

Suppurative pylephlebitis : being a clinical lecture delivered at Guy's Hospital on January 6th, 1900 / by J.H. Bryant.

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Publication/Creation

London : Ash, printers, 1900.

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REPRINTED FROM *The Guy's Hospital Gazette.*

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SUPPURATIVE
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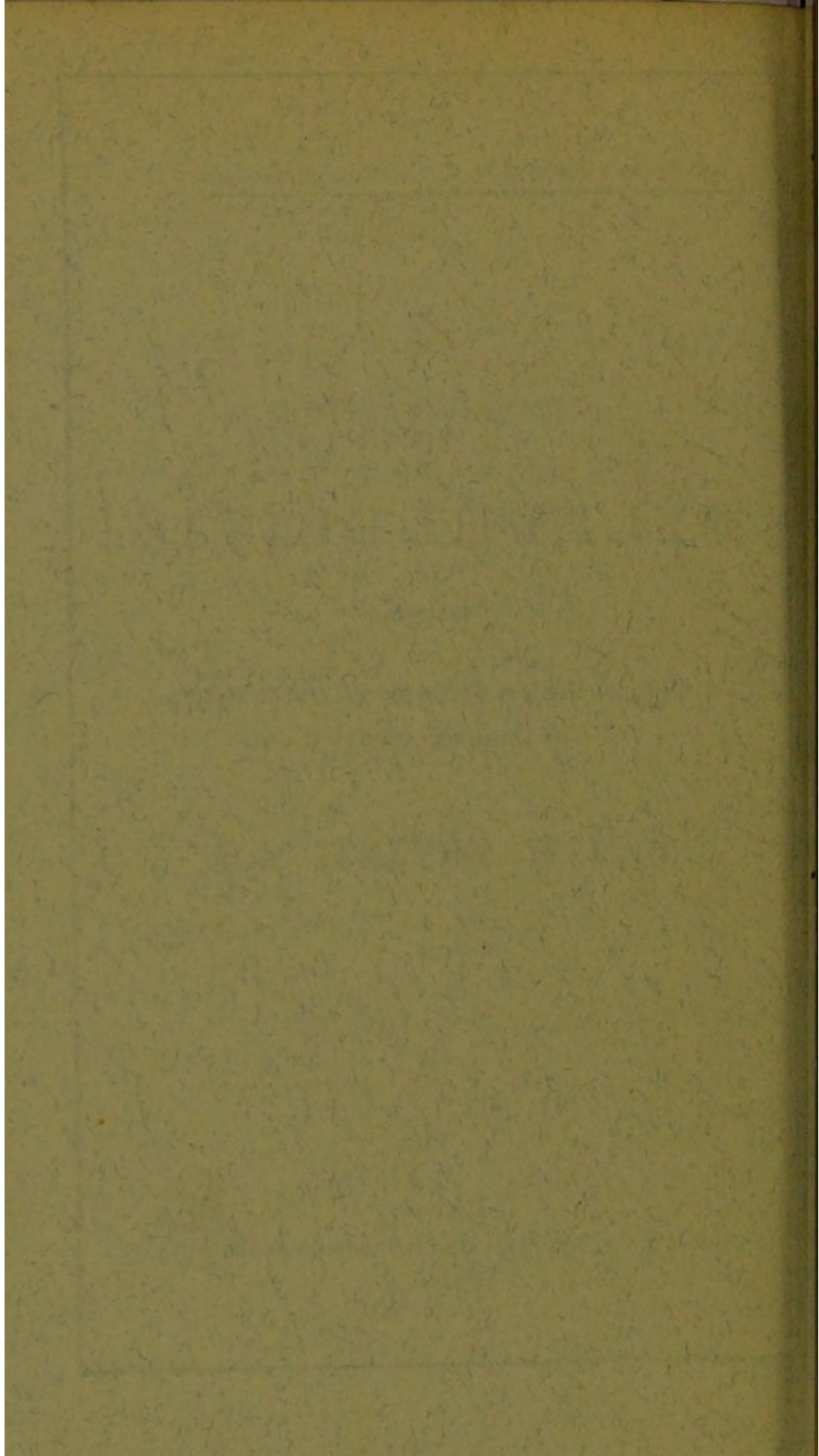
*A Clinical Lecture delivered at Guy's Hospital
on January 6th, 1900,*

By J. H. BRYANT, M.D.

LONDON :

ASH & Co., LTD., Printers, 42, Southwark Street, S.E.

1900.



SUPPURATIVE PYLEPHLEBITIS.

GENTLEMEN,—Last summer when I was in charge of Clinical, a case of Suppurative Pylephlebitis, that is, suppurative thrombosis of the portal vein, was admitted under my care. I have chosen this disease as the subject of my lecture to-day on account of its rarity, the obscure nature of the symptoms, the difficulties of making a correct diagnosis during life, and the very meagre description that you will find of it in the text-books. Suppurative pylephlebitis was the post-mortem diagnosis. The case was admitted as one of right-basal lobar pneumonia, and after careful physical examination and exploratory aspiration this diagnosis was thought to be most likely right up to the time of the patient's death, although it failed to explain in a satisfactory manner certain of the physical signs which were present. A few important negative statements have been added to the notes I shall read to you, which are taken from the excellent report of the case prepared by Mr. D. P. Watson. Perhaps there are some who will read this report and say, "Why was the disease not correctly diagnosed during life?" It is easy to be wise after the post-mortem examination. We unfortunately all make mistakes in diagnosis, and we shall continue to do so I am afraid, but we shall not fail to learn and profit by these mistakes. Do not be too ready to scoff when the post-mortem reveals a mistake in the diagnosis, endeavour to find out what points were missed, take

them to heart, and apply the lessons you have learned when you are faced by similar difficulties. You will learn more from the exposure of a mistaken diagnosis than from the confirmation of a correct one. I will read you the notes of this case, will then scrutinise the symptoms and discuss the diagnosis as far as possible, as we did in the ward, and then, lastly, if I have time, I will give you an account of the disease, based on twenty cases collected from our Records.

