INFANTILE HYPERTROPHY OF PYLORUS WITH SPASM CAUSING OBSTRUCTION.

o r

HYPERTROPHIC PYLORIC STENOSIS OF INFANCY.

B y

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C o n t e n t s.

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Prefatory Note.

I must acknowledge my great debt to Drs. McCracken and Ogden of Newcastle-on-Tyne for their kind permission to make any use of notes and specimens thought necessary.

To Mr Pringle I am indebted for permission to use his successful case of gastro-enterostomy and to Dr. Cowan for the case numbered 12.

Since this Thesis was composed, Mr Nicoll has informed me of a case operated upon by him in May 1911. Posterior Gastro-enterostomy was performed. The child died within 24 hours. The great interest of the case lies in the tremendous dilatation of the stomach. The peristaltic waves, he assures me, reached to the right iliac fossa.

I N T R O D U C T O R Y

INTRODUCTORY.

Few diseases remain such pathological mysteries as Hypertrophy of the Pylorus in infants.

A plurality of names and an ignorance of pathology often go together and this is well shown in this disease which is known variously as -

Congenital Stenosis of Pylorus.
Congenital Gastric Spasm,
Congenital Hypertrophy of Pylorus & Stomach-wall
Congenital Pyloric Spasm
Constricting Pyloric Hypertrophy
Infantile Hypertrophy of Pylorus.

Cautley (Diseases of Children p. 262) calls it "Congenital Hypertrophic Stenosis" because a hypertrophy or hyperplasia causing stenosis is the feature and is congenital.

The Hypertrophy, he thinks, is congenital, not the stenosis.

I have entitled this Thesis "Infantile Hypertrophy of/

of the Pylorus with spasm causing obstruction".

I call it "Infantile" because this does not commit one to the origin of the hypertrophy in uterine life, and although, doubtless, in the vast majority of cases, the condition is congenital yet it is by no means certain that, in a few cases, the hypertrophy does not develop after birth just as cases of pyloric hypertrophy with increase of the muscular element may develop in adults suffering from gastritis and gastric ulcer.

The condition is, in my opinion, undoubtedly due to an excess of development of the muscular coat of the pylorus whether arising from practically tumour formation or from simple hypertrophy.

I believe that the obstruction is not caused by the hypertrophy, i.e. is not simply mechanical, but is due to spasm whose gravity is increased by the fact that the contraction occurs in a muscle which is present to an excessive degree.

Burghard/

Burghard (See Section on Pathology) was easily able to pass a No x catheter through the hypertrophied pylorus at operation showing, therefore, that, in the absence of spasm, there was no obstruction presented by the increased muscle.

We may assume that, the greater the muscle increase, the stronger and more obstructive will the spasm be with a more complete and unyielding stenosis.

Even spasm by itself and without any hypertrophy of the pyloric muscle can produce obstruction as shown by the disease known as Simple Pyloric spasm which, although usually yielding rapidly to appropriate treatment, has yet caused death: and at Section no obstruction has been found caused by hypertrophy.

On wonders why such a definite clinical and pathological entity escaped notice until within very recent times.

If clinicians are blameworthy, much more pathologists who, it will be agreed, have less excuse for overlooking the condition than have physicians.

It/

It may be replied that the condition has increased greatly in incidence, as has been alleged of appendicitis; but surely the increase of cases cannot have been so large as to attract notice to the disease only within the last twenty years and to lead to the report of recoveries under medical or surgical treatment and of post-mortem examinations to a number which, in the literature of all nations, must amount to, at least, two hundred and fifty.

We may take it for granted that the disease is of no late development but rather that cases have been dying or exceptionally recovering in numbers yearly, unrecognised and diagnosed as marasmus, gastritis etc.

Even now the knowledge that there is such a condition is by no means general and fully 50% of general practitioners, out of twenty spoken to haphazardly by me, have been ignorant of its occurrence.

Its recognition is due, as so much of the advance of medical knowledge - more particularly clinical - ~~is due~~ to the increasingly painstaking accumulation of observations.

We should remember that even the comparatively common/

common gastric ulcer was really first described about the beginning of the nineteenth century.

West, in the preface to his text-book of 1848, proudly boasts that he had kept accurate notes of the diseases of 600 children out of 14,000 seen by him in nine years.

This then, I take it, is the explanation of the delayed recognition of Infantile Pyloric Hypertrophy.

To Osler is due the credit of having discovered what is probably the first recorded case.

The case was reported in the "Transactions of the Newhaven Medical Association.1788" by Hezekiah Beardsley as "Scirrhus of a Pylorus in an infant."

The symptoms notedwere constant vomitting, leanness and wizened old look of the child. The child lived to be four weeks old and post-mortem examination showed that "the pylorus was invested with a hard compact substance or scirrhosity which so completely obstructed the passage into the duodenum as to admit with the greatest difficulty the finest fluid." (Osler, Boston Med. and Surg. Journal.March.12th 1903. Annotation.Lancet.April 11th.1903. p. 1049.

Thomson/

Thomson unearthed two cases reported, one by Williamson in the London and Edinburgh Journal of Medical Science 1841. p. 23, the other by Dawosky in Caspar's Wochenschrift 1842 No. 7.

Williamson's paper was entitled a "Case of Scirrhus of the Stomach probably Congenital"; Dawosky's, a case of "Hypertrophy of the Submucous Cellular Tissue of the Pylorus in an Infant six weeks old."

I have not had the advantage of access to the originals but it seems all but impossible that the two mentioned by Thomson should have been anything but Infantile Pyloric Hypertrophy, although Scirrhus Carcinoma of the stomach has been reported as early as six weeks.

Beardsley's case also seems to have been a true Pyloric Hypertrophy.

Leaving out Landerer's Article (Ueber Angeborene Stenose des Pylorus, Tubingen, 1879) on congenital stenosis of the pylorus the next hint of the disease was by Maier in 1885 (Virchow's Archw 1885 Bd. c II. S. 413) who described a stenosis with hypertrophy and suggested a congenital/

congenital origin. Landerer's and Maier's Cases had all passed the early years of childhood.

Following the report in 1888 by Hirschsprung of two cases and Thomson's work in this country, the condition, pyloric hypertrophy, was frequently recognised.

According to Pfaundler (Pfaundler and Schlossman, Diseases of Children, Eng. Trans, Vol. III. p.200) no unquestioned case has been published in Latin and Slavonic countries (May 1905).

It seems impossible that the disease should be confined to the Teutonic, Scandinavian and Anglo-Saxon families.

Its incidence in the former nations may be less but, probably the knowledge of the disease has not established itself as yet.

Clinical Features and Course.

CLINICAL FEATURES AND COURSE.

The disease seems more common than certain of the other ailments seen in infancy. Heubner has calculated the occurrence in relation to other diseases at .5% and my experience in a year's work in a Children's Hospital, gives about the same figure.

SEX:- In the study of the disease in a number of cases one is struck immediately by its greater frequency in males. Heubner (Die Therapie der Gegenwart Oct.1906 p.433) in his cases found 31 boys and 8 girls. Starck (Zentralblatt fur Kinderheilkunde May 1909 p.156) in 12 cases, had 8 boys.

There is no doubt that there is a marked disposition for males to be oftener affected. Why this should be is, in the present state of our knowledge, inexplicable.

POSITION IN FAMILY:- Another striking feature in its incidence is that there seems to be a relatively large number of first-born children affected.

In many cases reported, the position of the child in the family is not mentioned and further investigation may show that there is no greater likelihood of the eldest being affected. In my cases there were four

first-born children. Davidson (quoted by Hutchison B.M.J. Vol.11 1910 p.1021) reported in 19 cases, 10 as first children.

FAMILY HISTORY:- Another point which my cases suggest is that it may be more common in illegitimate children.

Certain observers have suggested a familial tendency but the evidence in favour is weak. Peden. (Glasgow Med. Journal, 1889, Vol.XXI p.417) one of the earliest writers on the subject, mentions that three previous children were "great vomiters" and Henschel (cited by Cautley, Medico-Chirurgical Transactions, Vol.82, p.45) had four cases.

The information given by Cautley regarding Henschel's cases is meagre and exception can be taken to each as an example of hypertrophic stenosis. One lived to be 16 months, a sister to be 19 months. It may be argued that these children, if suffering from pyloric hypertrophy, would have died much sooner.

To the remaining two much stronger objections can be taken, as in one, a female who died aet 7 months, no post mortem examination was performed and the other, a brother, recovered. A fourth child had abnormalities of the pylorus.

Cautley mentions that none of these cases showed typical thickening of the pylorus. Starck (Ibid./

(Ibid. Cases 3 and 4) reports two cases in one family. Bendix (Ibid. p.1814) contents himself by saying he finds a distinct familial disposition for similar complaints.

An attempt has been made to assume a neuropathic family history, an exceedingly difficult thing to prove, and in this light, it would be exceedingly interesting to learn the after histories of cases recovering and attaining adult ages.

CONDITION AT BIRTH:- The child, as a rule, is a normal healthy child when born and there is nothing to suggest disease in the baby or in the family.

In a rapid survey of the cases, one is struck by the fact that so many are breast-fed, at least to begin with, and it is, in many cases, only when the child begins to vomit that artificial feeding is resorted to out of an idea that it is the mother's milk which is causing the symptoms.

ONSET OF SYMPTOMS:-

VOMITING:- The most striking symptom and the symptom which draws attention to the fact that all is not right with the child is the occurrence of vomiting.

In a few cases vomiting is preceded by diarrhoea but the number in which this antecedent diarrhoea has occurred is not large enough to allow us to assume other than that it is merely incidental.

The onset of vomiting in the majority of cases is in the second fortnight of life but it may date from birth as recorded by Burghard in 5 cases (Cases 2,6,7,9,11, Clinical Society's Trans.VolXL.)

In 52 cases the average day of onset of vomiting was the 17th day (Rotch.Pediatries.5th Ed.p.771) Kerr (Diagnostics of Diseases of Children.1907.p.114) says vomiting develops within the first three days of life.

The number of cases seen by any one observer is so small and the occurrence of vomiting is so likely to be disregarded at first by the mother that it is impossible to form any very strict rule.

Vomiting at first is disregarded or treated lightly. It is occasionally and as a general rule seems to increase gradually in frequency and violence. In a few cases vomiting by its gravity attracts notice and raises alarm almost/

almost immediately after its onset.

Generally it is first regurgitant; small quantities being ejected but, as time progresses, it becomes more violent, more frequent and more easily produced until quite small feeds and even water may cause emesis. In studying the literature of the subject one receives the impression that the character of the vomiting is pathognomonic. It is usually said to be forcible and projectile in type. In one case the vomitus was projected to a distance of three feet (B.M.J. 13th Oct. 1906 p. 940). Koplik (Diseases of Children p.507)says it is"characteristic and is simply regurgitant, at intervals, and in small quantities"

The only constant feature about the vomiting that I have observed is its persistence.

It varies greatly in frequency and in force and the vomitus in quantity and character.

Vomiting may be simply regurgitant, it may be interrupted i.e. vomiting may occur when small quantities are regurgitated, it may be projectile with varying quantities expelled, or vomiting may not occur for some considerable time and food accumulates to be ejected in one large vomit.

These characters may be present in the same child about the same time although if the case is closely observed/

observed one type will be seen to preponderate. It must be remembered that vomiting must be modified as to character by the amount of food taken, the quantity retained in the stomach and not entering the intestine, the strength of the child, the degree of catarrh present, and the condition of the stomach which here as in all obstructive diseases affecting the organ, will pass through the three stages of compensation, stagnation, and dilatation with retention.

As the disease and its effects progress and if the child live long enough, vomiting, which at first was probably regurgitant then forcible, becomes less projectile owing to commencing dilatation and one may congratulate oneself that the child is improving, while really the stomach is losing its "compensation". Again the frequency may diminish and again premature congratulations while the food is being simply retained in the stomach to be ejected perhaps once in 36 hours. This error should not be made if lavage is practised regularly and the quantity of the wash-out measured.

The frequency of vomiting may be modified by treatment. Still (Trans.Path.Soc.Lond.Vol.L.1899.p.89) found a marked diminution of vomiting when nasal feeding

was/

was begun. The child died five days after, so the decrease in frequency may have been the diminution that occurs in some cases before a fatal conclusion.

Batten's case (Lancet.Dec.2nd.1899, p.1511) of nasal feeding is not open to the same objection.

In this case whenever nasal feeding was dropped vomiting either recurred or increased. The child recovered to die later of Broncho-pneumonia. Lavage by the removal of irritating products of digestion and of fermentation will decrease vomiting. Vomiting may cease for some time on change of diet, (Robson and Moynihan Diseases of Stomach p.45). Moving the child about, as by carrying it, may precipitate emesis.

VOMITUS:- The character of the vomitus varies, but large coagula are present so frequently as to form a feature worthy of remark. The formation of large coagula is noteworthy and I have observed in practising lavage that the catheter is, if anything, more liable to be blocked by these clots than in other diseases. Towards the end the vomitus seems to become more fluid probably due to transudation from the walls of the irritated stomach. This increased fluidity will be associated with increase of the stomach content and Starck (Ibid p.160) has/

has proved this by lavage after a meal and measuring the result. The quantity of mucus present in the vomitus and in test feeds is striking.

