Pneumococcus peritonitis : paper read in the Section of Pathology, at the Annual Meeting of the British Medical Association, Cheltenham, July-August, 1901 / by J.H. Bryant.

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PNEUMOCOCCUS PERITONITIS.

PAPER READ IN THE SECTION OF PATHOLOGY.

At the Annual Meeting of the British Medical Association, Cheltenham, July-August, 1901.

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DURING the last fourteen months I have had the opportunity of investigating 3 cases of pneumococcus peritonitis, and in this discussion on pathological conditions caused by the pneumococcus I shall confine myself to a few remarks on the pathology of this rare form of peritonitis.

Very few cases of this nature have been recorded in England; in fact I have only been able to find one at all fully worked out.

Fisher, in a letter published in the *Lancet* on November 10th, 1900, in reply to a short leading article on the subject mentions the case of girl, aged 8, who was admitted into the Bristol Infirmary in 1899 suffering from acute peritonitis of five days' duration. Abdominal section was performed, but no cause was found to account for the peritonitis. She died on the following day, and at the necropsy no lesion of the abdominal or pelvic viscera was found, in fact there was no septic focus discovered anywhere.

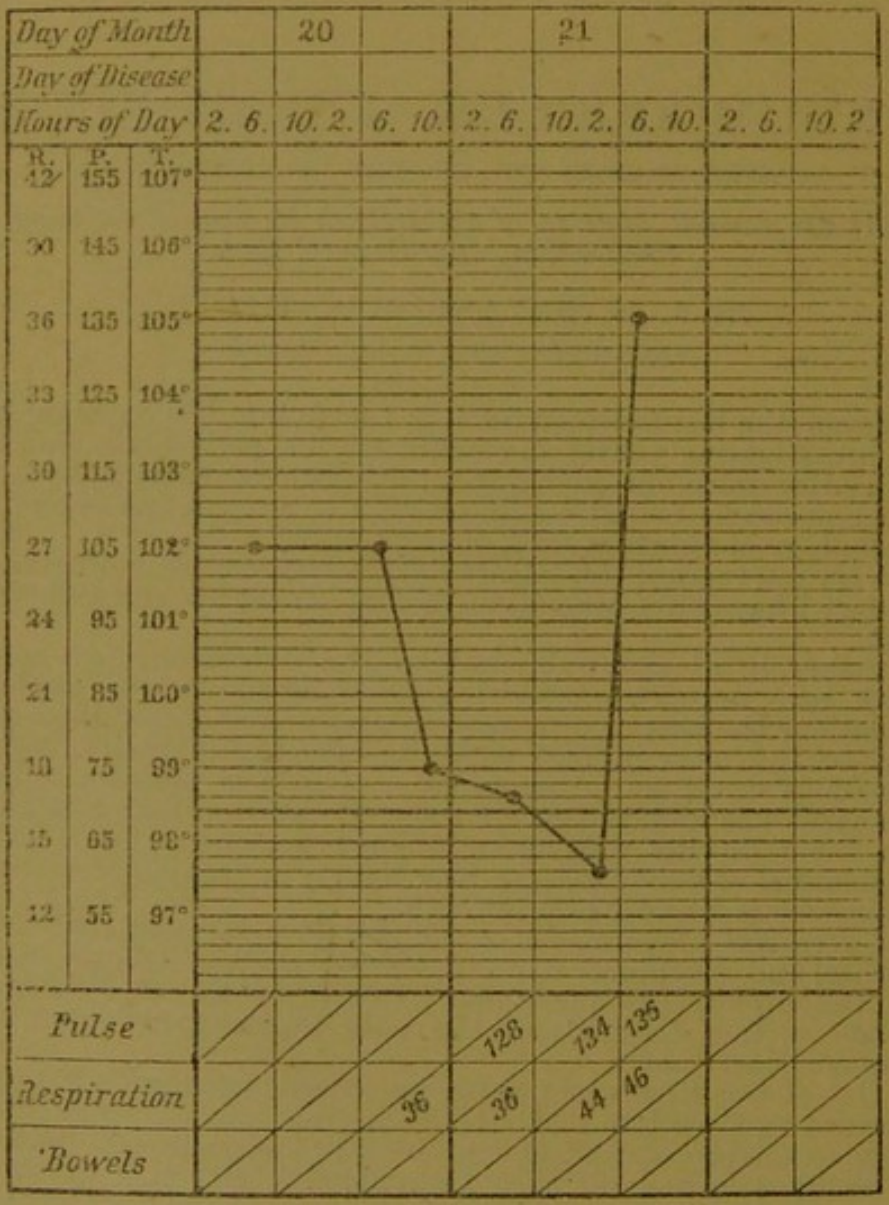
Coverglass preparations of the pus showed capsulated diplococci and streptococci, and cultures showed the presence of the diplococcus pneumoniæ. Most of the observations made on this disease and the cases reported have been by French observers.

