CONGENITAL PYLORIC STENOSIS*

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THE CAUSE of congenital pyloric stenosis is not known, although there are several theories. Fraser[1] subscribes to Thomson's[2] theory of the tumor being due to overaction of the stomach musculature and hence hypertrophy of the pylorus. Hurst[3] says: "It is clear that the obstruction is due to something which produces a much more powerful resistance than the simple absence of relaxation (achalasia). The only explanation is spasm." He believes "it possible that the tendency to spasm of the pylorus is the expression in an extremely exaggerated form of the constitutional condition called hyperthenic gastric diathesis, which manifests itself by hypertonus, hyperperistalsis and hyperchlorhydria, and occurs much more commonly in men than in women. The diathesis is often present in several members of a family—this would explain the rare instances in which two and even three or four members of the same family have hypertrophic pyloric stenosis." As opposed to Hurst's theory of hyperthenic gastric diathesis and those theories which deal with the basis of its being in a hypertonic child, one has the subsequent history of the cases that are cured by operation. My knowledge of the after-results is only of those cured by operative measures, and the subsequent history of these cases is that of a normal individual. In no case that we have been able to trace in our series has there been any subsequent history of digestive disturbance that would suggest a disability such as duodenal or gastric ulcer, or other disease which may be associated with hypertonicity. Strachauer,[4] and others, describe the lesion as having been found in a seven-month fetus and in a stillborn child. Cockayne[5] believes the condition has a genetic basis, though the way it is inherited is still uncertain and an environmental factor may be necessary for its production in addition to a generic one. The early age at which a well-marked tumor has been found, clinically and at operation, eight days in a case of this series, weighs heavily in favor of the condition being present before and at the time of birth; and this would seem to point to the genetic influence. Added facts, such as more than one case in a family (in our series five siblings apart from twins), speak strongly for the genetic etiology.

It is a condition that is most frequently found in the first-born of a family. In this series 51.8 per cent were first-born. This agrees with statistics published by other authors, but it seems to be a high percentage and challenges a rational explanation. The percentage of first-born children in the total number of births is greater in urban communities, where families are small. Yet even in such centers first-borns predominate (Table I).

There is reason to believe that more males are conceived than females,

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TABLE I

Order of Birth	Cases of Pyloric Stenosis		Average Births, Toronto	
	Number	Per Cent	Number	Per Cent
1st	144	51.8	4,748	41.2
2nd	61	21.9	2,730	23.8
3rd	41	14.7	1,627	14.1
4th	16	5.8	916	8.0
5th and over	16	5.8	1,476	12.9
Total	278	100.0	11,497	100.0

since a study of early miscarriages shows the percentage of males very high until figures at the time of birth show a slight preponderance only of males. Males are, therefore, less robust than females and have a poorer chance of surviving.

Twins, or a sibling twin, may have the condition. Sheldon[6] states that in 1,000 cases of congenital pyloric stenosis, 1–43.4 cases is a twin sibling, while the ordinary 1,000 birth-ratio is 1–80 births. In our series, one case in 36.3 cases is a twin, while the ordinary ratio for Canada is 1–83.4.[7] Cockayne[5] describes two pairs of twins who were first cousins; all four were proved to have congenital pyloric stenosis at operation. The occurrence of the condition in monozyotic or maternal twins is almost positive proof of a genetic factor being the producer of the condition. In the cases recorded,

TABLE II

SUMMARY OF 12 CASES OF TWINS WITH PYLORIC STENOSIS

Hospital for Sick Children, Toronto

Year	Initials of Twins	Sex of Twins	Type of Twinning
1914–1915	R. R.	♂* ♀	Fraternal
1917–1918	R. McG.	♀* ♂	Fraternal
1925–1926	B. M. (Twin died. Parents describe the twins as identical, December, 1939)	♂* ♂	Doubtful
	A. G.	♂* ♀	Fraternal
1927–1928	Harvey and Grant I. (Reexamined November, 1939)	♂* ♂	Fraternal
1930–1931	Ross and Donald McF. (Reexamined December, 1939)	♂* ♂	Fraternal
1933–1934	R. W.	♂* ♀	Fraternal
1935–1936	William and Harry L. (Reexamined December, 1939)	♂* ♂	Fraternal
1937–1938	P. W.	♂* ♀	Fraternal
1938–1939	Garry and Glen T. (Examined 1938)	♂* ♂*	Identical
	Robert and Richard G. (Examined 1938)	♂* ♂*	Identical
	Erla and Arla C. (Examined 1939)	♀* ♀	Fraternal

