PERITONITIS IN THE NEONATAL PERIOD*

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It has generally been assumed that peritonitis occurs but rarely in newborn infants. In the 1952 Charles West lecture (Moncrieff, 1953) on infection in newborn babies the condition was not mentioned. Thelander, who wrote a classical paper on perforations of the gastro-intestinal tract in newborn infants in 1939, stated that most paediatricians never saw a case, and in 1949 Low, Cooper and Cosby could only find 100 instances in the literature since 1761, when Morgagni first reported the condition in *De Sedibus et Causis Morborum*. During the last five years, however, many cases of peritonitis in infants have been described and it has become clear that a condition which was formerly thought to be a pathological curiosity, is in fact a not uncommon cause of disease and death in the neonatal period. The pathology and aetiology are, as yet, little understood and recent publications have caused confusion by the synonymous use of terms, such as meconium peritonitis, intra-uterine peritonitis, foetal peritonitis, and peritonitis in the newborn.

This paper is presented in order to describe our experience with neonates suffering from peritonitis, to evaluate the incidence of the disease, and to

classify the various types of the condition. It is based upon the case histories of 17 newborn infants seen by us during the last three and a half years. During this period we admitted about 250 babies to the Neonatal Surgical Unit at Alder Hey Children's Hospital; 98 of these had intestinal obstruction (excluding pyloric stenosis and imperforate anus). It appears, therefore, that about 7% of all newborn infants admitted for urgent surgical conditions and 17% of all cases of neonatal intestinal obstruction are suffering from peritonitis.

Of the 17 cases under discussion, seven were classified as meconium peritonitis, and 10 as acute bacterial peritonitis. There was a marked preponderance of girls, 12, or 70%. This is surprising as it is generally stated (Table 1) that perforation of the intestinal tract during the newborn period affects boys three times more commonly than girls (Thelander, 1939).

Physiological and Pathological Considerations

Meconium Peritonitis. It is generally accepted that meconium starts to accumulate in the intestine of the 4-month-old embryo, but there is some evidence that the 3-month-old foetus is already swallowing amniotic fluid and passing it along the

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TABLE 1
RESULTS IN PRESENT SERIES

Name	Age	Weight	Finding at Operation	Operative Procedure	Result
Meconium Peritonitis:					
Zena	10 hrs.	6 lb. 9 oz.	Volvulus of jejunum	Resection and anastomosis	Recovery
John	3 days	5 lb. 2 oz.	Ileal atresia	" "	Died 14 days later
Hilary	2 days	6 lb.	Meconium ileus	" "	Died 12 hr. later
Linda	3 days	5 lb. 2 oz.	Perforation of ileum	" "	Died three days later
Brian	3 weeks	9 lb. 12 oz.	Ileal obstruction due to bands	Division of bands	Recovery
Sarah	3 weeks	6 lb.	Ileal obstruction due to bands	" "	Recovery
Joan	2 days	5 lb. 6 oz.	Jejunal atresia	Resection and anastomosis	Died four days later
Acute Bacterial Peritonitis:					
Mary	2 weeks	6 lb. 9 oz.	Abscess round terminal ileum	Drainage and ileocolostomy	Recovery
Nancy	2 weeks	5 lb. 14 oz.	Abscess in R. iliac fossa	Drainage of abscess	Recovery
Stephen	8 days	3 lb.	Perforation of stomach	Closure of perforation	Died four days later
Jean	1 day	5 lb. 7 oz.	Gangrenous volvulus of ileum	Resection and anastomosis	Recovery
Lesley	5 days	5 lb. 3 oz.	Perforation of ileum	" "	Recovery
Lorraine	3 weeks	6 lb. 8 oz.	Pelvic abscess	Drainage of abscess	Recovery
Doreen	3 days	2 lb.	Perforation of Meckel's diverticulum	Resection and anastomosis	Died three days later
Alexander	3 weeks	7 lb. 9 oz.	Abscess in R. iliac fossa	Drainage of abscess	Recovery
Angela	1 week	6 lb.	Perforated duplication of ileum	Resection and anastomosis	Died four weeks later
Michael	4 weeks	7 lb. 9 oz.	Perforated ileum	Suture of perforation	Recovery

