M. D. Thesis.

Duncan Forbes M.A. M.B.
18 Morton R.
Bootle
Liverpool
Subject

Some Clinical Cases of disease in the large Serous Cavities with remarks and an essay on Suppurative Pneumonia.
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Introductory Remarks.

Typical cases of disease of the Serous Membranes present no difficulty in diagnosis. Constantly however in practice we meet with diseases which show a divergence from the usual type, or we may have two totally different diseases presenting the same physical signs and symptoms. It is in such that a large and varied experience is of advantage, as generally there is some symptom or some physical condition present in one and absent in the other by which a definite opinion may be formed. Yet occasionally even this is impossible and all that can be done is to wait until the disease has advanced a step further, until there is a further development of the disease and time unfolds its true nature.

In the following clinical cases I have endeavored to illustrate this. I have therefore selected not the everyday case of disease in the Serous cavities but such as have appeared to me more uncommon.
Two of each will be sufficient, namely:

two of the Pleura, two of the Pericardium,
and two of the Peritoneum.

All came under my own personal treatment, partly in my private practice,
and partly in my capacity as honorary Surgeon to the Booth's Hospital.

I have not entered into any unnecessary
lengthy details in regard to the
Clinical Cases but have confined
my remarks chiefly to the diseased
organ itself.

The paper concludes with an essay
on suppurative peritonitis, and the
treatment preventive and otherwise which
in my own experience I have found
most successful.
Clinical Cases.

Plena.

Case A. Primary Carcin of Lung & Thoja.

Mrs B, a lady of independent means, stout and well nourished, aged 56, and the mother of several children. The history I got from her was, that about a month before I first saw her she had been attended by another medical man for an attack of congestion of the lungs. She had had some spitting of blood at that time. Since then she had suffered from slight cough, shortness of breathing, had lost flesh and appetite and was unfit for any exertion. She was not confined to bed and occasionally took carriage exercises. On examination of her chest I found the left lung dull from the clavicle downward. This wooden dulness was the same anteriorly and posteriorly. The breath sounds could be distinctly heard only at the apex, and there
they were much impaired in comparison with those on the opposite side. On the rest of the lung they were slightly deficient. Local auscultation and resonance impaired. She suffered no pain and her temperature was normal.

On the right side the percussion note and respiration were normal. Heart and liver normal, excepting that the heart sounds were rather weak but I accounted for that by the fact that the lady was rather of stout habit. I suspected fluid in the left pleura and proceeded to measure the sides with the result that instead of an increase on the left I found that it was 1/2 inches less than the right from spine to mid-sternum.

On close examination I then observed that on the left side there was decided flattening of the chest as compared with the right instead of bulging as would be expected if there was a large quantity of fluid. Such being the case my diagnosis was that I had here a thickened pleura with consolidation of the lung, a condition
which frequently follows Pneumonia, and that this probably was what the old lady described to me as Congestion of the Lungs.

The treatment adopted was a tonic character. It was tried for a few weeks, without any special benefit. She left home to try change of air on my advice. She was absent about a month. When she returned, she was no better. She had lost flesh considerably. The thinner had a yellow tinge which she accounted for by saying that she had been exposed to the sea air and the sun in her bath chair while at Southport. The lung was in status quos. In addition there was now a new complication. She complained of a difficulty in swallowing or when she did take anything in the way of food the soon after vomited it. She was now taking nothing but liquids. She first experienced this difficulty in swallowing soon after she went to Southport. I now for the first time suspected malignancy, I believed that probably there was a malignant
grow the original somewhere in the lung or its neighborhood. I told my suspicions to the husband and suggested a consultation as I reasoned that the case was going to end fatally.

Dr. Bates of Liverpool saw the patient in consultation with me. He and I did think at first that there might be fluid in the pleural cavity but on careful examination came to the conclusion there was not. He agreed with me that the disease was one of the lung but would not give a definite opinion as to what that disease was.

The old lady got worse, her symptoms were aggravated. She was now having injections of beef tea.

Dr. Bates again saw the case with me.

On this occasion I introduced the point of a hypodermic syringe and removed a quantity of bloody serum. This I examined microscopically and found many multinucleated cells mixed with blood corpuscles. From the whole history of the case, the profuse emaciation un influenced by any treatment
the colour of the skin, the presence of blood and the other microscopical characters of the fluid removed, we concluded that without doubt the disease was malignant.

At Dr. Bates's suggestion but against my own conviction I aspirated and removed a few ounces of blood stained fluid without however any marked benefit. The patient in a few days sank and died. No F.M. could be obtained.

Remarks:

I believe that the growth started at the root of the lung, probably in the bronchial glands and that after spreading outwards to the lung and pleura it began to take an opposite direction, inwards. Then came the difficulty in swallowing. The growth pressing on the oesophagus and perhaps more or less infiltrating the stomach itself. That the disease commenced in the lung and not in the oesophagus or stomach was evident from the absence of all symptoms referable to the oesophagus or stomach for a considerable time, and
then confinement to the lungs. It was only when the disease advanced to near its termination that there was any apparent interference with the digestive organs.

