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Congenital hypertrophic pyloric stenosis, with report of nine cases from University Hospital, Omaha, Nebraska

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CONGENITAL HYPERTROPHIC PYLORIC STENOSIS, WITH REPORT OF NINE CASES FROM UNIVERSITY HOSPITAL, OMAHA, NEBRASKA

SENIOR THESIS

1933

BY

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CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

Congenital hypertrophic pyloric stenosis is a mechanical obstruction of the alimentary tract, due to a simple hypertrophy and hyperplasia of the circular muscle fibers of the pylorus, and infolding fibers of the longitudinal bundle, probably of congenital origin, manifesting itself clinically, soon after birth, by projectile vomiting, visible gastric hyperperistalis, constipation and progressive emaciation.

HISTORY

Congenital hypertrophic pyloric stenosis is by no means a new disease, but very seldom mentioned in the literature until after Hirschsprung's paper in 1887, following which much interest was aroused in the medical profession.

The first case on record was reported by Patrick Blair (as quoted by Still), in 1717. He gave a complete clinical history and autopsy report of pyloric stenosis before the Royal Society of London. In 1758, Christopher Weber described a case which died the sixth day after birth, and at autopsy showed a contracted cartilaginous pylorus. In 1771, Armstrong (as quoted by Ruhrah) reported that a child, aged three weeks, had died and at

autopsy had revealed a hard contracted pylorus. The child, he concluded, had died of spasm of the pylorus. Eleven years later, according to Osler, the first case reported in this country was that by Beardsley, in Transactions of the New Haven County Medical Society under the title of "Scirrhous of the Pylorus in an Infant". Following these early reports were those of Williamson in 1841 and Dowasky a year later. The report by Dowasky was the first to appear in German literature. No lively interest, however, was aroused until ten years later when Hirschsprung's paper appeared.

In 1887, Hirschsprung gave a complete clinical detail and accurate pathological findings in pyloric stenosis. In 1898, Cautley was able to find recorded but twenty cases, whereas, four years later, fifty cases had been reported and nineteen of these operated upon. In 1905, 125 cases were reported and three years later, Ibrahim collected 400 cases.

In 1906, Heubner stated that 0.5 per cent of his patients had this condition. Rosenhaupt found it in one out of 50 patients, and Hertz found 61 cases amoung 2,275 infants under one year admitted to his hospital at Copenhagen, an incidence of 2.7 per cent.

ETIOLOGY

The cause of congenital hypertrophic pyloric stenosis is still in the realm of conjecture, although many theories have been advanced.

The incidence in males is recognized by most observers as being approximately 3:1. Often occurring in the first born and the infants are most usually breast fed. Caulfield was the first to report familial incidence in successive generations of pyloric stenosis through a report of two cases. The occurrence in even three members of one family is not infrequent. Armstrong not only reported the condition early, but it was the third in the same family to die of the disease. Sauer and Finkelstein both report two in each of which three cases of stenosis occurred. Bilderbach reports cases in premature twins. Davis, Moore and Sauer each have reported cases in twins. Ashton, in 1929, reported a case of stenosis in a child whose mother, 23 years previously, had had the sam condition. Pfaunder, according to Ward, suggests the condition to be somewhat peculiar to the Anglo-Saxon race, as up to 1905 no unquestioned cases had been reported from Latin or Slavonic countries. However, at present, the condition seems to occur in all races but less frequent in

the negro. Lapage, up until 1924, had only found two cases of pyloric stenosis in Mongols.

In 1897, Thomson stated that "pyloric stenosis is not a muscular effection but a functional disturbance of the nerves of the stomach at the pylorus." Paul and Elterich considered the condition to be due to a disturbance of balance in the sympathetic and parasympathetic system at the outlet of the stomach. Strauss believes the tumor to occur in utero being brought about by the rhythmic contractions of the pylorus which doubtless start at this time, and is due to abnormal stimulation from the intrinsic or extrinsic nerves of the stomach.

