Congenital Hypertrophy of the Pylorus


1900.
Congenital Hypertrophy of the Pylorus

The number of cases of congenital hypertrophy of the pylorus reported in recent years render it probable that this condition is a fairly common cause of death in young children. The literature of the subject shows that an observer who has become familiar with the clinical symptoms of the disease, and is alive to the probability of its occurrence, will not have long to wait for examples. Many instances of "vomiting and maresmones" met with so commonly in practice probably come under this heading. The recovery reported by Batten (9) and Stel's case (26) in which gastro-enterostomy was successfully performed in a child eight months old lend additional interest to the subject.

The first case of this condition is recorded by Williamson of Keith (1) in 1841 and in 1842 Dawson (2) reported another case. The former regarded this condition as a peculiar hypertrophy of the cellular tissue, while Dawson described it as an induration and hypertrophy of the submucous tissue. No mention of an attheroscc case is found until Hirschspring (3) of Copenhagen publised...
in 1888 (3) published two cases—Herschung pointed out that great hypertrophy had taken place in the muscular tissue of the pylorus—W.K. Peddie of Glasgow. In 1889 published the next case (4).

In the discussion of the case which took place at the Glasgow Pathological Society, it was suggested that the condition was a vice of developmental growth. In 1891 G. Newton Pitt (5) published a case, and in this year four allied cases were published by Henshel (6) but one of these post-mortem showed the typical hypertrophy of the pylorus. Finally an epidemic of these cases occurred in 1896—John Thomson (7) published two cases, George three cases, one of which is a case of stenosis rather than hypertrophy; H. Finkelstein (8) one case the first in which the enlarged pylorus was felt and recognised during life; F. K. Hopp (9) one case; Schwirzer one case (10).

In 1897 two cases are mentioned by Fenwick (11) and in this year Schwirzer is said (Rilloin (12)) to have had a case on which gastro-enterostomy was performed. Ashby also recorded two cases.
and Monson published his third case (7) in 1898 Ballantyne & Hayne published one case, Cantley (17) two cases, and Still (18) published three cases in all of which the enlarged pyramids were felt during life. A specimen of this condition was also described from St. Bartholomew's Hospital Museum (16) and Meltzer published a case (14) in which gastro-enterostomy was unsuccessfully performed.

In 1899 Batten (19) recorded the first instance of recovery from this condition where the diagnosis was proved to be correct by post-mortem examination. In Nov. 1899 Abel (20) also published a case of recovery in which gastro-enterostomy was performed.

Dr. John Monson has had two more cases in his practice in Edinburgh, which he has kindly allowed me to re for these to.

Both occurred in March 1900, one in the wards of the Children's Hospital, the other in private practice. The symptoms were typical and in each case the diagnosis was confirmed by post-mortem examination.

I here give a short summary of these published. Only those are given in which the
Typical hypertrophy of the pylorus was found.

Case I. Williamson (1)
A healthy, born male child died from exhaustion when five weeks old after continuous vomiting, which began a few days after birth.
Post-mortem the pylorus was hard and thickened and the lumen admitted only a small probe. The thickening is stated to be chiefly in the mucous and submucous tissue. The submucous tissue forms nearly the whole wall of the pylorus.

Case II. Dvorak (2)
A child born healthy died at ten weeks from marasmus, preceded by convulsions. It was first seen when two weeks old. The history given was that until four weeks old the child had taken the breast well, but then began to vomit two or three times a day. The vomit consisted of curdled milk and mucus. Despite of treatment the vomiting increased and the child died a week after it was first seen.
Post-mortem the pylorus was hard and enlarged.
and about the size of a nut; the lumen
scarcely admitting a probe.

Case 3. Hirschsprung (3)
A female infant—died when thirty days old.
The child took the breast well until ten
days after birth, when vomiting commenced and
continued with great severity until death.
The bowels were constipated.
Post mortem, the desophageus was dilated
and its wall thickened. The stomach was
thickened throughout. The pylorus was a
form cylindrical swelling 2 1/2 centimeters
long. The lumen admitted a moderately thick
sound. All layers of the pylorus were
thickened but especially the muscular.
The mucous membrane was thrown into
six parallel folds.

