

CONGENITAL STENOSIS of the PYLORUS

by

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The subject of Congenital Stenosis of the Pylorus presents interest alike for the Physician, the Pathologist and the Surgeon. The first recorded observation was made in 1841 by Williamson of Leith. The symptoms described leave no doubt that the patient suffered from Congenital Stenosis of the Pylorus, which proved fatal when the child was 5 weeks old. The Stomach and Pylorus were removed post mortem and were exhibited at a meeting of the Edinburgh Anatomical Society, where the condition was considered to be Congenital Scirrhus of the Pyloric extremity of the Stomach. In 1842 Dawosky reported a case with similar symptoms, which proved fatal at the age of 10 weeks. At the post mortem examination a hard Pyloric tumour was found, the lumen of which would hardly admit a probe. Dawosky looked upon the condition as one of hypertrophy with induration of the submucous tissue. Landerer, in 1879, and Maier, in 1885, again called attention to the subject of Congenital Pyloric Stenosis by the publication of some observations they had made, both clinical and anatomical. But their cases

were all observed in adults, the two youngest patients being respectively 16 and 12 years of age, and they are now not generally regarded as true cases of Congenital Stenosis of the Pylorus. In 1888 Hirschsprung recorded two cases of his. Both patients suffered from symptoms similar to those which will be described below as typical of the condition and both died. From the post mortem examinations Hirschsprung was the first to recognise that the hypertrophy of the Pylorus was chiefly of the muscular tissue. In 1889 Peden showed a specimen at a meeting of the Glasgow Pathological and Clinical Society taken from the body of a child who had died from malnutrition, the result of Congenital Stenosis of the Pylorus. At a meeting of the Pathological Society of London in 1891 Newton Pitt showed another specimen taken from an infant 7 weeks old at death, and he drew attention to the hypertrophy of the muscular coat of the Stomach as well as the Pylorus. In 1891 Henschel published notes of four cases which possibly were cases of Congenital Stenosis of the Pylorus. One of them recovered without operation and the others died between the ages of 7 months and 2½ years. At the post mortem examinations of the three fatal cases none of them revealed the marked muscular hypertrophy which is usually met with at the Pylorus. Thomson in 1896 published notes of two fatal cases (see below)

and of another the following year with full descriptions of the morbid anatomy. About the same time as Thomson published his first two cases, Gran recorded notes of three cases. Soon afterwards Finkelstein published a paper on Congenital Stenosis of the Pylorus and gave notes of a case of his own and three cases from Heubner's private practice. In 1896 De Bruyn Kop and Schwyzer each reported a case and the following year Ashby published a paper on the subject with notes of two cases. In his book on "Disorders of Digestion in Infancy and Childhood" in 1897 W.S.Fenwick gave notes of two cases of Congenital Stenosis of the Pylorus, which he had treated. In 1898 cases were recorded by ~~Meltzer~~<sup>Meltzer</sup>, Batten and Rolleston, and in that year Stern had a case on which he operated. The following year, Still recorded three cases he had had under his charge, and Abel also operated on a case of Kehr's about the same time. Since then the literature on the subject of Congenital Stenosis of the Pylorus has been rapidly increasing, and a considerable number of cases have been recorded. The following have either written on the subject, or recorded cases - Blackadder, Cantley and Dent, Dival, Fletcher, Greeff, Kehr, Larkins, Lobker, Monnier, Nicoll, Pritchard, Rolleston and Crofton-Atkins, Saunders, Schmidt, Simonson and Southworth. A complete bibliography will be found below.

Owing to the kindness of Mr. H. J. Stiles and Dr. John Thomson I am enabled to give notes of their cases. The first three cases recorded have been published by Dr. Thomson and they are copied from the published notes. Three of the cases were treated at the Edinburgh Children's Hospital by Dr. Thomson when I held the position of House Surgeon to the Hospital and I had ample opportunity of seeing the cases. Mr. Stiles operated upon two of these three cases and I assisted him at the operations.

### Case. 1.

On 10th February 1894 I saw, with my friend Dr. Duddingston Wilson, a male infant, aet. 18 days, who for a week had been vomiting everything that was given him.

The child was much emaciated, but no other physical sign of disease could be found. The heart, lungs, liver, spleen, and other organs were all normal, so far as could be ascertained. There was no distension of the bowels, and no abnormality was discovered on palpation of the abdomen. The following are the main facts of the history:-

The parents were tolerably healthy, and none of their other eight children had been similarly affected. When about seven months pregnant the mother had influenza, after which she had never fully recovered her usual strength.

At birth the infant was well grown and well nourished, and during the first ten days of life he caused no anxiety. He was fed from the bottle, and his appetite was always good. He seemed to take as much milk as other babies, and appeared to be quite comfortable. He was, however, rather given to drinking greedily, and when he did so some of the milk came up afterwards; this happened several times a day. The urine

and the meconium were natural in appearance; and although the motions were always costive and somewhat scanty, they were otherwise normal in character.

When 3 days old he was given a dose of castor-oil, and vomited it. During the next few days he had three other doses of the same, only one of which he retained. These doses were given, not on account of any abnormal symptom, but merely because the nurse thought she had "never got him right cleaned out."

On the eleventh day of life he was given a fifth dose of castor-oil. It came up at once; and the child vomited everything that was given him from this time until he died. Generally the vomited matter consisted of the milk, etc., that he had swallowed. On one occasion, however, (thirteenth day of life), he brought up a little brown fluid "like beef-tea," and some hours afterwards passed a dark motion. He seemed to have no pain in his belly, but there was apparently some uneasiness caused by the passing of urine. His motions were usually quite normal in appearance, but very small in amount.

A great variety of treatment was tried, but all without the least effect on the vomiting. There was never any sign of blood in the vomited matters, and the infant showed no sign of severe pain. In fact, there was no change noticed, except increase of the emaciation

and weakness, until the child died on 20th February, aet. 28 days.

Post-mortem examination. Thoracic organs all perfectly normal. No fluid in peritoneal cavity. Liver congested, but otherwise normal. Gall-bladder full of dark bile. Spleen, kidneys, suprarenals, and pancreas normal.

Oesophagus dilated, so that, when it collapsed, it measures  $\frac{1}{2}$  in. in diameter. Stomach greatly distended, measuring  $4\frac{2}{3}$  in. from the duodenal end of the pylorus to the extremity of the cul-de-sac, and  $2\frac{3}{4}$  in. across near the cardiac end. The pylorus is very much thickened, so that it feels like a solid cylinder. It measures  $\frac{9}{16}$  in. in diameter and  $1\frac{2}{3}$  in. in circumference.

When the stomach is moderately compressed, the fluid in it readily passes up the dilated oesophagus, but none of it goes through the pylorus into the duodenum. On making an incision about 3 oz. of milk and water escaped. The gastric mucous membrane is congested, but otherwise seems normal; and no abrasion or scar of any kind can be found. The muscular wall of the stomach is greatly thickened, especially toward the pyloric end. Probe-pointed scissors readily pass through the pylorus; and, when slit up, the wall is found to be fully  $\frac{1}{6}$  in. thick. The mucous membrane lining it is not thickened, although thrown into folds



by the contraction of the muscular coat.

Dr. Robert Muir kindly examined microscopically sections of the pylorus and of part of the wall of the stomach. He reports that "on the surface of the mucous membrane there is found some mucus and cellular débris, but the mucous membrane itself is practically normal. The muscular layer of the pylorus is more than three times the normal thickness at that age, the greater part being composed of circular (i.e. sphincter) fibres, although some are irregularly arranged. The connective tissue between the fibres is exceedingly small in amount, and, proportionately to the muscular tissue, even less than in the normal condition. The condition is, therefore, one of pure muscular hypertrophy (Photograph I.): The serous coat is normal. The portion of stomach examined shows a similar condition of muscular hypertrophy, although less in degree"

The duodenum and the rest of the intestinal canal are quite normal, and are almost empty. There are a few soft, congested, slightly-enlarged mesenteric glands near the upper end of the bowel.

Case 2.

1896,

John M., aet.  $9\frac{1}{2}$  weeks; seen on 25th March, for persistent vomiting, with Dr. Edward Carmichael, to whose kindness I am indebted for the opportunity of taking the following notes.

As in the former case, no physical sign of disease of any of the organs could be discovered, but the child was thin and weakly.

He was the youngest of four children, the others being strong and healthy. The father also appeared robust. The mother was very neurotic, and suffered from some gynecological ailment. During the whole of her pregnancy she had suffered more or less from vomiting, which she said had weakened her a great deal.

At birth the baby seemed quite healthy in every way, and the meconium was normal in amount and appearance. During the first three days there seemed to be pain on micturition, but this afterwards passed off.

For the first six weeks or so the child was on the breast. His mother thought that she had very little milk to give him, but the nurse said that there must be a good deal, because the baby was always wetting his bib by putting up a little after each nursing. He rarely, however, was actually sick during the first few weeks.

When about 4 weeks old he began to vomit after almost every time he took the breast. He was gradually weaned in the next fortnight, and given, first, fresh cow's milk and water, afterwards condensed milk, Mellin's food, and a variety of other things; but the vomiting continued.

The vomiting usually occurred about half an hour or so after food was taken, and did not seem to be accompanied by any great pain or discomfort. Sometimes he would take several meals at intervals of an hour or two without vomiting, and would then apparently bring them all up at once. Shortly before I saw him he had ceased vomiting for some days, while getting nothing but veal broth and sherry whey. At the end of this time, however, a dose of castor-oil brought on the vomiting again. The vomited matters consisted of the liquids taken, along with yellowish mucus; there never was any trace of blood in them.

On some occasions he would drink as much as 4 table-spoonfuls from the bottle at a time; but for a fortnight before I saw him he had not taken more than from 2 to 4 teaspoonfuls at once.

During the first seven weeks of his life his bowels moved regularly, and his mother saw nothing in his motions, either in their appearance or amount. For the last fortnight there was very obstinate constipation.

The urine was copious in amount and appeared normal.

The child always slept remarkably well, and never seemed to wish for a drink between 10 P.M. and 5 A.M.

On the evening of 25th March the stomach was washed out with warm water, and the child's diet was limited to pancreatised milk. This, however, had no beneficial effect. About 12 o'clock on the following night he began taking fits, and he died about 5 A.M. next morning.

Post-mortem examination. 27th March, 9 A.M.

Body considerably emaciated; much rogor mortis. Heart normal. Lungs somewhat emphysematous, but otherwise fairly normal. Liver fatty and a little enlarged. Spleen, kidneys, suprarenals, pancreas, and bladder normal.

On opening the abdomen the lower border of the stomach is seen to reach within  $\frac{1}{2}$  in. of the umbilicus. The organ itself is collapsed, but seems unnaturally elongated, and is bent on itself at an acute angle about 2 in. from the pylorus. The bowels appear normal. There are a few slightly enlarged mesenteric glands.

The oesophagus, stomach, and duodenum were removed and examined.

The oesophagus is dilated, measuring, when collapsed,  $\frac{6}{16}$  in. in diameter.

The stomach is elongated, and slightly hour-glass shaped. It measures about 2 in. across at its widest part, and 6 in. in length from the duodenum to its cardiac end. The pylorus is distinctly enlarged, and feels almost quite solid; it measures  $1\frac{7}{8}$  in. in circumference and  $\frac{5}{8}$  in. in diameter.

The duodenum, when opened, appears quite normal in every way, and the papilla is normal and patent. The lower end of the pylorus seems quite closed when seen from the duodenum, and no fluid passes through it when the stomach is compressed.

When the pylorus is slit open, its thickening is found to be entirely due to enormous hypertrophy of the muscular coat, which measures  $\frac{3}{16}$  in. in diameter.

The stomach contains about a teaspoonful of tough yellowish mucus. Its lining membrane is congested, but presents no ulceration or abrasion. Its muscular wall is thickened very considerably towards the pylorus, while at the cardiac end this is not so marked.

Microscopic sections of the pylorus were made and submitted to Dr. Muir, who found "the mucous coat in a state of chronic catarrh; the muscular layer enormously thickened, and composed, as in Case I., of practically pure unstripped muscle; the serous coat normal in appearance and thickness."

### Case III.

On April 13th, 1896, I saw with my friend Dr. T. B. Darling a female infant aged twenty-seven days, who was suffering from constant vomiting.

She was somewhat emaciated, but seemed in every respect a well-developed child, and her heart and lungs were normal. On careful palpation a small, hard, very movable tumour could sometimes be felt in the epigastric region. This was, however, very indistinct, and no weight would have been attached to it had it not been that the symptoms present, taken along with the condition of the pylorus found in my two former cases, suggested its possible importance. The rest of the abdomen was normal, and not at all distended.

The parents were strong healthy people. They had been married for more than twelve years before pregnancy occurred. This was attributed to the presence of a gynecological abnormality which, at the end of that time, had been removed by operation. One miscarriage had occurred before this child was born.

During the early weeks of her pregnancy, the mother was travelling a great deal by sea, and was severely sick.

At birth the infant weighed 7 lbs. 2 oz., and seemed perfectly normal. There was very little breast

milk, and what there was did not satisfy her. She would remain at the breast for even forty or fifty minutes at a time without getting enough. She soon became exceedingly restless, and cried a great deal as if in severe pain. Her motions were very small, green and slimy. After persevering for a week or so her mother gave up the attempt to nurse her, and a mixture of cow's milk and water was given from a bottle. So far there had been scarcely any vomiting. At first the bottle seemed to satisfy the child much better than the breast had done; but vomiting began almost at once and rapidly became severe. It occurred very frequently, and the milk was expelled with great violence through the mouth and nose. Vomiting always occurred at once if she took more than two ounces at a time. Peptonised milk was tried for a while, but with no greater success.

Progress. About the 13th April a wet-nurse was procured and her milk was drawn off and administered to the baby through a Auvard's breast-pump. This proved, however, very little better than the other ways of feeding; the child returned about half of the milk she swallowed. Often, after taking a drink, she would immediately vomit it, but if put to the breast-pump again at once she would retain the second supply.

Stomach-washing with plain warm water was tried,

but this had not the slightest effect in stopping the vomiting. Bismuth was also given in fairly large doses with no result. A number of other fluids besides milk were then most carefully tried, and it was found that the stomach acted in the same way to all, whether it was breast-milk, diluted cow's milk, plain or peptonised, cream and whey, or chicken tea, etc. There was no ascertainable difference in the rapidity and certainty with which they were returned.

On April 17th, we commenced feeding the child by gavage. A soft rubber catheter was introduced into the stomach by the mouth every two hours, and 2 oz. of dilute peptonised milk was poured down it through a filter. At first this succeeded surprisingly in checking the vomiting - feed after feed being retained for the first two days, and only occasional meals being vomited. It was found, however, when the tube was introduced at the end of the two hours' interval, that instead of being empty as it should have been, the stomach usually contained nearly two full ounces of sour, slightly turbid fluid with a few small, soft curds in it. It was thus evident that although the stomach had still the power to partially digest its milk, there was extremely little absorption of its contents going on, and nothing was passing out of it through the pylorus. At this time there was very



little urine passed. It was found that the infant seemed more comfortable after the peptonised milk than after the same quantity of breast-milk.

On the 19th, there seemed to be much more fluid absorbed, as only from  $\frac{1}{8}$  to  $\frac{1}{4}$  of the quantity that was put in could be drawn off after two hours. The urine also passed much more freely, and within three or four days the child was found to have gained two ounces in weight.

On the 20th, she retained all the meals given through the stomach-tube, but one which she was allowed to suck from a bottle was returned at once. During the next few days, however, she began to vomit the food even when introduced with all care through the tube. This continued, and although a number of other foods were tried, there was no improvement. The child steadily emaciated and lost strength. Convulsions began on April 28th, and she died quietly on the morning of May 3rd.

The post-mortem examination took place on May 4th at 4 P.M., and was restricted to the examination of the stomach and neighbouring parts. The intestine contained a considerable amount of gas and a small quantity of liquid yellow faeces. The oesophagus seemed normal, not being dilated as in my two former cases.

