attended by no mortality, and by complete restoration of health in every case; the only acute case died shortly after operation, although transfusion was repeatedly used before and afterwards.

Syphilis.—Gummata of the spleen are sometimes seen, and the diagnosis is not always made before operation. They may be associated with a negative Wassermann reaction, and they may not react to anti-syphilitic treatment. Splenectomy has been performed for this condition on several occasions. W. J. Mayo has pointed out that some cases of syphilis resist all drug treatment, and that the spleen is the site where the spirochaetes lodge and defy treatment; he showed that splenectomy would permit a rapid cure by medical means.

Cysts, Tumours, etc.—Removal of the spleen for such conditions as cysts, tumours, and splenomegaly without symptoms follows general surgical principles, and need not be discussed.

In general, it may be observed that success in this operation depends upon two factors: a careful preparation of the patient by the usual methods, especially by blood transfusion, and a reduction of the size of the spleen by application of x rays or radium; the surgical outlook may so be considerably improved. Except in cases of splenic anaemia, the technical difficulties of the operation are inconsiderable.

PNEUMOCOCCAL AND STREPTOCOCCAL PERITONITIS*

BY

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Pneumococcal and streptococcal peritonitis should be a fruitful subject of discussion, for a review of the world's literature of the past five years shows much divergence of opinion, and I think it is probable that in this gathering there will be found no greater unanimity. These diseases are rather rare, and it falls to the lot of no one to have great experience in them.

London seems to be fortunate in its comparative immunity, if one may judge of the lack of any long series of published cases and from one's own hospital experience. In the past fifteen years at the Hospital for Sick Children, Great Ormond Street, there have only been twenty cases of pneumococcal peritonitis and twenty-two cases of streptococcal peritonitis. From a ten-year record at the Children's Hospital, Toronto, Duncan has reported thirty-four pneumococcal and thirty-two streptococcal cases. Obadalek has observed in his clinic at Brünn, in ten years, fifty cases of pneumococcal peritonitis. The French, judging from recent literature, are greatly interested in this condition, although no long series of cases have been reported. It would appear that the disease is becoming more frequent in France. It is reasonable to assume that there is a variation in incidence in different nations, and indeed different localities. There are certain facts upon which all are agreed: pneumococcal peritonitis is chiefly a disease of childhood, and is more common in the female sex: in its acute diffuse form it is a very fatal disease, whatever the treatment. There are three questions of great interest:

1. How does pneumococcal peritonitis arise?
2. How may it be diagnosed?
3. How shall it be treated?

* Read in opening a discussion in the Section of Diseases of Children at the Centenary Meeting of the British Medical Association, London, 1932.

MODE OF INFECTION

There are four possible ways by which the pneumococcus can reach the peritoneum: (1) By the genital tract in females. (2) By the blood stream. (3) By the intestine. (4) By the lymphatics through the diaphragm.

There is little evidence that a transdiaphragmatic spread is more than a possibility. I believe that the genital passage is the most common source of infection, for in no other way can the vast preponderance of female cases be explained. Moreover, no one has thrown any doubt on the careful work of McCartney and Fraser. It cannot be the only path, for the disease occurs in boys—of my cases, six out of twenty were males—and even in female cases the genital passages may be free of pneumococcal infection. I am informed by Dr. Nabarro that the pneumococcus is not often found in the vaginal discharges of London children: this may explain the small number of cases and the reduced proportion of females to males in comparison with the figures of other localities.

It is difficult to estimate the frequency of a blood infection, because the pneumococcus, when virulent, tends to establish an early septicaemia. If one considers the constant presence of the pneumococcus in the respiratory passages, and the frequency with which primary pneumococcal infections of the ears, pharynx, or pleura may be followed by septicaemia and secondary peritonitis and pericarditis, it is reasonable to suppose that sometimes the pneumococcus may be carried in the blood stream to the peritoneum, where it finds a non-resistant medium. It is not too fanciful to compare it to the behaviour of the staphylococcus. This organism may gain access to the blood stream of a child from some abrasion or sore, and it selects a bone for its attack and development. The clinical features and pathological findings in certain cases of pneumococcal peritonitis lend some support to the theory of a blood-stream infection. In the female case with a definite genital focus, the signs are first lower abdominal, and the pathological changes reach their fullest development in the pelvis. But in the boy, where this route can be excluded, the signs are more general, sometimes more marked in the upper abdomen, and the pathological changes are equally spread throughout, save for the influence of gravity on the inflammatory effusion. A blood-borne infection seems to be a likely explanation of this generalized distribution. The intestinal route has several supporters, but, I believe, without much reason. At first sight it is the most attractive, for the intestines bear the same relation to the peritoneum as the lungs to the pleura, and the migration of an organism through the intestinal walls is as easy to understand as an empyema in pneumonia. In its favour an instance has been reported of pneumococcal peritonitis following perforation of the stomach, and a case of intestinal resection at the Hospital for Sick Children was followed by a pure pneumococcal peritonitis. Bréchet quotes De Quervain, who operated on an encysted pneumococcal peritonitis in a girl of 10, and five months later removed her appendix and found that it contained pneumococci and colon bacilli. Others have drawn attention to an intensification of the infection in the ileo-caecal region.