The case of Mr B is of considerable interest as being one of malignant disease affecting primarily the lung and as far as I could learn these being no previous member of the family affected by Cancer. The lung is frequently affected secondarily but seldom primarily. I lately attended a case of malignant disease affecting the lung but the disease of the lung followed some years after an epithelium of the lip. It occurred in an elderly gentleman. The epithelium was removed by Bicknattock of Liverpool at a very early stage before any affection of the neighbouring glands. The disease never showed any signs of return in the neighbourhood of the primary site. When I first saw him he was suffering from symptoms very similar to those of Mr B. excepting that there was no marked difference in circumference or extent side. This followed the same course...
as the former, except that a nodule appeared on the liver showing that it probably had become affected. He died from a sudden attack of haemoptysis.

In both these cases when they came under my treatment there was a considerable quantity of fluid in the pleural cavity combined with a serious closure of the lung, and yet in neither had the actual disease been made out, for I understood that the one case had been treated for congestion of the lungs and the other for bronchitis. I make this statement with the view of showing how difficult the diagnosis of such diseases sometimes may be especially in the early stage, later in the difficulty is more easily solved as there is a more marked development of special symptoms, but I question much if there are not many cases of a like nature in which the cause of death is certified as being congestion of the lungs or bronchitis.

These two cases appear to me to be typical of malignant disease of the lungs or pleura.
The spitting of blood appearing perhaps only occasionally, the gradual evacuation, the physical signs referable to the lung itself, and the change in the appearance of the skin all point in one direction. It may be said that they are also common to other diseases. True, but there is one symptom, however, which is not absolutely diagnostic. I am always in the habit of considering strongly presumptive of malignant disease of lung and pleura, that is the presence of blood-stained fluid in the pleura. It will be understood I mean this to refer to a disease occurring idiopathically, not to fluid found in the pleura of traumatic origin. I have never known a case in which I have aspirated and found such fluid recover, neither in my own practice nor in that of those with whom I am in the habit of coming in contact.

In the case of Mr. B. the flattening of the side in which there was fluid and its diminished circumference was interesting and at the same time rendered the diagnosis more difficult. I believe there
must have been an increase on the healthy side, a compensating enlargement, the left lung which was diseased being unable to perform its natural function, rather than any actual flattening or diminution on the left. Still it is possible that there might have been, as some malignant growths while spreading themselves tend to destroy and contract the healthy tissues. I have observed this in the case of the liver in a man who died from malignant disease of the liver and peritoneum. There were numerous carcinomatous growths in the peritoneum, and the liver, though affected by the same disease, was small and contracted. Occasionally we get a combination of cirrhosis and cancer, and this may have had something to do with this patient’s liver as from his own account he had been a hard drinker.
Case B. Thickened Pleura and Collapsed Lung.

The following is taken from some notes I kept while resident Medical Officer to the Liverpool Workhouse & Fever Hospitals.

In many of its physical signs it resembled the former cases, and in many an accumulation of fluid.


The only history I could obtain was that he had often suffered with his chest before.

I found him sitting up in bed and breathing with great difficulty. His face had a profligate right-lung normal in resonance except the base where it was somewhat impaired. Moist rales of all characters could be heard from base to apex.

The sputum contained blood, and he evidently suffered from excessive coughing of right-lung.

The left side which was the important was dull from the clavicle downwards and the breath sounds could with difficulty be heard anywhere.

Succession was negative of fluid, yet the absence of the signs indicative of fluid on percussion is not a certainty that...
There is none as when the pleura is full or nearly so of fluid, no sound indicative of the presence of fluid can be obtained on percussion.

In this case if there was fluid the pleura must have been full or nearly so. The temperature was normal. The usual medicinal treatment was tried without any benefit. The following day, Aug. 16th, the man was cyanotic and evidently much worse. I introduced the point of a hypodermic into the left pleura but found nothing. Next morning the patient died, Aug. 17th.

Autopsy. The right lung was in the condition of expected, excess of congested. The left side of the thorax was almost empty. All that represented the lung being a small carniplied body situated close against the spine. The lung was not bigger than a good sized fist. The left pleura was enormously thickened. There was no fluid in the cavity.

The other organs were healthy, except the heart which was somewhat hypertrophied.

Remarks. Some years previously this man must have...
had an attack of pleurisy with a large accumulation of fluid in the cavity. The lung has been collapsed completely and never expanded. Deprived of its function, it has got less and less until it reached the size in which I found it.

The left lung was so entirely changed from its natural character, that I believe it could have been of no functional value to its owner. Hence I think that the respirating membrane which I heard though very indistinctly on the left side was communicated from this right, and that probably the greatly thickened pleura aided its transmission just as an accumulation of fluid aids the communication of sounds from one side of the chest to the other.

