disposal in the treatment of such disorders and here again I am afraid I shall be found in a somewhat negative or even destructive attitude.

In the treatment of all stomach disorders diet still holds the foremost place in the mind of the patient and frequently also in the mind of the practitioner. I believe in stomach disorders, as in most others, that the patient's appetite and his experience are his best guides and I am usually content with ordering such patients such articles of ordinary diet as they like and which they find agree with them. I very rarely prohibit meat, or cheese, or tea, or pastry of any kind, and if the patient likes pork and it agrees with him I ask him to take it. I usually prohibit all alcohol, as well as all what I may call manufactured non-alcoholic drinks, and I always enjoin the drinking of water in large quantities even at meals. The articles of diet against which I generally warn sufferers from gastric disorders (including ulcer of the stomach) are what are generally known as "slops," such as broth, soup, beef-tea, and porridge, raw eggs (and indeed uncooked food of all kinds), jellies and "messes" and second cooked things, and all foods out of bottles, tins, and boxes. I am sure dyspeptics are better without sugar and sweet things generally. I am constantly seeing people who cannot take milk in bulk and I think it is far from being an ideal food for adults either in disease or health. It is certainly no more digestible than meat and what are called milk diets by their bulk and insipidity are very soon distasteful to the patient. In the same way I find that well-made pastry frequently agrees much better than the bulky farinaceous puddings. Cold meat, boiled or roasted, can frequently be taken when hot meat disagrees. No case of gastric ulcer can, or should, be treated except in bed. I am accustomed even in this disorder to abstain from ordering milk diet. If there is great pain after food, especially if there is hæmatemesis, I prohibit all food and give only water, and plenty of it, by the mouth. I do not believe in rectal feeding; it is very distasteful and sometimes painful to the patient and it is of no real value. I have seen the most troublesome and offensive proctitis set up by nutrient suppositories. Many cases of hæmatemesis have been treated successfully in my beds by absolute rest and the administration of plain fresh water by the mouth and nothing else, unless a very little morphine is given, also by the mouth, to relieve pain and restlessness. I rarely use the so-called hæmostatics but in deference to public opinion I have occasionally ordered such inert substances as chloride of calcium to be given in large doses, also by the mouth, and I have occasionally used adrenalin which is a powerful and active substance and should be given with care.

Of the drugs in common use in stomach disorders laxatives occupy the first place in my estimation and of them I believe rhubarb is the best. Our old-fashioned rhubarb mixtures consisting of rhubarb, soda, and peppermint are excellent things taken after food for what we call the chronic, middle aged, constipated, flatulent dyspeptic. Most of the official preparations of rhubarb, including the powder, are too pungent and nauseating. I find the concentrated infusion most agreeable and efficient. I am one of those who believe that constipation is a real evil and cannot be treated too persistently, so long as it is treated intelligently. The salts of bismuth hold a deservedly high place in all painful and irritable conditions of the stomach, including ulcer, and, of course, cancer, but the old-fashioned bismuth mixtures are not nice things to take and I am glad to find that the practice of giving carbonate of bismuth in large doses simply stirred up in water is now very generally followed. I do not think that morphine is often necessary, except, of course, in the pains of cancer, and in that case any dose which is necessary to keep the patient at ease should be given ungrudgingly. If the bowels are kept acting daily by means of rhubarb and the kidneys are kept acting freely with water I am accustomed to give morphine in any dose that may be necessary. When large doses are required I prefer to give it by suppository. Such things as lactopeptin and taka-diastase and other artificial digesters are for the most part as expensive as they are useless and therefore unnecessary.

May I remind you that one of the most striking things we see in hospital practice is the astonishingly simple diets and medicines with which we manage to deal with our patients, and I believe just as successfully as with the more varied and expensive foods and medicines which so many think necessary in private practice, and it is from hospital practice that I have learned to believe in the simple

foods and medicines to which I have referred. I apply to the dietetic management of the stomach disorders the same rule which I ventured to advocate some years ago in the management of typhoid fever—viz., that no further restrictions in diet should be imposed than loss of appetite and impaired digestion indicate—that is, that the patient should take what he likes of ordinary articles of diet in so far as he knows they agree with him. Bulk is of much more consideration than kind in deciding what food should be given in gastric ulcer and all other stomach disorders.

In saying what I have said about food in stomach disorders I have not intended to shock what I know are the firmly held convictions of many whose authority to speak in these matters is far greater than mine. I have tried to tell you as clearly and as frankly as I could the methods which I constantly practise and which I constantly advocate and which I have no reason to believe have fallen seriously behind other and more orthodox methods in the measure of their success.

PNEUMOCOCCIC PERITONITIS IN CHILDREN: A STUDY.

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DURING the eight months in which we were working together at the East London Hospital for Children no less than four cases of peritonitis of the somewhat rare variety caused by the micrococcus lanceolatus came under our notice. The obscurity of the cases caused us to look up the literature on the subject, and the small number of cases reported in England and the slight attention which the condition has received in this country are our excuses for publishing the following account of the condition.

In 1885 da Bozzolo¹ described a case of infection of several serous cavities with numerous capsulated cocci in the exudate following on a nephritis. The case, however, was not fully worked out. In the following year Cornil² performed a necropsy on a case of pneumococcic peritonitis following pneumonia and associated with double empyema and pyopericarditis. During the next year or two isolated cases were here and there reported and in 1890 Nélaton³ undertook the first operation for the relief of the condition in a woman, aged 32 years, but without success. Galliard⁴ and Sevestre⁵ also operated during the same year on cases of pneumococcic peritonitis, Sevestre's case being a child with apparently primary involvement of the peritoneum. This case recovered and remained well. On the continent numerous examples of the disease now began to be recorded, for the most part from France. In 1903 von Brunn⁶ was able, in an excellent paper on the subject, to collect 57 cases of the disease in children and a further 15 adult cases. In the same year Jensen⁷ of Copenhagen published a lengthy article on the disease and in this he discussed the affection very fully. These two papers are up to now the most important contributions to the subject. Since the date of von Brunn's paper, Matthews⁸ and Bryant⁹ have brought forward fresh cases and have written on pneumococcic peritonitis; other papers have been contributed by Quervain¹⁰ and Ghon,¹¹ while cases have been reported by Panzacchi,¹² Brun,¹³ Powell,¹⁴ Taylor,¹⁵ Waldo,¹⁶

¹ Da Bozzolo: Centralblatt für Klinische Medicine, 1885.

² Cornil: Journal des Connaissances Médicales Pratiques et de Pharmacologie, 1886.

³ Nélaton: Bulletin Médical, 1890.

⁴ Galliard: Bulletins et Mémoires de la Société Médicale des Hôpitaux de Paris, 1890.

⁵ Sevestre: Semaine Médicale, 1890.

⁶ Von Brunn: Beiträge zur Klinischen Chirurgie, 1903, 29.

⁷ Jensen: Archiv für Klinische Chirurgie, 1903, 70.

⁸ Matthews: American Journal of the Medical Sciences, 1897, 113.

⁹ Bryant: Brit. Med. Jour., vol. ii, 1901.

¹⁰ Quervain: Centralblatt für Schweizer Ärzte, 1902.

¹¹ Ghon: Wiener Klinische Wochenschrift, 1904.

¹² Panzacchi: La Riforma Medica, 1904.

¹³ Brun: La Presse Médicale, 1901.

¹⁴ Powell: Brit. Med. Jour., vol. i., 1905.

¹⁵ Taylor: Transactions of the Clinical Society of London, vol. xxxvii.

¹⁶ Waldo: Brit. Med. Jour., vol. i., 1904.

Webber,¹⁷ Fisher,¹⁸ Dudgeon and Sargent,¹⁹ Davies and Langdon Brown,²⁰ and Duckworth and Marsh.²¹ As the result of a search of the literature we have been able to find records of 91 cases of pneumococcic peritonitis in children below the age of 15 years, including the cases reported above. The number of cases reported as occurring in adults is considerably less. The classification of these cases has been very difficult and not at all satisfactory, as in a very large number it is impossible with any certainty to decide as to which was the starting point of the infection. The question as to the possibility of infection taking place without the formation of a lesion at the point of entry of the micro-organism is of great importance in this connexion, and we believe that that possibility is now accepted. We decided to divide the cases into (1) those secondary to a definite pneumococcal infection of some other part of the body; (2) those in which the peritoneum was apparently the first point of attack of the organism; and (3) those in which owing to the rapid general spread of the infection it is impossible to decide as to which of the other groups they should fall under.

