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Congenital hypertrophic pyloric stenosis in infants

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By

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- DEFINITION -

According to Cecil (9) hypertrophic stenosis of the pylorus consists of an obstructive narrowing of the pylorus accompanied by, and apparently due to, hypertrophy of the pyloric muscle.

- HISTORY -

Undoubtedly cases of pyloric stenosis have occured since the beginning of time but they have not been recognized as such. Kellett (32) reports the first case by Fabricius Hildanus during the Shakespearean period. He entitled the case "Observatio singulanus de obstructione pylori." Kellett also located a case by Blair in 1717 and Weber in 1758. Knox (33) has made an intensive survey of the history of pyloric stenosis and finds the first case mentioned in 1777 by George Armstrong on a case from post-mortem findings in a child. Hezekiah Beardsley of the United States reported a case from post-mortem findings of a child in 1788. In 1841 Williamson of London reported a case describing it as Scirrhous of the stomach, and probably congenital. In 1842 Siemon-Dawosky reported a case in Germany and mentioned the projectile vomiting

connected with it. Finkelstein was the first to show a palpable tumor in these cases in 1896. Hirschsprung gave it our present day conception in 1887. In 1893 Cordua tried a jejunostomy for its relief. A gastro-enterostomy was done by Willy Meyer in 1898. Fredet successfully performed a submucous pyloroplasty in 1907.

Kellett (32) reports that the name pyloric stenosis was suggested by Landener in 1879. Loreta of Balogna was the first to propose an operation in 1882. He used digital dilatation. In 1886 Heinecke and Mikulicz in 1887 independently devised the procedure of pylorotomy, which was difficult to perform because of the thickness and hardness of the hypertrophic pyloric ring. The present day operation was first made by accident by Rammstedt in 1912. He had done a Fredet type of operation and his sutures tore out. The child's condition was so critical that he made no attempt to replace them, but merely tacked a piece of omentum over the pyloric incision and closed the abdomen. The patient recovered so well that he used this method from then on.

ANATOMY

The lower or right portion of the stomach is called the pyloric portion. It separates the cardiac portion of the stomach from the duodenum. It consists of the pyloric antrum and the pyloric valve. The pyloric valve is formed by a reduplication of the mucous membrane of the stomach, covered by a muscular ring composed of a thickened portion of the circular layer of the muscular coat according to Lewis (37).

Christopher (10) states that the pyloric canal in an infant measures 1.5 cm. long. The pylorus projects into the duodenum as the cervix does into the vagina. In pyloric stenosis the entire pylorus is converted into a tumor because of hypertrophy of the circular muscle layer. All other elements of the pylorus are normal.

The muscular coat of the stomach consists of two groups of fibers. The longitudinal fibers, which are continuous with the longitudinal fibers of the esophagus and which radiate in a stellate form from the cardiac orifice, are chiefly lost as they approach the pyloric antrum. The circular fibers, which form a thick muscular cylinder under the longitudinal fibers become more numerous as they approach the pyloric antrum and

at the actual outlet form a strong muscular ring which encircles the pyloric orifice and constitutes the pyloric sphincter. Romano (51) finds that the circular muscle is 2.5 mm. to 3 mm. thick. The mucosa of the stomach, pylorus, and duodenum is continuous.

- PHYSIOLOGY -

The sympathetic is generally considered to be inhibitory to the pyloric sphincter, and the vagus excitatory, but an opposite effect may be obtained from either nerve, the tonus level at the time appearing to be the determining factor according to Best (1)

Best (1) found that maintenance of a constant acidity in the duodenum did not prevent the pylorus from opening. On the other hand, when the duodenum was kept neutral or alkaline by means of fluid introduced through a duodenal tube rhythmical opening and closure of the sphincter occured in the usual way.

Most physiologists believe that the pylorus is patent for the greater part of the time and that evacuation of the stomach is definitely related to the peristaltic activity of the antrum. Water passes through the pylorus in squirts.

