Lumleian Lectures ON SOME FORMS OF VOMITING IN INFANCY.

DELIVERED BEFORE THE ROYAL COLLEGE OF PHYSICIANS
OF LONDON, MARCH-APRIL, 1925,

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LECTURE I.—VOMITING DUE TO OBSTRUCTION FROM DEVELOPMENTAL DEFECT IN THE UPPER ALIMENTARY TRACT.

PERSISTENT vomiting as a symptom of infantile disorders is frequent enough. It is the rule in dyspeptic states of all sorts as well as in infective pyrexial disorders. Yet, just because of the predominance and frequency of this symptom in many of the most common ailments of infancy, it is especially necessary to bear in mind the possibility that the vomiting may be due to other and local causes, to developmental failure high in the intestinal tract, to inco-ordination of the intestinal musculature, or to purely functional disturbance of the gastric movements.

THE CHARACTER OF VOMITING DUE TO DYSPEPSIA.

I exclude altogether from consideration the vomiting which is symptomatic of most states of dyspepsia in infants, even although that vomiting may be long continued and severe. In infancy the dyspeptic state is almost always associated with some coincident disturbance of the bowels, and the intestinal disturbance is generally more constant than the gastric. In health the infant's bowels are opened with the least possible disturbance, and the faecal material flows quietly from the anus with little apparent effort. In dyspepsia the character of the stools and the manner of defaecation are both altered. When fermentative changes are in excess the stools are acid and irritating, are hurried along the intestinal canal by powerful peristaltic waves and expelled from the anus with great violence, with the audible escape of gas and with the outpouring of much watery secretion from the bowel. Defaecation is then generally a painful process, as is shown by the facial expression of the infant, by the cry and the movements of arms and legs. The irritating quality of the stools may be gauged by the erythema and excoriations produced upon the skin of buttocks, thighs, and heels wherever the restless movements of the child bring it into contact with the dejecta. When, on the other hand, the putrefactive processes are in excess and the acidity of the intestinal contents is unduly diminished the reaction of the stools is alkaline, the fatty acids combine with calcium and magnesium to form soaps, and the stools become hard and scybalous. For the infant the ejection of such stools is a matter of difficulty. With great straining and much distension of the anus the stool, sometimes streaked with mucus or with blood, is painfully ejected. In a second group of infantile gastro-intestinal disorders the disturbance is due, not, as in dyspepsia, to changes in the reaction and character of the intestinal contents, but is the accompaniment of an infection, catarrhal or even ulcerative, of the mucous membrane of the intestinal wall. In catarrhal and ulcerative enteritis and colitis in infancy the disorder is also constantly manifested by changes in the stools, and especially by the presence of mucus, blood, and pus in considerable quantities, while defaecation is painful and often accompanied by tenesmus.

Both in dyspeptic and in infective diarrhoea vomiting of stomach contents may be severe and persistent, but the vomiting is then associated with characteristic changes in the stools and in the manner of defaecation. In the conditions of habitual and persistent vomiting which I shall consider it is to be noticed, in the first place, that the stools and the manner of defaecation will show none

of the characteristics of dyspeptic or of infective diarrhoea. In all of them vomiting alone is prominent, associated perhaps with a varying degree of constipation or with the passage of such stools as are characteristic of the state of inanition.

CLASSIFICATION OF THE CASES TO BE CONSIDERED.

Vomiting, then, so far as it forms a part of the picture of dyspepsia, so far as it is to be regarded as a reaction in the interests of the child designed to assist in the removal of irritating and harmful products of digestion, is excluded from consideration. In these lectures I propose to deal only with the cases of persistent vomiting which are dependent upon some disorder in the mechanism of the movements of the stomach and bowel. In some the disturbance is due to the presence of gross defects in the structure of the alimentary tube; in others neuromuscular inco-ordination imposes an obstruction to the passage of the intestinal contents little less yielding; in yet others the explanation is to be found in a variety of purely functional disturbances.

VOMITING DUE TO OBSTRUCTION FROM DEVELOPMENTAL DEFECT IN THE UPPER ALIMENTARY TRACT (*Lecture I*).

- (A) *Duodenal Obstruction*:
 - (1) *Intrinsic*—Duodenal atresia and stenosis.
 - (2) *Extrinsic*—Abnormalities of intestinal rotation and peritoneal fixation, giving rise to volvulus and kinking of the bowel.
- (B) *Pyloric Obstruction*: Pyloric stenosis of Landerer and Maier
- (C) *Oesophageal Obstruction*.

VOMITING DUE TO OBSTRUCTION FROM NEUROMUSCULAR INCO-ORDINATION (*Lecture II*).

- (A) *Pyloric Obstruction*: Hypertrophic pyloric stenosis, achalasia of the pyloric sphincter.
- (B) *Obstruction at the Ileo-caecal Valve*: Generalized intestinal spasm, achalasia of the ileo-caecal sphincter.
- (C) *Obstruction at the Anus and in the Rectum*: Hirschsprung's disease, achalasia of the ano-rectal sphincter.
- (D) *Obstruction at the Cardia*: Cardiospasm, achalasia of the cardiac sphincter.