Of the 91 cases 30 fall under the first group, 47 into the second, and in 14 instances it is impossible to place the case into either of these groups. As an example of the last group we may relate shortly a case reported by Stoos.²²

A girl, aged 11½ years, was suddenly seized with convulsions, vomiting, pain in the lower abdomen, and fever. Two days later there were râles and slight dulness at the left apex. On the next day the lower abdomen was swollen, and there were diarrhoea, herpes labialis, enlarged spleen, continued high temperature, and bad general condition. Acute gastro-enteritis was thought of, then typhoid fever, and finally tuberculous peritonitis. A large encysted abscess formed in the abdomen which was eventually incised (on the thirty-fourth day), and the patient in time recovered. In this case it seems to be impossible to determine the point of entry of the pneumococcus; there may have been pneumonia, or the peritoneum may have been infected from the bowel or in some other way. We have, therefore, on similar grounds, placed 14 of our 91 collected cases into the third group. Of the cases we report, Nos. 3, 4, 15, and 16 we regard as coming into our "primary" group, No. 8 into our "uncertain" group, and the remainder into our "secondary" group.

Etiology.—In discussing this question the primary and secondary cases must be considered separately and it seems advisable to discuss the latter first, as more is known of the method of spread of the pneumococcus to the peritoneum in this group, and the consideration of this will simplify the study of the etiology of the primary cases.

The secondary cases.—One of the most interesting and striking features brought out by a classification of the various primary lesions is the enormous preponderance of cases in which this lesion is pulmonary. We have collected 30 cases in which the peritonitis was definitely secondary in point of time, at least, to a lesion elsewhere, and in 22 of these cases (73 per cent.) this earlier lesion was in the lungs or pleuræ. The primary foci in the remaining eight cases were: in one case, the umbilicus; in two cases, sore-throat; in three cases, otitis media; and in two cases, intermuscular abscess not connected with bone (in the neighbourhood of the ankle and hip respectively). In the last seven of these cases the early focus was so far remote from the peritoneum as to make the blood stream the only possible path by which the pneumococcus reached the peritoneum. In four of these seven infection of several serous cavities occurred in each, affording additional evidence in favour of pneumococcic septicæmia. In the remaining case of extra-pulmonary origin the umbilicus was probably the source of infection; suppuration followed the detachment of the cord and peritonitis began shortly after. At the necropsy right-sided empyema was found. It is quite likely, though in the absence of blood cultures it cannot be proved, that the pleura was infected by the passage of the pneumococcus through the diaphragm with the lymph stream.

Much discussion has centred round the question as to

whether in cases of pulmonary and pleural origin the peritoneum is infected through the blood stream or by penetration of the pneumococcus through the diaphragm.²³ It has now been definitely established that, in pneumonia in man, pneumococci can be grown from the blood in practically every case (Ewing). This is strong evidence in favour of the peritoneum being infected from the blood stream, so strong that only on the most conclusive proof can the occurrence of the alternative trans-diaphragmatic mode of infection be established. Some work of Burckhardt,²⁴ which bears on this subject, must be considered. In one case he examined the diaphragm microscopically and found pneumococci in the pleural serosa and subserosa, while they were absent in the deeper layers. In a second case the organisms were found in great numbers throughout the whole thickness of the diaphragm. This case, however, had endocarditis and abscess in the shoulder-joint and was obviously of septicæmic type; the diaphragm was probably invaded from the blood stream. Experiments on animals showed that to effect penetration of the diaphragm by organisms from the pleura, virulent cultures must be used and the endothelium of the pleura must first be destroyed by a chemical poison—e.g., oleum terebinthinæ. Even in these circumstances, however, no peritonitis resulted. It must be remembered that pneumococci passing through the diaphragm by way of the lymph channels must proceed against the current in these vessels. We consider that the present evidence in favour of such a path of infection, when placed in opposition to the fact of the presence of septicæmia in practically every case of pneumonia, cannot be held to be of any weight. Of our 22 cases of pulmonary origin, in four remote lesions occurred late (otitis media, arthritis); these must have been brought about by blood-borne organisms. In the remaining 18 there is no direct evidence as to the path of infection. In 11 cases out of 30, therefore (seven out of eight cases of extra-pulmonary origin and four out of 22 cases of pulmonary origin), the infection of the peritoneum from the blood is certain (37 per cent.); in the remaining 19 that mode of infection is most probable.

Primary cases.—One of the most likely channels through which the micro-organisms may gain access to the peritoneal cavity in these cases is the bowel. The Fallopian tubes must also be considered in the case of female patients; as regards other mucous membranes—e.g., the tonsils and the middle ear—it is conceivable that the pneumococcus might gain the blood stream through them without causing a local lesion, and then first attack the peritoneum, perhaps at the same time or later implicating the other serous cavities. We must now consider what evidence can be brought forward in favour of the view that the peritoneum became infected from the bowel in the 45 primary cases we have collected. From the clinical side there is the fact that in 35 out of the 45 cases the onset of the illness was a sudden pain in the abdomen, with vomiting or diarrhoea; in 26 out of the 35 cases both vomiting and diarrhoea were noted. In a very few cases there is some history of an indigestible meal. It is open to argument that the vomiting, &c., were due to the commencing peritonitis but the very frequent occurrence of an early diarrhoea points in favour of these symptoms being those of an enteritis, rapidly passing on to peritonitis. In the remaining ten cases the reports are for the most part defective; in two of them digestive disturbances are reported without being defined.

The necropsy results are very scanty and neither prove nor disprove the theory. Of the 21 primary cases with a fatal result, in 15 there is no report as to the condition of the mucous membrane of the intestine; of the remaining six, in four it is spoken of as being healthy and in two cases swelling of Peyer's patches is noted. A swollen patch cut and stained showed in one case pneumococci in the mucous membrane only; in the other case no organisms were found. Thorough investigation of the state of the bowel wall is much to be desired in future cases. There is now no doubt as to the occasional presence of the pneumococcus in the lumen of the bowel. Weichselbaum, Flexner, and other authorities have pronounced definitely as to its presence. Normally it is killed by the gastric juice but disordered digestion, with marked reduction of the acidity of the gastric juice, no doubt enables the organism to reach the small gut. In adults numerous cases of enteritis due to the pneumococcus, in some instances associated with peritonitis and in others not, have been recorded, and there seems no reason

¹⁷ Webber: Brit. Med. Jour., vol. ii., 1904.

¹⁸ Fisher: THE LANCET, Nov. 10th, 1900, p. 1384.

¹⁹ Dudgeon and Sargent: THE LANCET, Feb. 25th (p. 473) and March 4th (p. 548) and 11th (p. 617), 1905.

²⁰ Davies and Langdon Brown: THE LANCET, Oct. 8th, 1894, p. 1017.

²¹ Duckworth and Marsh: Transactions of the Clinical Society of London, vol. xxxvii.

²² Stoos: Jahrbuch für Kinderheilkunde, 1902.

²³ Michaut: Gazette des Hôpitaux, 1901.