This man had thus practically only one lung and it is not difficult to understand how an attack of bronchitis or pneumonia would rapidly induce a fatal result.
II Pericardium

Case A. Blood & Pus in Pericardium.

Thomas Hazelhew, age 22 Police Constable admitted to the Booths Hospital under on September 13th.

History of injury. While cleaning a window on the lower floor of a house, in trying to change his position from the front to the side of the window he missed his hold and fell a distance of some feet. His left side coming in contact with some iron railings. He remained suspended with the iron in his side for about half a minute until lifted off. The railing was topped of a small iron ball about the size of an ordinary marble.

Condition on admission. Collapse somewhat respiratory, weak, profuse, breathing difficult and laboured.

A wound about two inches in length was situated on the left side between the fifth and sixth ribs in a line with the anterior wall of the chest. The ribs broken. Air rushed in and out of wound during inspiration and expiration.
The blood and air were yet churned up together. The direction of the wound was upwards and inwards towards the cardiac region. The heart was displaced upwards. Its action very irregular.

The wound was dressed antiseptically but not closed.

**Sept. 14**

Patient slept at intervals during night but had been restless and thirsty. Breathing fairly easy. Temp. 100.8 Pulse 116. Respiration 28.

Dressings soaked with bloody discharge. Dressings removed. Several ounces of bloody discharge escaped from the wound. Wound is perfectly aseptic.

Heart below injury dull on percussion anterior and posterior. No empty sound. No cough. No blood stained sputum.

Open beats on a level with ripple. Evidence of fluid in pericardial sac. Echocardi to be heard in neighborhood of heart.

The pleural cavity appearing empty of fluid and there being no further hemorrhage I closed the wound.

Evening temp. 101.2.

**Sept. 15**

Patient has had a good night. Temp. 100.2.
Feels comfortable.
Dressings removed. No discharge.
Chest wall markedly puffed up. Some moist rales at base. No cough. Heart & pulse in status quo.

From the 15th to 20th the case went on fairly well and I have no hopes that it might end favorably. The temperature however was variable and the pulse was never satisfactory.

After the 21st his breathing became more difficult. The condition of the lungs did not account for this. The right lung was healthy. The left pleural cavity appeared to be free from fluid. Posteriorly the left lung was resonant but in front the area of superficial dullness extended from the second rib to the sixth.

Heart sounds weak. I suspected fluid in the pericardium, although there was no bulging in the Cardiac region.

Sept 22. Patient unable to lie down.

Pulse very weak and irregular.

Prepares suspension - Sump 6° 99.8 E. 100. S.

Sept 30th: rapid breathing, somewhat delirious. Pulse scarcely to be felt.

Heart sounds very indistinct.
After consultation with my colleagues I aspirated the pericardium in the usual way, prepared as I found pus to lay it open and drain it.
The aspiration removed only about one and a half or two ounces of bloody fluid and without any benefit.
It thus became evident that although there was fluid in the pericardium, congestion by this fluid was not the cause of the failure of the heart's action and consequent dyspnea.
The fluid removed consisted chiefly of blood and pus capsules.

At 11: Patient died. Dr. Port-Watson could not be obtained.

Remarks: The opinion I formed was that the bullet penetrated the pleural cavity but not the lung. It then took a direction towards the pericardium and heart. The day following the accident I came to the conclusion that there was fluid in the pericardium. This fluid must have been either venous rapidly absorbed following a pericarditis, or it must have been blood the result of the injury. Inclined to the latter.
Opinion and the end justified it. As the fluid removed by the aspirator was so small and so benefit accrued from its removal compression by it could not have been the Cause of death. The Cause of death was failure of the heart—from inflammatory and degenerative changes in the heart—fibres themselves, the result of the injury.
Case B. Blood in the Pericardium

The following case is of interest in connection with the preceding as indicating the effect of simple mechanical pressure on the heart.

Edwin Pearson age 25—Carlton—admitted into the North Hospital under me Sept. 20, 1885—suffering from a stab on left side of chest.

The history of the injury was that while returning from a dancing class with some friends he was met by another young man who bore him some ill will, and he without any immediate provocation drew a knife and plunged it into his chest.

Pearson fell and was removed to the hospital.

On admission he was cold, skin moist, markedly restless, pulse very small, scarcely to be felt at wrist. He was not quite sensible. There was slight vomiting and desire to defecate.

There was a wound over the third left costal cartilage. Its direction was inwards and backwards. In superficial extent it was about 3/4 of an inch.
On examining no abnormal dulness could be detected on left side of chest and the heart sounds on auscultation seemed natural though weak. I did not again see him until after death.