SOME FORMS OF HABITUAL VOMITING IN INFANCY (*Lecture III*).

- (A) *Vomiting and Acrophagy*.
- (B) *Vomiting and Nervousness*.
- (C) *Vomiting and Idiosyncrasy against Cow's Milk*.
- (D) *Vomiting and Rumination*.

FREQUENCY OF THE CONDITIONS TO BE CONSIDERED.

Although such a condition as congenital occlusion of the bowel could hardly escape recognition so soon as *post-mortem* examinations came habitually to be practised upon a considerable scale—Osiander in 1797 was probably the first accurately to describe a case—it is only during the last twenty or thirty years that knowledge has slowly accumulated with regard to the majority of the disorders I shall have to consider. The successful application of the method of examination by Roentgen rays and an opaque meal in the case of young children is of comparatively recent date. The constant development of surgical technique to-day enables us successfully to submit the infant and even the newly born to operative procedures which in the past have invariably proved fatal, while in the case of the primarily functional disorders a clearer appreciation of the nature of the disturbance has enabled serviceable treatment to be devised. But, it may be urged, experience has seemed to show that many, if not all, of these conditions are of considerable rarity. Diligent search in the literature may find recorded perhaps one hundred or more examples of duodenal atresia or a similar number of oesophageal atresia—Spriggs in 1912 in his admirable paper quoted 328 examples of intestinal occlusion, of which 92 were in the duodenum. Of this total he himself provided 24 examples previously undescribed. The next year Edington added 7 very completely studied cases occurring within a short time of each other in his own practice. Jewesbury and Page in 1923 reported 4 cases of different varieties of duodenal obstruction in young infants met with in one year in St. Thomas's Hospital. In 1924 Smellie reported 3 cases of intrinsic duodenal obstruction studied by him in hospital practice during the same year. But the impressions derived from a study of the recorded cases are of little value in indicating the actual frequency of such conditions. Before 1900 the number of cases of hypertrophic pyloric stenosis

was insignificant; they were recognized by only a few physicians who had paid attention to the subject. To-day, although very many still escape detection, the great frequency of the disease is apparent, because a knowledge of its existence and symptomatology has become widely spread, and the lives of hundreds of infants may be said to be saved annually by timely operation. Of the frequency of all disorders in the first days of life we are relatively ignorant, partly because of their real obscurity, but partly because of a pessimistic attitude too widely spread towards the diseases of the newborn which tends to leave recovery to the test of time. Only if the child is born in an institution or hurried in the first days of life to a hospital is the diagnosis likely to be made. Braun calculates that in Germany imperforate anus, which is not likely to escape recognition, occurs about once in 17,000 births. Occlusion at a higher level is estimated by Theremin to occur once in from 16,000 to 55,000 infants. The value of these figures and estimates is very slight. It is at least possible that the frequency may be much greater. Complete atresia is more common in the records of *post-mortem* inspections than partial stenosis, but the partial obstruction is clearly the more elusive and the less likely to be differentiated from a state of marasmus or dyspepsia. Similarly, it is frequently found that spastic states of the bowel, of which the hypertrophic stenosis of the pylorus is the most common, are more apt to pass undetected if the obstruction is only intermittent and of slight degree. Such children waste, their immunity to infection falls and their tolerance for food diminishes, until death ultimately occurs from pulmonary disease or from a secondary state of true dyspepsia. With the exception of hypertrophic stenosis of the pylorus and of some of the minor functional disturbances, one can only conclude that the conditions which I shall describe are not common, but that they are less rare than is generally supposed, and that they are beginning to be identified in increasing numbers as attention is directed towards them.

I.—ORGANIC OBSTRUCTION OF THE UPPER ALIMENTARY TRACT.

A. DUODENAL OBSTRUCTION.

It is to duodenal obstruction that I shall devote most attention, not only because obstruction in that situation, taking all cases into consideration, is relatively common, but because the diagnosis, supported by a characteristic radiographic appearance, is often not difficult, and because, sometimes at least, we may hope to save life by means of a timely operation following upon early diagnosis. In the past the interest in these conditions has been mainly in their developmental and pathological bearings. In the future it will pass more and more to the bedside.

The cases to be considered fall into two groups, differing widely in etiology and to a considerable extent in symptomatology, in their treatment, and in their prognosis. They must therefore receive separate consideration.

Etiology.

1. Intrinsic Duodenal Obstruction: Duodenal Atresia and Stenosis.—In the alimentary tract, from the oesophagus to the anus, we meet with cases both of atresia and of stenosis which have been the subject of exhaustive pathological studies. Cordes, Spriggs, Kreuter, Kuliga, and Cautley have contributed valuable studies of the literature. In all situations we meet with three varieties of obstruction: (1) by a diaphragm or membrane, perforate or imperforate; (2) by the interposition in the length of the bowel of a fibrous cord-like structure which may or may not contain a narrow lumen; (3) by complete separation of the gut which terminates blindly and begins again after a gap which is completely unbridged.