The amount present in this affection in test feeds is certainly greater than in other gastric diseases. The quantity is modified quickly by lavage. Blood in streaks may be present in the ejecta or it may amount in rare cases to such an amount as to produce a coffee-ground appearance (B.M.J.Vol.II. p.940).

Bile, by the majority of writers, is said never to be present showing that an obstruction is present between the stomach and the bile duct papilla. Clogg, Nicoll and Pfaundler mention its presence in the vomitus on a few occasions but in a thorough search I cannot find what authority there is for the statement. Maybe it is one of these mistakes which creep into medical literature and is slavishly copied by other writers.

As a matter of fact bile is exceedingly rarely present in vomiting in infancy in quantities sufficiently great to be detected by the naked eye. I have examined the vomitus in one case only other than cases of hypertrophic strenosis. The case was one of strangulated hernia in an infant, vomiting was frequent and at operation the bowel was found gangrenous. Bile was absent from the vomitus/

vomitus.

CONSTIPATION:- Almost coincident with the onset of vomiting, constipation develops. Constipation is marked and constant in many cases. Pfaundler (Diseases of Children.p.201) mentions a case where no motion was passed for 12 days. The small quantity of food which escapes into the intestine is supposed by most writers to produce the constipation, for if there is little material in the bowel, little can be voided. The small amount of faecal matter found in the bowel at section is striking. It should be remembered that constipation is not present in all cases. It may alternate with diarrhoea and, as mentioned previously, frequently diarrhoea ushers in the close in medical and in surgical cases.

CHARACTER OF STOOLS:- The stools, which, at birth, are usually normal become in most cases small and contain little faecal matter. Melaena is rare. It would be interesting to investigate any increase in the time taken for material to pass along the bowels by means of the exhibition of dyes.

URINE:- Urine is decreased in amount due to loss of fluid by vomiting and it is striking in marked cases to observe how infrequently napkins have to be changed.

Napkins/

Napkins are said to be stained by excess of urates, I have not noticed any noteworthy difference. I have found marked acctonuria in two cases. The test employed was the iodoform one.

In both cases the amount of iodoform formed, estimated by the odour, was large and in one case, which recovered, it amounted to as great a quantity as that found in a case of appendicular abscess, dying rapidly of Delayed Chloroform Poisoning. No alcohol had been given to the infants.

Four other cases including one of pyloric spasm and one, an operation case, were used as controls to one of the cases of Hypertrophic strenosis.

The urine of the operation case gave a reaction just detectable by smell. The reaction in the case of pyloric spasm was stronger and in the child suffering from hypertrophic strenosis the reaction appeared immediately, and the odour was striking. The remaining two cases were negative. These results were secured on four different occasions and on different days and in addition the crystals were found microscopically. I may remark that the urine of the mother of one of the cases of hypertrophic strenosis was also tested. The Iodoform test was faintly positive/

positive. Owing to the possibility of the value of the test being vitiated by alcohol and ffom the fact that little trust can be placed in the mother's assurances, no stress can be laid on the reaction secured.

TYPES:- In the cases I have observed I have noticed two distinct types. In one which recovered/the child, although greatly emaciated, was bright, taking all interest in its surroundings for a baby. Peristalsis was markedly visible and the pylorus was easily palpable. In the others, which died, drowsiness and whining when roused, were equally striking. It may be said that these cases were examples of different stages. Between the case which recovered and one which died there was nothing to choose clinically.

The child is said to take the breast or bottle with avidity only to drop off easily satisfied. In one of my cases it was noticed that the child continually sucked the fingers. Pain is variable, it may be present or absent.

NERVOUS SYMPTOMS:- Tetany has been recorded as have convulsions. Little need be said about the general con dition of the child.

MARASMUS:- Marasmus in greater or less degree appears in undoubted cases sooner or later and the child may waste almost to a skeleton. In one case the weight on admission to Hospital was 3½ lbs. (Broadbent-Lancet Dec. 19 1908). The child died but Cooke's case (Lancet Jany. 11th 1908 p. 129) weighing 3½ lbs. recovered under medical treatment.

Improvement may set in suddenly after weeks and the weight improve quickly or the child may die of exhaustion following vomiting, of starvation, of enteritis or intercurrent disease.

ABDOMEN:- On examination of the abdomen it may be observed in advanced cases that the upper portion may be prominent contrasting with flaccid lower portion flattened from empty atonic intestines.

PERISTALSIS:- On repeated examination peristalsis may be observed when the upper abdomen will become prominent and waves will be seen crossing from left to right. Peristalsis may be slight or it may be strikingly visible so marked that an unobservant mother may notice it. It may even form a bulging visible through the clothes (B.M.J. Oct.8th 1910. p.1021). The waves are best seen after a feed, may be elicited by kneading or flicking the epigastrum and should be repeatedly looked for. In one of my cases they were most marked when the child fell asleep as if some inhibitory influence had been removed. The waves issue as a rounded swelling from under the left costal margins, travel across the abdomen above and to the right of the umbilicus, and may even travel down the duodenum while the position of the pylorus may be indicated by a constriction in the wave (Dent's case, quoted by Robson & Moynihan. Ibid. p.56.)

In one case at least peristalsis has been observed extending below the umbilicus (Clowes Lancet Aug.22nd 1908) The/

The waves number up to three per minute, sometimes are single and take 15-20 seconds to travel across the abdomen

An hour-glass appearance may be given to the stomach by the presence of two waves and this hour-glass appearance may also be simulated when a wave is situated beneath the division of the recti.

Kehr (Clogg Practitioner Vol. 73 1904.) reports the occurrence, after massage of the abdomen, of a hard tumour under the ribs reaching toright and disappearing under the right costal margins.

Presumably this was the contracting stomach. In one of the Cases I report I saw peristalsis only once, although I looked for the phenomenon repeatedly. The child was being fed per rectum so may be if food had been exhibited by the mouth peristalsis would have been stimulated. The occurrence of anti-peristalsis has been doubted. I have seen it once following the completion of the normal wave.

DILATATION:- Dilatation of the stomach may be evident and the stomach has been found large enough to hold 14 ozs. (Cantley B.M.J. Oct.13, 1906 p.940) and in another 15 ounces of fluid could be introduced into the stomach without inducing vomiting. (Guthrie, Reports of Soc.for Study of Disease in Children, Vol.III. p.4).

Percussion may show the organ is dilated, and in some cases/

cases it seems possible that the percussion note over the stomach may contrast with a duller note obtained over the empty intestines. I have never been able by percussion alone to say definitely that the stomach was dilated.

Percussion of the stomach in infants is difficult, and often is very liable to lead to error.

A better way to distinguish dilatation is by the use of a modified form of 'balottement' with the finger-tips. If performed gently but firmly the procedure if not prolonged does not seem to cause any discomfort to the patient. The hand should be warmed and, with the child feeding and lying comfortably, the finger-tips should be gently pushed with a slight prodding motion into the abdominal wall. If the child squirms the hand should not be removed, but time should be allowed for the child to become accustomed to the examining hand.

If the stomach is dilated there is a feeling of resilience and a sensation as if one were pushing a rubber bag.

The dilatation may be so great as to be easily appreciated by the hand laid flat upon the abdomen.

PYLORIC TUMOUR:- Palpation, in addition to discovering dilatation will, if thoroughly and repeatedly done, and if there is emaciation, reveal the presence of a tumour which is the pylorus. The pylorus is best sought for about 1½" to/

to the right and above the umbilious.

It can be pressed against the sides of the vertebrae and gives the impression of a small hard body, movable, and about the size of a hazelnut. It may seem elongated or rounded.

It has been said that, if the tumour be under the edge of the liver, it cannot be palpated.

Although a shortened mesentery has been reported, so short as to preclude anterior gastro-enterostomy (Case of Gastro-enterostomy, Clogg, Practitioner Vol. 73, 1904.), I do not think that the pylorus is ever so high up as to prevent palpation of it.

Miller (Lancet Jan.18th, 1908. p.188) suggests that difficulty in feeling the pylorus is caused by bending of the stomach so that the tumour lies behind the viscus.

According to Fenwick (Disorders of Digestion p.2) the pyloric end, when the stomach is full, moves downwards and to the right. We would expect then that, if the child was fed, the pylorus would become more easily palpable.

There is great difference of opinion as to the palpability of the tumour.

Starck (Ibid. p.155) puts the palpability at 30%. Nicoll (B.M.J. 1904 Oct.28th p. 1148) thinks that, in the majority of cases, nothing abnormal can be detected through the parietes. Pfaundler (Ibid. p.202) says a palpable tumour is present in the minority of cases. Still, (Lancet.March 11, 1905. p.634) out of 20 cases, palpated

the pylorus in 19. Robson and Moynihan (Diseases of Stomach 1906. p.46) think that the " mere position of the tumour is no bar to palpation" and that if it is under the liver examination under chloroform will reveal it.

In the cases seen by me the pylorus was palpated without difficulty save in one. In one case in addition to the pylorus being palpable the head of the pancreas and its body lying across the middle line could be felt.

The child, of course, was greatly emaciated. To discover the pylorus requires infinite patience, gentleness and a correct method of examination. Examination should be repeated.

In examining the child care should be taken that the child is lying comfortably preferably in the arms of a nurse, with the head and shoulders slightly raised. The child should be previously given a small feed and another should be kept in readiness. The abdomen should be kept protected and the extremities of the fingers of the warmed hand should palpate gently but firmly in the region of the pylorus. If the child should cry an attempt should be made to distract its attention and meantime what little pressure has been applied should be slightly decreased. After a little the child will probably become accustomed to the pressure which then should be stealthily increased.

The/

The attempt to palpate the pylorus should not be prolonged, for the baby may, in a future examination, associate the examiner's hand with the idea of discomfort.

I have attempted to palpate the pylorus per rectum in a child with symptoms pointing to hypertrophic stenosis. Chloroform was given and the finger introduced. No tumour was found. The child collapsed and its condition gave rise to great anxiety. Afterwards a diagnosis was made of pyloric spasm. I consider that the method of examination per rectum is too dangerous in these young infants.

I have already mentioned that in one case, the pylorus was palpated with difficulty. When I employed the method of examination I am about to describe, the tumour was easily felt. The baby was first given a small feed. Then it was placed in an easy position in a warm bath. While the child was in the bath the pylorus was palpated. I have tried the method in other children, healthy and otherwise. The water should be, to begin with, at a temperature of 92° and should be slowly raised to 103° and kept there. Care should be taken to prevent scalding as the temperature is being raised. The child should lie comfortably in the bath for some time when a splendid relaxation of the abdominal wall may occur permitting/

permitting of free palpation. The child as I have found, may even fall asleep in the bath, and the abdomen become quite flaccid. In older babies there may be struggling. The infant should then be kept in the water for some time then taken out and dried rapidly before a fire.

Then palpation should be attempted. If proper care is taken the method is free from risk and I do not consider it depressing.

RETENTION:- In the course of treatment it will be found that lavage shows a retention of food in the stomach. If there is a considerable accumulation of food it may be demonstrated by weighing the child before and after lavage.

On the only occasion where I had the child weighed specially, lavage made a difference of two ounces in the weight showing that the last feed given to the child two hours previously, had been retained.*

In test feeds and in lavage it will be noticed that mucus is increased as in any retention. The quantity of mucus present is modified by lavage.

*Fenwick (Disorders of Digestion of Children p.13) found that on cow's milk, the stomach was emptied in 2 to 2½ hours.

HYDROCHLORIC ACID:- I have elsewhere observed that writers differ as to the presence of free HCl. In two of my cases of hypertrophic stenosis, test-feeds were given. The test-feeds, consisting of three ounces of milk were given at 10 a.m. and were drawn off in 30 minutes. Lavage was not done during the preceding 24 hours. Controls were used, including two cases of pyloric spasm which were having lavage.

One case of hypertrophic stenosis was given a test feed once, the other had meals on three successive days.

On the cases of hypertrophic stenosis, free HCl was present. In the case where a test meal was given on successive days, the free HCl amounted to .03, .02, and .04 per 100 cc.

The controls with the exception of the cases of pyloric spasm showed no free HCl. The child whose amount of free HCl is recorded, was dismissed having slowly increased in weight from 5½ lbs. on admission, to 8½ lbs. The child had been an in-patient for 2½ months and in the last fortnight of residence there had been no vomiting. She relapsed, was re-admitted and during the second residence test meals were again given twice, when there was no free HCl but the total acidity was increased.

I/

I state these results of the second examinations -

1st Test Meal M.C. (Hypertrophic stenosis)	A.G.(Control)
Total Acidity in terms of HCl .3	.18
2nd Test Meal M.C. Hypertrophic stenosis	
Total acidity in terms of HCl .36	.18

With the first test meal there was one control with total acidity = .18.

With the second test meal in addition to the control which had already served, I had other two whose total acidities in terms of HCl.were respectively .07 and .24.

If total acidities are calculated in terms of lactic acid, the case of hypertrophic stenosis shows still of course, greater acidity again. But the milk forming the test meals was the same, therefore the increased amoint of acidity in the hypertrophic stenosis was not due to any lactic acid which may have been present in the milk.