One of the first cases of pneumococcus peritonitis was described by Cornil in 1886. In the same year Netter demonstrated the presence of pneumococci in the peritoneal fluid of patients who had died from pneumonia, although in these cases there was no evidence of any morbid changes in the peritoneum.

Since Cornil's case a number of examples of this form of peritonitis have been described by French authors.

Pneumococcus peritonitis is undoubtedly a rare condition, but I have no doubt in my own mind that if systematic histological and bacteriological examination of the pus from all cases of peritonitis were made, it would not be so rare as it appears to be at the present time.

Blackburn in his thesis mentions that Netter stated that of 151 pneumococcus infections noted by him there were only two in which the peritoneum was affected, and these two were in children. There is a form of peritonitis which has been, and is now, sometimes classed as idiopathic, a form in which no obvious primary lesion can be found to account for the inflammation of the peritoneum. I am strongly of opinion that these cases are nearly all, if not all, examples of pneumococcus infection. As far as peritonitis is concerned, the term "idiopathic" ought never to be applied to any case



unless most careful and exhaustive histological and bacteriological examinations have been carried out with negative results and inoculation experiments have failed to show the presence of a pathogenic organism. The following is an account of my cases. I will read them in the order in which they occur:—

CASE I.—Daisy S., aged 5 years, was admitted on May 20th, 1900, under the care of Dr. Newton Pitt for diarrhœa and vomiting. She had been a fairly healthy child but was subject to colds. When sixteen months of age she had an attack of whooping-cough, and when three years measles.

On Saturday, May 19th, she appeared to be perfectly well. The mother stated that she had eaten some raw rhubarb on this day. On Sunday morning, the 20th, about 8 A.M., she vomited freely, and about the same time suffered from diarrhœa. Both the vomiting and the diarrhœa continued to be profuse throughout the day, and she complained of "pain in her stomach." She was very fretful and only took a little milk and soda water. At 6 P.M. she was much worse, and at 7 P.M. she was admitted into the hospital.

Condition on Admission.—Temperature 102°, pulse 124, respirations 36. She was a well-nourished child, but was very fretful, and seemed to be wandering in her mind. She appeared to be in pain, and was very thirsty.

Respiratory System.—She had no cough. A pleuro-pericardial rub was heard over an area about 2 inches in diameter just below and external to the left nipple.

The Pulse was small and of low tension. The cardiac dulness was not increased. There were no *bruits*.

Alimentary System.—Lips dry; teeth good; tongue furred on the dorsum, but clean at the tip; papillæ red and prominent; fauces normal. The abdomen moved on respiration, and was quite supple. Palpation appeared to give her pain. The percussion note was normal.

Urine, acid, 1021. Small amount of albumen present. No blood, pus, or sugar.

The temperature came down to 99° until just before 6 P.M., when it suddenly rose to 105°. There was no diarrhœa after admission, but, even with the temperature down, she seemed ill, and was very fretful and thirsty. She vomited once during the afternoon for the first time since admission. At 5.45 P.M. she again vomited, and then went into a fit. She became quite pulseless, and remained collapsed for two hours in spite of the administration of oxygen and injections of brandy, strychnine, and ether, and a saline injection. She died at 7.45 P.M.