Pirie, in 1919, and Aldrich, in 1930, suggest the condition being due to a lack of balance between the secretions of the various endocrine organs in their process of development and involution which may result either in a relative or an absolute hyperadrenalism. He states that the pylorus and suprarenal develop about the third month. The suprarenal being larger than the kidney and since excessive secretion produces spasm in non-striated muscles, this would allow plenty of time for spasm induced by excessive secretion to bring about hypertrophy. Marine, in 1928, reports that a spontaneous involution of the cortex of the suprarenals occurs during the first two weeks of life and that the suprarenals undergo a distinct postnatal decrease in weight.

Cohen and Breitbart, in 1929, produced experimentally

in animals, characteristic physiologic pathologic change of the shock reaction consisting of smooth muscle spasm, edema and exudation. In addition, Ecker and Biskind have observed localized smooth muscle spasms in the rabbits' intestines in situ during anaphylatic shock. Cohen's and Breitbart's analysis of 27 cases indicated a familial tendency of 40.7 per cent. From their experiments, they concluded that the pathological condition in infantile pyloric obstruction is probably identical with that in allergy.

Brodie, in 1929, advanced the theory that a vitamin B deficiency may be a direct etiological factor. His experiments were carried out by feeding pregnant Albino rats on a diet deficient in Vitamin B. In four litters of rats, ten out of 23 showed pyloric obstruction. Furthermore, in the second generations, the incidence of obstruction was much higher. At autopsy, the stomach was enlarged and packed with a curd, and in all cases examined, the vagus showed myelin degeneration.

Apparently, after consideration of many etiological factors and theories, we are unable to form any definite conclusions as to what might be the exact cause. However, after an analysis of several thousand cases, the most outstanding etiological factors are: a. Most common in white race, b. Predominance of males, 3:1, c. Familial, and d. Usually first born infant. Since the medical profession

has become somewhat vitamin conscious, the vitamin B theory seems to be the most logical at the present time.

ANATOMY

The pars pylorica of the stomach extends from the incisura angularis in the lesser curvature, and a variable and inconstant notch on the greater curvature, as far as the pyloric opening. It differs from the body of the stomach in being more tubular in shape, and possessing thicker walls. The pyloric canal is a short, more or less tubular portion rather more than an inch in length, extending from the sulcus intermedius on the greater curvature to the pyloric constriction. The pyloric constriction marks the juction of stomach and duodenum, and there the various coats of these portions meet with one another.

The peritoneal covering of the stomach is continued onwards on to the first part of the duodenum. This serous coat is extremely adherent to the muscle beneath, and is tough and inextensible.

The muecular portion of the pylorus is composed of two layers, a rather thin outer longitudinal and a relatively thick inner circular group of fibers. The longitudinal fibers of the stomach are in part continued onwards into the longitudinal fibers of the duodenal coat,

but many of them bend inwards into the thickened ring around the opening, and spread out in diverging bundles, which interlace with the most superficial of the circular fibers, and some of them reach and terminate in the subjacent submucosa. In a study of the pars pylorica in 90 normal post-morten stomachs, Horton found in two instances, under one year of age, that the longitudinal fibers were absent in places, especially in the region of the greater curvature. The circular fibers are four or five times as thick as the longitudinal muscle. This layer is broken into numerous bundles by connective tissue septa, which usually run perpendicularly to the long axis of the pars pylorica. These septa are directly continuous with the submucosa. Horton also states that in his series of studies there was a definite break between the circular muscle of the pylorus and that of the duodenum.

Brown, in discussing the surgical anatomy of Rammstedt operation in 1931, describes the submucosa as an inconspicuous layer which makes the operation possible. This areolar layer exists to form a connection that will allow two structures to move freely on one another, one of these is muscle and the other mucosa. The areolar layer is well marked in the pyloric antrum, becomes still wider and looser up the pyloric canal, decreases markedly over the projection of the sphincter, ceases entirely at the

apex of the fornix, and then reappears again in the duodenum.

The gastric mucous membrane is continued into the mucous membrane of the duodenum at the distal margin of the sphincter. The mucosa is wrinkled into deep longitudinal folds throughout the pyloric canal until the opening into the duodenum is reached. Here the mucosa loses its deep folds and the size of the opening becomes slightly larger for a distance of about 1/8 inch where the duodenum is reached and the mucosa is thrown into folds again. The bore at this point is larger, but because of the lack of deep folds, it is not capable of marked increase in size as is other portions of the canal.

