Where the position of the appendix is known before operation to be abnormal, as in Case 49, a suitable incision is used.

Only one case of hernia has been recorded as a sequence of the operation. The following table shows why operation was undertaken and the result:

	Total.	Recoveries.	Deaths.	Mortality.
Acute appendicitis without peritonitis	22	22	0	Per cent. 0
Acute appendicitis with pelvic peritonitis	29	27	2	6.8
Acute appendicitis with general peritonitis	5	4	1	20
Acute appendicitis with localized abscess	14	14	0	0
Perforation of the stomach or duodenum	5	5	0	0
	75	72	3	4

When there was a localized abscess the appendix was not removed in four out of fourteen cases. When the bowel was friable and the appendix difficult to reach it was thought safer to leave it behind. No. 37 was a child of 6, who had general peritonitis and died two days after the operation. No. 45 was a woman of 47, who died of portal pyaemia thirty-four days after admission. No. 58, a boy of 5, had a localized abscess and a gangrenous appendix. He died of intestinal obstruction. All these patients were operated on immediately after their arrival at hospital. It is open to question whether in No. 58 life might not have been saved by waiting until the acute condition was over before operating.

Complications.

These were: pneumonia, 1; pelvic abscess, 1; faecal fistula, 1; subphrenic abscess, 1; hernia, 1. The faecal fistula occurred in the case of a girl of 8 in whom the small intestines were matted together in such a way that the appendix had literally to be dug out of a mass of adherent bowel. During the process a loop of intestine was opened into. A faecal fistula developed which was successfully closed a month after the original operation.

Advantages of Limited Drainage.

The following advantages are claimed for the method of limited drainage:

- (a) Shock is diminished as time is saved, and the abdominal parietes are not interfered with to the same extent.
- (b) The after-treatment is less painful. Nothing is more nerve-shattering for a patient than the daily dressing with moving of three large tubes.
- (c) The formation of a faecal fistula is less frequent, most faecal fistulas being due to ulceration of the bowel by tubes.
- (d) Adhesions are not likely to form, and intestinal obstruction as an early or later complication is thus avoided.

Two of the perforation cases mentioned in this list were subjected six weeks after the first operation to a second laparotomy for gastro-jejunostomy. In both cases the only adhesions found were over the site of perforation just beneath the liver, and they did not interfere at all with the mobility of the stomach. Would this have been the case if several tubes had been used?

Swabbing the fluid out of the general peritoneal cavity or irrigating it with saline or antiseptics are procedures more likely to do harm than good by disseminating septic material and injuring the already inflamed peritoneum.

The objection which might be raised against the method of limited drainage is the additional risks it involves. As an answer to this it will be seen that out of 75 cases only two patients had secondary abscess formation.

The first patient (No. 34) had acute appendicitis of ten hours' duration, and very few people would have hesitated in closing the abdomen; the infection may have been very virulent. The other case (No. 75) had a very high gangrenous appendix just beneath the liver; the peritoneal cavity was drained, but contrary to rule the tube was not put in Douglas's pouch but behind the caecum, in the right renal fossa. A subphrenic abscess developed later. I am inclined to think that this complication would have been avoided by merely draining the pelvis, as we did in all our other cases. A tube often acts as a focus of infection; it irritates the neighbouring peritoneum and renders it less capable of self-defence. If placed at a spot where the peritoneum is not already inflamed it is more likely to act as a drain and not as an irritating agent. That is the reason why in most of our cases we drain not the appendix site but Douglas's pouch.

Amongst our other cases several were closed which had free turbid fluid in the pelvis. The majority healed by first intention. A few—amongst whom was No. 70, a perforated duodenal ulcer of twelve hours' duration—had a discharge

from their wounds a few days after operation. In other words, when there is an abdominal wound and fluid within the abdominal cavity, this fluid, if it does not become absorbed, finds its way out easily via the wound.

These considerations lead me to the belief that a more restricted drainage of the abdomen than has been practised in the past involves no additional risks, whilst it entails advantages which make it a definite improvement in the surgery of emergencies.

SPLENOMEGALY WITH PROGRESSIVE ASCITES AND LOUD VENOUS HUM:

SPONTANEOUS CURE OF ASCITES BY LEAKAGE THROUGH
ABDOMINAL WALLS.*

BY

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The following case, in addition to having features of diagnostic interest, eventuated in such an unexpected way that it seems worthy of record.

History.