The patient was a boy, fifteen years of age, who was admitted on August 30th, 1899, for vomiting and pyrexia. On August 22nd he went to work and seemed in his usual health. At 1 a.m. on the 23rd he vomited and complained of pain in the right side. He felt feverish but had no rigors. He had eaten some green apples before going to bed. He did not remain in bed, but as he did not improve he was taken to see a doctor, who diagnosed gastro-enteritis, and ordered him to be kept in bed on a milk diet. He was very sick again on the 28th, and as the sickness continued and he seemed worse, he was admitted on August 30th. The bowels throughout have been freely opened and the motions very loose. There has been no cough and no expectoration.

Several years ago he was treated in King's College Hospital for a tuberculous ankle, and was kept in bed for some months. He was also treated at the same hospital for some disease of the left eye, for which iridectomy was performed. Two years ago he was treated for appendicitis by the same doctor, who sent him up for admission.

Condition on admission.—Temperature 101.6° , respiration 48, pulse 120. A fairly well developed boy. He seemed rather collapsed. His

face was flushed, his eyes sunken, his lips cyanosed, and his skin hot and dry.

The respirations were shallow, regular, 48. The lower part of the chest on the right side was bulged, the intercostal spaces were filled out, and this side of the chest did not move well on respiration. The tactile vocal fremitus was increased over the right lower lobe behind. There was absolute dullness on the right side, from the fourth space in front, and it extended round the chest in practically a straight line, being a little lower behind than in front. On auscultation the vesicular murmur was found to be absent over the dull area in front and over the lower part behind, that is, below the ninth rib; above this there was well marked bronchial breathing, and the voice sounds were rather ægophonic in character. Above the upper limit of dullness a few medium non-consonating râles were audible. There was no cough and no expectoration.

Pulse 120, low tension and running in character. The cardiac impulse was in the fifth space in the nipple line. Cardiac dullness normal. Sounds normal.

Tongue dry and coated with brown fur. Sordes on the lips and teeth. The abdomen moved well on respiration; it was a little rigid in the upper part, and there was slight tenderness on palpation below the right costal margin. The liver and spleen could not be felt. There was no abnormal area of dullness. There was no tenderness, and no tumour to be felt in the right iliac region.

Urine 1023, dark coloured, acid. No albumen, chlorides not diminished.

The patient was thought to be suffering from lobar pneumonia of the right lower lobe, complicated by some obscure hepatic or subdia-

phragmatic condition to account for the curious conformation of the right side of the thorax.

At 9 p.m. on the day of admission his temperature reached 104.6° , when he was sponged. On the 31st, at 10.30 p.m., he commenced to vomit, and the vomiting continued until 4 a.m.

On the 1st of September he was given minim doses of tincture of iodide every half hour, and the vomiting ceased. At 7 a.m. he became collapsed, cyanosed and almost pulseless. At 10 a.m. his temperature was 96° . On account of the bulging of the thorax on the right side, and the filling out of the intercostal spaces, the question of empyema, subdiaphragmatic abscess, and hepatic abscess all along had been considered, and it was deemed advisable to explore. No pus was found. Oxygen was administered, also musk and brandy, after which his condition improved for a time. Vomiting came on again, but was checked by tincture of iodide. The temperature rose to 103.6° , and patient sank and died at 10 a.m.

At the necropsy the body was found to be wasted. In the pleural cavity about a pint of clear serous fluid was found. The base of the right lung and the upper surface of the diaphragm were covered with recent lymph. The right lower lobe was quite airless from compression. There was no pneumonia. The heart weighed six ounces; it was healthy. The liver was firmly adherent to the diaphragm and to the parts which were in relation with it. The adhesions were firm and fibrous. The liver itself was much enlarged, and its upper level reached as high as the third rib, the lower edge did not project below the costal margin, and the organ weighed seventy-two ounces, or just double what it should have weighed. *In situ* it looked as if it had been dragged upwards by

means of its adhesions to the diaphragm, and possibly by actual permanent contraction of the diaphragm. It felt softer than normal, and the capsule was a good deal thickened. On section, multiple small foci of suppuration were seen, more particularly in the upper part of the right lobe, where they had run together in places. Throughout the right lobe little abscesses and collections of abscesses were seen. The left lobe was comparatively free. The portal vein and its branches contained sanious pus and ante-mortem thrombus; it was nowhere completely obstructed.

The spleen weighed four ounces, and was firmly adherent to the under surface of the diaphragm. The cause of the portal pyæmia was found in the appendix. The cæcum and appendix were bound down by firm, thick connective tissue. On making a careful dissection the appendix was found curled up in the form of the letter "O," and lying in the centre of the coil was an oval concretion which had ulcerated through the appendix, the hole in which was found. Another opening was found in the centre of the coil communicating with the cæcum, and, I take it, through this latter opening the pus of the appendicitic abscess discharged itself into the cæcum. The other abdominal viscera were healthy.

A specimen of pus was obtained from the liver for bacteriological examination, every precaution being taken to prevent the possibility of accidental contamination. Microscopical examination showed the presence of a few short bacilli; cultures showed the presence of the *bacillus coli communis*. No other organism was found.

I have read out rather fully the account of this case, and first of all, as I said, I will consider the symptoms and physical signs and

discuss their bearing on the diagnosis. A diagnosis must be based on a very careful estimate of the significance of the previous history, of the symptoms, and the physical signs.

The first point that I have to draw your attention to is the onset: it was acute, it commenced with vomiting and pain in the right side; and it is definitely stated that, the day before, the boy went to work and appeared quite well. He was admitted on the seventh day of his illness, and died on the ninth day.