Case 4. Hirschsprung (3)
A female child died when six months
old. She was put out to nurse at
two months and vomited frequently, in spite
of various changes of diet. At first
she had diarrhea. Death occurred from
Tubercular disease.

Post mortem the stomach was slightly dilated. The pylorus was a cylindrical swelling three centimetres long, and its lumen admits an ordinary pencil. The increase in size of the pylorus was due to hypertrophy of the muscular tissue

Case 5. W. K. Peden (4)

A healthy, well developed, child died at the age of three months. The child was brought up at the breast. When three days old vomiting commenced and in spite of all treatment continued until death. The tongue was clean, the breath sweet, and the vomited matter consisted only of the food taken. Large meals were vomited at once; small quantities were retained until a certain amount had collected in the stomach. Constipation was not obtrusive. No tumour could be felt. Post mortem the stomach seemed larger than normal. The pylorus was a sausage-shaped body with a lumen five millimetres in diameter. Microscopically
the submucosa was enormously thickened and the circular muscular fibres were also much increased.

Case 6. G. Newton Pitt (5)
A healthy well-developed child at birth died seven weeks old. It was admitted to the hospital on Feb. 25th suffering from vomiting and constipation. It was discharged relieved on March 13th and was readmitted on March 5th as the child had vomited continuously since it left the hospital and the bowels had not acted. Treatment relieved the constipation but had little effect on the vomiting which continued until death, preceded by convulsions.

Post mortem the wall of the stomach was hypertrophied and the pylorus formed a fusiform tumour 5/12 of an inch thick. which was found to consist of hypertrophied muscle — there was no stenosis.

Case 7. John Thomson (7)
A male child, well grown and nourished at
at birth died aged 28 days. The infant was bottle-fed and caused no anxiety until ten days old. The motions were scanty. Vomiting commenced on the eleventh day and continued until death from exhaustion. The abdomen was not distended and no abnormality could be felt on palpation. The vomited matter consisted simply of the food swallowed. Post-mortem examination showed the esophagus and stomach to be dilated. The mucous membrane of the stomach was congested and the muscular wall of the stomach especially towards the pylorus was thickened. The pylorus was mucous thickened and feels like a solid cylinder. It measured 9/16 of an inch in diameter, and 1 2/3 of inch in circumference and the wall was 1/6 of an inch thick. The lumen admits 1 or 2 pointed scissors. The mucous membrane is not thickened but thrown into folds by contraction of the muscular coat. Microscopically, the muscular coats of the pylorus are three times as thick as normal.
The increase being chiefly in the circular muscular fibres—the intermuscular connective tissue is small in amount. The condition is one of pure hypertrophy of the muscle fibres. The mucous membrane is normal.

Case 8. John Thomson (7)
A male child healthy in every way at birth died when nine weeks old. The child was fed at the breast till seven weeks old and always brought up a little after each meal, but during the first few weeks was rarely actually sick. At four weeks of age he began to vomit almost every time he took the breast. Various changes of diet were tried and in real broth and then, when the vomiting ceased for a few days but again returned and continued until death which was preceded by convulsions. The vomited matter consisted of the liquid taken and yellowish mucus. The bowels were regular for the first seven weeks but after that there was obstinate constipation.
Post-mortal the esophagus was dilated. The mucous membrane of the stomach was in a state of chronic catarrh, and the muscular wall of the stomach was thickened, especially towards the pylorus. The pylorus is enlarged and feels almost solid. The lower end of the pylorus looked at from the duodenum seems quite closed, and fluid passes when the stomach is compressed. The enlargement of the pylorus is due to enormous hypertrophy of the muscular coat. The pylorus measured $\frac{1}{8}$ of an inch in circumference, $\frac{5}{9}$ of an inch in diameter. The muscular coat was $\frac{3}{8}$ of an inch in diameter.