A single man, aged 25, was admitted to the Sheffield Royal Hospital on June 21st, 1916, suffering from haematemesis. He is a coal-miner by trade. Up to the onset of his illness he had always been actively athletic, playing football regularly. He is a life-long abstainer and non-smoker. He has not suffered from any previous illness except occasional attacks of "influenza."

On the day of onset, in April, 1916, he had worked for three consecutive "shifts" in the pit—that is, practically twenty-four hours without a break. During the third shift he felt pain in his stomach. For the next few days he was out of sorts, and then suddenly vomited about three pints of blood.

He was kept in bed for six weeks, feeling ill and weak. After this he began to improve, and was getting about, until the night of June 19th, when he felt sickly and restless. Next day he had another severe haematemesis in the evening, and again, on the morning of admission, a still larger one.

On admission (June 21st) he complied with all the characteristics of an early chronic splenic anaemia:

1. Spleen enlarged to below navel.
2. Absence of enlarged lymph glands.
3. Anaemia of chlorotic type: red cells 1,350,000.
4. Leucopenia (5,000).
5. Liability to copious gastro-intestinal haemorrhages from time to time.

At that time there seemed no reason to doubt the diagnosis of splenic anaemia. The question of splenectomy was considered, and my surgical colleague and I agreed to wait until the profound anaemia following haematemesis had passed off before advising operation. During the next few weeks he improved. There was no recurrence of bleeding.

About the middle of July, 1916, the abdomen was found to be somewhat fuller, and to contain free fluid. The quantity was not large and it produced no symptoms. It appeared to be the type of ascites not infrequently found in cases of splenic anaemia, without necessarily connoting cirrhosis of the liver.

A very loud venous hum was now detected for the first time over the lower half of the sternum. The area over which the sound was heard extended from the level of the third costal cartilage above to the tip of the ensiform cartilage below, and for a short distance on either side of the sternum. The sound at the upper level was a typical "bruit de diable," such as is heard in the neck of chlorotics. It became gradually louder as the lower end of the sternum was approached, and, suddenly, on reaching the ensiform cartilage, its intensity became extremely increased, so that it might be described as a continuous "roar." At this latter area a thrill could be felt on light palpation, disappearing on firm pressure. The sound was slightly increased during inspiration, but was continuous throughout the respiratory cycle. There were no visible enlarged cutaneous veins.

During this time the anaemia showed little improvement in spite of treatment, and he had one or two attacks of epistaxis. Towards October, 1916, the ascites began to increase very much, so as to necessitate tapping on October 20th. After this it refilled rapidly and the tapping had to be repeated nine times during the next three months, varying

* An account of this case was given at the meeting of the Association of Physicians held at Birmingham in May, 1921.

quantities, up to 13 pints, being removed each time. About Christmas the lower abdominal wall became sore, and on further attempts to tap him it was impossible to get any quantity of fluid away. Abdominal section was considered, but owing to the condition of his abdominal wall it was thought to be very undesirable.

After this the man's condition was deplorable. The abdomen was greatly distended, there was secondary oedema of the legs, and it was necessary to keep him almost continuously under morphine on account of the constant pain from distension. It seemed certain that he would die.

As regards diagnosis, the progressive ascites with rapid loss of flesh following his early symptoms of splenic anaemia made cirrhosis of the liver seem probable—in fact, a typical picture of Banti's disease, except for the rapidity of progress, which was measured in months instead of years. This view was supported by the presence of the loud venous hum over the sternum, such as is occasionally found in cases of hepatic cirrhosis.

About the end of January, 1917, a remarkable change gradually came over the scene. The abdominal walls had become greatly distended, and there were large numbers of "lineae atrophicae." Through these stretched areas of the lower abdominal wall the ascitic fluid began to ooze at various points. The oozing continued night and day for the next few weeks. Dressings were applied constantly to soak up the fluid, and during the next week or two the abdomen began to diminish in size. The pain became less, so that morphine was no longer required. His appetite returned and he put on flesh. Week by week he improved steadily, and in April, 1917, one year after the first onset of symptoms, and about three months after the oozing had begun, it ceased completely and he was able to get about. Since then he has made an uninterrupted recovery.

I examined him in May, 1921, four years after he had left hospital. He was then in good health, well nourished, and able to do his work and enjoy life, though somewhat less strenuously than before his illness. The spleen is just as large as it was when I first saw him in June, 1916. He does not look pale. There is no increased fragility of the red cells. There is no ascites. There has been no further haematemesis. The venous hum is now limited to an area about the size of a halfpenny, situated over the middle line opposite the sixth costal cartilage. It is much less loud than before. There is a very faint thrill on light palpation.