²⁴ Burckhardt: Beiträge zur Klinischen Chirurgie, 30, 1901.

why the same lesion may not occur in the case of children. Indeed, Jensen²⁵ speaks of two cases in children (he does not describe the cases fully and they are therefore not included in our statistics), one of membranous enteritis and diffuse pneumococcic peritonitis and the other of catarrh of the colon with diffuse peritonitis. Stoos²⁶ describes a case as proving infection from the bowel, but von Brunn thinks that the evidence is not conclusive. There was general fibrinous peritonitis, the solitary follicles and Peyer's patches were much swollen, the mucous membranes over them were necrotic, and the submucosa and serosa were much infiltrated. Bacilli and numerous diplococci were found throughout the whole thickness of the bowel wall and in the fibrinous peritoneal exudate. The bowel contained extraordinary numbers of diplococci, for the most part capsulated. No cultures were made. Von Brunn, on account of there being no evidence of reaction to the organism in the muscular layer, thinks that a post-mortem invasion is not excluded. Jensen made some experiments with a view to find support for the theory of bowel infection of the peritoneum in these cases. Two young mice and two young rabbits were each given through the mouth ten cubic centimetres of a highly virulent culture of pneumococci. One rabbit got diarrhoea, wasted, and died in four days. General fibrino-purulent peritonitis was found; the gut was strongly injected. There was no ulceration but the Peyer's patches were necrotic. Pneumococci were found in pure culture throughout the body and diffused through the gut wall. As the animal did not show symptoms of septicæmia he concludes that the general infection was late and that the peritoneum had become infected directly from the gut.

It is very likely that in some cases appendicitis is the starting point of the peritonitis. This method of origin is more particularly found in adults and a considerable number of these cases have been reported. In children definite evidence of appendicitis is rare. In a case recorded by Quervain²⁷ an encapsulated hypogastric abscess was opened and the pus contained pure pneumococci. Five months later an appendectomy was performed and pneumococci and other organisms were found in the lumen of the appendix. The wall of the appendix was normal and contained no organisms. Krogius, quoted by Quervain,²⁸ had a case—a girl, aged 14 years—of pneumococcic peritonitis in which the appendix was obliterated in the middle and perforated at the tip. He considered it likely that a pneumococcal appendicitis was the primary lesion. In none of the 21 primary fatal cases we have collected is there any mention of a diseased appendix; in the 24 cases which recovered the appendix was not seen. While admitting, therefore, that the appendix is the starting point of the peritonitis in a few cases we cannot endorse the view of Quervain who thinks that an affection of this organ is the commonest cause of pneumococcic peritonitis in these "primary" cases. Whether the pneumococci reach the peritoneum by traversing the gut wall or are taken up into the blood stream after penetrating the mucosa and thence gain the peritoneum we have no means of judging. The pathological evidence is *nil* and the experimental doubtful; apparently Jensen's experiment referred to above is the only one on the subject.

When considering the female genitalia as the starting point of the peritonitis in these cases mention must first be made of the sex incidence of the disease. Of all the cases females were affected in 73 per cent. and males in 26 per cent.; of the primary cases females in 84 per cent. and males in 16 per cent.; and of the secondary cases females in 67 per cent. and males in 33 per cent. It is very difficult to account for these figures. Brun, quoted by von Brunn,²⁹ ascribed a great rôle to the female genitalia as a portal of entry to the peritoneum for the pneumococci, relying on figures very similar to those given here. But of pathological support to this theory there is practically none. In two cases a congested tube was found; in a case recorded by Dudgeon and Sargent³⁰ pyometra was present with gonococci but no pneumococci in the pus. Slight vulvitis was present in one case and slight vaginitis in another. In the remaining cases the genital organs were normal. In the secondary cases, too, the same preponderance of females, slightly less marked, is found. In these cases the lungs are by far the commonest seat of the primary lesion and the female genitalia can have nothing to do with the pneumonia. One must suppose that when a larger number of cases are reported

this apparent excess of females affected will disappear. The remaining possible explanation of the occurrence of the primary peritoneal affection is that pneumococci have been absorbed through an apparently intact mucous membrane, septicæmia has occurred, and the cocci in the blood have found in the peritoneum a favourable situation for growth. With our present knowledge an opinion on the point cannot be given. It is now certain that in practically every case of pneumonia the pneumococcus is present in the blood stream. At what stage in the onset or course of the disease the organism gains the blood is not known. It is possible that in some cases the lungs may be infected by the blood stream and not *vice versa*, and if this be so the peritoneum may be in other cases a more favourable situation for the growth of the pneumococcus and the effect of the infection may be first manifested there.

Clinical characters.—In dealing with the clinical manifestations of the disease we are constrained to rely to a very great extent upon the analyses of the cases published by different writers and particularly to the writings of von Brunn and Jensen referred to above. Two of our own four cases were cases of primary peritoneal infection, these being Nos. 3 and 4. In the first of these cases a baby one month old was brought to hospital with a history of an acute onset of illness, the only symptoms being vomiting, followed a day later by diarrhoea and abdominal distension. These two symptoms persisted up to the time of death. The second case was complicated by the presence of renal disease, on which it appears the peritonitis was superadded as a terminal affection, and the only symptom of the latter was the presence of persistent abdominal pain. Of the other two cases which we ourselves saw the first is one of those cases of general pneumococcal infection with multiple local lesions, the signs of peritonitis being quite obscured by the extreme prostration and the severe thoracic infection. In the second case we have the picture of the onset of the peritonitis scarcely masked by an intermuscular abscess about the hip, and here again the symptoms noted in the first acute primary case were present—viz., vomiting and diarrhoea.

Two types of the disease have been separated on a pathological basis, the circumscribed and the diffuse, and a clinical entity given to each. This classification will be adhered to in our account, although it may be said that the difference of type is only one of degree. The circumscribed affection sets in acutely but soon passes into a chronic stage, in which the pus becomes encapsuled, marked by the mildness of symptoms. In the diffuse variety, on the other hand, no localisation of the pus takes place and it is noticeable for the violence and persistence of the symptoms. These two classes are further capable of division into primary and secondary groups, depending upon a previous absence or presence of a primary local infection elsewhere in the body.

Primary circumscribed pneumococcic peritonitis has three phases. Michaut³¹ described these tersely as (1) the meteoric; (2) the exudative; and (3) the perforative. The first phase is characterised by the acute onset in what has usually up to then been a quite healthy child of acute abdominal pain which may begin locally but usually spreads rapidly over the whole abdomen. With the pain comes vomiting which may only last for 12 hours but sometimes persists for days, according to Jensen for not more than six. The diarrhoea, practically always present, is severe and more persistent and may last all through this first stage. Fever is not, as a rule, very high and usually falls about the tenth day. The pulse curve corresponds to the fever curve. In short, the cardinal symptoms of the onset of pneumococcal peritonitis are these: severe abdominal pain, vomiting, diarrhoea, and fever. Albuminuria may occur but is uncommon. This first phase lasts from 10 to 14 days. The second phase is that of exudation into the peritoneal cavity with the formation of a local abscess. It is characterised by the signs of slow formation of fluid without marked pain, tenderness, or discomfort. Jensen³² drew an analogy with the formation of an empyema following the acute pleurisy of a pneumonia, while Lennander calls the condition "abdominal empyema." The encapsulation is first manifested by local swelling and dulness and on palpation there is usually some tenderness unassociated with muscular rigidity.

The situation of the abscess is usually sub-umbilical and placed rather laterally than mesially. If untreated it usually, in time, completely fills the abdomen, but in the

²⁵ Loc. cit.²⁸ Ibid.²⁶ Loc. cit.²⁹ Loc. cit.²⁷ Loc. cit.³⁰ Loc. cit.³¹ Loc. cit.³² Loc. cit.

epigastrium the stomach resonance may practically always be elicited, even with the most advanced distension. With great swelling of the abdomen the veins on its surface may become prominent. During this stage the temperature is slightly elevated, the diarrhoea has ceased, and constipation may be present. Vomiting is uncommon. The patient usually wastes and in advanced cases the abdominal distension may offer a marked contrast to the general emaciation. On the other hand, the general condition sometimes remains good. The third phase is only present if surgical intervention has not been attempted or allowed and depends upon the natural endeavour to evacuate the abscess. This usually points about one month from the commencement of the illness and in most cases by this time the child is gravely ill, the cachectic appearance and distended abdomen often suggesting advanced tuberculous peritonitis. The usual site of perforation is the umbilicus. Jensen states that in 25 cases of capsulated peritonitis perforation at the umbilicus occurred in seven and of eight similar cases following pneumonia the site of perforation was the same place in six. Perforation at other sites is rare, but the pus has been described as escaping through the vagina in several cases and through the bladder once.