North (46) found consistent chemical changes in the blood in cases of pyloric stenosis. The plasma

chlorides are decreased, the blood urea nitrogen is increased, and the carbon dioxide combining power is increased. The loss of chlorides is due to vomiting and a loss from blood to tissues. He has the following theories on the increased urea nitrogen; "1. Increased destruction of body protein due to the effect of some toxic substance or as a result of dehydration. 2. Decreased function of the kidney resulting from dehydration. 2. Decreased function of the kidney resulting from dehydration or toxic nephritis. 3. Hypochloremia resulting in elevation of the level of urea as a compensatory mechanism for maintaining the osmotic pressure of the blood. 4. Alkalosis following marked vomiting or the administration of alkalies for therapeutic purposes." North (46) finds that patients suffering from achlorhydria as a rule sh w changes in the blood similar to those in pyloric stenosis.

Morris (45) discovered that there is an increase in both plasma chloride and urinary chloride. The chloride of various tissues decrease about 50 per cent. He found that injections of 1 Gm to 2 grams of chloride subcutaneous resulted in almost complete retention while in a normal child there was a 90 per cent loss in the urine.

ETIOLOGY

There have as yet been no satisfactory explanations for congenital hypertrophic stenosis. No one is able to prove what causes the pathologic condition and why it should occur most often in male babies is unsolved. Many theories have been submitted and I will relate the more plausable ones. Engel (19) lists the following nine causes. "1. Congenital duodenal adhesions. 2. Peptic ulcer. 3. New growths, (a) carcinoma, (b) polyps, (c) tuberculosis, (d) fibroma. 4. Perigastritis 5. Perigastric adhesions. 6. Carcinoma of the pancreas and biliary tract. 7. Empyema of the gall-bladder. 8. Intragastric foreign bodies such as bezoar and hair-balls. 9. Spastic causes; (a) psychic causes, (b) local irritation, (c) reflex pylorospasm. Although any of these might have been the cause in one case it is a well known fact that such causes as carcinoma and peptic ulcer are exceedingly rare in infants. These causes would be more apropos to adult pyloric stenosis.

Robertson (50) states that he has bound no history of digestive disturbance such as duodenal or gastric ulcer. He concludes that it is probably genetic. His reasons are that it occurs at an early age, more than one case in a family, and its common occurrence in maternal twins. O'Donnell (47) reports a set of di-

zygotic twins and of two brothers, born two years apart, all of who were operated on for pyloric stenosis. Fabricius (21) reports four siblings with pyloric stenosis, all of whom had the same mother and three the same father. Cockanye (11) and Sheldon (52) both believe the cause is genetic. They agree that in the case of twins there is an environmental handicap which might be an inducing factor. Cramer (13) claims that since it occurs in the first three weeks of life it must be congenital for this amount of hypertrophy could not occur so soon.

Allen contends that it is quite likely due to injury to the central nervous system at birth. Flynn (24) believes it is due to a tendency of physicians to deliver the baby rather than allow the mother to give birth. The maneuver after the head is born, of depressing and traction in order to get a finger in the axilla of the anterior shoulder instead of allowing the uterine contractions to normally rotate the shoulders would be very likely to injure or irritate the parasympathetics. Yet Flynn's records show that it has occured in all types of deliveries, with all types of anesthesia. He also noted that none of the cases belonged to the three leading physicians of Houston, Texas, who deliver a large percentage of babies. One

anesthetist in Houston noted cases of neck stretching with pyloric stenosis following.

Einberg (18) thinks it is caused by trauma of the stomach from overdistention. Fraser (28), and Thomson (55) say it is due to overaction of stomach musculature.

Williams (59) believes the original is due to the defective maternal nutrition during pregnancy. He shows how many inants have alkalosis and dehydration with pyloric stenosis. He admits that part of this is caused by the vomiting. Most authorities believe all of it can be accounted by vomiting.

Cohen (12) lays the cause to an allergic phenomena. His investigations show the following results: (a) The family history of allergy. (b) Presence of other allergic reactions in the patients either at the time of pyloric obstruction or later in life. (c) Demonstration of specific skin reactions to definite allergens. (d) Therapeutic testing by the dietary change based on the evidence obtained by the immunologic methods used. He found 12 out of 27 or 44 per cent of cases showed allergic manifestations. Six infants studied were all found to show positive reactions either dermally or intradermally to egg, milk or cereals.

