MECONIUM PERITONITIS DUE TO A HOLE IN THE FOETAL INTESTINAL WALL AND WITHOUT OBSTRUCTION

BY

ALFRED WHITE FRANKLIN, M.B., B.Ch. F.R.C.P.

AND

JOHN P. HOSFORD, M.S., F.R.C.S.

(From the Departments of Paediatrics and Surgery, St. Bartholomew's Hospital, London)

The presence of meconium in the foetal peritoneal cavity has long been recognized as a cause of intense chemical peritonitis. The visceral peritoneum becomes thickened to form a membrane encasing the intestines; in it and in the thickened parietal peritoneum calcium is deposited, recognized radiologically (Neuhauser, 1944) and histologically (Falkenheim and Askanazy, 1892). In some cases there is a plastic peritonitis, in others gross ascites. Such changes are not infective, but infection is added when the normal intestinal bacteria appear on the third or fourth day of life. There is a mystery about the mechanism by which the meconium reaches the peritoneal cavity. Rupture of the intestinal wall does occur in both neonate and foetus in association with obstruction. Thelander (1939) has collected many reports of perforations at all levels of the gastro-intestinal tract. At the end of the proximal segment, at the lowest part of the normally functioning gut, there may be dilatation with or without muscular hypertrophy, and this whether the cause of the obstruction be atresia, stenosis, bands or aberrant arteries, or Meckel's diverticulum, malrotation with kinking of the gut, inspissated meconium, or paralysis of Hirschsprung type (functional obstruction). Sooner or later, without relief of the obstruction this segment perforates. A neonatal perforation, occurring after the arrival of intestinal bacteria, causes bacterial peritonitis and pneumoperitoneum with signs of severe shock leading to death of the baby.

Foetal perforation causing meconium peritonitis can be understood if it is accepted that the foetus swallows liquor amnii and the foetal intestinal wall moves in peristaltic waves. The statement made in 1915 by Rudnew (quoted by Low *et al.*, 1949), who collected 40 cases, is that in only half of them was there evidence of obstruction and, whether this proportion is accurate or not, a hole may be found without any obstruction. But why should there be a hole in the wall of what is apparently a normally formed and normally acting intestine? The mystery is further deepened by two more observations: cases of meconium peritonitis have been reported in newborn babies in whom the subsequent post mortem examination has failed to show any hole through which meconium could have escaped (Skiles, 1928); and in some cases, with a hole but without evidence of obstruction by inspissated meconium, pancreatic fibrosis is proved or suspected, as in Case 2 below. A full explanation to account for all these observations is still lacking. It is possible that a foetus with meconium ileus may by vigorous peristalsis rupture the gut and relieve itself by passing the meconium into the peritoneal cavity, and that the perforation may even heal in some cases before birth.

All cases of meconium peritonitis should be explored at the earliest convenient moment, after proper preparation of the baby, by corrective treatment of the fluid balance, vitamin-K injection, and, in the presence of gross ascites, slow abdominal decompression. The hole must be carefully sought and repaired. The surgeon must not relax because he knows that a hole is not always present. Congenital anomalies of the gut must be suitably corrected. Pancreatic efficiency must be tested by examination of stools and duodenal juice. There seems good reason to hope that with such an approach the majority of these babies will survive.

Diagnosis

The first problem is one of diagnosis. The picture may be typical of neonatal intestinal obstruction. In the cases reported here massive abdominal enlargement was the presenting symptom. Such enlargement, sometimes causing obstruction or delay in delivery, could be due to the presence of a solid tumour, such as a kidney; but dullness and a fluid thrill indicate fluid. Whether a large fluid-filled cyst or viscus (such as the bladder) can give the same clinical picture we cannot say. A scrotal swelling with an impulse communicating with the peritoneal cavity is presumptive evidence of free fluid.

The possible causes of ascites at the time of birth are meconium peritonitis or chylous ascites, but a bladder vastly distended with urine may be difficult to distinguish. Purulent peritonitis, while theoretically possible, must be extremely rare. A case of so-called foetal appendicitis diagnosed by a coroner (Jackson, 1904) does not read convincingly. Hill and Mason (1925) described as prenatal appendicitis a fatal case of meconium peritonitis due to perforation of the proximal part of the appendix. As the peritoneal fluid was sterile the case can hardly be regarded as one of infection. The gut is expected to remain free from bacteria from the time of birth until some time after 48 hours; and diagnosis and treatment should be carried out during this period.