Case 9. John Thompson (7)
A female child, healthy at birth, died when 47 days old. She was fed at the breast for a week, but as the milk was small in amount and did not satisfy, condensed milk was given also. When vomiting commenced at once. She came under observation when twenty
Seven days old and at that time nothing abnormal could be detected except a small movable tumour in the epigastric region. The tumour was indistinct and of no importance had it not been attached to it had not the symptoms, taken in conjunction with previous cases suggested the case might be one of hypertrophy of the pylorus. The abdomen was not distended. No treatment led to any improvement until the child was fed by a tube, when the vomiting improved for a short time. Sometimes it was found that two hours after a meal nearly the whole of the amount taken could be recovered by passing a stomach tube, showing that little passed the pylorus and that absorption in the stomach was interfered with. The vomiting soon returned and the child steadily lost strength and death occurred preceded by convulsions.

Post mortem the stomach was dilated. The pylorus is distinctly enlarged and feels almost solid. It measures 7/8 of an inch in length, 5/8 of an inch in diameter.
1 1/8 of an inch in circumference. The whole muscular layer was 3/16 of an inch in diameter. Microscopically there was enormous increase in the muscular fibres of the pylorus. The submucous tissue was also increased in thickness. No fluid passed through the pylorus when the stomach was compressed.

Case 10 and 11. John Thomson.

These cases occurred in Dr. John Thomson's practice in Edinburgh in March 1800— one in the wards of the Childrens Hospital and the other in private practice— The case at the Childrens Hospital was three weeks old at the time Dr. Nile performed pylorotomy. The child survived only a few hours. The other case was three months old at death which occurred the morning after pyloroplasty had been performed. The symptoms were similar to Dr. Thomson's previous cases and post mortem the same muscular hypertrophy of the pylorus was found.
Case 12.  
Gran (8)
A female child died at the age of four months. Vomiting began at 8 weeks and there was some diarrhea. When the stomach tube was passed four hours after a meal nearly the whole of the amount taken could be recovered. Farinaceous foods were better digested than mille. The vomiting increased in frequency and the child died from exhaustion.
Post-mortem the muscular layers of the pylorus were hypertrophied, especially the circular layer. The lumen was contracted measuring 3 millimeters in diameter.

Case 13.  
Gran (8)
A male child died at the age of four months. Severe attacks of vomiting and diarrhea were the chief symptoms. Post-mortem the stomach was dilated and the muscular layers of the pylorus were greatly increased in thickness. The lumen was stenosed.
Case 14. Finkelstein (9)  
A female child died when thirteen weeks old. She was fed on cow's milk. Vomiting commenced a few days after birth and in the third month became much more frequent. There was much constipation. The peristaltic movements of the stomach were plainly visible. A few days before death a movable tumour could be made out two centimetres above and a little to the right of the umbilicus. By inflation it was shown to be connected with the stomach.

Post-mortem

The pylorus was a hard cylindrical swelling forming a muscular intermediate piece between the stomach and duodenum.

The pylorus measured two centimetres in length and 1.5 centimetres in thickness. The mucous membrane was thickened and thickened, almost occluding the lumen. Histologically the longitudinal layer of muscular fibres was greatly hypertrophied.
Case 15. De Bruyn Kops (10)
A male child well nourished at birth, died when three months old. Vomiting began within a few hours of birth and continued until death from emaciation and anasarca.
Post mortem, the pylorus was much hypertrophied, the size of a marble and very hard. The lumen was much stenosed.

Case 16. Schwyzer (11)
A well-developed child weighing 8½ lbs died at eleven weeks. For two weeks the child took the breast well, and then began to vomit, occasionally also suffering from attacks of diarrhoea. The stomach was washed out during the sixth and seventh week and the vomiting ceased for a day, but returned again, followed by emulsions and death. Post mortem, the pylorus was a rounded tumour 2.4 centimetres in length, and 2.1 centimetres thick. The lumen admitted a probe 2 millimetres in diameter.
Case 17.  
Ashby (12)  
An infant—healthy at birth and for a week after—then began to vomit, became greatly emaciated, had convulsions, and died.  
Post-mortem the pylorus was enlarged and fremen stenosed. The enlargement was due to hypertrophy of the muscular fibres.