Christopher (10) and Boyd (3) state that hypertrophy always precedes the pyloric spasm, but it is a combination of the two that causes the trouble. On the other hand Hurst (30) and Mayo-Robson (40) contend pyloric stenosis is due to spasm and the hypertrophy, and it is difficult to understand why it should produce it in infancy. In the second place, the pyloric thickening persists at autopsy long after the symptoms have disappeared spontaneously or have been relieved by the indirect attack of gastro-enterostomy, whereas after the Rammstedt operation, which is directly curative, it disappears entirely.

Even vitamin defficiency has been shown to be a contributing cause. Brodie (4) found that rats receiving sufficient B_1 have never shown a case of pyloric obstruction. Rats fed a diet defficient in Vitamin B_1 had a 1.2 per cent mortality due to pyloric obstruction in the first generation and 22 per cent in the second generation. Autopsy showed degeneration of the vagus nerve. He therefore contends that it may be quite possible that mothers do not supply enough vitamin B in the milk.

PATHOLOGY

There is always thickening of the pyloric ring, and the lumen of the pylorus is never completely closed. Christopher (10) states that the circular muscle coat is five times its usual thickness, but all other structures appear normal. Flynn (24) finds the gross appearance of the pylorus to be a bobbin-shaped, indurated, ischemic tumor mass from one half to two inches in length, and one fourth inch thick. The mucosa is thrown into folds from the constricting encroachment of the lumen.

Romano (51) finds the normal structure is replaced by a cartilaginous-like mass from 22mm. to 38mm. in length and from 16mm. to 22mm. in diameter. The mass which is olive shaped, weighs twice as much as the normal infant pylorus. The hypertrophy is due to increase in both number and size of circular fibers. There is no intrinsic inflammatory change although infection may be superimposed when gastritis develops as the result of gastric retention and dilatation. ON the other hand Ladd (34) finds the mucosa and submucosa are essentially normal in infants under ten days, but after this they are edematous and show increased leukocytic infiltration.

SYMPTOMS AND SIGNS

The baby appears normal at birth, nurses well, and for the first week or two gains in weight. Vomiting is only occasional at first. After a few days it becomes projectile and more frequent. The appetite remains good but there is a loss of weight. The baby becomes constipated and there is a decreased urinary output. Stools are bile stained and small. The vomitus is usually free from bile. There is visible peristalsis in the epigastrium, the waves starting on the left and progressing to the right. These symptoms may have a sudden onset or disappear for a day or so.

A palpable tumor-like mass can usually be felt in the epigastrium, slightly to the right of the midline.

Williams (59) points out the clinical manifestations of dehydration which are always present after several days of vomiting. The skin is wizened, wrinkled, and has an inelastic condition. There is poor tissue turgor. The eyes and fontanelles are sunken. There will be an increased hemoglobin, red blood cell count, and hematocrit reading.

Findlay (24) finds a fall or complete absence of urinary chlorides is one of the most characteristic

features. It will fall even though there is no or little vomiting. He believes the chloride is held in the tissues by some toxin.

DIAGNOSIS

The diagnosis is best confirmed according to most authorities by palpation of a tumor. A. Brown (7) gives the following method. "The left hand should be placed behind the right side of the child's back and the fingers of the right hand should be placed from below upward and to the left so that they pass beneath the lateral margin of the right rectus muscle. Sometimes if an attendant makes pressure on the left side of the abdomen to force the stomach over to the right, the tumor may be palpated more easily." Downes (17) says that the secret of finding the tumor is to have the child perfectly quiet and completely relaxed and that relaxation can always be achieved by placing the baby in a tub of warm water. He suggests that it is helpful to press the fundus of the stomach with the left hand, while using the right to palpate the tumor. Donovan (16) finds his best results at palpating the the tumor when the stomach is empty. On the other hand Knox (33) says the best examination is made when the infant is being fed. Most authorities claim the tumor

is palpable in 94 per cent of cases, but then others say it is as low as 24 per cent.