The house surgeon told me he rallied about three hours after admission for a time but again collapsed into his original condition and remained in this collapsed state until 11.30 a.m. on the 21st Sept. When he died, twelve hours after admission and about twelve and a half after assault. His temperature was below normal 97.4° at 10 a.m. on the morning he died.

Autopsy: The third costal cartilage was severed, edge of left lung punctured. The pulmonary artery slightly grazed and the intra-pericardial portion of the aorta was penetrated. The puncture into the aorta was extremely small, in fact only the point of the knife could have entered. The pericardium was greatly distended.
with blood, and the heart firm and empty. A small quantity of blood was in the left pleura.

The other organs were healthy.

In the case of Hazelwood, blood was effused into the pericardium at the time of the accident, but not in such amount as to materially interfere with the functions of the heart. Death in him was due to injury to the heart-walls, leading to inflammatory changes and degeneration in the heart fibres themselves. Whereas, in the case of Pearson, there was no time for inflammatory change, nor was the quantity of blood lost at all sufficient to cause death.

Death was due simply to mechanical pressure of the clot of blood in the pericardium preventing the heart both from receiving or expelling blood.

I consider that in Pearson the shock was such that only a small amount of blood escaped into the pericardium and hence the inability to detect any abnormal dulness over the cardiac region, or that temporarily the very small opening into the heart may
have become occluded but that after his rallying, the circulation gaining strength, the opening in the aorta again became free and the pericardium completely filled.

Though in my experience I have not met with a similar case of injury yet I have several times observed the same thing in rupture of an aneurism into the pericardium, the intra-pericardial portion of the aorta having given way, blood filling the pericardium and causing death not by loss of blood but by pressure paralyzing the heart's functions.
Case 2. Incrusted Vesicular Collection in Peritonitis.

Mary evading a very unhealthy looking woman, age 36, married, 6 children admitted to the Booth Hospital under me on 14th 1888.

History. For months previously she had been confined, being attended by a midwife. The labor was a natural one. Six weeks after the birth she had a chill. A local doctor was called in and attended her as long as she could pay him.

The fews ensuing that she came under the care of the parochial doctor and finally gravitated into the Booth Hospital. She had never been well since her confinement. The nurses were regular. She had some difficulty in menstruation.

Temperature. Morning 98°. Evening 100.6.

Heart and lungs healthy.

Abdomen. The abdomen presented a large fluctuating swelling occupying chiefly the right side completely filling it and pushing the intestines to the left. It extended an inch.
above the umbilicus. Fluctuation was uniform throughout, excepting in the median line behind the pubis and for a short distance above, where there was a hard undulated mass apparently solid. This consisted of the enlarged utero and bladder matted together.

The original examination the roof of the cavity presented a wooden hardness. The isthmus was firmly fixed, being quite immovable and drawn upwards and forwards. The sound passed an inch further than the shoulder.

One of the most striking features of the case was the immobility of the swelling. It could be moved readily to one side or the other, neither upwards nor downwards. On a superficial examination this might easily have been mistaken for an ovarian cyst complicated with pelvic cellulitis, but I had never seen an ovarian cyst bound down by adhesions so firm and so intimate before. In fact the adhesion was such that I at once recognized the impossibility of success in any attempt to remove it were it ovarian.

At my request the house surgeon entered with his hypodermic to remove the fluid. It was a
clean serous fluid just like ordinary ascitic fluid. He said there was no pus in it—but I do not think he examined the fluid microscopically.

The temperature and the history led me to think that the tumor had some connection with her confinement, and that it might have originated in a pelvic peritonitis and cellulitis. I determined to lap the tumor, but instead of choosing the median line, I kept well to the outside of the sheath of the rectus. The reason for this was that the uterus and bladder were drawn up and fixed in front and fluctuation was more perceptible to the outside. While should I find pus there would be more free drainage from an opening well to the right of the median line.

The operation was performed in the usual way when what appeared to be pus escaping I dissected down and laid the whole cavity keep open. Instead of pus however the great mass of the fluid was serous almost clear with pus cells in it, and bloody lymph, such as we are in the habit of finding in pernicious peritonitis.
or pleuritis. The intestines were completely separated off from the ascitic fluid collection. The cavity was then washed out with an antiseptic, two drainage tubes inserted, and the wound dressed antiseptically. The amount of fluid removed was thirty ounces. 

The day following the operation her temperature was normal and continued so uninterrupted until the wound finally healed.

Six weeks after the operation she left the hospital and now is in perfect health.

Remarks. My opinion is that this fluid was intra-peritoneal. A pelvic peritonitis, accompanied no doubt by cellulitis, resulted in effusion of serous fluid. This became engorged by adhesions between uterine, bladder, omentum, and abdominal walls, and then separated off from the intestines. As it increased in quantity it gradually became purulent. If the fluid were extra-peritoneal it must have passed its way up from below and lain between the fascia transversalis and peritoneum but in my own experience I never found fluid of the same nature, if
the result of inflammatory action, except where it was in contact with the inner layer of a serous membrane, and just as we can get a localised empyema, the adhesions between the layers of the pleura being such as to confine the pus to one part instead of being generally diffused throughout the pleural cavity, so we may have a localised collection of purulent matter in the pleura, and just as a localised empyema is more amenable to surgical treatment than a general so with the pleumoneum.
Case B. Large General prevalent Collection in Peritoneum.