Case 18 & 19  
Sollon Fenwick (13)  
Mentioned briefly by Fenwick in "Disorders of Infancy & Childhood" London 1847.

Case 20  
H. Morley Petersen (15)  
An infant—healthy at birth—died when seven weeks old—Vomiting began at three weeks and continued in spite of all treatment until death.  
The specimen showed dilatation of the oesophagus. The stomach was thickened and the mucous membrane of the pylorus congested and swollen. There is great hypertrophy of the muscular tissue of the pylorus. This ceased suddenly on the duodenal side but tapered off gradually, gradually into the stomach. The pylorus measured 2 inches in diameter & felt very hard.
Case 21. Meltzer (14)

A female infant, well developed and healthy at birth, died when 44 days old. For the two first weeks after birth there was only occasional vomiting but it soon became more frequent and continued with intermissions until death. The vomiting was violent in character and occurred directly after nursing and at no other time. There was no bile in the vomit. The bowels were constipated but not obstinately. The stomach was not at first dilated but afterwards became so. Different kinds of treatment were tried without success and finally gastro-enterotomy was performed by Prof. Meyer. The child died a few hours after the operation.

Post mortem the stomach was dilated, the wall of the stomach was thin at the cardia but hypertrophied at the pyloric end. The pylorus was increased in size, and the lumen stenosed admitting only a probe one millimeter in diameter. The increase in thickness was due to hypertrophy of the inner muscular layer and the submucosa was also increased in thickness.
Case 22. Rolleston & Hayne (35)
A healthy born male child died at eight weeks of age. He was fed at the breast for a fortnight then on condensed milk. From the very first he vomited and the bowels were constipated. He child became extremely emaciated and died. No tumour could be felt in the region of the pylorus.
Post mortem the pylorus was much thickened. It was 3/4 of an inch long and the lumen was about the size of a No. 12 catheter.

Case 23. Cantley (37)
A male child died at 14 weeks of age. There was obstinate constipation from birth. Vomiting began when the child was three weeks old and continued in spite of change in diet. When the child came under observation at eleven weeks of age the chief symptoms were vomiting after food, sour breath, coated tongue and constipation. Under treatment the vomiting became less frequent...
and the constipation improved but the child grew steadily weaker. The vomiting again became worse and death took place from exhaustion.

Post-mortem the pylorus was a sausage-shaped swelling an inch in length distinctly limited above and below. On section the pyloric wall measured 3/8 of an inch in thickness. The mucus membrane was thickened especially at the duodenal end. The lumen of the pylorus admitted only a small probe. Compressing the stomach firmly, forced a small quantity of the contents of the stomach through the pylorus.

Microscopically, the main cause of the pyloric thickening is hypertrophy of the circular muscular fibres.

Case 24. Catterall (17)
A male child, healthy at birth, died at the age of seven weeks. In the first week the child was fed at breast but vomited frequently. In spite of all treatment the vomiting
continued and death occurred. The stools were green and offensive.
Post mortem, the stomach was distended and the splenic end of the stomach adherent to the spleen. The pylorus was a sausage-shaped humour. an inch long, with its lumen imperious. The small and large intestine absolutely empty. The hypophysis was chiefly as in the previous case in the circular muscular fibres.