The previous illness of appendicitis is an important point to consider, and it is also of importance to take notice of the diagnosis of the doctor who sent the boy into the hospital, viz., that the patient was suffering from gastro-enteritis. When I saw him in the ward the temperature was 104° , the respiration rate was 44, and the pulse was 140. I shall draw your attention to the ratio between the pulse, the temperature, and the respiration again. The other prominent symptoms were, the vomiting, the pain in the right side, the diarrhœa, and the dyspnœa.

The general appearance of the boy was interesting. He looked very ill, he was rather collapsed, his face was flushed, his eyes sunken, his lips cyanosed, and his skin hot and dry. Of the physical signs, the shape of the chest first attracted my attention. I noticed that it was not symmetrical; the right side was bulged, was much more prominent than the left, and the spaces on this side were filled out. There was less respiratory movement on the right side than on the left. The tactile vocal fremitus was increased at the right base, and there was absolute dulness extending round the chest on the right side (*vide* Diagram). There was

deficient entry of air below the ninth rib, and bronchial breathing above. There was no cough. We asked him whether he had ever had any cough or expectoration, but we could get no evidence of it.

The condition of the tongue was important; it was dry and covered with brown fur. With regard to the abdomen, it was not distended, but it was slightly rigid in the upper part. The liver and spleen could not be felt, and there was no marked tenderness. The iliac region was carefully examined, and there was no evidence of recent or old appendicitis. And then there is that statement about the urine—that the chlorides were not diminished. Those are the points on which we had to base the diagnosis, and taking all of them together we thought that the most likely one was pneumonia. But the pneumonia did not explain the bulging of the thorax or the filling out of the spaces. That is a condition which you do not get in a simple lobar pneumonia. We had to suggest something else to explain it, and we thought that it might be due to subdiaphragmatic abscess or possibly to hepatic abscess. Our chief reason for saying that it was not due to any subdiaphragmatic condition was the line of dulness. The upper line of the dulness comes, as you will notice, straight across in front and behind. With diseases beneath the diaphragm, you get the diaphragm pushed up, and the upper outline of the dulness is in consequence arched or dome-shaped. It comes something like this (represented on blackboard). On this account we thought that the *chief* condition from which this boy was suffering was some disease of the lower part of the right side of the thorax, and, as I have already stated, we thought it was pneumonia.

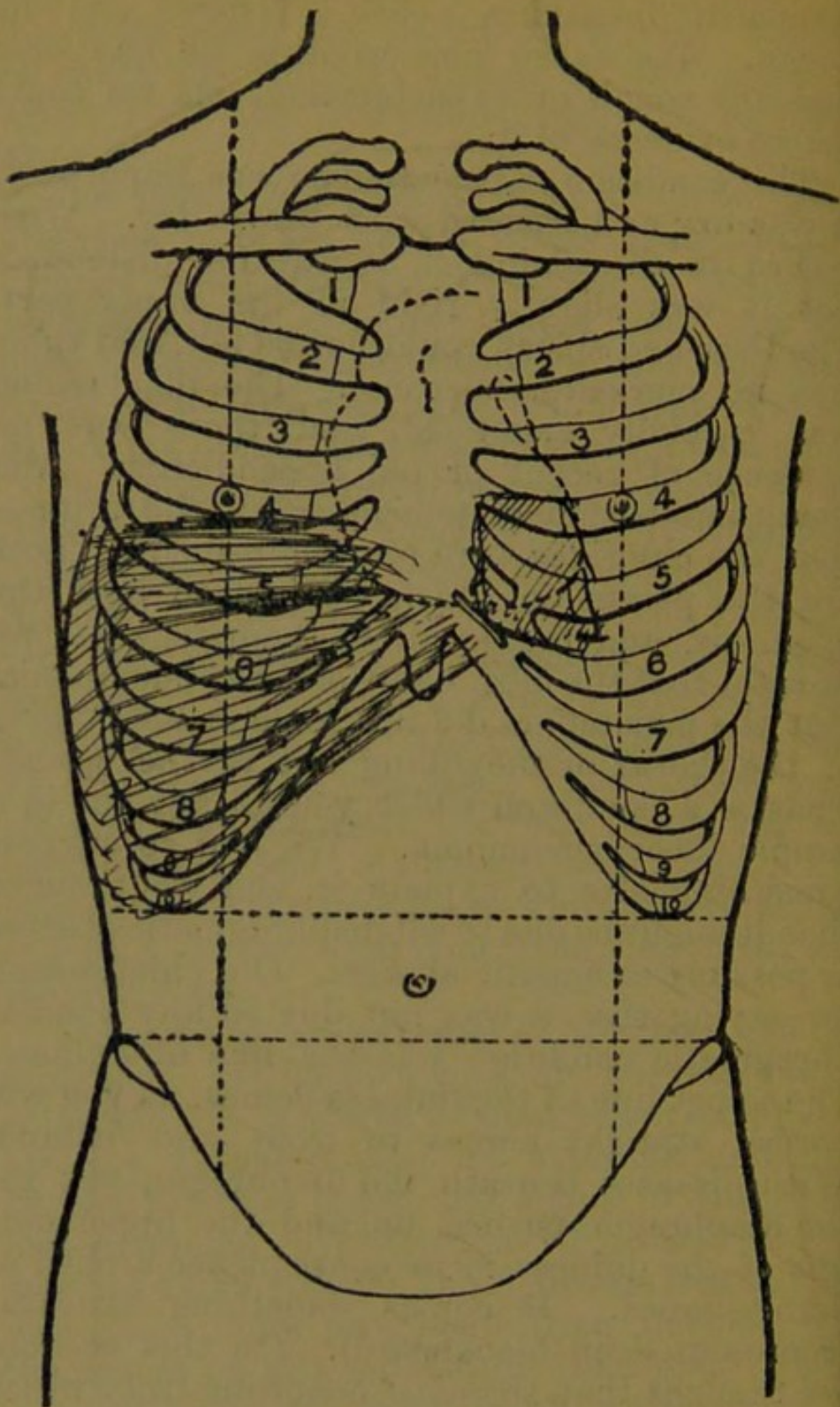


FIG. I.