The *post-mortem* examination was made on May 21st, fifteen hours after death. Rigor mortis was well marked, and there was marked hypostasis of the back, neck, and gluteal region. The extremities were much cyanosed. The body was well nourished. There was no anasarca. There was no otitis and no meningitis. The spinal cord, cervical glands, and thyroid were healthy. There was a good deal of subpleural ecchymosis on the upper two-thirds of the right lower lobe of the lung, and this part of the pleura was also covered with very thin flakes of recent lymph. There was also recent lymph covering the base of the left lung. There was no pneumonic consolidation.

The larynx, trachea, bronchi, and bronchial glands were normal. There was a little recent lymph covering the outer surface of the pericardial sac. The heart weighed 75 grams. There was no endocarditis, and the cavities were not dilated. The arteries were healthy. The mouth, pharynx, œsophagus, and stomach were normal. There was general acute peritonitis. The peritoneal cavity contained about 10 ounces of thin greenish yellow, opaque purulent fluid. The liver, spleen, and intestines were covered with small flakes of pale yellow lymph. The appendix vermiformis was quite healthy. There was no local collection of pus which would have caused the peritonitis, and there was no perforation of any of the hollow viscera. The pelvic organs appeared to be quite healthy.

The liver, gall bladder, and pancreas were normal. The lymphatic glands were swollen and sodden. The spleen weighed 54 grams, and was normal. The suprarenals were healthy. The kidneys weighed 83 grams, and were quite normal.

Coverglass preparations were made from the peritoneal fluid, and stained with methylene blue and with capsule stain. An almost pure growth of capsulated diplococci was seen. A few large long thick rod-shaped bacilli were seen also.

CASE II.—Thomas S, aged 45, a stone carver, was admitted on October 9th 1900, under my care for abdominal pain. He had always enjoyed good health, with the exception of an abscess on his knee, which he had when he was a boy. On October 1st he first noticed a pain in his epigastric region, which was accompanied by flatulence. From October 1st to October 3rd he suffered from constipation. He was able to work on the 1st, but on the 2nd had to give it up, and he went to bed until the 4th. He attempted to work again on the 4th, but soon had to give it up. On the 5th his motions were scanty and fluid. He remained in bed on the 7th, feeling very ill and suffering considerable pain, which was continuous, with occasional exacerbations which made him cry out. He was admitted on the 9th.

Condition on Admission.—Temperature 101.2°, pulse 100, respirations 36. He was very pale, and his eyes were much sunken. He complained of extreme tenderness all over his abdomen, but the muscles were not rigid. I saw him soon after admission, and diagnosed general suppurative peritonitis. I called in my colleague Mr. Dunn, who decided to operate at once.

On opening the peritoneal cavity about a pint of thick green fluid was evacuated, and it was found that the peritoneum was covered with flakes of recent greenish-yellow lymph.

On the following day he felt much better. He was taking peptonised milk well.

On the 11th he became very restless, and endeavoured to get out of bed. He gradually became worse, and died at 5 A.M. on the 11th.

The *post-mortem* examination was made ten hours after death. The body was rather emaciated. There was a vertical incision in the median line of the abdomen about 10 cm. in length, 5 cm. of which was above and 5 below the umbilicus. The brain and meninges were normal. The posterior surface of the right lung was covered with a thin layer of quite recent lymph. There was very marked subpleural ecchymosis over the posterior surface of this lobe. The lungs were engorged with blood, but there was no pneumonic consolidation. The larynx, trachea, bronchi, and bronchial glands were normal. There was no pericarditis. The heart weighed 302 grams. All the valves were competent, and there were no signs of recent or of old endocarditis. The right ventricle was very much hypertrophied, and the myocardium was firmly contracted. The left ventricle was not hypertrophied. The first part of the aorta was very atheromatous, and the coronary arteries were also thickened and atheromatous. There was a good deal of *post-mortem* digestion of the mucous membrane of the œsophagus. The cardiac end of the œsophagus was much ecchymosed. There was no ulceration. There was neither ulceration nor perforation of any part of the intestinal tract. The appendix vermiformis was quite healthy. There was general acute suppurative peritonitis. The peritoneal vessels were much injected, and all the viscera covered with peritoneum were clothed with thick layers and patches of greenish-yellow lymph. No local cause for the peritonitis could be found.