If the increased total acidity was not due to the milk, it must have developed in the stomach.

May not the variable results found, depend on the quantity of mucus in the stomach.

Foster (American Journal of Med. Sciences Vol.CXXXIII pp.303 to 306) has shown incontestably that HCl. combines with mucus or rather that mucus + pepsin combines with HCl and/

and Pavlov has stated that in a normal stomach as much as 25% of the original acidity may be neutralized by mucus.

As to the ultimate result in these cases, the child may, after showing no improvement for a considerable time despite treatment recover for no discoverable reason. When the issue is a fatal one the child may die of exhaustion as in Nicoll's case (See Diagnosis), it may continue in a mis erably marasmic condition to die of starvation, enteritis may occur or intercurrent disease such as broncho-pneumonia, or tuberculosis, may close its life. Some suddenly, and without warning, collapse.

MORBID ANATOMY.

MORBID ANATOMY.

The most striking point in the pathology of the disease is the presence of a thickened pylorus which according to Coats (Manual of Path. 1903 p.953) is often very firmly fixed owing to Thickness and inelasticity of the duodeno-hepatic ligament and in addition according to Pepper (Keating's Cyclopaedia of Children Diseases Supplement p.655) by thickening of the lesser omentum. In three cases I found the pylorus quite movable.

The pylorus on cutting is found to be hard and always is elongated. Stiles (B.M.J. Oct. 13, 1906 p.944) says the mass is more strictly fusiform and olive shaped than cylindrical and in his experience the greater the hypertrophy the more fusiform is the tumor.

In plate VI. Fig. c. I show a specimen photograph which shows this fusiform shape. The pylorus has been split through the lumen which is marked l. The ring-like form which is alleged to occur I believe to be the result of agonal or post-mortem contraction occurring in a practically normal pylorus. In an exhaustive/

exhaustive search I have not seen a ring form figured.

In one specimen removed from a child aet 18 months, who had never had stenotic symptoms, dying of Broncho-pneumonia following measles, I have observed a very marked ring-like pylorus which was more apparent on section.

In addition four portions of the bowel showed marked agonal contraction each part being about six inches long.

The stomach was submitted to the inspection of an expert who immediately pronounced the specimen an example of the ring-like form of hypertrophy.

In the museum of the Glasgow Royal Infirmary there is shown a specimen which seems to me to be a very modified example of the ring-like form arising from agonal contraction. The child from whom it was taken died of Tuberculosis.

It is shown with a marked example of pyloric hyper-trophy and I am unable to say whether the specimens are intended to exhibit the differences in the two forms or whether both are supposed to be examples of Hypertrophic Stenosis/

Stenosis.

All my 12 cases were of the elongated type of hypertrophy. The hypertrophy on section measured - Length in nine cases as follows:-

	Length	Thickness of wall
1.	1·5 c.m.	·4 c.m.
2.	3.0	·6
3.	1·9	.6
4.	1·8	.9
5.	1·6	.6
6.	1·7	.5
7.	1·5	.5
8.	2	:7
9.	2·5	·8

No measurement of wall, lumen and wall was taken and three were not measured.

It must be remembered that in some of these there would be post-mortem contraction in addition to the contraction which results from immersion in formalin.

They were all measured after section from above downwards.

A/

A striking feature was that the longer the pyloric thickening the less contracted appeared the stomach. Plate III. Fig.A. (Fig. D. is an exception).

The hypertrophy at the duodenum seemed to end abruptly in all cases, forming a marked projection into that viscus like the cervix uteri (Plate V. Fig.A and Plate VII. Fig.B). This appearance is not peculiar to the disease and is an exaggeration of the more or less complete annular valve found at the pyloro-duodenal junction in normal specimens and this valve according to Stiles (B.M.J. Oct. 13, 1906, p.943) is normally more marked in infants than in adults. In one case there seemed to be thickening of the duodenal wall as far as the bile - duct papilla (Plate VIII. Fig.A).

At the gastric end the tumour according to Cantley (Oct.13 B.M.J. 1906. p.940) has a distinct limit. I found that the tumour ends comparatively abruptly where the pyloric wall joins the stomach wall on the lesser curvature. Below it shades off rapidly into the wall of the stomach joining the greater curvature and at no point here can the thickening be said to have a definite limit.

I/

I would mention one exception to the above, viz:- a specimen not figured, which showed a markedly dilated stomach. Here the pyloric thickening ended abruptly on the gastric side as described by Cantley; Maier (quoted by Pepper, Keatings Cyclopaedia of Diseases of Children, Supplement, p. 654) considered that the sharp localization of the hypertrophy to the pylorus itself distinguished the congenital from the acquired form of pyloric stenosis.

I consider that the localization of the hypertrophy is no index to the congenital or the acquired form of stenosis.

In many specimens it is found to be impossible to force fluid from the stomach through the thickened pylorus and rarely it has been impossible to pass a probe through the lumen.

On looking at the pyloric canal from the stomach side there may be seen folds of mucous membrane disappearing into the canal and presenting a stellate, sometimes a whorled, sometimes an irregular arrangement (Plate IV. Figs. A.B.C.) As a rule one or two of these folds are especially prominent and on dividing the/

the pylorus longitudinally they may be seen projecting into the lumen and lessening the calibre. (Plate V. Figs.B.C.) These folds at times are congested. Hutcheson (B.M.Oct.8.1910.p.1022) seems to think that these longitudinal folds are not present in health.

Their only abnormality lies in their size and prominence for in the majority of healthy pylori removed carefully and withthe minimum of disturbance I have found folds present but much less marked. (Plate VI. Fig.A). According to Garrod (Clin. Journal 1906 Sept. 26, p.380) the most prominent fold corresponds with the greater or lesser curvature. In all of my specimens they were lateral. Garrod (Ibib p. 380) mentions that the presence of these folds increases the obstruction by diminishing the lumen. No doubt they do so if spasm is present but if the pylorus is actively dilating these folds, I would say, are smoothed out and in post-mortem specimens, if fresh, dilatation of the pylorus causes them to disappear, The folds are exactly similar to the rugae found in the normal stomach and they disappear just as these rugae disappear when the viscus is filled showing they have not the origin of the valvulae conniventes which are structural., The stomach may be dilated with/

with thinned walls or it may seem to be hypertrophied and contracted.

The capacity of the organ post-mortem was not estimated in my cases as measurements after death are absolutely valueless. Drewitz calculated the capacity of the stomach at one month at 99 c.cm; Pfaundler at 150. All but one of my specimens showed thickening of the stomach wall and this thickening was most marked about the pyloric antrum. "Pyloric antrum" is a term used rather loosely and indefinitely by anatomists but by it I mean the stomach for about one inch from the pylorus.

One would expect this hypertrophy of the antrum to occur for this portion resembles the intestine by the greater development of the circular coat. (Habershon Diseases of Stomach 1909. p. 32) and the main peristaltic work is normally carried on by the pyloric end of the stomach. (Hemmeter Diseases of Stomach. p. 31). The peristaltic work of the pyloric end of the stomach would be increased by the obstruction offered at the pylorus with the result that hypertrophy would occur.

The increase in the stomach wall is less marked about the fundus and at the cardia the hypertrophy may be more noticeable again. In one of my cases this hypertrophy was very striking (Plate VII. Fig. A. n.) The vestibule may be exceedingly similar to the fundus perhaps/

perhaps owing to commencing dilatation so that if the orifices be neglected it may be impossible to distinguish between the distal and proximal ends of the viscus. The lesser curvature may show a more acute angle than is usually found in the normal or the whole organ may approach the tubular in shape. (Plate III). These appearances, when we remember the ever changing shape of the normal organ in life, are not of any importance.

The stomach wall may show evidence of gastritis and there may be minute haemorrhage . Gastritis, oedema of folds, and dilatation are all secondary.

The organ is uaually found to contain an excess of mucus.

Lastly the oesophagus may be found dilated at its lower end.

The intestine which seems of smaller calibre than usual, is found empty as a rule. Its walls are thinned, tear easily and evidence of enteritis may be present.

In one case I found the shaven-beard appearance in Peyer's patches, found occasionally in cases of marasmus.

Parenthetically I may mention that only in this case and/

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and in one case of simple marasmus has this shaven-beard appearance been found in about 60 sections done by me. The liver, gall-bladder, ducts, spleen and pancreas seemed normal in three cases examined by me.

Lungs, in two of the three cases, showed areas of collapse as is often found in marasmus. As to the microscopic appearances of the pylorus the vast weight of evidence is in favour of the unfailing appearance of hypertrophy of the circular muscle-layer, but Finkelstein (quoted by Robson & Moynihan Diseases of Stomach 2nd.ED. p. 48) attributed the whole thickening in one case to increase of the longitudinal fibres. Cautley (Trans of Roy. Med. and Chir. Soc. Vol. 82. p.59) thought after seeing the diagrammatic representation by Finkelstein that the specimen had been cut obliquely and Cunningham (Reports of Roy. Soc. of Edinburgh Vol. XLV. Part I, Article No. II. p. 24, footnote) doubted if he had "correctly differentiated between the muscle layers in the wall of the pyloric canal". Hirschsprung (quoted by Cunningham Ibid p. 22) stated that relatively the longitudinal fibres were the more strongly developed.

Cunningham (Ibid p. 22) "would not hazard a decided opinion/

opinion as to whether they are both hypertrophied to relatively an equal extent or whether the circular sph. cylinder has undergone the greater degree of thickening" Then he contradicts himself further on by saying after mentioning examination of different specimens - "This would seem to indicate a relatively greater degree of hypertrophy in the longitudinal layer which I believe to be the case." At any rate he thought both layers were hypertrophied and thought a great amount of the thickening was due to contraction.

He also figures (Ibid Plate II. Fig. XIX.) a specimen where the greater hypertrophy was in the longitudinal layer.

All my specimens showed undoubted hypertrophy of the circular muscle and in one case, transverse section showed great increase of the longitudinal layer on the part of the pylorus corresponding to the greater curvature. (Plate IX.) Elsewhere in this pylorus the longitudinal layer did not seem increased. In other pylori there was increase of the longitudinal layer but the increased thickness was not so great as to exclude the possibility of simulation of hypertrophy by contraction.

I found in one specimen a degree of increase of the/

the muscularis mucosae so great as to leave no doubt but that it was due to hypertrophy.

As to the other structures I found they varied greatly in thickness depending evidently on degree of contraction. It was very striking to see how, in longitudinal sections of the same pylorus, the submucosa varied in thickness according as to whether the section passed through a longitudinal fold or not. I am of opinion it cannot be said definitely that, in my specimens, there was true increase in mucous, submucous, or serous coats.

It is quite possible that an ante-mortem oedema may produce an apparent increase in the submucous coat especially. Taylor & Wells(Diseases of Children p. 184) mention that the mucous and submucous coats are frequently increased in thickness, and Thomson (Scottish Med. and Surg. Journal 1897. Vol. I., p.515) reported the submucous connective tissue greatly increased in thickness. Still (3rd. Case Trans. Path. Soc. London, Vol.4, 1899, p.90) found the submucous coat about twice the normal thickness in one case. Holt (Diseases of Infancy & Childhood 3 ED. p. 325) says connective-tissue, submucosa, and mucosa may be increased. Pfaundler (Diseases of Children Pfaundler & Schlossman Trans. Vol. III. p. 204) says the serosa is never changed and doubts if there is any change in the connective/

connective tissue element. Gran (quoted by Lambert & Foster American Journal of Med.Science Vol.CXXXIV p.336) found the circular muscle twice as thick and the longitudinal layer showed no increase. All other tissue elements even serosa, were, according to Gran, approximately double the normal thickness.

Meltzer (New York Med. Rec. Aug.20th 1898) found in one case that the thickening at the duodenal end of the pylorus was due to fibrous hyperplasia in the submucosa; at proximal end the thickening was due to increase in submucosa and circular muscular layer.

It must be remembered before we can reconcile these varying statements that there is no definite measurements of the thickness of the wall of the normal pylorus nor of its layers, (Still, Trans.Path.Soc.London Vol.4,1899 p.91) found that the normal pyloric wall varied greatly in thickness); that these layers must vary in thickness according to the state of contraction of the part; that it seems reasonable to assume that muscle, which has the power of actively contracting, never or exceedingly rarely reduplicates, that the mucosa is constantly folded in the normally contracting pylorus and smoothed in the dilating pylorus and that the serosa probably also varies in same way but to a less apparent degree.

We/

We must be prepared to find great differences between sections - more especially in the mucosa - taken from normal pylori depending on whether the parts were in systole or diastole and, in the hypertrophied pylora, these differences would be accentuated owing to the greater and stronger contraction produced by the increased muscle. (We assume for the moment that the muscle is hypertrophied).