After spontaneous evacuation a cure may result, but more commonly there is some retention of pus and the abscess has to be thoroughly opened and drained. Von Brunn emphasises the diagnostic significance of spontaneous fistula formation at the umbilicus: "It is so frequently an occurrence in pneumococcal peritonitis as to be one of the most characteristic features. If, after symptoms of an acute peritonitis in a child, a discharge of pus occurs from the umbilicus, pneumococcal peritonitis must be thought of first of all." With regard to the other signs and symptoms during this phase the temperature is usually low, cedema of the legs may occur, while vomiting and diarrhoea are absent; there may be signs of pressure, especially on the pelvic viscera. Von Brunn states that in his first case interference was decided against on the surgical side on account of the absence of fever. Our 91 collected cases include 26 cases of the primary circumscribed variety.

Passing on to secondary circumscribed pneumococcic peritonitis, it may be said that the signs and symptoms vary in their intensity and diagnostic clearness, according as the peritonitis sets in immediately after the onset of the primary manifestation or some days later. Among our 91 collected cases there are 11 cases belonging to this class. Of these 11 the first stage was unrecognisable in no less than seven (64 per cent.) owing to the peritonitis setting in very shortly after the onset of the primary affection which in six of the seven cases was either pneumonia or broncho-pneumonia, and in the seventh case an arthritis. In another case the first stage was also missed, although the primary affection was apparently less severe, being a peri-articular abscess followed by the involvement of the joint. It may be pointed out, however, that to speak of the primary lesion being less severe may not be quite accurate, since in some of the cases at least the primary affection may be merely the first local manifestation of what is in truth a pneumococcal septicæmia. This general infection may in its severity be quite sufficient to mask to a greater or lesser extent the symptoms of the onset of the peritonitis. Of the remaining three cases (27 per cent.), in one the primary affection was pleurisy unaccompanied by pneumonia, the onset of peritonitis being well marked with pain, vomiting, and diarrhoea; in the other two there were broncho-pneumonia and pneumonia respectively as the primary lesions. An interval of some days elapsed and was followed by all the signs of an acute peritonitis. It follows from this masking of the acute symptoms that the majority of these cases were only recognised during the second or third stages which are the same as in the primary form and require no further mention.

Primary diffuse pneumococcic peritonitis is characterised by the acuteness of the onset, the severity of the symptoms, the rapid prostration of the patient, and in most instances his early death. In most of the cases reported its severity and early fatal result make it comparable with a perforative peritonitis. As mentioned above, the symptoms are those of the first or acute stage of the encapsulated variety but differ in their great virulence and persistence until the patient's death if no attempt be made to relieve the condition by laparotomy. Intense pain, vomiting, and diarrhoea set in suddenly, rapidly prostrating the patient, so that he speedily acquires the abdominal facies, with dry tongue, sordes, delirium, cold extremities, and cyanosis. The temperature

varies between 100° and 102° F. and the pulse between 140 and 160. If no relief be afforded death soon follows. Of the 91 cases which we have collected 21 were of this primary diffuse form, including Nos. 4 and 16 of our cases.

Secondary diffuse pneumococcic peritonitis was present in 19 out of our 91 cases. Jensen, on the other hand, only found it once in 58 cases. Of these 19 cases the signs and symptoms pointed so strongly to the presence of peritonitis in five that an operation was performed. Of the remaining 14 in four there was strong evidence of the presence of peritonitis but as no operation was performed it must be allowed that the evidence was not sufficiently strong to warrant surgical interference. In three there were signs or symptoms pointing to abdominal infection but these were not sufficient to permit a diagnosis of peritonitis. In two there was no evidence of peritonitis before death, whilst in five either there is no clinical history supplied (2) or no reference has been made to the condition of the abdomen (3).

From this abstract of the 19 cases it will be noted that, as might be expected, when the secondary peritonitis is of the diffuse variety it gives definite early signs of its presence in a much greater proportion of cases than when it is localised—namely, in nine of the 19 cases (47 per cent.). As further bearing on this matter it may be noted that amongst the 19 cases pericarditis was present five times, never as the only other local infection, but always in association with pneumonia, broncho-pneumonia, or empyema. It may be said, in fact, that the presence of pericarditis is a sign of a severe general infection (septicæmia) and is associated with the most grave symptoms. Hence it is worthy of note that in the five cases where pericarditis was present at the post-mortem examination no mention is made of signs or symptoms pointing to peritonitis save distension of the abdomen in one case; indeed, in three of the cases it is definitely stated that no clinical evidence of peritonitis was forthcoming. We consider that this evidence corroborates the view that in the severe septicæmic type of case the local manifestations are obscured to such an extent as to prevent their recognition clinically. When peritonitis has been diagnosed the symptoms have been for the most part the same as those of the primary form, acute abdominal pain, sickness, diarrhoea, and abdominal distension. The antecedent lesion in these cases was single in ten and multiple in nine. In addition to four cases in which pleurisy or empyema occurred as the only lesion found post mortem accompanying the peritonitis the latter was also present in conjunction with other local lesions in eight cases and was nearly three times as frequent as any other local primary manifestation.

Pathological anatomy.—Jensen epitomises the action of the pneumococcus by saying "it is pus-forming and fibrinogenous." Our own experience is all in favour of this statement and a reference to the conditions found at the post-mortem examinations in our own cases will bear this out. The pus, provided that no post-mortem infection from the intestine with bacillus coli has occurred, is odourless. Its fibrinous character is shown by the layer of lymph on the wall of the cavity and by the presence of jelly-like masses in the pus, this last appearance being due to an intimate mixture of the purulent and fibrinous elements of the exudate. The fibrinous layer covering the surface may appear simply as a layer of lymph, dull and whitish-grey, or, by the presence of pus in and between its layers, it may assume the form of a yellowish or greenish yellow gelatinous membrane which may usually be peeled away from the lining wall of the cavity. Sometimes it may be hæmorrhagic in character. Similarly, the main collection of pus, which is thick and yellow, or greenish yellow, may have floating in it the jelly-like masses mentioned, or flakes of lymph or hæmorrhagic masses, the last being due to the presence of blood between layers of fibrin. The above is the typical picture but in exceptional cases misleading characters may be found. In cases where the post-mortem examination has been deferred for more than 24 hours the exudate may be evil-smelling owing to post-mortem contamination. This was so in Case No. 4, reported below. In another case "the abdomen was full of thin, yellowish-brown pus which poured out as soon as it was opened" (Case 3). In both these cases, however, there was a very pronounced fibrinous element, and so constant is this character, so much a part of the action of the pneumococcus in a serous cavity, that Jensen has stated that the diagnosis can always be made on macroscopical examination of the pus.

The conditions found in the abdomen vary according as the peritonitis is diffuse or encapsuled. We can only speak from experience of the diffuse variety but give a synopsis of the accounts of other observers of the encapsuled variety. The local pneumococcic peritonitis of children, "the abdominal empyema" of Lennander, is usually subumbilical and rather more to one side than the other. Its limitations depend upon the early deposit of lymph shutting off the main part of the cavity of the abdomen. There may be more than one abscess. In a case given by Galliard³³ one abscess lay between the liver and diaphragm and another one was situated around the spleen. Again, in Sevestre's³⁴ case one abscess was localised to the true pelvis and another to the caecal region. If very large the abscess may fill the whole abdomen. In the first case given by von Brunn in his paper a large abscess had been opened, the pus pouring out under great pressure. He goes on to say, "the whole abdomen formed a huge abscess cavity in which the bowels, rolled into a little ball, lay almost like a foreign body. Liver, stomach, transverse colon, omentum, and coils of small intestine were all stuck together and lay in a mass as large as a man's head on the middle of the vertebral column." This is an extreme case. The serous membrane in these cases is injected, dulled, thickened, and covered to a varying extent with fibrin. Jensen believes that the encapsuled abscess begins as a diffuse peritonitis and its localisation depends upon early limitation by fibrinous exudate. Diffuse pneumococcic peritonitis was found post mortem in all our cases. A reference to Case 2 will supply the reader with a very good idea of the condition found in this variety. The condition of the serous surface varies with the length of time the abdomen has been infected. Exudation is usually rapid and deposit of fibrin takes place early. Although speaking from a very limited experience we believe that pronounced injection of the serous membrane does not take place early.