Robertson (50) watches for peristaltic waves. He feeds the infant glucose solution in a nursing bottle and watches the epigastric region fill out and waves of peristalsis sweep from left to right. When full the patient will vomit the projectile type.

Calvin (8) advises roentgen examination in cases that are not palpable. The amount of barium remaining in the stomach after four hours is the most important diagnostic finding. He found that 92.3 per cent of the patients on whom roentgen examinations were made and who were later proven to have pyloric stenosis at operations, roentgenograms showed from 60-100 per cent retention of barium in the stomach after four hours. Most doctors are not in favor of X-Ray. They believe it is both dangerous and unnecessary. Perforation has been known to occur during the passage of a barium meal. A film of an empty stomach will show a large, dilated, gas filled stomach, with relatively little gas beyond the pylorus according to Ladd (34). Ladd reports that fluoroscopic findings with a barium meal will show an enlarged stomach with rounded pyloric end in the case of pyloric stenosis. Peristaltic activity will be increased. There is little escape of barium into the duodenum.

A few authors believe that gastric lavage is as helpful as X-Ray, and far less harmful, in demonstrating gastric retention both as to fact and quantity.

DIFFERENTIAL DIAGNOSIS

Pylorospasm is most commonly confused with pyloric stenosis. Knox (33) points out the fact that pylorospasm has a less rapid onset, the course of the disease is not so severe, and vomiting occurs several days after birth. The vomitus may be bile stained. Potts (49) gives atropine and phenobarbitol which relaxes the pylorus of a patient with pylorospasm.

Congenital duodenal adhesions differ from pyloric stenosis in the following ways according to Knox (33). The vomiting begins immediately after birth; it is at first only regurgitated, then becomes projectile and cumulative in character. The gastric peristaltic waves come on after a few days and simulate true pyloric stenosis. There is no tumor formation.

The vomiting is at frequent intervals, it is small in amount and may contain bile in cases of gastritis. Furthermore fever is usually present and diarrhea rather than constipation occurs.

According to Ladd (34) the vomiting is not so forceful. It is also small in amount and at irregular

intervals. There is apt to be convulsions, spasticity and bulging fontanels.

Vomiting occurs in the first 48 hours of life in incomplete rotation of the intestinal tract.

It must also be remembered that unduly long feedings at the breast, and too bulky formulas may produce vomiting.

The five cardinal symptoms of pyloric stenosis, projectile vomiting, palpable tumor, visible peristaltic waves, constipation, and loss of weight must always be kept in mind when diagnosing pyloric stenosis according to Potts (49).

- INCIDENCE -

The consensus of opinion is that pyloric stenosis occurs in first born and male children. Ladd (35) reports 620 cases with 85 per cent males. Tallerman (54) reports 79 per cent of his cases were in male children. Potts (49) reports that it occurs five to one in males. Lothrop (38) and Cramer (13) find it to be seven times more common in males. Experimentation on rats by Brodie (4) brought forth the fact that 87.5 per cent of the cases were males. Ford (27) recorded 51.8 per cent in first-born children in her series of cases.

Most authorities mention that there is a tendency for it to occur in the same family.

Romano (51) finds no preponderance in any race. Downes (17) has found it to be chiefly in the Hebrews. It is more common among Anglo-Saxons and rare in the Latin races according to Wallgren (56). Folsom (25) finds it to be rare in negroes.

It seems to be more frequent in breast fed babies, but Davison (14) points out that the larger percentage of infants are breastf ed.

The average age of onset is 21 days but it may occur anytime from birth to the tenth week and there are a few cases on record in adults.

According to the records pyloric stenosis is on the increase but Wallgren reminds us that doctors are more aware of it than formerly.

- TREATMENT -

The authorities are able to agree perfectly on the clinical description of pyloric stenosis but there is considerable discord as to therapy. There are three groups; those who use medical therapy, the ones that advocate surgery and the group who uses a combination of the two. Most physicians belong to the

later. They usually try medical treatment for ten days and if there is no improvement then they do surgery. The surgeons feel that there method gets quick results and claim that most of the medically treated cases eventually come to them anyway.