Blood vessels of the pylorus are from two sources. The right gastric and gastric epiploic vessels, and in two planes a superficial and a deep. The superficial one shows on the surface just under the serosa, but they fail to make any visible anastomosis at the mid-point from their origins, as they do elsewhere in the stomach. Because of this fact, a bloodless line is formed guiding the surgeon to an important land mark. The deeper vessels mostly ramify in the mucosa. Their importance in surgical attack of the pylorus will be referred to later.

The nerve supply of the pylorus is the same as that in the stomach. There are two plexuses of nerves: the

myenteric plexus (of Auerbach) and the submucous plexus (of Meissner). They receive fibers from the celiac plexus and from the vagus indirectly through the celiac plexus.

PHYSIOLOGY

The original theory of the acid control of the pylorus was brought out by Cannon, in which he states that it is necessary to have acid to open the pylorus and acid to close the pylorus. Since this theory was advanced, many workers have shown that acid is not necessary. Campbell has shown that in certain cases, otherwise normal, there may be a complete achlorhydria, and yet the movements and emptying of the stomach may present nothing abnormal. Apperly suggests that the chief factor is the osmotic pressure of the gastric contents; the nearer this approaches to that of the saline constituents of the plasma, the more readily does the pyloric sphincter open.

Stimulation of the peripheral end of the vagus nerves may excercise varying effects on the gastric wall as well as on the sphincters. McSwiney and Wadge find that the effect of vagus stimulation depends upon the muscle tonus. In the pylorus, they obtained either increased or diminished contractions.

Klein states that the fluidity of the chyme in the antrum is of prime importance. He also states, in summarizing his article, that each advancing peristalic wave overcomes the tone of the pylorus and forces some of the chyme into the duodenum, regardless of whether the reaction of the chyme is neutral, acid or alkaline.

At the present time, there is no definitely accepted theory as to the mechanism of the pylorus. There is, however, probably several factors at work, and no single one could carry out the function alone.

PATHOLOGY

The pathology of hypertrophic pyloric stenosis has been quite well agreed upon from the first. As stated previously, Hirschsprung gave a rather complete description of the pathological picture in the above condition. There is a hypertrophy and hyperplasia of the musculture of the pylorus especially the circular fibers.

The pylorus appears as a hard, or firm rounded or olive-shaped tumor mass, covered with normal peritoneum and sharply demarcated from the distended stomach above and the empty duodenum below. It is white and bloodless with exception of a small vessel or two on the surface.

This swelling of the muscle takes place inside an

adherent and unstretchable covering of peritoneum, and consequently can go in two directions only, inwards toward the lumen, and lengthwise mainly into the duodenum. Another mechanical effect is a decrease in vascularity from compression of the vessels supplying the muscle fibers. Finally the muscle is changed in consistancy from its usual fibrous state to a crispness resembling that of young celery. The older types show marked toughness due to fibrous and increase vascularity.

Saunders, in 1902, reported two cases with autopsy record showing marked thickening of all the layers including the submucosa and mucosa. Dr. H. Ashby, in 1907, showed before the Manchester Medical Society, nine specimens illustrating congenital pyloric stenosis in infants. They all showed hypertrophy of the circular fibers of the pyloric sphincter, hypertrophy of the walls of the stomach, dilation, and marked gastric catarrh.

SYMPTOMATOLOGY

In most cases, the onset has always been within a few weeks after birth, usually from two to four weeks. However, Kaiser, in 1924, reported an unusual case in which vomiting occurred 14 hours after birth and persisted until operation was performed. The first symptom has been

"spitting up" or vomiting, accompanied almost from the start with marked constipation, and very soon a startling loss of weight. Vomiting is the most marked and constant symptom. It may occur during or immediately after a feeding or hours later, and the quantity vomited may far exceed the amount taken at a single meal. The vomiting may begin as projectile in type, or may become projectile after a few days, weeks or even a month of regurgitating the feedings. When the vomiting becomes forceful in character, the babe often forces the vomitus over the foot of his crib. Even though the child vomits most of its food, he seems to be eager for more to eat. Evidently, there is no nausea associated with the vomiting. Greer, in a report of 21 infants with severe vomiting, found 12 to be pyloric stenosis.

