MUSCULAR HYPERTROPHY OF THE PYLORUS IN INFANCY *

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This may be defined as an affection occurring in young infants characterized by forcible vomiting, constipation, visible peristalsis and frequently a palpable tumor at the situation of the pylorus produced by a marked increase in its muscle fibers. The disease has been described under the names "Hypertrophic Pyloric Stenosis in Infants," "Congenital Spastic Stenosis," "Congenital Pyloric Stenosis," and "Spastic Hypertrophic Pyloric Contracture."

Osler discovered a typical description of this disease written by Dr. Hezekiah Beardsley in a volume entitled "Cases and Observations by the Medical Society of New Haven County in the State of Connecticut," published in New Haven in 1787. It will not be out of place to quote liberally from this paper which is remarkable for the accuracy of observation and quaintness of diction.

"A child of Mr. Joel Grannis, a respectful farmer in the town of Southington, in the first week of its infancy, was attacked with a puking, or ejection of the milk, and of every other substance it received into its stomach almost instantaneously, and very little changed. The feces were in small quantity and of an ash color, which continued with little variation till its death. For these complaints a physician was consulted, who treated it as a common case arising from acidity in the prima via; the testaceous powders and other absorbents and correctors of acid acrimony were used for a long time without any apparent benefit. * * * I was at first inclined to attribute the disorder to a deficiency of bile and gastric juices, so necessary to digestion and chylification, joined with a morbid relaxation of the stomach, the action of which seemed wholly owing to the weight and pressure of its contents, as aliment taken in small quantities would often remain on it, till, by the addition of fresh quantities, the whole, or nearly all, was ejected; but his thirst, or some other cause, most commonly occasioned his swallowing such large draughts as to cause an immediate ejection, and often-times before the cup was taken from his mouth. * * * A number of the most respectable medical characters were consulted and a variety of medicines were used to little or no effect. His death, though long expected, was sudden, which I did not learn till the second day after it took place. This late period, the almost intolerable stench, and the impatience of the people who had collected for the funeral prevented so thorough an examination of the body, as might otherwise have been made. * * * I next examined the stomach, which was unusually large, the coats were about the thickness of a hog's bladder when fresh and distended with air; it contained about a wine pint of fluid exactly resembling that found in the vesicles before mentioned, and which I supposed to have been received just before his death. The pylorus was invested with a

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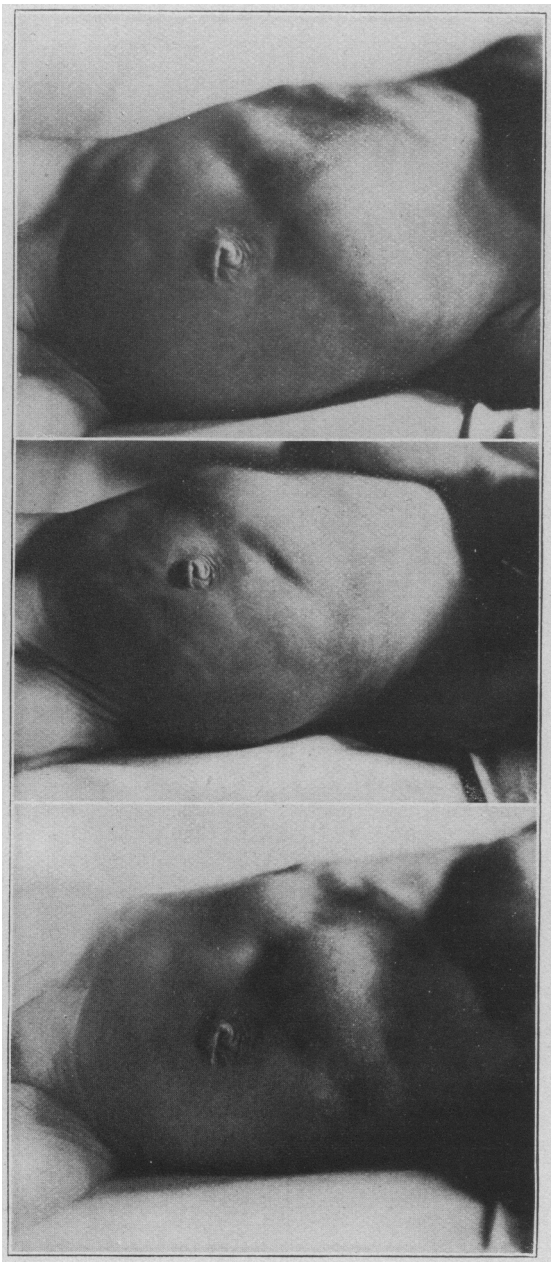

Figs. 1, 2 and 3.—Visible peristalsis of the stomach, Case 3.

hard compact substance, or schirrosity, which so completely obstructed the passage into the duodenum, as to admit with the greatest difficulty the finest fluid; whether this was the original disorder, or only a consequence, may perhaps be a question. In justice to myself I ought to mention that I had pronounced a schirrosity in that part before the child's death."

Williamson published an account of this condition in a 5-weeks-old infant with the autopsy findings in the London and Edinburgh *Journal of Medicine* in 1841.

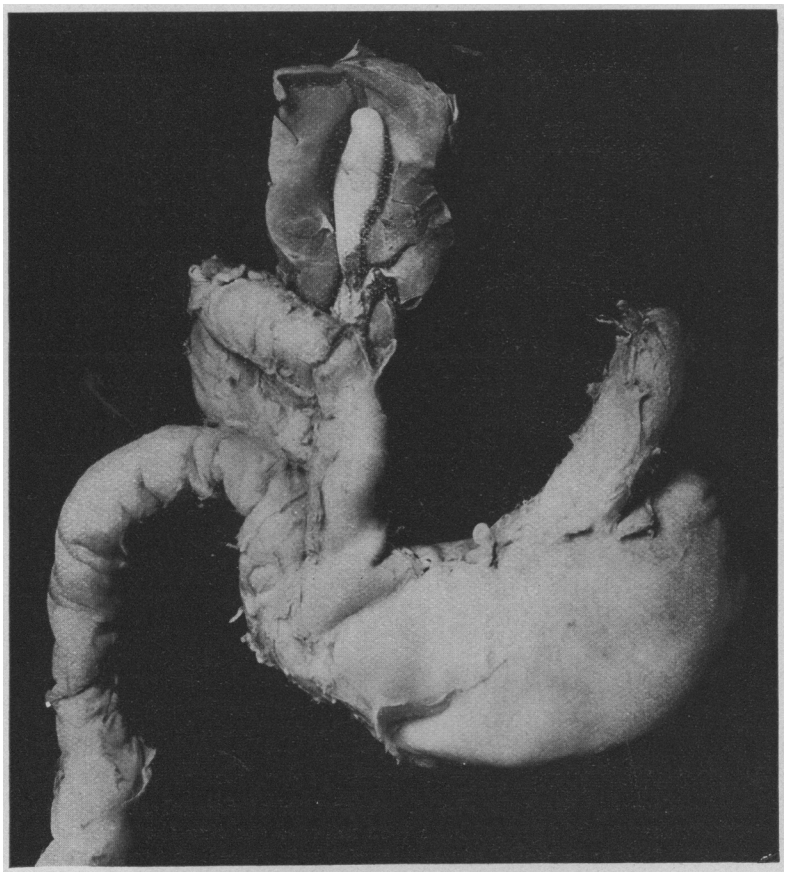

Fig. 4.—Case 4. Stomach, duodenum, fragment of liver and gall-bladder: the upper part of the small intestine is sutured to the stomach just below the pylorus (natural size).