gastro-intestinal tract (Davies and Potter, 1946). According to Keith (1933), it reaches the ileo-colic junction at four months and the rectum at five months of intra-uterine life. Meconium peritonitis can therefore theoretically occur during the last six months of pregnancy and Rudnew (1915) reported the condition in a 6-month-old foetus. Once meconium escapes into the peritoneal cavity it produces a chemical peritonitis. Numerous adhesions form, which, on microscopy contain meconium nodules and meconium-laden giant cells.

Two years ago we reported three cases of meconium peritonitis (Forshall, Hall and Rickham, 1952). We then agreed with Boikan (1930) in restricting the term 'meconium peritonitis' to those cases where 'meconium, calcified meconium, mucous droplets, foreign body giant cells, cells from the site of perforation, fibrous and fibrinous adhesions, and rarely lanugo hair, can be demonstrated in the peritoneal cavity'. Bendel and Michel (1953) defined meconium peritonitis rather similarly as a 'non-bacterial foreign body and chemical peritonitis occurring during intra-uterine or early neonatal life as a result of an abnormal communication between the bowel contents and the peritoneal cavity.' If the abnormal communication is still patent after birth the original sterile chemical peritonitis will soon become infected and a secondary pyogenic peritonitis will result. We do not think that these cases should be included under the title 'meconium peritonitis' as the clinical picture and the operative and necropsy findings are those of acute bacterial peritonitis, the presence of meconium being incidental.

Meconium peritonitis can be divided into two groups on an aetiological basis (Ramos, 1950). Group 1 includes all cases with intestinal obstruction. First, the cause of the obstruction can be in the lumen of the gut, i.e., meconium ileus, as in our Case 2, and in the cases described by Kornblith and Atani (1929) and Porter and Weeks (1915). Secondly, it can be in the wall of the gut, either an intestinal atresia, as our Cases 2 and 7, and numerous others reported in the literature (Neuhauser, 1944; Low *et al.*, 1949; Chandler, 1949) or, less commonly, an intestinal stenosis (Butler *et al.*, 1945). Thirdly, the cause of the obstruction may be outside the gut, for example, a volvulus as in our Case 1, and in cases described by Abt (1931) and Sturzenegger (1927), or it can be due to extrinsic congenital bands (Farr and Brunkow, 1925).

Group 2 comprises all those cases where there is perforation of the bowel wall, without distal obstruction. We have seen three such cases (Cases

4, 5 and 6) and numerous other examples can be found in the literature (Franklin and Hosford, 1952). There has been speculation as to the cause of intestinal perforation in the absence of obstruction. Davis and Poynter (1922) pointed out that perforation is by no means common, even in cases with intestinal atresia; the back pressure due to the intestinal block does, however, offer some explanation for a rupture of the thinned wall of the proximal intestine. In cases without intestinal obstruction one has to assume that there is an abnormality in the wall of the gut. Aplasia of the muscularis mucosa has been suggested as the cause of these perforations by von Sury (1912) and Moretti (1949), while Helbing (1908) and Paltauf (1888) postulated a primary vascular insufficiency of the affected intestine. Finally, Boikan (1930), Lattes (1943), and Maguire and Moore (1950) have brought forward evidence that a marked hypertrophy of the glands of Lieberkuhn, coupled with thinness of the intestinal wall and lymphoid hyperplasia, may be a predisposing cause of perforation. This would explain why the lower ileum is the common site of perforation as in our Case 4.

