(1) exploratory, to see if any adhesion of the stomach exists that may be freed; (2) a better closure of the abdominal wall, by close separate suture of the aponeurosis. Since my attention was called to this important detail by my colleague Dr. Grimsdale, during one of my operations in 1901, I have invariably practised it when closing openings in the linea alba, with the effect so far of preventing subsequent hernia. No. 5 in the first series is a case in point, in which no trace of hernia is to be found.

Case iv is that of a man, aged 37, who had been under the care of Dr. McCallum, of Kendal, for chronic indigestion which had lasted several years. He was admitted into the Kendal Hospital under the care of my brother, Dr. William R. Parker, who treated him by careful dieting and washing out the stomach for three weeks, and then sent him to me. The patient was very emaciated, and, not to keep him waiting, I operated the day after admission, on June 7th, 1902. On making an exploration through a median epigastric incision I found a state of affairs almost exactly like that found in Case iv of the series in Table I, but devoid of perforation into the peritoneum. An induration was found in the pyloric region, and cicatricial bands on the front of that region, indicating the healing of severe inflammatory change. There was no actual pylorus, the stomach being deformed and shrunk at that site, owing to contraction of ulcerative change. The stomach was opened and cut in two, the divided ends being reattached after removing a large adherent patch where chronic perforation, with adhesion behind, had occurred. Particular care was taken to use strong silk for the purpose, and two rows of sutures were put into the peritoneal surfaces, apposed, in addition to a previous row along the mucous edges. All through the operation I had a dread that the stitches might give way, and some of them actually did as I went on, and for that reason I put in an extra row all round, and in some places more still.

The patient did very well for two days, but throughout was subject to almost incessant biliary vomiting. It then became evident that he was not doing well; he became cold and collapsed, and died five days after operation, with symptoms, in addition, of abdominal tenderness, rapid further emaciation indicating probable peritonitis and septic poisoning.

A necropsy was made, extensive purulent peritonitis found, and a wide separation in the front of the attached surface, showing that here too the stitches had given way.

It is now pretty clear to me that this patient was probably not altogether fit to undergo the operation, owing to the previous exhaustion by starvation and pain. But for all that his progress for two days was satisfactory in all but the distressing vomit, which must have seriously tried the stitches. I am inclined to attribute the disaster mainly to the persistent vomiting, which, added to his previous weakness and malnutrition, prevented him from profiting by the operation. The same also seems to apply to Case ii of this series.

In similar cases where at the time I deemed the patients unfit to submit to any extensive operation, I have performed gastro-enterostomy with success. But even not always here. For instance, on December 12th, 1901, I operated in exactly the same manner on two men who had both suffered for a prolonged period from dyspepsia, vomiting, and other symptoms pointing to dilated stomach. In both cases I explored, and finding outside the stomach cicatricial evidences of internal ulceration and great attempts by Nature at spontaneous cure of the malady, I performed pyloroplasty, as the pyloric end of the stomach was narrowed, and hampered by the close proximity of a chronic ulcer, which did not seem to me capable of safe removal by excision. This did not take long, but in each case I also did gastro-enterostomy to relieve the pylorus and its neighbouring ulcer by a new and additional way out for the contents of the stomach.

One of the men, aged 43, somewhat feeble, died in three days of mere exhaustion apparently, while the other, aged 51, but more robust, made a good recovery, and reported himself in good health on February 26th, 1902, having started to work three days before. He also wrote on July 9th, and again on October 25th, 1902, saying that he could eat almost anything without any discomfort, and follow his employment.

In Table III I give these two and five other pyloroplasties, without further comment on the first five than the remarks in the table.

TABLE III.