No further mention of this disease in infants appeared in the literature until Hirschsprung, a Danish physician, reported two cases before the German Pediatric Society in 1887. He found that the pylorus was thickened and that the muscle fibers were increased. Pfaundler called attention to the fact that two distinct groups of cases had been classified

under one head. He showed that there was both a functional and organic closure of the pylorus and his views are now almost universally accepted. A careful examination of the literature by one of us (Shaw) in 1904 revealed 117 cases while Ibrahim collected over 400 cases in 1908 and a number of cases have been reported since then. This condition is therefore by no means uncommon and must frequently be overlooked.

Pfaundler found that the first vomiting or onset of the disease was between the fourth and fourteenth days in 50 per cent. of the cases, from the second to third week in 25 per cent., and from the third to sixth week

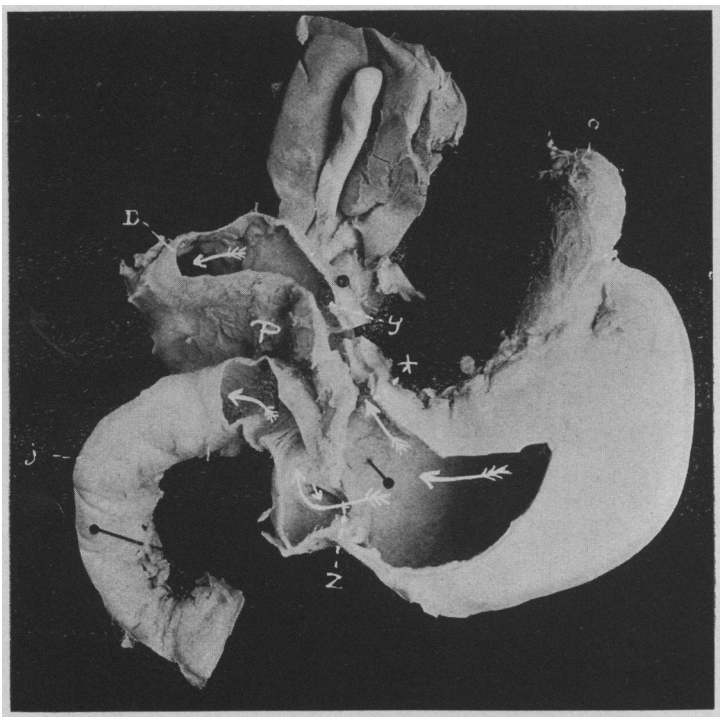

Fig. 5.—Case 4. Same as Fig. 4. A portion of the anterior walls of the stomach, duodenum and jejunum have been removed showing the stenosed pylorus with greatly thickened walls (x-y). Here, as indicated, fragments have been removed for sectioning longitudinally (Figs. 6, 7, and 8) and transversely (Figs. 9 and 10). The natural and artificial food channels are indicated by arrows. z, large, patent gastro-enterostomy opening; j, jejunum; d, duodenum; p, pancreas; o, esophagus (natural size).

in 25 per cent. Five out of nine of our cases started in the first and second weeks of life, two in the third week, one in the fourth and one after the fourth.

Boy babies are affected, according to Ibrahim, about four times as frequently as girls, but in our cases five of the nine were boys. Five of

our nine were first-born babies and in two families subsequent children have shown no gastric disturbances. In one family the first child is strong and healthy, the second child died after an operation to relieve the stenosis (Case 3) and the third child died when 6 weeks old from a typical attack of this disease. This is the only instance among our cases in which there appeared to be any family tendency.

Nearly all the cases of pyloric stenosis occur in breast-fed infants, which is contrary to nearly all the affections of the digestive tract.

SYMPTOMS

A healthy and robust child in the first weeks of life is suddenly seized with violent *vomiting* without apparent cause. At first this occurs irregularly but later increases in frequency, often after each feeding. Examination of the breast milk shows it to be normal. Modifications of cow's milk, changes in quantity or quality of the food or of the intervals of feeding have no effect on the vomiting. It is forceful and projectile

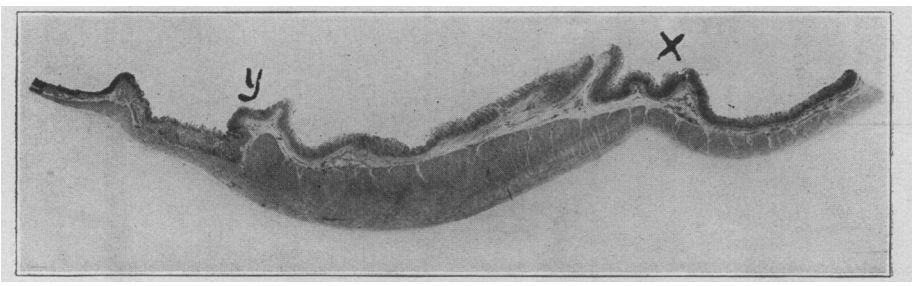

Fig. 6.—Case 4. Longitudinal section of wall of pyloric canal showing the greatly thickened muscular wall. The duodenal end of the "canal" is indicated by "y" and the gastric end by "x". These points are well marked, the former by the duodenal submucous or Brunner's glands, and the latter by a definite indentation; here the thickening of the muscle coat begins. Hematoxylin-eosin stain. (Mag. x 4.)

and occurs suddenly without warning. It is variable in amount, and the quantity of fluid thrown up is surprising. The child will apparently retain several of its feedings but suddenly will throw up all the previous feedings. Six ounces may be thrown up in a jet-like explosion. The vomited matter consists of slightly digested milk mixed with mucus. It never contains bile.

This vomiting is not controlled by the usual medicinal remedies. Lavage may reduce the vomiting to once or twice a day. The tongue remains clear and the breath sweet.

Constipation and impaired nutrition are directly dependent on the vomiting. The constipation is really deficient defecation resulting from the diminished intake of food and fluid. Cases have been observed in

which no bowel movement could be obtained for twelve days. The stools are small in amount, dark green, very offensive, and contain much mucus. The urine is scanty and high colored.

The *weight* of the infant is the index of his nutrition. The loss in weight in this disease can amount to 1 ounce or more a day. There is a progressive emaciation which is shown later by extreme exhaustion, subnormal temperature, depressed fontanel, sunken cheeks, etc.

The lower part of the *abdomen is flat* and even sunken in, while the upper part is distended and prominent. This gives the abdomen a characteristic appearance.

