INTESTINAL OBSTRUCTION IN THE NEWBORN*

BY

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From The Hospital for Sick Children, Great Ormond Street, London

There are many causes of intestinal obstruction in the newborn baby. Individually none of them are common conditions and yet together they make up a group of some importance. Rickham (1952) has estimated that up to 950 babies are born in Great Britain each year with congenital defects amenable to surgery and the obstructions form a large part of these.

This subject has been well discussed in several recent articles (Gross, 1953; Jolley, 1952; Santulli, 1954; Swain and France, 1954, etc.). In this paper I wish to refer to particular aspects which I have had the opportunity of observing during the past five years, mainly at The Hospital for Sick Children, Great Ormond Street.

Diagnosis

Delay in diagnosis is still an important factor in failure to relieve these obstructions. For example, of 21 recent cases of jejuno-ileal atresia admitted to this hospital, 18 babies began vomiting on the first day of life and yet only 17 were admitted by the third day. Why should this two-day lag period still exist?

The effects of obstruction are the same as at any other age. Absolute constipation occurs but the early passage of meconium may be apparently normal while the bowel below the obstruction is emptying. There is distension, but in the early stages it is difficult to assess in the naturally protuberant abdomen of the newborn, and in duodenal obstructions may be entirely lacking in a baby who vomits easily. Vomiting remains as the most reliable early sign in the majority of cases. There are many other reasons for neonatal vomiting and this seems to be the reason why clinicians may be slow to suspect obstruction. However, bile-stained vomit such as one sees in cases of obstruction below the bile ducts is not frequent.

Two large maternity hospitals were able to discover only two cases of true bile-stained vomiting occurring in the absence of an organic cause in mature babies during a year's observation during which about 4,000 babies were delivered. In a small series of 106 babies at Queen Charlotte's Maternity Hospital, London, every vomit was charted on a special form so as to ensure that small vomits were not passing unrecorded because the nursing staff considered them trivial. As would be expected a large proportion of the babies produced some vomitus but none was green (Table 1). Eight produced yellow vomitus which has often been attributed to bile staining. Four specimens of vomit were examined by Dr. G. H. Lathe. Bile pigments (Fouchet's reagent) were absent and the spectral absorption curve of extracts of vomitus indicated that the main pigments were of a carotenoid type known to be present in large amounts in colostrum (Dann, 1936). This may explain the misconception that bile-stained vomitus is not uncommon in normal newborn babies.

A second factor encouraging delay is the apparent well-being of the obstructed baby in the early stages. The newborn baby is prepared to live on his resources for some days and does not begin to look ill until secondary vascular changes develop in the distended or volved bowel, or until vomiting has been prolonged for some days. Thus there is a reluctance to transfer a fit looking baby to a surgical unit.

Fortunately, however, the most valuable confirmatory investigation is one which can be carried out in any hospital and with negligible disturbance to the patient. Plain radiographs of the abdomen are usually sufficient to clinch the diagnosis if properly interpreted. The basic feature in most cases is the presence of fluid levels in progressively distending

* Vomiting persisted over 24 hours in eight. There were no relevant abnormalities in the babies.

**Table 1**

<table>
<thead>
<tr>
<th>Colour of Vomit</th>
<th>White</th>
<th>Yellow</th>
<th>Brown</th>
<th>Green</th>
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</thead>
<tbody>
<tr>
<td>No. of Vomiting</td>
<td>12</td>
<td>8</td>
<td>3</td>
<td>0</td>
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* A paper given at the inaugural meeting of the British Association of Paediatric Surgeons in London in July, 1954.
loops of bowel. When the presence of obstruction is confirmed, laparotomy should follow. Delay for further study can be dangerous and the exact nature of the lesions will be better diagnosed by direct observation.

Barium meals are unnecessary and may be dangerous, except perhaps, in suspected incomplete obstructions. A small barium enema may prove to be of value in Hirschsprung’s disease. I have not found Farber’s test helpful. It is only positive in complete atresias which are clearly diagnosable by clinical examination and plain radiographs, and when negative only excludes this one cause of obstruction.

One must be on guard for other organic causes of neonatal vomiting of which the chief are birth injuries of the brain and infections. Cretinism can also cause constipation to the extent of obstructive vomiting, and cystic fibrosis of the pancreas may cause constipation and vomiting without necessarily progressing to the full picture of obstruction by meconium ileus.

Persistent vomiting may also occur in oesophageal hiatus hernia at this age.