MEDICAL

Medical treatment consists of diets, sedatives, antispasmodic drugs and correction of dehydration. Naturally this should best be carried out in a hospital which is usually too costly except for the rich or poor.

Most diets consist of thickened food, such as breast milk, thick cereals or ordinary formulas boiled with starches of various kinds. Concentrated foods of high caloric value are more frequently advised on the ground that children have limited starch digestion. Both types of diet clearly disregard the fact that by their use a more severe test is put upon the digestive system of an ailing child than of a normal, well child according to Romano (51) Patterson (48) claims that the stenosis will disappear at the age of six months if proper nutrition is maintained.

The older method of drug thearpy which is still preferred by some clinicians consists of sodium phenobarbitol 1/8 grain before each feeding. This is usually

combined with atropine 1/1000 grain. Cecil (9) points out that atropine alone is undesirable because it may produce distention of the bowel, dryness of the nose and throat, and cerebral stimulation.

Eumydrin (atropine methyl nitrate) is the drug of choice today. It is 1/50 as toxic as atropine and has a much longer action. Wallgren (57) uses a 0.6 per cent alcoholic solution which contains 0.1 mgm. of eumydrin in each drop. A drop of this solution placed on the surface of the tongue is rapidly absorbed and the treatment and absorption are not interfered with by vomiting. Dobbs (15) gives an oral dose of 1 cc. of a 1 in 10,000 aquous solution of eumydrin. The dose is gradually increased until six or seven cc. are given before each meal. The toxic signs of eumydrin are flushing of the face, and vomiting.

Dehydration is combated by giving fluids parenterally. Williams (58) gives 600-1200 cc. of normal saline intravenously at the rate of 20 cc. per hour. Nutritive enemas may be employed. It is striking to observe the number of authorities who forbid all water by mouth or permit it in small quantities only. Many believe, too, that drugs should not be administered until the water balance is correced.

Some authorities advise lavage if gastric retention is marked, others oppose it on the ground that it brings about a still greater loss of chlorides than has already been caused by vomiting. If it is employed, it should not be done with soda bicarbonate, because of the tendency to alkalosis which these children display.

Dobbs (15) has followed the rule that if no improvement is seen at the end of ten days then surgery should be tried. He has had twentyseven out of forty cases cured by medical treatment. Surgery cured eight out of forty. He lost one medical case and four surgically treated patients.

PRE-OPERATIVE CARE

The pre-operative care is so important that Romano (51) says the battle is fought and won before the operating room is entered. Fluids should be with held by mouth but should be given freely by hypodermoclysis and infusion. Saline solution is used to restore the electrolytes lost by prolonged vomiting, glucose is used to combat acidosis, reestablish the renal function and build up a carbohydrate reserve. Most surgeons do not like intraperitoneal injection for obvious reasons. Ladd (34) recommends glucose

solution, ten per cent, given intravenously at the rate of ten cc. per pound of body weight, and physiologic saline 15 cc. per pound of body weight. One should figure on maintaining the fluid intake at two and one half or three ounces per pound body weight per twentyfour hours.

Cachetic patients are transfused, the administration being li ited to ten cc. per pound of body weight.

The infant should be kept warm by wraping the extremities in wool and placing hot water bottles about the body during the operation. Ladd (34) recommends placing a hot water bottle under the babys back. He says that this also aidsin arching the back which is an advantage to the surgeon. The operating room should be as near 80 F as possible.

Gastric lavage thirty minutes before the operation is recommended by Ladd (34) and Levi (36). This cleans out the stomach, thus cutting down peristaltic movements and also makes for a cleaner field in case the mucosa is opened.

ANESTHESIA

Open drop ether is the anesthetic of choice. There are a few surgeons who prefer local anesthesia.

Meader (43) uses one half per cent novocaine. Romano (51) recommends local in patients with upper respiratory infections or patients who are poor risks. Brown (7) claims it takes ten cc of one per cent novocaine for a baby and this compares to 300 cc of one per cent novocaine in a 150 pound adult which is almost a lethal dose.