Mary Baldwin age 9½ years, of a tubercular family came under my care Aug. 17 a few years ago. She had always been a healthy girl previously, and had suffered from no special disease except the usual ailments of children.

The parents informed me that on the fourteenth she had eaten some berries or pods growing on waste ground, and that night she was attacked with pains in the bowels and vomiting.

On the fourteenth when I first saw her she lay in bed on her back with the legs drawn up and flexed. She complained of great abdominal pain. The abdomen was very tender even to the touch. There was no marked swelling. The bowels acted slightly, and there was a good deal of chills and vomiting of a yellowish fluid. There was very troublesome and persistent. Tongue Gum through rest of day. Pulse rapid. Respiration hurried.

Temperature 101°.

My diagnosis at this time was that the child was suffering from an attack of
Gastro-intestinal with probably more or less Peritonitis. Whether what she was said to have eaten had anything to do with her illness I could not say. The usual treatment was adopted. Opium internally and fomentation to the abdomen with a milk diet
This to some extent relieved but did not remove the disease.
Towards the end of August she had a slight attack of Pneumonia affecting the left lung. There were some moist rales to be heard over both lungs.
The abdomen now appeared distended but was uniformly tympanitic. The pain and straining continued but the vomiting was less frequent. Temp. 100 F. 101.8.
In the beginning of September (5th) I first noticed a new phase in the disease namely the presence of fluid in the abdomen. The fluid occupied the usual position & fluid free in the abdominal cavity. This rapidly increased in quantity and was accompanied & hectic rigor, fever, and profuse sweating. The girl now had become extremely emaciated.
September 15th I proposed a preliminary tapping but the parents would not hear of any operation unless I would guarantee a cure. I therefore discontinued my attendance, but promised to see the girl if they sent for me.

Towards the end of September, I was again asked to visit her. The pulse now could scarcely be felt, breathing very difficult, abdomen tense almost to bursting, fluctuation uniform throughout. All nourishment was returned. In fact it was evident to me, and to the parents, that unless something were done to relieve the child she could not live till morning. It seemed at best a forlorn hope, but I again suggested tapping and obtained permission.

Tapping in the usual way pus came through the cannula. I then with assistance laid the abdomen freely open for about two inches, washed out the cavity with a weak antiseptic and inserted a drainage tube.

I am sorry I cannot state the amount of pus removed as a large quantity was lost over the bed.
The intestines could be felt but matted together and pushed upwards. The wound was dressed antiseptically. The day following the operation she was able to take liquids, and now retained them. The temperature after a few days became normal. The drainage tube was gradually thinned and on Oct 12th I removed it altogether. The wound then rapidly healed excepting a small sinus. Oct 19th she had a rigor, and the temperature which had previously been normal again rose. Suspected peritonitis pus and opened up the original incision.

At this time I could not distinctly feel my finger in the abdominal cavity. Adhesions had during the process of repair formed between the intestines peritoneum and parietes completely shutting off a small abscess communicating with the colon. About two ounces of pus escaped. I then washed out the abscess with an antiseptic and again inserted a drainage tube.

Her progress after wards was uninterrupted.

The wound gradually though partly healed and though a long time in regaining her former
strength she is now her mother can as well as ever.

As regards the diagnosis, I believe the original one to have been correct, namely, enteritis, accompanied by peritonitis. The disease certainly ended in suppurative peritonitis, yet at one stage previous to the presence of pus in the abdominal cavity being confirmed, many of her symptoms were indicative of general tuberculosis. The tubercular history of the family, the cough, hectic and even fluid in the absence of tuberculosis, or mesenteric disease pointed to tuberculosis. The suddenness of the onset indicated a disease, which usually runs a more acute course, and this opinion the ultimate history established.

I lately attended a young girl about the same age whose symptoms from the first were exactly similar, excepting that she had eaten nothing more irritating than indigestible fruit food. The disease ran the same course and ended in fluid free in the abdomen, which I had no doubt was pus. The amount of fluid was attained
the quantity of the previous case.
I proposed abdominal section but
the parents declined any operation
and the child died.
This from the first in all respects resembled
the former. The duration from my first
attendance until the death of the
child was about six weeks.

Suppurative Peritonitis.