Malrotation

These conditions have been admirably described by Frazer and Robbins (1915) and Dott (1923), and Ladd’s (1932, 1933) procedure of unrotating the gut usually relieves the obstruction equally well.

When volvulus has been present for some time before birth the bowel has grown in its twisted position and its unwinding may be more difficult and will leave raw areas without complete peritoneal covering. Fig. 1 shows a peritoneal band found extending from the midline to the beginning of the jejunum. I think it is important after dividing the transduodenal bands of Ladd to continue clearing the first part of the jejunum lest obstruction at this point persist. I imagine this band to be an attempt at the formation of a ligament of Treitz in the presence of a misplaced duodenal flexure (Spencer, 1951). The ileocolic adhesion also shown is not uncommon: it may form a pocket between bowel and mesentry. One of our cases died of strangulation of a loop of ileum herniated through such an orifice. Results have been good, 12 of 15 cases in the past five years surviving, and the three deaths all in complicated cases.

Duodenal Atresia and Stenosis

Those duodenal atresias situated above the ampulla of Vater are likely to be diagnosed later as the vomit consists only of gastric contents and does not become bile stained. However, the very fact that the biliary and intestinal secretions are not lost and the fact that the lesion cannot lead to strangulation means that delay is here less serious than in atresia lower down.

More disturbing is the appallingly high incidence of mongolism in this series, 10 of a consecutive 32 cases studied by Bodian, White, Carter and Louw (1952). The condition can be recognized at birth by careful inspection of the facial, cranial, and manual stigmata. Treatment may then become a matter for ethical consideration.

Louw (1952) showed that in the earlier cases at Great Ormond Street there was a high mortality after gastro- and duodeno-jejunostomy and that this was not usually due to technical failure in the operation. It was the result of failure to manage the difficult 10 days until the anastomosis would transmit an adequate amount of milk to sustain the baby. Ehrenpreis and Sandblom (1949) greatly improved their results by the use of a transanastomotic tube to allow of early feeding. I have no personal experience of this and perhaps improved parenteral therapy should render it unnecessary, but the idea is attractive and our results still leave much to be desired—only five survivals out of 18 cases during the past five years.

Jejuno-ileal Atresia and Stenosis

Diagnosis is here straightforward but more urgent because delay causes vascular changes in the bowel wall by increased distension and eventual perforation. The results of simple anastomosis have been poor in our hands. Louw (1952) showed that in cases

![Fig. 1.—Drawing to show the binding down of the beginning of the jejunum and ileo-colic adhesion in a case of malrotation.](image)
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Treated reasonably early the main cause of death, unlike the duodenal cases, lay in the failure of the anastomosis to function.

Hoppu (1951) suggested that any primary anastomosis would inevitably be unreliable in bowel of this small calibre and that a prosthesis would be necessary to prevent plugging and functional obstruction. In experiments with baby rabbits of a comparable bowel size and structure he had a mortality of 58.3% from obstruction with end-to-end anastomosis. I repeated these experiments and found my mortality to be only 30% (Fig. 2) without the assistance of parenteral fluids or chemotherapy. The use of other types of anastomosis suggested that reasonable success could be obtained and that the results depended on the technical convenience of the method in the particular animal used rather than on the size of the aperture created.

I believe that the cause of failure in direct anastomoses in cases of atresia lies in the dilated and hypertrophied proximal bowel and not in the unexpanded distal bowel. The obstruction is an acute one superimposed on a chronic foetal one and as a result several inches of bowel proximal to the obstruction are not only dilated but also anatomically enlarged and hypertrophied. This gut contracts actively but is nevertheless ineffective in propulsion. It does not seem to close the lumen sufficiently to have a directional effect on fluid contents.

The first two cases of jejuno-ileal atresias on which I operated were both diagnosed so late as to necessitate resection of the proximal bulb of bowel for