* The symbol * indicates the condition.

there are many reports of pairs of twins each having the condition, but few have been studied sufficiently to prove the type of twinning present.[7] There seems to be a reasonable doubt that identical twins are not both affected when the condition is found in one. In our series, there were 12 pairs of twins, two pairs being identical twins. All four had a tumor, proved at operation. In all fraternal twins, one sibling only was affected (Table II). Multiple births are known to produce a disturbance of growth, and this may prove to be an environmental factor influencing the hypertrophy of the plyorus.

This series includes all cases of congenital pyloric stenosis reaching the Hospital for Sick Children. There were 28 cases which had no operative treatment, some being diagnosed only at autopsy, and who had received treatment for some other condition. Of those cases treated without operation, ten were discharged as improved and 18 died. Of these cases dying, the cause of death is given as bronchopneumonia, otitis media and septicemia, marasmus and acute intoxication. Of the cases submitted to operation, *viz.*, 402, 52 died subsequently from intoxication, malnutrition, decomposition, otitis media

TABLE III

CASES BY YEARS

	No. of Operations	Cured	Died	Not Operated Upon	Cured	Died
1914	2	2				
1915	1		1	1	1	
1916	3	2	1	3	3	
1917	7	5	2	2	1	1
1918	11	8	3	11	5	6
1919	9	8	1			
1920	6	4	2			
1921	15	10	5			
1922	15	12	3	1		1
1923	12	11	1	1		1
1924	27	22	5			
1925	14	12	2			
1926	31	24	7			
1927	15	10	5	1		1
1928	25	24	1	1		1
1929	30	28	2	1		1
1930	14	12	2	2		2
1931	20	19	1	1		1
1932	22	18	4	1		1
1933	14	14		1		1
1934	9	7	2	1		1
1935	19	19				
1936	10	10				
1937	12	12				
1938	23	21	2			
1939	36	36				
Totals	402	350	52	28	10	18

and general infection, and in a few cases, there was a peritonitis due directly to the opening of the duodenal mucous membrane (Table III and IV).

TABLE IV

CONGENITAL PYLORIC STENOSIS

Hospital for Sick Children, Toronto—430 Cases

Males 352—81.9% Females 78—18.1%

Place in family not known—13. Others as below

1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th
213	84	61	31	14	6	5	2	1	1
51.1%	20.1	14.6	7.4	3.3	1.4	1.2	0.47	0.24	0.24

	Treated by Operation	Died	Mortality
1914–1923 incl.	81	19	24.4%
1924–1933	212	29	13.0%
1934–1939	109	4	3.6%
1914–1939	402	52	12.9%
Not operated upon	28	18	64.3%

The symptoms of pyloric stenosis are occasionally found within a few hours after birth. Vomiting has been known to have occurred as early as this and to have continued through until the condition was cured. Of the symptoms that are present in the disease, vomiting will be found to be one of the most outstanding. It is of a special character. Inasmuch as it is the result of a pyloric obstruction, it will be large in quantity and be free of bile discoloration. Loss of weight, which occurs in the normal infant for two or three days after its birth, will be found to continue on until, at three weeks, the infant may be less in weight than at the time of its birth. In spite of the vomiting and the loss of weight, the patient will be found to have a splendid appetite. It takes its nourishment with avidity. The stool is found to be bile-stained, but may be small and scanty and bearing some relation to the amount of food that gets through the pylorus. The infant is dehydrated, its skin is shrunken, and it has very little subcutaneous fat. It is fretful and cries a great deal. Examination of the abdomen shows the abdomen scaphoid with visible peristalsis to be seen to sweep from left to right, gaining in size and activity with the increased amount of food taken into the stomach. When a fastigium has been reached an explosive vomiting occurs which partially empties the stomach. The vomiting is characteristic in that the propulsion is such as to throw the vomitus a considerable distance from the lips of the infant. A stream may be shot out as far as 18 inches. It comes out through the nasopharynx and nostrils. On palpation of the abdomen, it is possible to feel an enlargement in the pyloric region. The tumor is to be felt by examination of the hypergastric region on the right side. Digital examination, just below the level of the costal margin and just lateral to the edge of the rectus, may palpate a hard nodule about the size of a large hickory nut. This nodule is movable and is more easily felt when

the peristaltic wave in the stomach has lifted up the pyloric nodule to a position where it is more superficial.