No.	Date.	Name and Sex.	Remarks.
1	Aug. 14th, 1895	E. S. (F.) 38	Healed well. Return of symptoms 1899.
2	July 3rd, 1899	42	Pylorus explored; adhesions separated; pyloroplasty repeated. Healed well and quickly. January, 1900, wrote saying occasional indigestion and constipation. July 19th, 1902, wrote saying quite well; no stomach trouble; forgets what indigestion is like.
3	May 5th, 1897	J. D. (M.), 52	Ulcerated induration in stomach found. Suspected then to be carcinoma and not interfered with. November, 1897, called complaining of acid eructations. March 26th, 1900, wrote complaining that he was troubled more and more with his old trouble (dyspepsia and flatulence). June 18th, 1900, wrote saying that he was in fairly good health. This indurated ulcer was probably a gumma.
4	Jan. 23rd, 1899	G. L. (M), 21	Small unhealed ulcer found in stomach near pylorus; not interfered with. Reported quite well October, 1902.
5	Sept. 20th, 1899	H. B. (M), 46	An extraordinary case of superficial ulceration of mucous membrane over the right half of the stomach. Had gained weight two months later. A year later succumbed after gastro-enterostomy. Patient of Dr. Wilding, Hindley, Wigan.
6	Dec. 12th, 1901	T. H. (M), 43	Pyloroplasty and gastro-enterostomy. Died after 3 days; no repair.
7	Dec. 12th, 1901	R. L. (M), 51	Pyloroplasty and gastro-enterostomy. Made good recovery and was in excellent health July 9th and October 25th, 1902.

The Croonian Lectures

ON THE

NATURAL HISTORY AND PATHOLOGY OF PNEUMONIA.

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LECTURE III.—[ABSTRACT.] AGGLUTINATION.

ARKHAROW, Issaef, and Mosny found that pneumococci grew in a special manner in the serum of immunized animals. Early in 1895 I observed that the pneumococcus, in the form of diplococci, gave rise to a uniform turbidity in normal rabbit's serum; in immunized rabbit's serum, however, the cocci sank to the bottom in the form of a sediment, and the serum remained clear; these cocci were clumped together in masses, and sometimes formed very long chains, and I then stated that the mode of growth was a good indication of the protective power of the serum.

Eyre and I made a number of observations upon the agglutinative power of various serums, using different dilutions and testing both virulent and avirulent forms of pneumococci. We found that the quantity of agglutinin formed in the blood during the process of immunization was exceedingly small; for example, normal rabbit serum would cause agglutination in a strength of 25 per cent. at the end of twenty-four hours, while immune serum only agglutinated in the strength of 12.5 per cent.

We failed to find any definite relationship between the agglutinative power and the protective action of the various serums. Pane likewise found that the highest limit for agglutination were the following dilutions of the serum of an immunized rabbit, 1 in 15; of an immunized cow, 1 in 25; and of an immunized donkey, 1 in 30; while in order of protective power the donkey's serum came first, the rabbit's second, and the cow's last. Bezancon and Griffon¹ made observations upon the agglutinating power of the serum with the object of determining whether it could be used for diagnostic purposes, and the method which they adopted was to sow the

pneumococci directly into the serum and to examine it after growth had taken place. They found that the injection of rabbits with an attenuated pneumococcus was only sufficient to produce illness and not death. The serum during acute illness possessed agglutinating powers, which, however, soon disappeared after recovery. The most powerful serum only agglutinated when used undiluted, or diluted with broth to a not greater extent than 1 in 50. They also examined 39 cases of primary lobar pneumonia and 6 cases of secondary pneumonia. In every case agglutination was observed during some period or other of the attack, appearing for the first time, as a rule, just before the crisis, and persisting as long as five weeks after the crisis, although it sometimes disappeared in a week. They also found that the serum had different agglutinative powers with regard to different strains of pneumococci. They always found that it agglutinated best the pneumococci obtained from the mouths of the individuals from whom the serum was derived; indeed, in some cases it would only agglutinate these pneumococci, and from this they inferred that the pneumococcus infection always occurred from the pneumococci already in the mouth.

EFFECT OF SERUM UPON PNEUMOCOCCUS TOXINS.

Issaef states that immunized animals are more affected by toxins than non-immunized animals. He also states that the injection of serum together with the toxin is just as powerful in its effects as the injection of the toxin alone. Mennes, on the other hand, quotes experiments to show that the serum possesses antitoxic properties. These discordant results are doubtless due to the fact that the toxins that have hitherto been produced are so feeble that it is difficult to make satisfactory experiments with them.

BACTERICIDAL ACTION OF VARIOUS SERUMS.