Passing on to the condition of the viscera we note that in none of our cases was any macroscopic lesion of the stomach or bowel found. The intestines, including the appendix, were carefully examined in all but with a completely negative result. In two cases some enlargement of Peyer's patches was found. In each a single Peyer's patch was removed and a portion of the spleen at the same time. These were microscopically examined for organisms. One proved entirely negative but in the other Gram-positive diplococci morphologically identical with the pneumococcus were found both in the Peyer's patch and in the spleen. It must be clearly noted that in this case there was no trace of ulceration, the mucous membrane was not notably injected, and the organisms which we look upon as pneumococci were not found deeper than the mucous membrane of the Peyer's patch.

On leaving this division of the subject a few lines may be written upon the bacteriology of the cases we report. In two of our cases (Nos. 1 and 3) we relied upon the presence of Gram-positive diplococci, either alone or in chains, scattered in large numbers throughout the pus, this diplococcus being morphologically identical with the pneumococcus. In one of these cases the pneumococcus had already been grown from pus taken from the pleural cavity (Case 1). In another case (Case 2) we added to the evidence supplied by the examination of the pus the further proof mentioned above—namely, the presence of pneumococci in the spleen and a Peyer's patch. In Case 4 we cultivated from the blood, growing a Gram-positive diplococcus morphologically identical with the pneumococcus. Dr. J. W. H. Eyre, bacteriologist to Guy's Hospital, kindly examined this culture for us and stated that it gave a typical growth on blood agar but that inoculation into a rabbit did not kill the animal. Of our other cases quoted at the end of this paper, in seven the proof was either the presence of the pneumococcus in cultures from the blood or its presence in the peritoneal pus. In the other five the evidence is indirect, pneumococci being found in pus from an empyema or pyopericardium, and as macroscopically the peritoneal pus was similar in nature the conclusion was drawn that the same organism was the cause of the abdominal pus.

Diagnosis.—Michaut³⁵ has stated that whereas the diagnosis has frequently been made in the encysted form it has only once been made in the diffuse form. This statement is set down as an appropriate introduction to the discussion of diagnosis, since in most cases encysted peritonitis

can be diagnosed, whilst the diffuse may, and probably in the majority of cases will continue to be, extremely difficult if not impossible to diagnose correctly. The importance of the history is very great in many of these cases. This may be of the greatest diagnostic value, or its true meaning may only be recognised after the event. Experience has shown how obscure pneumonia may be in some cases, much more so a tonsillitis or conjunctivitis due to the pneumococcus. We recently saw a case of fever lasting over a week and falling by crisis, in which all the pulmonary signs recognised by skilled observers pointed to diffuse bronchitis only. Typhoid fever was thought of, but Widal's reaction proved negative. After the fall of temperature the patient expectorated a muco-pus which was examined for tubercle bacilli; these were absent but numerous bacilli and diplococci were present. The diplococci were Gram-positive and morphologically identical with the pneumococcus and we are of the opinion that what might have been a very obscure case but for this examination was a case of central pneumonia giving practically no physical signs of consolidation. This case occurred in an adult, but similar cases occur more frequently in children and we are inclined to look upon "cough and fever" preceding pneumococcic peritonitis as being highly suggestive of a previous pulmonary infection. In this connexion we may call attention to the second of our own cases where the boy had an abscess about the hip. This abscess was opened but unfortunately no bacteriological examination made of the pus. Looking back now on the case we consider that this inter-muscular abscess was the primary lesion and would emphasise the importance of a bacteriological examination in such cases. At a later date a baby was admitted into the Children's Hospital at Shadwell with several superficial abscesses which were opened. An examination of the pus showed the presence of pneumococci and we therefore drew off a sample of the blood from the median basilic vein and on blood serum pneumococci grew in pure culture. We would lay special stress on history in these cases and as being especially valuable when forthcoming in a case of acute peritonitis setting in suddenly and in an apparently previously healthy child. When the difficulty of diagnosis is considered and the heavy mortality necessarily resulting, no clue, however slight, can be disregarded.

The localised form of peritonitis will, as Michaut says, be diagnosed, since sooner or later an abdominal abscess will form, but unless the history of the acute attack or, in some cases, of a preceding pneumococcic affection elsewhere is obtained the diagnosis of the abscess may not be made until it is on the point of bursting at the umbilicus. This is unsatisfactory, for it means that the child has been subjected to a continued infection for a long period, at times up to a month and in many cases is in a weak and emaciated condition before the surgeon operates. The diagnosis should be made, if possible, and the condition relieved during the acute stage or during the early part of the quiescent period. The cardinal symptoms to rely upon are the acute onset of the vomiting and diarrhoea and abdominal pain, associated with a sudden rise of temperature. Herpes labialis appears to be so uncommon that its absence is quite unimportant, but it has occurred in a few cases. This local form of the disease has to be diagnosed from other forms of peritonitis, including that following appendicitis, from typhoid fever, and in its second and third stages, from tuberculous peritonitis.

From other forms of peritonitis which, save for that arising primarily from the appendix, are uncommon there will be great difficulty in diagnosis in the absence of any history. The same may be said with regard to the diagnosis from appendicitis, since the pain is chiefly in the right iliac fossa in many of these cases, while in many cases of appendicitis, especially in the early stages, the pain is not localised. Further, in very young children knowledge as to the situation of the pain is often unobtainable. In many such cases operation will be withheld and expectant treatment adopted. It is of importance that in such cases the second period should not be missed but the abscess formation treated as soon as recognisable. From typhoid fever the diagnosis may prove difficult if diarrhoea persists after the vomiting has ceased and the temperature remains raised. The absence of the rash and of splenic enlargement, the presence of a leucocytosis as opposed to a leucopenia, and the fact that a Widal examination proves negative, are all points against the illness being typhoid fever. There can be no doubt that in the past

³³ Loc. cit.³⁴ Loc. cit.³⁵ Loc. cit.

many cases of encysted pneumococcal pus have been looked upon as cases of tuberculous peritonitis. It is somewhat remarkable that on looking through the reports of cases at the East London Hospital for Children for the last ten years not a single case of intraperitoneal abscess due to the pneumococcus, operated upon and cured, has been recorded as such. We are of the opinion that such cases must have been treated but, the pus being looked upon as tuberculous, no bacteriological examination was made. There can be little doubt that in the later stages, when the child is very emaciated, the appearance is almost typical of tuberculous peritonitis. As points in diagnosis von Brunn calls attention to the importance of a history of the acute stage and the fact that in tuberculous peritonitis masses can be felt which are absent in the pneumococcal form. In the chronic stages he says only puncture can decide the question.

The diffuse form of pneumococcal peritonitis has to be diagnosed from a severe and acute attack of typhoid fever and from peritonitis following perforation of a viscus. The same emphasis may be laid on the importance of history in this form as in the local, but with the primary form there is little doubt but that in most cases the diagnosis will be next to impossible. We have no statistics at hand with regard to the relative frequency of acute diffuse peritonitis due to the pneumococcus and that due to perforations of the bowel. Such figures will no doubt be supplied in the future and prove valuable. For the present we can only say that as the commonest causes of acute perforative peritonitis in children appear to be perforation of the appendix and typhoid and tuberculous ulcerations of the bowel, in the absence of any history one of these conditions will still be diagnosed, but that if the peritonitis is recognised and an operation follow the character of the pus should lead to a diagnosis in most cases at once. Jensen in his paper states that of 16 cases of operation on the diffuse form eight were operated on for appendicitis.