The most characteristic symptom and one that is pathognomonic of this condition is the *visible peristaltic contraction* of the stomach. This has been termed "stomach stiffening" and is due to the strong muscular contraction of the narrow pylorus. The stomach appears to stand up on itself. This is seen during sleep as well as when the child is awake. The wave starts from the left side and is about the size of a hen's egg. The protruding part is often surrounded by deep retraction in the abdominal

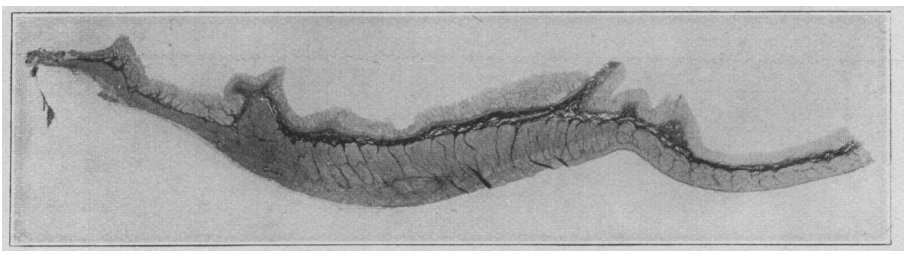

Fig. 7.—Case 4. Section same as Fig. 6, stained by Van Giesen's connective tissue stain. The large amount of muscle tissue is in marked contrast to the connective tissue which appears black. (Mag. x 4.)

wall. Before it disappears to the right another wave appears where the first started. Hour-glass contractions may occur owing to the two waves running close behind each other. This visible peristalsis apparently is not painful, which is not the case with spastic contraction of the pylorus with which it is often associated.

An equally characteristic and pathognomonic symptom is a *palpable enlargement of the pylorus.* This is not regularly present especially in early cases. The pylorus was markedly enlarged in all of our six patients operated on, and in none of them could the pylorus be palpated through the abdominal wall.

Dilatation of the stomach is present in advanced cases depending on the degree and duration of obstruction but is absent in the early stages. as the food is ejected as soon as the stomach is overdistended. The stomach contents may be retained over several feedings and then ejected, showing that little or no food passes out of the stomach.

Several authors emphasize the presence of *hyperacidity*. The increased acidity may be due to hyperchlorhydria or an increase in fatty acids.

PATIENTS OPERATED ON

CASE 1.—E. S., first child, breast-fed from birth. Vomiting commenced about the third month; admitted to St. Margaret's December, 1902, aged 6 months: weight 15 pounds 8 ounces; uncontrollable vomiting, visible peristalsis, constipation. The symptoms continued despite treatment and there was a steady loss in weight. Operation (anterior gastro-enterostomy) at the Child's Hospital by Dr. Elting in June, 1903. Weight 12 pounds 1 ounce; "Stomach greatly dilated,

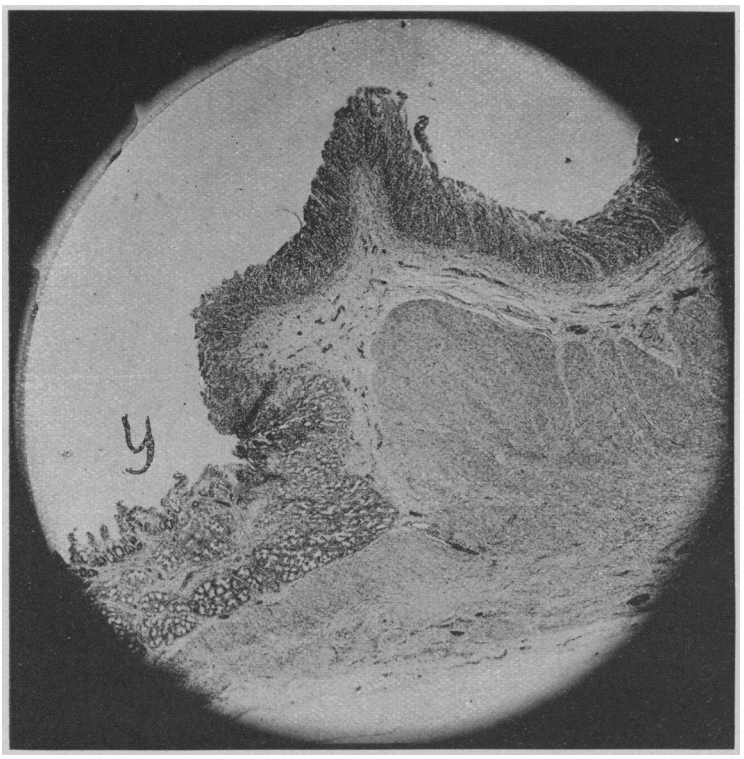

Fig. 8.—Case 4. Portion of Fig. 6 showing duodenal end of wall of pyloric canal. The submucous (or Brunner's) glands begin at "y," to the left is duodenum and to the right the pylorus. The thickness of the walls is in marked contrast. Minute black foci scattered through the submucosa, muscularis, and serosa indicate the inflammatory changes above referred to. (Mag. x 40.)

pylorus firm and thickened, lumen greatly reduced and stenosis pronounced." No bile was ever detected in the vomitus before operation but was present afterwards.

The child made a good recovery and gained 9 pounds in five months, and is now a large, well-nourished girl without any stomach trouble.

CASE 2.—E. B. (referred by Dr. Pearson of Schenectady); first child; breast-fed. Vomiting commenced at the end of the first week and was projectile in character; visible peristalsis and constipation.

Operation (posterior gastro-enterostomy) by Dr. Elting, Nov. 23, 1906, when the child was three weeks old. Died Nov. 28, 1906. No post-mortem allowed. Operation protocol reads as follows:

"On opening the abdominal cavity, a large distended stomach ballooned out. The intestines were collapsed and appeared as mere ribbons. There was complete stenosis of the pylorus due to a tumor the size of a small pigeon's egg and regular in outline. It was impossible to force gas through at this point. After anastomosis the intestines began to dilate and fill with gas."

CASE 3.—M. M., referred by Dr. Maby of Mechanicsville; second child; first child was strong and healthy. This child was breast-fed and commenced vomiting at three weeks. It was admitted to St. Margaret's Nov. 1, 1909. There was visible peristalsis (Figs. 1, 2, 3). Vomiting was controlled by lavage, but the stomach retained all food. There was constipation. Weight at birth 7 pounds; at five weeks 5 pounds 14 ounces.

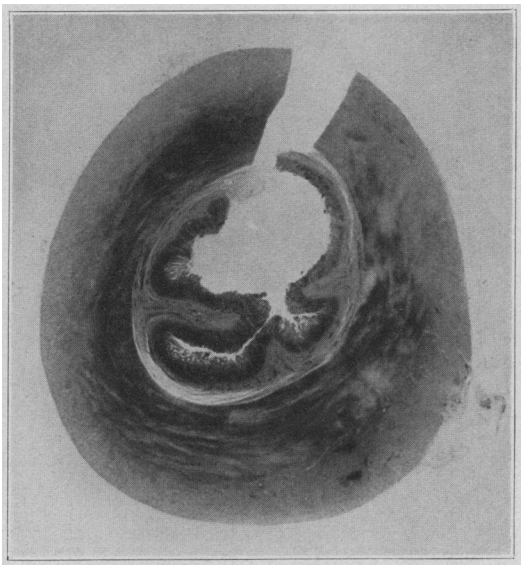

Fig. 9.—Case 4. Transverse section of walls of pyloric canal showing the greatly thickened muscular coat. Hematoxylin and eosin stain. (Mag. x 6.)