In 1890 Baring and Nissen showed that the blood serum of a normal rabbit possessed no bactericidal action, the method which they adopted being that of plate cultivations. They found that the serum of a normal guinea-pig as a rule had no bactericidal property, but the serum of one guinea-pig which they examined possessed marked bactericidal properties. Kruse and Pansini in 1892 repeated these experiments, and obtained similar results. The serum of a sheep has marked bactericidal properties, and so too has the serum of immunized rabbits. Eyre and I made a number of observations upon this subject,² using normal rabbit serum, normal fowl serum, normal guinea-pig serum, the serum of an immunized rabbit, and the serum of an immunized pony. We obtained exactly similar results to the other observers in reference to the normal rabbit serum and to the normal guinea-pig serum; the normal fowl serum never possessed any bactericidal properties; the serum of the immunized rabbit and of the immunized pony in some cases possessed definite bactericidal properties, and in other cases possessed none.

Pane found that the serum of an immunized animal acted most powerfully when it was mixed with a cultivation and the mixture injected. The serum of an immunized cow and of an immunized donkey, on the other hand, acted more powerfully when the serum was injected directly into the circulation and the cultivation injected under the skin. His explanation is that the immunity produced by the serum was an active immunity, and that the rabbit serum produced its effect more rapidly than the serum of other animals. It is quite possible, however, that the difference may be due to the presence of a specific additment in the rabbit serum which is absent in other serums.

Mennes³ found that the serum of immunized rabbits by itself exercised no bactericidal influence. If, however, to this serum there were added normal white blood corpuscles or the white blood corpuscles of an immunized animal the growth was hindered. He also found that when leucocytes were added to normal serum to which pneumococci were added, they did not englobe the cocci, but if in place of normal serum immune serum were used, the leucocytes were capable of englobing the cocci; the immune serum so acted upon the cocci as to render them sensitive to the action of the leucocytes.

METHOD OF RECOGNIZING PNEUMOCOCCI IN LESIONS.

The microscopical examination of sputum is not satisfactory

for determining the presence of pneumonia; Gamaleia states that out of a number of observations the two cases in which the largest number of pneumococci were found in the sputum were not cases of pneumonia. Some years ago I made a number of observations with reference to this point, and I found that it was exceedingly difficult to detect pneumonia by a microscopical examination of the sputum. Lindsay (M.D. Thesis, Cambridge) has recently made an extensive series of observations in this connexion with similar results. The method of inoculation appears to be the only certain method of finding the cocci, but in dealing with sputum we have the difficulty that pneumococci are frequently normally present in the mouth. Honl⁴ states that by injecting pneumococcus sputum under the skin of the ear of a rabbit, a marked oedema occurs in twenty-four hours; the swelling consists of leucocytes, fibrin, and cocci. The sputum from other diseases does not produce the same effect, inasmuch as the mouth pneumococci are either present in too small numbers or are not sufficiently virulent. Gamaleia examined by the method of inoculation the lungs in 12 cases of pneumonia, and in each case demonstrated the presence of the pneumococcus.

PNEUMOCOCCI IN NATURE.

Netter⁵ found pneumococci in the dust from hospital wards by injecting guinea-pigs; in two animals pneumococci were found after death. Emmerich also isolated pneumococci from the dust of a ward, and Uffelmann in the air of a cellar. Maximovitch⁶ found pneumococci in the dust of a hospital. Eyre and I, by means of inoculated experiments of dust obtained from various sources into mice, demonstrated the presence of the pneumococcus in dust obtained from a hospital ward and also from a laboratory cupboard, but obtained negative results with specimens of dust from the street, entrance hall of another hospital, and some of its wards.

Pneumococci were frequently found in the mouths of healthy individuals; Netter found them in 15 per cent. of the saliva of healthy persons, Sternberg in 20 per cent. Eyre and I examined the apparently normal saliva from 13 perfectly healthy individuals, and from seven hospital patients suffering from such diseases as occupation paralysis, chlorosis, etc., 20 persons in all; by injecting the saliva subcutaneously into the rabbit we isolated the pneumococcus from the saliva of 6 of these, that is, 30 per cent. Netter⁷ and Kurth found it in the nose of healthy individuals. Gasperini found it in 8 out of 10 healthy conjunctival sacs.