Representing areas dull on percussion anteriorly.

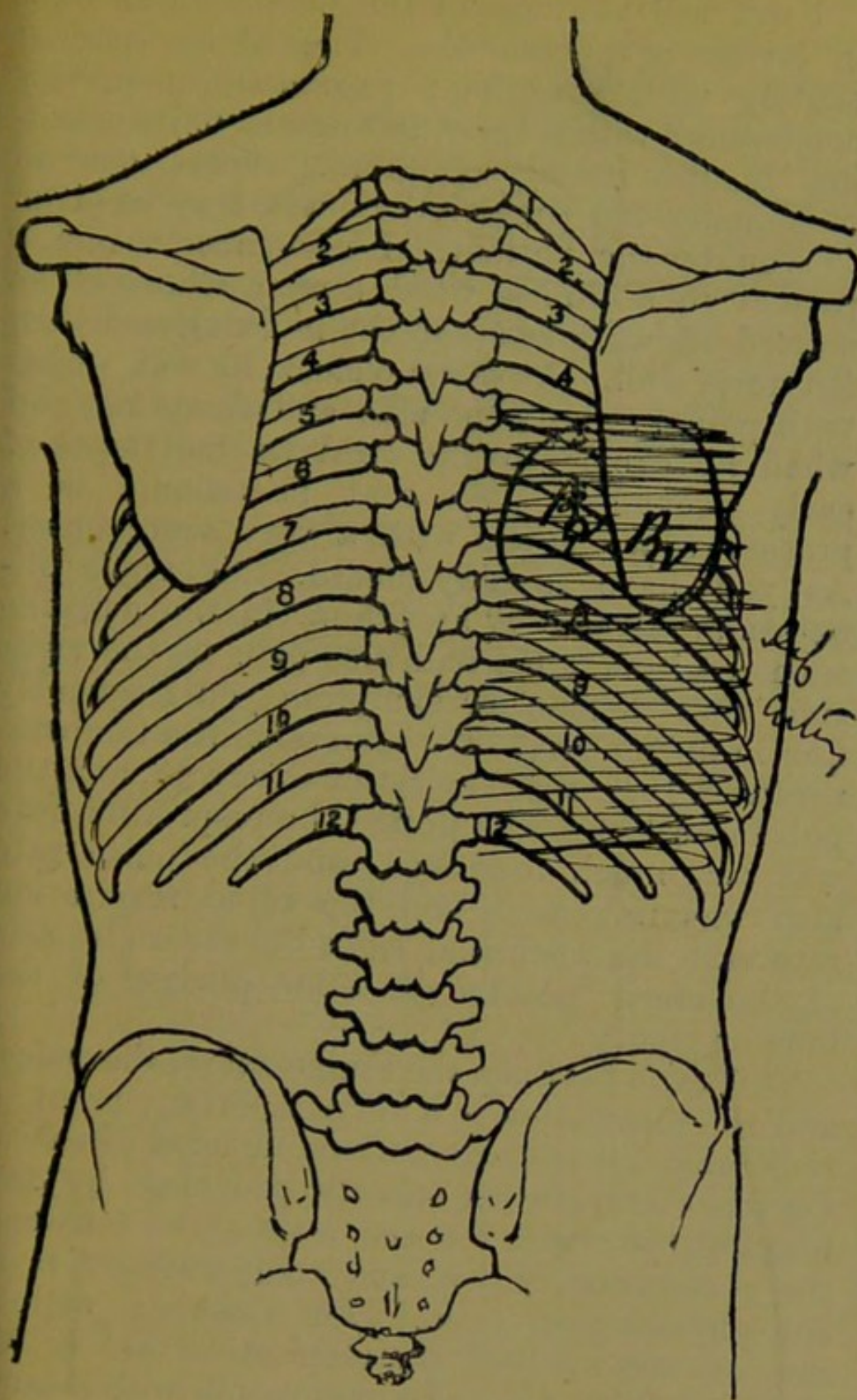


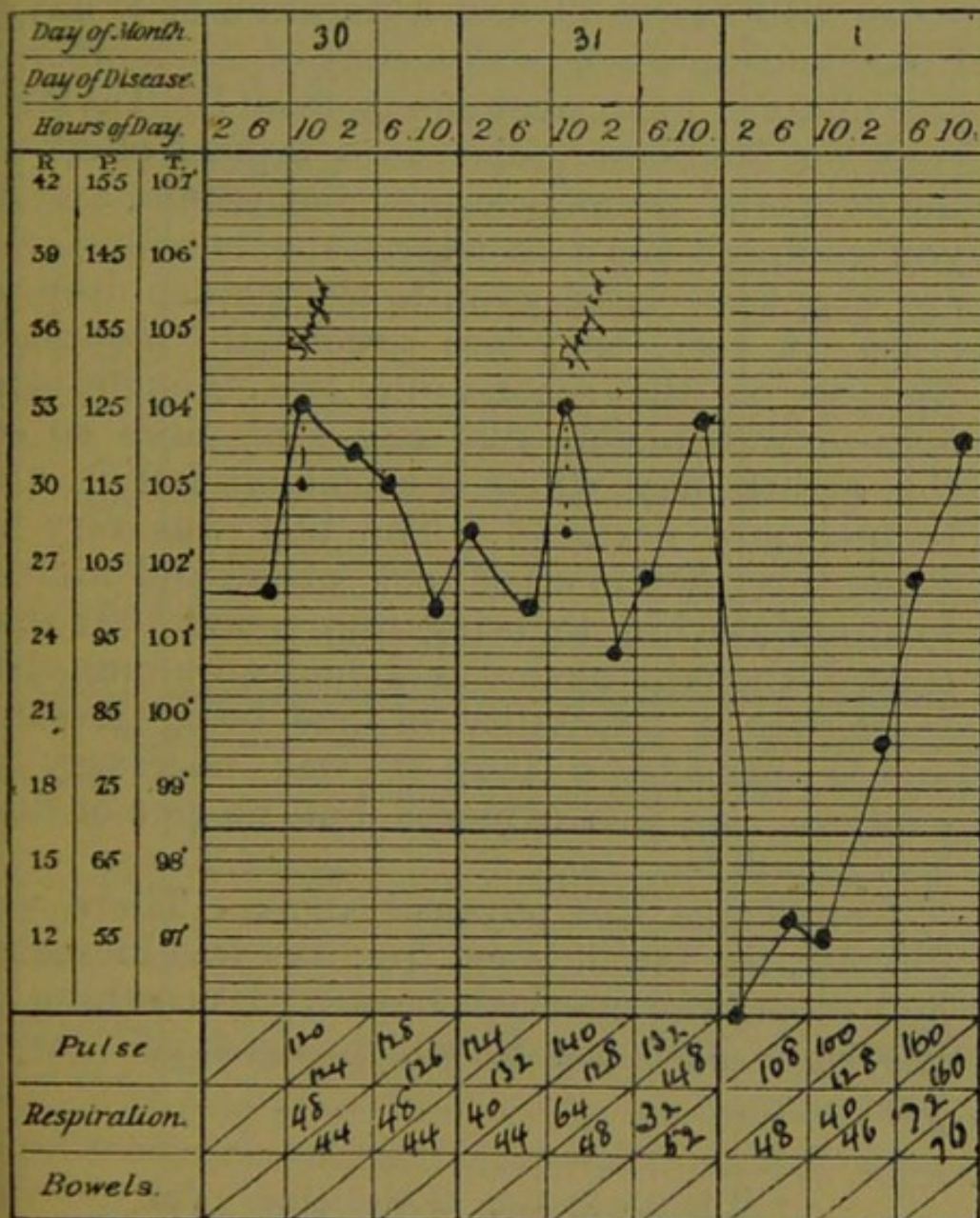
FIG. II.

The same, posteriorly.

I will tell you now of the points which were in favour of pneumonia. The illness started acutely with vomiting; pneumonia generally commences with a rigor, but occasionally vomiting is the initial symptom. Sometimes in pneumonia the time of the onset may be given to you by the patient. I remember seeing a patient in a clinical ward, a man, who said he started his work at six in the morning and then felt quite well. At seven o'clock he was seized with pain in the right side and shivering, and when he came into the ward he had signs of early pneumonia. So that pneumonia is a process which starts with a very acute onset. As you have already heard, this boy's illness started acutely at 1 a.m. in the morning with vomiting. He had also pain in the right side, that is another symptom you frequently find in pneumonia. On admission the temperature was 104° , the respirations were 48, and the pulse was 120. You notice the respiration rate was very much increased, and the pulse rate also was increased, and the rapid respiration rate with the abnormal signs on the right side of the chest, pointed to some disease of the lung or pleura.

This ratio between the temperature, the pulse, and the respiration is a very important point to remember; it is characteristic in some diseases. On the temperature charts provided by the hospital, you will see the ratio given between the respiration, pulse, and temperature; it is the physiological ratio. For instance, with a temperature at 104° , the respiration rate is 33, and the pulse 125. In pneumonia with a temperature of 104° , the respiration rate may be 40, or possibly more than that, and the pulse rate may be lower than the physiological ratio, *e.g.* 100. In typhoid fever, the ratio is also

very striking in the ordinary run of cases, with a temperature of 104° , the respiration rate may be 28—the respiration rate usually depends, however, upon the amount of bronchitis present; in some cases, providing that there is marked bronchitis, the respiration rate may be over 40—and a pulse rate, which is quite low, of about 80. In very bad cases of pneumonia when there is severe toxæmia, the pulse rate may be high, 120 or more. This boy was extremely ill, and we thought that the high pulse might possibly be explained by a severe toxæmia, and