The stomach was large, measuring about  $4\frac{1}{2}$  in. in

length from the duodenal end of the pylorus to the cardiac extremity, and  $2\frac{1}{4}$  in. across at the larger end. It contained a considerable amount of liquid food which had been given shortly before death, and a great deal of very tough mucus. None of the fluid could be squeezed through the pylorus by compressing the stomach. The pylorus, before it was opened into, felt like an almost solid cylinder, although it was not quite so hard as in my two former cases. It measured about  $\frac{7}{8}$  in. in length,  $\frac{5}{8}$  in. in diameter, and  $1\frac{1}{2}$  in. in circumference. On opening the stomach the wall was found to be greatly thickened towards the pylorus, but thin at the cardiac end. The thickening was due mainly to increase in the muscular coat. The gastric mucous membrane seemed normal, and there was no trace of any ulceration past or present to be found on it.

Microscopical sections of the pylorus and adjacent stomach-wall were made for me at the Laboratory of the Royal College of Physicians. The condition present resembled, in most respects, that described and figured in my former paper as found in the other cases. The epithelial layer of the mucous membrane was normal, and the great bulk of the thickening of the part was due to enormous increase in the inner circular layer of the muscular coat, while the increase in the outer longitudinal layer was comparatively slight. The

whole muscular layer measured  $\frac{3}{16}$  in. in diameter. As in the other cases there seemed no increase in the intra-muscular connective tissue. There was, however, one striking difference in this case, namely, that here the submucous connective tissue was greatly increased in amount. Where this was least marked, it was fully  $2\frac{1}{2}$  times, while in some places it was as much as 4 times the normal thickness. In the part of the stomach-wall adjacent to the pylorus, of which a section was made, this increase of submucous connective tissue was even greater than in the pylorus. The serous coat was normal.

#### Case IV.

M. H. Girl. Aged 6 weeks on admission to the Edinburgh Children's Hospital on April 26th, 1900. Complaint - Vomiting and wasting. Father and mother alive and healthy. There was another child in the family, but she died of meningitis when she was 17 months old. At birth patient seemed a healthy child and was well nourished. The labour was natural. The child was put on the breast for 2 or 3 days. but there was very little milk and so barley water (2 parts) and cow's milk (1 part) were given. The child seemed to do very well for a month and when four weeks old, was very fat. The bowels were regular and the motions were natural. There did not seem to be any trouble with digestion. Patient slept well.

History of Present Illness. When patient was four weeks old she began to vomit. At first it was not often and it always came on between 10 and 30 minutes after a feed. The child did not seem ill apart from the vomiting. But the vomiting became more frequent and at the same time the child began to be troubled with flatulence. At first the vomiting would occur once or twice a day, but it became more frequent until all the food seemed to be vomited and the vomiting became more forcible. Sometimes the vomit was

unchanged milk, and at other times curds, depending on how long it had been kept in the stomach. Patient had never had any diarrhoea. Lately there has been considerable constipation. The child did not seem to pass much urine during the last fortnight. The mother thinks there has been very little, if any, pain. The child always seems hungry.

State on Admission to the Children's Hospital.

Height 21 in. Weight 6 lbs. 12 oz. The child is very emaciated and extremely feeble. Fontanelle is depressed. Skin is soft. Thorax normal in shape. Heart and Lungs are normal. The abdomen is rather flat in appearance. No gastric peristalsis seen. The Pylorus is readily **felt** and evidently thickened. The vomit has a slightly greenish tinge. Patient put on raw meat juice and barley water.

April 23. Vomiting has continued in spite of small feeds being given and changes made in the diet. The patient is rapidly sinking. The Pylorus is **felt**, but no gastric peristalsis has been seen.

Evening. The parents refused to leave patient in Hospital. She was therefore taken home this afternoon.

The child died two days later and a post-mortem examination was given.

Post-mortem examination. The body is very emaciated. Heart and Lungs normal. The stomach is rather large and its walls are thickened. The Pylorus is hard, thickened and somewhat elongated. The lumen of the Pylorus admitted a probe 3m.m. in diameter. The stomach and Pylorus were cut open (Plate II) and the thickening is seen to be due to muscular hypertrophy.

Case IV.

M.W. girl. Healthy at birth, born Jan. 7th, 1900 at full time. Normal labour. Father and mother alive and healthy. Three other children alive and healthy. One child died at 7 weeks of age from vomiting, which was similar to the patient's. The child was put on the breast. There was vomiting almost from the first. The mother's breast began to inflame and so the child was put on condensed milk, but there was no improvement in the vomiting and there was wasting. After this had been tried for about a week boiled cow's milk and barley water were tried. The vomiting was no better and sometimes was very forcible. Most of the food was returned. Sometimes the food would remain down an hour, but usually it was vomited at once. The vomit does not seem ever to have contained any blood or bile. The vomiting did not improve, and there was progressive emaciation, and the child was admitted to the Edinburgh Children's Hospital on Feb. 25th, 1900.

State on admission to Hospital. Temp. 98°

Weight  $5\frac{1}{2}$  lbs. Child is small, thin and emaciated. Skin, dark and soft. Cry is vigorous. Fontanelle slightly depressed. Tongue and lips clean. Abdomen not distended. No projection at any part. No

peristaltic waves made out on the abdominal wall. No tumour felt. Chloroform given and with deep palpation an elongated swelling about the <sup>thickness</sup> ~~size~~ of a man's finger was felt a little to the right of the middle line and about midway between the ensiform cartilage and the umbilicus.

Feb. 26th, 1900. Child cried a lot last night. Peptonised milk given, but child vomited several times. Bowels moved several times during last 24 hours. Motions small and dark-coloured. General vitality is good.

Feb. 27th, 1900. Child very quiet. Pupils dilated. Fontanelle depressed. Weight  $5\frac{1}{2}$  lbs. Vomiting persists in spite of changes in food. No peristalsis seen. Temperature is continuously subnormal.

Feb. 28th, 1900. Vomiting not so violent, but patient seems weaker. Patient is fed by stomach-tube now and a little brandy added to food. Fontanelle more depressed. Bowels regular. Motions small and dark in colour. Pulse 130. Temperature subnormal.

Mar. 2nd, 1900. Child is losing ground. Vomiting continuous. Nutrient enemata given 4 hourly. No peristaltic waves seen. Diagnosed as Congenital Stenosis of the Pylorus and operation advised.

Mar. 7th, 1900. Parents agree to operation now.



Child is more emaciated and <sup>the</sup> vomiting, though occasionally better for a day, is on the whole much about the same.

Evening. Mr. Stiles operated at noon. The stomach did not appear to be enlarged, but its walls were very thick for a child of patient's age. The pylorus was hard, thickened, and elongated, and felt like an almost solid cylinder 1 in. long. The pylorus was first excised. On the duodenal side it looked like an Os Uteri, and on the gastric side it gradually thinned into the stomach. (Plates III, Fig 1 & 2). It was found impossible to get the Duodenum up to the posterior wall of the stomach and so ~~an~~ an anterior-gastro-duodenostomy done. After the operation the child cried pretty heartily and did not seem collapsed at all.

But the child suddenly became worse about 8 P.M. and died.

P.M. The anastomosis was perfect, though Mr. Stiles thought it should have been nearer to the greater curvature of the stomach.

On examining the Pylorus microscopically there was seen to be marked hypertrophy of the muscular tissue. The hypertrophy was chiefly of the circular fibres. (Plates IV & V.)

## Case VI.

A.L. Boy. Born Feb. 5th, 1900. Labour was normal. The child appeared healthy at birth. It was the only child. The baby was put on the breast. On second day the weight was  $7\frac{1}{2}$  lbs. The nipples were a little sore and the child did not seem anxious to take the breast. The bowels were constipated. Dr. Darling, who was attending the mother, noticed on the thirteenth day that the child seemed to be starving. That day he ordered the child to have some peptonised milk as well as the breast, but he vomited after the former. The weight on the fourteenth day was 6 lbs. 12 oz. The vomiting continued and patient was kept on the breast alone. He was rather better when on the breast alone for a few days, but mouthfuls of milk would frequently come up. On March 4th, 1900 (27 days old) the weight was 7 lbs. 2 oz. The food was then varied (whey, barley water etc.) but the vomiting was rather worse. Dr. John Thomson saw the case in consultation on March 13th. The weight was then 6 lbs. 5 oz. The child looked emaciated. The abdomen was not distended. A resistance was felt in the region of the Pylorus, but no definite tumour was made out. 2 oz. peptonised milk was given and two hours later the stomach tube was passed. The whole of the 2 oz.

of milk just given was withdrawn, as well as some from the previous meal. No peristaltic waves were seen over the stomach.

Mar. 14th, 1900. Mr. Stiles did a posterior gastro-enterostomy. The stomach was found to be large and the Pylorus was hard and large.

Mar. 15th, 1900. Child seemed to be doing well until 6 a.m. today, and then began to be rather uneasy. He then suddenly collapsed and died at 8 a.m. He had been given nutrient enemata every four hours since operation.

Post-mortem examination. Stomach distended. It was full of dark blood (about 2 oz.). The same amount was also found in the Duodenum. The operation wound was quite satisfactory. The bleeding had evidently been from the anastomosis. There was no blood found below the Duodenum.

The Pylorus was thick. It was 3.3c.m. long. (Plate VI.). The muscular layer was 4m.m. thick. The peritoneal and mucous layers were normal. The stomach was 11c.m. long and 6½c.m. broad at the widest part. It was rather elongated and the fundus was too marked for a child of its age. The walls of the stomach were thickened except at the Cardiac end. The other organs were normal. Death was evidently due to Haemorrhage. A photograph is given showing the stomach opened from the front exposing the anastomosis. (Plate III fig. 3.).

Case VII.

M. F. F. Girl. 25 days old on admission to the Edinburgh Sick Children's Hospital on March 20th, 1902. Complaint - vomiting and wasting. The vomiting had been present for about 13 days. Father alive and healthy. Mother alive, but said to have a weak heart. She had suffered for four months during the Pregnancy. There are two older children in the family, and they are alive and healthy. At birth the patient seemed all right. The labour was natural, but the patient was born about 3 weeks prematurely. She did quite well until 12 days old.

Present Illness. Up to the 12th day patient seemed quite well. There was no vomiting and the bowels acted naturally. She was taken out of doors on the 8th day. From birth patient was put on the bottle, the food consisting of 1 part of milk to 2 parts of water. On the 12th day the child began to vomit. She vomited practically the whole of the feeds for several days. Benger's food was then tried without any improvement. Veal tea with Barley water was next used and there was a little improvement. But it was only temporary. Sherry whey was given a trial, and for two days the vomiting was less frequent and all the food did not seem to come up. For two days before admission patient was put on Horlick's malted milk, but

the vomiting persisted. The day before admission only two tablespoonfuls were given two hourly and the vomiting was hardly so persistent. The patient, before admission to Hospital, has been rather sleepy. She seemed to have no pain. She is gradually getting more emaciated. There has been no diarrhoea, and the motions have been fairly natural. The urine was high coloured and scanty. The tongue has never been furred and patient has not had thrush. She has been troubled with flatulence. The vomiting has mostly occurred one hour after food. The stomach was washed out by Dr. John Thomson the night before admission to Hospital. The vomiting was less frequent after this until admission.

Condition on Admission to Hospital on March 20th, 1902. Very emaciated, but otherwise healthy. Heart and lungs healthy. Abdomen flat and soft. No peristalsis seen. Liver was felt about a finger's breadth below the costal margin in the right nipple line. Something hard felt in the region of the Pylorus. Temperature 97°

March 20th, 1902. 6 p.m. Mr. Stiles has operated on patient this afternoon. A muscular thickening, about 1 in. long and cylindrical in shape was found at the Pylorus. Loreta's operation was done. Dressing forceps were passed through the Pylorus which was stretched. Something was felt to give way, but

no rupture could be made out anywhere. The details of the operation will be described later. The patient seemed cold after the operation and there was some collapse. Nutrient enema given - albumen and water,  $\frac{1}{2}$  oz. of each - but it was not all retained.

6.45 p.m. 1 oz. saline solution injected subcutaneously. Child seems warmer and improved generally.

8 p.m. Child worse. Gr.  $\frac{1}{100}$  Strychnine given subcutaneously.

11 p.m. Tinct. opii  $m\frac{1}{2}$  in starch given as enema.

11.30 p.m. The following enema given - albumen and water, each 1 oz. whisky  $m20$ . Almost all retained.

March 21, 1902. 6 a.m. Subcutaneous injection of Saline (1oz.) given  $2\frac{1}{2}$  hours ago. Nutrient enema now repeated and retained. To have subcutaneous injection of Saline (1oz.) every 6 hours, and nutrient enema every 4 hours.

March 22, 1902. Temperature went up to  $100^{\circ}$  last night and is  $101^{\circ}$  this morning. Enemata nearly all retained. Mouth feeding started this morning. Patient got 1 oz. peptonised milk. Most of it returned with some bile-stained fluid. Bowels washed out with Saline, and Tinct. Opii  $m.i.$  in starch used before nutrient enema given.

2.45 p.m. Albumen and water, equal parts, given

by the mouth. Only swallowed 2 drachms. Subcutaneous injections of Saline continued, but amount increased to 3 or 4 oz. every 6 hours.

8.45 p.m. Patient does not take albumen by mouth - makes no attempt to suck. A small rubber tube was passed to the back of the mouth and fluid passed along it, but it simply came out at the other side of the mouth. Child has become much worse. Temperature has dropped to 97.8°. Pulse scarcely felt - weak and irregular. Enemata retained.

9 p.m. Child died.

Post-mortem examination. Marked emaciation. Peritonitis present. Wound on stomach found to be all right, but a rupture was found on the posterior aspect of the Pylorus .

Death from Peritonitis, the result of rupturing the Pylorus at the operation.

### Case VIII.

A.S. 4½ weeks on admission to the Edinburgh Sick Children's Hospital on May 4th, 1902. Complaint - Vomiting. Father alive and healthy. Mother alive but said to be anaemic. Patient is the only child of parents. Forceps were used to deliver the child. He looked very healthy at birth. Patient was fed on milk and water for the first five days, then he was put on the breast for one week. The mother had little milk so patient was put on barley and oat flour water with milk from the bottle. This did not seem to suit him, so he was put on milk and water alone - 1 part of milk to 2 of water - but patient constantly vomited. Two teaspoonfuls of whisky were given during the first two weeks of life. The bowels were usually constipated. No fits and never any attacks of diarrhoea.

History of present illness. About two weeks ago the child began to vomit. The time of vomiting varied. Sometimes it would occur at once after a feed, but at other times not for an hour. Patient was constipated. Soon after birth the motions were dark coloured, but lately have been rather light. The vomiting continued in spite of changes in diet. Sometimes it was violent and this was generally when a feed had been kept in the stomach for an hour. Very rarely was most of a feed



kept down.

State on Admission to Hospital on May 4th, 1902.

Weight  $6\frac{1}{4}$  lbs. Child is poorly nourished. Complexion pale. Skin soft. Sneezes occasionally. Vomits soon after taking milk. Cries when the abdomen is palpated. Head well-formed. Anterior fontanelle slightly depressed. No cranio-tabes. No facial irritability. Hair plentiful and light-coloured. Eyelids rather puffy. Nose and ears well-formed. No discharges. Abdomen slightly tumid. No enlarged lymphatic glands made out. Temperature  $96.4^{\circ}$ .

Respiratory and Circulatory Systems - Thorax well formed. Apart from slight evidence of Bronchitis, nothing abnormal.

Alimentary System. No teeth. Gums and lips rather pale. Tongue moist. Vomits after taking food. Food mostly all returned. Hepatic dulness normal. Whole abdomen is resistant. Epigastrium seems tender. No peristaltic waves seen. Pylorus not felt.

9. 5. 1902. Stomach been washed out daily. Patient given peptonised milk by the mouth. Nutrient enemata of egg albumen given. Subcutaneous injections of Saline given night and morning. But child seems to be losing ground and the vomiting persists. Distinct peristaltic waves seen passing from left to right

over the region of the stomach.

10. 5. 1902. Mr. Stiles operated this morning. On deep palpation under the anaesthetic Mr. Stiles was able to make out a distinct tumour a little to the right of the middle line and below the margin of the liver. Loreta's operation was done. The Pylorus was first stretched with metal bougies up to No. 19 and then with a tracheal dilator. No rupture could be felt or seen, and the Pylorus seemed well dilated. After the operation the patient was rather collapsed. A subcutaneous injection of Saline was given (1 oz.). Put on nutrient enemata.