No doubt the frequent occurrence of diarrhoea in the early stages of the illness has played some part in suggesting an intestinal source. Against it is the fact that the pneumococcus is an inconstant inhabitant of the bowel, and if it were an inhabitant, there is no reason why it alone of organisms should migrate through the walls. The intestinal wall is impervious to organisms save through trauma or ulceration. The diffuse nature of the peritonitis is also against a local infection. I cannot believe that an intestinal lesion plays an important part in this disorder.

DIAGNOSIS

The clinical features of pneumococcal peritonitis are on the whole very characteristic. The illness may begin with overwhelming suddenness, but more usually there are two or three days of unexplained and indefinite malaise before the child is brought under medical supervision. There may have been some catarrhal signs: vomiting and diarrhoea and abdominal pain are early symptoms. The pain is general, of moderate severity, and not of a colicky type. The temperature is often high, higher than that associated with appendicitis. The pulse and respirations are raised. The abdomen soon becomes distended, and this distension is rather central. There is general abdominal resistance, but a much less degree of wooden rigidity than is found in peritonitis due to appendicitis. The abdomen has a doughy feel, and is everywhere tender. The child looks ill, flushed, and anxious, and there is often some cyanosis. It is said that herpes is frequent, but I have not seen this. Often the quick respirations and the general appearances suggest an early pneumonia, but examination of the chest is negative, or only shows those signs associated with a severe septicaemia.

All degrees may be found. In milder cases the abdominal symptoms and signs may be so indefinite that no diagnosis is made until two or three weeks later, when a localized collection of pus is found in the abdomen, which reveals on incision and examination pneumococcal pus.

It is sometimes possible in female cases to recognize the local origin. If seen sufficiently early, it may be possible to deduce from the symptoms and signs that the infection is primarily pelvic, spreading upwards to the whole abdomen. Tenderness and rigidity are at their maximum above the pubis, but with the ascent of the infection the signs become more diffuse.

There is a leucocytosis of between 15,000 and 40,000, and polymorphs predominate. Gibson considers the blood count to be characteristic, but other observers do not agree that it can be relied upon to differentiate a pneumococcal from a septic peritonitis. Ellison, Tidy, and Williams diagnosed a case as pneumococcal peritonitis by an agglutination test. The diagnosis from streptococcal peritonitis is exceedingly difficult and may be impossible. In my experience, streptococcal peritonitis is always more virulent in its attack and development. The appearance of the child is even more toxic, but cyanosis is absent. Diarrhoea is often present and the abdominal signs are very similar. There is the same tumescence with a doughy rigidity and general tenderness.

This differential diagnosis is of minor importance, for the same principles of treatment will apply to either of these forms of primary peritonitis. It is a very different matter when we come to consider the distinguishing features of pneumococcal or streptococcal peritonitis from the peritonitis due to acute appendicitis. It is of the utmost importance that this differentiation should be made. In advanced cases it may be impossible on physical signs alone, but in the average case I believe that a critical examination of the history and appearance of the patient enables a correct diagnosis to be made. In appendicitis in children the pain at onset is always central, even when the appendix is concealed behind the caecum or within the pelvis. Careful inquiry will usually elicit this fact. At a later stage, when the peritoneum is affected, there is always a hard rigidity, even when the abdomen has begun to be distended. Again, careful examination will show a maximum point of tenderness in the right iliac fossa wherever the appendix is situated.

I have very recently had a unique opportunity of testing the value of physical signs. Two cases were admitted on my operating day, within one hour. Both were boys.