The liver weighed 1,367 grams. There was a good deal of subscapular ecchymosis. The organ was very pale from fatty degeneration and infiltration. The gall bladder and pancreas were normal. The mesenteric glands were enlarged. The spleen weighed 106 grams. The suprarenal capsules were normal. The kidneys weighed 250 grams; they were healthy.

The following report of the bacteriological examination was made by Mr. Pakes:

"Pneumococci and streptococci were present, the former as well as the latter being found in the cutures from the heart blood of a mouse inoculated with the original culture of the peritoneal fluid."

CASE III.*—Harriet S., aged 4, was admitted into Guy's Hospital under my care on May 11th, 1901, for vomiting and abdominal pain. She had always enjoyed good health until Thursday evening, May 9th, when she was seized with severe vomiting after taking her supper. She vomited about six times during the night. The vomiting 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 to the hospital and was at once admitted. She had not vomited since Thursday evening. The bowels were opened on Friday, and the motion appeared to be natural. There was no discharge of blood or mucus from the rectum.

On admission the pulse was 140, the temperature 102.8°, and respirations 40. 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 pneumococcus 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 the condition was due to diaphragmatic pleurisy or pneumonia.

During the night the child 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; it was with great difficulty that she could retain any food.

On Sunday morning, 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 was removed.

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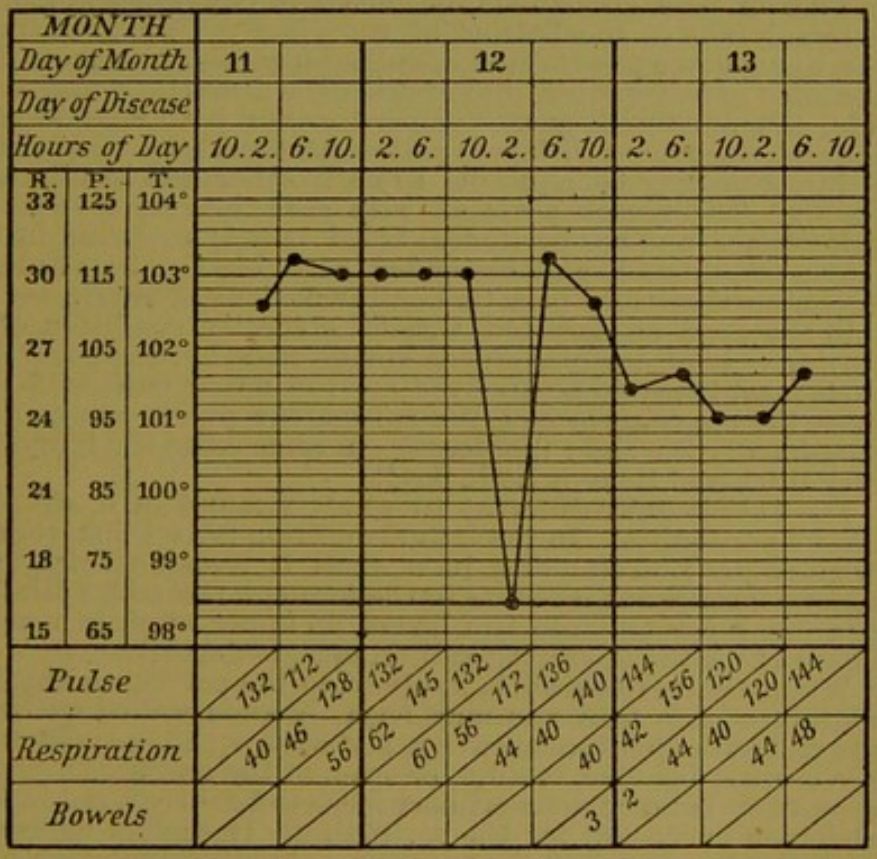
* This case was read before the Society for the Study of Disease in Children.

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 coverglass 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 Monday afternoon at 4.30 P.M.

I made the *post-mortem* examination 21 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 grams. 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, 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 840 grams and the spleen 54 grams; they were both normal. The kidneys weighed 102 grams and were quite normal.