In many of the reported cases of meconium ileus no sign of a perforation can be found at operation and one has to assume that the perforation seals itself off during intra-uterine life (Peiser, 1908; Davis and Poynter, 1922; Flesch, 1925). In six of our seven cases no perforation could be demonstrated. We were, however, able to show by serial section that there were deep ulcers with complete destruction of the muscular layers in the proximal distended segment of the gut, and that in Case 1, which had a volvulus, the walls of the involved gut showed incomplete layers of musculature which were replaced by granulation tissue containing giant cells and calcified meconium. It is therefore reasonable to assume that in many instances there is a perforation *in utero* which closes before birth.

The question of trauma as an aetiological factor in the causation of meconium peritonitis has been discussed very fully in the literature. Rupture of the gut due to trauma usually occurs during and after birth, and although some meconium may exude into the peritoneal cavity, bacterial peritonitis will quickly supervene. We have seen only one case where trauma was the cause of peritonitis in the newborn, and nowadays such cases of mismanagement are rare. Russell (1928) reviewed the cases reported to be caused by trauma. Most of them were described in the nineteenth century by German workers (Zillner, 1884; Paltauf, 1888) and at that time the medico-legal aspect of the condition was widely discussed. Lee and MacMillan (1950)

in a critical review of the published cases doubted if trauma were an aetiological factor in many of them.

Whatever the cause of the entrance of meconium into the peritoneal cavity, the effect is always the same, namely, a plastic peritonitis with numerous adhesions binding down the intestine. These adhesions may cause kinking of the intestine and hence obstruction, as shown in two of our patients (Cases 5 and 6).

Acute Bacterial Peritonitis. Under this heading we have included all those cases where a gastrointestinal perforation occurred during or after birth and where no meconium was found in the peritoneal cavity. It is well known that acute peritonitis can occur in early infancy without gastrointestinal perforation. This is usually due to blood-stream infections with such organisms as streptococcus, gonococcus or pneumococcus (Gubern Salisachs, 1951). Peritonitis due to *Bact. coli* is very rarely seen as a complication of gastroenteritis. Such cases have become uncommon and only rarely come to the surgeon's notice.

Four of our nine cases came first to our notice when a large intra-peritoneal abscess had formed in the right iliac fossa or the pelvis (Cases 8, 9, 13 and 15). At operation, no attempt was made to demonstrate the site of perforation, and as all the infants survived and subsequent barium follow-through studies did not reveal any abnormality, we are not certain if any part of the gut ruptured. It might have been a perforated lower ileum, a Meckel's diverticulum, or an appendix, or due to blood-stream infection, although the age, clinical picture and localization of the abscesses are rather against this hypothesis. In the other six cases, the site of rupture was seen at operation. We found a rupture of the lesser curvature of the stomach, a perforation of a gangrenous volvulus, two ruptures of the lower ileum, a perforated Meckel's diverticulum and a perforation in an ileal duplication respectively.

In discussing acute bacterial peritonitis in the newborn, the following aetiological factors have to be considered:

PERFORATED PEPTIC ULCER. Perforation of peptic ulcers have not infrequently been encountered during the newborn period. Bird, Limper and Mayer (1941) collected from the literature 42 cases of peptic ulcers in newborn babies. Duodenal ulcers were twice as frequent as gastric ones. The presenting symptom was either bleeding or perforation. In 1953 Greene and Gose collected 20 reported cases of perforated ulcers and added two more which had been under