Blood and urinary tests have no characteristic changes of diagnostic value.

The diagnosis of this condition should be relatively simple. The history of loss of weight, vomiting, visible peristalsis, in an infant, is to be considered as a probable case of pyloric stenosis. The earliest case that has been operated upon by the writer was one eight days of age. It was found to have a well-marked and definite tumor. The symptoms may not appear until two or three weeks after birth and then a history is given of a sudden onset. At operation, a well-marked tumor may be found, which makes it difficult to explain why there should have been no obvious symptoms before the sudden onset.

In the diagnosis of a case of pyloric stenosis the examiner should, in suspected cases, have the patient stripped of all clothing, lying on its back, with a good light on the abdomen. The infant should be given some sweet water or glucose solution in a nursing bottle, and, as it nurses, the upper part of the abdomen should be observed. As the patient fills his stomach the epigastric region will be seen to fill out, and, presently, waves of peristalsis will be observed to originate in the left side of the abdomen and sweep across to the right. With an increasing volume entering the stomach the waves will be seen to increase in magnitude and speed. When they become very pronounced the patient is seen to stop nursing, draw its legs up and cry, and eventually when the waves have become of great magnitude, explosive vomiting occurs. The stomach being emptied of the larger part of its contents, the child will immediately again take the bottle in an endeavor to get some nourishment. It is an opportune time to palpate the right hypocostal region to examine for the presence of the plyoric tumor. The left hand lifting in the right flank of the patient assists the second finger of the right hand to identify the tumor in the epigastrium. An examination which has demonstrated visible peristalsis with projectile vomiting and the identification by palpation of a pyloric tumor may be considered as sufficient for diagnosis of an obstructive congenital pyloric lesion. A thin barium mixture may be given in order to obtain roentgenographic verification, but this type of examination is not to be encouraged as there is a certain danger associated with the giving of barium to an infant who has pyloric stenosis. The liability of the barium producing an acute obstruction in the very narrow lumen of the pylorus is a real danger. A proper physical examination of the infant will give all the needed information to enable one to make an accurate diagnosis.

The condition is best treated by operation. It has been claimed that the condition is amenable to well-supervised medical treatment. Yet such treatment, when successful, is prolonged and difficult, demanding the utmost in the parents' devotion in the care of a crying infant over many months. Whereas operative interference relieves the obstruction of the pylorus im-

mediately, and one may expect the function of the stomach to be immediately restored in large part or completely. This condition lends itself perfectly to surgical interference. It is surgery's best operative procedure. Any infant who has pyloric stenosis should be operated upon for the relief of the obstruction.

Steps must be taken to make it a good operative risk. The most important factor in the treatment of the infant is an early recognition of the condition. Surgery undertaken before there has been serious interference with the patient's health gives the best results, as is well demonstrated by the reports of Barrington Ward.[8] When the patient has been allowed to experience a starvation of sorts for weeks its general condition will be much affected. Dehydration will be present and it must be overcome by the administration, intravenously, of suitable fluids, such as normal saline, glucose 5 per cent, and blood. When acidosis is present whole blood transfusions are indicated. The infant can then be considered safe for operation. There are no contraindications for operation except the unfavorable condition of the infant, and this can generally be corrected by intravenous therapy in from 24 to 48 hours, to a degree that will permit the surgical procedure.

When operation is finally decided upon, the question of an anesthetic becomes of importance. In this series, almost all types of anesthetics have been tried. The experience of the writer is that ethyl chloride and ether, or ether without the ethyl chloride to initiate anesthesia, is much the best method. Local anesthesia has not proved satisfactory, as it is very difficult to restrain a struggling infant. Inasmuch as the operation is simple and can be performed in a very short period of time under favorable conditions, a general anesthetic is the best. It will be found most useful to have a small section of board about 24 inches long and six inches wide. This board is padded. The infant is laid upon this, with the legs bound to the board, somewhat in the manner of a papoose. This arrangement controls the infant, so that if it be not deeply anesthetized it cannot flex its thighs or move its legs. Before the anesthetic is begun a catheter should be introduced into the patient's stomach and the stomach contents withdrawn. The stomach having been emptied, the anesthetist may proceed with the administration of ether. Conforming to the size of the infant's face, a simple wire mask covered with ten thicknesses of gauze is used.