The embryological studies of Tandler, Kreuter, Forschner, Odermatt, and others have done much to make clear the origin of atresia and stenosis of the alimentary tract. These studies take us back to the fifth week of embryonic life—to a time when the bowel is a simple tube stretching from the mouth to the allantois, and showing but little differentiation into its separate parts except for a slight widening of the stomach and some convolution in that part which is to become the small intestine. The coats of the bowel wall are in no way differentiated, and the

central lumen is lined by a layer of primitive rounded endodermal cells. At this stage there sets in a rapid proliferation of the primitive epithelium, which results in the complete obliteration of the temporary lumen. Only some weeks later does a permanent lumen form by the fusion of certain vacuoles or cleft-like spaces which appear in the solid cord of endodermal cells. The epithelium lining this permanent lumen becomes differentiated into that characteristic of the various parts of the intestine, and the submucous and muscular coats of the bowel become distinct. The growth of the mesenchyme and the differentiation into muscular and fibrous coats may begin at a time when the occlusion by primitive epithelium is still present. This process of obliteration of the lumen of the bowel has been shown to occur normally in the oesophagus of the embryos of many animals (Marshall, Balfour), in the oesophagus of the human embryo (Kreuter), and in the duodenum of the human embryo (Tandler). Forschner has attempted to show that exceptionally it may occur in all situations in the bowel. He has argued very convincingly that the ingrowth of the mesenchyme and the persistence of epithelial occlusion will account for the three types of obstruction, representatives of each of which are found from time to time in most, if not in all, of the segments of the intestinal canal which are developed from this primitive tube. If the persistence of the foetal obliteration of the lumen of the primitive gut involves but a minimal length of bowel the diaphragmatic variety is produced, and the mesenchyme may or may not penetrate within its folds. In specimens examined microscopically it is found that the diaphragm in some cases consists mainly of a double layer of epithelium placed, as it were, back to back, while in others the epithelial layers are separated by fibrous tissue and muscle. If, on the other hand, the obliteration extends for some distance along the length of the bowel, the mesenchyme grows closely around the mass of embryonic epithelium, which atrophies and disappears, as such tissue tends to do, notably in the case of the vitelline duct, leaving no trace of its presence. If, as the result of changes in further development, the slender cord of fibrous tissue, enclosing perhaps the remnants of muscle and glandular tissue, is sundered, the two parts of the bowel at birth would be completely separated.

In the duodenum, as in the oesophagus and rectum, we are spared the necessity of discussing an extensive literature concerned to prove that atresia and stenosis by a diaphragm or by the interposition of a fibrous cord may be the result of foetal accidents such as volvulus and intussusception, or of foetal diseases such as peritonitis. If we were dealing with occlusions of the intestine at a lower level, where the bowel has a greater mobility, such factors would have to be considered, although even there it is difficult to understand how a volvulus or an intussusception could disappear, leaving no trace of its presence save a diaphragmatic or cord-like occlusion of the bowel, or to believe that the inflammatory changes involved in peritonitis could take place at so early a stage of foetal life. The belief that duodenal atresia and stenosis are due to a primary fault of development is supported by the frequency with which other developmental anomalies are found in association with the duodenal obstruction (11 out of 57 cases, Cordes).

Bland-Sutton has emphasized that the parts of the bowel which are most prone to exhibit developmental defects are those in which the developmental process is particularly complicated, the seat, as he says, of an "embryological event," as, for instance, in the neighbourhood of the vitelline duct or around the bile papilla. In many cases of duodenal atresia it is clear that the obstruction is closely connected with the outgrowth of liver and pancreas from the primitive gut. Not only are many obstructions found in the near neighbourhood of the papilla, but abnormalities of the common bile duct are frequent accompaniments. Thus, Buchanan describes a case in which the bile duct opened upon the upper surface of a perforate diaphragm, the pancreatic duct entering below. Edington figures his histological examination of a similar case in which the diaphragm was traversed by a channel which was lined by columnar epithelium, continuous with the ducts in the pancreatic tissue above and with the

lumen of the bowel below. Smellie describes a case of complete isolation of the stomach and first part of the duodenum which ended blindly. The gall bladder was in the correct position. The cystic and the hepatic ducts, joining to form the common duct, gradually merged into the second part of the duodenum, so that it was not possible to say where the common duct ended and where the duodenum began; presumably it was just below the entrance of the pancreatic duct. Many other cases of a like nature could be quoted. Often abnormality of the bile duct leads to a double outflow, bile entering both below and above the obstruction. In many cases of duodenal obstruction in which the main orifice of the bile duct is found to be above a complete obstruction the intestines have contained bile. Similarly, bile has been vomited when the papilla is situated below a complete obstruction. That bile so vomited may be due to swallowed amniotic fluid containing bile, or that the bile found in the intestine below an obstruction reached that situation at a time antecedent to the development of the obstruction, are possible, if somewhat unlikely, explanations. According to Savariaud, bile does not normally reach the duodenum until the fifth month. The presence of a minute accessory duct is probably often overlooked, and is the explanation of the presence of bile on what would appear to be the wrong side of a complete obstruction. Spriggs, Cordes, and Cockayne have dealt fully with this point of the distribution of bile in congenital duodenal obstruction. In other cases it may be that the condition is in reality one, not of complete atresia, but of stenosis, and that there exists a minute channel which has escaped detection. The difficulty of detecting minute fistular communications after death is considerable. There is a specimen in the St. Bartholomew's Hospital Museum from an infant, aged 9 months, showing a diaphragmatic septum in the duodenum. There is no evidence of an orifice in this diaphragm, though the history of the case and the duration of life seem to prove that some opening must have existed. We may conclude, with Cordes, that since the region of the bile papilla from an early period in the life of the embryo undergoes radical formative changes as the result of which the liver and pancreas develop, epithelial obliteration in the manner described, which may occur in any situation, is peculiarly prone to affect the near neighbourhood of the ampulla of Vater, and to be accompanied by abnormality in the distribution of bile ducts and pancreatic ducts.