their care. The great Cruveilhier is often credited with having described the first case in 1829, but Siebold (1825) reported a perforated peptic ulcer in a newborn infant three years previously. The first successful operation for this condition was performed by Léger, Ricard, Léonard and Piette in 1950. Since then Kellogg, Abelson and Cornwell (1951), Brink and Keyser (1952), and Moncrief (1954) have reported further successes. Perforation of a gastric ulcer *in utero* was described by Lee and Wells (1923). Several theories about the aetiology of peptic perforations have been advanced: Wright and Scott (1950) thought that ulcers might develop because of the extraordinary high acidity of the stomach contents during the first few days of life. The gastric acidity reaches adult values during this period (Miller, 1941). Herbut (1943) postulated a defect in the gastric musculature and Russell (1940) considered trauma to be an aetiological factor. In Case 10 of our series there was a large rent in the lesser curvature which had caused gross pneumoperitoneum (Fig. 1). The obstetric history of this 3 lb. premature baby is of interest. There was a prolapse of the cord and the child was delivered as a difficult breech delivery at home. No cause for the perforation was found at operation and the defect was closed by sutures. The baby died four days after operation and at necropsy an extensive tear in the falx and massive extradural haemorrhage

FIG. 1.—Case 10: radiograph of abdomen, child erect, showing gross pneumoperitoneum.

were found. The immediate cause of death was a thrombosis of the inferior vena cava causing infarction of the liver, spleen, stomach and duodenum. It is likely that the original tear in the lesser curvature was due to the same vascular catastrophe. Our experience with this baby is of interest in view of Guthrie's (1942) theory that the anoxia associated with prolonged labour leads to devitalization of the duodenal mucosa. Our case certainly had a difficult and prolonged delivery. Intracranial haemorrhage in association with perforation of a gastric ulcer has also previously been reported by Gottlieb, Chu and Sharlin (1950).

ACUTE APPENDICITIS. Acute appendicitis is a great rarity in this age group. In 1945 Etherington-Wilson could only discover five cases in the literature and reported a sixth. He overlooked a case reported by Ch'eng and K'ang (1937). All these children died shortly after birth. Since then one more fatal case (Creery, 1953) and one case surviving operation (Meigher and Lucas, 1952) have been reported.

PERFORATED MECKEL'S DIVERTICULUM. Perforation of Meckel's diverticulum in the neonatal period has occasionally been described. Ungari and Valiani (1952) had a fatal case where the perforation was due to ectopic gastric mucosa. Another fatal case was described by Rosza and Gross (1953).

FIG. 2.—Case 17: rupture of small omphalocele. The protruded small intestine is strangulated and black.

In Case 14 of our series there was a small gangrenous area near the base of the diverticulum which had perforated. Normal intestinal mucosa was found on section.

PERFORATED INTESTINE. Perforation of the small or large intestine is the cause of the majority of cases with peritonitis in the newborn. As mentioned before, the lower ileum is the site most frequently involved. In our Cases 11 and 17 such a perforation was found and in Cases 8, 9, 13 and 15 it is possible that the peritonitis was due to a similar cause. In considering the aetiology of these ruptures, Gross and Ferguson (1952) have pointed out that the gastro-intestinal tract in newborn infants shows a greater development of its secretory and absorbing surfaces than of its musculature. In Case 11 there was a marked thinning of the intestinal wall around the site of perforation. Defects in the muscular coat of the intestine have been considered as the cause of some neonatal intestinal perforations, especially when associated with increased intestinal pressure due to partial obstruction (Qvigstad, 1950).

Trauma as a cause of perforation of the intestine has already been discussed under meconium peritonitis. In Case 17 of our series trauma may have been an aetiological factor.

This baby was perfectly well until he was 8 days of age, when the shrivelled remnants of the umbilical cord dropped off. It was then noticed that a loop of bowel protruded through a small defect at the umbilicus. By the time the child was admitted to our unit, the whole of the small intestine had prolapsed. It was black and appeared not viable (Fig. 2). As it was obviously impossible to resect the whole length of intestine, it was returned to the abdomen and the defect was repaired. To our surprise, the child made an uninterrupted recovery and was discharged 10 days after operation. Two and a half weeks later the child was readmitted as an emergency with a history of vomiting for 12 hours. On examination he was very shocked and cyanosed, the respiration was grunting and the abdomen was enormously distended and silent. Some mucus was passed per rectum. A radiograph of the abdomen revealed free gas under the diaphragm. At operation the abdominal cavity was found to be filled with faeces and pus and numerous dense adhesions had formed between the coils of intestine. There was a rent, half an inch long, in the wall of the lower ileum. The rent was sutured transversely and the abdomen closed. A biopsy from the edge of the intestinal defect revealed no evidence of specific infection or thrombosis on microscopy. The child made a good recovery.