Operative Procedure.—The skin is prepared by washing with soap and water, drying, and then applying 2½ per cent iodine in 70 per cent alcohol. Standing on the right of the patient, an incision is made parallel to the right costal margin and about one-half inch below it. The inner extremity of this incision should be lateral to the edge of the rectus, and the incision need not be longer than 2½ inches. The skin and deep fascia having been divided, the external oblique is split in the direction of its fibers, the internal oblique is then identified and split in the direction of its fibers. The transversalis fascia and peritoneum are generally closely applied and can be divided as

one. When this incision has been properly made through its entirety, the liver will be found to be exposed and will be blocking any attempt on the part of the abdominal parietes to extrude through the wound. A finger can then be passed around the edge of the liver and one can immediately identify the hard tumor of the pylorus. A pair of ring forceps without rubber or a pair of sponge forceps are then used to grasp the stomach, and by delivering part of this the pyloric tumor is, usually, easily drawn through the wound. It is sometimes found that the tumor is very large and the wound may have to be enlarged to permit the delivery of it outside the abdomen. The left thumb and forefinger then grasp the duodenum at the lower extremity of the tumor and squeezing the fingers together presses the lower part of the tumor presenting in the duodenum toward the stomach. The tumor will be found to have an avascular line on its superior border. This superior border is concave, the whole tumor having the appearance of a large cashew nut. A simple incision about half an inch long and just deep enough to allow the engaging of the closed tips of tendon scissors is all that is required. The closed points of these tendon scissors engaged in the wound are spread. The tumor will be found to split from end-to-end, and through its thickness, until the mucous membrane of the pylorus is seen appearing as a white and glistening structure in the bottom of the split. When the tension is taken off the scissors in the split tumor this mucous membrane will be found to bulge into the split. It is not necessary to divide every last strand of the tumor at its lower extremity, and it is courting danger to attempt it. All pyloric tumors project into the duodenum, as a cervix into the vagina. When one endeavors to split the extremity of the tumor at its duodenal end, he will almost invariably open the mucous membrane and have soiling of the wound from stomach content, in addition to hemorrhage coming from the torn membrane. When this event occurs it becomes necessary to suture this small opening as it has been known to produce peritonitis, which has ended fatally. The writer believes that the squeezing of the duodenal end of the tumor, in such a fashion that it is expressed towards the stomach, permits one, when the tumor is split with the scissors, to have it sufficiently divided for all practical purposes. When the tumor has been split in the avascular region, it will be found there is no hemorrhage occurring that requires any attention. The tumor is dropped back into the abdomen. No steps are taken to cover the split in the tumor by grafts or fat or strips of muscle. The lower edge of the liver slides back into position, and one can close the peritoneum and different muscle layers without interference of abdominal contents extruding through the wound. The writer uses No. 0 catgut to close the different layers, and No. 000 chromic catgut in the skin. The wound is dressed by applying a small gauze strip over bismuth-formic iodide powder. This dressing is slightly larger than the wound. Mastisol is applied, surrounding the wound, and a section of gauze bandage is placed over the dressing and pressed on to the drying mastisol and trimmed off, so that the

whole dressing is not more than two inches wide and three or four inches long.

Following the operation a transfusion of whole blood is administered when it is felt dehydration or shock requires it.

Postoperative Feeding.—One-half ounce of breast milk four hours after operation. Feeding increases one drachm every four hours until a total of 3 oz. q. 4 h. x 5 is given, unless the child vomits, in which case the amount of feeding remains the same without any increase at the end of the four-hour period. After the amount of the feeding has reached 3 oz. q. 4 h. x 5, it is changed back to the feeding given previous to operation, or started on 2 per cent L.A.M., 4–5 oz., q. 4 h. x 5.