2. Extrinsic Obstruction: Abnormalities of Intestinal Rotation, etc.—In the duodenum at the duodeno-jejunal flexure we meet with a second group of cases of obstruction, due also to developmental anomalies but of a very different nature. In the state of development characteristic of the human embryo of the fifth week, the three parts of the intestinal canal are clearly marked by their separate blood supply—the fore-gut digestive in function, the mid-gut absorptive, the hind-gut excretory. The blood supply of the mid-gut is the superior mesenteric artery, which crosses directly to the umbilicus and vitelline duct. A considerable part of the mid-gut lies outside the abdomen, traversing the ring which is to be the site of the future umbilicus. The pre-arterial segment of the mid-gut elongates at a very much greater rate than the post-arterial, which contains the caecum. By the tenth week the coils of intestine come more and more to occupy the abdominal cavity and to leave the umbilical sac. The pre-arterial coils enter first and pass over to the left side of the abdomen behind the transverse colon and behind the mesenteric vessels, so that they come to lie, not on the right, but on the left side of the mesentery. The superior mesenteric artery crosses the pre-arterial gut at a point near the termination of the duodenum, and the primitive relation of this vessel, lying as it does between the duodenum and the colon, is never lost. When later the caecum and ascending colon leave the sac they occupy a situation on the right side of the mesentery, while the descending colon maintains its primitive position on the left.

Derangements of this intestinal rotation—non-rotation with a left-sided caecum and appendix, reversed rotation with the duodenal loop crossing in front of the artery, and with the colon most posterior of all, malrotation with reversed rotation of the pre-arterial segment, and arrested

rotation of the post-arterial segment—are met with from time to time. The abnormal disposition of the intestine implies abnormal attachments and fixation of the peritoneal folds, and this may or may not give rise to untoward results. Undue fixation may cause kinking of the bowel, especially at the duodeno-jejunal flexure, or there may be direct pressure upon the duodenum by the superior mesenteric artery or by abnormal peritoneal folds. On the other hand, undue want of fixation predisposes to volvulus. Dott, in a very valuable paper, has pointed out how widely this characteristic volvulus neonatorum differs from the usual variety of acquired volvulus. Acquired volvulus is confined to a limited portion of gut, sharply defined above and below. The bowel involved suffers from compression or occlusion of its blood vessels, and can be easily recognized as a greatly congested and widely distended portion of the intestine. In volvulus neonatorum the extent of bowel involved is so great as to render its recognition difficult. There is obstruction only at one end of the twisted bowel—the duodenal end. The lower extremity in ileum or colon suffers no occlusion. The degree of vascular congestion is slight. Collapse rather than distension is likely to occur, and only the upper duodenum and the stomach become greatly distended. In general the symptoms are those occasioned by duodenal obstruction.

Symptomatology.

1. Intrinsic Duodenal Obstruction: Duodenal Atresia and Stenosis—Sex.—In contrast to the rule in hypertrophic pyloric stenosis, where more than four-fifths of the whole occur in males, boys and girls seem equally affected.

Prematurity and other Malformations.—In congenital duodenal stenosis or atresia the infant is not infrequently premature, sometimes the subject of other congenital malformations, yet most often strong, healthy, and well formed in other respects at the moment of birth.

Age.—The age of the infant at which serious symptoms first attract attention depends upon the completeness of the obstruction. In the great majority the occlusion is complete, or all but complete, and unless relieved by operation life is not prolonged beyond two or three weeks. Many have died about the ninth day. In only a very few is a painful existence maintained for a few months. Smellie's case lived nearly three months; Buchanan's eighteen months; Cautley's eighteen months; von Koos's thirteen months before operation and two months after. Silcock, Perry and Shaw, and Moore have recorded the accidental finding of duodenal stenosis, thought to be congenital, in the *post-mortem* examinations of adults. In general, in spite of these occasional exceptions, it may be said that if operative interference is to save life the diagnosis must be established within a few days of birth.