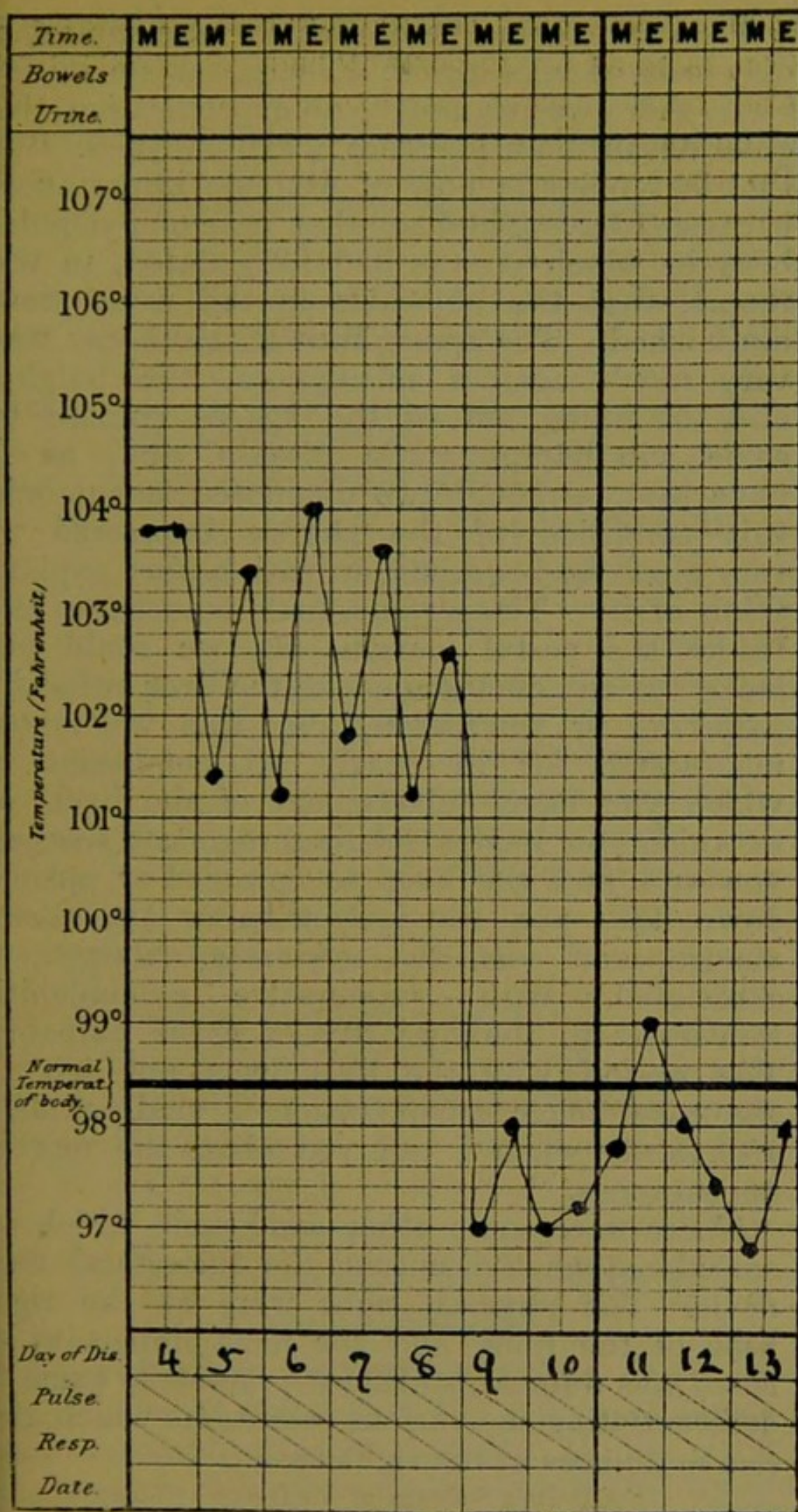


so we looked upon the temperature and respiration ratio as an important sign of pneumonia. And now I want particularly to draw your attention to the course of the temperature. On the first, second, and third days, you will notice (from the chart) that it was a continuous temperature. And on the second day of admission, that is, on the eighth day of the disease, there was a sudden drop which we thought was the crisis.

The crisis in pneumonia may come on as late as the eighth day.* When looking up our cases of pneumonia for Dr. Pye-Smith, I found that out of 316 cases 39 had a duration of 8 days. I have put up (here) also a copy of the temperature chart of lobar pneumonia, from Dr. Taylor's book on Medicine. This is a typical chart of the course of temperature in pneumonia, and if you compare the two charts the temperatures are about the same, even to the sudden drop or crisis, in fact, they are practically identical. And so on the second day we thought the disease was pneumonia, and still more so on the eighth day when the sudden fall in the temperature occurred. The boy was very ill when this pseudo crisis took place, and we had to stimulate him to bring him round. At that time I thought again, as I had all along, that there was a possibility of pus being present, and I considered that it was necessary to explore. He was explored but no pus or fluid was found.

Next come the physical signs. There was dulness, the tactile vocal fremitus was increased and there was marked bronchial breathing at the right base, all of which signs you get in pneumonia.

* Allbutt's System of Medicine. Vol. v.



Then as to some points which were against a diagnosis of pneumonia. There was no initial rigor, there was no cough, no expectoration, the chlorides in the urine were not diminished. But that last point is row of extreme importance, because I have found another case of pylephlebitis in which it was definitely stated, in the report, that the chlorides in the urine were diminished. The appearance of the thorax was very much against pneumonia, the bulging and the filling out of the spaces pointed to some sub-diaphragmatic disease, and, as I have said, although we diagnosed pneumonia this condition of the thorax impressed us very much and we had no satisfactory explanation of it. There was a little tenderness just below the costal margin, but we could not make out any enlargement of the liver either by palpation or percussion, and this was most misleading, for in nearly all conditions in which you have enlargement of the liver, or in which you have a subdiaphragmatic abscess, you will find the liver so enlarged or pushed down, that you can feel it below the costal margin, and even the moderate enlargement associated with suppurative pylephlebitis renders it in the majority of cases palpable. The reason why the liver was not felt in the case under consideration I shall explain afterwards, when I tell you about the morbid appearances.

There were some other conditions which we considered when discussing the differential diagnosis. The physical signs were at the right base of the lung, and the most important of them was dulness, so that it practically came to differentiating the various conditions which give rise to dulness at the right base.

Dulness at the right base.

A. Diseases of the lung and pleura.

1. Consolidation of the lung.

- a.* Lobar pneumonia.
- b.* Diffuse broncho-pneumonia.
- c.* Fibroid lung.
- d.* Basal phthisis.
- e.* Malignant growth.
- f.* Hydatid.
- g.* Large infarct.

2. Diseases of the pleura.

- a.* Pleuritic effusion {
 - Hydro-thorax {
 - Bright's disease.
 - Chronic valvular disease of the heart.
 - Simple serous effusion (inflammatory).
 - Seropurulent effusion.
 - Purulent effusion.
 - Hæmothorax.
- b.* Thickened pleura.
- c.* Tuberculous pleurisy.

B. Subdiaphragmatic diseases.

1. Subdiaphragmatic abscess.

2. Certain diseases of the liver.

- a.* Abscess.
- b.* Hydatid.
- c.* New growth.
- d.* (Suppurative pylephlebitis and pyæmic abscesses.)

First of all, to distinguish between these two main divisions diseases of the lung and pleura, and subdiaphragmatic diseases of the liver. In cases of subdiaphragmatic abscess and disease

of the liver you may get some local bulging of the lower part of the right side of the thorax and filling out the intercostal spaces. On percussion, as the enlargement pushes up the diaphragm, you get a dome-shaped outline to the dulness. Palpation of the abdomen generally reveals enlargement of the liver. In the case under discussion the dulness was straight across, and the liver was not felt.