REMARKS.

The venous hum was first heard in July, 1916, soon after the second attack of haematemesis, at the same time that a certain amount of fluid was first detected in the abdomen. How long the murmur had been present is uncertain. When first heard it was so loud and obvious that it could hardly have been missed had it been present when he was examined on admission about a month earlier.

Thayer¹ has given a detailed account of recorded cases of venous hums heard over and about the abdomen. These vary widely in position and degree according to circumstances. They may or may not be associated with visibly enlarged cutaneous veins. In four cases the sounds described correspond so closely to that in my case that their similarity of origin seems probable.

Rolleston² quotes a case under Ogle at St. George's Hospital of a man, aged 58, who died of cirrhosis of the liver. A loud murmur had been heard during life behind the sternum, and at autopsy the left internal mammary vein showed a varicose condition in this region. It then turned inwards to the falciform ligament and ran into the left lobe of the liver, joining the left branch of the portal vein.

He also records a case of his own, a man aged 43, who died of cirrhosis of the liver. There was a continuous bruit in the epigastrium, resembling the "bruit de diable" heard in the neck. The falciform ligament contained a thin-walled vein as large as the little finger. He says: "When the hum is loudest over the lower end of the sternum above the lower margin of the liver it is probably produced in a dilated coronary vein, or in a dilated communication between the internal mammary vein and branches joining the portal vein."

In all these cases the presence of cirrhosis of the liver was either proved at autopsy or was strongly suspected during life. So far as I can ascertain, no case having a similar loud venous bruit audible in this particular situation—namely, over the xiphisternum—has been recorded in any other pathological condition than cirrhosis.

Spontaneous Restoration of Portal Equilibrium.—I have not been able to find a record of any case of ascites which has similarly cured itself by multiple leakings through the

abdominal wall. Jenner³ published a case in which, after several tapings, the abdominal wall close to the navel burst with a loud report, but whether the result was satisfactory or not I do not know.

DIAGNOSIS.

As the patient is still alive the actual pathological conditions must, for the present, remain a matter of speculation. It is possible to exclude certain alternatives. Thus there is no question of alcohol, as the man is a life-long abstainer. Syphilis may also be excluded, as the blood gives a definitely negative reaction. Gaucher's disease is rare, and in the absence of any family history, known occurrence in childhood, or typical stigmata of the disease, such a diagnosis is not supported by the facts of the case. Familial splenomegaly is negated by the absence of family history, of jaundice, and of increased fragility of the red cells. The absence of jaundice also excludes biliary cirrhosis.

The diagnosis, therefore, seems to rest between:

1. A primary portal cirrhosis—not due to alcohol.
2. Thrombosis of the portal vein.
3. Splenic anaemia (with or without cirrhosis of the liver).

As regards (1) primary portal cirrhosis (not alcoholic), we may set the rarity of such a condition in adults, the absence of any early dyspeptic symptoms, the very large hard spleen preceding any recognizable ascites, and the complete cessation of progress when portal equilibrium was re-established.

There are several points in this case which seem to fit in with the diagnosis of (2) portal thrombosis. In the first place there is the sudden onset of symptoms. His first abdominal pain came on whilst he had been at hard manual labour at the "coal-face" for nearly twenty-four hours. We may assume, therefore, that he was not anaemic to any extent previous to that time. This alone is not pathognomonic, because it is familiar knowledge that in typical splenic anaemias patients usually do not present themselves for treatment until the spleen is already very large, and must therefore have been enlarging for some time without producing symptoms sufficient to require treatment. If, however, this abdominal pain marks the onset of portal thrombosis, the occurrence of severe haematemesis a few days later would also be explicable on the same grounds.

I am informed by his medical attendant, Dr. West Jones of Eckington, that the spleen was enlarged to a considerable extent whilst he was attending him before admission, but there is no exact record as to when it was first noticed. The onset of progressive ascites in the late summer would suggest further extension of thrombosis to other branches of the portal vein. The course and progress of the case are also consistent with this diagnosis. His profound anaemia when first seen in June, 1916, could be sufficiently accounted for as "post-haemorrhagic."

If the diagnosis of portal thrombosis is correct it would seem that now his obstruction is relieved he is in much the same condition as a man who has recovered from a femoral thrombosis, whose only inconvenience is the mechanical damage which the obstruction has caused to the affected leg. In his case the mechanical damage is an enlarged spleen and probably a good many abdominal adhesions.