SURGICAL TREATMENT

The accepted method of treatment of pyloric stenosis seems to be surgical. Romano (51) mentions that the condition is largely mechanical and is thus best corrected by a mechanical measure. Most men are willing to try medical treatment for a few days if the case does not seem too severe. Brown (6) states that the earlier the operation is performed the less shock results and the more rapid the convalescence. He has had several cases come to operation after futile attempts had been made under medical treatment. The result being that the infant was in worse condition than if operation had been performed when the diagnosis was first made.

Pyloroplasty was the early surgical measure. This consisted of cutting through the mucosa as well as the other tissues. This resulted in too high a

mortality. Later a V-shaped submucous pyloroplasty was advised and succussfully done.

In 1907 Fredet did a submucous pyloroplasty by means of a straight incision over the superior anterior part of the pylorus three fourths of inch long and extending through the serosa and mucosa. The incision is made here due to less vascularization. The longitudinal incision was then transformed into a transverse wound by the use of sutures put in parallel to the long axis of the bowel.

In 1912 Rammstedt omitted the transverse suture leaving the pyloric wound open. This is the accepted method today. Some doctors cover the exposed mucosa with a flap of tissue.

Brown (6) who believes that speed is of utmost importance claims that his average time in twenty cases was seven and one half minutes.

Williams describes a few aids in technique. He delivers the tumor by introducing the left index finger into the abdominal cavity, then grasping the tumor with a special spatula. The muscle of the pylorus is divided by first incising the longitudinal coat with a scalpel and then splitting the muscle of the circular coat by the aid of a special pair of blunt, flat bladed forceps.

Boice (2) has a two stage operation. The stomach is divided in half. The upper one half is anastamosed to the duodenum. A month later the distal portion of the stomach and the pylorus are removed.

Cases are on record where the muscle fibers were not completely separated. The symptoms continued until the abdomen had been reopened and the fibers cut. McLanahan (42) states that Dr. William A. Fisher suggested making a small incision on the anterior wall of the stomach through which a curved Kelly clamp would be introduced into the pylorus. The jaws of the clamp could be spread while in the pyloric canal to assure the operator that sufficient space was present. McLanahan passes a catheter number ten or twelve into the stomach then at the operation he works it through the pylorus.

Engel (19) gives a method of repair for accidental incision of the mucous membrane as follows: " a parallel incision is made about one half cm. alongside of the primary incision through the pylorus to the mucous membrane. This narrow strip is sutured to the oppowall of the first incision after the mucous membrane, which was cut, is first sutured. This affords a firm closure and yet, virtue of the second incision, the

primary purpose of relieving the obstruction is accomplished."

Most authorities close the incision with continuous chromic catgut, size 00. Potts (49) has discontinued the use of tension sutures because of stitch abscesses about them.

The original skin incision may be made one half inch below the right costal margin and paralled to it. Two and one half inches is long enough. Others make a four cm. incision through the right rectus muscle. beginning just below and one and one half cm. to the right of the xiphoid cartilage.

One of the most convincing examples in favor of surgery for pyloric stenosis is cited by Brooks (5). He saw a case of identical twins with congenital hypertrophic stenosis. In one obstruction was so severe that surgery was resorted to at once; in the other, it was less severe and the child was carried along on medical treatment. At the end of the sixth year of life no one could have surmised that the children were twins, for the child who had been treated medically had never been strong or well and at this age he was at least two years behind the other twin in growth and development.

POST OPERATIVE TREATMENT

The first essential of post operative treatment is to maintain body heat. This is done with hot water bottles and blankets. The head should be lowered until there is complete recovery from the anesthetic.

There is a difference of opinion as to when feeding should begin. Most authorities agree that it should be shortly after the patient has recovered from the anesthetic. The common routine is sterile water followed by breast milk, whey, barley water or other formulas, as the pediatrician may advise. The important thing is that the feedings are small. Gastroenteritis is usually due to the administration of large quantities of food before the alimentary canal has regained its normal tone.

