

## **A case of pneumococcal peritonitis / by J.H. Bryant.**

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## A CASE OF PNEUMOCOCCAL PERITONITIS.

By J. H. BRYANT, M.D.

HARRIET S—, aged four years, was admitted into Guy's Hospital under my care on May the 11th, 1901, for vomiting and abdominal pain. She had always enjoyed good health until the evening of May the 9th, when she was seized with severe vomiting after supper. She vomited about six times during the night. The sickness ceased on the following morning, but she appeared to be very ill, and would not take her food. On the morning of the 11th, as she was much worse, and had not taken any food, she was brought up to the hospital, and was at once admitted. She had not been sick since the evening of the 9th. The bowels were opened on the 10th, and the motion appeared to be natural. There was no discharge of blood or mucus from the rectum.

On admission the pulse was 140 to the minute, the temperature  $102.8^{\circ}$  F., and the respirations 40 to the minute. She appeared to be very ill, and had a drawn and anxious expression. The abdomen was tense, rigid, and very tender all over. There was no particular pain or tenderness in the right iliac fossa. There were no physical signs of pleurisy or pneumonia. I saw the child soon after admission, and came to the conclusion that she was suffering from acute peritonitis or diaphragmatic pleurisy, and I suggested the possibility of a pneumococcal infection on account of the absence of any localising symptoms of appendicitis or other diseases. Mr. Dunn saw the child with me shortly afterwards, and decided



not to operate, chiefly on account of the absence of vomiting and constipation, and because he was inclined to the view that her condition was due to diaphragmatic pleurisy or pneumonia.

During the night she was decidedly worse; the abdomen became more rigid and tender, but the drawn anxious expression was not so well marked. She also vomited three times, and it was with great difficulty that she could retain any food.

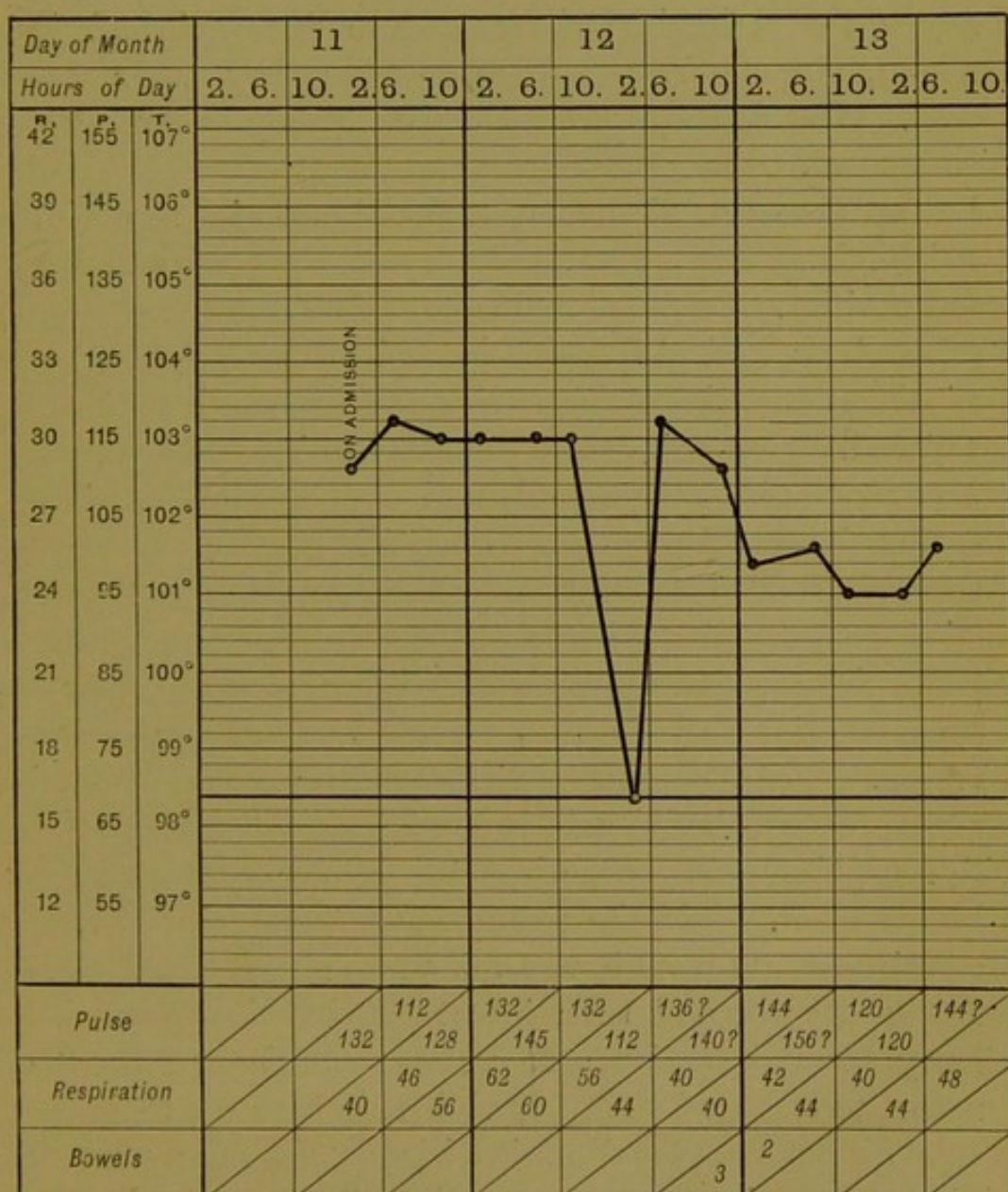
On the morning of the 12th Mr. Dunn saw the child with me again, and decided to operate. The abdomen was opened in the median line, and general peritonitis was found. There was a good deal of slightly turbid fluid in the peritoneal cavity, and the intestines were covered with flakes of pale greyish-yellow lymph. The fluid had no odour. The appendix vermiformis appeared to be a little swollen, and it was removed. A subsequent examination showed no ulceration, and the swelling was no more than could be accounted for by the peritonitis. It was certainly not the primary focus of the peritonitis. The peritoneal cavity was washed out, and a drainage-tube was left in. I examined some of the peritoneal fluid, staining some cover-glass preparations with Maconochie's capsule stain, and found a number of capsulated diplococci, which I considered were pneumococci. Cultures were also taken, but no growth resulted.