There is considerable evidence, both clinical and pathological, that cases of portal thrombosis simulate splenic anaemia very closely. Parkes Weber⁴ recorded such a case in a woman. The onset dated from a confinement in August, 1910. She had ascites, enlarged spleen, and severe haematemesis five years later, from which she never recovered. At autopsy thrombosis of the portal tract was found. There was no cirrhosis of the liver. Galloway⁵ also recorded a case in a man, aged 28, with splenomegaly, leucopenia, recurrent haematemesis, and ascites. Splenectomy in his case was followed by death in ten days. At autopsy there was no cirrhosis of the liver, but definite portal thrombosis.

Similar cases have been known and recorded previously. Langdon Brown⁶ in his review of pyelethrombosis and suppurative pyelephlebitis, mentions a case which was admitted into St. Bartholomew's in 1872 and had been diagnosed as portal thrombosis twenty years before admission. The patient was a woman, aged 48. She had pain in the left side twenty-six years previously, following the birth of her second child. Haematemesis occurred at intervals; the spleen reached to the navel. At autopsy the portal vein was found to be a mere cord with fine lumen. The liver was normal. It is interesting to note that in the report of this case no mention is made of ascites having been present, in spite of the extent of the thrombosis.

As has been pointed out by others, there is no certainty, even if portal thrombosis is the only lesion found at autopsy, that it has caused the condition or even preceded the onset of splenomegaly. Even if it has done both we are still left in doubt as to what has been the cause of the thrombosis.

On the whole I am inclined to look upon this as a case of "portal thrombosis." Possibly the further course of the case may show more definitely the underlying pathological condition. Meanwhile the question arises, Should splenectomy be advised now? Considering the very large size of the spleen, the probable existence of considerable adhesions in the abdomen, and the fact that he is practically free from symptoms, I think the answer should be in the negative.

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THE RECORD OF A BRIEF EXPERIENCE WITH THE SACHS-GEORGI TEST.

BY

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*With an Historical Account of the Development of Flocculation
Tests for Syphilis,*

BY

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OUR only excuse for placing the following notes on record is the fact that hitherto comparatively few collated results of parallel Wassermann and Sachs-Georgi tests have appeared from laboratories in this country. It will doubtless be the policy of the near future to institute carefully planned investigations on the great scale dealing with parallel complement-fixation and flocculation tests with a view to the selection of the best working methods and the correlation with clinical experience of what may prove to be the future routine serological test for syphilis. Pending the appearance of such massed statistics it may be of interest to record the capabilities of a flocculation test performed with material sent from outside sources to the diagnosis department of this institute accompanied by clinical data of varying completeness. Our series comprises only 265 serums forwarded to the institute during the period July to October, 1921. One of us (B.) carried out the routine Wassermann tests, while the other (P.) undertook, solely as a matter of research, the parallel flocculation tests.

METHODS EMPLOYED.

Flocculation Tests.

Two antigens were employed throughout ("D" and "L"), and the results with both are summarized. Antigen "D" was that of Bordet and Ruelens (1919), with the addition of cholesterolin as recommended by Dreyer and Ward (1921), and the latter's technique was strictly adhered to so far as qualitative diagnosis was concerned, no attempt being made in this short series to probe the possibilities of quantitative evaluation. A dropping apparatus was used, and for the first 122 serums five tubes containing dilutions of serum varying from 1 in 1.25 to 1 in 26.4 in a total volume of 25 drops were put up. Later, as the results from the first tube were indefinite, and as a large quantity of serum was required, the first tube was dispensed with. The mixture of antigen, saline, and serum was incubated at 37° C. for seven hours in a water-bath, and the results then read. One batch of antigen was employed throughout, and was preserved in a cool incubator at a temperature of about 22° C. Antigen "L" was simply an alcoholic extract of guinea-pig's heart muscle, to which was added a 1 per cent. solution of cholesterolin in the proportion of 9 parts of heart extract to 1 of the cholesterolin solution.

For the first 132 cases four tubes were put up, containing in a total volume of 26 drops a constant quantity of pure serum (6 drops) and 20 drops of a dilution of antigen varying from 1 in 20 to 1 in 80. For the remaining half of the series the antigen was kept constant in the four tubes (20 drops of a 1 in 40 suspension) and the serum was varied from 1 drop to 6, the total volume being made up to 26 drops.