Faber (20) with holds both water and food for twentyfour hours after the operation. He contends that peristalsis is completely inhibited for several hours and depressed for twentyfour hours after pylorotomy according to barium meals given patients. He does give fluid parenterally the first day.

Engel gives 100 cc. of normal saline solution by bowel before putting the patient to bed. Moynihan (44) recommends passing a catheter into the stomach

immediately upon completion of the operation and putting in two ounces of water.

Meader (43) starts fractional feedings two to four hours after return from the operating room. He starts with one dram of water, then two hours later one dram of artificial feeding. At two hour intervals the feedings are increased in one dram doses until one ounce of fluid is given. If the feedings are all retained, they are then increased slowly until the child is taking three to three and one half ounces every four hours.

Knox (33) uses the following routine:

For example if the operation is at 12:30 PM.

3:30PM	30 minims of water	
4:00	1 dram of water	
4:30	2 " " "	
5:00	2 " " "	
5:30	2 " " "	plus 1 dram breast milk
7:30	3 " " "	
9:00	2 " " "	" 2 " " "
10:30	4 " " "	
12:00	2 " " "	" 3 " " "
1:30AM	5 " " "	
3:00	1 " " "	" 5 " " "
4:30	6 " " "	
6:00	1 " " "	" 6 " " "
7:30	7 " " "	
9:00	1 " " "	" 7 " " "
10:30	1 ounce of "	" 1 ounce " "
12:00M	1 dram of "	
1:30PM	9 " " "	
3:00	1 " " "	" 9 drams " "

Then he gives one ounce of breast milk every three hous and water between feedings, not to exceed

one ounce. If the infant is doing well, then he should be put to the breast for three to five minutes to start with. If breast milk is not obtainable, condensed milk (1-9 or 1-10) may be used.

Some of the authorities say that the infant can receive better care in the hospital. Others like to get them out of the hospital as soon as possible because of possible infection. Lothrop (38) recommends that someone hold the child in order to insure body warmpth, and he believes this is best done by the mother at home. Donovan (16) on the other hand says the infant should be handled as little as possible and even recommends feeding via dropper for the first five days without picking up the baby.

- COMPLICATIONS -

Although the complications that occur with operations for pyloric stenosis are now at a minimum they must still be looked out for. Accidental opening of the mucus membrane with out its repair or recognition will result in peritonitis. Hemorrhage from the cut edges of the pylorus may take place if one has not been careful of hemostasis. Infection of the wound may take place if the princi-

ples of asepsis and antisepsis are not observed carefully. Pneumonia (postoperative) is possible, although it rarely occurs.

CAUSES OF DEATH

The above complications make up most of the death causes. The larger percentage were caused by peritonitis, due to perforation of the duodenum or secondary to other foci of infection, shock, wound rupture and gastro-enteritis. Undernourishment, dehydration and inadequate preoperative preparation increase shock and collapse, and predispose to infection and unsatisfactory healing.

PROGNOSIS

The prognisis for pyloric stenosis cases has improved immensely in the last twenty years. Potts (49) reports 78 patients between 1923 and 1932 with a mortality rate of 12.8 per cent. From 1933-1936 he had 56 patients with a mortality rate of 3.5 per cent. Lothrop (38) reports that Childrens Hospital and Infants Hospital in Boston had a total of 425 cases from 1915 to 1931 with a mortality rate of 6.3 per cent. Ladd (35) had 635 cases with 5.4 per

cent mortality.

Probably preoperative preparation has as much to do with surgical mortality as anything. Thelonger a patient goes without treatment there is a certain point reached where even the best preoperative care will have little effect due to the damage already done.

McGahee (41) ranks the mortality according to when the surgery is done. His figures show no mortality for those treated during the first week, five per cent in the second and third, fifteen per cent from the fourth to the sixth, and twenty per cent after that period.

- CONCLUSIONS -

The consensus of opinion seems to be that pyloric stenosis in infants is co:genital and that the ypertrophy comes before the spasm. The important points on the part of the clinician is recognition of the pathology, adequate preoperative care, prompt operation by an experienced surgeon and careful post-operative management by a competent pediatrician.

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