The younger, aged 2½, had been ill for three days with abdominal pain and vomiting. His mother stated that the pain was at first above the umbilicus. He had had some diarrhoea. His temperature was 99.6°, pulse 132, and respirations 56. His breathing was a little laboured. The abdomen was distended. There was a general board-like rigidity and general tenderness. The tenderness was a little more definite over McBurney's point. The diagnosis was made of peritonitis, secondary to appendicitis, and at the operation a gangrenous perforated retrocaecal appendix was found with general peritonitis. The appendix was removed and the peritoneal cavity was drained and recovery followed.

The second patient, a boy aged 6½, had been quite well until six days before. He then complained of malaise, developed a temperature of 101°, and had several frequent and slimy stools. The following day an enlarged gland of the neck was noted, and he had a severe epistaxis. Three days before admission he began vomiting and the respiration became rapid. He complained of pain in the epigastrium, and his general condition deteriorated. On admission he looked ill. His pulse was 136, temperature 100.8°, and respiration 80. There were no signs in the chest sufficient to account for his condition. The abdomen was distended, very tender in every area, especially in the epigastrium, with general resistance not amounting to a hard rigidity. The diagnosis was made of streptococcal peritonitis, and under gas and oxygen a small incision was made to verify the diagnosis, and a tube was placed in the pelvis. Anti-streptococcal serum was given intravenously, and all measures taken to combat the infection, but the patient died the next day. At the necropsy a small amount of purulent fluid was found in both pleural cavities; there was general peritonitis, but most of the peritoneal fluid had drained away.

These cases were very similar in their appearance, and it was only the slight but definite difference in their physical signs that enabled a correct diagnosis to be made. Operation in the first case undoubtedly saved life; in the second no treatment, operative or non-operative, would have had effect. With whatever care clinical observations are made there must always remain a few cases in which an element of doubt remains, and it was for these cases that Lipshutz and Lowenburg devised peritoneal puncture. I have no experience of the method, and most recent writers, while commending it, confess that they have not given it a trial. Duncan employed it in twelve cases without accident.

TREATMENT

It is in the early severe and diffuse form of peritonitis that the problem of treatment is most open to discussion. In the subacute and chronic forms, with localized abscess, the surgical indications are sufficiently clear. It is merely necessary to open and drain the abscess and take measures to improve the resistance of the patient. In the diffuse type, without localization, the problem is that of the septicaemia. During the past few years there has been a strong veering of surgical opinion towards the adoption of a conservative attitude in the early stages. France, Canada, and America lead the way, followed less confidently by Germany, and as for Great Britain, we shall hear to-day.

The opponents of intervention urge that operation is not reasonable, because there is no intestinal leak to be stopped and no diseased organ to be removed, and because drainage cannot be effective in a diffuse peritonitis with matted coils and shut-off collections of pus. They hold with much reason that the performance of a set operation is just sufficient to break down the last resistance of the child and determine a fatal issue. In other septicaemic states, where, for instance, a blood infection has followed a cutaneous lesion or a cellulitis, incision of the primary area before localization is the worst possible treatment.

The matter is difficult to prove by statistics, for the acute types of peritonitis are very fatal whatever the treatment; and it may be that successful treatment, piloting such cases into a chronic stage with residual abscess, is merely the natural survival of the fittest.

The main argument in favour of immediate operation in all cases is the difficulty of diagnosis. It may not be possible to be certain that the peritonitis is not secondary to appendicitis. Many cases have been reported where an erroneous diagnosis of pneumococcal peritonitis has been made and a diseased appendix found at operation. In the two cases I have already mentioned which were admitted to hospital within an hour, the withholding of operation in the first case would have meant certain death from appendicitis and peritonitis; in the second case, operation made no difference to the inevitable fatal termination.

My rules of conduct would be these:

If the diagnosis is certain and if the infection as shown by the physical signs is diffuse, then operation is contraindicated.

If the diagnosis is certain and if there is reason to believe that the infection is most intense in one area—for example, the pelvis in girls—then a simple drainage operation is performed. If the diagnosis is uncertain, especially in males, then a quick laparotomy under gas and oxygen, to determine the condition of the appendix and to institute pelvic drainage, is the safer treatment.

The intra-abdominal manipulations should be reduced to a minimum. It is usually possible to realize the nature of the infection at once on opening the peritoneum. The straw-coloured odourless fluid, the bright red roughened peritoneum, the fibrinous patches, some filmy adhesions between the slightly distended coils, form a picture that cannot be mistaken.

In the absence of much exudate it might be justifiable to close the peritoneum, although I have not had the opportunity of testing this procedure.