Having distinguished between those two main divisions we will next consider these diseases separately. With consolidated lung, the typical physical signs would be, deficient movement, increased tactile vocal fremitus, dulness, and bronchial breathing, all of which were present.

In the disease of the pleura you may get filling out of the spaces, deficient movement, displacement of the cardiac impulse, tactile vocal fremitus absent, dulness, and deficient or absent entry of air. There was no marked displacement of the cardiac impulse and the bronchial breathing was so loud that pleural effusion was considered unlikely.

The question of diffuse bronchopneumonia was considered, but the course of the temperature and the dulness and bronchial breathing without moist sounds were considered points very much against this view.

Fibroid lung is a disease which may come on after pleurisy or empyema, and it is often associated with bronchiectasis, but there is generally evidence of that, *e.g.*, bronchial breathing, loud râles, and the expectoration of a large quantity of sputum, especially in the early morning, sometimes as much as half a pint or more. It has not an acute onset.

Basal phthisis was considered in this case on account of the tubercular history, but basal

phthisis is exceedingly rare; and, again, in this disease the bronchial breathing is associated with consonating râles, and the onset is not acute. With regard to the rarity of the disease, I have only seen one case of basal phthisis, and I think it is the only case that has been in the hospital during the last eight years.

Growth was not seriously considered on account of the acute onset and the age of the patient.

Hydatid was considered on this account. Hydatid most frequently invades the liver, and it may spread through the diaphragm and then involve the lung. In this case the enlargement of the thorax was so general, and there was no other evidence of hydatid. There was no expectoration. With hydatid of the lung there is generally hæmoptysis, and sometimes expectoration of daughter cysts, scolices, and hooklets.

A large infarct may occur in cases of heart disease, but if you look at this diagram (produced) the heart is not increased in size, the heart sounds are normal, and there is no evidence at all of a cardiac lesion.

Diseases of the pleura. With pleuritic effusion you do not, as a rule, get such a large amount of bronchial breathing as we found in this case, and it was so loud that we did not think it was due to pleuritic effusion.

Thickened pleura. That was not considered on account of the bronchial breathing being so loud. Nor was tuberculous pleurisy considered for the same reason.

And now I want to show you how it was that we went wrong in this case. The most unusual thing about it was that the liver was so large and yet could not be felt. The post-mortem showed that the liver was firmly adherent to

the wall of the thorax ; and I should imagine that the lower part of the liver first became attached to the thoracic wall, so that when it subsequently enlarged it could not come down into the abdomen. To show you how unusual this symptom is, of the twenty cases which I looked up, the liver was definitely stated to be enlarged in 60 per cent. of them and could in all those cases be felt below the costal margin.

Another point of similarity between pneumonia and this disease is, that an acute onset is not at all unusual. An acute onset was noticed in 60 per cent. of the cases of suppurative pylephlebitis.

The relation of a previous attack of appendicitis is important ; 40 per cent. of the cases analysed were due to appendicitis. The previous history of appendicitis, the condition of the thorax, the pyrexia and the general appearance of the boy should have put us on the track of suppurative pylephlebitis.

The actual cause of the suppurative pylephlebitis in this case, I take it, was appendicitis. Suppurative pylephlebitis is nearly always caused by some suppuration or some ulcerative lesion in the area drained by the portal circulation. I could not trace any thrombosis of the vessels in the immediate neighbourhood of the appendix, but pus was found in the portal vein. It may have taken place in this way ; there must have been some septic absorption resulting from the appendicitis, possibly the vessels of the appendix became inflamed, thrombosis followed, the thrombus became infected with pyogenic micro-organisms, a piece or pieces of this thrombus or colonies of micro-organisms became loose, and were carried off into the portal vein, and from thence into the liver, and in this manner little pieces of infected thrombi lodged in the

liver substance, in the small portal vessels, where they set up inflammation and suppuration, causing the formation of multiple small abscesses in the liver. In some cases a thrombus is found in the vessels immediately adjacent to the seat of the lesion, and it may be traced right up to the main branches of the portal vein, so that the way in which the liver is affected in this disease is by means of emboli or by a septic thrombus spreading from one of the mesenteric veins into the main branch of the portal circulation.

The usual post-mortem appearances in cases of suppurative pylephlebitis are these:—The portal vein is not, as a rule, entirely occluded, but it contains pus and is thrombosed, and that explains why it is that you do not get ascites; ascites is a very rare condition in suppurative pylephlebitis. The liver is usually enlarged. As I told you, in the cases which I looked up, in 60 per cent. the liver was enlarged, and it contains as a rule a number of small abscesses, and these abscesses can be traced directly to small branches of the portal circulation. The inflammation spreads to the capsule of the liver and causes adhesions between the liver and diaphragm. The spleen is sometimes enlarged, and it is not at all uncommon to find the pleura affected. I have analysed the weights of the liver; the greatest weight found was ninety-eight ounces, and the smallest forty-two ounces, the average being sixty-six ounces. In the twenty cases the diaphragm was described as being adherent to the liver in ten cases; it was stated not to be adherent in two, and it was not mentioned in the other eight cases.

No abnormal changes were noted in the heart. The spleen was enlarged in six of the cases, and its greatest weight was twenty-three

ounces. Pleurisy at the right base was found in eleven of the cases; there was an effusion in five of the eleven, the effusion being purulent in three. Another condition which is sometimes found associated with pylephlebitis is peritonitis; that occurred in half of the cases.

The organisms found were, the bacillus coli communis, and in one of the cases streptococci and a bacillus; the bacillus was not identified. In one case some diplococci were found; they were also not identified. Staphylococci have been found, and in a case of dysentery the amoeba coli.