Prognosis.—It appears to us on looking through the collected cases that prognosis depends on three factors: (1) whether the peritonitis is local or diffuse; (2) whether an operation is or is not performed and, if performed, whether or not at a suitable time; and (3) the previous presence or the onset of a severe complication. Whether the peritonitis is primary or secondary, providing there is no other complication, does not appear to affect the prognosis to any material extent according to our table. The following is the table which speaks for itself:—

Variety of peritonitis.	Number of cases.	Number of cases operated on.	Number of cases not operated on.	Result.		
				Recovery.	Death.	Uncertain.
Primary local ...	26	26	0	22	3	1
Secondary local ...	11	10	1	9	1	1
Primary diffuse ...	21	10	11	2	19	0
Secondary diffuse ...	19	5	14	3	16	0
Origin un- } local ...	8	8	0	6	2	0
} diffuse...	6	3	3	1	5	0
Total ...	91	62	29	43	46	2

45 of the 91 cases had encysted peritonitis, of which 44 were operated on, and of these 37 recovered and six died; in two the result is uncertain. The other 46 cases were of the diffuse variety, of which 18 underwent operation. Of the 18 cases operated on six, or 33·3 per cent., recovered, whereas all those not operated on died. The one case of the local variety not operated on recovered by spontaneous evacuation of the pus. The above table shows that recovery occurred in 86 per cent. of those with the encysted form, whilst only 14 per cent. of the cases with the diffuse variety survived.

Treatment.—In dealing with treatment we shall be very brief, simply giving the conclusions arrived at by a study of the cases reported and a consideration of the pathological condition found at post-mortem examinations. There can be no doubt but that when once pneumococcal peritonitis has been diagnosed a laparotomy should be performed. Jensen says, "Operate before a distinct abscess has formed," but acknowledges that the early stages may be very difficult to diagnose. Remembering, however, that there is an acute stage in localised pneumococcal peritonitis in which the

indications for operation may not be strong enough to lead to immediate intervention, it may be hoped that in the future such cases may be watched and the persistence of fever, of some abdominal tenderness, and perhaps diarrhoea, may lead to an earlier diagnosis of the condition and earlier operation. At operation the cause of the peritonitis may, in the great majority of cases, be recognised by the character of the pus, non-odorous, greenish, and fibrinous. It is sufficient in the localised form to treat the abscess in the simplest way, inserting a large drain and allowing it to heal from the bottom. With the largest of abscesses there is no trouble. In the acute diffuse form the abdomen will be opened in many cases for peritonitis of unknown cause. The character of the pus should make the diagnosis clear. It is as yet impossible to advise any exact line of treatment but, if the pus is not evil-smelling, mopping out the cavity and closing it without drainage would appear to be the wisest course. In the event of the pus being foul-smelling from infection with bacillus coli the perforation of some viscus would suggest itself and this would resolve the treatment into that special to perforative peritonitis.

Summary.—1. Pneumococcal peritonitis is a comparatively rare disease in children but in the past many cases have not been recognised. 2. It is in about a third of the cases secondary to some remote pneumococcal lesion; of these affections of the lungs and pleuræ are by far the commonest, the middle ear being the next most common site. The infection is carried to the peritoneum in the blood stream. 3. In the remaining two-thirds the peritoneum is probably infected from the bowel in the vast majority. No microscopic lesion is, as a rule, found. 4. In half the cases the pus is encysted; in these the diagnosis is fairly easy, the prognosis is good, and the treatment is laparotomy and drainage. 5. In the other half of the cases the peritonitis is diffuse, the diagnosis is very difficult, and the prognosis very gloomy. The treatment is laparotomy. 6. The pathological appearances are characteristic.

The cases which we personally observed and for permission to publish which we are indebted to the medical and surgical staff of the East London Hospital for Children were as follows:—

CASE 1.—A boy, aged four years, was brought to the hospital in March, 1905, with the history that seven weeks before he had had tonsillitis, followed by pneumonia. This got better after 14 days, but on the sixteenth day he had a relapse. He had since been getting worse and latterly had had some pain in the chest and abdomen. The boy on admission was pale and thin and had great dyspnoea. The left chest was obviously full of fluid and a piece of rib was resected, a pint of pus, creamy and odourless, escaping. Great improvement in the boy's condition followed the operation. During the next week the temperature was irregular, occasionally reaching 101° F. at night. On the ninth day after admission he was worse and suddenly became very collapsed, but revived on receiving stimulants. On the eleventh day he had two similar attacks. There were slight impairment of resonance and deficient breath sounds at the right base and the general condition was worse. During all this time the empyema which had been opened was draining well. On the thirteenth day the breath sounds at the right base were stronger and the general condition was unchanged. On the fifteenth day the patient looked much worse. The right limit of cardiac dullness, which had been extended to the right by the fluid in the left chest and had never recovered its normal position, was two inches from the right edge of the sternum and there were marked dullness and resistance at the right base behind. The question arose as to whether there was a right-sided empyema or a purulent pericarditis. The child died before the arrival of the visiting physician.

At the necropsy the right pleural cavity was found to be obliterated, save for a small pocket behind and at the base which contained from one to two ounces of thick greenish pus. The left empyema had been drained perfectly. There were firm adhesions between the pleural and pericardial surfaces. Pyopericarditis was present, with a thick layer of lymph on both visceral and parietal surfaces. The mitral valve showed old endocarditis. There was myocardial degeneration. On opening the abdomen general diffuse peritonitis was found. There was a pocket of pus between the liver and the diaphragm, just to the right of the coronary ligament. The other organs were normal. Films of the pus from the abdomen showed diplococci morphologically identical with the pneumococcus. The pus from the empyema grown on blood serum gave a Gram-positive diplococcus indistinguishable from the pneumococcus.

CASE 2.—A boy, one year and five months old, had a swelling over the left trochanter which the mother had noticed four days before. On examination the child was found to be a small wasted infant; the temperature was 101·6° F., the pulse was 156, and the respiration was 40. There were no physical signs in the chest. There was a large fluctuating swelling of the size of a man's fist over the left trochanter; the skin over it was reddened. Next day the abscess was opened and the pus evacuated. No communication with the hip-joint could be found. Three days later, as the temperature had not fallen at all (it was then 104°), the abscess cavity was explored under an anæsthetic and one or two small loculi were opened up. These ramifications of the abscess cavity penetrated the intermuscular planes of the buttock in all directions. Counter incisions were freely made. That evening the infant began to vomit and diarrhoea set in. Next day the vomiting and diarrhoea continued; no cause could be found and the child died on the following morning.

At the necropsy the lungs were tough and fibroid, possibly the late results of a pneumonia. At the lower and posterior part of the right pleural cavity there was a trace of pus between the pleural layers which were coated with lymph. The left chest, the heart, and the pericardium were normal. The abdomen was the seat of a diffuse peritonitis. The pus was thick, greenish in colour, and very rich in fibrin. The peritonitis seemed to be most pronounced around the cæcum and the ascending and descending colon. A small shut-off collection of pus was situated between the ascending colon and the cæcum on the outer side and the spine on the inner side, the mesentery and the small intestine bounding it in front. There was lymph on the under surface of the diaphragm. The intestines were normal, as were the liver, the spleen, and the kidneys. There was slight enlargement of the mesenteric glands. The abscess in the left hip was extra-articular and unconnected with bone. No diseased bone could be found. Films of pus from the peritoneum yielded a Gram-positive diplococcus morphologically identical with the pneumococcus. The same organism was found in sections of the spleen stained by Gram's stain and Bismarck brown and in the mucous membrane of a Peyer's patch, of which sections were made.

CASE 3.—A male infant, one month old, was brought to the hospital with the history that three days before he had suddenly been seized with sickness. Previously he had been healthy and had been fed on the breast only. The first day of his illness he had vomited three times and diarrhoea set in on the next day. On the morning of his admission patches of redness were seen all over the body. The child was very wasted, erythematous macules and papules covered the body, the abdomen was greatly distended, and large veins were coursing over the surface. The skin was dusky. There were severe vomiting and diarrhoea. The temperature was 100° F. on admission. The child died in three hours, the temperature having risen to 103°.

The necropsy showed congestion of the lungs; otherwise the thorax was normal, as were the brain and ears. The abdomen was full of thin, yellowish-brown pus, and in places there were quantities of semi-solid, greenish, jelly-like pus. Peyer's patches were a little swollen but there was no ulceration of the bowel. The liver was somewhat fatty but the other organs were normal. Pus from the peritoneum stained by Gram's stain and counter-stained by Bismarck brown showed numerous Gram-positive diplococci morphologically identical with the pneumococcus.