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

Remarks.

Pneumococcus peritonitis may be classified in two ways:

1. Primary, when the peritoneum is the first structure to be involved.

2. Secondary, when the peritoneum is infected as a complication of some other pneumococcus lesion.

Or according to the distribution: (*a*) Local or encysted; (*b*) general or diffuse.

Morbid Changes.

In the general or diffuse form the abdomen is distended, partly from the presence of fluid in the peritoneal cavity and partly from tympanitic distension of the intestines. On opening the peritoneal cavity a fair amount of turbid, light greyish-yellow fluid may be found. If this is collected and allowed to stand, the upper portion becomes much clearer, and a thin pale, greyish-yellow deposit of pus cells and flakes of lymph falls to the bottom of the glass. Coverglass preparations of the pus stained with Maconochie's stain show the presence of capsulated diplococci. The intestines are, as a rule, distended with gas. The peritoneum, both visceral and parietal, is covered with pale yellow flakes of lymph and coils of intestine may be adhering to each other and to adjacent viscera. The lymph peels off with ease, leaving a slightly dull surface, and the peritoneal blood vessels are found to be congested, dilated, and distended with blood. In virulent infections there may be actual extravasations of blood.

In the local or encysted variety a similar condition of a portion only of the peritoneal cavity may be seen, and the parts most likely to be involved are the upper surface of the liver, the upper surface of the spleen, the pelvic cavity, and occasionally the right iliac fossa. In some cases the lymph is very thick and there is little fluid.

The fluid is quite different to what is found in a streptococcus peritonitis. In the latter condition it is thick yellow or green pus; on standing in a glass it remains thick and opaque throughout, whereas in the former it resembles diluted pus and separates into two layers, an upper, slightly opaque liquid and a thick, greyish-yellow, opaque deposit.

When the peritonitis is localised to the pelvis, an abscess containing this thin pus may form, become walled off from the rest of the peritoneal cavity, and open spontaneously into the vagina, bladder, rectum, or even at the umbilicus. As a rule, there is no obvious primary focus to be discovered in the abdomen to account for the peritonitis. In each of my cases pleurisy was found at the necropsy; it varied in degree and extent; in some cases it appeared to be secondary to the peritonitis, whereas in others it looked as if it was a part of a general infection, and had the appearance of having been affected at the same time as the peritoneum. In none of my cases was the pericardium affected, and in none of them was there any sign or evidence of pneumonia or broncho-pneumonia.

Pericarditis, endocarditis, meningitis, pneumonia, broncho-pneumonia may in some cases be associated with the peritonitis.

Pneumococcus peritonitis rarely occurs in adults. One of my cases was a man aged 45. Dieulafoy found only 2 cases in adults out of 17. Ménétrier and Legroux report 2 cases, one a man aged 32, and the other a man aged 35. Témine, in his thesis published in 1898, reports only 3 cases occurring in the adult.

Cassaet draws attention to the fact that in adults men are more liable to be affected than women. In children, on the contrary, girls are much more frequently attacked. Two of my cases were girls aged 4 and 5 respectively.

Quéhart, in his thesis, collected 33 cases, of which 27 were girls and 6 boys. Of these, 15 were between the ages 2 and 5; 12 were between the ages 5 and 10; 6 were between the ages 10 and 15.

Brun also draws attention to the fact that girls are more frequently affected than boys, and mentions 11 out of 14. Hagenbach-Burckhardt reports 2 cases, both of which were girls, aged 2 and 6 respectively. Audion also reports 2 cases, girls aged 3 and 2½ respectively.

There appears to be no very obvious reason why the peritoneum should be attacked by the pneumococcus. The disease usually begins suddenly in children who are apparently quite well. In 2 of Quéhant's cases there was evidence of abdominal injury, 3 were secondary to pneumonia, and 1 to sore throat. In the remaining 25 it was apparently primary, there being no obvious cause or source of infection.

In 1 of Audion's cases the peritonitis followed double broncho-pneumonia and otitis. Broca has published 2 cases which followed attacks of appendicitis; Galliard, 1 case secondary to salpingitis; Guido Banti and Weichselbaum, cases associated with the presence of abdominal neoplasms.