Davidson (quoted by Hutchison B.M.J. Oct. 8, 1910. p. 1022) found the individual muscle-fibres not only increased in size but found them broader, and Peden (Glas. Med. Journal 1889 Vol. XXI. p. 418) in his report of a case describes the circular muscle-fibres as showing in some areas enormous hypertrophy of individual muscle-cells. I have been unable to corroborate this. I would point out as suggestive of no real increase in size Muller's observation mentioned in Archiv. Fur Der gesammte Physiologie Band CXVI. p. 252 (quoted by Hertz Lancet April 22nd, 1911. p. 1055). Müller found that the length of muscle fibres of the filled stomach of a frog was 1⅓ to 3 times that of the fibres in the empty and contracted stomach. I may also mention here that Müller found that the fibres of the contracted stomach/

stomach were arranged in from 15 - 20 layers, and in the full stomach in 2 - 3 layers.

In one of Still's cases (quoted in Ellbutt's System of Medicine p. 516) the wall of the duodenum in immediate neighbourhood of the pylorus was also slightly thickened. He does not mention which layer was affected. In one of my cases the longitudinal muscular layer of the duodenum was found distinctly thickened and this was the more striking (Plate X B) as there seemed to be no hypertrophy of the longitudinal layer of the pylorus. Further the longitudinal layer of the duodenum in this specimen was thicker than the longitudinal layer of the pylorus contrary to my usual finding in normal pylori. Here it should be remembered that the longitudinal layer of the pylorus is not so continuous with that of the duodenum as some of the text-books would have us believe but that as Cunningham has pointed out (Ibid p.15 & 16) and as is very apparent, many of the longitudinal fibres of the pylorus dip into and disperse among the circular fibres.

Remembering Müller's observation on the arrangement of muscle/

muscle fibres in the contracted stomach it would be hazardous to say definitely in some cases whether the stomach wall generally was hypertrophied or not. But as to the pyloric antrum I think there is no doubt but that there is usually hypertrophy of the muscle here.

In only one of my cases could the cardiac sphincter be said to be definitely thickened (Plate VII. Fig. A). The cardia belonged to a stomach which was not in systole to any appreciable degree, so in all probability there was a real increase in its layers although section did not show that any particular element was increased relatively.

Lastly there may be evidence of gastritis usually most marked about the pyloric half of the stomach and in the intestine even marked macroscopic evidence of enteritis, while the mucosa is usually thinned.

PATHOGENESIS.

Pathogenesis.

Many explanations of the origin of the disease have been advanced but none seems satisfactory.

The most important Theories of the origin of the disease and of the post-mortem appearances are that:-

1. There is no increase in amount of tissue and that the condition during life is one of functional spasm while the post-mortem condition is due to agonal contracture (Pfaundler, quoted by Koplik Diseases of Children p. 512).

2. There is a true increase of tissue.
   (a) That this increase is one of simple excess due to attempt of nature to produce an effieient sphincter (Cantley).
   (b) That there is a true tumor formation (Löbker).
   (c) That the hypertrophy is a reversion to original type (Murray)

These observers believe that the excess of tissue is of foetal origin.

Bernheim-Karrer (quoted by Starck Ibid p. 160) seems to believe that the stenosis is caused by the hypertrophy./

hypertrophy. Others, such as Cautley, suggest that there is spasm grafted on the hypertrophy thus producing stenosis.

3. There is some primary cause which produces hypertrophy and this hypertrophy is organic.

A. That the primary cause of the hypertrophy is spasm arising from

a. Hyperaesthesia of the stomach caused perhaps by ulcer (Shattock Proc.Roy. Soc.Med.Vol LL p.88 et seq.)

b. Disordered nerve function producing antogonistic muscular action (Thompson)

c. Hyperchlorhydria (Knopfelmacher).

d. Reflex irritation from Phimosis.(Bendix)

B. That the primary cause of the hypertrophy which is here thought to be compensatory is to be found in anatomical defects such as -

a. a narrowing of the lumen either primary or produced by disease e.g. Haemorrhage into the tissue.

b. An abnormal position produced by shortening of the duodeno-hepatic ligament and mesentery. (Schotten, Pitt)

As to Pfaunddler's suggestion that there is no true hypertrophy, I have already cited a case which simulated the ring-like form of the disease and here the agonal contracture did not permit of confusion between that pylorus and a hypertrophied one on careful examination.

One would expect if the condition was one of functional spasm alone, to be able to palpate the pylorus in ordinary marasmic cases when spasm had been produced by feeding the child on a diet yielding large and firm coagula. These coagula would give rise to a firm spasmodic closure of the pylorus and pyloric antrum if Cannon's observations (American Journal of Physiology Vol 1 1898 p.369, 370, 382) on the functions of the stomach are correct: and this pyloric spasm would be palpable in marasmic cases as a pyloric tumour if Pfaundler's hypothesis was true.

I have been unable to palpate anything in these marasmic cases simulating the tumour found in pyloric hypertrophy and always if the tumour has been palpable there has been found a thickened pylorus post-mortem.

In two cases, ending fatally and where in life the pylorus was not palpable, although from the other symptoms a diagnosis of hypertrophic stenosis was made, the/

the stomachs were removed, bottled and reported to have thickened pylori. When two years after I came to cut open the organs I discovered they were illustrations of the comparative uselessness of bottling specimens to be admired for they were both examples of agonal contracture, and of a mild degree. They showed no evidence of true hypertrophy of the wall of the pylorus.

If Pfaundler's view was correct it is probable although two cases are too few to found an argument upon that, in these two cases just mentioned, where spasm was present during life and where no pylorus was palpable, the condition of contracture would have been perpetuated in death and would have simulated the increase of the pyloric wall seen in hypertrophic stenosis.

Again in the cases operated upon and dying of peritonitis, no disappearance of Pfaundler's spasm has occurred.

The degree of thickening is too great to be the result of spasm of the infantile pylorus.

We will now consider Cautley's view, that it is a simple redundancy of foetal growth, Murray's suggestion that it is a reversion to an earlier type, and Ibrahim's that the hypertrophy is a persistence of a foetal condition.

I/

I cannot see in what way a simple increase in the amount of muscular tissue could produce obstruction. Burghard (Clin. Soc. Trans. Vol XL p.123) in ten cases at operation found no difficulty in passing a No. 8-10 catheter through the lumen of the pylorus and in the same volume (p.104) Sutherland says "at operation it has frequently been found that the hypertrophied pylorus can and does relax and will often often admit the little finger without difficulty.

Following from Burghard's assertion we assume that even spasm is not produced by the hypertrophied muscle for a hypertrophied muscle would produce a permanent degree of spasm if it produced spasm at all; we would have the permanent cause with the permanent effect and no cure at any time by medical means. Batten's case is an example. There his patient who had recovered and become a normal child died, about 9 months later of Broncho-pneumonia. At the post mortem examination the pylorus was found typical (Lancet Dec. 2nd 1899.p.1899).

Even if hypertrophy were primary, as Cautley believes, there would be no reason why spasm producing obstruction should arise from that hypertrophy. There is no true stenosis, as I hope to show, and in the circular muscle would remain the same inhibitory power - whatever/

whatever inhibition may be, and in the longitudinal muscle would remain the same contracting power to produce dilatation.

Here I assume that the longitudinal fibres normally dilates the pylorus by pulling on its wall whose circular muscle action has been inhibited. Physiologists have next to nothing to tell us of "how" the pylorus dilates; what is the mechanism of its opening.

For stenosis to occur in the hypertrophied pylorus of Cautlet there must be spasm.

Hypertrophy of the pylorus has not been reported in an infant who has died of another disease and who has not suffered from symptoms of obstruction and, until one is described I am unable to see why spasm should be so peculiarly selective as to be grafted on a muscle which, although hypertrophied, is yet always normal with the possible exception of enlargement of its fibres.

These arguments apply also to Murray's and Ibrahim's theories. Ibrahim thought from the examination of three premature children living several weeks that the pylorus had a greater relative amount of muscular tissue in the 7th and 8th months than at a later stage of development and/

and that this condition persisted after birth.

Cunningham (Ibid. p.24) found the quantity of muscle if anything, smaller.

It may be objected that cases of pyloric hypertrophy may occur and not show symptoms until adult life.

Maylard)Lancet Dec.17th 1904, p.1709) has reported a case which he calls "Congenital Hypertrophic Strenosis in the Adult".

The man had had symptoms for three years. He died about $1\frac{1}{2}$ years after operation and microscopically the muscular coat of the pylorus was well developed and hypertrophied. There was also chronic gastritis.

If Maylard intends this case to be accepted as a "Congenital Hypertrophy of the Pylorus" showing no symptoms until adult life, I would point out the greater possibility of the hypertrophy being due to the gastritis without assuming there was ulceration.

No case of pyloric hypertrophy has been described in the adult without an explanation of its origin in some other gross pathological lesion. The objection has been raised to the theory that the disease is a congenital aberration that malformations elsewhere are not associated with the pyloric hypertrophy. I would point out/

that abnormalities as a rule arise from arrest or deficiency. (In the literature I find only five examples of associated abnormalities to which I can add one, a case of patent foramen ovale.)

The theory of Lobker that the increase of muscle is a tumour formation, is not feasible.

A myomatous neoplasm has its fibres arranged in a non-functionating, heterodox manner; while in pyloric hypertrophy the myomatous increase, retains the functionating, orthodox histological arrangement.

As to Bernheim-Karrer's view that the stenosis is caused by the hypertrophy, I have already shown that there is no true mechanical obstruction offered by the hypertrophy and, in addition, on post-mortem examination the pyloric lumen is often as large as the normal. Again if the hypertrophy by itself produced obstruction, we should not expect intermissions in the symptoms such as vomiting and constipation.

We had better here consider the theories of the hypertrophy being compensatory and arising from anatomical aberrations.

First, as regards the narrowing of the lumen primarily, or secondarily, by disease, I have shewn that there/

there is no narrowing. There is no absolute measurements for the lumen of the normal pylorus, but in face of Burghard's assertion and when I mention that in two of my cases the lumen admitted a lead pencil with ease, and in another case, treated to a fatal conclusion in the Glasgow Royal Infirmary, it was the diameter of a No. IX catheter, we must accept as proved the view that there is no congenital narrowing structurally.

Observers, who have thought that there is a true narrowing of the lumen, have possibly been misled by examining specimens preserved in formalin. None of my specimens preserved in formalin retained the permeability of the pyloric canal. Birmingham (Journal of Anatomy and Physiology Vol.XXXV p.46 and 47) found that in contracted stomachs in formalin, the normal pylorus was closed and Cunningham (Ibid. p.16) says that this closure is almost invariably, no matter what the condition of the pyloric canal may be.

Pitt's suggestion that there is a shortening of the duodeno-hepatic ligament causing flexion of the intestinal canal so producing obstruction and then compensatory hypertrophy, is fascinating. I have found no shortening of the ligament or mesentery as I have mentioned/

mentioned in the Section on Morbid Anatomy. If there occurred this shortening, we would not expect the intermission in the disease which occurs on change of diet etc., and the pyloric tumour would not be so palpable, and would not be found so close to the umbilicus.

The position of the pylorus and any flexion present after death is no criterion of abnormal position and flexion in life.

We had better rule out here the theory that the hypertrophy arises from spasm of the pylorus set up by reflex irritation from phimosis.

In the discussion of the theory that hypertrophy is caused by spasm, two questions must be answered.

Does spasm occur and can this spasm produce hypertrophy of the muscle.

Simple pyloric spasm in infancy is a well accepted entity.

The peristaltic restlessness of the stomach of Kussmaul, the anti-peristaltic restlessness described by Glax, Cahn, and the case reported by Bentegue (quoted by Einhorn, Diseases of Stomach p. 493) of a man with spasmodic contraction of the pylorus following Kerosene swallowing cured by exploratory operation, prove the occurrence of disordered motility of the stomach and spasm.

Moynihan (Duodenal Ulcer 1910 p.105) found spasm of the pylorus in two cases operated on under local anaesthesia only. Eve (Lancet, June 27th 1908 p.1824) has found a spastic contracture of the pylorus in three adults at operation.

Hawkins (B.M.J. Jan. 13th 1906, cases 2 and 3) has proved the occurrence of spasm in the intestine and in his Case No. 3 there is proof that there may be hyperexcitability of the intestine.

Cannon (American Journal of Physiology Vol. 1 1898, p.369 and 370) also observed that spasm, or at least very strong contraction seemed to occur in the pyloric antrum when solid particles approached the pylorus so that these particles were gripped.

Then the other question arises, can spasm from whatever cause arising, produce hypertrophy, and this again can be answered in the affirmative.

Kemp (Diseases of Stomach and Intestines pp.219 and 272) Boas (Diseases of Stomach 1907 p.505); Einhorn (Diseases of Stomach p.38); Habershon (Diseases of Stomach p.407 and Med. Soc. Trans. Vol.XXVI p.210) agree that pyloric hypertrophy may follow spasm produced by gastric ulcer.

Boas (Ibid p.505) and Einhorn (Ibid. p.185) say

it/

it may follow spasm in cases of chronic gastritis and Hemmeter also (Diseases of Stomach p.615) cites a case where hypertrophy of the muscle was found on post-mortem. examination.