The vomitus is invariably free from bile. There may be, however, streaks of appreciable quantities of brown or red blood, due either to superficial submucous hemorrhage or the slight erosions or ulcers of the mucosa. Microscopic and bacterologic examinations reveal nothing constant.

The next most important symptom is some degree of emaciation which may be either severe or mild, depending upon the degree of obstruction at the pylorus. The rapidity and degree of weight loss are considered impor-

tant aids in determining prognosis. If the weight loss equals one-fourth the birth weight, the child's condition is critical. "Quest Statistics", as given by Sauer, tend to show that a weight loss of more than 34 per cent is incompatible with life.

Almost with the onset of vomiting, constipation begins. At first, the bowel movements may only be decreased in frequency and amount, but shortly, if the obstruction continues, a severe constipation sets in. The patient may have no stools, or there may be the characteristic starvation stools. At times there has been observed an alternating constipation and diarrhea. Osler mentions in his text, cases with terminal diarrhea. It has been suggested that some of the toxic manifestations are due to this constipation, however, according to Abt, these changes probably occur in the proteid metabolism of the body proteids.

Scanty urine is another ourstanding symptom, and at times precedes the constipation. It is highly colored, and may deposit a brick red uric acid sediment. Acetone and diacetic acid may be found.

Alkalosis, in congenital hypertrophic pyloric stenosis, has been brought out especially by Maizels and McAuthur. Their examination of 33 infants with vomiting and marked gastric delay, as shown by the roentgengrams,

was as follows: the plasma pH and the plasma bicarbonate to be normal or increased, while the plasma chloride and cell chloride are often diminished. Also the volume of the blood is often decreased and the relative cell volume increased, and the amount of cell chloride is usually lowered to a relatively greater extent than the plasma. The urine was alkaline and almost chloride free. It is also important to note that alkalosis and chloride reduction are rare in non-obstructive vomiting. One or more of the above changes were present in 88 per cent of their cases. Alkalosis may also be diagnosed clinically especially by shallow breathing, depressed and irregular with frequent long apnoeic pauses. Further evidence of alkalosis might be noted in the appearance of general hypertonicity and such evidence of tetany as carpopedal spasm and positive Chvostek.

Cowie has shown experimentally that alkali delays opening of the pylorus in the normal infant.

Moore has added to the above cardinal symptoms, subnormal temperature and cold extremities. These, he states, usually appear before the baby is two weeks old.

Henske, in a report of 46 cases, states that in three of these, a temperature ranging from 103 to 105 degrees was present at the first examination, and the administration of intra-peritoneal normal saline gave

immediate relief. This, he states, is not uncommon, and designates it as inanition or dehydration fever.

PHYSICAL SIGNS

The physical findings give almost as clear a picture as does the symptomatology. The most important being, visible gastric peristalsis.

Visible peristalsis may occur as an early or late sign. It signifies an attempt of the gastric musculture to force food through a narrowed pylorus. These waves are most pronounced following the administration of food or water into the stomach. They occur just above the navel and pass from left to right across the abdomen, fading always as the pylorus is reached. Gastric waves give no indication as to the severity of the disease. There may only be one wave present at a time, but as high as three might be observed distinctly on the surface of the abdomen.

Palpation of a tumor mass has brought about much discussion in the literature. By some, it is stated as being the most important single finding, while others state that a definite tumor mass is palpable in only 40 per cent of the cases. Finkelstein was the first to palpate the hardened pylorus. He described it as "a round, hard easily circumscribed and somewhat movable mass a little larger

than the end phalanx of the little finger." Abt and Strauss do not stress the fact of a palpable tumor, while Holt, in a series of 141 cases, lists palpation of a tumor mass as the fourth most important finding.

As the roentgengram and fluoroscope have crept into all branches of medicine, so has it become a valuable adjunct in examination and diagnosis of pyloric obstructions. The roentgenographic examination is made in the following manner: the opaque meal should be given the child by bottle or stomach tube, and the stomach fluoroscopically observed from time to time with the child in various positions. It will be noted that the pyloric end of the stomach rounds out, but maintains a clear cut, sharp outline, often without a suggestion of the meal passing through the pylorus. At times, small quantities pass through the pylorus. However, the fact that some of the meal passes through must not be regarded as evidence of a competent pylorus. Strauss states that if 50 per cent of the bismuth remains after four hours, the diagnosis of pyloric stenosis is made, while if 70 per cent passes, the case is probably pyloro-spasm. He believed the fluoroscopic examination was the most important means of making an accurate diagnosis. De Buys, in 1913, found in normal infants that the food left the stomach varying between 1 3/4 and 2 hours, being the same with human and cows milk.