No apparent cause was found in any of my cases; the first indications of the disease in all 3 were abdominal pain and vomiting. In Case III there was a history of the child having eaten a quantity of raw rhubarb, which may have started the gastro-intestinal symptoms. In a good many cases there is a history of a chill.

The Source and Channel of Infection.

Goldenberg, Weichselbaum, Wolf, and others have demonstrated the important fact that pneumococci can frequently be found in the mouths of perfectly healthy people. Bezançon and Griffon have also reported the result of their investigations on the throats of healthy persons. They used the serum of a young rabbit for their culture medium, and found by making bacteriological examinations of the secretion from the tonsils of forty healthy persons, under various conditions, that pneumococci were present in every case.

There are several possible channels of infection, namely:

1. The alimentary canal.
2. The uterine organs.
3. The blood vessels.
4. The lymphatics.
5. The skin.

I have already alluded to the fact that pneumococci are frequently found in the mouths and throats of healthy people, and therefore must be constantly swallowed with the saliva and food, thus getting into the alimentary canal as easily and probably as frequently as into the respiratory tract. A possible explanation of the fact that the peritoneum is so rarely affected by this organism is that the normal digestive fluids are inimical to the growth and development of the pneumococcus. If, however, there is severe gastro-intestinal disturbance caused by the ingestion of some unsuitable and indigestible food, which may have caused gastro-enteritis leading to severe vomiting and diarrhœa, there will be not

only a general lowering of the resistance of the body but also a local lowering of resistance in the stomach and intestine and also an altered condition of the gastro-intestinal secretions. Such a series of changes might well predispose to the growth and development of the pneumococcus in the gastric and intestinal mucosa, and might also allow of the passage of the organisms by means of the blood vessels and lymphatics to the peritoneum itself, and in this way a peritonitis might be brought about. That the pneumococcus does actually attack the mucosa is proved by the case of pneumococcus enteritis published by Boulay.

The question may be asked why the pleura is so much more frequently affected than the peritoneum? The answer to this, I take it, is that by far the most frequent manifestation of a pneumococcus infection is pneumonia, and the close relation of the pleura to the lung is the explanation of it so frequently being involved.

2. *The Uterine Organs.*

The very large proportion of girls to boys (Quéhant, 27 girls to 6 boys; Brun 11 girls, 3 boys; Hagenbach Burckhardt, 2 girls; Bryant, 2 girls; Fisher, 1 girl) affected by this form of peritonitis is very suggestive that the uterus and Fallopian tubes are the channels by means of which the pneumococcus gains access to the peritoneal cavity. It is not easy to explain, however, why it is that when adults are attacked they are nearly always men.

Boulay has noted the presence of the diplococcus pneumoniæ in the cavity, and in the walls of the uterus, an important observation which indicates that the micro-organism may easily reach the peritoneum through the Fallopian tubes or the lymphatics or blood vessels of the uterus.

Another point which favours the view that this is often the source and channel of infection is the fact that the pelvic peritoneum is much more extensively involved than the rest of the peritoneum, even when the peritonitis is diffuse and that when the peritonitis happens to be local or encysted the pelvic peritoneum is frequently the part which is affected.

It has been well established that peritonitis may result from gonococcus infections of the uterus and Fallopian tubes and in cases of tuberculous peritonitis, even in young children it is not unusual to find advanced tuberculous disease of the uterus and Fallopian tubes, and from their appearance it would seem that they were the first parts to be affected, and were the primary focus of the disease. Peritonitis also frequently follows septic conditions of the uterus, the result of parturition or abortion. These examples of primary disease of the uterine organs illustrate their importance as sources and channels of infection.

There were no obvious changes in the uterus and Fallopian tubes in my two cases, and, unfortunately, no bacteriological examinations were made, so that there is no conclusive evidence to show what part these organs played in the production of the peritonitis.