We have found this treatment satisfactory and see no reason why we should change it. Recently, it has been suggested that no feeding be given for 24 hours following the operation. This is based on the observation that infants vomit during the first 24 hours when they have been fed, and that barium administered shortly after operation is not emptied promptly from the stomach. In spite of this suggestion we feel that the infant should be fed four hours after operation and that occasional vomiting should not interfere with the routine of feeding.

We have had the cooperation of the staff in pediatrics, in the care of these cases. The cases are admitted to their wards, and when operation is performed the pediatrician still directs the feeding and general care. Without this cooperation the problem would be very difficult for the surgeon.

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DISCUSSION.—DR. WILLIAM E. LADD (Boston, Mass.): There is no question but that the advance in treatment of congenital pyloric stenosis in the last 25 years has been one of the most striking improvements in the whole surgical field. I find this a difficult paper to discuss because the treatment has become so well standardized, because there are few controversial points, and because I can find little or nothing to disagree with in Doctor Robertson's paper.

RESULTS IN 765 CASES OF PYLORIC STENOSIS

Years (Inclusive)	Number of Cases	Deaths	Mortality Per Cent
1915–1922	125	15	10.4
1923–1928	150	11	7.0
1929–1931	151	3	2.0
1932–1935	162	8	4.9
1936–1939	177	1	0.56

During the early part of the period, after we had adopted the present operation of pyloromyotomy, I believe our high mortality was largely due to having poor risk patients and to an inadequate knowledge of how to combat dehydration. Both of these handicaps have been largely overcome. The pediatrician now realizes that this condition is a surgical problem and refers the patient earlier, and our knowledge of fluid balance has been greatly improved. However, in spite of this situation, complications still arise which sometimes prove fatal.

In the earlier series, reported in the literature, some fatalities were reported from hemorrhage from the cut pylorus. This can be avoided by selecting, for the incision, the bloodless area on the superior surface of the pyloric sphincter. Cutting through the mucous membrane at the duodenal end of the pylorus is an error which it is easy to make. I think every member of my staff, including myself, has made this mistake once—but only once; and in no instance has a peritonitis resulted. This error is serious only if it is not recognized—then a fatality will probably follow.

Disruption of the wound has taken place several times in our series and in one instance caused a fatal peritonitis.

The several factors involved in this failure of wound healing are probably subclinical scurvy, low protein and edema, and technic of making and closing the incision. During the last two years we have given all these patients vitamin C before and after operation to overcome the first factor. We have probably paid too little attention to the question of low serum protein. We have varied our technic of wound closure. The last type of closure employed has consisted in medial retraction of the rectus muscle and closure by layers with silk supplemented by stay sutures to, but not through the peritoneum. Doctor Robertson's suggestion of a gridiron incision under the costal border and outside the rectus muscles has many obvious advantages and I plan to adopt it at the next opportunity.

The suggestion recently raised by Faber and Davis, of San Francisco, that feeding be withheld for a period of 24 hours, or more, does not appeal to me as being sound. Our present feeding regimen which is similar to Doctor Robertson's has proved eminently satisfactory.

DR. ALBERT O. SINGLETON (Galveston, Tex.): I wish to discuss one phase of the problem of pyloric stenosis which Doctor Robertson has so ably presented, and this has to do with the incision which is employed.

The accompanying illustration (Fig. 1) illustrates the type of incision we have found very advantageous. It is made quite far to the right, since the pylorus in infants will be found much further to the right of the midline than in adults. With the posterior sheath of the rectus split in a transverse direction, sufficient room is obtained. With the pylorus delivered into the wound, there is no room for the evisceration of the small intestine and omentum, which may happen in these infants with their tremendous intra-abdominal pressure. This is particularly noticeable under local anesthesia which we are in the habit of using. Also, postoperative disruption cannot occur. Doctor Robinson's suggestion of making the incision still further to the right, even outside of the rectus muscle, may be a very good one.

DR. EDWARD J. DONOVAN (New York, N. Y.): I was very much interested in Doctor Robertson's paper on pyloric stenosis, since he has brought out a great many facts, particularly in regard to the twins, that I have not heard discussed in relation to pyloric stenosis before. We have had no identical twins in our series, and my experience with this condition consists

of about 425 cases that I have operated upon personally. Our cases have followed those of Doctor Robertson very closely. We have found that the condition occurs about five to seven times more often in boys than in girls, that is, there are between 14 and 18 girls in each 100 cases. All nationalities are represented, and in a city where there is a large colored population there are about two colored children in each 100 cases.