Also, the bismuth might remain as long as 7½ hours in the stomach of a normal child. Thus the roentgenographic examination presents graphic evidence of pyloric obstruction that is confirmatory of clinical findings.

DIAGNOSIS

The diagnosis of congenital hypertrophic pyloric stenosis is usually easy. However, there are a few other conditions which might cause some trouble. Conditions which simulate stenosis are: a. Congenital narrowness of the lumen of the pylorus, b. Adhesion bands, short mesentery or anomalies in the hepato-duodenal ligament, all are relatively rare conditions, but may be accompanied by pyloric hypertrophy, c. Congenital retroperitoneal hernia and d. Cardiospasm. A. and b. respectively can usually be differentiated from stenosis on the basis that they occur immediately after birth, while two to three weeks usually elapse before symptoms of true stenosis appear.

Burt and Tyson, in 1930, reported a case of duodenal obstruction which very closely simulated pylored stenosis. They think the most outstanding point in the differential diagnosis is palpation of a tumor mass. The character of the vomiting and contents of the vomitus are

important. That, in stenosis, is early, projectile and never contains bile, while in duodenal atresia, bile is always present unless the obstruction is above the ampulla of vater. In such a condition, roentgengraphic study is of value.

Congenital retroperitoneal hernia have presented obstructive symptoms, however, there is a large tumor mass which is soft and fixed in position.

Cardiospasm may be present at birth, or may develop later. It is associated with vomiting either during active swallowing or immediately following it, as in cases of esophogeal atresia.

TREATMENT

Treatment of congenital hypertrophic pyloric stenosis can be divided into two groups: those treated medically and those treated surgically. However, neither of the treatments alone will suffice for all cases, but most satisfactory results are obtained by correlation of both treatments.

While some authorities speak of the disease as curable by medical treatment, others state that it is incurable unless surgically treated. It is quite clear, therefore, that at least two different classes of cases are referred to under the same name: one, simple spasm of the pylorus

associated with hypertrophy of the pyloric end of the stomach, not simply of the pyloric ring, the other, in which the spasm, stenosis and hypertrophy are associated with hyperplasia in the wall of the stomach itself.

Medical treatment is advocated primarily by those men who believe in the theory that the causation of pyloric stenosis is due to spasm entirely.

Heubner was the first to report cures of cases by dietary measures. He used corn meal mush feedings at frequent intervals. Birk, in 1914, was probably the first to introduce thick feedings. Then in the spring of the same year, Sauer used Farina paste successfully. Since that time, the medical treatment has followed the trend of Sauer's teachings. Sauer collected 497 cases which had been treated medically with a mortality of 8.9 per cent. He also collected 761 cases which were treated surgically with a mortality of 12 per cent. Ernberg and Hamilton, in 1921, reported 57 cases treated medically with two deaths, a mortality of 3.5 per cent. Porter reported 26 cases with a single death. He used Farina in ten successive cases without having to resort to surgery and with apparent cure.

In 1922, Haas published a paper on the atropine treatment of pyloric stenosis. He suggested that atropine was specific and advised treatment of all cases with that

drug. He used only freshly prepared atropine. One minim of a 1:1000 solution was used in the feeding if the child did not show any idiosyncrasy to the drug. Two drops are added to the second feeding until the desired effects are obtained or taxic symptoms present. As high as 1/50 to 1/85 of a grain can be given in 24 hours. The atropine treatment is continued as long as necessary, with a gradual reduction of the drug until there is not more need for it. Many have used the treatment as suggested by Haas, but since the surgical treatment has been so markedly reduced, little of this type of therapy is being used exclusively.