Strictly speaking this term should be
confined to Peritonitis ending in Suppuration,
or pus in the peritoneum, but generally
speaking it is applied to all cases
in which pus is found in the abdomen
accompanied by more or less Peritonitis.
The Peritonitis thus may not be the
primary cause of the pus, but— the cause
that of the Peritonitis as when an intra-
peritoneal abscess finds its way into
the peritoneum and Peritonitis results.
Suppurative Peritonitis may be due to a
variety of causes. It may be idiopathic
as from exposure to cold. Such a cause
has sometimes been doubted but I recollect
of having under any treatment, when resident medical officers in the Liverpool Infirmary hospital, such a case in a man in whom the autopsy showed a considerable amount of sero-purulent fluid in the peritoneum and no other attributable cause could be ascertained.

Other causes are, inflammation spreading from neighbouring structures as in Gastro-intestinal or Pelvic cellulitis, ulceration of stomach or intestine, rupture of any of the internal viscera, tearing of oesophagus at the peritoneum, injuries and operations. It may accompany general septicemia as in so called Peroneral Fever.

Intussusception or torsion of the bowel may give rise to suppurative peritonitis, but generally these conditions, unless promptly treated, end fatally before the formation of pus can be diagnosed.

The Symptoms of Acute Periculent Peritonitis are very variable, but this depends very much on the stage of the disease. Vomiting and abdominal pain and tenderness are two of the most constant.
symptoms, yet I have seen a case of peritonitis in which death took place on the seventh day, and in which only on one occasion was there any vomiting.

The pulse may be rapid, full or weak and intermittent, the temperature above normal or subnormal.

In the early stage I have generally found the temperature high, and all the accompaniments of pyrexia, while in the later stages the pulse becomes weak and intermittent, the temperature subnormal and the skin cold and clammy.

The symptoms of supplicative peritonitis likewise vary according to the cause; thus when it follows the extension of inflammation from neighbouring structures, there is high fever, while when it is due to the bursting of an abscess into the abdominal cavity we may have from the commencement a stage of collapse with weak pulse and subnormal temperature from which condition the patient may never rally.

The stage of collapse is more evident
if the pus is putrid.

Generally speaking when there formation of pus accompanies a simple peritonitis, as in either of the recorded clinical cases the pus is not putrid, and in such cases a much more favourable prognosis may be given.

I am always much more anxious in regard to a case of Suppurative Peritonitis where the stage of collapse shows itself rapidly as I generally fear in those putrid pus. I know that some surgeons say that in Suppurative Peritonitis the pus is generally putrid. Such has not been my experience in primary fecal Suppurative peritonitis, then that has been so the putrid matter has usually been conveyed from somewhere beyond the peritoneum.

The hope of a successful issue after operation is much lessened when the pus is putrid. All abscesses are less amenable to treatment when putrid but what I mean is that putrid pus has a much more injurious effect on the peritoneum than ordinary non-putrid pus. The peritoneum...
when purulent pus has been in contact with it becomes coagulated, more friable and easily bleeding, whereas in non-purulent collections (non-purulent) the peritoneum has always appeared to me to be rendered clearer and thickened. Hence there is less likelihood of absorption should the abscess become purulent after operation.

Pus in the peritoneum, the result of supplicative peritonitis may be either generally diffused throughout the abdominal cavity or it may be encapsulated or localised.

The foregoing clinical cases illustrate both varieties. At times the abscess is multilocular. In fact, purulent accumulations in the peritoneum present the same varieties as are to be found in the pleura.

Acute purulent peritonitis is usually attended an extremity of fatal disease. Yet I think if the patient can resist the early acute stage, and the first presence of pus in the peritoneum there is every chance under proper surgical treatment of a successful issue.

The treatment of this abscess is the same as
that of any other abscess, namely the opening and free drainage of its cavity, with thorough washing out. In an empyema, I never wash out the pleura, unless the abscess is putrid, but in suppurative joints it, I always do, whether putrid or not, and my reason is, that otherwise it is difficult to get rid of the purulent lymph, which lodges among the intestines or low down in the pelvis, and keeps up a constant discharge preventing the wound from healing and aiding putrescence. In some cases I delay the washing out for twenty four hours. The collapse at times is so great before we can obtain permission for abdominal section that it would be injudicious to add to the risk of washing out. In these cases I may content myself by opening the abdomen & inserting a drainage tube. The following day I wash out the joint, and if the abscess is multilocular break down the partitions so as to convert it as much as possible into a single abscess cyst.

The success of any operative procedure in this disease depends considerably on the stage of the disease at which the operation
is undertaken.

I do not consider that in either the case of Baldwin or Hardy a surgeon in the early stage would have been justified in opening the abdomen. Such treatment would only aggravate the mischief. Acute peritonitis generally ends favorably without the formation of pus, but besides even though pus is present when in small quantity it is impossible to diagnose. We may suspect it, but we cannot be certain. In addition I do not think that a small quantity of sero-purulent fluid in the peritoneum of aseptic increases much the danger in itself of a case of peritonitis, excepting in so far as recovery will not ensue until it is removed by operation or otherwise. What I mean is that if death is to ensue in the early stage of acute peritonitis, it is the peritonitis which will be the cause and not the pus. There is therefore nothing to be gained by the too early opening of the abdomen. After the formation of pus there is generally to a considerable degree a cessation of the incessant pain.
The same relief is felt if the inflamed peritoneum as in any inflamed tissue when pus begins to form.