Two simple tests should help differential diagnosis—aspiration with examination of a sample of the fluid, and a plain *x*-ray film of the abdomen, particularly the lateral view. In meconium peritonitis the fluid is thick, albuminous, heavily bile-stained, and contains fat, cholesterol, and epithelial squamae on microscopy. In chylous ascites the fluid appears milky-white and has a chemical composition resembling chyle. Urine should be recognized on chemical testing.

The *x*-ray film in meconium peritonitis shows calcification as a fine tracery. The extent of aeration of the gut is also seen, but within the first 24 hours, unless one loop of intestine is grossly dilated, obstruction cannot be diagnosed. If the air cannot be seen in the rectum in the lateral view at or after 24 hours an obstruction in the intestinal tract is presumed to be present.

Two cases of meconium peritonitis with ascites, without intestinal obstruction, successfully diagnosed and treated, are described in some detail. In both, examination of the aspirated ascitic fluid established the diagnosis. A communication was assumed to exist between the lumen of the gut and the peritoneal cavity which required immediate closure before the arrival of bacteria in the bowel. What was uncertain before operation was whether the gut was normal or congenitally obstructed, and when and why the hole had appeared. The record of Case 1 has been published under the charge nurse's name as the first of its kind successfully treated in England (Gover, 1950).

Case 1

A male child was delivered normally at home, July 19, 1950, by Dr. Lucy Elliott. A normal boy had been born two and three-quarter years before, and the mother had been well during this second pregnancy. There was some delay in the second stage and after the birth of the head. The baby breathed spontaneously and the colour rapidly became

pink. After a stitch had been put in the mother's perineum the baby was again examined, and the colour found to have changed to a greyish pallor. Respiration was very rapid and the abdomen extremely distended. The baby was brought immediately to St. Bartholomew's Hospital and admitted; aged 2 hours.

On examination the baby was cyanosed and breathing rapidly, the abdomen was grossly distended with obvious superficial veins on the anterior wall, dull to percussion, with a fluid thrill. A diagnosis was made of free fluid or possibly fluid in a large cyst or dilated viscus. The abdomen was aspirated and 5 ml. of heavily bile-stained fluid removed from the right iliac fossa. In the fluid were seen red and white blood corpuscles, lipoid - containing phagocytes, a few cholesterol crystals, and "Maltese cross" cholesterol esters. There were no visible organisms, and cultures were sterile. Fouchet's test and the delayed direct van den Bergh reaction were positive. There was no tryptic activity, but a trace of amylase and much protein. Abdominal *x*-ray films at 5 hours (Figs. 1 and 2) showed areas of calcification characteristic of meconium peritonitis. The stomach and part of the intestine contained air, which had not progressed through to the rectum, an appearance normal for the age of the baby. The character of the fluid and the *x*-ray evidence of calcification established the diagnosis of meconium peritonitis, and a communication between the gut lumen and the peritoneal cavity was assumed.

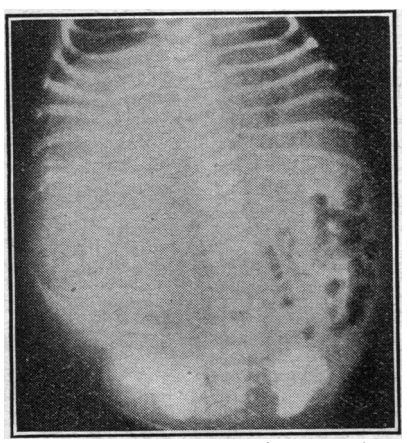

FIG. 1.—Case 1. A.P. radiograph taken when the patient was 5 hours old.