Barnett, following the suggestion of Dr. Sauer, has used phenobarbitol combined with thick feedings in cases of projectile vomiting. He thinks that the vomiting of this character is a stimulation of the "vomiting center" and that phenobarbitol has the power to diminish its excitability. Phenobarbitol can be given in 1/4 grain doses with each thick cereal feeding, however, he advises beginning with 1/8 grain with each feeding, increasing the dose if necessary. If 1/4 grain of the drug with each feeding does not control the vomiting, then he suggests immediate surgical relief. With all his cases, he used one drop of 1:1000 atropine with each feeding.

A therapeutic measure which has recently been

brought before the medical profession is that used by Barbour. He advises three to five minute applications of the roentgen rays to the upper part of the chest. In his experience, if the obstruction is due mainly to spasm, there follows within 24 hours temporary or permanent relief. If the amount of obstruction caused by the tumor is extreme, little or no abatement of the symptoms occur. He reports 37 cases who failed to respond to internal therapy. In four of the cases, there was little or no change of symptoms, and a Rammstedt operation was performed. In the 33 other cases, the vomiting and visible peristalsis subsided following irradiation.

Previously a definite dividing line could be drawn between those patients treated medically and those treated surgically, and up until 1912 the mortality was about 50 per cent regardless of which road the physician traveled. At present, most cases are treated surgically after they have been given a fair chance on medical treatment. The mortality has been markedly reduced, and ranges between 3 and 10 per cent depending upon the authorities.

SURGICAL TREATMENT

The history of surgical attack upon pyloric stenosis is quite interesting and, as other medical or surgical

problems, has required considerable time with advancement of various types of operation.

Probably the first was in 1887, an operation described by Loreta. The operation was known as "Divulsion of the pylorus" in which an incision was made into the anterior wall of the stomach and graduated sounds were passed into the pylorus. Then in 1893, Cordua operated his first case in which he performed a jejunostomy. Until 1906, Loreta's operation was performed 21 times with 6 deaths and 15 recoveries. In 1897 Schunyzer and Meltzer ~~in 1898~~ were others who practiced surgical intervention. The first successful case of gastro-jejunostomy for this disease was performed by Lobker in July, 1898. Also in 1898, Meyer, of New York, performed a gastro-enterostomy on a child with stenosis, and one week later Stern, in Germany, performed a similar operation. Paterson, in 1906, reported that there had been 25 gastro-jejunostomies performed with 14 deaths and 11 recoveries. Dent, in 1902, advocated single pyloroplasty and reported recoveries from his operation. Shaw and Elting, in 1904, recorded 39 operations with 17 recoveries.

In 1906, Paterson states, "with our present knowledge, it is difficult to decide whether pyloroplasty or gastro-jejunostomy is the better operation." Whether the remote results will prove as satisfactory after pyloroplasty as after gastro-jejunostomy, we do not know.

Dufour and Fredet, in 1908, published results in 135 cases, including those reported by Shaw and Elting, with a 50 per cent mortality. Dufour and Fredet, in 1908, successfully performed their pyloroplastic operation on two infants with hypertrophic pyloric stenosis. Weber, two years later, described the identical muscle splitting operation without incision of the mucosa and with transverse suturing of the longitudinally incised muscularis. The operation of choice, until 1911, was gastro-enterostomy which resulted in a mortality of about 50 per cent. Behrend (as quoted by McGregor), in 1910, incised the pylorus and fed an infant through a catheter passed through the mouth down through the pylorus.

In 1911, Rammstedt performed the operation by making an incision in muscularis down to the mucosa leaving the two margins of the wound free. It is quite interesting to note that this operation would probably not have been performed until a later date had it not been for the fact that he was unable to approximate the two margins of the incision and left the wound gaping with the mucosa bulging into it.

There are, at present, few modifications of the Rammstedt operation, notably the operation performed by Strauss in which he divides the plexuses of Miessner and Auerbach, and in a series of 191 cases had a mortality of only 3 per cent. However, most surgeons, at present, use

the so called Fredet-Rammstedt operation.

First, before consideration of the operating technique, it is important to outline the pre-operative treatment which, according to recent authorities, had done more in reducing the mortality than any advances in surgical skill.

Pre-operative care must be based on reason and not on any routine procedure. Were early diagnosis is made, much less in the way of pre-operative care is required. However, in the majority of these patients, vomiting has caused a loss in blood chlorides, which may result in alkalosis. The starvation, incidental to prolonged vomiting has caused a depletion of the glycogen reserve in the body and a resulting ketosis.