11. 5. 1902. Patient seems to be doing well. Cries loudly. Anterior Fontanelle not depressed. Enemata not retained during the night, but child has retained two this morning. 3 oz. Saline injected subcutaneously. 1 oz. of fluid given by the mouth - egg albumen 1 part and water 3 parts. To be continued every two hours, and nutrient enemata also given four hourly.

Evening. No sickness. Nutrient enemata mostly retained.

12. 5. 1902. Patient doing well. Enemata retained. No sickness. 1 oz. egg albumen and water given hourly during day. Temperature continues subnormal. Pulse fairly good. Patient cries loudly.

Subcutaneous injections stopped.

16. 5. 1902. Strength is well maintained. Bowels regular. Motions well formed. No sickness, in fact patient has not vomited since night of operation. Patient now having  $1\frac{1}{2}$  oz. peptonised milk every two hours. Enemata continued.

24. 5. 1902. Patient seems to be holding his own. Slight thrush. Cries very loudly. Feeds increased to 2 oz. two hourly. Enemata stopped - tend to irritate.

29. 5. 1902. Patient is progressing. He has put on 12 oz. in weight during the last 6 days. Today some undigested food in the motions.

30. 5. 1902. Vomited a little today. Stools rather better.

31. 5. 1902. A very little milk returned - not with violence. Dressing changed and stitches removed. Wound quite healed.

4. 6. 1902. No more vomiting, but weight not increased during the last four days. Taking food well. General condition good.

18. 6. 1902. Child doing well. Good appetite. Sleeps well. Getting stronger. Gaining weight.

27. 6. 1902. Child is steadily gaining weight. No vomiting. Looks well. Taken out daily. A little constipation.

4. 7. 1902. Patient discharged cured. No vomiting. Diet peptonised milk. Weight - 8 lbs. 10 oz.

Patient was brought from time to time to see Dr. Thomson at Hospital. The child was quite healthy. Seen last on Dec. 5th, 1903, aged 8 months. Weight then was  $25\frac{3}{4}$  lbs. Looked extremely well. No trace of rickets.

Case IX.

A. M. Boy. Born May 27th, 1902. At birth 4 lbs. Dr. Chalmers said child was healthy at birth though small. Only child. Father and mother alive and healthy.

July 8, 1902. Weight 6 lbs. 2½ oz. Child been doing well up to now.

Then the child began to vomit. He had been fed on milk (1 part) and water (2 parts) with a little cream. The vomiting continued and Dr. Thomson saw patient in consultation with Dr. Chalmers on July 20th 1902. Before this time lime water and barley water given with milk and it improved vomiting for a little, but got worse again. On July 17th a wet nurse was obtained and the child took the breast well for a day or two, and then vomited irregularly during the day. On July 20th, 1902, the child was very emaciated. Abdomen rather distended. Peristaltic waves seen over the stomach. No tumour felt. Peptonised milk tried with whey. Patient had a good night and slept well. He did well for a few days and then became worse again and vomited several times.

July 30, 1902. Patient vomits occasionally. Looks fairly well. Weight 5 lbs. 13½ oz.

Aug. 1, 1902. Vomited violently through the

nose. Stomach washed out. It contained  $1\frac{1}{2}$  oz. fluid. There was some spasm of the oesophagus caused by passing the tube. The wet nurse again tried. Child looking pinched.

Aug. 5. 1902. Very sick during last night. Stomach again washed out. 2 oz. coagulated milk withdrawn. Patient seemed easier after stomach washed out. Child is wasting.

Aug. 8. 1902. No improvement. Stomach washed out. Passing of tube caused pain owing to spasm. Peristaltic waves seen passing over the abdomen from left to right. Started as an elevation on the left side and passed towards the right. When wave got to the Pylorus the elevation died away. No pain apparently, but deep palpation elicited some tenderness.

Aug. 11. 1902. No improvement. Peristaltic waves marked. Pylorus felt. Dilatation of stomach rapidly increasing and lower border reaches to below the umbilicus. Operation advised.

Aug. 13. 1902. No improvement. Child rapidly wasting. Vomiting continues. It is not very violent. Weight 5 lbs.  $8\frac{1}{2}$  oz. Mr. Stiles operated about noon today. Loreta's operation done. There was rupture, about  $\frac{1}{3}$  in. on the anterior aspect of the Pylorus. It was sutured. The stomach was very large. The child

remained unconscious for a long time after the operation. A subcutaneous injection of Saline was given.

Evening. Temperature 99.8°. Nutrient enemata given and also subcutaneous injections of Saline alternately.

Aug. 14. 1902. Temperature 101°. Fairly good night. Child looks comfortable, but rather irritable. Nutrient enemata and subcutaneous injections of Saline continued.

Aug. 15. 1902. Child looks pinched and collapsed. Marked peristalsis seen after child put on the breast.

Aug. 16. 1902. Patient looks better. Temperature coming down. Sleeps well. No vomiting. Weight 7 lbs. 2½ oz. with dressing on.

Aug. 20. 1902. Child not so well yesterday. He looks pinched and miserable. Lost weight. Stomach distended. Some of the stitches cutting.

Aug. 23. 1902. Temperature 102°. Child weaker and collapsed. Bowels rather loose. Rectal injections stopped. Kept on the breast. Subcutaneous injections of Saline continued. A little squinting. Fontanelle depressed. Seems almost moribund.

Aug. 24. 1902. Child a little better. Temperature 99°. Diarrhoea better.

Aug. 25. 1902. Much improved, though rather restless. Temperature been up, but now coming down. Motions are slimy.

Aug. 26. 1902. Patient better and happy. Subcutaneous injections of Saline continued. There is a general improvement.

Aug. 28. 1902. Patient is making progress and gaining weight. After this there was no vomiting and patient became a healthy-looking child.

Oct. 1902. Weight 25 lbs. Well grown child. No vomiting. Complete recovery.



Case X.

Baby Y. Boy. Healthy child at birth. Weight  $6\frac{1}{2}$  lbs. Natural labour. Father and mother alive and healthy. First child. Dr. W.E.Frost attended the mother at the confinement, and I am indebted to him for the notes up to the operation. There was apparently nothing wrong with the child up to 8 weeks of age, except that occasionally a little food was returned. There was no violent vomiting. During these 8 weeks diluted cow's milk was given. When 8 weeks old the child began to vomit violently. During the first day the vomiting was not frequent - about three times in the 24 hours - but after that it was more frequent. It generally came on after a feed, but sometimes not for an hour. The milk was then peptonised, but this caused no improvement. Other foods were tried, but with the same result. There was rapid emaciation. Dr. Frost, from the nature of the vomiting, thought the child was probably suffering from Congenital Stenosis of the Pylorus, though no peristaltic waves were seen and it was not certain whether the Pylorus could be felt. On July 27th, 1902, Mr. Stiles saw the patient and agreed with Dr. Frost's diagnosis. Mr. Stiles felt an enlarged Pylorus. Operation was advised.

July 28, 1902. Child 9 weeks old. Mr. Stiles

operated. Enlarged Pylorus distinctly felt under chloroform. Stomach did not seem much enlarged, but the Pylorus was thick and hard. Loreta's operation was done. Lombard's oesophageal dilators were used for stretching the Pylorus. There was no rupture. The child stood the operation well, and soon recovered from the anaesthetic. Subcutaneous injections of Saline (about  $1\frac{1}{2}$  oz.) were given 6 hourly, and nutrient enemata were given 4 hourly. The child seemed to do well, and 24 hours after the operation 1 drachm of diluted peptonised milk was given by the mouth. This was repeated in 2 hours, and, since there was no vomiting, it was increased to  $1\frac{1}{2}$  drachms every hour on the 3rd day after the operation.

Aug. 2. 1902. No vomiting. Child doing well. Feeds increased to 2 drachms hourly and subcutaneous injections of Saline and nutrient enemata (with a little brandy) were continued. There is a little oedema of the eyelids. Bowels constipated. Urine fairly plentiful. Heart seemed a little weak.

Aug. 4. 1902. OEdema of hands and feet now present. Child seems bright. No vomiting. Gets 6 drachms peptonised milk every hour. Nutrient enemata stopped. Castor-oil given for constipation. The temperature had risen a little after the operation, but is now  $98.2^{\circ}$ .

Aug. 6. 1902. OEdema still present. No sickness. Feeds increased to 1 oz. hourly. Child is progressing.

Aug. 8. 1902. OEdema not going down. Child vomited once yesterday and once this morning. It was not violent.

Aug. 10. 1902. Child vomited a little yesterday and today. Sterilised milk and Barley water, equal parts, now given. Bowels not so constipated. Child not so bright.

Aug. 11. 1902. No improvement in vomiting. About half the food seems to come up. Nutrient enemata (with a little brandy) started again. No subcutaneous injections given.

Aug. 12. 1902. Child is very fretful. Some constant sickness. Not much food comes up at a time. Child is wasting. Possibly a recurrence of Stenosis.

Evening. Child very collapsed. Temperature 96°. Pulse very feeble. Hot bottles used and strychnine injected subcutaneously.

Child died at 11.30 p.m.

Post-mortem examination. No peritonitis. Wound in stomach healed. Stomach wall thickened. Little or no dilatation of that organ. The muscular coat gradually thickened towards the Pylorus. The Pylorus is hard and thick. Stricture is about  $\frac{3}{4}$  in.

long and the muscular coat at its thickest part measures  $2\frac{3}{4}$  m.m. The Stricture has an Os Uteri appearance when looked at from the Duodenum, owing to an abrupt termination of the Pyloric Sphincter on that side (Plate VII). The thickening is due to an increase in the muscular tissue. (Plates VIII & IX).

Case XI.

P. B. E. Boy. 5 weeks old when admitted to Edinburgh Children's Hospital on Nov. 11th, 1902. Complaint - Vomiting since 9 days old. Father and mother alive and healthy. There was another child, who is 9 years old and is quite healthy. The mother had a miscarriage at the 6th month between the two children. The child was healthy at birth and the labour was natural. Patient was 9 lbs. at birth, and was put on the breast.

Present Illness. Child very healthy, fat child at birth. He was put to the breast on the second day. He took it readily and well. There was no vomiting or diarrhoea until the 9th day when patient suddenly vomited. The milk was ejected with considerable force. The vomiting then continued after each feed. Dr. Thatcher was called in. Magnesia was given but the vomiting persisted. The child was kept on the breast. Even in two days the mother said she noticed the child getting thinner. Patient was then taken off the breast for two days and Barley water was given. There was vomiting immediately after each meal, but it was not so violent. The child was put back on to the breast. Vomiting continued though not quite so bad. Some of the milk seemed to be retained. Patient was

again taken off the breast and given diluted cow's milk (not boiled) with lime water and sugar in it. 1 oz. was given every two hours. Most of this was vomited though sometimes he would keep a feed without sickness. During this time the patient was rapidly becoming emaciated and not sleeping well. He did not cry out. The bowels were moved daily. The motions were green, but not very offensive. The cow's milk mixture was continued for four days and then Bengers' food was tried. Everything was vomited and so after one day the cow's milk mixture was tried again. Later Mellin's food was tried. Dr. John Thomson then saw child in consultation. At that time gastric contents not "shot out", but seemed to "well up." The vomiting usually took place immediately after feeds, but sometimes not for an hour. The vomit was usually curdled milk, but sometimes was a straw coloured fluid. During the three days before admission to the Hospital patient had convulsions - usually one every two hours the first day, and then more irregularly on the other two days. The convulsions were general.

On admission to Hospital - Nov. 11th, 1902 - patient weighed 7 lbs. Well grown and developed. He had a healthy skin and complexion, but was extremely emaciated and pinched; rather irritable and hungry,

but bright. The head was well shaped. Fontanelle was depressed. No cranio-tabes present. Features and body generally, well-formed. No enlarged glands. Cry loud and strong. No cough. Pulse 112 to 120. Temperature 98°.

Respiratory and Circulatory Systems. Thorax well formed. No abnormal features made out.

Haemoporitic System. Spleen not enlarged. No enlarged lymphatic glands.

Nervous System. Normal.

Skin. Soft and natural.

Digestive System. Lips of good colour. Child obviously thirsty and wanting to be fed. Food given, but soon vomited forcibly. Motions rather white and scanty, not offensive. Abdomen - contracted, but looked normal. No fulness over the region of the stomach. Felt what seemed like a contracted and thickened Stomach and Pylorus..

Nov. 11. 1902. 1 p.m. Stomach washed out. Then 1 oz. peptonised milk with 10m. Brandy given through tube. Child not sick after this. Subcutaneous saline injection given. Pulse before injection 132, now 112. Patient put on 1 oz. peptonised milk with 10m. Brandy every 2 hours. Rectal injections of Sterile water given every 4 hours.

Nov. 12. 1902. Since admission yesterday patient has had 9 oz. Saline solution injected subcutaneously and has retained 2 oz. of Saline enemata. 13 oz. peptonised milk given by the mouth. Only had one slight fit, at 10 o'clock last night. The child looks stronger and cries very loudly. Passed urine freely and sweated. He has had three small motions, natural in colour and consistence. No peristaltic waves seen on the abdomen.

Nov. 13. 1902. Child slept very well during the night. Has had two fits since yesterday. Slightly sick after feed last night - about  $\frac{1}{2}$  oz. of feed returned. Took all feeds well until 5 a.m. today, when very sick after feed. Child sick again at 10 a.m. All the feed was returned and it was sour. Bowels not moved since yesterday. Child looks well. Pulse fairly good volume. Temperature 98.4°. When child vomits, food usually wells up.

Evening. Vomited three times since morning. No fits. Weight 7 lbs. Stomach washed out. Considerable amount of curds removed. To be given egg albumen, peptonised milk, and raw meat juice, 3ii each, with Barley water, 3i, every 2 hours.

Nov. 14. 1902. Morning. No more fits. Sick 4 times since last night. Still weighs 7 lbs. Marked peristalsis seen this morning over the region



of the stomach passing from left to right. (Pylorus felt just to the right of the middle line below the margin of the liver). Mr. Stiles seen case with Dr. Thomson. Operation to be done tomorrow.

Evening. Child frequently sick. Not so bright.  
Nov. 15. 1902. Had a good night. Only sick a little. Has retained rectal injections - 1 oz. every 4 hours. No subcutaneous injections given today - some still not absorbed. Eyes rather puffy. Pulse 140 - fair volume. Peristaltic waves distinctly seen on the abdomen.

11 a.m. Mr. Stiles operated. Pylorus very hard and large. Loreta's operation done. Bougies, 11-19, first used to dilate Pylorus and then Tracheal dilation used. Child seemed to stand operation well. Subcutaneous injections of Saline to be continued - 2 to 4 oz. about every 6 hours.

Nov. 16. 1902. Child seems to be doing very well. Pulse 140 - volume very good. Child warm; sleeps well. Vomited dark-coloured fluid at 7 a.m. Nutrient enemata (2 drachms raw beef juice and 1 oz. Saline) every 4 hours. Fed by the mouth. Raw beef juice and barley water with 10m. Brandy given. Sick soon after.

12.30 p.m. Fed again but vomited everything.  
Temperature 101:

12.50 p.m. Patient suddenly died.

Post mortem examination. Body emaciated. Heart, lungs, liver and kidneys normal. Oesophagus not dilated. Stomach - Rather enlarged. Walls thickened. The muscular coat is hypertrophied. About 1 in. from the Pylorus it is  $1\frac{1}{2}$  m.m. thick. It gradually thickens as it passes into the sphincter (Plate X). Pylorus is hard and thick. It measures  $\frac{7}{8}$  in. long. The thickening is due to muscular hypertrophy, being chiefly in the circular fibres (Plate XI). The sphincter has an Os Uteri appearance when looked at from the Duodenum.

#### Case XII.

H. G. Boy. Admitted into Edinburgh Children's Hospital, April 6th, 1903, aged 4 months.

Complaint - Vomiting and emaciation. The vomiting has been going on since 2nd day of life. Father alive and healthy. Mother used to have dyspepsia, but not since marriage. Three other children in family - alive and healthy. No specific history can be made out. Labour - chloroform and instruments. At birth the baby is said to have been small but plump. The

mother thinks he was heavier than now, but child was not weighed. The mother says she had plenty of milk.