In retrospect, it seems possible that after the first

FIG. 3.—Case 16: perforation in a duplication of the ileum. The probe points to the site of perforation.

operation the partially devitalized area of gut became surrounded by adhesions. Before the second operation these adhesions may have been ruptured by minimal trauma, such as lifting the child or compressing the abdomen.

Perforation of a duplication of the intestinal tract is another rare cause of acute bacterial peritonitis. Only one of the 68 cases of duplication reported from the Boston Children's Hospital had perforated.

FIG. 5.—Case 16: the excised segment of ileum with duplication. The perforation can be seen on the right side.

Resection was performed and the child died (Gross, 1953). We have quite recently seen a case with a duplication of the lower ileum about 1 ft. in length. The proximal end of the duplication ended blindly, while the distal end opened into the main lumen of the ileum. There was a round perforation of about 3 mm. in diameter (Figs. 3 to 6). The child, a girl weighing 5 lb. 9 oz., had been quite well for one week after birth. She then started vomiting and

FIG. 4.—Case 16: the mesentery of the ileum and its duplication have been divided. The perforation is marked by the probe.

FIG. 6.—Case 16: the anastomosis between the proximal and distal part of the ileum has been completed.

six hours later collapsed; she was resuscitated. Eighteen hours after the onset of the illness straight radiographs of the abdomen showed multiple fluid levels. There was no gas under the diaphragm. She was admitted to our unit 24 hours after the first vomiting. On admission she was very shocked and toxic with a high temperature. The abdomen was distended and there was definite tenderness in the right iliac fossa. A provisional diagnosis of bacterial peritonitis, probably due to intestinal perforation, was made. At operation, the whole length of the ileum with its duplication was resected and an end-to-end anastomosis was performed.

PYOGENIC PERITONITIS. Finally, pyogenic peritonitis may occur secondary to gangrene of the bowel as in our Case 10, in which there was a gangrenous volvulus of the mid-ileum; a similar case was described by Arnheim (1945).

BILE PERITONITIS. This is only mentioned in order to complete the picture. It is excessively rare; I could only find one case reported in the literature (Byrne and Bottomley, 1953). The peritonitis was due to a ruptured choledocus cyst.

The aetiology of peritonitis in the newborn is summarized in Table 2.

Diagnosis

The signs and symptoms of meconium peritonitis are those of intestinal obstruction in the newborn. In cases associated with atresia of the gut, the clinical picture will develop shortly after birth, but in cases where the obstruction is due to strictures or bands symptoms might not come on for some time. Two patients in our series, Cases 5 and 6, had no trouble until they were 3 weeks old. Bile-stained vomit, abdominal distension and constipation are

the presenting symptoms and their severity depends upon the site and completeness of the obstruction.

With a patent intestinal perforation at birth, the symptoms of acute bacterial peritonitis will soon supervene. Shock, cyanosis, rapid, grunting respiration, abdominal distension, bile-stained vomit, oedema of the flanks and scrotum, and occasionally in the absence of obstruction, the passage of blood and mucus per rectum, are described by Thelander (1939). Not all these symptoms may be encountered in the same patient, but some are always present. Shock is very marked when the perforation is recent, especially in small babies. Cases 10 and 14 in our series, weighing 3 lb. and 2 lb. respectively, were good examples of infants suffering from profound shock. Abdominal distension with or without visible peristalsis, was observed to a greater or lesser extent in all our babies. Rapid, grunting respiration and cyanotic attacks are usually secondary to abdominal distension causing pressure on the diaphragm, but may also be due to inhalation of vomit. In three children, Cases 10, 12 and 17, absence of the liver dullness due to pneumoperitoneum was noticed. Oedema of the flanks and genitalia was observed in four infants, Cases 8, 9, 13 and 15. In two of them, the oedema was associated with a blotchy erythema of the abdominal wall. We have not encountered a neonate suffering from peritonitis who passed blood per rectum, but Cases 13 and 17 passed quantities of mucus before the pelvic abscess was evacuated through the rectum. In the last four cases under discussion, the infection localized as a large intraperitoneal abscess and a mass could be felt in the right iliac fossa in two cases. The palpation of this mass clinched the diagnosis.