Operation (posterior gastro-enterostomy) by Dr. MacDonald, Nov. 4, 1909. Weight at that time 5 pounds 9 ounces. Child died Nov. 5, 1909; no post-mortem allowed. At the operation a firm tumor was found at the pylorus. The stomach was distended and the intestines collapsed.

A third child was born last September. Two weeks later it began to vomit. There was visible peristalsis and Dr. Maby wrote that it was a typical case of hypertrophic pyloric stenosis. On account of their experience with the other child they would not permit an operation, and the child died in October when 6 weeks old.

CASE 4.—C. D.; referred by Dr. Kurth, Schenectady; first child was breast-fed; began vomiting at two weeks; admitted to St. Margaret's Jan. 8, 1910, when five weeks old; weight 7 pounds, 8 ounces. There was visible peristalsis and constipation.

Operation (posterior gastro-enterostomy) by Dr. Elting, Jan. 8, 1910. Weight 7 pounds 1 ounce: "Markedly dilated stomach with a definite hard and firm construction at the pylorus. There is comparatively complete obstruction." The child made a good recovery and was discharged Feb. 6, weighing 8 pounds, 9 ounces. Returned to hospital Feb. 24, with severe ileo-colitis. There was no vomiting. Died of exhaustion Feb. 28, aged two months, three weeks. Autopsy by Dr. Ordway (Figs. 4, 5, 6, 7, 8, 9, 10).

CASE 5.—T. K., referred by Dr. Shaw of Lansingburg. First child and breast-fed. Vomiting commenced on the tenth day. There was visible peristalsis and constipation. The child was admitted to St. Margaret's March 15, 1911, and weighed 7 pounds, 3 ounces, at three weeks. Weight at birth was 8½ pounds.

Operation (posterior gastro-enterostomy) by Dr. Elting, March 19. Weight 6 pounds, 12 ounces. Died March 21. No post-mortem allowed. At operation there was dilated stomach, collapsed intestines, and a large pyloric tumor.

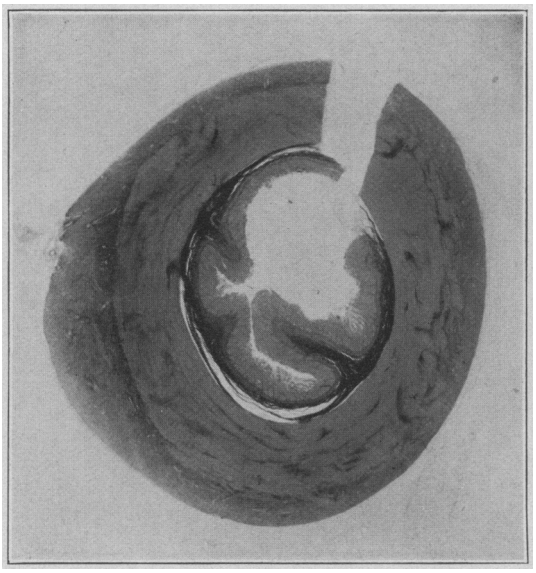

Fig. 10.—Case 4. Section same as Fig. 9 stained by Van Giesen's connective tissue stain. The large amount of muscle tissue is in marked contrast to the connective tissue which appears black. (Mag. x 6.)

CASE 6.—M. C., referred by Dr. Lamont of Catskill; second child; first was strong and healthy; child was breast-fed and began vomiting at two weeks. Weight at birth was said to be 8 pounds; child was weaned at three weeks and all methods of feeding were employed. There were visible peristalsis, constipation and vomiting. The emaciation was extreme.

Admitted to St. Margaret's April 17, 1911, at 8 weeks and weighed 5 pounds, 11 ounces. The vomiting was controlled by lavage but the entire previous feedings would be removed at the next lavage. The child lost 8 ounces in four days.

Operation (posterior gastro-enterostomy) April 21, by Dr. Elting. The operation showed dilated stomach, collapsed intestines and very large pylorus. The stomach was removed and sections prepared by Dr. Ordway (Figs. 11, 12, 13, 14, 15, 16, 17).

CASE 7.—H. P., first child, was breast-fed for first three weeks. Vomiting commenced at second week. Weight at birth 7½ pounds. Admitted to St. Margaret's Aug. 21, 1906, when 6 weeks old and weighed 6 pounds, 14 ounces.

There were visible peristalsis and constipation. The peristalsis was visible for several months and there was a palpable pylorus. Weight September 2, 6 pounds, 7 ounces. The child improved slowly. Lavage was given twice a day and the child was nourished by rectal nutrient enemata. Weight at discharge December 20, 1906, 9 pounds, 5 ounces. He is now a strong, healthy boy.

CASE 8.—R. C. was the second child and weighed at birth 7¾ pounds. He was breast-fed at first and later had a wet-nurse. Vomiting came on suddenly at four weeks. There were visible peristalsis and constipation. Lavage was employed by the mother. The weight at six weeks was 6 pounds. The condition lasted five months, and the child is now apparently healthy and strong.

CASE 9.—E. G. was the second child. The first was strong and healthy. Breast-fed for eight months. Mixed feeding was begun at five months. Weight at birth was 8 pounds. Lowest weight was 5 pounds, 8 ounces.

Vomiting commenced at the third week. There were visible peristalsis and marked constipation. A pyloric tumor was very plainly felt. Weight at 9 months was 8 pounds, 11 ounces. The mother used lavage twice a day for several months.

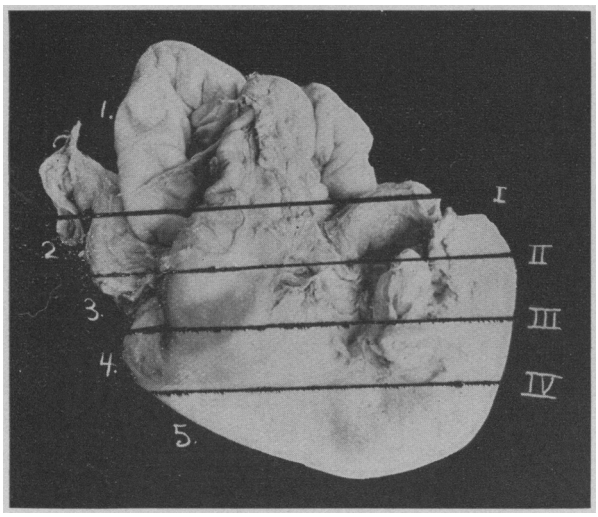

Fig. 11.—Case 6. Stomach, duodenum, and adjacent portion of small intestine of infant. The jejunum is sutured to the posterior wall of the stomach near the pylorus. I-IV indicate the location, and the plane of the sections which divide the specimen into five fragments (1-5) see Figs. 12 and 17 (5/7 natural size).