And now with regard to the etiology of this disease. Eight out of the twenty cases, that is 40 per cent. of the cases, were due to appendicitis, two cases were associated with gall-stone and suppurating gall-bladder, two were secondary to gastric ulcer, one was secondary to duodenal ulcer, one was associated with empyema, one was a case of ulcer of the rectum and one a case of suppurating ovary, in two cases the cause was not found, in one case the cause was an operation for hæmorrhoids, and in one it was dysenteric ulceration of the colon. So that there are a large variety of causes even in these twenty cases. In other cases the disease has been found to be due to sloughing of the cæcum, abscess of the spleen, ulceration of the common bile duct extending to the portal vein, inflammation of the umbilical vein of infants, dysentery, and faulty operations for hæmorrhoids. Fagge described a case which was secondary to suppurating mesenteric glands, and the same authority also quotes a case of Frerich's, due to a fish-bone perforating the mesenteric vein. This disease has been known to follow also ulceration of the cervix uteri.

One of the most striking things, I think, concerning this disease is, that none of the textbooks describe it as being secondary to typhoid fever, by far the commonest disease in which we get ulceration of the intestine in this country. Why it should follow on some form of ulceration and not on typhoid fever does not seem very easy of explanation. It appears very extraordinary that with such extensive ulceration of the intestine, as in typhoid fever, you never see this disease.

Then with regard to the sex of the patients, eleven cases were in males and nine in females. The ages varied from fifteen to sixty-two; the average age was twenty-nine. An analysis of the ages showed that eight of the cases occurred in the second decade, five in the third, two in the fourth, four in the fifth, none in the sixth, and one in the seventh. So that thirteen of the cases occurred in persons up to thirty years of age.

The onset was acute in twelve cases, and it was said to be gradual in two; it came on with pain in the right side in two of the cases, there was abdominal pain in two, it commenced with a rigor in three, and with vomiting in four. Pain in the back, pain in the loins, diarrhoea, acute pain in the loins; an indefinite onset sometimes with irregular chills, fever and sweating—these are the early symptoms that you may find.

It is interesting also to note for what symptoms the patients were admitted. One patient was admitted for vomiting and pyrexia, two for pain in the right side of the chest and pyrexia, one for pain in the right hypogastric region and jaundice, one for biliary colic, two for abdominal pain, and one for discharging sinus in the groin.

And then as to the symptoms of the disease. The aspect of the patient is very striking in most cases. The majority show wasting, and sunken eyes. The patients are thin, they generally have an anxious expression, some of them are jaundiced, they nearly all look very ill, some of them are restless, some are collapsed. Osler describes a "muddy complexion" as being characteristic of this disease. Many of the patients have the features of a general pyæmia.

The temperature in this disease varies considerably. The highest temperature recorded in the twenty cases which I looked up was 107° and the lowest 96° . Sometimes the temperature is continuous and is not associated with rigors. Rigors occurred in 50 per cent. of the cases.

Then there is the relation between the temperature and pulse and respiration. Generally the pulse rate is high, so is the respiration rate, and the pulse rate is, if anything, above the physiological ratio.

Vomiting is a very common symptom, and, as I have already mentioned, occurred in 60 per cent. of the cases; and it is mentioned in the text-books also as being a most important symptom of the disease.

Diarrhœa is a common symptom, and it occurred in nine of my cases.

Constipation is noted in some of the cases, viz., in seven of them, delirium in three, jaundice in eight, that is, in forty per cent. Pepper, in his "System of Medicine," mentions that three-fourths of the cases are jaundiced. Dr. Fagge says that pylephlebitis often produces jaundice, and it does so much oftener than a single abscess in the liver.

The physical signs. The tongue is described as being dry and coated with brown fur. The abdomen is generally a little distended, and

there may be some rigidity over the right hypochondriac region, some tenderness is usually noticeable in that position, and patients often complain of pain in that region. As a general rule the liver is enlarged, and if you can feel the liver it is usually tender. The enlargement of the liver is uniform in the generality of cases; occasionally you may be able to feel little soft nodules on the liver. I remember seeing, some years ago, a patient suffering from suppurative pylephlebitis, she came in with a high temperature and rigors, she was jaundiced, the abdomen was a little distended, and the liver was enlarged, the edge could be felt on a level with the umbilicus, and on its surface little nodules which were soft, and one of them which was much larger than the other fluctuated. This one was explored with a hypodermic needle, and some pus was drawn off. A diagnosis of pyæmic abscesses in the liver was made. I performed the necropsy on this case, and found portal pyæmia, pyæmic abscesses in the liver, and the cause was a suppurating gall-bladder. The gall-bladder had given way at its attachment to the liver, and just above the spot where it had perforated there was a small abscess in the liver, which had set up a general portal pyæmia.

The urine is generally normal. Albuminuria was noticed in four cases, and in those cases in which jaundice was present the urine contained bile. On the whole, the urine did not show any striking or characteristic changes.

A note, to the effect that the chest was bulged, was contained in three of the reports, so that, apparently, it is not such an unusual symptom as I thought it was. One case showed falling in of the chest, it was associated with empyema.

The duration of this disease is rather an interesting point; it is very variable—from five to 296 days. Dr. Goodhart described a case which lasted for 296 days, and in this last mentioned case signs of healing in the liver were found. The average duration, including that case, was fifty-six days, but if you exclude it, the average duration was forty-three days; the shortest was five days and a few hours. This case which I have read to you to-day was the next shortest, namely, nine days.

A correct diagnosis during life was rare. One case was diagnosed as gastro-enteritis, another pneumonia, another gall-stones and biliary colic, two as typhoid fever, one as appendicitis and hepatic abscess, and only two correctly. The diagnosis of suppurative pylephlebitis is not at all easy. The remarks I have made to-day will, I hope, help you if you ever come across this rare disease. The treatment, I regret to say, is only symptomatic. The only hope of cure would be an early eradication of the cause.