Dehydration must be combated by salt solution given by hypodermoclysis. This supplies fluids for the body tissues and aids in the restoration of electrolytes lost by persistent vomiting. Intravenous administration of glucose solution will account for removal of ketonic acids, and promotes re-establishment of renal functions. A 5 to 10 per cent solution may be used, giving 50 to 60cc every 5 hours until 150cc have been given. In the extremely cachetic patients, blood transfusion just prior to operation is advisable. The use of 10cc of the whole blood per pound of body weight is given.

Because of the marked degree of emaciation, the loss of body heat must be prevented. This can be accomplished by placing the patient in warm blankets beneath which some form of heat may be constantly applied. The legs, arms and chest should be wrapped in flannel bangages.

ANESTHESIA

The anesthesia of choice, recommended by most surgeons, is ether by the open drop method. Other types of anesthesia may be used. Novocaine alone adds to the risk by prolonging the time of operation and may interfere with the healing of the wound. Abt and Strauss reported 221 cases treated surgically, of which 187 were given ether and 34 cases local anesthesia. They state, "where local anesthesia was used, a large amount of purulent discharge occurred." In 10 of 14 cases, which showed wound infections, local anesthesia was used.

TECHNIQUE OF OPERATION

The incision, about two inches long over the outer and upper border of the rectus, exposes the lower edge of the liver, which is held aside to expose the greater curvature of the stomach. This edge of the stomach is drawn

into the wound with rubber covered forceps and followed until the pylorus presents, when the olive shaped, hard, hypertrophic mass is brought into the wound, the index finger of the left hand hooked over its upper surface and the pylorus drawn well out into view. The right end of the incision in the pylorus is determined by the clearly visible junction of the whitish, opaque, and solid pylorus with the bluish, translucent and flaccid duodenum. Extending the incision well up on the pyloric antrum, following the bloodless line between the superficial blood vessels.

The mucosa is exposed by blunt dissection. This is done by spreading the muscle which is easy near the stomach and in the pylorus, but when the duodenal end is reached, much care must be taken. Here the zone of adhesion is the danger, this ring is where the serosa, muscle and mucosa are all bound together and will split together or not at all. The mucosa usually bulges into the line of incision and the operation is completed. Many writers suggest the administration of 100 to 150cc of normal saline into the peritoneal cavity before closure of the abdominal wound.

Advantages of the Rammstedt operation over other previous surgical procedures are many. It requires less time, produces less shock and only a small incision is necessary. The gastrointestinal tract is not opened, and the danger of infection is minimized. Probably the greatest

advantage is the restoration of the continuity of the gastrointestinal tract. The average time required for the operative procedure is about 15 minutes, however, A. F. Brown, in a report of 20 operated cases, performed the operation in an average time of 8½ minutes. While in one case, which was in critical condition, he performed the operation in 3½ minutes.

Dangers of the operations are few, but have been brought out especially by Brown. He relates that the superficial vessels seldom cause trouble, while there are two of the deeper vessels which are almost invariably seen in the muscle during operation. One is met running across the duodenal end of the incision, just at the apex of the fornix of the mucosa, to which it forms a very useful indication. The other appears in the deeper layers of the muscle towards the other end of the wound, and occasionally causes trouble from bleeding.

Inadequate incision in the pylorus and opening into the duodenum are other dangers. There are few accounts in which it was necessary to make a second operation because the first was not complete. However, this occurred once in the small series of cases which have been operated at the University of Nebraska Hospital.

Wallstein reports the following finding from necropsy examination of 25 infants' stomachs who previously had been

subjected to Fredet-Rammstedt operation. Twenty-four hours following the operation there is a wide gap between the cut edges of the muscular coat. The wound is healed within 9 days. The pylorus is relaxed within 2 weeks. The stomach has returned to normal size within one month and gap between the cut edges of muscle coats has practically disappeared in six weeks. In contrast to the operation of gastro-enterostomy which leaves the pylorus unchanged, the Fredet-Rammstedt operation cures the pyloric lesion. Healing is brought about by the cells of the serosa and submucosa but the unstriped muscle cells take no part in the process.