We do not know any more about the etiology than anyone else, but have felt, because we have had several cases in premature infants with pyloric tumors that were very easily palpable, that the tumor was congenital in origin, and an associated pylorospasm caused the onset of symptoms between the

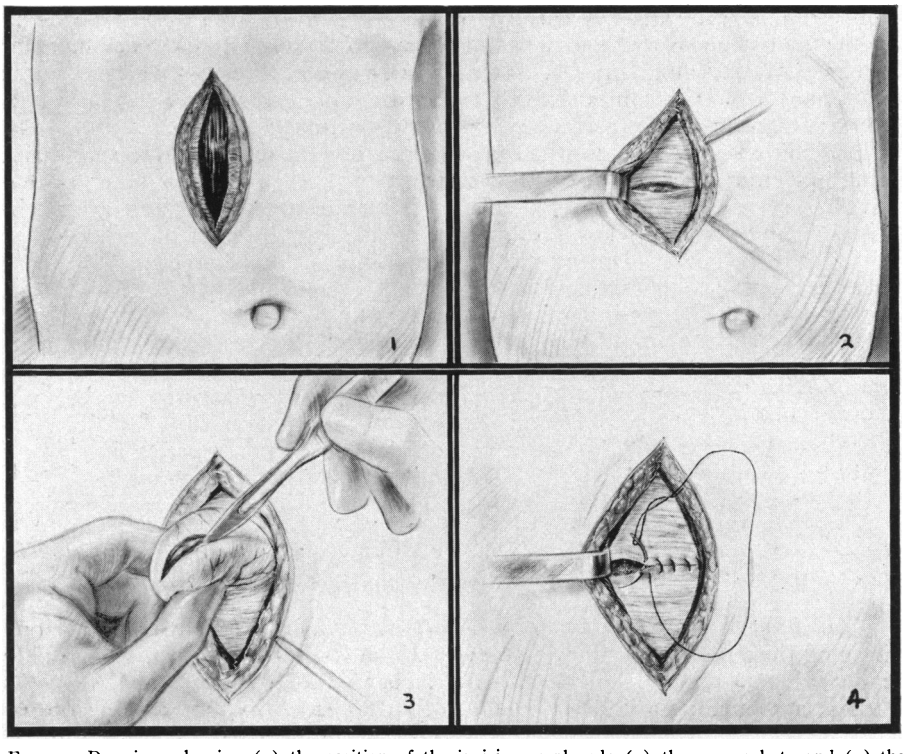

FIG. 1.—Drawings showing (1) the position of the incision employed; (2) the approach to and (3) the delivery of the pyloric hypertrophy; and (4) the method of closure of the wound.

second and fifth weeks. We employ the Fredet-Rammstedt operation with a very high right rectus incision and retract the right lobe of the liver upwards in order to reach the pylorus. I think Doctor Robertson's incision may be better than the right rectus because we have occasionally found that it is difficult to deliver the pyloric tumor through this high right rectus incision. I will try some cases using Doctor Robertson's incision.

The operation that we perform is exactly the same as that done by Doctor Robertson except that we use a curved mosquito clamp to spread the muscle surfaces instead of scissors.

We believe in feeding these babies very promptly ofter operation. We give them 15 cc. of water two hours after operation, and their first feeding, which consists of breast milk and barley water, three hours after operation.

Very few of these babies vomit after operation, if you empty the stomach completely in the operating room.

We use ether in all cases and pass a stomach tube and completely empty the stomach before we make the abdominal incision.

We have concentrated most of our attention on bringing the mortality down. When I started my series we were still operating upon them as emergencies. I remember very well going down to the Babies' Hospital one Sunday afternoon and operating upon one who had been sent in by a pediatrician, and the indication for operation in those days was that the baby was vomiting everything. No one seemed to give any thought as to whether the baby was able to stand such an operation or not. We have, in recent years, concentrated a great deal of our attention on the preoperative preparation, particularly the restoration of the fluid balance, and feel that the amount of vomiting is unimportant. Sometimes it takes between four and five days to prepare these babies for operation. In 1931, I reported my first 100 cases with one death and have had no deaths since then. In 1935, I reported 243 cases with the same one death. Last year, I reported 350 with the same death, and since that report I have had about 60 more and have lost none.