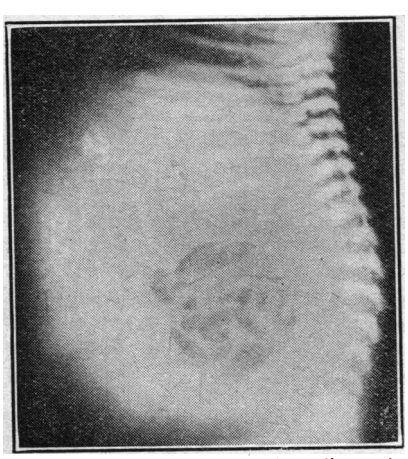

FIG. 2.—Case 1. Lateral radiograph taken when patient was 5 hours old.

Treatment and Progress

The baby was given "synkavit," 2.5 mg. intramuscularly, distaquaine penicillin, 150.000 units daily, and streptomycin, 30 mg. six-hourly. A cannula was left in the peritoneal cavity through which 350 ml. of fluid slowly drained in eight hours. At 24 hours the abdomen was opened (J. P. H.) through a right paramedian incision, the baby being anaesthetized with ether and oxygen. A large quantity of thick brown fluid was drained away and flakes of lymph and some small solid pieces were swabbed out, leaving the large peritoneal cavity as empty as possible. The parietal peritoneum was grossly thickened by organizing lymph, and in places felt quite rough from the deposit of calcified material. The intestines formed a relatively small mass in the left hypochondrium firmly matted together by and covered over with thick fibrin. In the middle of the mass at a level above and to the left of the umbilicus could be seen a circular hole with mucous membrane pouting out of it. The intestines were matted so firmly together that it was impossible to decide into what portion of the intestinal canal this hole opened. The latter was closed with fine catgut in two layers, the deeper being continuous and the superficial interrupted. The abdomen was closed in layers without drainage. A subcutaneous infusion of N/5 saline with "hyalase" was started. The baby's condition at the end of the operation was very good.

The second day passed uneventfully On the third day feeds of water and then expressed breast milk were given with a Belcroy feeder. The subcutaneous saline was stopped after 48 hours, the streptomycin after six days, and the penicillin after seven days, when the stitches were removed. The baby passed some pale meconium on the third day, followed by a little dark blood. Thereafter bowels were opened three to six times daily. On the second and third days after operation streptokinase (2 ml.), kindly supplied by Dr. I. A. Cathie, was injected into the peritoneal cavity.

The weight on admission was 5 lb. 3½ oz. (2.4 kg.), including ascitic fluid. The baby was discharged home on the fourteenth day, fully breast-fed and weighing 5 lb. 8 oz. (2.5 kg.).

The baby's progress has since been entirely normal. At the age of 7 months he weighed 20 lb. (9.1 kg.), was on a normal diet for his age, and appeared to be developing normally. A barium follow-through examination was reported as normal; calcification was still visible in the right hypochondrium. At 21 months he weighed 28 lb. (12.7 kg.).

Stool at age of 2 years gives complete digestion of the standard gelatin substrate at a dilution of 1 in 1,600. On microscopy a wet film of the stool appears normal. These findings rule out pancreatic fibrosis in this case.

Case 2

This child, a male, was born at Queen Charlotte's Maternity Hospital, on September 13, 1951. The mother had previously had two normal girls. She was of rhesus-negative blood group without antibodies. The second stage of labour (L.O.A. presentation) was delayed unaccountably. This delay was found to be due to gross distension of the abdomen. The birth weight was 8 lb. 15½ oz. (4.1 kg.), and the baby appeared cyanosed and distressed; the respiration rate was 80 a minute and the pulse 150. The abdomen was very large, dull to percussion, with a fluid thrill. The scrotum was large, containing fluid which communicated with the abdominal cavity. There was a soft swelling superficial to the abdominal muscles in the left iliac fossa, the nature of which remains unknown, but which had disappeared by the age of 5 months. The lower limbs were not oedematous. An abdominal *x*-ray film showed a minute speck of calcification in the right hypochondrium. The baby was admitted to St. Bartholomew's Hospital at the age of 4 hours. Aspiration of thick bile-stained fluid from the right iliac fossa confirmed the diagnosis of meconium peritonitis.