History of Illness. On the first day patient got a little sugar and water and did not vomit. On the second day the child was put on the breast, and that night he vomited a large quantity of milk. After that the child vomited every day, but only mouthfuls at a time until a fortnight old. When a fortnight old the vomiting became worse. He began to bring all the milk up when he got it - "like a fountain." It seemed sometimes to be more than he could have taken and was very sour. "He was never satisfied." When patient was a month old (Jan. 5. 1903) a doctor who saw him ordered Nestle's food during the day and the child was to be kept on the breast at night. This was done for a week, but the child vomited the same. He vomited after about every second feed now, and seemed then to bring up the whole two feeds. When the child was five weeks old he was taken off the breast altogether and kept on Nestle's food only. It was only given three hourly at first and in small quantities, but later he had to get more. Patient continued to vomit. When  $6\frac{1}{2}$  weeks old the patient was put on Allen & Hanbury's No. 1 food instead of Nestle's milk. For one or two days he seemed a little better, and then the vomiting became worse than before. On

Feb. 5th, 1903, when patient was just over 8 weeks old, Dr. J.G.S. Jamieson of Brechin saw him. He stopped the Allen & Hanbury's food and put the child on Barley water alone. Dr. Jamieson describes the case at this stage as follows:- "It was thin to a skeleton, abdomen protuberant, pained and very sleepless;" "put on barley water and raw meat juice. Vomiting almost ceased; pain less and slept more." Then he tried peptonised milk diluted, one to 4 or 5 parts. "The vomiting became more violent but not so frequent as formerly." Then Fairchild's peptogenic milk powder used (30 minutes action) and a little Panopepton during the night. There was always a little vomiting. With every change of diet there was about 36 hours of improvement and then the patient became rather worse again. The feeding was regular and clean. The abdomen continued protuberant." Dr. Jamieson "by auscultation and percussion made out the stomach reaching to within  $\frac{1}{2}$  in. above the umbilicus. Peristaltic waves can be seen in epigastric region passing from left to right. Baby has not put on any flesh. Present weight 6 lbs. 7 oz. Bowels have been rather constipated. During the last five weeks the mother thinks the child has been freer of pain and slept better. The vomiting has continued, though now rather less frequent than formerly. The vomiting is more

marked when patient lies on the right side, but he seems to like lying better on that side." Yesterday patient had two big vomits in the day and several small ones. Bowels moved five times yesterday.

State on admission to Hospital. Height 20 in. Weight 7 lbs. 3 oz. Patient is a very small, emaciated, old looking baby. A little excoriation on left buttock. Very cross: always crying. Cry is strong. There are no enlarged lymphatic glands. Head well developed. Fontanelle slightly depressed.

Circulatory and Respiratory Systems. Thorax well shaped. Expansion good. Nothing abnormal made out.

Digestive System. Tongue clean. Child seems hungry. Abdomen looks relatively distended. Some peristaltic waves seen, beginning in left lumbar region and going across abdomen below the level of the umbilicus. No abnormal resistance felt. Liver made out extending to one finger's breadth below the costal margin in the right nipple line. No thickened Pylorus felt. Temperature 98°. Pulse 136. Patient put on 2 drachms raw meat juice with 1 oz. Barley water every two hours.

April 7, 1903. Patient kept on raw meat juice and Barley water and has not vomited once since admission; but he is losing weight. Stomach washed out.

Only a little mucus with remains of raw meat juice came up. No peristaltic waves seen today. To have peptonised milk alternately with raw meat juice and barley water.

Midnight. While child being fed very evident peristaltic waves seen. They commenced well to the left at the costal margin and ran to right just above the level of the umbilicus. Waves about  $1\frac{1}{2}$  in. x 1 in. Child has not been sick.

April 8. 1903. 4.30 a.m. Child had feed at 3 a.m. Took it as usual, but rather slowly. Just now child suddenly collapsed, became very cold and blue and pulse with difficulty felt. Gaspd very infrequently. Strychnine Gr.  $\frac{1}{200}$  given subcutaneously. Hot pack used. Child did not respond to stimulation and died now.

Post-mortem examination. Extremely emaciated. Abdomen distended. Ladder pattern visible chiefly above umbilicus. Some excoriation around anus and over thighs. On opening the abdomen the greater part of its extent is occupied by coils of small intestine, only a small strip of the liver is visible. No large intestine is seen. On the left side a considerable portion of the body of the stomach is seen reaching to the surface and extending from the costal margin to the level of the Iliac crest. On turning the coils

of small intestine to the right the whole stomach is exposed. It extends vertically downwards and is sharply bent on itself at its lower end. (Plate XIII Fig 1.) The lower extremity is below the level of the Anterior Superior Iliac Spine, and about  $1\frac{1}{2}$  in. above the upper margin of the Symphysis Pubis. The Pylorus is very long and thick -  $1\frac{1}{8}$  in. long and fully  $\frac{1}{6}$  in. thick at the widest part. The stomach measures vertically  $4\frac{5}{8}$  in. and transversely  $1\frac{3}{4}$  to 2 in. The stomach contained some fluid but none could be squeezed through the Pylorus. On opening the stomach its walls are found much thickened and its mucous membrane pale and thickly covered with mucus. The OEsophagus is dilated, measuring  $\frac{5}{8}$  in. in diameter in its collapsed state immediately above its junction with the stomach. (Plate XII Fig 2.) In the greater part of its extent it measures nearly  $\frac{1}{2}$  in. in diameter, but about the level of the 6th Cervical vertebra it rapidly narrows. The spleen, liver and kidneys present no abnormal features. The large intestine is much contracted. Heart and lungs normal. Thymus is small. Death evidently due to starvation due to Congenital Stenosis of the Pylorus.

Case XIII.

W. S. Boy. Born March 10th, 1903. Normal Labour. Child healthy at birth. Father and mother healthy. They have had 7 other children, 6 of whom are alive and healthy and one died from Bronchitis. The mother was rather weak during the pregnancy. Child began to vomit on the second day. Since the fourth day of life, the patient said to have vomited almost everything. Very rarely would he keep a feed down. The vomiting was violent, and sometimes the gastric contents were squirted through the nose with considerable force. The mother did not notice any waves on the abdominal wall. The child was kept on the breast for a fortnight, but in addition got some oat flour water night and morning. On the seventh day the latter was replaced by Allen & Hanbury's food. This seemed to make the vomiting more, so it was stopped and the child kept on the breast alone until a fortnight old. Then he was taken off the breast because the vomiting was so bad, and peptonised milk was given. The vomiting persisted and the child was admitted to the Edinburgh Children's Hospital on April 4th, 1903.

State on Admission to Hospital. Weight 9 lbs. Healthy looking child and fairly well nourished.



Complexion good; skin moist; no eruptions. No enlarged lymphatic glands. Child good-natured. Head well formed. Fontanelle slightly depressed. No cranio-tabes. Thorax well formed. Circulatory and Respiratory Systems normal.

Abdomen - not distended; soft. No dulness or tenderness. Gastric peristalsis seen. (Plate XIII.) Wave  $1\frac{1}{2}$  x 1 in. passes from left costal margin to middle line. Only occasionally seen. No tumour felt. Liver 1 finger's breadth below costal margin in right nipple line.

Temperature 99°. Pulse 135. Respirations 38.

Digestive System - Tongue clean. Mouth healthy. Vomiting shortly after each feed, sometimes bringing up the whole feed. The food is occasionally kept down for an hour or an hour and a half. The vomit is ejected through the nose and mouth with considerable force.

6. 4. 1903. Stomach tube passed and 2 oz. brown fluid drawn off. Stomach washed out with sterile water.  $1\frac{1}{2}$  oz. Barley water and 2 drachms raw meat juice then introduced into the stomach. It was vomited as soon as the tube was withdrawn.

7. 4. 1903. Child no better. Yesterday half the fluid swallowed was vomited. Still on barley

water and raw meat juice. Child is losing weight. No distinct peristaltic waves seen over the stomach today.

8. 4. 1903. Vomiting persists. Peptonised milk tried alternately with barley water and raw meat juice. Child looks worse. Fontanelle depressed. Lost 4 oz. in weight during the last 2 days. Peristalsis seen this morning. Subcutaneous injection of Saline given this afternoon.

9. 4. 1903. No improvement. Operation decided upon. Subcutaneous injections of Saline given every 4 hours.

11.30 a.m. Mr. Stiles has just operated. Liver was very large. Stomach did not seem much enlarged. Pylorus felt hard - almost cartilaginous - and thickened. Loreta's operation was done. Lombard's tracheal dilator used for stretching Pylorus. Child stood the operation well. Pulse was quite good at the end of operation.

3.30 a.m. Pulse continues good. Subcutaneous injection of Saline given and to be continued every 6 hours. Rectal injection of raw meat juice and water also given and to be continued four hourly.

April 10. 1903. Temperature 102° 8. Pulse 140 - fairly strong. Rectal injections retained. Patient is a good colour. Bowels moved. Stools dark in

colour.

1 p.m. Temperature 106°. Pulse fairly strong though quick. Fontanelle depressed. Child seems fairly well in spite of high temperature.

Evening. Temperature keeps up and pulse still rapid. Child sleeps very well and seems comfortable.

April 11. 1903. Temperature 103°4. Pulse 160. Child had a fairly comfortable night. No vomiting. A little albumen water given by the mouth.

Evening. Temperature 103°. Pulse about 160. Good colour. No sickness or pain.

April 12. 1903. Had a good night. Peptonised milk given last night. No sickness. Temperature keeps up.

April 13. 1903. Child vomited once during the night. Appears comfortable. Temperature 104°6. Pulse 160; not so strong. Abdomen somewhat distended.

Evening. Abdomen more distended. Pulse about 180. Mr. Stiles saw patient and decided to re-open abdomen. A large amount of serum escaped with a quantity of flaked lymph. It seemed to come from the Iliac regions. Some pus found in the region of the Pylorus. The intestines were fairly healthy looking. There was a little lymph on them. There seemed to be no rupture at the Pylorus and the wound on the anterior wall of the stomach seemed all right. The abdominal cavity

was washed out with Sterile lotion. A drainage tube was inserted and the abdominal wound closed except where the drainage tube was. Dressing applied. Patient seemed no worse after the operation. Subcutaneous injections of Saline and nutrient enemata to be continued.

April 14. 1903. Child had a fairly good night. Temperature keeps up. Pulse 170 - feeble. No vomiting.

1 p.m. Child appears in pain. Restless. Some collapse.

Patient died at 11 p.m.

Post-mortem examination Stomach did not seem enlarged, but walls thick. Wound on its anterior wall water-tight. No rupture of Pylorus. Pylorus was long and thick. A large metal bougie could easily be passed through it. General peritonitis present. Other viscera normal.

Case XIV.

C. B. Boy. Born April 3, 1903. He was the third child of the family. The second child had suffered from Intussusception. Patient was a healthy child at birth. He was fed on peptogenic milk. When 5 weeks old he weighed 10 lbs. and there was then no vomiting. On May 20th, 1903, the child began to vomit. Dr. T. Thyne saw him and treated him by various changes in diet. The bowels became constipated. At first the vomiting was not frequent or violent. But soon it became more violent and the food was shot out of the stomach. On June 7th the weight was 9 lbs. The loss of weight and the vomiting continued. Dr. John Thomson saw the patient in consultation with Dr. Thyne on June 11th, 1903. The child was then thin, but fairly vigorous. The Heart and Lungs were normal. Marked peristaltic waves were seen passing across the abdomen. The Pylorus was not felt. Horlick's food was given.

June 15, 1903. From the 11th to the 13th patient was better, but then severe vomiting set in. Patient has lost 6 oz. in a week. The stomach tube was passed and a large amount of curdled milk was removed. Marked peristalsis seen.

June 16. 1903. Mr. Stiles operated this afternoon. A curious callosity was found on the Anterior wall of the stomach. The Pylorus was hard and thick. Loreta's operation was done. Lombard's dilator used. 1 oz. water and 2 drachms raw meat juice passed by a tube into the duodenum at the operation. A subcutaneous injection of Saline was given after the operation.

Evening. Patient seems to be doing well, though. Temperature has gone up to 102°.

June 17. 1903. Temperature keeps up. Child had a restless night. Urine plentiful. Subcutaneous injections of Saline given every 6 hours, and nutrient enemata (with Brandy) given 4 hourly.

June 18. 1903. Patient had a good night. Temperature 98°4 this morning. Nutrient enemata not retained. Food given by the mouth. There was no vomiting. Bowels moved: stools green. Bowel washed out. Child to have small feed 2 hourly consisting of raw meat juice, sugar of milk and a little brandy. Some oedema of face and hands.

June 19. 1903. Patient had a good night. Bowels washed out. 6 drachms of food given hourly. Temperature normal.

June 20. 1903. Peptonised milk given. It

disagreed with child, so returned to raw meat juice. Child fairly comfortable, but looks rather pinched.

June 21. 1903. Child looking pinched and grey. Stomach was a little distended. Motions slimy and green. 2 stitches cutting. Stomach washed out.

Evening. Child looking rather better. Castor-oil given. Subcutaneous injection of 7 oz. of Saline Stitches removed. Temperature 101°. Raw meat juice continued.

June 22. 1903. The subcutaneous injection caused pain. Patient cried most of night, but sleeping this morning. Bowels moved. Stools better.

June 23. 1903. Patient looking pinched. Diarrhoea. There has been considerable vomiting.

June 25. 1903. More vomiting. Child looks pinched.

Evening. Child weaker and collapsed. Hot bottles used. Brandy given by mouth. Nutrient enemata also given.

June 27. 1903. A little vomiting still. Child is wasting.

June 30. 1903. Vomiting had stopped but began again. Stomach washed out. Curdled milk removed. Raw meat juice peptonised milk and brandy given by mouth.

July 3. 1903. Patient been better. Vomiting stopped. Stomach washed out. Some curdled milk removed.

July 5. 1903. Vomited violently last night at 9 p.m. The vomiting continued most of the night. It was forcible.

July 7. 1903. Good day yesterday. Stomach washed out. But patient is worse today and looks pinched. Evidence of Stenosis recurring. Weight 7 lbs. 9 oz.

July 8. 1903. Patient continued to vomit. No doubt the Stenosis had returned. Mr. Stiles saw case and advised gastro-enterostomy. Operation done about 1 p.m. Stomach was found adherent to the Liver. The Pylorus was hard. Posterior gastro-enterostomy done. 1 drachm water and 5 min. Brandy given hourly by the mouth. 2 oz. Saline and 10 min. Brandy given per rectum every 4 hours.

Evening. Temperature 101°. Child pale and anxious-looking but otherwise well. No vomiting. To get 1 drachm raw meat juice,  $\frac{1}{2}$  oz. water and 5 min. Brandy by the mouth hourly. Rectal injections continued.

July 9. 1903. Patient had a good night. Milk tried, but seemed to cause flatulence so stopped. Child very thirsty. Raw meat juice continued.



July 10. 1903. Patient doing very well. Temperature normal. Bowels well moved. Peptonised milk  $\frac{1}{2}$  oz., raw meat juice 1 drachm, and brandy 5 min. given every 2 hours.

July 13. 1903. Progress continues. Patient hungry. No vomiting. Motions good. Sleeping well.

July 16. 1903. Child doing well. Sutures removed on 14th.  $2\frac{1}{2}$  oz. peptonised milk given 2 hourly. Child gained  $2\frac{1}{2}$  oz. in 24 hours.

July 22 1903. Weight 8 lbs.  $7\frac{1}{2}$  oz. Child happy and thriving. No vomiting. Sleeps well. Motions rather green.

July 31. 1903. Weight 8 lbs.  $10\frac{1}{2}$  oz. Bowels all right. The child has gained 2 lbs.  $7\frac{1}{2}$  oz. in 5 weeks.

Sept. 3. 1903. The patient has done very well. No vomiting. Weight 11 lbs. 2 oz. Given 4 oz. peptogenic milk every 3 hours.

Oct. 27. 1903. Child is quite well. Weight 14 lbs. 2 oz.

March 1904. Patient is now a healthy child. No evidence of recurrence of Stenosis.

Case XV.

J. W. Boy First child. Healthy at birth. Weight 7 lbs 10 oz. Normal labour. Father and mother healthy. Continued healthy until 9 days old. Then he began to vomit. This became worse and when a month old he was vomiting repeatedly during the day. Weight then 9 lbs. 3 oz. He was given diluted cow's milk and taken into the country. For a few days he vomited a little each day. He was very hungry. Dr. Thomson saw patient in consultation on May 21st, 1903. He was then 34 days old. Temperature was 102° and there was some vomiting after each meal then. Weight was 8 lbs. Stomach washed out.