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<tr>
<th>EXPERIMENTAL ANASTOMOSES OF ILEUM OF BABY RABBITS</th>
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<tr>
<td><strong>1. SIDE TO SIDE. 2 cm. LONG. 1 LAYER.</strong></td>
</tr>
<tr>
<td><strong>91% SUCCESS</strong></td>
</tr>
<tr>
<td><strong>2. END TO END. 1 LAYER.</strong></td>
</tr>
<tr>
<td><strong>Bowel 0.4 - 1.0 cm across.</strong></td>
</tr>
<tr>
<td><strong>70% SUCCESS</strong></td>
</tr>
<tr>
<td><strong>3. VERY OBLIQUE, BACK TO BACK. 2 cm LONG. 1 LAYER.</strong></td>
</tr>
<tr>
<td><strong>60% SUCCESS</strong></td>
</tr>
<tr>
<td><strong>4. END TO END. 2 LAYER. WITH LARGE CUFF.</strong></td>
</tr>
<tr>
<td><strong>67% SUCCESS</strong></td>
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Fig. 2.—Results of experimental anastomoses of ileum in 70 young rabbits.
incipient gangrene; both survived. The third, a case of high jejunal atresia, was diagnosed early and in excellent health and I performed a simple end-to-oblique anastomosis. All went well until the feeds were increased to full requirements when vomiting recurred. Several trials showed that the anastomosis would only pass about half the food required. A second laparotomy was performed at which the anastomosis was found to be patent although it was not functioning. A long lateral anastomosis was made around it. Nevertheless, the same trouble was experienced after operation and the baby died aged 5 weeks with a widely patent anastomosis, full expansion of the distal bowel, and yet persistence of the distension of the bowel proximal to the anastomosis.

During the baby's life the ward sister pointed out to me that during feeds the milk could be felt to enter the dilated bulb rapidly but that it stayed churning there until part was returned by vomiting. There was no paralytic ileus but the bulb was ineffective in propulsion.

Animal experiments are in progress to study this phenomenon. Fig. 3 shows the principle underlying the apparatus which I have called a 'gut pump'. The outlet is raised 1 cm. above the inlet so that the gut has to do measurable work to transmit the fluid. It was noted that the regular longitudinal contractions recorded in the usual isolated organ bath preparations had no propulsive value but that the intermittent stripping contractions in which the segment contracted down hard from end to end produced a sudden passage of fluid. Normal bowel worked best with an inlet pressure of about 2-5 cm.

The next stage was to make a hypertrophied loop of bowel by reversing a segment of ileum, restoring continuity by two end-to-end anastomoses and sacrificing the animal a few weeks later. The bowel above the reversed segment enlarged and hypertrophied as shown in Fig. 4. In the organ bath normal bowel worked best at 2-5 cm. inlet pressure transmitting 45 ml. in 10 minutes. The hypertrophied segment, although moving vigorously, was quite inefficient at this pressure, transmitting only 4 ml. in 10 minutes. If the inlet pressure was raised to 6 cm. then the normal bowel was over-stretched and failed, whereas the hypertrophied segment became most efficient, sending over 106 ml. in 10 minutes. In other words, the hypertrophied segment has adjusted itself to abnormal obstructive conditions and is inefficient when a normal intestinal gradient is restored.

The clinical and experimental observations led me and some of the surgeons at the hospital to undertake routine resection of the hypertrophied segment of bowel before anastomosis. Other surgeons per-

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* Deaths included five early post-operative deaths and one 'biochemical' death.

Three cases were not operated upon: two similar cases associated with meconium ileus, volvulus and meconium peritonitis which died during attempted resuscitation, and one case which died soon after operation on an associated oesophageal atresia. This infant also was premature, had kernicterus and other abnormalities.
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sisted with simple anastomosis and relied on careful parenteral fluid therapy to keep the baby alive until the bowel function was re-established. The results are shown in Table 2, two-thirds of the cases surviving after resection and only one-third without it.

The technique is illustrated in Fig. 5. From 15 to 25 cm. of bowel are resected and the proximal bowel sucked clear of content which may otherwise tend to inspissate and block the anastomosis. The distal bowel is inflated with saline to clear any concretions and to exclude any further atresias. Anastomosis is then performed with one layer of interrupted silk mattress sutures. The proximal bowel is cut sufficiently obliquely to make the opening 2 cm. long. The distal bowel is cut along its antimesenteric border for a like distance. The bowel is controlled by stay sutures and no clamps are used.

Aetiology

I have studied all our cases of jejuno-ileal atresia whether or not there were associated and more impressive anomalies such as meconium peritonitis. In this way I think that a progression of cases can be shown from simple complete atresia with a blind bowel end to cases with only a slight lesion of the bowel wall, and that these throw some light on the aetiology. Thirty-three cases of jejuno-ileal atresia have been admitted during the past five years.