Radiology can help in the diagnosis of these conditions and a straight radiograph of the abdomen

TABLE 2
PERITONITIS IN THE NEONATAL PERIOD

(1) Meconium Peritonitis		if perforation patent after birth	(2) Acute Bacterial Peritonitis	(3) Bile Peritonitis
Group 1 with intestinal obstruction (i) In lumen of gut, i.e., meconium ileus (ii) In wall of gut, i.e., atresia, stenosis (iii) Outside gut, i.e., volvulus, hernia, bands			(i) Acute appendicitis (ii) Perforated Meckel's diverticulum (iii) Perforated peptic ulcer (iv) Perforation of intestine (v) Gangrenous bowel (vi) Trauma (vii) Bloodstream infection (viii) Infected haemoperitoneum (ix) Transmural infection	
Group 2 without intestinal obstruction (i) Defect in muscularis (ii) Vascular accident (iii) Hypertrophy of glands of Lieberkuhn

FIG. 7.—Case 12: perforation of the ileum. The radiograph in the erect position shows air under the diaphragm.

often reveals the nature of the condition. Kirchhoff in 1932 was the first to describe a pneumoperitoneum when radiographing a newborn baby. It is obvious that only cases with a patent intestinal perforation will show free gas in the abdomen. Three of our cases were diagnosed on this radiological finding (Figs. 7 and 8).

In meconium peritonitis the extruded meconium frequently calcifies within the peritoneum. Litten (quoted by Boikan, 1930) stated that this calcification can occur within 24 to 48 hours. This phenomenon can be reproduced experimentally by introducing meconium into the peritoneal cavity of rats (Rubovits, Taft and Neuwelt, 1938). Calcified masses can occasionally be observed in radiographs of the abdomen (Fig. 9). This diagnostic radiological sign was first recognized by Neuhauser in 1944. As we have previously pointed out (Forshall, Hall and Rickham, 1952), calcification is often so slight that it cannot be seen on a straight abdominal radiograph, although it can be demonstrated in x-ray films of the excised tissues.

Rarely, intra-abdominal calcification is discovered accidentally in babies not suffering from intestinal obstruction. Olnick and Hatcher (1953) report the case of a baby of 2 months of age whose radiographs showed calcification in the abdomen and scrotum. The diagnosis was confirmed by scrotal biopsy.

Calcification may occasionally occur in meconium within the lumen of the bowel, the peritoneum being unaffected (Camp and Roberts, 1949). The differential diagnosis can only be made at operation.

A straight radiograph of the abdomen may also be helpful in those cases where a mass can be felt on palpating the abdomen. In Case 15 of our series there was some doubt if a palpable mass was an enlarged, displaced kidney. An intramuscular pyelogram showed normally functioning kidneys and a soft tissue shadow displacing the gas-filled intestine (Fig. 10).

Treatment

The treatment of peritonitis in the newborn is the treatment of the underlying condition. This is not the place to discuss treatment in any detail, as it would involve discussing the management of all types of intestinal obstruction in the neonatal

FIG. 8.—Case 14: perforated Meckel's diverticulum. The radiograph of the abdomen in the erect position shows a small air bubble between the liver and the diaphragm.

