CONGENITAL DUODENAL OBSTRUCTION: MAL- 
ROTATION OF THE INTESTINE

REPORT OF CASE *

B. S. DENZER, M.D.
NEW YORK

In this report the clinical rather than the developmental aspects of congenital duodenal obstruction will be emphasized. It seems that enough cases have been recorded to warrant giving a more definite clinical picture, and this may serve to center interest in them as clinical cases rather than as pathologic specimens. Unfortunately, the first few days of life are a veritable shadowland for diagnosis and treatment. The infant that seems barely holding on to life may be a thriving youngster a few days later, and, on the other hand, sudden and unexplained exitus of a baby apparently not very ill is an all too frequent occurrence. Similarly, at this time of life exploratory laparotomy is an adventure. It is, therefore, with some hesitancy that the effort is made to group the striking clinical features of congenital duodenal obstruction and to suggest that at least some of the cases may be brought within the reach of surgical assistance. Perusal of the literature, in addition to the study of a single case, forms the background for the discussion and conclusions.

From the clinical standpoint, the possible duration of life of infants suffering from duodenal obstruction is of first importance. As far as this is related to the anatomic anomaly, the duration of life is dependent on whether there is atresia (complete occlusion of the intestinal tract) or merely stenosis (partial obstruction). Of thirteen cases of stenosis collected by Kreuter in 1905, four patients lived for a week or more.¹ A search of the literature since 1905, probably not complete, of cases of partial obstruction in which the patients live more than a week, has revealed an even greater number.² Four infants died during the second week of life, and three during the second month; four others lived from four to nine months, one for twelve months,

* Received for publication, Aug. 15, 1922.
* From the Pediatric Service of the New York Nursery and Child's Hospital and the Pediatric Department of Cornell University Medical School.
1. Two infants lived seven days, one lived twelve days and one six months.
2. This refers only to infancy and not to those cases of obstruction discovered in later life, due to bands, anomalous peritoneal fossae, malrotations, etc.
one for eighteen months and one for two years.\textsuperscript{3} The mere duration of life is emphasized because it indicates that in some cases there is at least time for surgical intervention.

Efforts at treatment have been made. Colwell, in 1912 recorded five cases in which enterostomy had been done, although unsuccessfully. Commenting on the reports of Keith and Buchanan of infants living nine and eighteen months, he remarked, “it would seem that cases should be subjected to laparotomy, and, if possible, gastro-enterostomy should be performed.” Indeed, since then such an operation has been done successfully. Ernst, in 1916,\textsuperscript{4} operated on an infant 11 days old. There was an obstruction “at the upper side of the transverse mesocolon” and Ernst performed a duodeno-entero anterior anastomosis. Eleven months later the child was a “vigorous well developed youngster.”

The symptomatology of cases living more than a week depends on the location and degree of obstruction. When the obstruction is situated below the ampulla of Vater, the vomitus is bile stained. This is emphasized by nearly all the authors who considered the symptomatology of the cases they reported.\textsuperscript{5} Bradford believes that “vomiting occurs after the lapse of a longer period following feedings than does the vomiting caused by stenosis higher in the tract.” In nearly all cases there was difficulty in excluding pyloric stenosis, and in many cases that was the diagnosis made. Tumor was mentioned by only a few authors; it was a vague sign.\textsuperscript{6} Peristaltic waves were more frequently found. Fecal stools, the occurrence of the residue of milk digestion or the continuance of meconium stools beyond the usual period were helpful in determining partial or complete obstruction. Prolongation of life, despite an almost complete obstruction was a surprising


5. A possible exception to this statement is Van Der Bogert’s case. In an infant, 4\frac{1}{2} months old, with a duodenal obstruction 2\frac{1}{2} inches from the pylorus “there was no appearance of bile in the vomitus, although unfortunately no chemical tests were made.”

feature. In Cautley’s case, in which the child lived twelve months “the second part of the duodenum was extremely stenosed, merely admitting the passage of a probe.” And Spriggs, performing a necropsy on an infant 9 months old, found a diaphragm occluding the lumen of the duodenum; and, as he remarked, although there was “no evidence of an orifice in this diaphragm, the history of the case proves that some opening must have existed.” The uncertainties of abdominal diagnosis, in general, at this early age, combined with variations depending on the position and degree of obstruction, make it very difficult to outline a clear cut clinical picture. One might be tempted to offer a crude rule of thumb, that a symptom complex suggesting but not typical of pyloric stenosis or pylorospasm, in a desperately sick infant, should raise the question of an obstruction in the upper part of the small intestine.

The etiology of duodenal obstruction has been fully reviewed. Many of the anomalies and the theories to explain them are referable to conditions within the lumen of the intestine, valves, folds of mucous membrane, failure of absorption of embryonic epithelial plugs, etc. The case to be reported was not studied from that angle, and although it is extremely unlikely, such a factor might have been operative in addition to malrotation.

REPORT OF CASE

History.—Baby R. was born on the obstetric service of the New York Nursery and Child’s Hospital, Dec. 24, 1920. Toxemia of pregnancy necessitated delivery by cesarean section during the eighth month of parturition. The premature baby weighed 6½ pounds and appeared well developed. When 7 days old the child was transferred to the pediatric service with the history of 1½ pounds loss of weight and vomiting of all food since birth; the vomitus was dark green in color and not projectile.