The Vomiting.—The persistent vomiting of bile-stained fluid in a newly born child should always arouse the suspicion of duodenal obstruction. The vomiting begins upon the first, second, or third day of life, so soon as food is taken in considerable quantities. It occurs with great regularity soon after a meal. Although vomiting is profuse, it lacks the explosive character of the act in hypertrophic pyloric stenosis. In more than 80 per cent. of the recorded cases the vomit is brightly stained with bile. In the remainder, when the obstruction is above the papilla, there may be no bile in the vomit. In the later stages haematemesis is common. Bilious vomiting is rare in hypertrophic pyloric stenosis; I have met with it only once.

The Stools.—Some degree of constipation is the rule, but the complete absence of meconium and the passage of only a little glairy mucus, as is the rule in intestinal occlusion at a lower level, do not occur. Generally it is noted that on one or more occasions a little meconium, not very abnormal in appearance, has been voided, but that as the days pass there is no sign whatever of the usual change to yellow milk stools. In hypertrophic pyloric stenosis the change to milk stools always takes place in the usual way, although at the height of the disturbance the stools may again assume a character which reminds one of meconium—that is, they become the slimy, deeply pigmented stools of complete inanition. Microscopic examination, in cases of complete obstruction, may show the absence of lanugo.

The Examination of the Rectum.—In duodenal stenosis or atresia the examining finger encounters nothing very abnormal in the rectum. In occlusion of the lower alimentary tract the rectum and pelvic colon are often quite undistended, so that the finger cannot be passed more than 2 or 3 inches from the anus. It is to be remembered that rectal atresia is always situated within 2 inches of the anus, so that confusion should not arise. In several instances the inability to pass the finger into the rectum has led to the erroneous diagnosis of a rectal rather than an intestinal atresia. For the same reason in an ileal or colic obstruction it is usually possible to inject only minimal amounts of fluid into the bowel. In duodenal obstruction, on the other hand, the same difficulty is less likely to be encountered.

The Abdomen.—In duodenal obstruction the abdomen seldom shows any general distension. Only the epigastrium may be full and the outline of the stomach, with faint indications of peristaltic activity, may be apparent. Immense peristaltic waves, such as gradually develop in the later stages of hypertrophic pyloric stenosis, are not seen. On the other hand, it is important to notice that the visible peristalsis may be appreciable in the first days of life, while in hypertrophic pyloric stenosis it develops slowly after an interval, usually of some weeks. In intestinal occlusion at a lower level the abdomen may be greatly or irregularly distended, and visible intestinal peristalsis rather than gastric peristalsis is the rule. Auscultation over the distended abdomen will often disclose a great variety of loud gurgling sounds and borborygmi, while in duodenal obstruction there is unusual silence.

The General Condition.—After the first few days of life emaciation and dehydration proceed rapidly. Soon the temperature becomes subnormal, the extremities cold and cyanosed, and the pulse rapid. The facial expression is anxious and the child looks very ill, contrasting with the eager, alert, if pained, expression of the baby with hypertrophic pyloric stenosis. Anuria is common. Many are jaundiced more deeply than is the rule with the newly born.

The X-ray Picture.—In nearly all cases an x-ray examination after a bismuth meal will show a quite characteristic picture, which can hardly fail to make the diagnosis clear. The duodenum is seen as a widely distended pouch, ending abruptly at the site of the obstruction. By an x-ray examination, and by careful clinical examination, it ought to be possible to avoid confusion with intestinal occlusion at a lower level, in which constipation and the abdominal distension rather than the vomiting are the prominent features. For this reason I have excluded consideration of ileal and colic atresia.

The following case, which came under my care in the children's department at Guy's Hospital, is an example of congenital duodenal stenosis, with complete or almost complete obstruction.

The baby, a girl, weighed at birth 3½ lb. She was a first child, born after one miscarriage. On the first and second days nothing occurred to arouse anxiety. On the third day vomiting began, and continued with increasing severity after every breast feed. The vomit was deeply bile-stained. The baby was seen by me on the fifth day. I was told that up to that time the bowels had opened twice and a little meconium, not noticeably abnormal, had escaped. Rectal examination showed nothing abnormal. There was no visible peristalsis of the small intestine, and although close inspection seemed to show the outline of the distended stomach no definite peristaltic waves were seen. An examination after a bismuth meal showed a dilated stomach with nothing leaving the pylorus. Thirty minutes later a second examination showed the outline of the greatly distended duodenum (Fig. 1), which ended abruptly at a point apparently in its second part. Two small opaque spots below suggested that a small amount of the meal had passed an obstruction which was barely perforate. On this day, the fifth of life, the infant's weight was 16 oz. less than at birth, although very little meconium had been passed. That

afternoon Mr. Steward performed a posterior gastro-enterostomy. There were a few ounces of straw-coloured fluid in the peritoneal cavity, but no other evidence of peritonitis. The site of obstruction was marked outside the gut by a paler area, perhaps half an inch long, above which the bowel was greatly dilated and engorged, while below it was collapsed and empty.

For the first six days after operation bilious vomiting continued, although in decreasing amount. The bowels were open on the day after the operation. Three weeks after the operation x-ray examination showed the opaque meal issuing from the stoma, while the distended outline of the duodenum was still apparent.