PATHOLOGY

Introduction

Secretion and digestion are often regarded as the most important functions of the stomach; absorption and assimilation, it is agreed, are chiefly performed elsewhere. We are apt to overlook the functions of storage and motility. The importance of these is emphasized in many animals; in the rat, for example, a large part of the stomach is lined by stratified squamous epithelium with a distinct horny layer, the secretory glands being situated in the small pyloric portion. The muscle coats are

more evident at the pylorus. The circular muscles are usually distinct and reach their maximum thickness just before the beginning of the duodenum. The oblique fibers are not so well defined and may merge into the circular or the longitudinal muscles.

Discussion of Pathological Findings

In these cases most writers describe a tumor occupying the position of the distal part of the pylorus. We shall designate this as the "pyloric canal," which connects the smaller funnel-shaped, or pyloric, portion of the stomach with the duodenum. The proximal end is indicated by a constriction (Fig. 6 x) and the distal by the duodenal submucous glands.

The tumor-like enlargement varies from 1.5 cm. to 3 cm. in length and 0.75 to 1.5 cm. in thickness. It is described by Scudder as a resilient

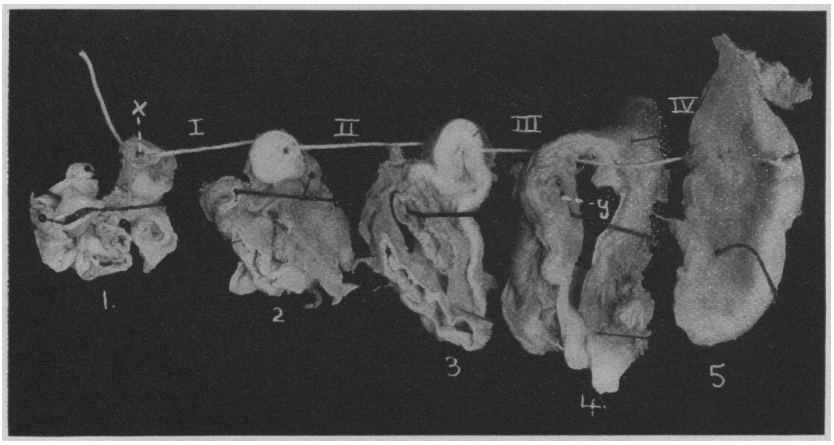

Fig. 12.—Case 6. Five sections showing four serial surfaces 1-I, 2-II, 3-III, 4-IV (see also Fig. 17). A white cord passes through the natural food channel, the walls of which at the pyloric portion of the stomach are greatly thickened (sections 2 and 3); the duodenum is of normal thickness (section I-"X"). A dark cord passes through the gastro-enterostomy opening (section 4-"y"), and indicates the course of the artificial food channel (½ natural size).

cylinder about the size of a hickory nut. Its consistency is very firm, so that Dilz speaks of it as a cartilaginous tube. The tumor is composed of the greatly thickened muscle coat of the "pyloric canal," chiefly of the inner or circular layer of fibers. Our measurements[1] in general agree with those of other observers. No difference was found in the length of the nuclei in the normal control case and in the two cases of hypertrophy of the pylorus, although the thickness in the latter two was somewhat less. The caliber and patency of the lumen of the "pyloric canal" is variable and the measurements depend in part on the method by which

1. See table of measurements.

the specimen is preserved. In Case 4 a tapering probe was gently inserted through the "canal" and where the least resistance was encountered, the circumference of the probe was found to be 1.3 cm. By a similar method Wolbach in one case found a lumen of 2 cm. in circumference. The canal in Case 4 was carefully packed with absorbent cotton and after hardening in 10 per cent. solution of liquor formaldehydi (formalin) the diameter of the lumen was 2 x 3 mm. In Case 6, on the other hand, the specimen had been preserved in toto in 10 per cent. solution of liquor formaldehydi and the diameter of the lumen, which was practically obliterated by folds of mucosa, was 0.14 x 0.49 mm. A normal control hard-

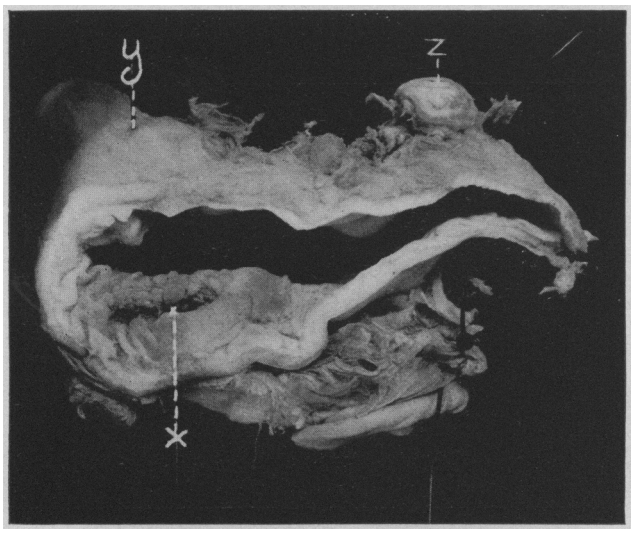

Fig. 13.—Case 6, Section 4, surface 4-IV, showing large patent gastro-enterostomy opening at "x"; the outer surface of a portion of the pyloric thickening is seen at "y"; "z" indicates the esophageal opening (natural size).

ened by slowly injecting through it and then immersing in 10 per cent. solution of liquor formaldehydi measured 4.0 x 4.9 mm. in diameter.

The test of functional patency by hydrostatic pressure is fallacious, for the redundant folds of mucous membrane may act as valves; these and the subsequent shrinkage due to the preserving fluid may give a false appearance of almost absolute impermeability to the pyloric canal. There is little doubt, however, that the lumen is encroached on by the ridges or folds of mucosa projecting into it. In some cases the mucous membrane is hypertrophied.[2] Dilated, cystic and flattened glands with excess of

2. See measurements, Cases 4 and 5, table of measurements.

mucus were noted by Wolbach. Our cases showed an occasional dilated gland, but not more marked than in control cases. Longitudinal folds or ridges project into the lumen of the pyloric canal. Case 4 showed three marked folds and Case 6, four; in the control there were two. Dilz describes three ridges of mucosa projecting into and completely occluding the lumen. Cervix-like projections of the mucosa similar to those bulging toward the duodenum may also be found at the proximal end of the pyloric canal. They occur, though less marked, in normal cases. It is these folds and ridges which form the so-called "pyloric valve."

Various inflammatory changes of the pylorus have been described in these cases. Larkin believes there is an increase in connective tissue.