Lastly, the early stage is not the most favorable time for operation. It is after the inflamed peritoneum has become coated by lymph, and thickened, with the intestines more or less matted together. It is then, that if after operation the pus should become putrid there would be less likelihood of absorption.

In my experience prevalent collections in the peritoneum have shown no greater tendency to become septic than in the pleura.

An empyema may hurt externally or through the lung into the bronchi and become purulent and pus in the peritoneum may become the same by finding its way into the intestine or opening externally but the presence of pus in the peritoneum does not in itself predispose to putrescence.

I know that there is a prevalent opinion among some medical men, that there is something peculiarly virulent in pusulent fluid from the peritoneum, as Virchow
by their special care to avoid any injury from the knife during a P.M. when there is evidence of peritoneal peritonitis. This how can altogether depends on what was the cause of the peritonitis. If it was a case of peritonitis arising in the same way as either of the clinical cases then the pus has no special value. It becomes different if the condition of the peritoneum is only a symptom of a general disease, as for example if it resulted from the absorption of putrid matter from the uterus as in septicemia following parturition.
Here we find pus in the peritoneum as also elsewhere, and then it is of a most septic and virulent character, but not more so than that found anywhere else in this disease.
I do not here include that form of puerperal peritonitis, which follows difficult labor, or which results from the extension of inflammation from the cellular tissue, but such as results from the absorption of putrid matter and may be an accompaniment of general
Septicaemia.

I have sometimes been struck by the extreme virulence of that form of peritonitis, which results from violence during labor. Death results rapidly, occasionally even before the formation of pus. I was lately asked to see a case of this nature, where the medical attendant had justly feared attempted to deliver and failing he turned and brought away a dead child with great difficulty. The second day the patient developed peritonitis and died on the third. The autopsy showed that it would have been impossible to deliver the body of a living child. In such very acute cases we either find no pus or very little. There is excessive congestion of the whole of the peritoneum, serous effusion and great distension of the abdomen from flatus. In fact the distension of the abdomen from flatus is one of the most striking features and seems to give the patient most annoyance. Hence in the foregoing case I inserted a small forceps and cannula, and allowed the flatus to escape with temporary relief but
not with any permanent benefit. Some surgeons have spoken of opening the abdomen in such a case. This proceeding would be worse than useless, because there is little fluid of any kind to be evacuated, the intestines would still remain distended, and the peritonitis continue probably in a more aggravated form. Further the case would be complicated by the difficulty of retaining the intestines in their distended state in the abdominal cavity. Peritonitis of such a nature is best treated & opinion. The pyo-pelvic state furnishes another class of cases which sometimes result in suppurative peritonitis. I mean those abscesses from pelvic cellulitis. These abscesses usually find their way externally but occasionally they open into the peritoneum. The symptoms of such an accident are collapse with diffused pain and peritonitis which runs a very acute course. This follows from the bursting into the peritoneum of an abscess anywhere in its neighborhood, as where pus forms in peritonitis or
or in an ovarian cyst.

Few cases in surgery require more promptitude and decision, first, early to make an incision into any abscess in the neighborhood of the peritoneum, and secondly if such an accident should happen as that the pus find its way into the peritoneum, then immediately to lay open the abdomen wash out thoroughly its cavity, and introduce a drainage tube, at the same time attending to the primary abscess and its cause.

No matter how great the collapse there should be no delay. This is the only chance for the patient and if done early enough the peritonitis which follows may only be slight.

Thus sudden the man into the peritoneum previous healthy is entirely different from pus effused after an attack of peritonitis or from a serious collection becoming prevalent. Practical experience teaches this. In this one condition we have a most violent peritonitis with pus present from the commencement.

Whereas when the pus has followed the peritonitis, the serious movement as it have previously endeavored to show seems to become more tolerant.
Penetrating wounds of the abdomen make many additions to the list of suppurative peritonitis. At the north end of Liverpool, where all the large docks are situated, and where all nationalities are represented, the knife is not an uncommon method of settling a quarrel, and I have had a fair share of such cases to treat.

Neither the knife used nor the clothes of the injured man are the cleanest and therefore it is not surprising that penetrating wounds of the abdomen sometimes end in suppurative peritonitis. The knife may simply penetrate the abdominal cavity or it may implicate the intestine or other internal organ or involve some blood vessel and thereby lead to haemorrhage into the cavity.