The commonly quoted view of their origin is that

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**Fig. 5.—Illustrating technique used in 'end-to-back' anastomosis (after Denis Browne, 1951).**
layer to be lost and it is replaced by a loose fibroblastic tissue. Furthermore, other observers such as Johnson (1910) were unable to confirm the presence of such a solid stage in the intestine below the duodenum. Also we have found epithelial squames in the lumen of bowel below a complete atresia (Fig. 6). These have to be swallowed to reach the lumen and the embryo has no such squames on its skin to be swallowed until the third month (Keith, 1948) well after the time of the suggested solid stage. Farber's test is a crude one, and Emery (1952) has recently pointed out that with other staining methods a few squames could be recognized in the meconium of cases of atresia.

Another suggested cause was some vascular accident in utero, and Davis and Poynter (1922) described endarteritic obliteration of the arcades of Tandler (1900) and Forssner (1907) of persistence of the solid stage of development of the bowel mucosa. But persistence of a mucosal septum is the least common type of atresia in this region. Indeed our histological studies have shown it to be the first

![Fig. 6.—Epithelial squames in the lumen of the bowel below a complete atresia. (×570 approx.)](image)

![Fig. 7.—Bismuth oxychloride injection of vessels in the region of ileal atresia.](image)

![Fig. 8.—A microscopic nodule of meconium peritonitis on the serosa in a case of ileal atresia (also shows loss of mucous membrane). (×55 approx.)](image)

corresponding to the atretic areas in one case. The vessels of four consecutive cases were injected using a technique based on that of Barlow, Bentley and Walder (1951). I found no abnormality of the feeding vessels beyond a variation in calibre comparable to that of the bowel supplied (Fig. 7).

I suggest that a more likely cause of atresia would be an accident to the bowel after its formation, such as incarceration of the physiological umbilical hernia with resulting nipping or adhesion of parts of the intestine and a predisposition to volvulus of the mass thus formed.
To consider this hypothesis: first, can an accident to formed bowel result in atresia? One typical ileal atresia with a complete gap was found to contain the remnants of an intussusception with a gangrenous apex in its distal bowel. Further examination showed microscopic foci of meconium peritonitis similar to that shown in Fig. 8. The cause of the atresia would appear to be clear in this type of case of which others have been described. Second, does incarceration of the physiological umbilical hernia ever occur? The baby depicted in Fig. 9 was born with an exomphalos into which two open ends of bowel protruded as a spontaneous ileostomy. Around them in the sac lay meconium debris and necrotic remnants of the intervening bowel. Third, is there any evidence of abnormality in the process of reduction of the hernia and replacement of the bowel within the abdomen in these cases? The mesentry was abnormal in nine of the 21 in which it was adequately examined. Also volvulus of a part of the small intestine was present in 11 of the 21 cases. Severe foetal peritonitis was present in five cases and localized adhesions in five more. Microscopic evidence of meconium peritonitis was found in several other cases with no obvious peritonitis. Furthermore, three cases had an associated cystic fibrosis of the pancreas and yet another had long segment Hirschprung's disease with no ganglia up to the level of the ileal atresia. Both these conditions would produce foetal obstruction and a tendency to dilatation and volvulus of the bowel.

Fig. 10 illustrates a case of volvulus with changes in the wall at the site of twisting, all the layers being lost except the serosa but without actual stenosis.

Fig. 11 illustrates a case of volvulus with severe stenosis of the segment looped around the base. This phenomenon has been observed at either end of the volvulus so that the volvulus cannot be explained as a result of loading of the bowel above a performed atresia.

Fig. 12 depicts an atresia with a cord running between the lumina above and below the lesion. Such cases have shown microscopical meconium peritonitis compatible with nipping in the neck of the umbilical hernial sac causing damage to the bowel wall.

Fig. 13 depicts a complete atresia with a gap between the bowel ends. This type has also shown microscopical meconium peritonitis and some cases have had an isolated segment or segments of bowel between the two ends. Here nipping off at the neck of the sac may have caused complete separation of the bowel and in some cases the intervening segment has grown large enough to persist as a recognizable isolated mass of bowel.

A mucosal septum is rare in jejuno-ileum, but I have seen two cases outside this series. Again, such a lesion could result from external pressure causing fusion of the mucosal walls.

This concept of a causation of the jejuno-ileal atresias is only a hypothesis, but I suggest that it fits more of the observed facts better than the other hypotheses. As a practical point, such an embryologically late accident to the alimentary tract would be less likely to be associated with serious lesions in other systems. This is borne out in fact; for example, the association with mongolism found in duodenal atresias is not present. The only mongol in these 33 cases was a baby who also had duodenal atresia, and other serious lesions in other systems were absent.