May 23. 1903. Vomiting continued. No pain apparently. Constantly having fits. Temperature was 101° 8. He was pale and eyes sunken. Fontanelle depressed. Stomach was felt contracted and hard. Stomach tube passed and 3 oz. brownish yellow fluid withdrawn. Washed out. Egg albumen and water and a little brandy given. No peristaltic waves seen. Saline injection given per rectum.

May 24. 1903. Peristalsis seen last night over the stomach. Child not comfortable. Vomiting continued. Temperature over 100°. Washing out stomach relieved.

May 27. 1903. Child worse. Gastric contents putrid and green in colour.

May 29. 1903. Marked peristalsis seen over stomach. No food seems to be getting through Pylorus. Operation decided upon. Temperature 105°. Loreta's operation done by Mr. Stiles. Pylorus had typical appearance. 2 oz. Saline put into Duodenum. Subcutaneous injection of Saline given later. Temperature nearly normal at night.

May 30. 1903. Child very ill. Not comfortable. Nutrient enema with a little brandy given.

1 p.m. Child collapsed and died now.

Post-mortem examination. Thymus small. Heart and Lungs normal. Liver large and fatty. Kidneys normal. Stomach large -  $4\frac{1}{2}$  in. x 3 in. Pylorus typical of Congenital Stenosis of Pylorus. Bowels empty.

Death from collapse.

Case XVI.

Male child. Born Dec. 18. 1903. Father and mother are alive and healthy. Patient is their only child. The labour was long and difficult. The child was born 2 or 3 weeks prematurely. He was small, but looked quite healthy. Double inguinal hernia was noticed the day after birth. Patient was put on the breast at first, but he at once began to vomit. The vomiting was irregular and not forcible for about 3 weeks. Then child was taken off the breast and put on the bottle. When 3 weeks old patient weighed 6lbs. When he was taken off the breast diluted cow's milk was given. For the first day with this there was no vomiting, but it gradually came on again. Allen & Hanbury's food and Horlick's food were tried, but the vomiting did not improve. It became more frequent and very violent. Sometimes the gastric contents were shot out through the nose with considerable force. After a feed, occasionally the food would be kept in the stomach for an hour and a half and then most of the food taken would be returned. Dr. John Thomson saw patient in consultation with Dr. T. Thyne when the child was 11 weeks old. There was marked emaciation and constipation. Gastric peristalsis was seen passing from left to right. By washing out the

stomach periodically and giving small quantities of food at a time, the vomiting improved for a time, but when the child was 14 weeks old it became worse again. The mother said that the way the food was ejected resembled a lemonade bottle being opened. When patient was 14 weeks old, Mr. Stiles saw him. He saw gastric peristaltic waves but could not feel the Pylorus. He advised operation.

April 1. 1904. Weight 5 lbs. 12 oz. This morning Mr. Stiles did a posterior gastro-enterostomy. Child stood operation well. The operation was done about 11 a.m. During the afternoon there was no vomiting and patient was given some sips of sterilised water with a little brandy in it. Nutrient enemata were given 4 hourly and subcutaneous injections of sterilised normal saline were given 6 hourly (about 2 oz. at a time).

April 2. 1904. Patient had a good night. No vomiting. Slept well. 1 drachm of milk given this morning. There was no vomiting after it and so child now getting 2 drachms of milk hourly.

Evening. Patient seems to be doing well. No collapse or vomiting. 1 oz. of milk to be given hourly.

April 3. 1904. Progress is uninterrupted. Bowels moved. No vomiting. Child seems well.

April 5. 1904. Progress continues. A little milk was returned yesterday, but none since. Child very bright. Weight 5 lbs. 12 oz.

April 8. 1904. Child is making good recovery. No vomiting. 2 oz. diluted cow's milk with a little brandy given every 2 hours.

April 12. 1904. Sutures taken out 2 days ago. Wound quite healed. There has been just a little food returned during the last 2 days. Weight 7 lbs. 2 oz.

April 15. 1904. No more vomiting. Child is looking very well. Weight 7 lbs. 10 oz. Patient has continued to do well. There is no vomiting and weight is increasing.

## Etiology and Pathology.

1. Conditions at birth. The affection occurs in infants apparently healthy at birth. The labours have mostly been normal. It is a striking fact that very few other congenital deformities have been found in infants suffering from Congenital Stenosis of the Pylorus. In Ashby's second case, the patient also had an imperforate anus. Still's first case showed slight webbing of the toes. In Schwyzer's first case there were two areas of narrowing of the large intestine. Cautley's first case had an Inguinal Hernia and at the post-mortem examination a cyst was found in the right Cerebral Hemisphere. At the post-mortem examination on Still's third case right Hydronephrosis with dilatation of the upper part of that Ureter was found. Stern found in his case Congenital dilatation with hypertrophy of the large intestine. Mr. Stiles had a case a few weeks ago (last case given above), and the child also had a double Inguinal Hernia. It should, however, be mentioned that the child was prematurely born. It is worthy of notice that Ballantyne, in all the cases of mal-formed foetuses he has examined, has never met with a Congenital Stenosis of the Pylorus.

2. Onset. The condition may begin to manifest itself soon after birth, but it may be latent for some weeks. De Bruyn Kop's case began to vomit within two days of birth. The vomiting also began on the 2nd day in one of Dr. Thomson's cases. Still had a case which showed no symptoms for 6 weeks.

3. Family History. Most of the cases recorded have been children of healthy parents. Except in the cases of Henschel, only one member of a family has suffered from Congenital Stenosis of the Pylorus. Henschel recorded notes of three members of the same family having suffered from this affection.

4. Sex. Of the cases recorded, where the sex is indicated, there seems to be no preponderance of cases in either sex.

Some authorities (Cautley and Peden) look upon the condition as an excess of development or local gigantism. Thomson maintains that it is a neurosis of development which tends to antagonistic action of the muscular coats of the Pylorus and Stomach, the Pyloric sphincter not relaxing but remaining in a state of spasm or over action, which gives rise to secondary hypertrophy.



Rolleston writes that "it seems reasonable to combine these views so far as to believe that there is some congenital hyperplasia of the Pyloric sphincter, and that spasm supervenes on this and is largely responsible for the symptoms manifested."

R.W.Murray and Flynn point out that the Pyloric Antrum in Mammals represents the gizzard in birds. In some Edentates like the Great Ant Eater and the Armadillo there is a very considerable development of the circular fibres surrounding the Pylorus and they suggest that it is possible that the Congenital hypertrophic Stenosis may be an example of return to the Pyloric type of the Edentates.

Pritchard maintains that the obstruction to the passage of the food through the Pylorus is due to spasm caused by irritation by the gastric contents.

The exact causation of the condition is not quite clear, but one of two things seems certain -

I."That it is a primary developmental hyperplasia,"  
or

III."That it is a secondary hypertrophy resulting from over action."

Cautley says that the hyperplasia may be "due to a simple redundancy of growth. Nature, in her extreme anxiety to provide an efficient sphincter, has over-exerted herself and produced too great a quantity of

muscular tissue." He also adds that "if the condition be due to Pyloric spasm, the affection should be amenable to treatment short of operation." Thomson replies as follows:- "I doubt very much, however, whether there exists in human pathology, or, indeed, in Nature, a single instance of a localised true hypertrophy of muscle, being certainly or even probably, primary in character. By true hypertrophy of any muscle, I mean such hypertrophy as results in increased power to perform its normal function." He excludes Pregnancy. There is no doubt that there is some hypertrophy at the Pylorus at birth. In a case of Ashby's, where death occurred on the fifth day, he reports that "there was marked hypertrophy, although it was not so marked as in the cases where death had been later." Simonshon has observed the same condition in a child 36 hours old at death. In addition to this hypertrophy present at birth, it is generally agreed that it goes on increasing after birth. Thomson maintains that it is due to "a functional disorder of the nerves of the Stomach and Pylorus leading to ill-co-ordinated and therefore antagonistic action of their muscular arrangements." He also says that "although it is a matter of dispute how much, if at all, the liquor amnii swallowed by the foetus contributes to its nourishment, it is generally admitted that a large amount of fluid passes normally

through the stomach of the foetus during the later months of pregnancy, and it is reasonable to suppose that its passage occasions a considerable degree of peristaltic action. Excessive or irregular action of the pyloric muscle would inevitably cause some amount of obstruction to the outflow of fluid from the stomach, and this would easily account for the hypertrophy of the muscular tissue in the stomach wall." Against Pritchard's contention that the spasm is caused by irritation by the gastric contents, it must be pointed out that the same effect is caused by all kinds of food and that the quantity rather than the quality of the food seems to be the greater disturbing influence.

The general consensus of opinion is in favour of Dr. Thomson's view.

### Morbid Anatomy.

Oesophagus. Sometimes a little dilatation of the lower part of the oesophagus is present (seen in photograph); and there may be some hypertrophy as well.

### Stomach and Pylorus.

In most of the stomachs from cases of Congenital Stenosis of the Pylorus there has been some dilatation observed, varying in degree in different cases. I was present at the post mortem examination on one of Dr. Thomson's cases where the dilatation was very marked. A photograph was taken and is shown below. In some cases no dilatation of the stomach is made out, and this may be due to some muscular contraction present in the organ at the time of death, or to the fact that no dilatation had taken place. In most of the cases considerable hypertrophy of the stomach wall has been observed. This is most marked at the Pyloric end and it gradually diminishes towards the Fundus. Sometimes indeed, the fundus is rather thinner than normal. The

Pylorus is enlarged and feels hard (almost cartilaginous). It has been described as "sausage shaped", and "like an almost solid cylinder". On opening into the stomach the thickening of its wall can easily be made out, and with the naked eye it can be seen to be due to muscular hypertrophy. The mucous membrane presents no striking appearance, but it may be a little congested. There is no marked catarrh or ulceration present. The mucous membrane of the stomach is thrown into longitudinal folds near the Pylorus - radiating from the Pyloric orifice. There is usually a fold corresponding to the greater curvature of the stomach and this is the most prominent. The lumen of the Pylorus varies in size. Sometimes only a fine probe can be passed through, but in other cases a rod 3.5m.m. in diameter was easily passed through ~~each~~. It is worthy of notice here that in all the cases in which Loreta's operation has been performed, there is no record of any difficulty in passing the dilating forceps or a fairly large metal bougie through the lumen of the Pylorus. The degree of thickening at the Pylorus does not seem to bear any direct relationship to the size of the Pyloric orifice. The thickened Pylorus gradually becomes thinner as it is traced into the stomach, but on looking at it from the Duodenum it is seen to terminate abruptly and has an Os Uteri appearance.

When opened up the Pylorus is seen to be considerably thickened. As Still has pointed out it is hard to estimate the normal limits of thickness of the Pylorus in Infants and he has made a valuable contribution to the subject by examining minutely the Pylorus of several infants where death has taken place from other causes than Congenital Stenosis of the Pylorus. From his observations there can be no doubt as to the increase in the thickness of the Pylorus in this affection, though it varies in degree.

Microscopically - The thickening in the stomach wall is found to be due to hypertrophy of its muscular coat. The Pylorus, on microscopic examination, shows no change in the serous layer. There is marked hypertrophy of the muscular coat. With the exception of Finkelstein's case the hypertrophy has been almost entirely confined to the circular fibres. Finkelstein reported that in his case the hypertrophy was of the longitudinal fibres. Cantley suggests that he mistook the circular for the longitudinal fibres by the section not being cut exactly in the longitudinal direction - a mistake which might certainly occur. In some cases an increase in the thickness of the submucosa has been noted by Still, Thomson and others, but this is not the rule. The mucosa is usually normal in thickness, though it may be a little thinner than normal owing to pressure.

The cases given above will give a good idea of the thickening of the muscular layer. The Duodenum and the rest of the Intestinal Canal are usually quite normal. There may be a few congested, slightly enlarged glands in connection with the upper part of the alimentary tract. The other organs are quite normal. There is always marked general emaciation.

### Clinical Features.

The infant is almost invariably healthy-looking and fat at birth. The symptoms do not usually begin to develop for about a week, though in De Bruyn Kop's case they began at once. The first manifestation is vomiting. At first it is not violent and may be infrequent. It may only be present after a big feed. But it soon becomes more frequent and increases in severity. It persists in spite of changes in the kind of food, and even when a small amount is given at a time. In the latter case the food gradually accumulates in the stomach and ultimately most of it is returned. The hypertrophy of the stomach increases, and after a few weeks the food is "shot out" of the stomach with considerable force. The usual remedies used to allay vomiting in Infants are of no avail. Sometimes washing out the stomach produces a slight improvement, but it is very temporary. When the vomiting has become very violent, the little patient seems to suffer pain. The vomited matter consists of the food taken in. This is usually milk, and if returned at once it is unchanged. If it has been retained in the stomach for some time it undergoes acid decomposition. There is rarely any trace of blood in the vomit. Simonsohn reported that blood



had been present in the vomit from his case.

There is no bile in the vomit.

The bowels are constipated. The stools show the presence of bile and there is usually some mucus when the condition has lasted for some weeks. The urine is scanty in amount. The eyes become sunken. The temperature soon becomes constantly subnormal and the extremities are cold. The Fontanelle becomes depressed. Emaciation is inevitable with the symptoms. The patient loses weight, becomes weaker and soon passes into a state of marasmus.

As the patient becomes thinner and hypertrophy and dilatation of the stomach develop, an examination of the abdomen reveals marked physical signs. The epigastric region may be seen bulging and the outline of the stomach may be seen standing out. If the stomach wall contracts, the fulness in the epigastric region disappears. Peristaltic waves are not infrequently seen passing along the abdominal wall from left to right in the region of the stomach. These are not usually seen until the vomiting has persisted for some weeks. In one of Dr. John Thomson's cases I saw peristaltic waves when the patient was three weeks old. The abdominal wall is lax and there is no difficulty in palpating the abdomen. The thickened Pylorus can sometimes be felt, though usually not for a few weeks after

birth. It is felt as a rounded or elongated tumour below the liver, a little to the right of the middle line. An anaesthetic may be necessary to make it out. When the stomach is contracted it may be made out almost like a tumour. The size of the stomach can be made out as follows:- Pass a rubber catheter or tube into the stomach and sometimes, when the abdominal wall is lax and thin, it can be felt passing along the greater curvature to the Pylorus. The stomach can then be filled with fluid or inflated with air, and its outline distinctly made out. When the stomach tube is passed periodically, it shows that when the gastric contents ~~is~~<sup>are</sup> not vomited ~~they~~<sup>are</sup> often retained for a long time and accumulate. Thomson has pointed out that this "would seem to indicate not only that the stomach is impassable at these times", "but that there is something interfering with the absorption of fluids by the stomach."

When the vomiting is once thoroughly established, death usually takes place before many weeks. If the vomiting is not very severe and emaciation is slow, the child may last some months.

### Diagnosis.

The chief points to be kept in mind in making a diagnosis in cases with similar symptoms to those described above are:- Firstly, the child is healthy at birth and without any apparent cause begins to vomit; secondly, the vomiting continues in spite of changes in diet, and a large proportion of the food is returned; thirdly, there is usually no blood or bile in the vomit; fourthly, there is progressive emaciation; fifthly, there are visible gastric peristaltic movements; sixthly, there is a tumour corresponding to the Pylorus. The difficult cases to diagnose are those in which no gastric peristaltic waves can be seen and no tumour in the region of the Pylorus felt. Many children, especially those fed artificially and who have extreme dyspeptic troubles, vomit a good deal and lose weight, but they improve when we change their diet and get a good nurse. By the success or failure of such measures a diagnosis can usually be made with a fair amount of certainty.

### Prognosis.

Well marked cases are almost invariably fatal unless operative treatment is resorted to, hence the choice of this subject as a surgical thesis. It is true that one or two typical cases have recovered, but they are exceptional. Batten recorded a case of his, which, with careful treatment and without operation, recovered completely. The child died some months later from Pneumonia and at the Post-mortem examination a thickened Pylorus with hypertrophied muscular walls was found. Some cases have been reported as cases of Congenital Stenosis of the Pylorus in which the symptoms were not typical and it is doubtful if the diagnosis were correct. The duration of life depends upon the amount of food which passes through the Pylorus. Cautley considers that four months is the average duration of life of infants suffering from this affection. Other observers put it at less. Undoubtedly some cases, in which the obstruction is not marked or only intermittent, may live for many months.