Treatment and Progress

Intramuscular injections were given of synkavit, 5 mg., distaquaine penicillin, 150.000 units repeated daily from September 13 to 20, and streptomycin, 125 mg. daily in four doses (September 13–23). By slow drainage through a cannula 14 oz. (400 ml.) of bile-stained fluid was removed from the peritoneal cavity. This fluid is described as yellowish brown and glutinous, alkaline to litmus, specific gravity 1020. Microscopical examination showed some red and some white blood corpuscles, epithelial cells, mucus, fat globules, and occasional plaques of degenerate squamous epithelium. Fouchet's test for bilirubin, and the direct delayed and the indirect van den Bergh reactions were strongly positive. Fat and a trace of cholesterol were present. Protein was calculated at approximately 4.7 g. per 100 ml. (including mucus); chloride (as NaCl) 550 mg. per 100 ml.; urea 330 mg. per 100 ml. Cultures were sterile.

At 24 hours the abdomen was opened (J. P. H.), the baby being anaesthetized with oxygen and ether. The intestines

were found matted together with bile-stained fibrin. The gut was normal in size for the age of the baby. There was nothing to suggest any obstruction. No hole could be found, and after a search the abdomen was closed without drainage. The post-operative condition was good, but it deteriorated on the fifth and sixth days. The abdominal circumference on the latter day at the level of the umbilicus was 18½ in. (46 cm.). An abdominal *x*-ray film (Fig. 3) showed a fluid level. Gas and bile-stained fluid were withdrawn from the peritoneal cavity. On culture this fluid grew *Bact. coli* and *Str. faecalis*, both sensitive to chloramphenicol.

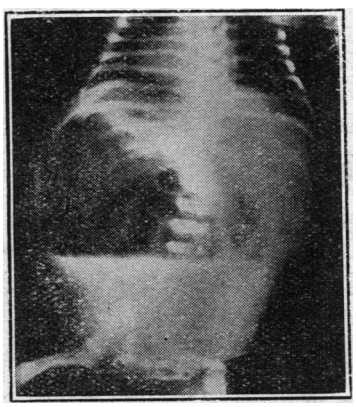

FIG. 3.—Case 2. Radiograph taken aged 6 days, showing a fluid level.

On the seventh day (September 20) the abdomen was reopened through a new incision, and this time a hole similar to that in Case 1 was found; this was sewn up and the abdomen cleaned up and closed without drainage. Four days later the lower part of the wound broke down and discharged faeces for three weeks. Chloramphenicol was given by mouth in suspension from September 21 to October 8, 125 mg. daily in three doses, and 62 mg. a day from October 8 to 15.

The mother attempted breast-feeding and seemed to provide enough breast milk, but the bowels were opened from six to nine times daily and there was no gain in weight. The weight, which had dropped to 6 lb. 11 oz. (3 kg.) on September 26, had increased only to 7 lb. 2 oz. (3.2 kg.) by October 20. Although the motions were loose they were neither fatty nor offensive. Tests for trypsin were negative in the stools (October 22) and in duodenal juice (October 25). Although these findings do not make certain a diagnosis of pancreatic deficiency or fibrosis at this age, the baby was given pancreatin granules (2 g. a day), and began to gain weight at once. He was discharged home on November 5, fed partly breast and partly a half-cream milk, aged 7½ weeks, and weighing 8 lb. 4 oz. (3.7 kg.) with the wound completely healed.

An abdominal *x*-ray film at this time showed marked calcification (Fig. 4) of the parietal peritoneum, as compared with the doubtful speck seen seven weeks earlier.

He was weaned from the breast at 4 months. Re-examined at 5 months of age, he was a well-grown normal baby weighing 18 lb. (8.2 kg.), with a very large appetite, and taking 12-oz. (340-ml.) feeds of full-cream dried milk four times a day. The duodenal juice contained no trypsin, and, although progress has been so satisfactory, the diagnosis of fibrosis of the pancreas has been made.