These authorities cited prove that hypertrophy in the adult can arise from spasm and we should remember that smooth muscle seems to be prone to functional hypertrophy. The greater relative and more rapid development of hypertrophy in the infantile pylorus would result because it occurs in a growing organism. As Coats (Manual of Pathology 5th Ed. p.259) puts it in his general reference to Hypertrophy "The effect will be greater when the stimulus is applied during the period of normal growth than after the state of maturity has been reached."

It may be objected that as spasm occurs in children to a mild degree, there should be intermediate stages of hypertrophy of the pylorus. There may have been intermediate stages of hypertrophy but the hypertrophy would have been so reduced that the thickened pylorus may have been mistaken for one in agonal contraction.

Cautley has raised the objection to the occurrence of pyloric hypertrophy from spasm, that no hypertrophy occurs in other sphincters from over-action

Goodsall/

Goodsall and Miles (Diseases of Anus and Rectum 1905 Vol.II p.261) mention the occurrence of hypertrophy of the external sphincter and (Vol.1.p.213) say that the sphincteric muscles become hypertrophied from over-exercise and excessive strain.

It has been objected that as the symptoms pointing to hypertrophy appear soon after birth, therefore, there is not time for such great hypertrophy as has been found on post-mortem, to develop.

It does not follow, because the symptoms appear soon after birth, that the hypertrophy in all cases is also present at birth.

There may be spasm to begin with and the hypertrophy may develop later. In the case mentioned by Goodhart and Still (Diseases of Children p.143) which developed at five months, there may have been no hypertrophy at birth. Dent according to Cautley (B.M.J. Oct.13th 1906 p.941) has met with an example in a 7 months foetus. Ballantyne does not mention a foetal example.

We must take it for granted that the hypertrophy is present, in the vast majority of cases, at birth and develops in foetal life.

It/

It should be remembered that birth is not the beginning of life that it is only a stage in life and that it is quite possible, nay, most probable that the stomach functionates in uterine life. From the vertical fusiform bulging the fore-gut the stomach rapidly develops and already by the 5th or 6th week the adult form as we know it, is clearly indicated, so there is ample time for hypertrophy to occur in the pylorus in utero, if the necessary stimulus is applied. The fact of the presence of urea in the liquor amnii, and that the child may urinate soon after birth is no proof that the child swallows liquor amnii in utero but hairs and vernix caseosa could not be found in the intestinal canal if the stomach did not functionate, and if the foetus had not swallowed them with liquor amnii.

Ballantyne observes "Of the swallowing of "liquor amnii there can be no doubt" (Ante-Natal Pathology Foetus p.153, also see page 161).

Hyperacidity of the gastric juice has been advanced as the cause of spasm. Despite Knopfelmacher, (quoted by Cautley, British Journal of Children's Diseases May 1908 p.181) having found hyperacidity and my own results, in face of the analyses of Muller and Wilcox in five cases of Hypertrohic stenosis (LANCET, Vol. II 1907 14th Dec. p.1671) finding HCl. variable, of

Fenwick/

Fenwick (Disorders of Digestion in Infancy p.316) who found it normal and of the cases of Congenital stenosis in adults reported by Russell (B.M.J. July 11th, 1908) by Maylard* (B.M.J. July 11th 1908) where there was no hypertrophy ~~by~~ but hyperacidity, we must dismiss the idea that hyperacidity can cause hypertrophy. (I can find the report of no case where simple hyperchlorhydria produced hypertrophy). Lambert and Foster (Journal of Med. Science Vol.CXXXIV p.335 to 349) give cases where with increased HCl. there was no hypertrophy at operation.

When we remember that Muller and Wilcox (Ibid. p.1671) found ferment activity usually high and that the coagula are usually large denoting increased secretory activity, it suggests that the increase of HCl. is not caus~~ed~~al but that it is compensatory.

Theeffect of the increased HCl. and ferment activity might be to increase the digestive powers of the stomach and so prepare the food more thoroughly for egress through the stomach.

Thomson's Theory (Edin. Hosp. Reports, Vol. IV 1896 p.122) of the origin of the hypertrophy from spasm from faulty development of the nervous mechanism regulating/

*- Mr Maylard did not in his article, mention anonums of HCl but he informs me he found it variable

regulating contraction and relaxation of the pylorus is fascinating but "functional" is a word used to cloak deficient knowledge of pathology and "functional" diseases are decreasing and will continue to decrease in number in inverse ratio to our recognition of etiology.

He assumes that the disordered function begins in utero and evidently the hypothesis is that inhibition in the circular layer of muscle does not occur while the longitudinal layer contracts, pulls on the tonic or spastic circular layer with the result that a sort of vicious circle is set up and the over-action of the opposing layers of muscle causes hypertrophy in them.

The theory receives some support from the series of cases where a neuropathic family history has been thought to be present, from the few instances where more than one case has occurred in a family, from the fact that the higher and later nervous system - the last acquired - is the first to suffer functionally and from the possibility of some lesion occurring in a nerve with such a long course as the vagus.

I am of the opinion that Shattock unwittingly came nearer the truth than he dreamed of, but, instead of the hyperaesthesia or hyperexcitability - for want of a better name - being on the stomach side, I am persuaded that/

that it is on the duodenal side of the pylorus.

We must first consider the physiology of digestion.

Pavlov (Work of the Digestive Glands Translated by Thompson 2nd Ed. p.187 et seq.) has shown that acid chyme passes intermittently through the pylorus into the duodenum where by a reflex action it stimulates contraction of the pylorus and so prevents egress of more food until, the acid being neutralized, the pylorus relaxes and allows a further escape of food from the stomach.

The neutralization of the acid chyme takes place by an increased flow of succus entericus, pancreatic secretion and of bile.

We are not here interested in the question of whether it is the acid in the duodenum or secretin and the elusive prosecretin which is the excitant of pancreatic juice (Pavlov Ibid. p.132 to 137) or whether they act together or whether the acid acts by stimulating secretin or pro-secretin.

Pavlov (Ibid p.187) has shown that a solution of sodium bicarbonate introduced into the stomach may be kept there indefinitely by injecting into the duodenum gastric juice. He has also shown (Ibid p.187) that the passage of acid solutions out of the stomach is markedly slower in dogs with a pancreatic fistula. These two effects are due to/

to closure of the pylorus by stimulation of the duodenal reflex.

According to Hedenhain (quoted by Tigerstedt Text book of Physiology p.264 trans.) and Pavlov (Ibid. p.112 and 113) water is an excitant of the gastric juice.

When we recollect that duodenal ulcer is usually close to the pylorus (Guthrie Rankin B.M.J. July 23rd 1910 p.181) that duodenal and gastric ulcer occurs in infancy and have been described as occurring on or before the 15th day (Dreschfield in Albutt's System p.556 and Fenwick Disorders of Digestion in Infancy p.288) and even at 30 hours (Path. Soc. Trans. Vol.XXXII, p.79) and that duodenal ulcer has probably only to be looked for that its greater frequency may be recognised, and that melaena neonatorum frequently occurs without any visible pathological change, it suggests that the duodenum is perhaps more prone to disorders in infancy than we think.

Remembering that water excites a secretion of gastric juice and that liquor amnii is swallowed by the foetus we have a secretion of HCl. in utero. This HCl. passes into the duodenum stimulates contraction of the pylorus and secretion of the pancreatic juice and secretin, then is neutralized with the result that the pylorus dilates.

But/

But remembering Pavlov's experiments on the dog with the pancreatic fistula and his experiment where gastric juice was trickled into the duodenum already mentioned, and remembering that the great function of the pylorus seems undoubtedly the protection of the duodenum from the acid chyme of the stomach as the reflex regulating the closure ofnthe pylorus, acts from the duodenum., assume some hyperaesthesia or hyperexcitability of the duodenum or rather some aberration or deficiency in alkalinity of the secretions poured into the duodenum, then the HCl would remain unneutralized in the duodenum, would stimulate spasm of the pylorus, and so would cause hypertrophy.

To put it in another way if, as suggested by Cunningham the opening of the pylorus is an active process caused by contraction of the longitudinal layer pulling on the inhibited circular muscle, it would only require a deficiancy of alkalinity in the duodenum to produce spasm in the circular muscle while, at the same time, the longitudinal layer would be contracting, so there would be an antagonistic muscular action, not nervous in origin as Thomson thinks, but chemical and due to chemical defect on the distal side of the pylorus. I see no reason why the digestive glands should not functionate if uterowhen, in all probability, the thymus, thyroid and suprarenals do.

It/

It may be suggested that the deficient alkalinity of the duodenum would persist and prevent a cure by medical means.

To this I would reply there is a wide limit to the vis medicatrix naturae and that it is not impossible there occurs a compensatory increase of the other secretions so neutralizing the deficiency of the perverted one. Then the spasm disappears following on the removal of the cause the hypertrophy remains - maybe to disappear ultimately - and the circular muscle acts normally despite its bulk.

The occurrence of a chemical compensation receives some little support from the sudden improvement which occurs in some of these cases and even in the cases ending fatally where there is a cessation of vomiting with diarrhoea, is it not possible that this compensation occurs that a cure of the spasm results but that chyme is poured into an intestine unable by disease and disuse to deal with it, enteritis results, and the child is overwhelmed.

I am persuaded that the cause of the spasm, which produces the hypertrophy, is to be found on the duodenal side, and we must wait on an extended knowledge of the chemistry of the alimentary canal and its perversions before an absolute elucidation of the problem can be/

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be secured.

The occurrence of acetonuria or rather hyper-acetonuria in my cases does not seem to be suggestive save, maybe - to fire wildly - of some disorder of metabolism, or of secretion.

According to Mann (Phsiology and Pathology of Urine 1908 p.213) it occurs in ordinary gastric disease and occurs constantly in inanition.

It may be suggested that it hastens the exit of cases which end fatally and if, as has been said of the acidosis in cyclical vomiting where acetonuria is one of the features and where the acidosis is said to produce the vomiting, this acetonuria increases the vomiting, it is an indication for appropriate treatment.

D I A G N O S I S.

DIAGNOSIS.

The diagnosis of the disease presents great difficulty in the early stages and in the absence of definite symptoms, if marked peristalsis is absent and if no pyloric tumour is palpable, the diagnosis cannot be assured.

Surgeons have been known to operate where no hypertrophy was present.

The case reported by Hall (Cautley Diseases of Children p. 261) seems to be an example.

The child had been vomiting from birth. At seven months gastro-enterostomy was performed: the child died and section showed gastritis with a plug of mucous blocking the pylorus.

In, at least, two cases intussusception has been suggested as a diagnosis. (Guthrie's Case Reports of Society for Study of Disease in Children Vol.III. p.3) (Park West quoted by Clogg Practitioner 1904 Nov. 4th, p. 634).

Diagnosis is founded upon:-

1./

1. Age
2. Vomiting
3. Constipation and character of stools
4. Visible peristalsis
5. Dilatation of Stomach
6. Palpable pyloric tumour
7. Marasmus.

With the exception of the pyloric tumour all the other signs may be present in other diseases and the tumour may be simulated, it is said, by enlarged glands, or by a contraction of the pyloric antrum.

As to the age: if a child, within the first year of life, is suffering from any of the symptoms or presents any of the signs tabulated, the possibility of the disease should be remembered.

This possibility would be increased if the child showed any of these signs and was in the first few weeks of life.

It is very improbable that the child, if aged 8 months and over, would be suffering from Pyloric Hypertrophy.

No/

No weight can be attached to the character of vomiting, unless it is unmistakably projectile, as all kinds of vomiting occur in this disease.

If vomiting is forcible it is decidedly suggestive; otherwise the importance of vomiting lies not in its character but in its persistence.

Constipation, as I show in Chart 1, is not always, although usually, present.

The frequency of enteritis in the later stages should be remembered.

The stools, if small, consisting of mucus and containing little faecal matter, point strongly to Pyloric Hypertrophy, but, at the same time, it should be noted that wide variations from this general rule occur.

Visible peristalsis of the nature described in the Section on Symptomatology may be said to be pathognomonic of obstruction at the pylorus and this obstruction will probably be due to Pyloric Hypertroply.

Slight peristalsis may be simulated by contraction of/

of the abdominal wall as the child squirms, but, to the careful observer, the difference should be evident.

A slight degree of visible peristalsis may occur in comparatively healthy babies, more especially if they are bottle-fed.

The transverse colon may show peristalsis though in the reverse direction to that of the stomach: an unusual arrangement of the colon, so that the wave passes from left to right and still down the viscus, may cause difficulty (Case mentioned in Lancet 1905, March 11. p.634).

Dilatation of the stomach is a late development of the disease and also may occur in artificially fed infants.

The dilatation, to be of any importance in diagnosis, should be permanent, not variable.

The sign, which clinches the diagnosis, is the palpation of the pyloric tumour. Opinions as to the palpability of the tumour vary greatly (See Symptomatology)

I believe that, sooner or later, if emaciation of a moderate degree is present and if relaxation of the abdominal wall is secured, it should be possible to palpate the pyloric tumour definitely in all cases.

The/

The degree of emaciation need not be so extreme as to be a contra-indication to operation. If, granted emaciation and relaxation of the abdominal wall, the tumour cannot be felt then the case is not one of Hypertrophic Pyloric Stenosis.