On the twenty-fifth day after the operation the child was sent home, weighing 8 lb. 5 oz. When 3 months old, during hot weather, the infant suffered severely from an attack of diarrhoea and vomiting, and, as might be expected from the conditions produced by the operation, the vomited matter was bile-stained. Fig. 2 is a photograph of this baby, now apparently healthy, at the age of 8 months.

FIG. 1.—Congenital duodenal stenosis, showing dilatation of upper duodenum.

FIG. 2.—Photograph of the infant with congenital duodenal stenosis taken when aged 8 months.

Péhu and Auberge, who have recently exhaustively reviewed the literature of duodenal obstruction in childhood, divide the cases into two groups: cases of stenosis and atresia, here called "intrinsic," which they term "cases incompatible with existence"; and cases, here named "extrinsic," which they call "cases compatible with existence." I have recounted this success in detail because I feel that the view implied in this nomenclature is altogether too gloomy. *Post-mortem* records show a very large number of cases which almost certainly might have been saved in the same way had the diagnosis been established in the first days of life by means of the characteristic x-ray picture. In the literature, however, only one other

case is reported as having been saved by timely operation. In 1916 Ernst of Copenhagen successfully performed duodeno-enterostomy, which would appear to be the operation of choice in a case of duodenal atresia.

2. Extrinsic Duodenal Obstruction: Abnormalities of Intestinal Rotation, etc.—Age.—The symptoms in extrinsic cases are often less complete and less characteristic. The history in general is one of recurrent attacks or bouts of severe vomiting of bilious material, together with abdominal pain, dating back in many cases to earliest infancy, but often beginning in later life. It is clear that the anomaly of intestinal rotation and of peritoneal fixation can be present without producing any obstruction, but that it does powerfully predispose to a variety of accidents, such as volvulus, ptosis, and kinking of the duodeno-jejunal flexure, or duodenal obstruction by peritoneal bands and by

the superior mesenteric artery as it passes into the root of the mesentery.

Of twenty-eight cases of duodenal obstruction of this variety recorded in the literature as being relieved by operation, and reviewed by Duval and Gatellier, in six the subjects were children. Dubose's case was 8 weeks old, Frank's 11 months, Downes's 4½ years, Ombrédanne's 11½ years, Kotzareff's 12 years, and Foisy's 14 years. Rixford has recorded successful reduction of volvulus at 5 years of age. Poynton

has recently published four cases in children occurring within a short time of each other in his own practice, all of which, however, proved fatal. Since all the characteristic features of a localized intestinal volvulus are absent, he rightly emphasizes the likelihood of confusion with that more common cause of bouts of uncontrollable vomiting in childhood, the so-called cyclical vomiting.

In very many cases, however, it would appear that the first attack of duodenal obstruction is fatal. Of Dott's series of thirteen cases in which duodenal obstruction had occurred, collected from the literature, symptoms developed within a few days of birth in six. In his own two cases the infants died on the twelfth and sixth days of life, immediately after an unsuccessful attempt to reduce the

obstruction by operation. Of the four cases published in 1923 by Jewesbury and Page, occurring in young infants admitted to St. Thomas's Hospital within a short time of each other, one was an example of atresia and one of extrinsic duodenal obstruction by arterial pressure. Both of these died. Two others, in which the extrinsic obstruction was by kinking of the duodeno-jejunal flexure by peritoneal bands, were saved by operation. I agree with Dott that in all probability extrinsic obstruction is more common in the newborn than is generally supposed. Such cases are sure to be confounded with dyspepsia and treated by dietetic prescriptions. The danger to life must vary with the precise nature of the accident to which the obstruction is due. It is likely to be much greater in volvulus than in obstruction by peritoneal folds or bands.

Vomiting.—The vomit is always bile-stained. In the later stages haematemesis may occur. The act follows closely upon the intake of food.

The Stools.—Meconium is voided normally after birth, and if the onset of the obstruction is delayed for some days the change to milk stools may take place normally. Later, constipation will be severe and the stools become those characteristic of a state of inanition.

The Abdomen.—Distension will only affect the epigastrium, where the outline of the stomach and perhaps gastric peristalsis may be dimly apparent.

The X-ray Picture.—The diagnosis is only likely to be made with certainty by means of an x-ray examination. It must be directed to the elucidation of two points. Normally the duodenum empties with great rapidity. For a few seconds, perhaps five or six, the issuing shadow will be seen to pause in the first part of the duodenum immediately beyond the pylorus. The remaining parts of the duodenum are traversed in the space of six or eight seconds more. In extrinsic duodenal obstruction in older subjects the duodenum is seen to be greatly dilated, and to empty with difficulty and very slowly. Peristaltic waves are observable, and sometimes reverse peristalsis with return of the food through a "forced" pylorus. In infants something of the same may be seen, if perhaps in less developed form. Secondly, in cases of doubt attention should be paid to the position of the caecum and ascending colon. If there is a left-sided caecum, or if it is otherwise abnormal in situation or unduly movable, the probability that we are dealing with a case of obstruction from abnormality of peritoneal fixation is greatly strengthened. For this examination a bismuth enema is valuable. Such an examination in an older child may be necessary to distinguish the condition from one of cyclical vomiting.