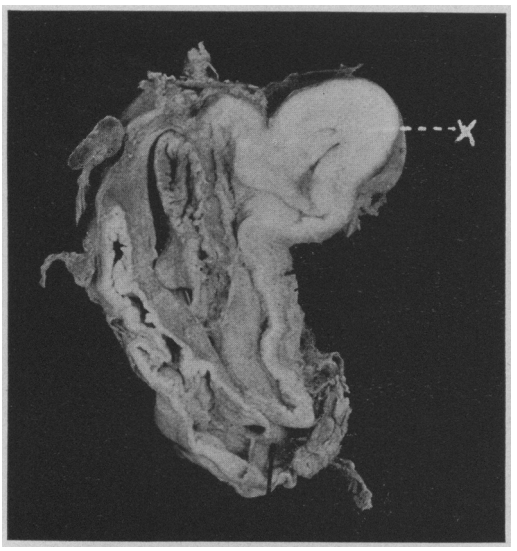

Fig. 14.—Case 6, Section 3, surface 3-III, showing great thickening of the pyloric walls in the vicinity of "x". The walls of the cardiac portion of the stomach are of normal thickness (natural size).

Weill and Pelm consider the hyperplasia of the pyloric muscles secondary to an inflammatory process, while Fredet and Guillinot think that an inflammatory swelling of the mucosa and spasm merely aggravate the stenosis.

In our cases, one, Case 4, showed, microscopically, moderate chronic and slight acute inflammatory changes in the mucous, muscle and serous coats; in Cases 6 and "X" (control) there was no evidence of inflammation of the walls of the pyloric canal. The stomach, in cases of pyloric stenosis in infants, has been described as larger than normal by some writers and others have found it smaller; its wall also in some cases is thinned and in others increased in thickness. In Case 4 the muscles of

the stomach wall near the pyloric canal were 0.7 mm. and in the control 0.9 mm. in thickness.

The duodenum, when mentioned, has been regarded as normal. In our own cases the thickness of the muscle coat was 0.49 mm. in Cases 4 and 6 and in that of the control "X," 0.16 mm.; this, however, may be within normal limits.

AUTOPSY AND LABORATORY FINDINGS

CASE 4.—Post-mortem examination, Bender Laboratory A-10-17, by Dr. Ordway, for Dr. Shaw, at St. Margaret's House, Feb. 28, 1910, at 9:30 a. m.

C. D., aged 2 months and 3 weeks, 7 hours post-mortem.

Body is that of a fairly well developed, poorly nourished, emaciated male infant. Bony framework is everywhere prominent. Skin hangs in folds. Eyes

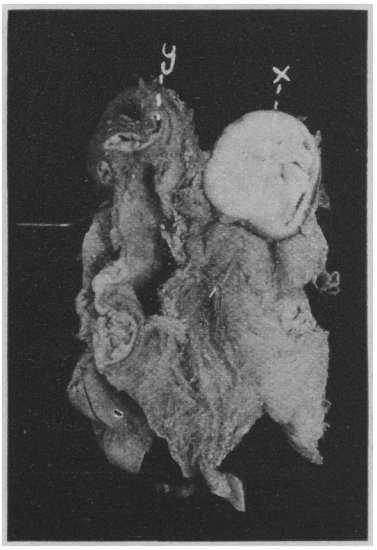

Fig. 15.—Case 6, Section 2, surface 2-II, showing great thickening of the pyloric walls at "x". The jejunum is seen at "y" (natural size).

are sunken. External orifices are negative. There is very slight lividity of dependent portions and no *rigor mortis*. The abdomen is prominent, slightly elevated above the level of the chest. In the median line between the ensiform and umbilicus there is a whitened linear scar 7 cm. in length. There is no edema. The axillary and inguinal glands are slightly enlarged, distinctly palpable; otherwise the superficial glands are negative.

Note.—Autopsy incision restricted to surgical incision above mentioned.

Peritoneal Cavity.—The right lobe of the liver, anteriorly, in the median line and the omental tissue are adherent to the inner aspect of the operative wound above described; otherwise the peritoneal cavity is normally smooth, moist, and glistening. The pleural and pericardial cavities, the heart, lungs, spleen, liver, pancreas, adrenals, kidneys, aorta and genital organs are normal.

Gastro-Intestinal Tract.—The upper portion of the cardiac end of the stomach to the left of the esophagus is adherent to the adjacent peritoneum of the diaphragm by delicate fibrous adhesions. The stomach is small, does not reach

the umbilicus and is empty. Rugæ are prominent. The walls of the pylorus are greatly thickened for a distance of 2 cm., having the consistency of a firm, hyperplastic gland and forming a distinct ovoid tumor-mass in the region of the pylorus. This is in marked contrast to the walls of the duodenum, which are thin and collapsed, and to the walls of the cardiac portion of the stomach, which are of normal thickness. A tapering probe is inserted through this thickened portion of the pylorus without difficulty until a point 1.3 cm. in circumference is reached; that is, the circumference of the pyloric opening after death, with practically no pressure, is 1.3 cm. On the posterior wall of the stomach 1.5 cm. below the pyloric opening, which is marked by protruding folds of mucous membrane, is a large opening into the jejunum; the latter is firmly adherent to the posterior wall of the stomach (Figs. 4 and 5. The diameter of this opening is 1.4 cm. Pressure on the gall-bladder causes yellowish fluid to exude into the duodenum from the ampulla. The small and large intestines contain a small amount of bright yellow, semi-solid material with numerous

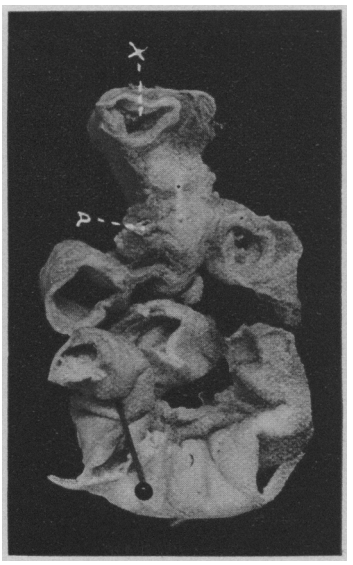

Fig. 16.—Case 6, Section 1, surface 1-I, showing duodenum "x"; adjacent to the pylorus is the pancreas "p"; the other openings represent sections of the duodenum and coils of jejunum (natural size).

light yellowish shreds. Peyer's patches and solitary follicles are prominent. The intestines are markedly distended by gas.

Anatomic Diagnoses.—Emaciation, hypertrophy of pylorus, gastro-enterostomy; healed incised wound of abdominal wall, adhesions of omentum and liver to abdominal wall.

Microscopic Examination.—The stomach, duodenum and adherent jejunum are carefully filled with cotton saturated with 10 per cent. solution of liquor formaldehydi and these organs hardened in as nearly normal shape as possible. Particular care is taken that the stenosed pyloric and the artificial gastro-enterostomy openings may be prevented from distortion by shrinkage (Figs. 4 and 5). Fragments removed from the wall of the "pyloric canal," as indicated in Figure 5, were cut in longitudinal and transverse sections and stained by hematoxylin and eosin and Van Giesen's connective tissue stains (Figs. 6 to 10). The great thickening of the wall is due to an increase in muscle tissue and

chiefly to the circular fibers. There is slight atrophy of the glands in the mucosa and an increase in the interglandular tissue. An occasional gland is distended by lymphocytes and polymorphonuclear leukocytes. Scattered through the submucosa, muscularis and serosa, are numerous foci of lymphoid cells and few polymorphonuclear leukocytes; in many places similar cells distend the perivascular lymphatics. There is no increase in connective tissue. In the serosa there is a small amount of old yellowish pigment.[3]

CASE 6.—Michael C. Bender Laboratory, A-11-42. Post-mortem examination performed by Dr. Donhauser and restricted to surgical incision and examination of stomach, duodenum and jejunum.