Case 25. Still (18)
A male infant, a fine child at birth, died when twelve weeks old. He was fed at the breast till 7 weeks of age but always regurgitated some of his food. Vomiting after nearly every meal began at six weeks and the child wasted. He was admitted to hospital when ten weeks old. There was no constipation. Under treatment the vomiting was less in frequency, but still was of daily occurrence. The child improved for a time in spite of
Several convulsions. The temperature was subnormal during the last ten days. Rectal feeding was tried but did not relieve the vomiting. The abdomen was flat and sullen and a few days before death a tumour suggesting a thickened pylorus was felt in the right hypochondrium. Post mortem, the stomach was not dilated. The stomach wall was thickened, the thickening increasing towards the pylorus. There was a tumour-like enlargement of the pylorus which felt like a pylorus affected with stricture. The wall of the pylorus was seven millimetres thick, the muscular part of the wall five millimetres and the mucosa and submucosa together two millimetres. The lumen of the pylorus was from four millimetres in diameter. The microscope showed the thickening to be due to hypertrophy of the circular muscular fibres.

Case 26. Still (8)
A male infant a fair child at birth and
For six weeks afterwards died when fourteen weeks old. He was fed at the breast with the addition of condensed milk on account of large appetite. There had been no regurgitation of food but when six weeks old he began to vomit. Change of diet diminished the vomiting for a time but not permanently. He was admitted to hospital when twelve weeks old. The abdomen was slightly full and peristaltic movements of the stomach, which the mother had previously noticed, were very marked at times passing from right to left across to right across the epigastrium. Peristalsis increased during the last ten days of life and the day before death the hard pylorus could be distinctly felt in the right hypochondrium. The temperature was subnormal, there was almost daily vomiting, the bowels were regular; the child gradually became weaker and died. Post mortem the stomach was slightly dilated, and the wall of the stomach hypertrophied especially toward the pylorus.
The pylorus was considerably thickened and almost cartilaginous to the touch. The mucous membrane was not thickened but thrown into longitudinal folds by the contraction of the pylorus.

The measurement of the pylorus were circumference 4.75 centimetres, thickness of wall 5.1 millimetres, length of pylorus 2.5 centimetres. The lumen of the pylorus was over 3.5 millimetres in diameter.

Microscopically, the thickening was due to hypertrophy of the circular muscular fibres. The mucosa and submucosa were normal.

Case 27. Still (18)
A male child thought to be quite healthy until three weeks old died at 14 weeks of age. Vomiting commenced at three weeks and continued with slight temporary improvements until death. The Bowels were constive. On admission to hospital when twelve weeks old the child was emaciated, the eyes sunken and the fontanelle depressed. Peristalsis was
If the stomach was visible from left to right, and the lower border of the stomach was 3/4 of an inch above the umbilicus. A hard tumour could be distinctly felt in the region of the pylorus. Vomiting occurred after almost every meal; the temperature was subnormal. Nasal feeding commenced five days before death. Diminished the vomiting but the child was now very weak, the bowels had become loose and death occurred.

Post mortem the pylorus was thickened feeling as if infiltrated with malignant growth. The stomach was thickened, especially towards the pylorus, the thickening being evidently muscular.

The diameter of the pylorus was 1.4 centimetres, its length was 2.4 centimetres. The muscular portion of the wall was 4.5 millimetres thick; its lumen was normal admitting a probe 3.5 millimetres in diameter.

Microscopically the thickening of the pylorus was due chiefly to hypertrophy of the
Circular muscular fibres, but in this case the longitudinal muscular layer was also hypertrophied and the submucosa was about twice the normal thickness.

Case 28, Ballon (19)
A male infant, healthy at birth and five weeks afterwards, died at eleven months of age from gastro-enteritis and broncho-pneumonia with the following history. At 5 weeks of age while breast-fed vomiting commenced without apparent cause and the child lost flesh and was constipated. On admission to hospital at eleven weeks of age there was considerable emaciation, the abdomen was flaccid, tympanic, clean, and temperature subnormal. Peristaltic movements of the stomach passing from left to right were visible, and a firm transverse mass like the pylorus could be felt in the right hypochondrium. This was not always palpable when there was no peristalsis. Nasal feeding lessened the vomiting and the child improved
but at the first attempt to feed by the mouth the vomiting returned; a subsequent attempt was successful and from that time the child made steady progress until at nine months of age he was plump and healthy. When eleven months old he died, after a few days illness, from gastro-enteritis and broncho-pneumonia.