The condition of one who has received such an injury is collapse and this is so whether the intestine is damaged or not. A piece of omentum frequently protrudes through the wound, which may prevent...
the entrance of air.
I lately read an article in one of the medical papers in which a surgeon advocated the opening the abdomen in all penetrating wounds and examining the intestine to see whether it was injured or not. Theoretically this treatment would be good but practically it does not present a very favourable aspect. Firstly because not one man in ten would allow such an operation unless he knew that his bowel was injured, and that without the operation there was no hope for his recovery. Further abdominal section without good grounds for it is running a very serious risk. No surgeon would open the knee joint if he could obtain as good a result by the aspirator. Secondly in most penetrating wounds the intestine escapes, and hence it would be wrong to add to the danger by an operation which in most cases would he worse than useless. Even though in one case a wound of the intestine
was found after abdominal section and be removed, this would not make up for two that died without any injury to the bowel.

Certainly if there is any just ground to believe that the intestine has been penetrated the only treatment applicable is to open the abdomen and excise the torn bowel.

The intestines lying free in the abdominal cavity are less resistant than the pancreas, and fortunately generally remain undamaged, unless a long and sharp pointed instrument is used.

In dealing with a penetrating wound of the abdomen the piece ofomentum protruding usually first calls for attention. There are different ways of dealing with this but I have always been in the habit of ligaturing it in two halves with chromic gut, cutting it off and returning the stump into the abdomen. The omentum protruding is much congested and has been lying exposed to the air and in contact with the clothing for some time. In addition it is frequently torn by the knife and may
have received septic matter from it. Therefore it is much better removed. The wound I close with antiseptic silk carrying the suture through the peritoneum on each side.

In most cases such a wound when treated in this way will readily heal, without any bad results, if there is no wound of the intestine for considerable vessel injured leading to haemorrhage into the peritoneum.

If however severe peritonitis follows which does not readily yield to treatment, or if the patient does not soon rally from the state of collapse following on the injury I suspect either that the intestine is injured or that there is some vessel bleeding into the abdominal cavity, or that septic matter from the knife or otherwise has been conveyed into the peritoneum, and I immediately open the abdomen, and examine the interior. The epigastric artery is the vessel which in my experience has most frequently given trouble.

Some months ago I was asked by a medical
friend to see a young man who had been stabbed. He and his brother had a quarrel and the younger made use of a knife with which he was cutting tobacco. When I saw him it was five days after the injury. The wound was in the left region exactly in the course of the epigastriae acting. He was suffering from peritonitis with abdomen distended, and tympanitic in front but dull posteriorly. I suspected fluid in the abdominal cavity and from the history and site of the wound believed the fluid was partially blood which had escaped from the epigastriae. I suggested opening the abdomen and washing it out, and at the same time if we found any vessel had been injured to endeavor to ligature it. In this proposition the patient entirely objected and he died two days afterwards. The autopsy showed that the epigastriae had been completely divided. There was a large amount of blood in the abdominal
cavity, some pus and extensive peritonitis but no injury to intestine. In this case had the abdomen been opened, washed out, the epigastrium tied, and a drainage tube inserted a very different result might have been looked for even five days after the injury.

The fact that the wound is in the immediate neighborhood of the epigastrium is not a justification for abdominal section on the mere supposition that perhaps it may be wounded and blood have escaped into the peritoneum. If this is done we may often expose the patient to an unnecessary risk.

A young man, a bus driver, was admitted to the Booth Hospital last August suffering from a stab in the abdomen. The house surgeon thinking that the epigastrium was injured sent for me.

The injured man was in a state of collapse, cold, pulse scarcely to be felt. The wound about an inch in superficial extent appeared to be immediately over the course of the epigastrium.
It was a penetrating wound and about two inches of omentum protruded from the condition of the patient and the position of the wound. I suspected internal haemorrhage. However, I thought I would close the wound and wait results. I therefore lifted the omentum, cut it off, and retained the stump in the usual way afterwards stitching up the parieties. In a few days the man was perfectly well having had no untoward symptom. The epigastric must have had a very severe escape. Had he remained in a collapsed state for a longer period than was attributable to the shock from the injury itself, then I would have felt justified in abdominal section feeling sure that internal haemorrhage was going on. Blood clot is extremely liable to degenerate into pus. Therefore when a large amount it ought never to be left in the abdominal cavity if it can be removed without much additional danger. In large amount—
It is much more liable to become pus than to be absorbed, and is one of the common causes of supplicative peritonitis.

Lastly, the healing or reparative process after operation in supplicative peritonitis is of interest. This process was well illustrated in the case of Mary Baldwin, where having occasion to open up the original incision I was unable to feel any ping among the intestines, the purulent collection having become completely separated off from the general abdominal cavity.

The final closing of such an abscess is by granulation, but the adhesions which form between the layers of peritoneum play a most important part in localising the pus, confining it to the lower portion of the abdominal cavity and the site of the external wound, where it can obtain free exit.