Meconium Ileus

Several cases have now been added to the 14 recently published from The Hospital for Sick Children, Great Ormond Street (Nixon, 1953). All of our cases have had fibrocystic disease of the pancreas, and it seems that inspissation of the meconium is due to the abnormality of the mucus in this disease rather than to pancreatic achyilia per se.

Fortunately the severity of the intestinal lesion is not necessarily associated with similarly severe pulmonary lesions. The successful case reported is now 4 years old and she is a fit girl who has had no severe respiratory infections. Modern care of this disease can make the saving of a case of meconium ileus well worth while.
FIG. 10.—Lesion in the ileal wall at site of volvulus. Only the serous coat persists.

FIG. 11.—Congenital volvulus of part of small bowel with severe stenosis at site of volvulus shown in original position and after unwinding.

FIG. 12.—Ileal atresia with cord between lumina above and below.

FIG. 13.—Typical hypertrophied and dilated terminal bulb above an ileal atresia. This operative specimen shows the extent of the resection carried out.
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In my previous report I suggested that the immediate threat to life was perforation of the maximally dilated loop of bowel due to over-distension, in some cases, an added volvulus. The simplest means of overcoming this risk is an ileostomy which must be above the plug of meconium. Natural infection of the bowel contents will soon liquefy them and pancreatic instillations may assist.

This method was successful in the case mentioned. However, in meconium ileus we are dealing with another form of acute chronic obstruction and the hypertrophied loop may be inefficient in propulsion though viable. Hence resection with a double-barrelled ileostomy as practised by Gross (1953) would seem more reliable.

Many of our cases have had pre-natal volvulus, often with perforation and meconium peritonitis and sometimes an associated atresia. Some of these have seemed to present insoluble problems.

**Hirschsprung’s Disease**

Rapid deflation of the abdomen on rectal examination or washout may make the diagnosis obvious. If not, and the lesion is discovered at laparotomy, then we have found that in this age group the site of the cone of narrowing bowel may be misleading. During foetal life the meconium can be driven down into a long aganglionic segment so that a colostomy may be sited too low in the bowel. The management of this particular problem in Hirschsprung’s disease does not seem to be settled. Possibly an ileostomy followed by early re-operation with facilities for examinations of frozen sections may be the answer.

**Management in General**

Again, I wish only to stress certain particular personal opinions.

I believe that the use of gastric aspiration before and during transfer to a surgical unit would save some of the unfortunate pulmonary inhalation complications which seem so unnecessarily to jeopardize the chances of these babies.

Attempts at pre-operative resuscitation should not be unduly prolonged. Urgent operation is indicated in almost every case. Resuscitation should be started but can be continued during the operation. This sounds unorthodox but the babies do stand up to it. Several times I have seen some accident such as perforation or volvulus occur during the few hours devoted to pre-operative treatment so that the patient has died even before operation could be attempted. If intestinal intubation were practicable in the newborn the urgency would be lessened but gastric intubation is an inefficient means of decompressing the small bowel.

A full laparotomy is almost always used and I prefer a transverse supra-umbilical muscle-cutting incision. It makes prompt atraumatic elucidation of the pathology much easier and avoids the risk of missing the not uncommon second lesion.

Therapy with fluids should be conservative. Blood and plasma for shock and nourishment are life saving. Plasma should be given regularly until oral feeds are adequate to prevent protein depletion. This is a much easier task than to attempt to correct an established depletion. Water should be given sparingly, not more than 40 ml per lb per day for maintenance and in an electrolyte solution such as half strength Darrow’s solution which will not supply too much sodium chloride. There is a practical danger of giving too much to keep the drip running. The resulting diuresis may get rid of some of the excess water but may also deplete other components of the body fluids not so easily replaced, even if frank drowning by sodium retention does not occur.

Nor is there any need to hurry the oral feeding. The operation soon relieves the obstruction and converts the situation to one of simple starvation which a newborn baby stands well. The aim should be, as Gross puts it, ‘to keep the baby on the dry side’.

I would like to thank all the members of the medical and nursing staff of The Hospital for Sick Children, Great Ormond Street, for their cooperation and advice and for allowing me to operate on many of their patients. My particular thanks are due to Mr. Denis Browne and to Dr. Bodian, and to the Research Committee for grants which have enabled me to continue this work. My thanks are also due to Mr. Derek Martin and Mr. P. G. Cull, of the Department of Medical Illustration, for their excellent work.

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Finally, I should like to express my great debt to the late Professor Sir James Spence for many valuable discussions. Although I left Newcastle several years ago, he was never too busy to help.

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