bulness in flanks, reported in</td>
<td>5</td>
</tr>
<tr>
<td>Pancreas palpable</td>
<td>1</td>
</tr>
<tr>
<td>Glycosuria</td>
<td>1</td>
</tr>
<tr>
<td>Blood in stools</td>
<td>1</td>
</tr>
<tr>
<td>Pulse between 80 and 110 at first</td>
<td>21</td>
</tr>
<tr>
<td>Pulse between 130 and 160 later</td>
<td>20</td>
</tr>
</tbody>
</table>

Prognosis.

The prognosis in these cases is very unfavourable, though not absolutely hopeless. Out of 21 cases, 2 only recovered, in both cases after operation. This works out to a mortality of 91 per cent. Of course in an obscure
disease like Acute Haemorrhagic Pancreatitis, there is always some doubt about the diagnosis, unless confirmed by a necropsy, but the case reported by Nash, (Jesuit Nov 1, 1902, p. 119) is so typical of the condition that there is no room for doubt. The other case of recovery (Milne, J. Med. Sept 12, 1897, p. 66) is not so clear, but was most probably correctly diagnosed of the remaining 19 cases, one—Mayrham—survived ten days, but the majority succumbed before the 4th day. The following table shows the time from the onset of the acute pain till death.

<table>
<thead>
<tr>
<th></th>
<th>died on the</th>
<th>1st day</th>
<th>2nd day</th>
<th>3rd day</th>
<th>4th day</th>
<th>5th day</th>
<th>6th day</th>
<th>7th day</th>
<th>8th day</th>
<th>9th day</th>
<th>10th day</th>
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<tbody>
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21
The pancreas is enormously enlarged, is usually surrounded by a large collection of blood clot, and may show many grey necrotic areas. In my first case the organ weighed 1 lb. 5 oz. but this is considerably in excess of the usual weight—10 to 12 oz. It is extremely friable, deep claret red in colour, and is obviously enormously distended with blood. The body and tail are much more affected than the head, which is often moderately healthy. The polar flexures is generally surrounded by blood clot and is often pressed upon or displaced.

On microscopic examination the interlobular connective tissue is found to be enormously distended with blood. Indeed in many sections nothing but blood can be distinguished. The outline of the cells lining the acini are
often completely observed; in
other places the cells are very
granular and their nuclei do
not take the stain; again in
other parts the lobules appear healthy.
The tissues are infiltrated with many
inflammatory cells, these being
particularly numerous round the
necrotic lobules. Scattered here
and there are patches of orange
coloured, feathery crystals. The
vessels are thrombosed, the arteries
empty. Sometimes bacilli can
be demonstrated in the section.\footnote{Bryant
listed Nov 1900 k 1342} In seven
cases, cultures made from the
pancreas, have shown growths of
bacilllus coli communis.

The occurrence of a large
collection of bile stained fluid in
the biliary cavity, in these cases,
have several times given rise to
the suspicion that there had been
a rupture of the bile passages. I
have collected 5 cases in which
this condition was present. In Case
though there were some finks of bile coloured fluid in the abdomen, there was no bile staining on the bowels. The bile probably escaped into the peritoneal cavity by the lymphatics, owing to the sudden and inflammatory condition of the gall bladder and duct.

In four cases the liver showed slight fatty degeneration. As in these cases gall stones were found, the fatty condition was probably not consequent on the Acute Pancreatitis.

The curious condition known as "fat necrosis" was present in all but 2 of the 21 cases of Acute Haemorrhagic Pancreatitis reported since 1893. Fat necrosis was first described and named by Baker in 1882 (Virchow's Archiv, xc 520).

This change is usually confined to the fat which lies in relation to the peritoneum. In nearly all cases it is present in the subperitoneal fat, in the great omentum, in the fat surrounding the pancreas.
and in the perinephric fat. In a curious case described by Bryant (January Nov 1900 p 1344) fat necrosis was also found in the pericardial and mediastinal fat. This is the only case on record in which such a condition existed. Fat necrosis may occur apart altogether from pancreatic disease, and has been observed during laparotomy for ovarian tumours (Hawkins Lancet 1893 p 361). Small dead white areas are found, varying in size from a pin's head to a crown piece, scattered over the fat in the abdomen. The cause of the condition is very obscure.

Hlava produced acute haemorrhage Pancreatitis and fat necrosis by injecting the Klebs-Löffler bacillus into the pancreas (Centralblatt für Pathologie 1897 viii p 792). Fleischer in the Journal of Experimantal Medicine 1897 vol xi p 416, concludes that "the escape of pancreatic secretion into the peripancreatic tissues is the origin of the necrosis," He also.
States that "the fat splitting ferment" of pancreatic secretion is demonstrable at certain stages of the pathological process. This might explain the occurrence of fat necrosis in the peritoneal fat, but does not offer any explanation of the necrosis which undoubtedly sometimes occurs in the mediastinal and pericardial fat. It is perhaps possible that the steapsin is carried from the peritoneal cavity by the lymphatics, thus causing distant necrosis. If Flenser's conclusion is correct, that fat necrosis is caused by the direct action of the fat splitting ferment of the pancreas, then it is almost certain that the lymphatics must be the channel of conduction.

Differential diagnosis:

The difficulties surrounding the diagnosis of this rare disease have been greatly exaggerated in the past.
It is true that very few of the recorded cases have been diagnosed before death, but this seems to be due more to the rarity with which the disease is met, than to any inherent obscurity of the signs and symptoms. Anyone who has seen one case of this most distressing malady, would have no difficulty whatever in recognising the condition again. The conditions which most resemble Acute Haemorrhagic Pancreatitis are perforated gastric ulcer and acute intestinal obstruction.