Post-mortem considerable broncho-pneumonia was found. The stomach wall felt unusually thick. The pylorus was firm and hard; the lumen admitted a probe 4 millimetres in diameter. On section the pylorus was 1.5 centimetres in diameter; the total thickness of the pyloric wall was 5.5 millimetres; the muscular coat 4 millimetres, the mucosa and submucosa together measured 1.5 millimetres. After hardening sections were cut and stained, and the micrometric measurements of the different layers were compared with those of a normal pylorus in a child 12 months old, and the comparison showed that the circular muscular layer
in this case was greatly increased in
thickness.

Case 29. Batten (19)
Dr. Batten also mentions a second case
of this disease which was under his
care and ended fatally, at 14 weeks.
The enlarged pylorus was in this case also
distinctly felt previous to death.

Case 30. Abel (20)
A child born of healthy parents and
health, for three weeks after birth,
in the fourth week was taken with
tocolic, constipation and later with vomiting.
The vomited matter contained no bile.
Treatment led to no improvement. At
intervals the upper part of the abdomen
was much swollen. The swelling
subsided after vomiting. When the
child was admitted to hospital there
was vomiting after food. No trace of
bile in the vomit. The motions were
plenty and greenish black. At time
a sausage like tumour could be felt extending
from the splenic to the hepatic flexure of
the color. The tumour is rather hard, elastic and tympanitic on percussion — slow peristalsis can be distinctly traced over the surface of the tumour. Indentation right edge of the tumour a round hard obstruction can be felt, but not distinctly located, which disappears when the tumour subsides. The diagnosis of mechanical pyloric obstruction was made and the operation of gastro-enterostomy according to Wolfers method with a jejunum loop was performed. At the operation the stomach was found dilated and the pylorus was a smooth round very hard tumour 3.5 centimetres in length and about the same in circumference. The lumen of the pylorus did not admit a probe 3 millimetres in diameter. The muscular coat of the stomach is thickened and the mucous membrane swollen and a part of the coat of the stomach closes up the pyloric valve. The child bore the operation well, took its food, gained weight and there was no return of the vomiting. On the eleventh day after the operation it was doing well.
Wolfe
Hensal describes three cases (6), Gran (8) a third case besides the two given, and Ashley also a second case (12) but as the typical muscular hypertrophy is not mentioned they are probably cases of Stenon with a different pathology.

General Considerations
Family History
Under this heading there is nothing particular to note. The mother in several cases had been out of health during pregnancy and in one case several previous children had been ronies. Most of the children were born healthy.

Sex. The disease is fairly evenly distributed between the two sexes.

Age at which first symptom appeared
In two cases vomiting commenced at birth.
In seven cases --- in the first week.
In six cases --- in the second week.
In three cases --- in the third week.
In three cases --- in the fourth week.
In one case --- in the fifth week.
In two cases --- in the sixth week.
In one case vomiting did not begin until the eighth week.
Symptoms
The first and most prominent symptom of the disease is vomiting. Commencing without apparent cause, sometimes within a few hours from birth, but as a rule beginning before the end of the first month, it steadily increases in frequency and severity. At first it may occur but once or twice a day and only after a large meal and might be mistaken for regurgitation so common in infants; in the end the smallest quantity is vomited at once. Treatment may lead for a time to improvement, but the vomiting almost invariably returns. It is more affected by the quantity than the quality of the food taken, though in case 12 Fran observed that primrose food was better digested than milk and Gentley found that soured milk was better retained than ordinary milk. As a rule large meals are returned at once, and small quantities are returned until a certain amount has collected in the stomach when they are promptly expelled with
great violence. Ballin noticed that the act of swallowing seemed to start the vomiting as when the child was fed with exactly the same food by means of a tube the vomiting ceased.

The vomited matter consists of the food taken more or less altered by digestion; it contains no bile. Often if a stomach tube be passed two or three hours after a meal, nearly the whole of the food taken can be recovered.