DR. EDWIN M. MILLER (Chicago, Ill.): I hesitate to discuss a paper presented by one who has had a much wider experience than I in this field. During the past 20-odd years my experience has been limited to about 40 Rammstedt operations. My object in discussing this paper is to say that I feel one seldom has an opportunity to grossly and microscopically examine the pylorus of an infant upon whom this operation has been performed some time previously. This opportunity has come to me recently in a case that I operated upon in September, 1939—an infant, at that time two months old, weighing less than birth weight, presenting a typical picture in all respects with a definitely palpable pyloric tumor.

Last month this infant died of some cerebral disturbance, and they found in the records that I had operated upon the baby six months previously. They were kind enough, therefore, to turn the specimen over to me for examination.

I made a photograph, which was not a very good one, showing the duodenal mucosa and the anterior surface of the pylorus. I was surprised to find, on pathologic examination, how smooth this area was and the very little scar in this region. Transverse sections were made through the central portion of the pylorus, and the gross appearance across the center of the pylorus, apparently, differs somewhat, I would say, from the conception as illustrated in one of Doctor Robertson's photographs. Microscopically, it is difficult to decide where the incision had been made; that is, it was difficult for our pathologist to decide. He could not positively say, after thoroughly looking at this section a few days ago, where the incision had been made, but because of the thinner area in one quadrant I imagine that is the point where the operation had been performed.

DR. WARREN H. COLE (Chicago, Ill.): Apparently we all agree that one of the most important features in bringing the mortality rate down in this disease lies in the fact that we are paying more attention to the electrolytic and fluid balance and bringing them up to normal before submitting these patients to operation. As some of the discussers have already mentioned, it is important to evacuate the stomach before the child is sent to the operating room. This will help the anesthesia and, certainly, will lead to a

smoother postoperative course; it may be necessary to evacuate the stomach again at the end of the operation if the child has swallowed a great deal of air.

It is likewise agreed that either general or local anesthesia may be used. I prefer local, and if, preoperatively, a mild sedative, such as second or nembutal is given, the local anesthetic will behave much better. I have never seen any ill effects from mild sedation of this style.

A second very important feature in the progress of the child postoperatively lies in the question as to how well the feedings are retained. I am quite convinced we can control this phase of convalescence to a large extent by watching the amount of feedings. I heartily agree that we should start feeding these children a few hours after operation, and should increase the amount rapidly up to about a normal feeding. However, if vomiting becomes significant during the course of the first few days, feedings should be reduced immediately to one-half the original amount, and increased gradually. In my experience this will almost invariably stop the vomiting. We will certainly grant that if over a given period of time a child took ten ounces of food and vomited eight, the situation would be much worse than taking five and vomiting none.

I should like to heartily endorse Doctor Ladd's statement about the use of vitamin C. I think this phase of therapy is tremendously important, and when you add closure of the wound with silk to that precaution, the incidence of wound disruption will be reduced practically to zero.

I wish to call attention to a final, and not insignificant point, namely, that a good spirit of cooperation with the pediatrician must be attained lest he attempt to treat these infants too long medically.

DR. WALTER ESTELL LEE (Philadelphia, Pa.): It would seem that nothing could be added to this discussion of the subject of congenital pyloric stenosis because everyone at the present time seems to be in agreement. I would like to express my personal appreciation of the suggestion of Doctor Robertson that a gridiron incision should be made in the subcostal region to the right of the semilunar line. I too have found, as Doctor Ladd has said, that the muscle-splitting incision is usually too far medially to be ideal.

May I suggest the addition of two procedures to the technic: For some ten years, we have operated entirely under local anesthesia. After the preliminary administration of luminal or nembutal, we give the child, shortly before operation, a sugar teat to suck. You may be surprised to hear that in the Quaker City the so-called sugar teat is made up of ten drops of paregoric and ten drops of whiskey or brandy to one ounce of a 5 per cent glucose solution. You will, likewise, be surprised how the babies take to this mixture, and also, more or less, amused by how promptly they become happily intoxicated.