### Treatment.

If a diagnosis of Congenital Stenosis of the Pylorus can be made with a considerable degree of certainty, there can be no doubt as to the advisability of operative treatment. It is the surest way of saving the patient, and although cure may occasionally take place without operation it is not certain that it will be permanent. But in cases where no gastric peristalsis is seen and no tumour in the region of the Pylorus felt and the diagnosis cannot be made with any degree of certainty, palliative treatment should be tried. The only drug which seems to be of any value in severe vomiting in children is opium, given in small doses. Ashby suggests that small doses of Calomel may be of value. The most important thing is that the child should have good nursing. Everything must be clean and the food given in a small quantity at a time. There is less likelihood of the child vomiting when fed by a spoon or pipette than when put on the bottle or the breast. If the vomiting persists, the patient should be fed by a rubber tube passed into the stomach, preferably through the nose. If the vomiting becomes less marked or stopped, the amount given at each feed can be increased. A choice in food can be made from the following:- Barley water, egg albumen, raw meat juice, milk and such preparations as Allen & Hanbury's malted food. Whatever

form of food is given, it should not be in a too concentrated form. Sometimes washing out the stomach periodically is useful. Subcutaneous injections of sterilised salt solution ( $\frac{3}{4}\%$ ) may with advantage be given night and morning. About 2 oz. is usually a sufficiently large amount for each injection. Nutrient enemata should be given about every 4 or 6 hours if the vomiting is severe. About 1 oz. or  $1\frac{1}{2}$  oz. should be given in each enema. It is a good practice to wash out the bowel daily, and if the enemata tend to set up any irritation, an injection of starch with 1 min. of Laudanum will be found helpful.

If, in spite of palliative treatment, the vomiting persists and the emaciation continues, it is wise to lose no time in having recourse to an operation.

Having decided upon operative treatment, the question arises which is the best operation. There are at present three operations which are done by different surgeons for the relief of Congenital Stenosis of the Pylorus, viz. Stretching the Pylorus (Loreta's operation), Gastro-enterostomy, and Pyloroplasty. Pylorotomy was done in one case by Mr. Stiles, but he now, with other surgeons, regards the operation as too severe for infants in the weakened state in which they mostly are when an operation is to be done for Congenital Stenosis of the Pylorus. It will probably

be best to first describe the three operations mentioned above and then to consider their relative merits. In describing the operations it will be assumed that there is no defect in the technique of the operations. The greatest care must be taken in preparing the skin, and in keeping the field of operation aseptic. Inside the abdominal cavity sterile swabs, wrung out in sterilised salt solution at the body temperature should be used. Before proceeding to operate, it is wise to wash out the stomach. Chloroform is generally the best anaesthetic to use.

1. Stretching the Pylorus. Loreta's Operation.

The best instrument for the operation is Lombard's tracheal dilator. It is three-pronged and the blades are not too narrow, so that they do not cut the tissues. Burghard has introduced a special pair of forceps for stretching the Pylorus. It is three-pronged, but the blades are rather narrow and more likely to cut the tissues than Lombard's dilating forceps. Both instruments are made so that the force employed can be regulated.

An incision about  $1\frac{1}{2}$  in. long is made in the middle line of the abdomen, and situated about midway between the umbilicus and the ensiform cartilage. The surgeon then grasps hold of the stomach and draws it outside the abdominal wound (Plate **XIV** Figs. 1 & 2.) Around it sterilised moistened swabs are packed to

protect the general peritoneal cavity, should there be any escape of gastric contents when the incision is made in the stomach. The anterior wall of the stomach is then raised and folded a little so that it can be transfixed. (Plate XV Fig 1. ). This fold is made so that when it is transfixed the incision lies in the long axis of the stomach, about midway between the Greater and Lesser curvatures and its right extremity about 1 in. from the Pylorus. The incision ought to be about  $\frac{1}{2}$  in. long. The assistant holds the stomach so that the wound gapes and there is no escape of gastric contents. The apex of the dilating forceps is then passed through the gaping wound, (Plate XV Fig. 2. ), and through the Pyloric Sphincter. The forceps are then slowly opened and the Pylorus gradually stretched. Should there be any difficulty in passing the blades of the forceps through the Pyloric Sphincter, metal bougies should first be used, beginning with one small enough and going on until a No. 18 has been <sup>a</sup>passed. If there has been any rupture at the Pylorus, it must be closed by silk sutures and a layer of Lembert sutures used to invert the edges. The opening in the stomach is next closed by a layer of silk sutures passing through the whole thickness of the stomach wall. The edges are then inverted by a layer of Lembert sutures. The stomach



is returned to the abdominal cavity and the abdominal incision closed. The best method of closing it is by silk worm gut sutures passed through the whole thickness of the abdominal wall, with a few fine horse hair sutures between them for the skin edges. If there should later be any distention of the abdomen, the silk worm gut sutures are apt to cut the skin. A good method of getting over this disadvantage is by passing one end of each suture through a small piece of fine rubber tube before the suture is tied. A dressing of sterilised gauze is applied.

2. Gastro-enterostomy. The Posterior is the better operation. Most surgeons consider that it drains the stomach better, and the anastomosis is supported by the posterior wall of the abdomen. The mesentery may also be a little short for the anterior operation. This may not be apparent at the operation, because the Stomach and Transverse Colon are empty, but when they are full there may be a little dragging on the anastomosis. The best position on the Jejunum to make the anastomosis is about 8 in. from the Duodeno-jejunal flexure. The site chosen on the stomach for the anastomosis should be nearer the Pylorus than the Fundus and not far from the Greater curvature though sufficiently far to avoid the larger branches

of the Gastro-epiploic vessels. The Isoperistaltic is better than the Antiperistaltic operation because a "vicious circle" is less likely to develop.

An incision about 2 in. long is made in the middle line of the abdomen, having its lower extremity at about the level of the umbilicus. The Stomach and Transverse Colon are drawn outside the abdominal wound, thrown upwards, and grasped by the assistant's right hand, so that his fingers press the posterior wall of the stomach against the Transverse Meso-colon. An opening is then made in the Transverse Meso-colon opposite that part of the posterior wall of the stomach where the anastomosis is to be made. This opening should be made some distance from the attachment of the Meso-colon to the Colon and between the larger branches of the Colic vessels, in this way avoiding haemorrhage as much as possible. The edges of the opening are fixed to the posterior wall of the stomach by about five catgut sutures. The surgeon then finds the part of the Jejunum he wishes, and the best way to find it is by following the Duodenum to the Duodeno-jejunal flexure, and then the Jejunum will be made out passing from there. The openings in the Stomach and Jejunum are to be made in their longitudinal axes and in the latter it should be situated opposite the mesenteric attachment. The Jejunum is laid in apposition

with the posterior wall of the stomach. Two fixation silk sutures are first used, situated about  $1\frac{1}{4}$  in. apart from each other. Then between these a continuous Lembert suture of fine silk is used to unite the peritoneum of the Jejunum to that of the Stomach. Before opening into the Stomach and Jejunum the general peritoneal cavity should be protected by packing moist sterilised swabs at the body temperature, around the site of the anastomosis. Incisions about 1 in. long are then made in the Stomach and Jejunum about  $\frac{1}{8}$  in. from the line of the Lembert suture. Probably the easiest way to do this is by transfixing the parts by means of a tenotomy knife. The incision in the Stomach should be a little longer than that in the Jejunum, because the Stomach is to some extent held on the stretch by the assistant. The edges of the wound adjacent to the Lembert suture should then be united by a continuous silk suture passing through all layers. The free edges of the wound should then be united, first by a continuous silk suture passing through all layers, and then by a continuous Lembert suture to invert the edges. The ends of these sutures are tied to the fixation sutures at the extremities of the anastomosis. A careful examination of the anastomosis should then be made, to see if there are any weak places where there might be an escape.

Additional Lembert sutures should be put in such positions. When the surgeon is satisfied with the anastomosis the viscera are replaced and the peritoneal cavity closed in the manner described above under Loreta's operation. A dressing of dry sterilised gauze is applied to the wound.

3. Pyloroplasty. An incision about 2 in. long is made in the middle line of the abdomen, midway between the umbilicus and the ensiform cartilage. If the stomach presents it should be pushed to the left and the Pylorus drawn to the abdominal wound. There may be some difficulty in getting the Pylorus out, but it should be drawn as far into, or outside, the wound as possible. Moist sterilised swabs are packed round it so as to protect the general peritoneal cavity. A longitudinal incision is then made through the Pylorus. It should be about 1 in. long and extend through the Duodenal end of the Sphincter. The edges of the opening are then drawn apart and the extremities of the incision sutured in apposition by a silk suture passed through all layers. The longitudinal incision is thus converted into a transverse slit. The edges are united by interrupted silk sutures passed through all layers. Then a row of Lembert sutures is used to invert the edges. The Pylorus

is then replaced and the abdominal incision closed as described under Loreta's operation. A dry sterilised dressing is applied to the wound.

Before closing the abdominal incision after any one of the above operations some sterilised normal saline solution at the body temperature can be put into the peritoneal cavity.

After Treatment. The child is put back to bed and kept thoroughly warm with hot blankets and hot bottles. Care must be taken that the latter are not allowed to burn the skin. A nutrient enema containing a little stimulant should be given after the operation. It should not exceed  $1\frac{1}{2}$  oz. in amount. Nutrient enemata can be given every 4 hours as long as it is considered necessary. Subcutaneous injections of sterilised normal saline are often very beneficial in the treatment of these cases after operation, and they should be continued until the patient is able to take a fair amount of nutriment by the mouth. About 2 to 4 oz. should be injected every 6 hours. It is generally considered that the feeding by the mouth can be tried earlier after Gastro-enterostomy than after either of the other operations. After Gastro-enterostomy if the patient shows no tendency to vomit, sips of sterilised water can be given in 12 hours. If

there is a tendency to vomit nothing should be given for 24 hours, and then only sips of sterilised water at first. If there is no vomiting, diluted egg albumen should be given. At first only 1 drachm should be given each hour. If this has been done for about 12 hours with a satisfactory result, the amount should be doubled. By the end of the second day after operation the child should get 2 drachms of fluid every hour. Then  $\frac{1}{2}$  oz. of diluted raw meat juice or peptonised milk can be given instead of egg albumen. If Loreta's operation or Pyloroplasty has been done it is well to wait 24 hours before giving anything by the mouth, but after then the above treatment should be adopted. There is a growing tendency to start feeding by the mouth sooner after the operation, and as a rule the absence or presence of vomiting may be taken as a safe guide. But the amount given at a time should not be large. In the last case Mr. Stiles operated upon (Gastro-enterostomy) the patient got  $\frac{1}{2}$  oz. of peptonised milk hourly 24 hours after the operation and with no bad effect. On the fourth day after operation 1 oz. or  $1\frac{1}{2}$  oz. of peptonised milk can be given every 2 hours according to the age of the patient. If it is considered advisable, a little stimulant can also be given by the mouth during the first few days. The amount of food given should be

gradually increased until about the 12th day the patient is getting a little less than the usual amount of food given to a child of the same age. Should there be any recurrence of vomiting the amount should be decreased again and if necessary a change made in the kind of food. The subcutaneous injections of saline and the nutrient enemata should be continued as long as the patient seems to be in need of them. Should the bowel become irritated by the enemata, it should be washed out and an injection of starch with 1 min. of Tinct. Opii given before the nutrient enema. It is wise to look at the abdominal wound about the seventh day, but if the stitches are not cutting they should be left in until the tenth day. After then a pad of gauze should be put on the cicatrix and a binder applied, though not so tightly as to cause discomfort.

The greatest care must be taken as to the amount, regularity and quality of the food given to the patient for some months after operation.

### Choice of Operation.

I have been able to collect records of 37 operations done on 36 patients suffering from Congenital Stenosis of the Pylorus. Owing to the great kindness of Mr. Burghard and Mr. Stiles I am enabled to give the results of the cases they have operated upon.

A Table showing the results of the cases operated upon is given below. The case reported by Hausy as Congenital Stenosis of the Pylorus in a boy, aged 11 years, and on whom he successfully performed gastro-enterostomy is not given in the Table because it is not generally regarded as a true case of Congenital Stenosis of the Pylorus. The same applies to Rosenheim's case, a child of 6 years of age, on whom Sonnenburg did a Pyloroplasty with recovery.

There have been 17 cases reported as having had Loreta's operation done and of these 10 recovered, 6 died, and 1 recurred (Case XIV above). Mr. Burghard has had 6 cases which have all recovered after Loreta's operation. He writes to me as follows:- "I prefer Loreta's operation because it is simple, rapid and - in my experience - efficient. Should it be necessary to find a substitute for it I should certainly try gastro-enterostomy, but I cannot help thinking that in these young children there must be a distinct death-rate to the operation." At the time of operation Mr.



Burghard feeds the patient. "A meal is put into the Duodenum by a Catheter before the stomach incision is closed. The Pylorus is pushed up to prevent it escaping while the stomach is being sutured." After doing Loreta's operation on 8 cases Mr. Stiles has given the operation up in favour of Gastro-enterostomy. Mr. Stiles found it difficult to estimate the amount of atretching necessary. In one of his cases the symptoms recurred(Case XIV) and a gastro-enterostomy was done with a successful result. In another case (Case XIII) there was a rupture of the Pylorus posteriorly which set up Peritonitis. No rupture could be seen at the operation. Of Loreta's operation it may therefore be said that if the surgeon can be sure that he will hit the happy medium of stretching the Pylorus sufficiently without causing any rupture posteriorly where it cannot be seen, then it is a very satisfactory operation.

Gastro-enterostomy has been performed in 13 cases, of which 7 recovered and 6 died. In one case a Murphy's button was used and was the cause of death. A Murphy's button should not be used because it may either cause obstruction or fall back into the stomach and remain there. Those who object to gastro-enterostomy do so on the ground that it takes longer to do than either Pyloroplasty or Loreta's operation, and that

it necessitates a greater exposure of viscera, and consequently a greater degree of shock is associated with it. Mr. Stiles considers that the disadvantage of a rather greater exposure to shock is more than counterbalanced by the certainty that there will be no recurrence of symptoms after it, which cannot be said of Loreta's operation.

Weill and Pehu say that gastro-enterostomy is essentially the operation to be preferred.

Mayo Robson and Moynihan write that "the operation of choice in all such cases is clearly gastro-enterostomy."

Pyloroplasty has been performed in 6 cases of Congenital Stenosis of the Pylorus, of which 5 recovered and 1 died. Mr. Dent has performed the operation 4 times with successful results. He prefers it to Loreta's operation because the exact amount of injury done to the parts is known, and it can be done at least as quickly. He objects to gastro-enterostomy for the following reasons:-

1. "That it necessitates a considerable exposure of the abdominal contents."
2. "That the operation must be more protracted than either dilatation of the Pylorus or Pyloroplasty."
3. "That the incision has to be prolonged further down towards the umbilicus and consequently there is greater likelihood of a ventral hernia developing."

J. Murray has done Pyloroplasty once in a case of Congenital Stenosis of the Pylorus and he says that nothing would induce him to do it again. He considers the operation exceedingly difficult on account of the Pylorus being so thick. He experienced the greatest difficulty in getting the ends of the incision in apposition.

Monnier considers that Pyloroplasty should not be done on account of the thickness of the Pyloric wall.

Mayo Robson and Moynihan say: "Pyloroplasty, on account of the great thickness of the Pylorus and its rigidity in the whole circumference, is inapplicable."

Mr. Burghard writes of his cases, "I have never seen a Pylorus that I should have cared to do a Pyloroplasty on."

Mr. Stiles, judging from the cases he has operated on, considers Pyloroplasty very difficult.

Of these three operations it will be seen from the above that the choice really lies between Loreta's operation and Gastro-enterostomy. In favour of the former is the fact that there is probably less shock associated with it, and in favour of the latter it must be admitted that the result is more certain.

Table showing Cases Operated upon.