The dilutions of serum in the tubes were therefore 1 in 3, 1 in 6.5, 1 in 13, and 1 in 26. The serums had been previously heated at 54° C. for 90 minutes. The tubes were incubated for five hours in a water-bath at 37° C. and the results were read 20 minutes after

removal from the bath. They were reread after standing at room temperature for twelve to fourteen hours.

One batch of this antigen "L" was used throughout and experience showed that preservation in the cold (about 4° C.) was most satisfactory. Results with both antigens were recorded as "standard" (easily visible to the naked eye), "trace" (visible with lens), and "negative." At each test controls were used—namely, one Wassermann-positive serum, one Wassermann-negative serum, also previously tested positive and negative Sachs-Georgi samples. The antigen was also put up alone to exclude any non-specific precipitation.

Wassermann Tests.

These were performed with the antigen employed here—namely, an alcoholic extract of guinea-pig's heart muscle, to which is added before the test a 1 per cent. cholesterolin solution in the proportion of 5 parts of extract to 4 parts of the cholesterolin solution. Of this antigen a 1 in 90 dilution was used.

RESULTS.

Total number of serums tested	...	...	...	265
Of these, Wassermann positive	...	...	...	91
Positive flocculation ("L")	...	...	...	86
Positive flocculation ("D")	...	...	...	74

The degree of agreement may be otherwise expressed for the two antigens used, thus:

W+ L+ ...	...	83	W+ D+ ...	...	71
W- L- ...	...	171	W- D- ...	...	171
W+ L- ...	...	8	W+ D- ...	...	20
W- L+ ...	...	3	W- D+ ...	...	5
Total ...	...	265	Total ...	...	265

Thus, comparing the Wassermann and the flocculation tests by "L" antigen the total number of discrepancies in the series was 11, or 4.1 per cent., and in the case of "D" 23, or 8.6 per cent. The 91 positive Wassermann tests are classified as 70 "full positive," 10 "partials," and 11 "slight partials." Corresponding to this grouping the results with "L" and "D" are as follows:

	Total Positive.	Full Positive.	Partial Positive.	Slight Partial.
W ...	91	70	10	11
"L" ...	83	67	9	7
"D" ...	71	61	5	5

Thus 5 of the 8 missed positives with "L" antigen correspond to partial and slight positive Wassermann reactions, and half of those missed by "D" antigen are similarly accounted for. Only three serums yielded positive results with both "L" and "D" antigens, while the corresponding Wassermann tests were negative. In all three the flocculation tests were recorded as "trace" reactions. One was from a latent case under treatment and in which the Wassermann test had been positive seven months previously; another was untreated and was stated to have a rash on trunk and perforation of palate (certainly a suggestive history); while the third came with no particulars.

Treated and Untreated Cases.

One hundred and fifteen of the series were stated to be treated cases, while 150 were entered as untreated or "no particulars." They are thus grouped:

	Total.	W+.	"L"+.	"D"+.
Treated ...	115	31	28	23
Untreated ...	150	60	55	48
Total ...	265	91	83	71

Primary Cases.—There were 20 cases of primary sore, in 9 of which spirochaetes were found. They yielded 9 positive results by all three methods.

Only 5 cases of nerve syphilis appear in the list, with a yield of one positive result with the Wassermann test, the corresponding flocculation tests proving negative.

Discussion.

It has been customary to take the discrepancy percentage as a measure of the efficiency of a flocculation test, and on this basis the results from our small series compare favourably with those recorded by other workers in this country who have published results within the last eighteen months. The discrepancy percentage, however, must in the meantime be regarded solely as a provisional measure of efficiency. The ultimate interpretation of discrepancies between complement-fixation and flocculation tests must, as Lesser (1919) has wisely pointed out in a paper dealing with results by three different methods (Wassermann, Meinecke, and Sachs-Georgi), rest with the clinician, whose record of the past, present, and future clinical course of the case and response to treatment will materially assist the serologist to appraise with some confidence the results from different methods and to eliminate those which prove unsatisfactory. The flocculation test may indeed prove, as Dreyer and Ward suggest, to fit the clinical facts even better than the Wassermann. Even now, with some sixteen years' experience of the Wassermann test, doubtful reactions are constantly occurring (especially in treated cases), and the correlation of these with parallel flocculation tests may serve to place the latter on a proper footing with respect to the clinical condition in the diagnosis of which it is designed to participate. So far, the agreement