Dott has discussed the operative procedure indicated if laparotomy reveals a neo-natal volvulus. The pressure can be relieved by the reduction of the volvulus, as was successfully done in Rixford's case. Dott favours the delivery of the intestines and their rapid untwisting, deciding in each case whether to return them in the position of malrotation or to complete the rotation to the normal position by hand. He advises fixation of the caecum as an additional security against recurrence. If on account of adhesions reduction is impossible, a gastro-enterostomy should be performed. In obstruction by peritoneal bands it may only be necessary to free the duodenum from their pressure.

B. OBSTRUCTION OF THE PYLORUS.

True organic stenosis at the pyloric valve from congenital defect is occasionally found in adults. Landerer in 1879 and Maier in 1885 were the first to record a series of cases of this defect which had in common an absence of the ring-like thickening due to the musculature of the pyloric sphincter, while the mucous membrane passed inwards at the point to form a valve-like diaphragm pierced by a quite small opening. In Landerer's cases the pyloric canal and stomach showed dilatation, but little or no hypertrophy. Maier described at least one similar case without hypertrophy, but added a large number in which, in addition to the valvular obstruction, there was present a considerable hypertrophy of the muscle of the whole pyloric canal and pyloric part of the stomach. It seems likely that cases in adults of pyloric stenosis and hypertrophy described by Maylard, Barling, and others in this country and thought to resemble the common hypertrophic pyloric stenosis of

infancy, were in reality of the type described by Landerer and Maier in Germany and recognized by Russell in this country. It is interesting to recall that Hirschsprung, in calling the attention of the profession to the common hypertrophic stenosis of infants, made the opposite mistake, and was at first under the impression that he was recording cases in infancy of the organic stenosis of the type described by Landerer and Maier. In hypertrophic pyloric stenosis there is present, of course, no true organic stenosis at all. Within the rigid spastic musculature of the pyloric canal there lies a lumen in every way normal, but so folded that on transverse section the outline appears shaped like a star or rosette. Since Hirschsprung's first communication innumerable cases of hypertrophic pyloric stenosis have been encountered and recorded, and the great frequency of the condition is now fully appreciated. On the other hand, even among adults the records of cases of organic stenosis remain very few. In infancy, I know of but one undoubted case, that described by Schäfer. It is characteristic of this form of pyloric obstruction that the symptoms begin at some time during later childhood, but not during infancy.

Schäfer's *post-mortem* examination showed closure of the pylorus by a membranous diaphragm composed of mucous membrane with a central lumen which permitted the passage of only a very narrow sound. During life a litre of malted milk had passed the obstruction daily without giving rise to any appreciable discomfort. A most minute examination showed no trace of the pyloric ring sphincter muscle, but in its place a fold of mucous membrane; this was confirmed by histological examination.

Faber has recently reviewed the literature; it may be remarked that the condition seems almost completely to have escaped attention in this country.

I am unable to add to the single observation in an infant accidentally made by Schäfer, but I show two specimens from adults typical of this congenital hypoplasia of the pyloric ring muscle.

The first is the stomach from a man 38 years of age, who had suffered from the age of 12 with abdominal pain, vomiting, and tetany, of great severity. He came under the care of Dr. Fawcett, to whom I am indebted for the specimen, but died suddenly before operation could be undertaken. The stomach is very greatly dilated. The pyloric canal has been distended and taken up into the body of the stomach, much as the cervical canal is taken up by the pregnant uterus at the commencement of labour. Separating the small duodenum from this huge stomach is a membranous diaphragm, perforated by a small aperture.

This man's father, who also came under Dr. Fawcett's care, suffered apparently from a similar defect of development. At the age of 63 he too was admitted for attacks of abdominal pain and vomiting of "fifty years' duration." At times as much as nine pints of fluid had been removed from the stomach by the pump. The state of his kidneys, however, made operation inadvisable.

I show also a specimen from the museum of Guy's Hospital, of which I think the description in the catalogue is misleading. It shows the pyloric end of a very greatly dilated stomach, which measured 30 in. by 9 in., from a man, aged 38, who had suffered with symptoms of pyloric obstruction since the age of 12. The specimen shows the same hypoplasia of the pylorus, with a fold of mucous membrane in the form of a diaphragm, perforated by a small central aperture.

It is, I think, clear that these two specimens, so alike in every detail, both in their clinical history and in their *post-mortem* appearances, are examples of the congenital pyloric stenosis of the type described by Landerer and Maier.

ŒSOPHAGEAL OBSTRUCTION.

In order to complete the list of causes of persistent vomiting in infancy due to anatomical defect, it is necessary to say a word as to the condition of Œsophageal stenosis or atresia. Cautley has recently reviewed the literature so exhaustively that there is little that can be usefully added.

Etiology.—A study of the developmental defects which involve the Œsophagus entails also a study of similar defects in larynx and trachea.