Stomach, duodenum and portion of adjacent small intestine of an infant. The specimen was received in toto in 10 per cent. solution of liquor formaldehydi. It is somewhat shriveled by the preserving fluid and measures 7x5 cm. In the region of the pylorus is a hard tumor mass measuring 2x1.5 cm. The jejunum is sutured to the stomach on the posterior aspect near the pylorus (Fig. 11). Serial sections as indicated, I, II, III, IV, are made and

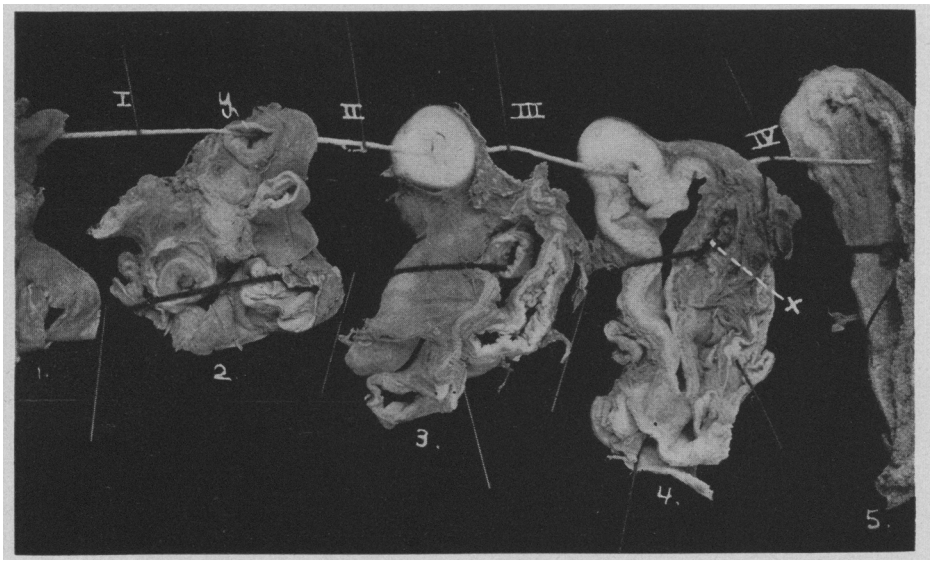

FIG. 17.—Case 6. Three sections and portions of two others showing three serial surfaces, 2-I, 3-II, 4-III. White cord passes through the natural food channel, the walls of which at the pyloric portion of the stomach are greatly thickened (3 and 4); the duodenum is of normal thickness (2 "y"). A dark cord passes through the gastro-enterostomy opening (4 "x") and indicates the course of the artificial food channel (5/7 natural size).

Note.—The surfaces here seen are the reverse of those shown in Figures 12 *et seq.*

divide the specimen into five fragments 1, 2, 3, 4, 5 (Fig. 12). A white cord passes through the natural food channel, the walls of which at the pyloric portion of the stomach are greatly thickened (Figs. 12, 14, 15, 17), sections 3 and 2. The wall of the duodenum is of normal thickness (Fig. 12, Section 1 "X", and Fig. 16).

3. These inflammatory changes may have been secondary to the healing of the gastro-enterostomy wound. Patient 6 died before such change could have occurred.

A dark cord passes through the gastro-enterostomy opening and indicates the course of the artificial food channel (Figs. 12 and 17). The thickening of the pylorus is not abrupt on the stomach side, but involves almost the entire pyloric portion of the stomach and gradually increases in thickness as the duodenum is approached. At the junction of the duodenum, where the submucous (Brunner's) glands begin, this thickening is at a maximum and here ends abruptly. The gastro-enterostomy opening is large and the canal is patent. The intestine and stomach are firmly approximated and there is no leakage or suppuration.

Microscopic Examination.—Fragments removed from the thickened wall of the pyloric canal, cut in transverse and longitudinal section, and stained by hematoxylin and eosin, show no pathologic change other than the great increase in the thickness of the muscle coat, chiefly of the circular fibers. These fibers appear normal.[4]

CASE "X" (Control).—Post-mortem examination by Dr. Kellert. Bender Laboratory A-11-47. May 25, 1911. Name unknown. Female infant, 8 months old. Child died with cerebral symptoms. Examination restricted to chest and abdomen, all the organs of which appeared normal. Stomach and duodenum hardened by carefully injecting and then immersing in 10 per cent. solution of liquor formaldehydi. Fragments were removed from the wall of the apparently normal pyloric canal, sectioned transversely (Fig. 18) and longitudinally (Fig. 19,[5] and stained by hematoxylin and eosin. Microscopic examination reveals no pathologic changes. Although this child was 8 months old, it was taken as a control to Cases 4 and 6, which were, respectively, 11 weeks and 8 weeks; notwithstanding this great difference in the age, the walls of this normal pyloric canal, as shown in Figures 18 and 19, and as indicated in the table of measurements, are in striking contrast to those of the pyloric canals in the cases (4 and 6) of muscular hypertrophy of the pylorus.

TABLE OF MEASUREMENTS, CASES 4, 6, AND "X" (CONTROL)

	Case 4 mm.	Case 6 mm.	Case "X" (Control) mm.
Length of pyloric tumor "pyloric canal"	16	18	11
Thickness of tumor, external diameter	12	14	9
Thickness of muscle layers	2 to 3	3 to 3.5	1 to 1.6
Thickness of circular fibers	1.64	2.57	0.98
Thickness of oblique and longitudinal fibers	0.56	0.45	0.16
Diameter of lumen	3.0 x 2.0	0.49x0.14	4.9x4.0
Width of serosa	0.16	0.13	0.08
Thickness of mucosa and submucosa, from top of folds	1.8 to 3.5	3.5	2.0
Thickness of mucosa and submucosa, base of folds	1.06	1.1	0.82
Thickness of epithelium	0.41	0.90	0.49
*Mucosa, number of folds	3	4+	2+
Stomach, thickness of muscle coats near "pyloric canal"	0.7	—	0.9
Duodenum, thickness of muscle coats near pylorus	0.49	0.49	0.16
Muscle nuclei, width	1.9 to 2.5	2.0 to 2.5	3.8
Muscle nuclei, length	28 to 34	28 to 34	28 to 34

*When small irregularities are present as well as the distinct folds, they are indicated by + following the number of folds designated.

4. See also table of measurements.

5. Owing to a mistake in reproducing, Figures 6 and 7 were reduced from mag. x 6 to mag. x 4, while the "control," Fig. 19, was reduced from mag. x 6 to mag. x 5½. Because of this the contrast is not evident in these longitudinal sections, but is shown well when Figs. 9 and 10 are compared with Fig. 18.