Whether operation is undertaken or not, immediate steps must be adopted to combat the intense septicaemia. The organism should be rapidly typed in the manner suggested by Armstrong, and the appropriate serum administered. I will leave to others in this discussion, more qualified to speak, the question of the appropriate amount and frequency. Blood transfusion is considered of great value, but I must confess, in my own cases, I have found it a little disappointing. Perhaps one expected too much. Fluids must be administered in large quantities, and glucose solution intravenously is of great value. Mercuriochrome and mercury perchloride intravenously have seemed to do good in some cases.

STREPTOCOCCAL PERITONITIS

I must give a separate word to that very fatal condition streptococcal peritonitis. I have alluded to it more than once in discussing pneumococcal peritonitis, and it has many points of resemblance. The clinical features and pathological appearance are very similar, and differ only in degree: 71 per cent. of the cases were in females. Its origin corresponds very closely. I believe the vaginal route of infection is very common, and infection of the peritoneum by the blood stream accounts for all the other cases. Its septicaemic complications are very severe. In my experience, recovery with or without subacute encysted intra-peritoneal collections never occurs. Operation has no effect. In fifteen years there have been twenty-two cases at Great Ormond Street: twelve were operated upon, ten were not, but all terminated fatally.

One can only hope that, with the passage of time, the streptococcus will become less virulent, or the human immunity greater, and that the disease will become more benign. Hopefully one may recall the vast reduction in the number of cases of fulminating osteomyelitis due to the staphylococcus which have occurred in our own memory. In the severe streptococcal peritonitis, operation is not demanded, save for the exclusion of an appendix peritonitis. Serum, blood transfusion, and the administration of large quantities of fluids will be the better line of treatment.

THE EFFECTS OF ANAESTHETICS ON THE LIVER *

BY

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Attention was first directed to "chronic chloroform poisoning"¹ shortly after the discovery of anaesthetics. Since that time interest in the action of this drug on the liver has been more or less constant. Stiles and M'Donald² have reviewed the earlier literature and given their findings, and Whipple and Sperry³ have contributed most invaluable data; yet I shall not recount extensively the inceptive investigations, but rather the more recent ones concerning the effects of several chemical substances which are used in anaesthesia.

Latterly, considerable information has emanated from the Mayo Foundation on the physiology of the liver (^{4 5 6 7 et al}), and the value of functional tests of this organ.^{4 8} What with partial and total removal of the liver, heart-lung-liver and heart-lung-leg perfusions, denervation, studies on detoxication, and so forth, our knowledge of the several known vital functions of this great organ has been made much more clear and a rational evaluation given to the many tests, of which it may be said with authority that Rosenthal's⁹ is the most dependable in a relative and comparative sense.

It has been shown¹⁰ that brief periods of chloroform anaesthesia are sufficient to produce immediate and delayed toxic effects on the liver in the dog; that half an hour of chloroform causes injury which requires eight days for functional recovery, while it takes as long as six weeks for return to normal after two hours of chloroform anaesthesia; that disturbances of function could be demonstrated with the bromsulphalein test long after pigment metabolism had returned to normal; that ether produces a definite but transitory impairment of function, recovery being complete usually within twenty-four hours; and that nitrous oxide and ethylene do not produce any change in this dye test for hepatic function or any disturbance of pigment metabolism, but when there is a reduction in the oxygen supply during the administration of these gases, liver function is so deranged that it takes about six days to come back to normal. At this juncture a word may be said on the topic of cyanosis. Nothing but harm comes to aerobiotic cells through deprivation of oxygen; take, for example, the finding of Koehler, Brunquist, and Loevenhart,¹¹ that anoxaemia produces a more marked acidosis than has been seen in any other condition correlative with depression to all functional activity. This is the point at which one may put it down most axiomatically that any measure of functional perturbation in any one part of an organism is in direct proportion to the damage going on in all of the rest of that being, and, as the liver is such an important structure, it may be looked upon as a good index.

Large doses of morphine cause considerable, but transitory, impairment of function of the liver in dogs,¹⁰ but in the human being,^{10 12} with whom the therapeutic dose is from thirty to fifty times smaller in size, it is believed that no such damage occurs. Amytal, a representative of the malonyl-urea derivatives, has very little action upon liver function, comparing very favourably with ether in this respect.¹³ Tribromethanol^{14 15} produces only a mild parenchymatous degeneration of the liver after repeated administration. With this drug, fatty changes in the liver occur occasionally. When they do occur they are very slight in the normal dog.

* Paper read in the Section of Anaesthetics at the Centenary Meeting of the British Medical Association, London, 1932.