FIG. 9.—Case 7: Straight radiograph of the abdomen showing intra-abdominal islands of calcification.

period. It is hardly necessary to stress that shock, dehydration and electrolyte disturbances must be corrected if these small infants are to survive operation. Most of them need intravenous therapy and continuous gastric suction for some hours pre-operatively until their blood chemistry has come back to normal and their general condition has improved. At operation, bands or adhesions should be divided if they cause demonstrable obstruction. Gastric perforations should be closed by suturing the defect, but when encountering intestinal perforations, we prefer resection of the affected loop of gut and end-to-end anastomosis. Intestinal atresia, or stenosis, should also be treated by wide resection of the affected segment and end-to-end anastomosis. The numerous intraperitoneal adhesions may prevent a thorough inspection of the distal intestine and produce subsequent intestinal obstruction, but we

did not attempt to separate these bands at operation as it was felt that this would greatly increase operating time, produce a considerable amount of bleeding and shock, and lead to the formation of new adhesions. We have usually thought it wiser to close the abdomen after resection and anastomosis had been completed. In those cases surviving operation, no further obstruction developed.

Where there is a large intraperitoneal abscess, this should be drained by the most convenient route. The abscess wall should not be disturbed and we believe it is unwise to search for the site of perforation. There is, of course, danger of a faecal fistula developing subsequently if this treatment is employed. One of our four cases of abscess developed such a fistula post-operatively; this closed spontaneously after a week. The three cases which were treated according to this principle had a very satisfactory convalescence. In Case 8 no mass was felt pre-operatively and the baby was operated upon for intestinal obstruction of unknown origin. At operation, an abscess surrounded by coils of lower ileum was found in the right iliac fossa. The

FIG. 10.—Case 15: radiograph of the abdomen showing a constant shadow in the right iliac fossa which displaces the gas-filled intestine. It was found at operation to be an intra-peritoneal abscess in the right iliac fossa.

abscess was drained, but the densely adherent loops of gut were still obstructed. Rather than dissect out the adhesions, it was felt that a short-circuiting side-to-side ileo-transverse colostomy offered the best chances for survival. The child made an uninterrupted recovery. She has been followed up for over three years and has no further symptoms.

Post-operatively, a prolonged period of ileus is to be expected. Intravenous therapy, continuous low-pressure gastric suction and vigilant control of any changes in the blood chemistry will, in our experience, overcome this dreaded complication.

Mortality

In this series of 17 cases there were seven deaths, a mortality of 41.2%. This high figure compares unfavourably with a mortality of 19.8% in 81 cases of intestinal obstruction without peritonitis in newborn babies operated upon during the same period of three and a half years. It appears, therefore, that peritonitis in the newborn carries an extra risk and is, in our experience, next to meconium ileus, the most fatal of the intra-abdominal catastrophes in this age group.

The causes of death were as follows: three children died of bronchopneumonia. One of them had, in addition, a congenital malformation of the heart and another was very premature, weighing only 2 lb. The causes of death in the other four were gastro-enteritis, overwhelming toxæmia, thrombosis of the inferior vena cava and intestinal fistula producing inanition. In retrospect, two of these deaths might have been prevented, if at the time we had known more about the correct post-operative management of these infants.

In conclusion, we should again like to stress that peritonitis in the newborn is not a very rare condition. It is still frequently not diagnosed during life and only discovered at necropsy. In a survey of neonatal deaths in Liverpool during 1949 several records of such cases were found (Rickham, 1952). Once diagnosed, the condition is curable by operation and should be associated with a reasonable chance of survival.

I should like to thank the paediatricians of the Liverpool region who referred these cases to us and whose vigilance in detecting surgical emergencies in newborn babies has played such a big part in lowering our operative mortality.

I should like to acknowledge Miss I. Forshall's help in preparing this paper. We collaborated closely in

treating these infants and Miss Forshall performed half the operations.

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