Of still greater importance is the presence of pneumococci in the lungs of healthy individuals. Dürck⁸ examined the lungs of 13 children who had died from some other disease; he found pneumococci 12 times, in most cases mixed with other bacteria; in the thirteenth case other bacteria were found, but not the pneumococcus. He also examined the lungs of dogs and horses, and out of 15 cases he found pathogenic bacteria, only 1 being sterile; among these bacteria were pneumococci. Boni⁹ found that the lungs of guinea-pigs were almost always sterile, but in certain cases pneumococci were present. Dogs almost always have bacteria in their lungs; in 20 cases only 6 were sterile, the remaining 14 contained bacteria, and of these, 5 contained pneumococci.

OTHER LOCALIZATIONS OF THE PNEUMOCOCCUS.

Pneumococci are found not only in the affected lung, but also frequently in other parts of the body. Nazari¹⁰ found cocci in the blood during life in 4 out of 17 cases; he also examined the spleen, but only found pneumococci present there when they were present also in the blood. F. W. White, of Boston, examined 19 cases of pneumonia during life: in 3 of these pneumococci were present and they were all fatal, and 7 other cases proved fatal although no pneumococci were found. Cohn¹¹ examined 32 cases: in 9 of these cases pneumococci were found, 7 of which were fatal: in the remaining 23 no pneumococci were found, and of these 5 were fatal. Banti¹² says that cocci are frequently present in the blood in pneumonia, and indeed believes that they are probably always present. Pernice and Alessi¹³ found pneumococci in the blood and various organs in 2 cases of fatal pneumonia. We can thus readily understand how it is that with pneumonia metastatic lesions are frequently found. Again, the pneumococcus may actually enter through the lungs and produce metastatic lesions without leaving behind any trace

of inflammation in the lung itself. This we know experimentally, as the following experiment performed by Eyre and myself shows: a rabbit was anaesthetised and a catheter passed through the larynx into the trachea. A cubic centimetre of a broth cultivation of the pneumococcus was poured down the catheter. The animal was killed fifteen days later: there was marked pleurisy and pericarditis, but the lungs were quite normal. The pneumococcus may also enter the body in other ways than through the lung, consequently various lesions are caused by it and not preceded by pneumonia, such as septicaemia, pleurisy, endocarditis, arthritis, meningitis and cerebritis, nephritis, parotitis, peritonitis, pericarditis, colitis and gastritis, cellulitis, osteomyelitis, acute otitis media, and conjunctivitis.

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MEMORANDA:

MEDICAL, SURGICAL, OBSTETRICAL, THERAPEUTICAL, PATHOLOGICAL, Etc.

TWO CASES OF CONSUMPTION PROBABLY INFECTED BY TUBERCULOUS MILK.

IN the course of my investigation of cases of consumption in connexion with the voluntary notification of phthisis in Manchester, the following history has been recorded which strongly points to infection by means of tuberculous milk.

The patient, E. S., male, aged 17, was visited on November 1st, 1902. The illness commenced in December, 1901, with loss of appetite especially in the morning. He had no cough then, but began to lose flesh very slightly. This continued up to the third week in May, 1902, when an ischio-rectal abscess developed which was opened. After this, the patient began to lose flesh rapidly and has continued to do so up to the present. The abscess discharged for about three months and left a fistula which was operated on about seven weeks before the date of my visit. One week after the operation as the patient was drinking a large quantity of liquids, diabetes was suspected, the urine was examined and found to be loaded with sugar. He had been drinking a large amount of water at his work for some weeks previously, but this had not been observed at home, so that the diabetes had certainly not been present for more than three months. There was no cough before the operation on the fistula; the patient and his mother are both certain on this point. After the operation, cough developed rapidly, and the sputum now contains tubercle bacilli. There is no family history of consumption, with the exception that the patient's sister, aged 5 years, died of tuberculosis on July 2nd, 1901. This child was removed from home to another address on April 18th, 1901, where she remained until her decease, and where E. S. also stayed for about two months at the same time. For some weeks before her removal the girl had suffered from loss of appetite and some vague symptoms of discomfort in the abdomen, but she had no cough then; in fact, she never developed a cough. On May 26th she complained of epigastric pain, and vomited. The abdomen was hard and tender, the tongue furred, and the pulse small and quick. Appendicitis was diagnosed, and an abscess ultimately formed which began to discharge through the umbilicus. On June 26th, 1901, an operation was performed, and an abscess evacuated. After the operation, from the condition then found, the conclusion was come to that the disease was tuberculous, and the mother was informed of this fact at the time.