Without going so far as Still (Lancet 1905, March 11, p.633) who considers that diagnosis cannot be made in absence of marked visible peristalsis and thickening of pylorus, I would say although I believe the tumour will be felt if diligently searched for, that a diagnosis may be made from marked visible peristalsis alone.

There is just a possibility of enlarged glands being mistaken for the pylorus.

I have not seen a case of definitely palpable abdominal glands in an infant.

It is possible that in a case of marasmus the head of the pancreas might be thought to be the pyloric tumour. In my case, already mentioned, the pyloric tumour could be slipped about under the fingers while the head of the pancreas seemed fixed.

It/

It is possible that information might be secured from the use of Bismuth meals. No work in this branch seems to have been done, and I was deterred from investigating it by a belief that, to some children, Bismuth in any form and in any but small doses is exceedingly dangerous.

Marasmus does not seem to be present in every case. Nicoll (B.M.J. 1904, Oct.29, p.1148) mentions a case where diagnosis of Pyloric Stenosis was repeatedly dismissed during life owing to absence of marasmus.

Post-mortem, pyloric hypertrophy was found. The case is exceptional and one would be justified in dismissing the possibility of pyloric stenosis in a similar child.

The disease with which pyloric hypertrophy is most usually confounded is pyloric spasm: with this I include gastritis which, with a narrow canal and oedema, might produce obstruction.

Pyloric spasm and pyloric hypertrophy in their earlier stages and before marasmus is established may be identified in their symptoms.

We/

We cannot distinguish between the characters of vomiting in the two diseases: the movements of the bowel, although constipation is much less likely to be present in Pyloric spasm, may give us no aid in diagnosis.

If peristalsis is slight it may be either affection. Little help is gained from test-feeds.

HCl seems to be always increased in Pyloric spasm (Muller & Wilcox Lancet 1907, Dec.14th.) variable, most probably, in Pyloric hypertrophy.

I found free HCl acid present in two cases diagnosed as pyloric spasm. Four analyses in all were done in these cases and the test-feeds were withdrawn in 20 minutes.

However, there is little practical importance in distinguishing the two diseases, in the early stages at least, for treatment in both follows much the same lines and the results of treatment may establish the diagnosis of pyloric spasm from the fact that spasm usually yields rapidly to appropriate treatment.

Indeed, even if a diagnosis of pyloric spasm be made and the child recover the case may really have been one of slight/

slight hypertrophy.

Habit-vomiting would not as a rule lead to emaciation and peristalsis would probably be absent and if present would be slight.

Vomiting with gastric catarrh and constipation, arising from unsuitable food would be amenable to dietetic treatment.

Atresia and new-growth of the pylorus may be omitted as excessively rare.

Ashby & Wright (Diseases of Children 1905 p.58) report a case of congenital obstruction of the jejunum. The child lived for some months, and bile was present in the vomitus.

Had the stenosis been situated above the bile-duct papilla the case, by an observer who did not attach importance to the presence of a pyloric tumour, might have been labelled hypertrophic stenosis.

Any obstruction of the lumen of the œsophagus might be confounded with hypertrophic stenosis.

Observation would settle the diagnosis. In the former there would be no peristalsis visible and no pyloric/

pyloric tumour: vomiting would tend to occur immediately after swallowing food and finally a sound would prove the oesophageal obstruction.

Febrile diseases might cause doubt for an inconsider--able time.

P R O G N O S I S.

P R O G N O S I S.

Prognosis.

Observers differ greatly as to the mortality occurring in cases treated medically. Heubner (quoted by V. Starck, Zentralblatt fur Kinderheilkunde 1909, May 5th Vol.XIV, No. 5, p.155) claimed 19 recoveries in 21 cases and Feer (ibid) had six deaths in thirteen cases.

Starck himself claims eleven recoveries in twelve cases.

Hutchison (Clin. Journal Sept.9th, 1908) out of 14 cases treated medically in private, had 13 recoveries.

In the sixty cases of these observers there was a recovery rate of 83.3% which approaches closely to Koplik's estimate of 85%. (Diseases of Children p.516).

Ibrahim (quoted by Bendix, Madizinische Klinik 28th Nov. 1909 p.1815) had collected up to 1908, 232 cases.

He puts the mortality rates as follows:-

Mortality	for Medical treatment ...	46.1%
"	of 83 cases in Germany ...	22.9%
"	of Heubner's Cases -(21)	9.5%
"	for operative treatment (138)	54.3%

Heubner (quoted by Holt, Diseases of Infancy and Childhood 3rd Ed. p. 325) thinks the vast majority recover under/

under medical treatment and Hecker and Trumpp (Diseases of Children Eng. Trans 1907 p.375) are of opinion that complete recovery may occur in many cases in spite of most alarming symptoms. Ruhrah (Diseases of Infancy and Childhood 1905 p.125) thinks that "practically all cases die unless treated surgically.

Graetzer (Practical Pediatrics. Eng. Trans. p.65) says that even operation is usually futile. Rotch (Pediatrics 5th Ed. p. 772) puts the mortality rate under medical treatment at 80% and Nicoll in the "Practitioner" (Nov. 1910 p. 659 and p. 660) seems to suggest that for pyloric hypertrophy medical treatment is useless. Nicoll's meaning is somewhat obscure.

Voelcker (Clinical Soc. Trans. 1907 Vol.XL p. 108 et seq.) reports seven cases with two recoveries and mentions (ibid p. 118) that out of 39 cases treated in Gt. Ormonde St. Hospital 34 had died, a mortality of 87%.

Thomson had 4 recoveries in 17 cases treated medically.

Cautley (B.M.J. Oct. 13th 1906 p. 942) had seven cases with seven deaths.

Leaving out Voelcker's 7 cases, which may possibly have been included in his figure of 39, we get a mortality of 85.7% as opposed to the recovery rate, already mentioned/

mentioned, of 83.3%.

The figures, from which I have calculated these percentages, have been taken from representatives of the two opposing camps advocating surgical and medical treatment respectively, otherwise they are not selected but have been taken at random.

The discrepancy between the figures is too great to be explained by the results of treatment.

The recovery rate of 83.3% is explained when we recollect the probability of wrong diagnosis. It is probable that cases of simple pyloric spasm and gastritis have been included. As the diagnosis of the cases on which the mortality of 85.7% is calculated have been verified by post-mortem examination, we must conclude that it is at least correct for these particular cases.

As for operative treatment Stiles (B.M.J.12th Oct. 1906 p. 948), in 84 cases treated by operation found a recovery rate of 46.4%.

Rotch puts the mortality rate of operation at 47.7% and Nicoll, in 1906, about 50%. The last named seems now (Nov1l910) to be of the opinion that the recovery rate of operation is somewhat higher.

In any event the prognosis seems to be bad and

in/

in any individual case it is exceedingly difficult to prophesy the result.

One of the greatest aids in the formation of a prognosis is, I believe, the facies. If the facies gives evidence of toxaemia the outlook is bad but if, despite the vomiting and other symptoms, the child remains bright there is distinct hope. None of the cases I have seen die, was at any time alert, but, on the contrary each lay whining constantly, or was very drowsy.

I am persuaded that the condition of the alimentary canal is of great importance, of greater importance as a general rule than the frequency of vomiting.

Decrease of vomiting without retention is good prognostically but it should be remembered that frequently a few days before death vomiting decreases and often ceases.

Marked dilatation seems rarely to occur save in fatal cases.

If peristalsis disappears gradually and if this is associated with other evidence of improvement the child may receover.

Peristalsis may not be seen in the few days preceding/

preceding death due to, I think, loss of the motor power of the stomach. The size of the pyloric tumour is no indication.

The condition of the motions is some indication of an improvement. The stools approach more or less the normal. Enteritis if at all acute is usually fatal. Nervous symptoms, such as convulsions and tetany, usually foreshadow a fatal conclusion.

The weight seems of little value unless a steady increase occurs. I have previously mentioned a case of Nicoll's which died although the child remained plump.

Cooke (Lancet - 1908 Jan. 11. p.129) reports a case weighing 3½ lbs. which recovered.

The weight may show a slight increase, not due to retention, and child may be dying (See Chart II.Child was admitted 11th Oct., died 14th Oct.)

In few diseases is it so difficult to calculate the probable result and the cases, which seem most hopeless, may give most pleasant surprises.

It seems that cases can recover, show no symptoms then relapse. Nicoll (Practitioner,Nov.1910 p. 661) cites a case which was demonstrated as á dietetic cure.

Operation/

Operation had to be subsequently undertaken.

I have had experience of a somewhat similar case which, after two months, again developed symptoms. On the second occasion there was no peristalsis or palpable pyloric tumour and the symptoms rapidly subsided. The child developed a sharp attack of Broncho-pneumonia, when in Hospital for the second time, and made a good recovery.

See-fisch (quoted by Carpenter B. M. J. Feb. 8th, 1908, p. 323) has reported three cases which had to be operated on before they were twelve years old, and, on the contrary, Starck (Ibid p. 157) has had four cases well, five, five, three and four years after cessation of treatment.

It seems possible that if the cause of the spasm can disappear the hypertrophy would vanish and the future of the child would then not be prejudiced.

T R E A T M E N T

T R E A T M E N T.

It may be that with increased knowledge of the disease and of its treatment all cases will be retained by the physicians and the necessity of operative interference will disappear entirely.

At present treatment is largely empirical and is confined to the treatment of symptoms and sequelae.

In considering treatment we should always bear in mind the pathology or what we know of the pathology of the disease

The occurrence of retention, of dilatation and gastritis should always be remembered and the probability that spasm is increased by these complications. If spasm is increased vomiting will become more marked. No ideal diet has as yet been evolved and Nicoll (Practitioner Nov.1910 p.661) seems to be the only authority who puts any trust in any particular diet, and he is very half-hearted in his faith.

Most observers think that one food after another should be tried that no food is ideal and that if one does not benefit the infant recourse must be had to another.

One/

One writer Sheffield (Mod.Diag. and Treat. of Diseases of Children 1911 p.138) suggests that a food yielding large coagula should be given. The large coagula might, he thinks, mechanically dilate the pylorus and so produce cure. The action of the large coagula, on the contrary would be to cause increased spasm.

Cannon (Ibid. p.370) observed that the pylorus had a tendency to remain closed when hard particles arrived at the antrum. From this the ideal food is one which yields a small soft curd. Mother's milk is often out of the question in these days and recourse is had to cow's milk which should have its fat reduced so as to aid in the prevention of irritation of the stomach.

Fats have a tendency to undergo fermentation. Milk sugar should be added and sodium cibrate the former to supply the plade of fats to a certain degree and the latter to prevent formation of large coagula.

It is possible that, as long as the diet is simple and seems/

seems to be rational, it is desirable that the food should not be varied and that the infant should not be used as a test-tube.

Meltzer (quoted by Hemmeter Diseases of Stomach p. 662) advises a slightly larger amount of milk than normal in the first stage of disease. The idea is that this increased amount of food would stimulate the muscular wall of the stomach and so more nutriment would be driven into the duodenum.

I would say that the result of this overloading of the stomach would be to hasten the passage of the first stage and would lead to a greater degree of spasm with an increased obstruction.

Koplik (Ibid p. 516) advise that the breast should be given at long intervals and short nursings. This suggestion seems to have a better foundation than Meltzer's.

The best results seem to occur when the child is fed frequently with small amounts. Gardner (Lancet 1903 Jan. 10, p.100) fed his patient every 20 minutes.

It would seem better that the child should not be so frequently disturbed.

Feeding/

Feeding hourly is usually suggested and in quantities up to one ounce. At night the child should be fed two-hourly.

Batten's result, already mentioned, with nasal feeding is suggestive.

It seems as if in his child the act of swallowing stimulated the vagus and so spasm with vomiting occurred.

In Britain lavage is generally used. In Germany they seem to do as well without, if statistics prove anything. The number of times that lavage is practised varies, but speaking generally, it is recommended twice daily and if the child's condition improves it is very gradually and cautiously decreased, but is still employed for weeks.

The advantage of lavage is that it removes stagnating food from the stomach and any toxins that, arising from decomposition, may produce irritation and increase spasm.

If gastritis is present lavage alleviates the disease reduces oedema, and so increases the permeability or the possibility of permeability of the pyloric canal.

Nervous/

Nervous symptoms, tetany and convulsions, seem to be more frequent where lavage is not employed. (Case Lancet Aug. 19th 1905).

Lavage by aiding in preventing gastritis will help in the prevention of dilatation a sequela which, it is evident, will prejudice the child's chances of recovery under either medical or surgical treatment.

By preventing decomposition and entrance of a chyme irritating in character into the duodenum, the possibility of the development of enteritis will be lessened and the bowel will be kept in a condition more suited to deal with the chyme when the passage is established.

The advantage to be secured from the use of lavage far outweigh the disadvantages.

A catheter used in lavage has been known to break (Clin. Soc. Trans. Vol.XL). The possibility of a lung infection can be largely prevented by the use of a catheter of No. 10 - 12 size.

Lavage should be with normal saline solution of a temperature of 75 ^{o} F. or a solution of Sodium Bi-carbonate 5 grammes to 1 ounce. No more than 3 to 4 ounces depending onsize of the stomach, should be introduced, and care should/

should be taken that coagula do not block the catheter.