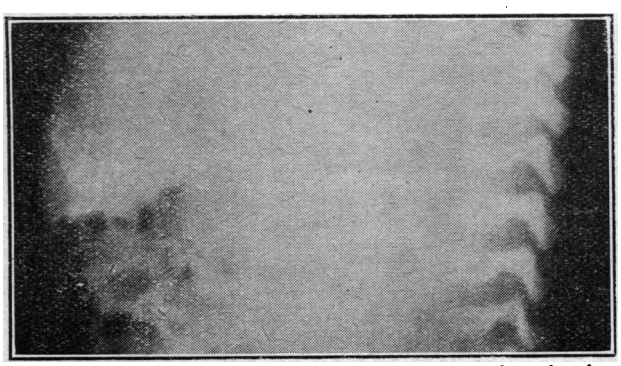

FIG. 4.—Case 2. Radiograph taken aged 7 weeks, showing calcification of parietal peritoneum.

The difficulty in this case arose from the failure to find a hole in the gut and to sew it up at the time when the gut contents were sterile. The control of *Bact. coli* infection by chloramphenicol certainly contributed greatly to the eventual recovery of the baby, but comparison of the two cases points to the importance of early diagnosis and to treatment within the first 48 hours. We have decided, should no hole be apparent on the next occasion, to pass a soft rubber tube into the stomach and slowly bubble oxygen through, if necessary filling the abdomen with sterile normal saline, on the principle used to find small punctures in bicycle tyres.

Conclusion

Perforation of the intestine in the foetus in the absence of any recognizable obstruction, with escape of bile into the peritoneal cavity, seems in these cases to have led to intense chemical peritonitis. The intestines were found embedded in a thick fibrin layer, so that it was impossible even to recognize in what anatomical part of the intestine, large or small, the lesion existed. In the first case there was marked calcification in the parietal and visceral peritoneum at birth; in the second this appeared during the course of observation, though Dr. Rohan Williams was satisfied that the *x*-ray film at birth showed a minute but definite amount. In neither case was there bacterial peritonitis at 24 hours, but in the second this occurred later. In neither was there clinical evidence of obstruction at the time of the peritonitis or at any later period. The first patient appears to be a normal boy. The second is a case presumably of pancreatic fibrosis. Why there was a hole in the intestine in either case is difficult to understand.

The cases are reported because circumstances enabled the diagnosis to be made within a few hours of birth. This allowed the successful treatment of what has so often been recorded as a neonatal calamity.

REFERENCES

Falkenheim and Askanazy, M. (1892). *Jb. Kinderheilk.*, **34**, 71.
Gover, E. M. (1950). *Nursing Times*, **46**, 934.
Hill, W. B., and Mason, C. C. (1925). *Amer. J. Dis. Child.*, **29**, 86.
Jackson, W. F. (1904). *Amer. J. med. Sci.*, **127**, 710.
Low, J. R., Cooper, G., and Cosby, L. (1949). *Surgery*, **26**, 223.
Neuhauser, E. B. D. (1944). *Amer. J. Roentgen.*, **51**, 421.
Rudnew, W. (1915). Quoted by Low *et al.* (1949).
Skiles, J. H. (1928). *J. Amer. med. Ass.*, **90**, 1618.
Thelander, H. E. (1939). *Amer. J. Dis. Child.*, **58**, 371.

Every three months the Rockefeller Foundation issues a bulletin of how it has spent its money that quarter. In April–June this year the Foundation gave away about ten million dollars (about £3½m.), some of it to institutions, some to individuals for special inquiries, some to Americans, but a great deal to various European and Eastern countries. In Britain, the Burden Neurological Institute at Bristol received £9,000 for four years' research in neurophysiology, St. Thomas's Hospital Medical School £4,500 for an inquiry into the relation between human physical form and physiological function, the Institution of Civil Engineers £12,000 for training and research in public health engineering, and Cambridge University £600 to pay for an assistant in *x*-ray crystallography to Sir Lawrence Bragg. Special dollar grants went to Dr. S. C. Truelove (Oxford) to visit the U.S. and Canada to study experiments in medical education ($2,525); Miss L. P. Yule (Department of Child Life and Health, Edinburgh) to observe American methods of social work ($2,025); Dr. D. A. Sholl and Professor B. Katz (London) $400 and $460 respectively to visit neuroanatomical and physiological research centres in the U.S. The Nuffield Institute for Medical Research at Oxford received $1,800 to buy a special *x*-ray tube, the University of Glasgow $6,700 for special biochemical equipment, and the University of Leeds $2,450 for biophysical research. This was the aid for the quarter to medical research in Britain.