CASE 4.—A girl, aged five years, had had nausea and pain round the umbilicus ten days before admission, with difficulty in micturition, but no scantiness of urine. She had previously been quite well and had never had scarlet fever. There had been a little vomiting for two days. Seven days before admission the feet and legs began to swell and the swelling had been since increasing and spreading upwards. The bowels had been constipated. On the morning of admission redness of the legs and thighs was noticed. On admission at midnight she was seen to be a well-nourished girl. The face was pale, not puffy, and there was no appearance of facies abdominalis to be noted. The mucous membranes were of good colour but the tongue was very foul. The patient complained of severe pain all over the abdomen. There was great oedema of both legs, of a slightly solid character, which extended upwards in the abdominal wall rather more on the left side than on the right. The skin over the lower part of the abdomen and the thighs was red in patches. The temperature was 102° F. and the pulse was 148, not running. There were no physical signs in the chest. The abdomen was considerably distended and moved fairly well with respiration in the upper part and a little in the lower part. There was much general tenderness but the appendix region could be easily palpated and no thickening was felt. There were no dulness in the flanks and no signs of fluid. A catheter was passed and a small amount of urine was drawn off which was loaded with albumin. Nothing abnormal was felt on rectal examination. On the next morning the patient was more comfortable. The physical signs remained the same, save that now the red area comprised a circle of about eight inches diameter, with the vulva, which was a little excoriated, at the centre. The edge of the patch was definite but was not raised. The temperature was 100° and there had been no sickness. The bowels had not been opened. Calomel and salines were ordered. Milk was taken well. Later, ten grains of compound powder of scammony were ordered. During the same night the patient suddenly collapsed and died.

At the necropsy the chest was normal save for congestion of the lungs. The abdomen contained about a pint of evil-smelling fluid just becoming turbid and a small quantity of flaky yellow lymph in the left side of the pelvis. There was general peritonitis and no localised collections of pus could be made out. The genitalia and the appendix were normal. There was congestion of the intestines but no ulceration. Peyer's patches were a little enlarged. The spleen and liver were normal. The kidneys were a little enlarged. The cortex was injected and showed fatty change. The capsule stripped readily and the stellate veins were congested. The vena cava was normal. The brain was not examined (by request). Sections were cut and examined microscopically of the spleen, the kidneys, and a Peyer's patch. The kidneys were in a state of early tubal nephritis. No organisms were seen in the sections nor in those of the spleen and Peyer's patch. Films of the pus (from the yellow flaky lymph) stained by Gram's method yielded a Gram-positive capsulated diplococcus morphologically identical with the pneumococcus. The blood in the femoral vein when cultivated gave the same organism.

A search through the records of the hospital was made in order to see what other cases of the disease had been met with and resulted in the discovery of 12 cases which are briefly summarised as follows.

CASE 5.—The patient was a male, aged 16 months. He had had cough and diarrhoea for some days. He died shortly after admission. At the necropsy broncho-pneumonia was found throughout both lungs, also pyopericarditis, thick lymph on the upper and lower surfaces of the right lobe of the liver, meningitis, and double otitis media. Pneumococci were found pure in films from the brain and the pericardium and in films from the peritoneum pneumococci and bacillus coli communis (necropsy 52 hours after death). In films from the lungs there were pneumococci and other organisms.

CASE 6.—This patient was a female, aged 23 months, who had been vomiting for four days. She was admitted for severe broncho-pneumonia, of which the signs were typical. The abdomen was prominent, distended, and resistant. There was constant vomiting. The patient died a few days later, the signs being unchanged. At the necropsy both the pleural cavities contained lymph, less than one ounce of fluid being present; the pericardium was normal and the liver and

spleen were coated with lymph. There were general suppurative peritonitis and caries of the right petrous bone (there had been no aural discharge while in hospital). The brain was hyperæmic but there was no meningitis. In films from the ear and the peritoneum chains of strepto-diplococci, morphologically identical with the pneumococcus, were found.

CASE 7.—The patient was a male, aged five months. He had a sudden onset of screaming and fits. There was no diarrhoea or vomiting. He had flatulence and great abdominal distension. On admission there were râles at both bases; there was an impaired note at the right base. Cardiac dulness was increased; the sounds were pure. There was no marked tenderness of the abdomen and no tumour could be felt. The head was retracted and rigid. The patient died in 24 hours. At the necropsy there were small diaphragmatic empyemata at both bases, also pyopericarditis; the spleen was coated with lymph and there was a little turbid sero-pus in the pouch of Douglas. There was suppuration in the right middle ear, also suppurative meningitis. The pericardial pus showed pneumococci and a few staphylococci on film preparations.

CASE 8.—The patient was a male, aged 20 months. He was admitted with signs of left empyema. This was evacuated. Six days later sickness began; there was no diarrhoea. Sudden death occurred ten days after operation. At the necropsy there were some adhesions in the left pleural cavity and some lymph; the right was healthy. The pericardium was normal. In the peritoneum there were four ounces of pus and thick yellow sediment in the pouch of Douglas, lymph thickly coating the liver and the spleen; there were no adhesions between the coils of intestine. The ears were normal, also the meninges. Pneumococci were present pure in the pus from the empyema.

CASE 9.—The patient was a female, aged three years and nine months. There had been a gradual onset for 14 days. She had headache, drowsiness, and anorexia, and five days before admission she became worse, with severe pain in the abdomen. Next day she had diarrhoea and vomiting, the former for one day, and the latter had continued since. She had constipation on admission. She looked very ill, had facies abdominalis, and the knees were drawn up. There were fine rhonchi in both lungs. The heart was normal. The abdomen was very distended; there was no marked tenderness; it was very rigid, especially on the left side, and resonant all over. There were severe diarrhoea and vomiting. On the next day the patient was weaker and there was less distension. On the following day the distension practically disappeared, diarrhoea was severe, the patient was very restless, and death ensued. At the necropsy there was double empyema, three ounces of pus in all, pyopericarditis, general purulent peritonitis, thick lymph on the upper surface of the right lobe of the liver, the under surface of the left lobe, the upper and outer surfaces of the spleen, the under surface of the stomach, and the intestinal folds. Films of the pleural, pericardial, and peritoneal pus showed pure pneumococci.

CASE 10.—The patient was a female, aged four years and eight months. She had had whooping cough five months before; one month before she had had pain in the chest, dyspnoea, and wasting. On admission there were signs in the right lung which led to operation for empyema 14 days later. The abdomen was normal and the spleen was palpable. 18 days after admission the abdomen became slightly swollen and tender. The child did not improve and died a few days later. At the necropsy in the right pleura was an old empyema cavity; the lower lobe of the right lung showed croupous pneumonia. With regard to the left pleura there was semi-solid greenish lymph from the base to the apex. There was pyopericarditis (greenish pus). In the abdomen there were two ounces of turbid fluid, with flakes of lymph in places, for the most part round the liver. There was some oedema of the meninges. Cultures of the heart's blood yielded pure pneumococci.

CASE 11.—The patient was a male, aged 21 months. He had been feverish for nine days. Six days ago he vomited and had cough and delirium and cried before micturition. On admission he had cyanosis and was very ill. There were signs of fluid at the left base. Two ounces of turbid fluid were aspirated. Three days later ten ounces were withdrawn, followed by death on the next day. At the necropsy, in the left pleura there was greenish lymph and croupous pneumonia at the lower lobe of the left lung. In the right pleura there were turbid fluid and general coating of lymph. Pyopericarditis was present. There was recent lymph and roughening of the peritoneal covering of the spleen. The remainder of the peritoneum was normal. Films of the pericardial and left pleural fluids showed pure pneumococci.

CASE 12.—The patient was a male, aged 20 months. Ten weeks ago he had had broncho-pneumonia. Since then he had been wasting, had anæmia, and a cough. He was admitted with signs of right-sided empyema which was operated on and two and a half ounces of pus were withdrawn. 11 days later he had broncho-pneumonia at the right apex and two days later death ensued. At the necropsy he had broncho-pneumonia in the right lung, the empyema cavity draining well. There was broncho-pneumonia in the left lung. The pericardium and meninges were normal. In the abdomen there was half a pint of green oily pus; there were no adhesions or roughening of the peritoneum. The films of pus from the empyema showed pneumococci alone.