The child does not appear to be in pain except after vomiting; usually the breath is sweet; the tongue clean and there are no signs of gastric retention except as a secondary occurrence in the later stages of the disease. The temperature is often subnormal. The bowels in the majority of cases are constipated, but in four cases diarrhea is mentioned.

The general symptoms are those of slow starvation, gradual wasting, weak pulse, loss of strength, cold skin, and depressed fontanelle.
Physical Signs

The abdomen is usually flat and indurated. Peristaltic movements of the stomach can sometimes be seen passing from left to right across the epigastrium. In some cases a tumour has been felt and recognized as the enlarged pylorus during life. The earliest age at which this has occurred was at 27 days of age in case 9. It may be possible to make out the enlargement of the stomach which often occurs in these cases.

Diagnosis

The chief points are:
The vomiting coming on without apparent cause in an otherwise healthy child; the absence of the signs of jaundice; the persistence of the vomiting in spite of treatment; there is no bile in the vomit; the temperature is subnormal; peristaltic movements of the stomach may be visible; the signs of a dilated stomach; by passing a tube two or three hours after a meal nearly the whole of the amount taken may be recovered.
whereas normally the stomach of a child should be empty in this time; finally, the presence of a tumour in the position of the pylorus would confirm the diagnosis as other tumours in this region are rare. Should there be any difficulty in palpating the abdomen it would be advisable to examine under an anaesthetic.

Diagnosis
The large mortality in the cases reported under the diagnosis was very unfavourable. The two cases of recovery reported last year show that the condition is not necessarily fatal.

Treatment
Feeding with a naso-tube is the only treatment that has produced any improvement. It should be continued for some days after the vomiting has ceased and then an attempt should be made to feed the child by the mouth, and if this fails the naso-tube should be again used for a time. The child
requires to be kept thoroughly warm.

If after a fair trial nasal feeding fails and an abdominal section confirms the diagnosis gastro-enterostomy should be performed.

Morbid Anatomy

The condition for sometime before death has been practically one of starvation and as a consequence there is great emaciation. In one case there were tubercular disease of the lungs and in another dilatation of the pelvis of the right kidney and ureter.

As a rule no disease is found outside the alimentary canal and this below the pylorus is perfectly normal. The one constant condition found was the hypertrophied pylorus, and this in all cases but one, case 14, was due almost entirely to hypertrophy of the circular muscular fibres. In considering this point it is as well to remember, as still (18) has pointed out that the normal pylorus in young children varies
Considerably in thickness and that the variations mainly depend on variations in thickness of the circular muscular coat. The hypertrophy found in these cases bore no constant relation to the duration of life.

The esophagus was in some cases dilated in others normal. The stomach is often dilated and the wall hypertrophied especially toward the pylorus, while at the cardia it is thinner than normal. The mucous membrane of the stomach is normal without there has been gastric catarrh when the usual signs are present.

The pylorus is greatly increased in size. It forms an almost solid cylindrical tumour varying in dimensions. Cautley says that roughly it may be taken to be about the size of the last joint of the little finger. To the touch the pylorus is hard and firm and feels like solid muscle. Opeing the duodenum and looking at the pylorus from below, the wall of the pylorus projects into the duodenum, and
and the lumen seems almost closed. The view from the duodenum has been compared to the oesuter, but Still (13) has pointed out that the normal pylorus has this appearance though in a less degree. In longitudinal section the thickening of the pyloric wall is well seen and also that the increase in thickness is chiefly due to hypertrophy of the muscular tissue. The mucous membrane is thrown into longitudinal folds by contraction of the hypertrophied muscle. There is no fibrous structure of the canal. According to Still (13) though his observations are rather limited in number the diameter of the normal pylorus in the first year of life as seen post mortem is 3.5 to 4.0 millimetres. If this is accepted as correct there was no stenosis in nine of the cases recorded as it is either mentioned that there was no stenosis, or the diameter was above 3.5 millimetres in this number. In twelve cases where the lumen was either below below 3.5 millimetres in diameter
If it is stated there was stenosis, it is probable that the stenosis was produced by contraction of the hypertrophied muscle. Microscopic examination showed marked hypertrophy of the circular muscular fibres. The longitudinal layer of muscular fibres was in some cases slightly increased in thickness, in other cases normal. The exception to this rule was in Fickelstein's case in which there was great hypertrophy of the longitudinal muscular layer. The intermuscular connective tissue is stated by some observers to be increased in amount, by others it is described as less than normal. The mucous membrane is usually normal. The submucous tissue in some cases was increased in others no alteration was observed.