Keith has discussed the results of abnormality in the development of the tracheo-Œsophageal septum. De Vries of Amsterdam has figured diagrammatically the possible results of failure of this septum to develop normally, and has found in Continental literature cases recorded which illustrate every variety. By far the most common form is that in which atresia of the Œsophagus is combined with an opening between the lower Œsophagus, below the atresia, and the trachea. The opening may be wide or it may be narrow. A similar atresia or stenosis

of the larynx with a communication between the normal oesophagus and the lower trachea or a bronchus is also encountered. On the other hand, atresia of oesophagus without communication with the trachea is very rare. Atresia or stenosis of larynx without communication with oesophagus may also occur. A small fistulous communication between the two passages, due to developmental error, may be present without other defect, or it may complicate the more usual oesophageal atresia. Traction diverticula may represent such a fistulous communication in an incomplete form.

Symptoms.—In the form most frequently met with—oesophageal atresia, with communication between the lower oesophagus and the trachea—death invariably follows within a few days. The child, though sucking strongly and hungrily, is able to swallow only the first few mouthfuls; thereafter the attempt to swallow leads to a free regurgitation from the mouth and the nose. The attempt to take food, moreover, has commonly to be given up because of asphyxial attacks or attacks of choking and coughing. A sound is held up, very often at a distance of some 10 cm. from the gums. The shadow of an opaque meal shows clearly the obstruction in the oesophagus. Death is commonly from an inhalation bronchopneumonia, due to the fistulous opening between trachea and the lower segment below the obstruction. In a few cases the respiratory movements have served to bring about great inflation of the stomach with air which has passed along the same route. The existence of this communication serves to make even the desperate expedient of gastrostomy useless and inadvisable.

In oesophageal obstruction the regurgitation of the milk is so clearly due to a failure to swallow that the condition can hardly be confused with any other. Even in diaphragmatic hernia, with the stomach within the thoracic cavity, the difficulty does not concern the act of swallowing, although here again we may meet with asphyxial attacks which are produced by the attempt to take food. In oesophageal stenosis, as distinct from atresia, symptoms are seldom prominent during infancy while the diet remains fluid. The cases present themselves for examination in later childhood.

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PUERPERAL SEPSIS IN WALES.*

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THE tables and diagrams exhibited, taken in conjunction with those for previous years, show that Wales has a consistently high maternal mortality rate, and that the Welsh figures are much higher than the combined figures for England and Wales. Further, the fact emerges that whilst the "sepsis" death rates are somewhat higher in Wales than in England and Wales, the chief disproportion is from "other causes." I have to thank the Registrar-General for permission to quote the figures in the table relating to 1923, which have not yet been published.

Distribution for Wales and for England and Wales of Mortality of Women in Childbirth, per 1,000 Children Born Alive, distinguishing Septic and other Causes, 1921, 1922, and 1923.

	1921.		1922.		1923.	
	Wales.	Eng'land and Wales.	Wales.	England and Wales.	Wales.	England and Wales.
County Boroughs—						
Sepsis	1.35	1.54	3.32	1.62	1.94	1.41
Other causes ...	2.99	2.40	3.34	2.28	2.67	2.60
All causes ...	4.34	3.94	6.56	3.90	4.61	4.01
Other Urban Districts—						
Sepsis	1.72	1.27	1.31	1.27	1.49	1.25
Other causes ...	3.47	2.76	3.77	2.68	3.92	2.67
All causes ...	5.19	4.03	5.08	3.95	5.41	3.92
Rural Districts—						
Sepsis	1.80	1.25	1.45	1.16	1.51	1.15
Other causes ...	4.55	2.97	3.83	2.85	4.54	2.72
All causes ...	6.35	4.22	5.28	4.01	6.05	3.87
All Districts—						
Sepsis	1.67	1.38	1.75	1.38	1.59	1.30
Other causes ...	3.68	2.53	3.68	2.43	3.83	2.51
All causes ...	5.35	3.91	5.43	3.81	5.42	3.81

The three types of population compared are—

- (1) County borough populations, which are truly urban in character.
- (2) Other urban districts, which contain a large number of people living under semi-rural conditions.
- (3) Rural districts, the bulk of whose populations are country dwellers.

It will be seen that in England and Wales as a whole the death rate from sepsis is lowest in rural districts, and becomes higher proportionately as the urbanization of the population increases. With regard to the death rates from "other causes," the position is reversed. The more highly urbanized populations show the lowest maternal mortality from "other causes." This may be attributable to the better medical and surgical facilities available and to the provision of maternity beds.

In Wales the sepsis death rates show no regular variation with the type of population concerned; but regarding deaths from "other causes," the table shows how much greater is the risk for the rural mothers than for the mothers in the urbanized communities.

The first report of the Welsh Consultative Council draws attention to the fact that Wales is a rugged and mountainous area, consisting for the most part of rural counties sparsely populated, with exceptional difficulties

* Paper preliminary to a discussion on puerperal sepsis at the British Congress of Obstetrics and Gynaecology, London, April 22nd.