The other suggestion has resulted from the observations of one of my assistants, Doctor Summey. In the past, one of our greatest difficulties, and most embarrassing complications, has been the failure to obtain primary union of the celiotomy wound in but a small proportion of cases. This involves merely the skin and the subcutaneous tissues, not the muscle sheaths, and we have not as yet had a wound disruption. After the removal of the skin sutures these infections promptly heal and we have had no herniae develop. Doctor Summey had been experimenting independently; and I was, eventually, informed by the nurse that they never had a breakdown in their wounds, as I did in mine! The explanation, apparently, is due to the fact that they have been using through-and-through, unabsorbable mass sutures

which pass through skin, subcutaneous tissues, muscle and peritoneum, and which are then tied over rolls of gauze after the skin edges have been approximated by interrupted mattress sutures of silk. Since employing this technic I have had primary union in every case. This, of course, is in line with Doctor Lahey's contention that in the closure of celiotomy wounds it is not necessary to employ layer sutures, and that through-and-through sutures of nonabsorbable material, down to the peritoneum, result in the lowest incidence of wound complications. It seems reasonable that the tissues in infants may not be capable of handling too much foreign body in the form of suture material.

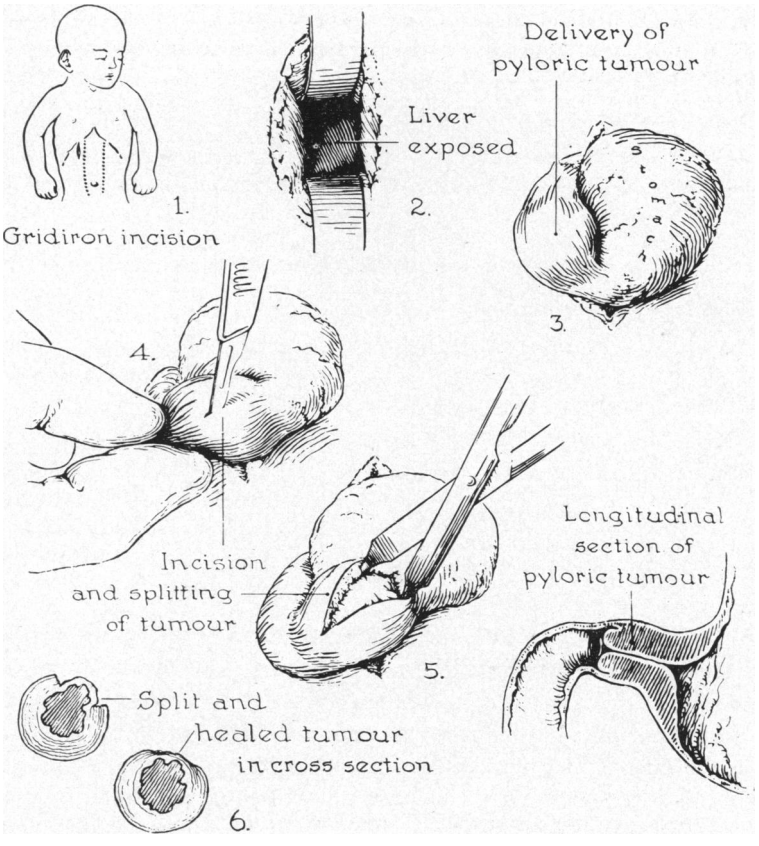

FIG. 1.

DR. D. E. ROBERTSON (Toronto, Canada, closing): It may be of interest to demonstrate by a drawing the gross specimen from a child who died of pneumonia three or four months after operation, which is a true representation of the condition present in the stomach (Fig. 1). In the pathologic specimen, just a thin layer of fibrous tissue covering the mucous membrane is to be seen. It is quite smooth externally and there is no muscle layer to be found.

In regard to the incision, I do believe that the best incision is the gridiron incision which can be closed by layers, and I think one should use No. 0 chromic catgut. I use No. 000 chromic catgut in the skin. Chromic catgut is used as I believe it is less irritating than plain catgut. When a sterile dressing is applied over the wound, with mastisol to hold it on, the wound need not be disturbed for ten days to two weeks.