After putting the child back to bed the pulse became very feeble, and stimulants were administered. In the afternoon she had an attack of profuse diarrhœa, which was stopped with a starch and opium enema and bismuth. As the pulse did not improve saline enemata and infusions of saline solution into the axillæ were administered. During the night she became very restless, and continued so all the following morning. She died suddenly on the afternoon of the 13th, at 4.30 p.m.

I made the post-mortem examination twenty-one hours after death. Rigor mortis was well marked. The body was rather wasted and anæmic. There was general



acute pleurisy, both lungs being covered with light greyish-yellow, thin recent lymph. There was a small quantity of thin, slightly turbid serous fluid in both pleural cavities. There was no pneumonic consolidation of any part of either lung, and no evidence of any commencing pneumonia. The larynx, trachea, and



bronchi appeared to be normal. There was no pericarditis. The heart weighed 51 grammes; it was healthy. The arteries were normal. There was general acute peritonitis. The peritoneal blood-vessels were congested.



The coils of intestine were adhering to each other by means of pale greyish-yellow lymph. In the pelvis there was a little turbid serous fluid. There was no local lesion to account for the peritonitis. There was no ulceration of the stomach, duodenum, or small or large intestine. The condition of the appendix vermiformis has already been mentioned. There was no suppuration of any of the abdominal viscera. The liver weighed 184 grammes, and the spleen 54 grammes; they were both normal. The kidneys weighed 102 grammes, and were quite normal.

Cultures were taken from the blood in the right ventricle, from the spleen, and from the pleural and peritoneal cavities. Pneumococci were found in pure culture in the first three, and with staphylococci in the last. Microscopical preparations from these sources also showed capsulated diplococci. Mr. Pakes examined the cultures and cover-glass preparations, and confirmed the opinion that the diplococci were pneumococci.

*Remarks.*—The cause of death was pneumoccal septicæmia, for pneumococci were found in the heart, blood, spleen, pleural and peritoneal cavities. The question naturally arises as to the source and channel of the infection. There was no obvious primary lesion found in any part of the body, and the clinical and pathological evidence pointed to the peritoneum as the first structure to be attacked. Did the pneumococci first gain access to the peritoneum through the alimentary canal and cause acute peritonitis, and then infect the blood, or was it a primary blood infection, the peritoneum being the first structure to be attacked? I have brought forward this case as one of pneumococcal peritonitis, as all the symptoms pointed to the peritoneum as the structure to be first implicated, there being no indication of the lungs or pleura being involved; and the post-mortem evidence also corroborated this view, for the morbid changes were more marked and advanced in the peritoneum than in the pleura.

The case I have just recorded makes the third which I have had the opportunity of investigating during the last eighteen months. The appearance of the lymph, and the character of the fluid, and the clinical history in each of these cases reminded me very forcibly of several cases I had seen some years ago, in which no local lesion was found, and which were classified under the vague and unsatisfactory heading idiopathic peritonitis. I am of opinion that a large proportion, if not all, of these so-called cases of idiopathic peritonitis would have proved to have been due to pneumococcal infections had they been examined from a bacteriological point of view.