POST-OPERATIVE TREATMENT

Post-operative treatment is very important. Fluids are given as pre-operative and food by mouth is started within three to four hours following operation. Breast milk is probably the most satisfactory and should be given in 1½ to 2 dram doses every 2 hours, increasing the amount slightly with each feeding until the patient is receiving the necessary food requirements. If vomiting occurs, food is with-held for a single feeding or thick feeding might be substituted. The child may be set up to a 45 degree angle while fed allowing gravity to assist the flow of

liquids from the stomach.

The operative mortality has been much reduced since 1911, when Rammstedt first performed his operation. Abt and Strauss report a mortality of 3 per cent in 221 operated cases. Clapton and Hartman 16 per cent mortality in 81 cases, while Bowling and Dowens had a mortality of 15 per cent in 454 cases. Mixter had a 9.5 per cent mortality in 195 operative cases. The most recent article is that published by Lanman and Mahoney who analyzed 425 operated cases with a mortality of 6.4 per cent. From 1915-1923, the mortality on 125 cases was 10.4 per cent, from 1923-1928 on 150 cases a mortality of 7 per cent, from 1928-1931, 150 cases with a mortality of 2 per cent. These men attribute their low mortality in recent years to better pre- and post-operative care, rather than better surgical technique. Donovan reports 119 operated cases with 7 deaths, a mortality of 5.9 per cent.

SUMMARY

During the past twenty years, little has been added to our knowledge concerning the diagnosis, etiology or pathology, however, there has been a marked advancement in the pre- and post-operative care of those infants. The mortality has been reduced from 50 per cent in 1911, to

about 6 per cent or less at the present time. The mild cases, or "pyloroespasm", are treated by dietary and medical means while the severe cases, or "pyloric stenosis", are treated surgically. The X-ray therapy, as advocated by Barbour, should be kept in mind.

Report of Nine Cases From University Hospital,
Omaha, Nebraska.

Case No.	4125	10141	24203	25474	33934	34129	35515	36047	37196
Sex & Color	M-W	F-W	M-W	F-B	F-W	M-W	M-W	M-W	M-W
Age at Entrance	3 mo.	4 wks.	11 wks.	5 mo.	5 wks.	8 wks.	4 wks.	6 wks.	4 wks.
Wt. at Entrance	8# 2 oz.	7# 4 oz.	6# 13 oz.	5# 8 oz.	7# 1 oz.	7# 11oz.	8# 3 oz.	7# 8 oz.	7# 13oz.
Treatment	Sur-gical	Sur-gical	Med-ical	Sur-gical	Sur-gical	Sur-gical	Sur-gical	Sur-gical	Sur-gical
Projectile Vomiting	+	+	+	+	+	+	+	+	+
Gastric Peristalsis	+	+	-	+	+	+	+	+	+
Palpable Tumor	+	+?	-	-	+	-	+	+	-
Constipation Scanty Urine	+	+	+	+	+	+	+	+	+
Operation	Rammstedt		Lactic acid → Cereal		Rammstedt		-		
Anesthesia	Ether	Ether		Local	Local	Local	Ether	Ether	Ether
Wt. at Operation	4# 12oz.	6# 2 oz.		5# 4 oz.	6# 9 oz.	7# 12oz.	7# 2 oz.	7# 7 oz.	7# 9 oz.
Wt. at Dismissal	8# 2 oz.	8# 11oz.	9# 10oz.	10# 2 oz.	8# 12oz.	8# 11oz.	8# 7 oz.	8# 2 oz.	8#
Duration in Hospital	39 days	54 days	43 days	41 days	44 days	16 days	17 days	20 days	14 days

Remarks

Case 10141 - X-ray positive for stenosis.
Case 33934 - This patient continued to vomit after first operation. Second operation performed 30 days later. Patient did not vomit following this operation and left the hospital 14 days later. Patient's brother had been operated for stenosis two years previously.

Case 25474 - X-ray negative for stenosis. At operation, a stenosis was found and addition constricting bands about the duodenum.

Average time in Hospital - 32 Days.
Average gain in weight after operation - 2# 4 oz.
Average age at entrance - 8.2 weeks.
Mortality - Negative.

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