Pathology.
The following theories have been advanced to explain this condition:
1. That the hypertrophy of the pylorus is due to spasm the result of some irritant in the stomach after birth (ulcer, hyperacidity). Dawson.
2. The condition is simply a redundancy of foetal growth. Nature has produced an excess of muscular tissue (Adams), an excess of all the tissues of the pylorus (Mellinger).

3. There is a congenital stricture of the pylorus followed by hypertrophy of the stomach especially at its pyloric end (Finkelstein, de Bary, Rohr, Fanderau).

4. That there is a functional disorder of the nerves of the stomach, probably due to delayed or imperfect development, leading to ill-coordinated and therefore antagonistic action of the muscular arrangements of the stomach. That the functional disorder begins in utero and leads to increased action of the pyloric sphincter and consequently to hypertrophy. That the passage downwards of the amniotic fluid which the foetus is supposed to swallow is the exciting cause of the increased muscular action.

(John Thomson)
The objections to the first theory are that life in these cases is often very short and it is highly improbable that the amount of hypertrophy found could take place in the time; that the degree of hypertrophy found bears no constant relation to the child's life; that no cause of irritation can be demonstrated; usually, there is no gastric catarrh, varying the diet having little effect. The fact that in Batten's case nasal feeding led to improvement without any alteration in diet seems to prove that the condition is not due to an irritant in the stomach.

The second theory is supported by still observations that the normal pylorus varies in thickness and that the variations are due to variations in thickness of the circular muscular layer. If however the disease is due to a permanent malformation it seems improbable that the symptoms in some cases should be so long delayed. The case of recovery reported by Batten and other probable recoveries reported by Finkelstein (19) and Robin (21) is a still stronger objection.
The third theory that there is a "congenital stenosis" is contradicted by the fact that in many cases no stenosis was found.

The last theory, suggested by Dr. John Monson, has received most support. It agrees with our experience that muscular hypertrophy is the result of increased work and it affords an adequate and reasonable explanation of the symptoms during life, and the post mortem appearances found. But the disease has a nervous origin is rendered probable by the fact that in some cases the vomiting seems to be started by the act of deglutition. In case 28 when the child was fed by a tube the vomiting ceased but at once commenced when feeding by the mouth was attempted. The nervous disorder must vary in intensity, appear in different cases and from time to time in the same case, as in some cases the symptoms are present from birth, while in others they are delayed for weeks. In cases of recovery we must
Suppose that the stomach regains its normal innervation. The theory is difficult to prove or disprove, but it at any rate affords a reasonable explanation of the disorder. Meltzer in the Medical Record Aug 1890 has advanced the following objections to Thomson's theory:

1. That theory supposes muscular tissue only to be affected whereas the submucous tissue is also hypertrophied.
2. A nervous theory did not account for the hyperplasia in the submucous tissue.
3. The assumption that obstruction of the pylorus leads to hypertrophy is without foundation.

His criticisms do not appear to me to invalidate the theory. The submucous varies in thickness in normal cases and the amount of thickness found in some cases might well be due to distention brought about by spasm of the sphincter pylori. Although no actual proof can be given that obstruction of the pylorus leads to hypertrophy, it is not unreasonable
to suppose such a result, and it is the usual explanation of the hypertrophy found in hypertrophic stenosis in adults. Therefore, in most of those so far advanced we explain the symptoms in those cases were no stenosis was found.
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