After washing out and before the catheter is removed a feed should be introduced.

I have not used lavage oftener than once daily in cases of hypertrophy stenosis. Some babies seem to dread it and in these cases it is more depressing than is generally thought.

In my second case the one which recovered, after washing with Sodium Bicarbonate solution I used a solution of Calcium Permanganate. I was led to use this by observing that in cases of subacute and chronic gastro enteritis with stinking stools it seemed often to be most efficaceous. It was useless in infantile diarrhoea.

Theoretically it has twice the disinfecting power of the Potassium Salt.

I suggest that it acts by its disinfecting power in the stomach and that a small quantity escapes into the bowel and continues its action there but to a much less degree. It is possible that its former use in anaemia was justified when the anaemia was due to bowel intoxication.

As to other treatment Bendix (p.1816) applies poultices to the epigustrum before meals and warm applications are often/

often used. He also advises frequent warm baths,

Enemata to supply the loss of fluid by vomiting, to clear the lower bowel occasionally, and for alimentation may be employed. Schwyzer (quoted in Trans. Med. Chir. Soc. London Vol.82, p.62) kept a patient alive for 14 days with rectal feeding and Cooke (Lancet Jan.11. 1908. p.128) gave only salt and water per rectum for 7-10 days. A "dummy" of cotton wool dipped in water was used. The child which weighed 3½ lbs. recovered.

Of treatment by drugs, I have little experience. Alkalis, in the hands of British observers has not been successful. They seem to be indicated, more especially in the presence of hyperacidity. Maybe the doses employed have not been large enough.

Heubner (Ibid. p.438) used opium and atropine; Bender (Ibid. 1816) opium and magnesium and potassium carbonate; Starck (Ibid. p.161) Valerian and in severe cases morphine hypodermically.

Oedems may occur and if due to the weakened condition may in addition to general trestment be treated with adrenalin and stimulants. The oedema is an expression of the child's general condition.

I have used Eau-de-mer of Dr. Quinton subcutaneously

but/

but with the exception of an evanescent stimulation I cannot say it benefited these cases. Lastly the weak condition of the child and the subnormal temperature should be remembered. and artificial means used to maintain the body heat. In my opinion the occurrence of diarrhoea is an indication rather for immediately reducing the quantities of food given than for drug treatment of the diarrhoea.

The consideration of surgical treatment and of medical versus operative interference I have retained for discussion in the Appendix.

A P P E N D I X.

A p p e n d i x.

Operative measures employed are -

1. Pyloroplasty.
2. Divulsion.
3. Gastro-enterostomy.
4. Gastro-enterostomy and Divulsion.
5. Pylorcetomy.

PYLOROPLASTY:- The advantages of pyloroplasty are that it restores the natural passage that only a very small portion of the abdominal contents need be exposed, and that the operation can be done rapidly and, if the lumen is not entered, aseptically.

The objections to pyloroplasty are that the lumen seems to completely block for 24-48 hours and vomiting during this time will tend to increase with the result that the scale may be turned against the child. It has happened that one of the folds has been removed to provide a more patent canal. I do not think there is any necessity for this removal and indeed it is bad practice for there will be an increased possibility of scar formation with resulting contraction. It seems to be possible that in a markedly elongated pylorus the wound required would be excessive/

excessive.

DIVULSION:- Divulsion seems rather crude. Here one requires to dilate the pylorus so that rupture of the muscular fibres occur. The amount of force required cannot be gauged accurately. It seems possible that, in a few days the pylorus simply overstretched might again contract and produce symptoms. Relapse has occurred necessitating gastro-enterostomy (Stiles Lancet May 28th 1904). (Mackay Intercolonial Med. Journal of Australia quoted by Clogg). Then the wall has been ruptured completely and the duodenum in two cases, at least, has been perforated. Divulsion would lead to as much soiling of the peritoneum as gastro-enterostomy.

GASTRO-ENTEROSTOMY:- Gastro-enterostomy, if properly performed and by an expert operator, seems to me to be the procedure approaching most closely the ideal.

The wound made should be large enough to allow for contraction in a dilated stomach. There does not seem to be after the first few weeks any greater tendency for bowel irritation to occur than in the other operative procedures.

Mikulicz (quoted by Clogg Ibid. p.652) reported a case of multiple duodenal ulcers in a child aet

2/

2 months following a gastro-enterostomy.

It would be exceedingly interesting to see, if in after life, the gastro-enterostomy wound closes and the pylorus becomes normal. It seems possible.

I have collected 44 cases of gastro-enterostomy 20 of pyloroplasty and 27 of divulsion, a total of 91 cases. I purposely refrain from burdening this Thesis with statistics of operation. They are of no use in proving anything when cases are so few in number. The mortality rate I may mention in these cases excluding cases dying after one week, was 40.6%.

The question of medical or surgical treatment in these cases is exceedingly difficult and it can never be said that a case recovering under surgical treatment would not have recovered if medical means had been employed. Medical cases are not selected, surgical cases are to a greater or less degree. Many cases come under treatment so late that little or no hope of benefit by any procedure can be entertained, I believe that operation should not be undertaken until the pyloric tumour is palpated. Hall (already quoted) and Scudder (Note B.M.J.19th Sept.1908) have operated on cases which were not hypertrophic stenosis.

The latter performed gastro-enterostomy on a child 7 days old. The infant recovered. In any

operative/

In any operative procedure given, treatment by medical means but without lavage, should be continued.

PYLORECTOMY:- It is not necessary to discuss pylorectomy, performed once with a fatal result, nor the combination of pyloroplasty and divulsion, save to mention that if the two be employed together, it seems that the result would be more certain.

Gastro-enterostomy and divulsion, I would say, is an unnecessary combination.

If a child is marasmic and continues to lose weight and is manifestly not improving, I would advise operation if the operator could be selected. Otherwise the chances of recovery are probably greater under medical treatment.

As time passes and technique improves, almost all these cases will be submitted to operation unless, in the meantime, some improvement in results of medical treatment, is obtained.

I am unable to understand why in the hands of Starck and Hutchison medical treatment has given such good results, unless they have diagnosed some cases wrongly.

C A S E S.

C A S E S.

1. I.G. Aet 12 weeks, 1st. child, Male. Admitted 11th. October, 1910. Died 15th October, 1910. 5 lbs. 14 oz. Baby healthy at birth. Vomiting began in second week and now occurs after every feed. Weight on admission 5 lbs. 14 oz. Pylorus palpable. Marked Peristalsis, Systolic murmur at base. On day of admission vomited thrice. Vomitee next day frequently. On 13th October had gained two ounces, weighed after wash-out. Lavage daily. Diarrhoea. Temperature on morning of 14th. 101°. 100° at 6 p.m. (See Chart II).

POST-MORTEM EXAMINATION - Marked thickening of pylorus. Folds marked. Stomach enlarged and wall thickened. Patent foramen ovale. Microscopically great increase of circular muscle. Longitudinal uncertain. Mucosa and submucosa thickened but may have been due to contraction.

2. M.G. $\frac{3}{12}$. Female. Illegitimate. Breast-fed for one month $\frac{9}{9}$. Admitted November 25th, 1910. Weight 5½ lbs. Vomiting in 3 weeks. Constipation. Vomiting frequently after meals. Five weeks before admission convulsions, which were frequent for 14 days. Two attacks since. Two miscarriages. snuffles at birth. Interstitial keratitis.

On 4th. December weight before washing out two hours after feed 5 lbs. 13 oz. After washing out 5 lbs. 11 oz.

Child/

Child slowly improved. Peristalsis was visible and tumour was palpable. Recovery. Free H Cl. present. Acetonuria. Readmitted with return of vomiting maybe due to unsuitable food. Soon recovered. During residence Broncho-pneumonia. (Chart I. 1st. residence).

3. G.W. Female. Admitted August 7th, 1910. Weight 6 lbs. 4 oz. Aet $\frac{2}{12}$. Vomiting since birth. Constipation established at 2 weeks. Mother noticed a lump moving across abdomen, one month before. Peristalsis marked. Antiperistalsis seen on one occasion. Pylorus palpable. On several occasions waves were distinctly more visible when child fell asleep. Constipation on admission. Stools became more frequent, but not definitely diarrhoeal. No vomiting in last 3 weeks of life. Died September 2nd, 1910. Lavage once daily. Free H Cl. present. Acetonuria. POST-MORTEM - Appearances typical, stomach dilated. Microscopically circular muscle and longitudinal muscle on lesser curvature hypertrophied. (See Text and Plate IX). Chart III.

4. R. C. Male. 1st. Child. Admitted July 24th, 1908. Weight 6 lbs. 4 oz. Vomiting at 2 weeks. Breast-fed for 14 days, then Benger's Food, Barley water, Nestle's Milk &c. Stomach dilated. Peristalsis visible. Pylorus palpable/

palpable. Collapsed and died 24th July.

POST-MORTEM - Pylorus enormously thickened. Longitudinal layer of duodenum hypertrophied. (Plate X).

5. J.W. Male. 2 months. 5th. Child. Weight 7 lbs. 12 oz. Admitted April 27th, 1909. Vomiting at 3 weeks. No peristalsis visible and no pylorus palpable. In last six days of life Temperature. No note as to condition of lungs &c. Died April 27th, 1909. Pylorus found typical on section.

6. J.B. Male. 10th. Child. Weight 8 lbs. 11 oz. Admitted August 20th, 1908. Has gone back for 3 months. Bowels opened every third or fourth day before admission. Breast-fed for three weeks. Lower border of stomach at umbilicus. Pylorus palpable. Marked visible peristalsis. During residence in last 10 weeks motions on an average passed thrice daily. Lavage

POST-MORTEM Lumen admitted pencil. Ileum with swelled injected mucous membrane and engorgement of vessels and lymph-glands.

7. B.K. Male. Aet. 5 weeks. Weight 7 lbs. Admitted September 14th, 1909. Vomited after breast. Bowels costive. Peristalsis visible and pylorus palpable. In last week of life bowels moved 4-5 times daily. Died October/

October 5th, 1909. No report as to whether post-mortem examination permitted.

8. J.M. Male. 10 weeks. Weight 5 lbs. 6 oz. Admitted 14th November, 1909. Vomiting frequent for 5 weeks before admission. No diarrhoea or constipation. 1st. Child. Peristalsis visible. Lower margin of stomach reaches below umbilicus. Pylorus palpable. Died November 24th, 1908. Bowels moved on an average thrice daily while in residence. No post-mortem report.

9. W.P. Aet. 9 weeks. Male. Weight 8 lbs. 12 oz. Admitted 1st. April, 1908. Vomiting with marked constipation since three weeks after birth. Peristalsis seen. Pylorus palpated. On 5th. April temperature rose to 104·8, and Child was collapsed. Bowels constipated for 2½ weeks of residence then became looser. In last 2 weeks of life bowels moved on an average 4 times daily. Died August 18th 1908. Weight on admission was 8 lbs. 12 oz. then dropped to 7 lbs. 12 oz. in 18 days. On July 7th. was 7-10 oz. increased slowly to 9½ lbs. on 14th August. Dropped to 9 lbs. on day before death. Temperature rose to 101·6° before death.

POST-MORTEM EXAMINATION - Pylorus typical. Admitted a pencil.

10./

10. ? ? - Female. 1st. Child. Death at 7 weeks 1911. Vomiting onset at 3 week. Delivered by Caesarean Section Pylorus palpable and Peristalsis seen once. Fed per rectum Head of Pancreas and body distinctly palpable. Died. Post-Mortem appearances typical.

11. J.G. 1st. Child. Male. Aet. 7 weeks. Mr. Pringle's Admitted 1st March, 1811. Vomiting for two weeks before admission. Bowels did not move for four days before admission.
4th March, 1911. - Posterior Gastro-enterostomy. Pylorus thickened.
21st. March, 1911 - Weight 8 lbs. No record of weight on admission.
19th May, 1911. Reported well with exception of whooping-cough. Chart IV.

12. M.S. - Female, Weight 6 lbs. 4½ oz. Aet. 4 weeks. Dr. Cowan's S.F. Aet. 2½ months. Admitted 10th August 1910. Peristalsis noticed once. Died 2nd. September 1910.
Dr. Cowan was away from home, and case was not diagnosed.
POST-MORTEM - Pylorus typical. Orifice admitted a No. IX. catheter.

13. In another case all that is known about the child is that it was a male and illegitimate. Died.

In 12 cases treated medically there was one which recovered. If we omit case I. and Dr. Cowan's undiagnosed case there were 9 cases which died despite treatment. The cessation of constipation of vomiting should be noted. Two children were illegitimate.

In 13 cases three were females and at least four cases were first-born.

In the case of several specimens, discovered unlabelled, there were no notes, and no clue as to the time of their residence in the Hospital could be discovered.

P L A T E S.

P L A T E S.

PLATE No. 1. shows dilated stomach.

Point of director's entry is situated above the pylorus.

" No.II. Dilated stomach.

The hypertrophied pylorus can be seen peeping out through tear in the duodenum.

" No.III. Stomachs with hypertrophied pylori.