PATHOGENESIS

There are a number of theories and conjectures concerning the pathogenesis of this condition. The earlier writers considered it a congenital organic defect due to excess of development. Cautley claimed that this was a primary hypertrophy, an instance in which "Nature in her extreme anxiety to provide an efficient pyloric sphincter, has over-exerted herself, and produced too great an amount of muscular tissue."

Flynn considered it an atavistic deformity analogous to the gastric mill in insects and crustacea and the gizzard in birds, or a reversion to the edentate pylorus seen in the armadillo. The assumption that there was a true tumor has not been proved histologically.

The term "congenital" is misleading as no true hypertrophy has been found in fetal life or at birth. The symptoms rarely if ever appear before

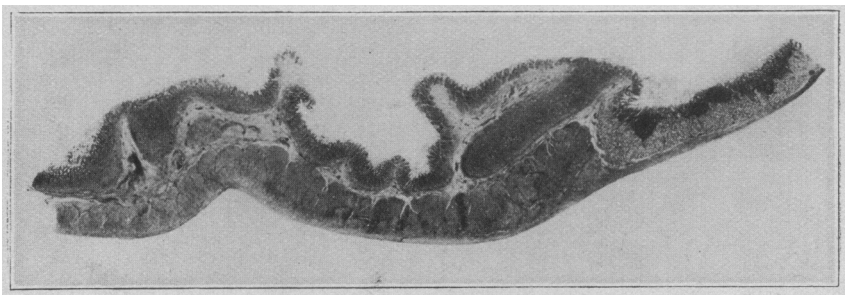

Fig. 18.—Case "X" (Control).—Longitudinal section of wall of normal pyloric canal. Compare with Fig. 6 (Mag. x 5½).

the end of the first week, which is irreconcilable with the hypothesis of a congenital obstacle.

The later writers, as a result of the study of a large number of cases, are almost unanimous in the opinion that the hypertrophy or muscular hyperplasia is a secondary manifestation. This is the result of a spasm or incoordination of the gastric muscles. The spasm may start as a reflex spasm of the gastric mucosa incited by erosions, fissures, over-activity, hyperacidity, too high fat in the food, increased nervous irritability, etc.

Still has advanced an ingenious theory that the spasm is a kind of "stomach stuttering" in which the muscular action of both ends of the stomach is antagonistic. This he claims is analogous to the disturbance of coordination which is evident in hiccough or in the stuttering which is almost physiologic in the young child who is learning to talk.

Case 1 of our series shows conclusively that spasm of the pylorus will produce a marked hypertrophy.

COURSE

The course is slowly progressive and the condition may last from three to twenty months. The duration depends on the degree of stenosis. In cases in which recovery occurs the vomiting gradually subsides, constipation improves and the baby begins to gain in weight. This disease must be considered a very grave affection. Dr. John Thomson's statistics show that four children recovered out of seventeen treated medically, or a mortality of about 76 per cent., while eight recovered out of twenty-three treated surgically, or a mortality of 65 per cent. These figures are a little higher than those given by Ibrahim, who showed a mortality of 46 per cent. of patients treated medically and 54 per cent. surgically. In this connection it may be stated that successful surgical cases are more liable to see the light of publication than those resulting fatally.

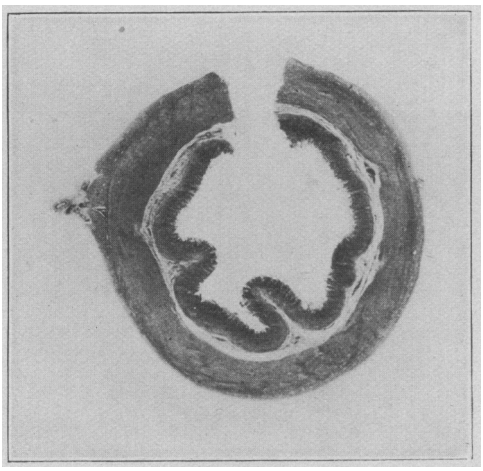

Fig. 19.—Case "X" (Control).—Transverse section of wall of normal pyloric canal. Compare with Fig. 9 (Mag. x 6).

DIAGNOSIS

The diagnosis is not difficult. The history, age of the child, visible peristalsis and other symptoms should at once arouse suspicion. In acute gastric conditions there is always more or less temperature and there is no constipation. It should not be mistaken for dyspepsia from overfeeding, or the habitual vomiting of Finkelstein, in which there is a congenital hyperplasia of the gastric mucosa. The latter condition is caused by too high fat in the food and the vomiting comes on immediately after feeding. True congenital malformations are rare. These are the result of an arrest in development or inflammatory process in fetal life. The baby only lives a few days after birth. The vomitus will contain bile if the obstruction is below the pylorus.

The condition most liable to be confounded is that of functional spasm of the pylorus or pylorospasm. Vomiting is the chief symptom in both these conditions, but in the simple spasm the stools are more frequent and bulky and the child may even have diarrhea. It occurs later in infancy and there is never any marked visible peristalsis and never any palpable pylorus. There may be bile in the vomitus at times.

TREATMENT

The treatment should be individual and each case must be studied intelligently.

The *dietetic* treatment consists in using the breast milk if possible. If the milk is rejected immediately after nursing, it might be pumped from the breast and given in small quantities with a medicine dropper. Some authors advocate giving it ice cold. If the breast milk contains too much fat it can be allowed to stand a few hours and the fat removed and the bottom milk given. Peptonized skimmed cow's milk will sometimes be retained. Whey, malt soup, albumin-water, acid-milk, etc., may also be tried. It is best to give small amounts at frequent intervals. Feeding by gavage should be employed.

The *mechanical* treatment is most important. Washing the stomach out several times a day with cold water removes the contents of the stomach and prevents fermentation and other changes and also tends to relax the muscles of the stomach. Any intelligent mother after a few lessons can perform lavage successfully. In two of our cases the mothers kept this up for many months without any difficulty. The use of hot compresses over the abdomen after feeding has also been recommended.

The *drug* treatment is unsatisfactory. Alkalies can be administered after feeding to reduce the acidity. The use of small doses of atropin, opium or cocain about half an hour before each feeding has been recommended by some authors.

Surgical treatment should be employed when no further good can be done by conservative methods. Surgery has added much to our knowledge of the pathology of this condition. There are several operations recommended to relieve this condition. A posterior gastro-enterostomy seems to be the choice of most surgeons.

SUMMARY AND CONCLUSIONS

1. The pyloric tumor is due to a thickening of the walls of that portion of the pylorus adjacent to the duodenum—the pyloric canal.

2. This thickening is caused by an hypertrophy of the muscle coat, chiefly the circular fibers.

3. The cause of this hypertrophy is not evident.

4. Inflammatory changes may or may not be present.

5. Spasm of the (hypertrophied) muscles, due to local or reflex cause, may be a factor in producing stenosis.

We are indebted to Dr. Ellis Kellert for assistance in the technical work and to Mr. J. A. Glenn for photomicrographs.

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