CASE 13.—The patient was a male, aged four weeks. The umbilical cord dropped off on the twelfth day; for a few days there was a slight discharge. On the eighteenth day there were vomiting and diarrhoea with green stools, and swelling of the abdomen. He had since vomited often, but the diarrhoea was less marked. On the twenty-sixth day swelling of the abdomen was more marked; the patient seemed in great pain and the urine was scanty. Discharge from the navel began again on the day of admission (the twenty-eighth). On admission the patient was found to be small and thin; there was no abdominal facies, the pulse was good, and there was no oedema. The abdomen was very distended, tense, reddish blue, and the veins were distended; there was a yellow discharge from the umbilicus. No tumour was felt; there was a fluid thrill. The umbilicus was protuberant. The whole of the right side of the abdomen was dull, save just above the pubes, and dulness extended one inch to the left of the middle line and was not moveable. The chest was normal. Under an anæsthetic five hours later the abdomen was resonant all over. At the operation a "cyst" presented in the wound covered with thickened peritoneum, which was incised; a large quantity of pus escaped. Death occurred on the next day. At the necropsy in the right pleura were blood-stained fluid and shreds of lymph; there was no

pneumonia in the right lung; the diaphragm was markedly cedematous. The left pleura was normal. The intestines were matted together and pressed against the spine. There were an empty potential cavity in front, little lymph, and pus in the splenic region and between the stomach and the colon—pale yellow. The umbilical vein was normal. Films of the pus showed pneumococci only.

CASE 14.—The patient was a female, aged eight years. She had had a cough for one month, with two days of vomiting, some delirium, and pain in the left side and fever. She had been worse for a week. On admission she was thin, blue, very ill, and had rapid respirations and clubbed fingers. The left chest was obviously full of fluid. The abdomen was distended and tender. Operation was performed for empyema and a pint of pus was evacuated. Diarrhoea and vomiting occurred during the next five days; on the sixth day there were distension and tenderness of the abdomen, which increased rapidly. There was constant vomiting, the face became abdominal in expression, and death followed on the twelfth day. At the necropsy there was an old empyema in the left pleural cavity; there was no pneumonia on the left side. In the right pleura was an empyema, with three ounces of greenish pus. There were general peritonitis and coils of bowel loosely adherent, covered with recent green lymph. On the under surface of the diaphragm on the left side was a thick layer of drier lymph. Film preparations of the peritoneal fluid showed the presence of Gram-positive diplococci along with other organisms.

CASE 15.—The patient was a male aged seven weeks. For two days before admission he had been restless and irritable. The day before the abdominal wall was noticed to be red. There had been no vomiting. On admission there was jaundice and the patient had a dull and earthy expression. The abdomen was prominent and cedematous in its lower half; the skin was red and glazed. There was free fluid in the abdomen. The spleen extended down to the umbilicus; a friction rub was heard over it. The liver was two fingers' breadth below the ribs. The abdomen was tender. The infant vomited twice and died within 24 hours. At the necropsy the chest was found to be normal, also the ears and the brain. There were six ounces of green turbid fluid in the abdomen, and lymph in the pelvis and over the organs. The liver was large, firm, jaundiced, and light green in colour. There was recent lymph on the spleen. The appendix was normal. Films and cultures from the pus yielded pure growth of pneumococci.

CASE 16.—The patient was a male, aged four and a half years. For three months he had had a puffy face. Five days before admission he had general cedema, pain in the abdomen, and constant vomiting. On admission he had cyanosis, great cedema, but no signs in the chest. The abdomen was distended and there was free fluid. On the next day there were impairment of the note at the left base and pain and tenderness in the abdomen; two days later death occurred after repeated vomiting. At the necropsy each pleural cavity contained about four ounces of thin pus. There was no pneumonia. In the abdomen there were 10 ounces of thin pus and there was lymph round the spleen. The kidneys were greatly enlarged and pale, the stellate veins were engorged, and the cortex was diminished. Films and cultures from the peritoneum gave pure growth of pneumococci.

ACHONDROPLASIA: ITS OCCURRENCE IN MAN AND IN ANIMALS.

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THE disease which in this country is usually distinguished by the name of "achondroplasia" has many other synonyms. Although this title, which was originally bestowed by Parrot, is not a perfect one it is commonly used, as it is shorter than some and more correct than others. An objection to it has been raised because it suggests that there is no growth of cartilage cells at all, and this is obviously a mistake, as there is plenty of cartilaginous activity but not in the direction of increasing the length of the limb bones. Kaufmann (1892) therefore suggested the term "chondrodystrophia foetalis hyperplastica"; this, though more correct, is unlikely to come into general use owing to its length. Other terms, such as "foetal rickets," "congenital rickets," and "foetal cretinism," are now no longer employed as they suggest an entirely fallacious view of the cause of the disease.

The subjects of this condition are short and stunted in their growth but as the limb bones are chiefly affected they fall under the category of micromelic dwarfs, as distinguished from microsomic, where the growth of all the bones is arrested. Judging from the statuettes of the ancient Egyptian god Pthah, many of which can be seen in the British Museum, the disease is of very ancient origin. Some of the old Egyptian metal workers are also represented as dwarfs of this type. The figure of the god Bes (Fig. 1), although very like an achondroplastic dwarf, is thought by some to show the appearances of a cretin. Charcot and Richer¹ have also demonstrated that several of the old masters have represented in their pictures dwarfs having the characters of the disease. Velasquez painted pygmies

evidently suffering from achondroplasia in some of his paintings for the Court of Philip IV. M. Poncet and M. Leriche² have gone much further, however, and have suggested that achondroplastic dwarfs represent vestiges of a race of men who lived in bygone times and have now disappeared. They regard many of the existing pygmy races as examples of atavistic return to an ancient race of dwarfs who were to be found all over the world. The Akkas and Obongos are cited as types of a "physiological, hereditary, and racial achondroplasia."

FIG. 1.

Figure of Bes.

Now, the Akkas are no doubt pygmies but are most beautifully formed and proportioned and their skeletons do not exhibit the slightest abnormality. The skull, the hands, and limb bones are all perfectly formed and do not show a trace of those changes which are pathognomonic of achondroplasia. The limb bones in particular are long and slender and not short and thick and, moreover, do not show the abnormal radio-humeral ratio. M. Poncet and M. Leriche state that these natives have a tendency to separation of the big toe from the rest of the toes and they quote this as being an approximation to the *main en trident* of achondroplasia. But in the scale of evolution the negro more nearly approaches the anthropoid apes than does the white man and the separation of the big toe is more and more marked the nearer we approach the apes and is seen in all negroes. Moreover, it is also seen to a certain extent in those who walk about barefooted. Although these authors give many other reasons in support of their theory they are not in the least convincing. Besides, to state that a disease (for a congenital deformity must be classed as such) is "physiological" is begging the question altogether, as a pathological state cannot be physiological.

Etiology.—The cause of achondroplasia is entirely unknown. Rickets, syphilis, and alcohol, all of which have been given as causes at various times, have not apparently anything to do with the production of the disorder. Cretinism also, is entirely distinct from it. Infants having the disease are frequently stillborn or die in early childhood, but probably many more reach maturity than was formerly thought. Thus, in the 34 cases which I have collected 18 of the subjects were over 17 years of age, seven were over 30 years, and two were over 40 years.

The disease is occasionally hereditary and it has been noticed that more than one member of a family may be affected (see Table III., cases 10 and 11, 16 and 17, 18 and 19). Porak³ mentions the case of a woman who had the disease and gave birth to an infant similarly deformed. Boeckh⁴ also published a case of a female dwarf evidently suffering from achondroplasia whose father and several other relatives had been dwarfs with similar proportions. In some cases, also, it has been found that one or both parents, although not actual examples of the disorder, are, nevertheless, of extremely small stature. Thus in Poncet and

² Revue de Chirurgie, December, 1903.

³ De l'Achondroplasie, Clermont, 1890.

⁴ Archiv für Gynaekologie, Band xlii., 1893.

¹ Les Difformes et les Malades dans l'Art, Paris, 1899.