Age at Operation	Date of Operation	Operation	Surgeon	Result	Remarks
				Death	
				Recovery	
6½ weeks.	Mar. 7. 1900	Pylorotomy & Ant. gastro-duodenostomy	H. J. Stiles	1	Death from shock 8 hours after operation.
5 weeks, 2 days	Mar. 14. 1900	Gastro-enterostomy. (Post)	"	1	Death from haemorrhage 13 hours after operation.
3½ weeks	Mar. 20. 1902	Loreta's Operation.	"	1	Death from Peritonitis due to rupture of Pylorus.
5½ weeks	May 10. 1902	Do.	"	1	Recovery complete.
9 weeks	July 28. 1902	Do.	"	1	Probably a recurrence of Stenosis.
11 weeks	Aug. 13. 1902	Do.	"	1	Child now healthy.
5½ weeks	Nov. 15. 1902	Do.	"	1	Death from collapse 2 hrs. after operation.
4 weeks, 2 days	April 9. 1903	Do.	"	1	Death from Peritonitis.
6 weeks	May 29. 1903	Do.	"	1	Death from collapse 24 hours after operation.
10½ weeks	June 16. 1903	Do.	"	1	Recurrence of stenosis so gastro-enterostomy done.
14 weeks	July 8. 1903	Gastro-enterostomy (Post)	"	1	Complete recovery.
14 weeks	April 1. 1904	Gastro-enterostomy (Post)	"	1	Child doing well.
7 weeks	.	Loreta's Operation.	Burghard	1	
5 weeks	.	Do.	"	1	Complete recovery. Child died since from Bronchitis.
8 weeks	.	Do.	"	1	
11 weeks	.	Do.	"	1	
26 weeks	.	Do.	"	1	
8 weeks	Oct. 1902	Do.	"	1	Still's case.
7 weeks	June 3. 1897	Gastro-enterostomy (Anter.)	Stern	1	Death from shock
6 weeks	.	Gastro-enterostomy (Murphy's button).	Meyer	1	Metzler's case Death from Obstruction.
	.	Gastro-enterostomy	"	1	Alder's case. Died a few hours after operation.
10 weeks	July 25. 1898	Gastro-enterostomy (Poster)	Lobker	1	Child healthy 2 years after operation.
7 weeks	June 6. 1899	Do.	"	1	Death from Peritonitis.
6 weeks	Jan. 28. 1899	Gastro-enterostomy (Ant.)	Fritzche	1	Monnier's case
8 weeks	Oct. 27. 1899	Do.	Kehr	1	Abel's case.
8 weeks	April 1. 1900	Gastro-enterostomy (Poster)	"	1	
8 weeks	July 25. 1899	Loreta's Operation.	Nicoll	1	
8 weeks	Dec. 18. 1900	Do.	Schmidt	1	
10 weeks	Oct. 18 1900	Pyloroplasty	Braun	1	
5½ weeks	Aug. 19. 1901	Gastro-enterostomy	Trantenroth	1	
4½ months	Oct. 1902	Pyloroplasty	J. Murray	1	Guthrie's case
6 weeks	Aug. 2 1903	Loreta's Operation	G. L. Chiene	1	Death from shock a few hours after operation

Table (Contd.)

No.	Age at Operation	Date of Operation	Operation	Surgeon	Result		Remarks
					Death	Recovery	
32	3 months		Gastro-enterostomy.	C.W. Towns- C. end. w. g. and end			Death in 23 hours after operation
33	8 weeks	June 10. 1902	Pyloroplasty	Dent		1	Cautley's case
34	6 weeks	Aug. 19 1902	Do.	..		1	.. ..
35	.	.	Do.	..		1	.. ..
36	.	.	Do.	..		1	.. ..

DESCRIPTION of the PLATES.

Plate I. Microphotograph (x200 diameters).  
Longitudinal section of muscular coat of Pylorus,  
showing circular fibres cut transversely (b) and long-  
itudinal fibres (a) cut longitudinally. (From Case I.)

Plate II. Drawing showing Stomach, Pylorus and  
Duodenum opened up. The longitudinal folds of mucous  
membrane are seen and the muscular hypertrophy of the  
Pylorus is evident. (From Case IV.)

Plate III. Figs. 1 & 2. Show Pylorus resected  
from Case V.

In Fig. 1. Pylorus is exposed from the gastric  
side.

In Fig. 2. Pylorus is exposed from the duodenal  
side.

Fig. 3. Stomach opened from the front showing  
anastomosis after gastro-enterostomy. (From Case VI.)

Plate IV. Microphotograph(x 8 diameters).  
Transverse section through Pylorus near Stomach.  
Marked hypertrophy of muscular coat is seen. (From  
Case V.)

- a. Longitudinal muscular fibres.
- b. Circular                   ,,           ,,
- c. Submucous coat ...           ..
- d. Mucous Membrane.

DESCRIPTION of the PLATES. ( Ctd.)

Plate V. Microphotograph ( x 35 diameters).

Same as Plate IV.

Plate VI. Photograph. Shows Pylorus opened longitudinally. (From Case VI.)

Plate VII. Microphotograph ( x 8 diameters). Longitudinal section at gastro-duodenal junction - stomach to left and duodenum to right. (Case X).

- a. Longitudinal muscular fibres cut longitudinally.
- b. Circular           ,,           ,,           ,, transversely.
- c. Submucous coat
- d. Mucous Membrane.

Plate VIII. Microphotograph ( x 35 diameters). Transverse section at Pylorus. (Case X.)

- a. Longitudinal muscular fibres cut transversely.
- b. Circular           ,,           ,,           ,, longitudinally.
- c. Submucous coat.
- d. Mucous Membrane.

Plate IX. Microphotograph (250 diameters). Same section as Plate VIII showing muscular coats.

- a. Longitudinal muscular fibres.
- b. Circular           ,,           ,,

DESCRIPTION of the PLATES. (Ctd.)

Plate X. Microphotograph ( x 8 diameters).  
Longitudinal section through stomach near gastro-pyloric junction. (Case XI). Letters same as Plate VIII.

Plate XI. Microphotograph ( x 35 diameters).  
Transverse section through Pylorus (From Case XI).  
a. Longitudinal muscular fibres cut transversely.  
b. Circular           ,,           ,,           ,, longitudinally.  
c. Submucous coat.  
d. Mucous Membrane.

Plate XII. Fig. 1. Photograph showing marked dilatation of the stomach secondary to Congenital Stenosis of the Pylorus. (From Case XII).

Fig. 2. Same, but also shows some dilatation at the lower extremity of the OEsophagus.

Plate XIII. Photograph showing gastric peristalsis. (From Case XIII).

Plates XIV & XV. Photographs showing the steps in Loreta's operation.



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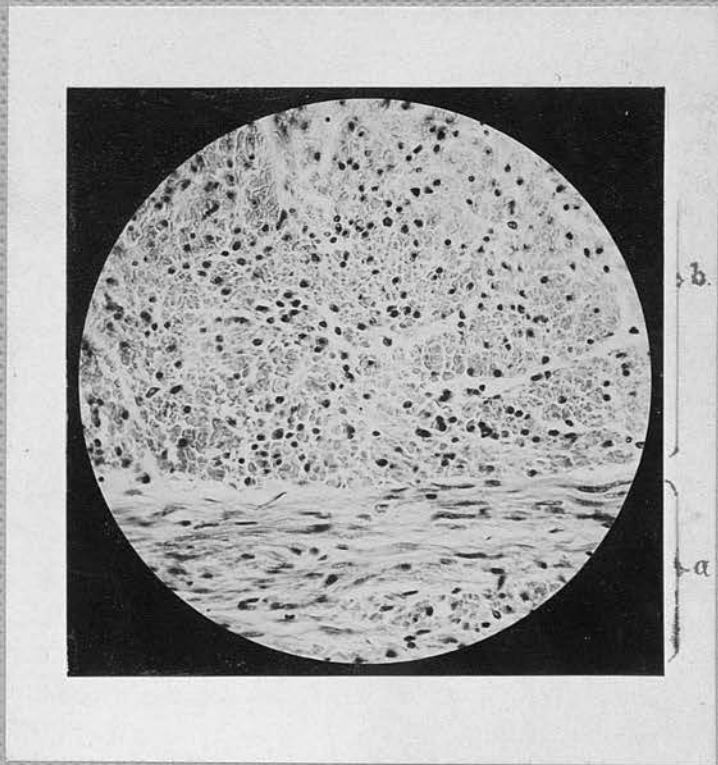
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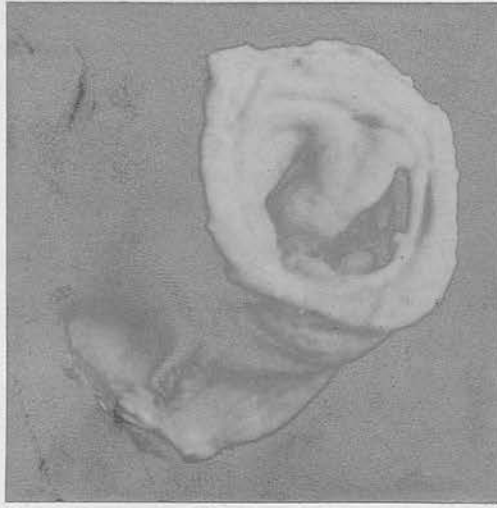


Fig 1.

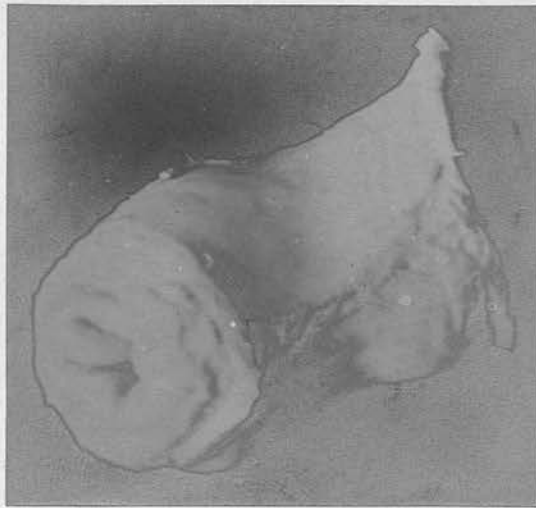


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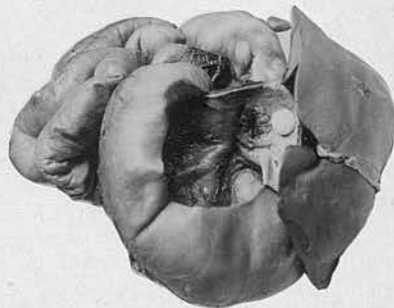
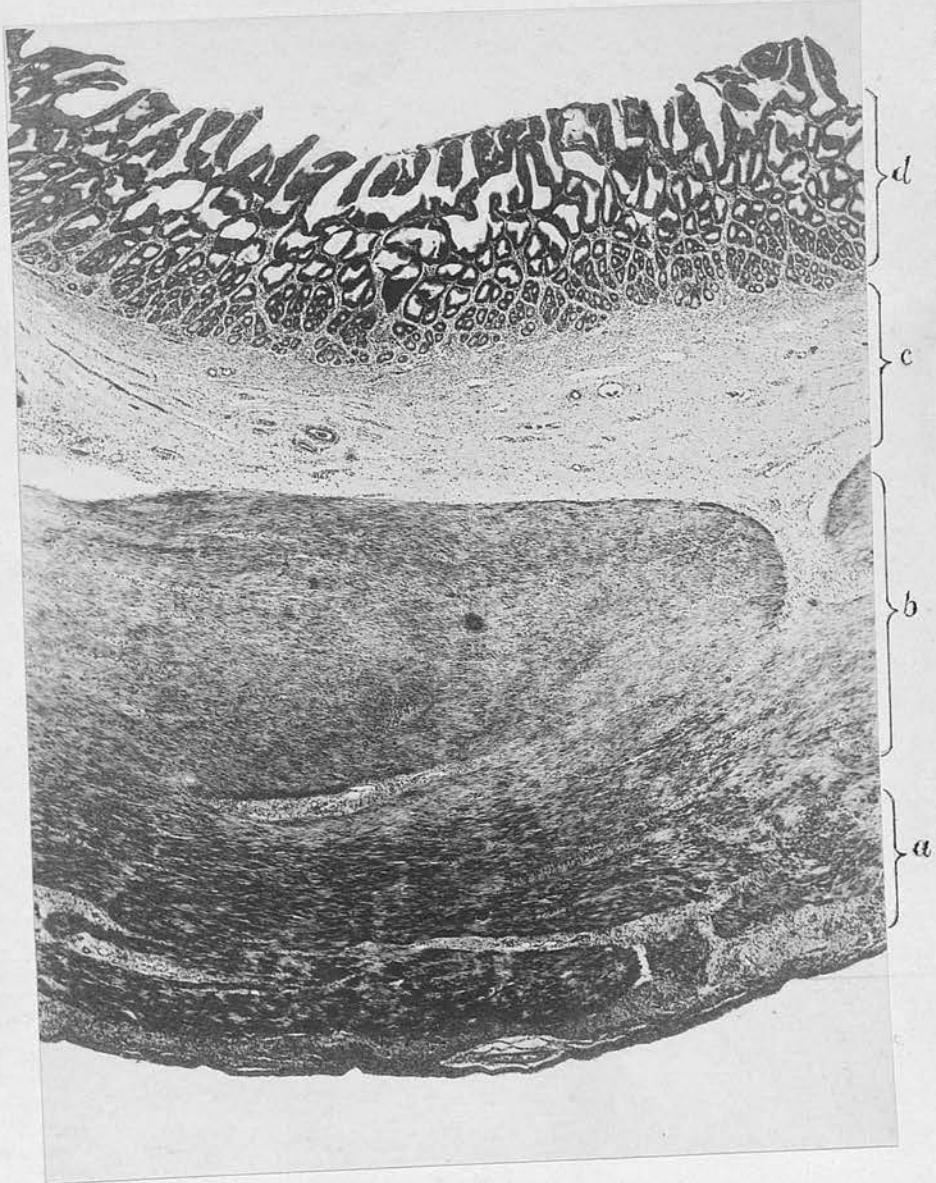


Fig 3.