Neither of these two patients drank much milk, probably not more than 3 oz. each daily; but the milk supplied to this family has been almost continuously tuberculous for over two years, and at one period especially (March, 1901) was very markedly so. Specimens of the milk which the family was using were taken on September 5th, 1900, and found by Professor Delépine to cause tuberculosis in the guinea pig; from

a fresh milk supply on March 4th, 1901 (two cows with tuberculous udders found by Mr. Lloyd, Veterinary Surgeon to the Manchester Corporation), and again from the first milk supply on January 18th, 1902 (one cow with tuberculous udder found by Mr. Brittlebank, M.R.C.V.S., Veterinary Surgeon to the Manchester Corporation). When the address of the two patients was changed in the end of April, 1901, the new milk supply was again tuberculous, for on July 11th, 1901, the farm from which the milk was obtained was visited by Mr. Brittlebank, and a well-marked case of tuberculosis of the udder found. The milk from this cow was also found by Professor Delépine to cause tuberculosis in the guinea-pig. The cow in question was slaughtered, and the udder submitted to examination by Professor Delépine, who reported the lesions in the udder to be most extensive, the mucous membrane of the acini being very markedly tuberculous. In the opinion of Mr. Brittlebank the udder must have been affected with tuberculosis for at least three or four months.

The disease in both these cases began in the abdomen, and in the case of the girl never reached the lungs, while in the case of the boy the lungs were only affected secondarily. No source of infection apart from milk could be ascertained in either case.

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A CASE OF HAEMORRHAGIC NEPHRITIS.

THE following notes may prove of interest. I publish them in the hope that some one may offer an explanation of what seems to me an obscure case:

K. A., aged 32, multipara, single, was transferred at term from the Birkenhead Workhouse to the Infirmary on May 11th, 1902, and was there delivered of a male child. I was called to her after the expulsion of the placenta, as the nurses had some trouble with the membranes. These, however, I removed without difficulty. I then noticed that in front of the well-contracted uterus, but unconnected with it, was a second tumour of similar size and consistency—obviously, from its position, the bladder. I passed a soft male catheter and drew off several ounces of what was apparently almost pure blood. The anterior tumour on this decreased somewhat, but remained still distinctly palpable, being about three fingerbreadths above the pubes. I then learned for the first time from the patient that her water had been "red like blood" for some days before labour set in. Except for this statement, and the fact that she had been troubled with slight dribbling from the bladder during her pregnancy, no other urinary history could be elicited.

The following morning the anterior tumour was still *in situ*, and the visiting medical officer who saw the patient with me washed the bladder out with hot boracic solution, which cleared out several clots, followed by tinct. ferri perchlor. The next day the patient could micturate naturally, and the blood which was passed with the urine was less in quantity; but during the whole course of her illness the urine was never free from blood. The blood was never intimately mixed with the water, and came most freely during the first part of the flow. There was no actual pain on micturition, but the patient complained of slight discomfort during the act—this was probably due to the presence of some urethritis. The bladder gradually decreased in size, but always remained palpable. Its cavity was evidently contracted, as during irrigation it only retained about 8 cz. of fluid.

On May 20th the patient complained of pain in the left calf, and on examination one of the veins of the leg was found to be thrombosed. There was nothing further of note in the course of the case, which terminated fatally on June 10th.

I may say that there was never any sign of purpura haemorrhagica, nor was there loss of blood from any source otherwise than per urethram. The heart and lungs presented no abnormality. The blood, unfortunately, was not examined, but the patient never looked markedly anaemic. During the last fortnight of her illness there was irregular evening pyrexia, but the temperature never rose above 101° F.

Treatment was mainly symptomatic, with a view to controlling the haemorrhage and keeping up the patient's strength.