3. *The Blood Vessels.*

There is no doubt that many cases die as a result of a general pneumococcus septicæmia, and then the question naturally arises in such cases whether there was a general blood infection from the first, with a primary peritoneal manifestation and subsequent morbid changes in the pleura, pericardium, and

other structures; or whether, as a result of a general blood infection, all these structures were attacked at or about the same time.

Case I appears to have been an example of a general blood infection, and the symptoms point to the peritoneum as being attacked quite early in the disease, for the symptoms were:—A sudden onset, with vomiting, diarrhœa, and abdominal pain. On admission a pleuro-pericardial rub was heard, and it would appear from this, that the pleura most probably was involved at the same time, or at least very soon after the peritoneum. In Case II the clinical evidence points to primary infection of the peritoneum, with a secondary implication of the pleura, which may have been due to a general blood infection, or to a local infection through the lymphatics.

Case III died from pneumococcus septicæmia, pneumococci having been demonstrated in the heart, blood, spleen, peritoneum, and pleura. The clinical and pathological evidence pointed to the peritoneum as the first structure to be involved, the pleura probably being soon after infected. Whether it was a general blood infection from the first, or whether the peritoneum was first infected and then the blood, there is no positive proof. I consider, however, that both the clinical and pathological evidence is in favour of a primary infection of the peritoneum, probably through the alimentary canal, with a subsequent general blood infection leading to general acute inflammation of the pleura.

4. *The Lymphatics.*

Very frequently when making *post-mortem* examinations it is found that the upper surface of the liver is firmly attached to the under surface of the diaphragm by means of fine fibrous adhesions, and that the base of the right lung is also fixed to the upper surface of the diaphragm in a similar manner; on the left side the spleen and base of the left lung may be similarly attached to the diaphragm. In many of these cases a definite history of pneumonia or pleurisy may be traced, and I am of opinion that the majority are examples of cured local pneumococcus peritonitis, the primary lesion being pneumonia or pleurisy, and the peritonitis being caused by a secondary infection through the lymphatics.

With regard to the lymphatics as a channel of infection, a few months ago I made a *post-mortem* examination on a case of right pleuro-pneumonia, and found the right lung adherent to the adjacent part of the pericardial sac by means of recent lymph. There was commencing peritonitis, but only the outer two-thirds of the pericardium, covering the right ventricle, was involved; the most advanced changes were noticed on the outer part of the right ventricle, which was covered with small flakes of lymph; then internal to this came an area of pericardium, dull and granular in appearance; then a zone showing increased vascularity, and finally the pericardium covering the outer third of the right ventricle and the whole of the left ventricle, was smooth and shiny, and appeared to be perfectly healthy.

I do not consider that in any of the three cases I have recorded the lymphatics were the primary channels of infection. In Case I the symptoms point to a general blood infection. In Case II the pleura without much doubt was infected at a subsequent date to the peritoneum, and it is possible that the pneumococci were conveyed through the lymphatics

of the diaphragm, as the bases of the lungs were the parts most involved.

To sum up, then, it appears that when the peritoneum is infected through the lymphatics it is nearly always secondary to pneumonia or pleurisy, and the part of the peritoneum most involved is that covering the upper part of the liver and spleen. In some of the cases of local pelvic peritonitis also the organisms may be conveyed from the uterine cavity to the peritoneum, by the lymphatics, and not by the Fallopian tubes; in fact I consider that when the infection is carried by means of the lymphatics, the peritonitis is much more likely to be local than general.

5. *The Skin.*

The skin is another possible channel by means of which the pneumococcus can reach the peritoneum; but there have been no cases, as far as I know, published which point to this manner of infection. An umbilical fistula, abscess, or other lesion of this part of the abdominal wall, or old adhesions binding the intestines to the parietal peritoneum lining the anterior abdominal wall, might predispose to such a mode of infection.

Taking into consideration the symptoms, the physical signs, and the pathological changes found in my cases and in those reported by others, I am inclined to the view that in primary diffuse pneumococcus peritonitis, the pneumococcus reaches the peritoneum either through the alimentary canal, or uterine organs. I consider that undoubtedly some of the cases are manifestations of a general pneumococcus blood infection, and that others start as a peritonitis, and die from a subsequent pneumococcus septicæmia.

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