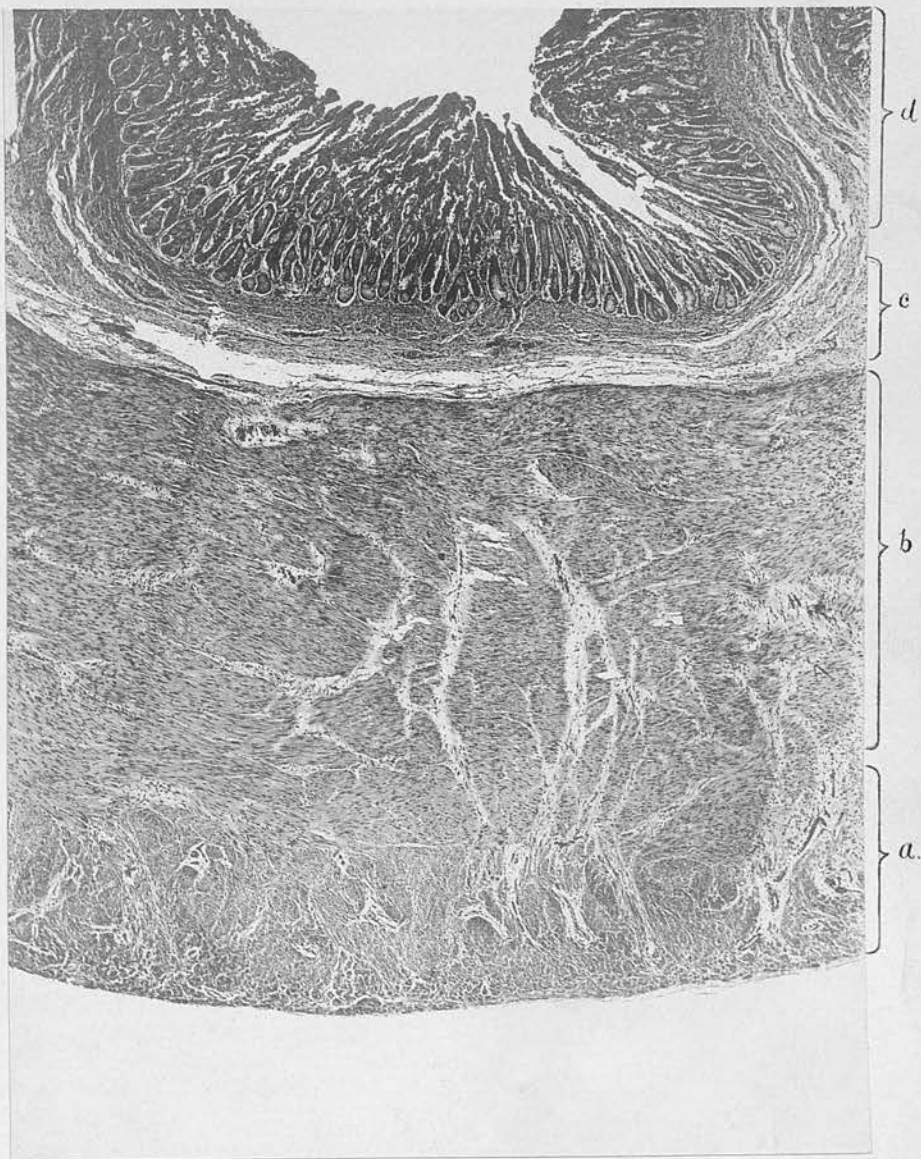


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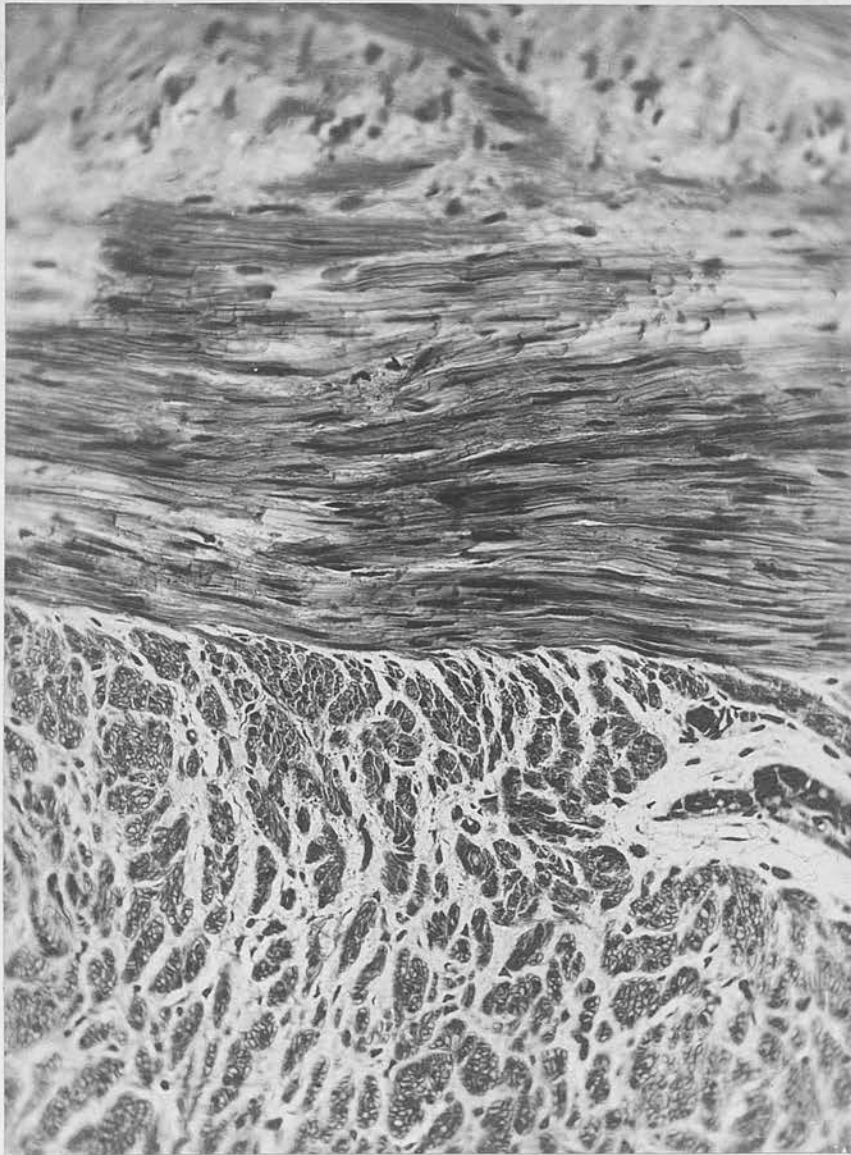




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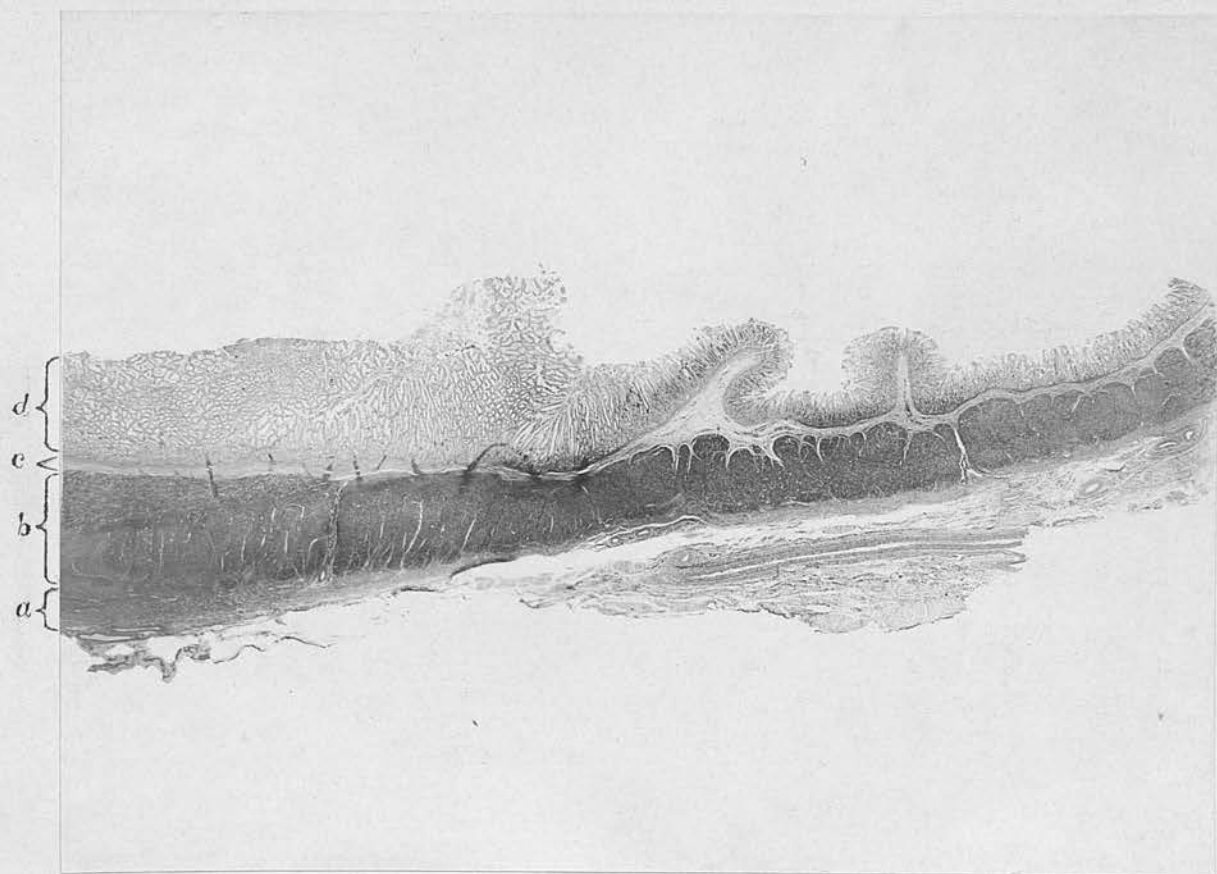


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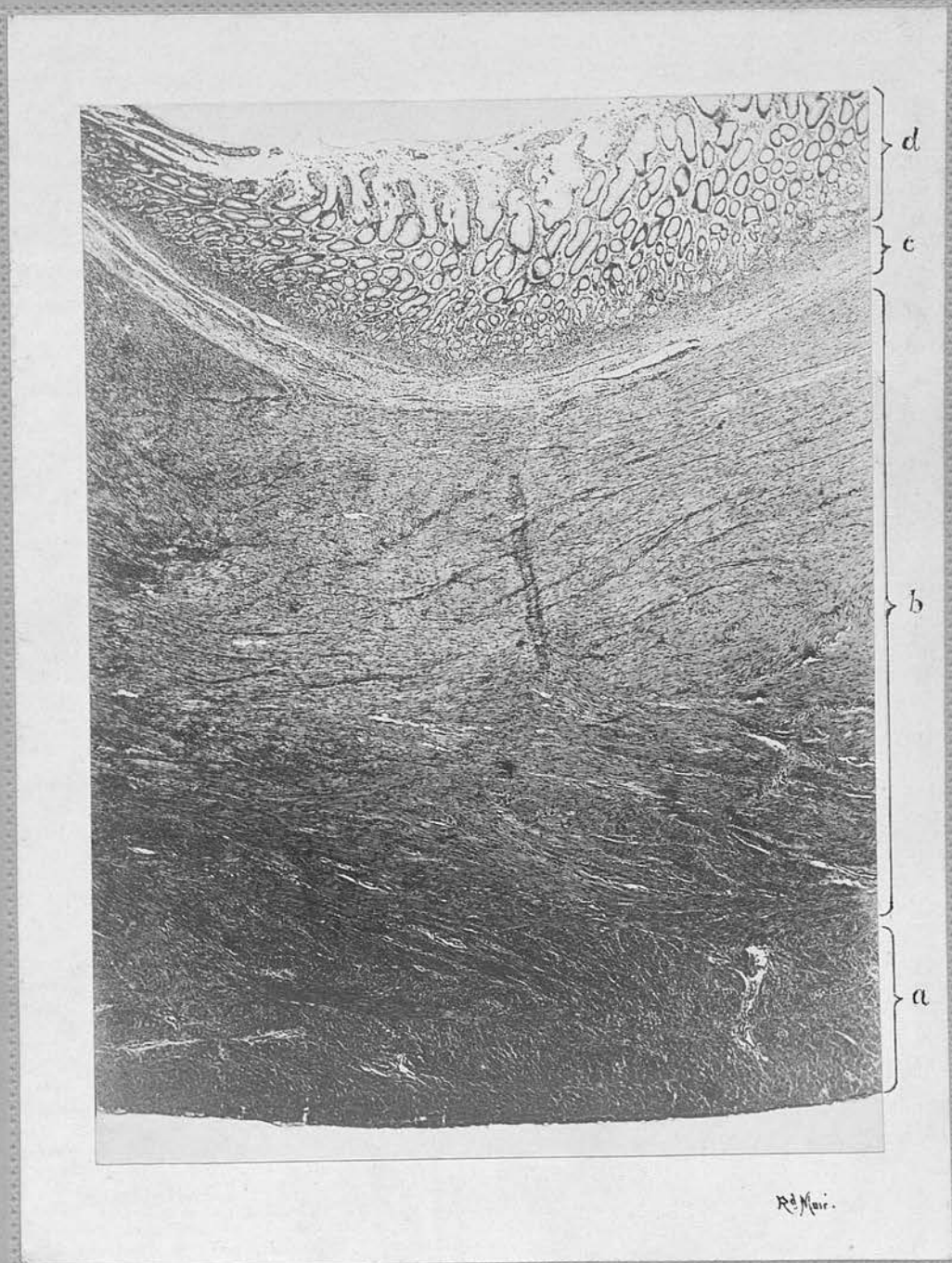


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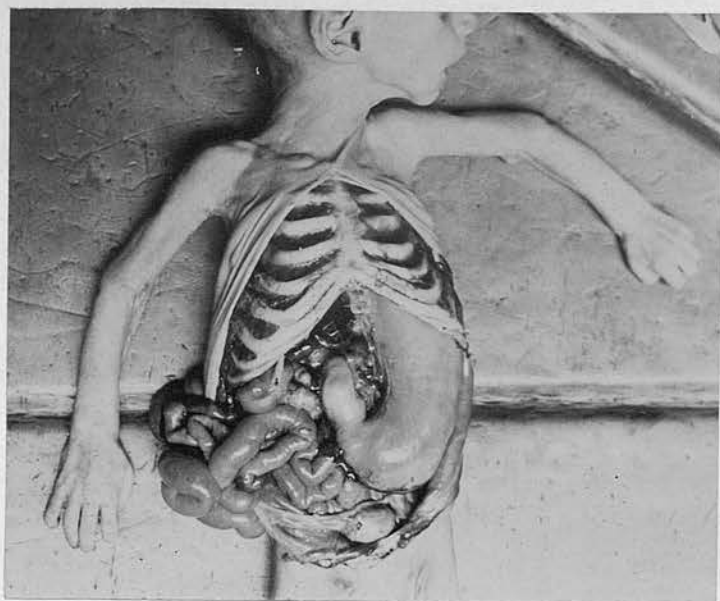


Fig 1



Fig 2.



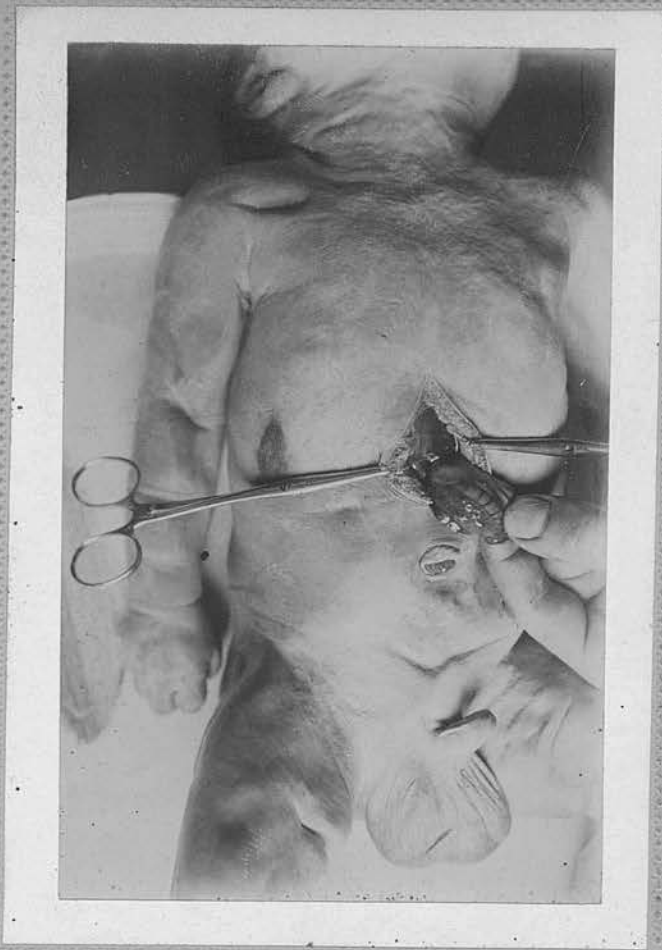


Fig 1



Fig 2



Fig. 1.

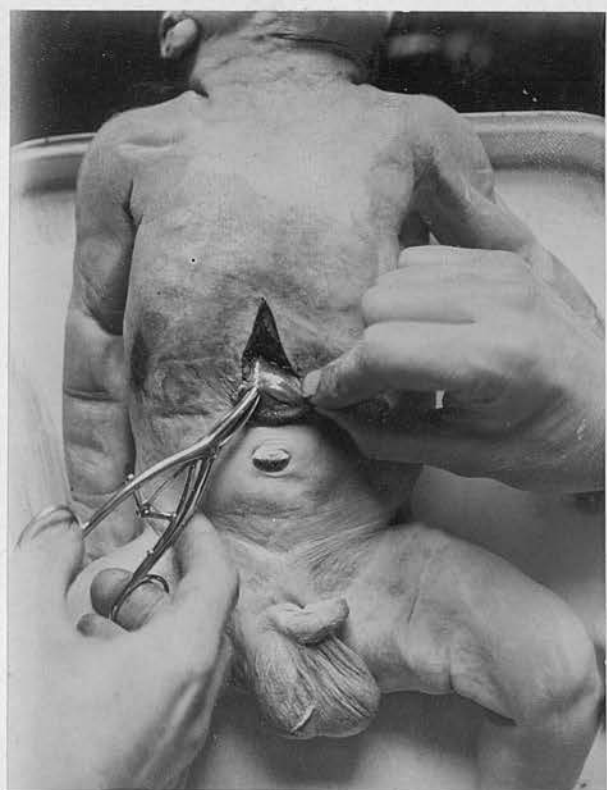


Fig. 2.