**Perforated gastric ulcer**

In this disease the patient is usually a female between the ages of 17 and 35; while Acute Haemorrhagic Pancreatitis usually attacks stout middle-aged males. The history is of the utmost importance, as instead of the repeated attacks of biliary colic which so often precedes the
onset of the latter, there have been the well known symptoms of pain and vomiting immediately after meals, and perhaps of haematemeses. The pain in both cases is sudden and severe in onset, but is worse in Acute Haemorrhagic Pancreatitis, and is associated with a much more speedy and profound collapse. The site of the pain is also of importance as after perforation it is felt all over the abdomen, while in the other it is always above the umbilicus and to the right of the middle line. The muscles of the abdomen are hard and board-like, and respiration is costal in type in cases of subacute gastric ulcers, while in Acute Pancreatitis the "boaroiding" is slight and the respiratory movements of the abdomen are almost undisturbed. In the former case the pulse is hard and wing and varies from 110 to 120 per
minute, in the latter it is usually almost imperceptible and not so rapid at first — go to 100 — but becomes faster as the disease progresses. The temperature in perforated gastric ulcers usually rises rapidly to even 104° or 105°, then falls to slightly above normal (Oxler) while in Acute Haemorrhagic Pancreatitis, it is at first either normal or only slightly raised, afterwards rising to 100° or 101°.

The Hippocratic face is very well marked in the gastric condition, but is not seen in the other. The liver dulness is not interfered with in Acute Haemorrhagic Pancreatitis.

Acute Intestinal Obstruction

There is usually but little difficulty in eliminating this condition, when examining a case of Acute Haemorrhagic Pancreatitis. The history, the swelling limited to the upper part of the abdomen, the absence of the Hippocratic
Faces, and the free passage of flatus generally clear up any doubt. The constipation is not so marked or so obstinate as in intestinal obstruction, and the bowels usually move spontaneously on the second day of the illness. The profound and immediate collapse so typical of Acute Haemosuccuous Pancreatitis is absent and in this condition the vomited matter is "bilious", never faecal.

The following table shows at a glance the important differences in the symptoms and physical signs of the three diseases.

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Acute H. Pancreatitis</th>
<th>Perforated G. Ulcer</th>
<th>Acute I. Obstruction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain</td>
<td>Extremely severe</td>
<td>Severe</td>
<td>Cælitis at first</td>
</tr>
<tr>
<td>Age</td>
<td>middle aged</td>
<td>14 to 35</td>
<td>various</td>
</tr>
<tr>
<td>Sex</td>
<td>male</td>
<td>female</td>
<td>various</td>
</tr>
<tr>
<td>History</td>
<td>biliary colic</td>
<td>gastric ulcer</td>
<td>none</td>
</tr>
<tr>
<td>Site of pain</td>
<td>right hypochondrium</td>
<td>Various</td>
<td>various</td>
</tr>
<tr>
<td>Collapse</td>
<td>Profound</td>
<td>Less profound</td>
<td>Slight at first</td>
</tr>
<tr>
<td>Vomited</td>
<td>bilious</td>
<td>bilious</td>
<td>bilious, bilious, faecal</td>
</tr>
<tr>
<td>Constipation</td>
<td>not absolute</td>
<td>none</td>
<td>absolute</td>
</tr>
<tr>
<td>M. Latus</td>
<td>free</td>
<td>free</td>
<td>none</td>
</tr>
<tr>
<td>Symptom</td>
<td>A. B. Pancrochat</td>
<td>P. Castorbury</td>
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<tr>
<td>Distention</td>
<td>Above undertone</td>
<td>General</td>
<td>General</td>
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<td>Respiratio</td>
<td>Costo-abornal</td>
<td>Costal</td>
<td>Costal</td>
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<tr>
<td>Pulse</td>
<td>90-160 at first</td>
<td>110 to 120 all</td>
<td>100-120 very</td>
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<tr>
<td>Temperature</td>
<td>Early normal</td>
<td>Early 105-106</td>
<td>Very stable</td>
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<tr>
<td>Facies</td>
<td>Everted</td>
<td>Hypertrophic</td>
<td>Hypertrophic</td>
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<tr>
<td>Liver dulness</td>
<td>Not obscured</td>
<td>Obscured</td>
<td>Not obscured</td>
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<tr>
<td>Pulse</td>
<td>Infratable hard</td>
<td>Hard very rapid</td>
<td>Faint feable</td>
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**Treatment**

It must be admitted that at present the treatment of this very fatal disease is unsatisfactory. The first indications are, of course, to treat the very urgent symptoms. The pain must be relieved by large doses of morphia and the collapse combated by stimulent enemata and injections of strychnine and digitalis. The bowels should be cleared by a simple enema.

As soon as the condition of the patient warrants it, the abdomen should be opened, the gall bladder incised, the common
bile duct explored for a calculus, and a probe passed down the duct into the duodenum to make certain that there is no obstruction in the ampulla of Vater. The gall bladder should then be stitched to the parietal peritoneum and drained. The gall stones which are almost certainly present should be removed. Under this treatment a case reported by Nash of Bedford recovered (Lancet Nov 1 1902 p 1192).

In the British Medical Journal, Feb 14 1905 p 307, Ettler Jones relates a case treated by laparotomy and excision of the pancreas, followed by recovery. These two cases are certainly hopeful and in all probability in the future the treatment of Acute Haemorrhagic Pancreatitis will fall more and more into the rapidly extending domain of surgery.

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