Course.—Dr. Anderson, the resident physician, noted that the baby was emaciated, the fontanel slightly depressed, the abdomen sunken and the liver and spleen palpable. The infant lived twenty-six days after admission to the pediatric service. Although occasional days passed without vomiting, persistent bilious vomiting was the striking symptom. Lavage on numerous occasions also revealed bile stained fluid, at times as much as from 3 to 4 ounces of “thick pea soup material.” At first, the stools were yellow and indicated the passage of food through the intestines; toward the end they became watery and green. The roentgen-ray examination was not conclusive. It showed no peristaltic waves, an atomic stomach and a threadlike shadow outlining the course of the duodenum. Three days before death, definite peristaltic waves were seen. No tumor could be felt. Atropin was administered for a few days. It is hardly necessary to detail the supportive measures employed—saline solution subcutaneously and into the peritoneal cavity, and transfusion—and the care in matters of feeding.

Fig. 1.—The liver has been pulled upward and the transverse colon downward. This gives the impression that pressure occurred lower down in the duodenojejunal than was actually the case. (1) Sharp duodenojejunal angle; (2) jejunum; (3) lower part of ileum; (4) band attaching cecum to posterior abdominal wall.
Diagnosis.—The diagnosis lay between pyloric stenosis and obstruction below the ampulla of Vater. Bilious vomiting, pointing to relaxation of the pyloric sphincter, was the striking symptom and most of those who examined the infant favored the diagnosis of obstruction. A second transfusion preliminary to exploratory laparotomy was planned when the infant died.

Report of Necropsy.—Aside from the intestinal tract, the necropsy is not of interest. The small intestine occupied the entire right half of the abdomen. The cecum was situated to the left of the midline beneath the transverse colon (Fig. 1). Drawing the cecum downward and to the right, brought out a firm, rather tough band, attaching the cecum to the posterior abdominal wall. The ileum entered the cecum from right to left. The stomach was much dilated and the pylorus was wide and flabby. The dilatation of the first portion of the duodenum was even more pronounced than is indicated in the sketch. The third portion of the duodenum, instead of curving gradually upward, turned to the left, creating a sharp duodenojejunal angle. To express these relations differently, the third part of the duodenum was missing and the duodenojejunal angle was formed by the transverse or second part of the duodenum and the continuous loop of the intestine. As this loop of intestine continued downward, it emerged, clamped between the last few inches of the ileum dorsally and the cecum and ascending colon ventrally. One may picture the ileum and the ascending colon as the blades of a scissors (Fig. 2). The posterior or dorsal blade is formed by the ileum, the anterior or ventral blade by the ascending colon, the ileo-caecal region forms their point of attachment and the duodenum is clamped between them. Figure 2 shows the ascending colon cut and rolled back, uncovering the duodenum as it crosses the ileum. Thus, three factors contributed to the production of the obstruction: (1) the sharp duodenojejunal angle; (2) the squeezing of the jejunum between ileum and colon; (3) the dilatation of the first portion of the duodenum was even more pronounced than is indicated in the sketch.

8. The sketch was made from the preserved and somewhat shrunken specimen.

Fig. 2.—Ascending colon cut and pulled back to show mechanism of obstruction. (1) Dilated duodenum; (2) duodenojejunal angle; (3) jejunum; (4) ileum.
(3) the binding down of the cecum to the posterior abdominal wall. The fixation of the cecum prevented free mobility, and a freely movable cecum might have exerted less pressure on the small intestine.

It is simpler to describe the anatomic conditions determining the obstruction than to trace their pathogenesis. The explanation of the mechanism of obstruction rests on the determination of the primary factor in the malrotation of the cecum and ascending colon. It will be recalled that in early fetal life the alimentary canal is a vertical tube. As development proceeds, the colon, which is at first on the left side, rotates about the mesenteric axis and the cecum passes across the duodenum and finally descends to the right iliac fossa. "This rotation may be arrested temporarily or permanently in any position during its circuit." 9 In the case presented there are two possibilities. First, there might have been a localized peritonitis that produced a fibrous band maintaining the cecum in the position characteristic of an earlier period of fetal life, that is, to the left of the median line (Fig. 1). According to this explanation, the fibrous band, or rather the inflammation responsible for it, would be considered the primary factor preventing normal rotation of the large intestine. Or, secondly, there might have been some other unknown primary factor interfering with normal rotation; according to this assumption, the fibrous band would be considered the result of a secondary contraction of a loose peritoneal fold. The sharp duodenojejunal angle was surely secondary; most probably it was due to the drag exerted by the small intestine. The loops of intestine hung from the mesentery as from a pedicle instead of spreading out fan shaped in the normal fashion. In either case failure of the intestine to rotate completely was the immediate cause of obstruction since it left the cecum in such a position that pressure upon the duodenum was unavoidable.

DISCUSSION AND CONCLUSIONS

Duodenal obstruction in infants, and especially in new-born babies, embraces too many variant factors to warrant generalizations. The most that can be expected, as far as diagnosis is concerned, is the determination of the presence of obstruction, whether it is partial or complete, and its approximate location. Recording the symptomatology may develop a clearer clinical picture. The results of surgical intervention depend chiefly on the complexity of the lesions. Surgeons who have seen the specimen doubt very much whether anything could have been done in the case described. In general, gastro-enterostomy is

the operation indicated. The duration of life in some infants with duodenal obstruction and the successful performance of gastroenterostomy by Ernst suggest that under favorable circumstances some cases may be amenable to surgical treatment. The cause of the obstruction in the case reported was malrotation of the intestine; whether the malrotation was primary or due to a fibrous band binding the cecum to the left abdominal wall, is controversial.