" No. IV. Pyloric antra and entry to proximal end of pylorus.

Notice large fold disappearing into canal in specimen B.

A - whorled. B - Stellate. C - Irregular.

" No. V. In A is shown absence of fold.

B. pylorus which had only one fold

C. Pylorus which had three - two of which are seen in the photograph.

" No. VI. A. shows a normal pylorus - note rugae.

B. & C. show fusiform shape of pylorus.

" No. VII. A. is thickened cardia (observe the double arrow.)
Photograph enlarged.

B. Cervix uteri appearance formed by the hypertrophied pylorus projecting into the duodenum.
Photograph enlarged.

PLATE No. VIII. A. Micro photograph of section of pylorus and duodenum. In centre of lumen is a fold cut across. Mucous membrane thickened. Duodenal wall thickened, but striking point is the increase of the pyloric muscle.

B. Is a noraml pylorus, age as "A"

PLATE No. IX. Micro-photograph to show the hypertrophy of the longitudinal muscle of the pylorus on the quadrant corresponding to its greater curvature. To get the correct position x should be lowermost.

Section is transverse.

PLATE No. X. B. Shows the marked hypertrophy of the longitudinal muscle of the duodenum.

A. Normal duodenum for purposes of comparison. Age as "A"

Sections are longitudinal.

C H A R T S.

C h a r t s.

CHART I. Note frequency of vomiting.
Note weight on admission when peristalsis was marked and pylorus was palpable. Weight did not mount above weight on admission for 14 days. In 4th week a steady gain set in.
Bowels were never really constipated, illustrating that with hypertrophic pyloric stenosis there need not be constipation.

CHART II. No constipation in last three days of life.
Increase of weight.
The temperature often found at end.

CHART III. Vomiting frequent. Constipation for 1st 14 days then bowels moved the 3 - 4 times daily.
No vomiting in last 14 days of life. Pylorus became permeable.
Thermometer did not rise for six days.

CHART IV. Mr Pringle's case of successful gastro-enterostomy.

CHART V. Dr Cowen's case.
Persistent sickness. No vomiting during 1st sox days of residence. No increase of weight.

BIBLIOGRAPHY.

Bibliography.

1.	Batten.	Lancet.	December 2nd 1899.
2.	Gardner	"	January 10th 1903.
3.	Murray	"	January 24th 1903.
4.	Annotation	"	April 11th 1903.
5.	Stiles	"	March 28th 1904.
6.	Maylard	"	December 17th 1904.
7.	Morison	"	December 24th 1904.
8.	Still	"	March 11th 1905.
9.	Rob.	"	June 23rd 1906.
10.	Cooke	"	January 11th 1908.
11.	Clowes	"	August 20th 1908.
12.	Broadbent	"	December 19th 1908.
13.	Carpenter	"	June 26th 1909.
14.	Dent	"	" "
15.	Nicoll	British Med, Journal	April 23rd 1898.
16.	Paterson	" " "	November 3rd 1906.
17.	Nicoll	" " "	September 1st 1900.
18.	Gardner & Atkins	" " "	December 21st 1900.
19.	Cautley	" " "	May 31st, 1902.
20.	Campbell	" " "	June 25th, 1904.
21.	Cleveland	" " "	May 7th 1904.
22.	Scudder (Note)	" "	Sept. 19th 1908.
23.	Carpenter	" " "	1908 Vol.I.p.322.
24.	Hutchison	" " "	Ocotber 8th 1910.

25. Dent. Brit.Journ.of Children's Diseases. 1904 Vol.1.

26. Fisher & Neild. Bristol Med.Chir.Journal 1904 p.123.

27. Guthrie & Murray) Reports of Soc.for Study of Diseases in Children. Vol.III.

28. Thomson. Edin.Hosp.Reports. Vol.IV. 1896.

29. Williamson " Monthly Journal of Medical Science Vol I. 1841.

30 Shaw & Elting) Archives of Pediatrics. December 1904.

31. Nicoll. Glas.Med.Journal. Vol.65, 1906.

32. Peden " " " 1889. p.416.

33. Hunter " " " Vol.70 1908.

34. Thompson. Scot.Med. and Surgical Journ. 1897.

35. Cautley & Dent.) Medico-Chirurgical Trans.Lond. Vol 86.

36. Nicoll " " " Glas. Vol III 1902.

37. Meyer (Note) Practitioner 1899. p. 200.

38. Clogg " Vol.73, 1904.

39. Schwyzer, N.Y. Med. Journal. Nos. 21, 1896.

40. Meltzer " " Record. Aug.20th 1898.

41. Giles, Australasian Med. Gaz. 1904.

42. Scudder & Quinby) Jour. of Amer.Med.Assocn. May 27th 1905.

43. Garrod. Clinical Journal 1906 Vol. 28.

44. Still. Trans.Path.Soc.London. Vol. 50.

45. Pitt " " " " Vol. 63.

46. Fisk — Annals of Surgery. July 1906 Vol.44.

47. Thomson. — Clin.Exam.and Treat.of Sick Children. 1898.

48. Still. — Abbutt's System of Med. 1907 Vol.III.

49. Cautley. — Diseases of Children 1910.

50. Ashby & Wright — " " " 1905.

51. Goodhart & Still. — " " " 1905.

52. Robson & Moynihan — Diseases of Stomach 1904.

53. Cheyne & Burghard — Manual of Surg. Treat. 1906 p.281.

54. Koplik. — Diseases of Infancy & Children. 1910.

55. Pfaundler & Schlossman — Diseases of Children. Vol. III.

56. Starck. — Zentralblatt fur Kinderheilkunde May 1909.

57. Bendix. — Medizinishe Klinik 28th Nov., 1909.

58. Engel. — Deutsche Medizinische Woch. 22nd July 1909.

59. Heubner. — Die Therapie der Gegenwart. Oct. 1906.

60. Hammer. — Prager Medizin ische Woch 1899. Vol.24.

61. Davosky. — Caspar's Wochenschrift 1842 No. 7.

62. Fritsche. — Munch Med.Woch. Vol.86, 1899.

63. Stern — " " " " 24 1898.

64. Henschel. — Archives furkinderheilk. Vol.13 1891 or 2.

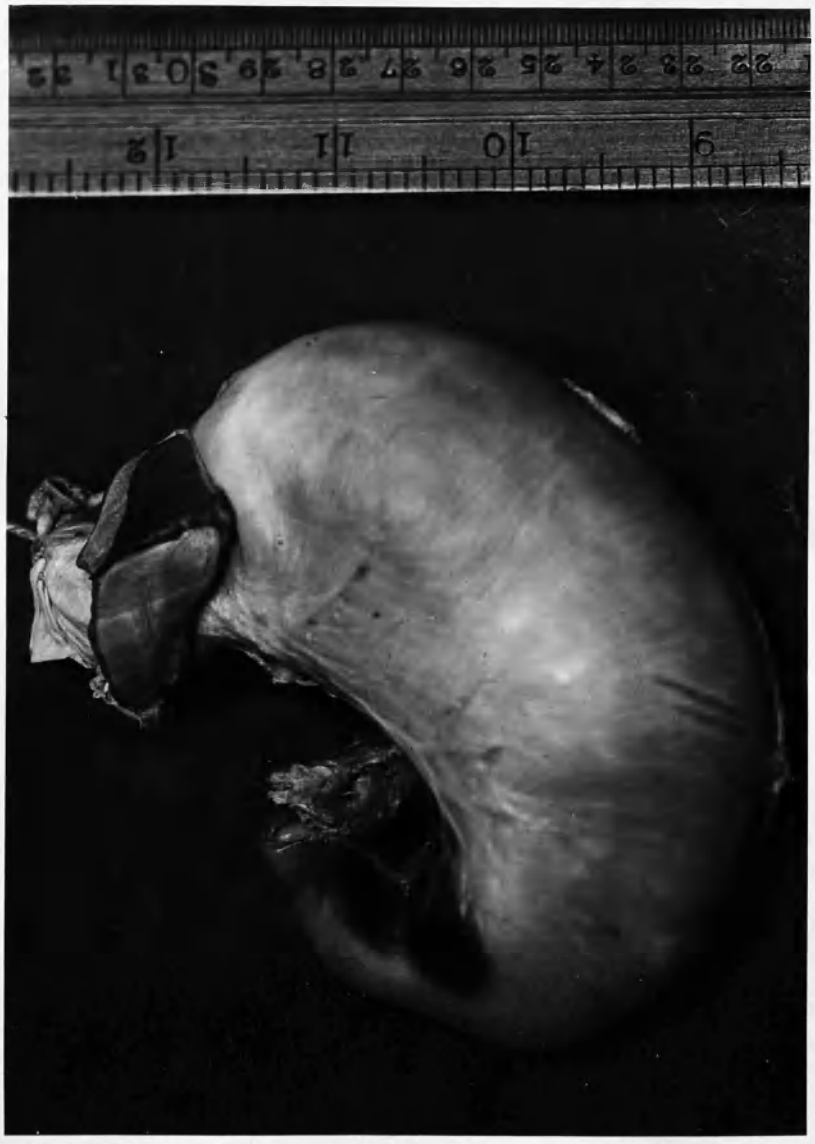
65. Hirschsprung. — Jahrb d Kinderheilk. 1888.

66. Finkelstein — " " " 1896.

67. Gran — " " " 1896.

Plate No. I.

Plate No. II.



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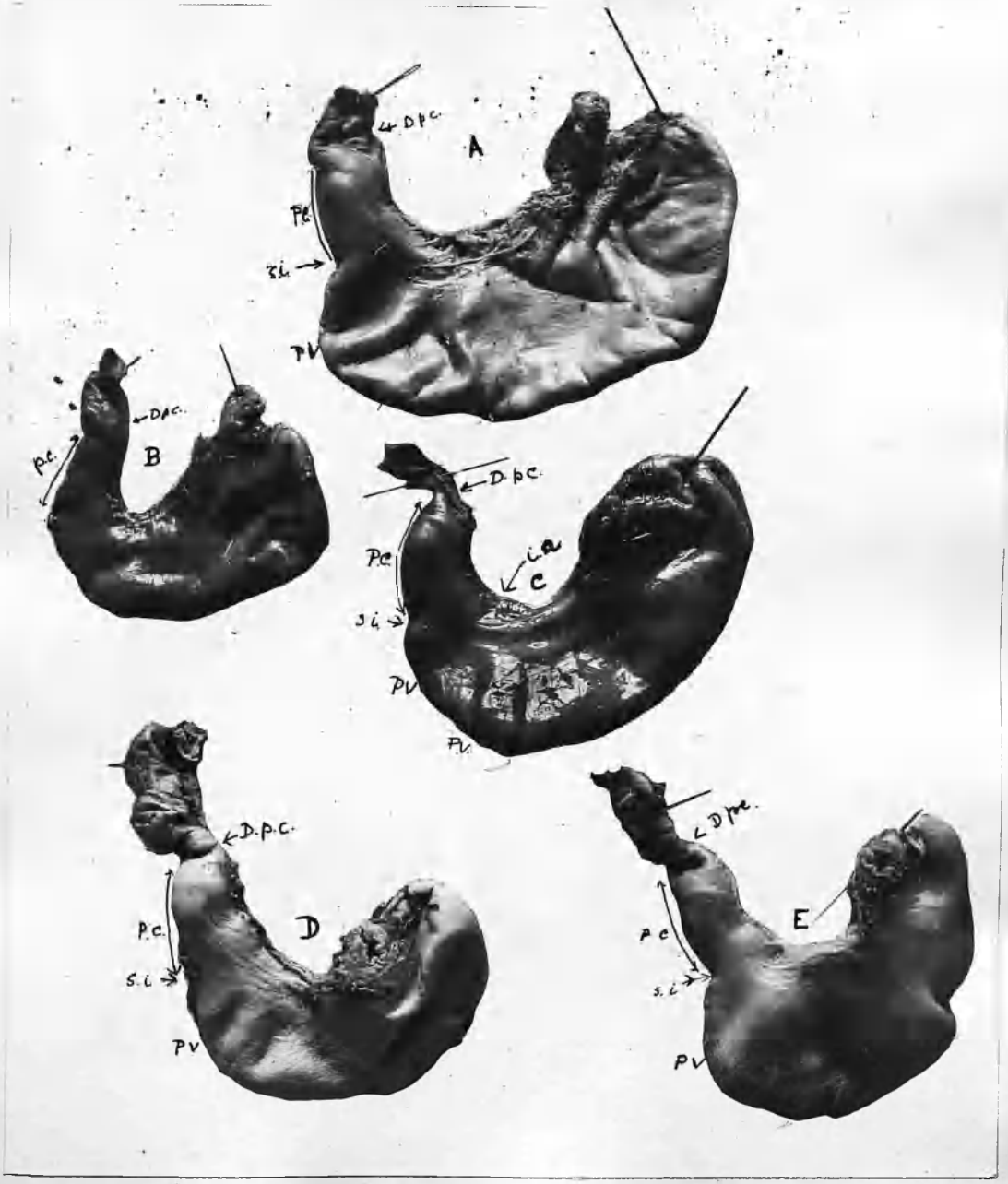

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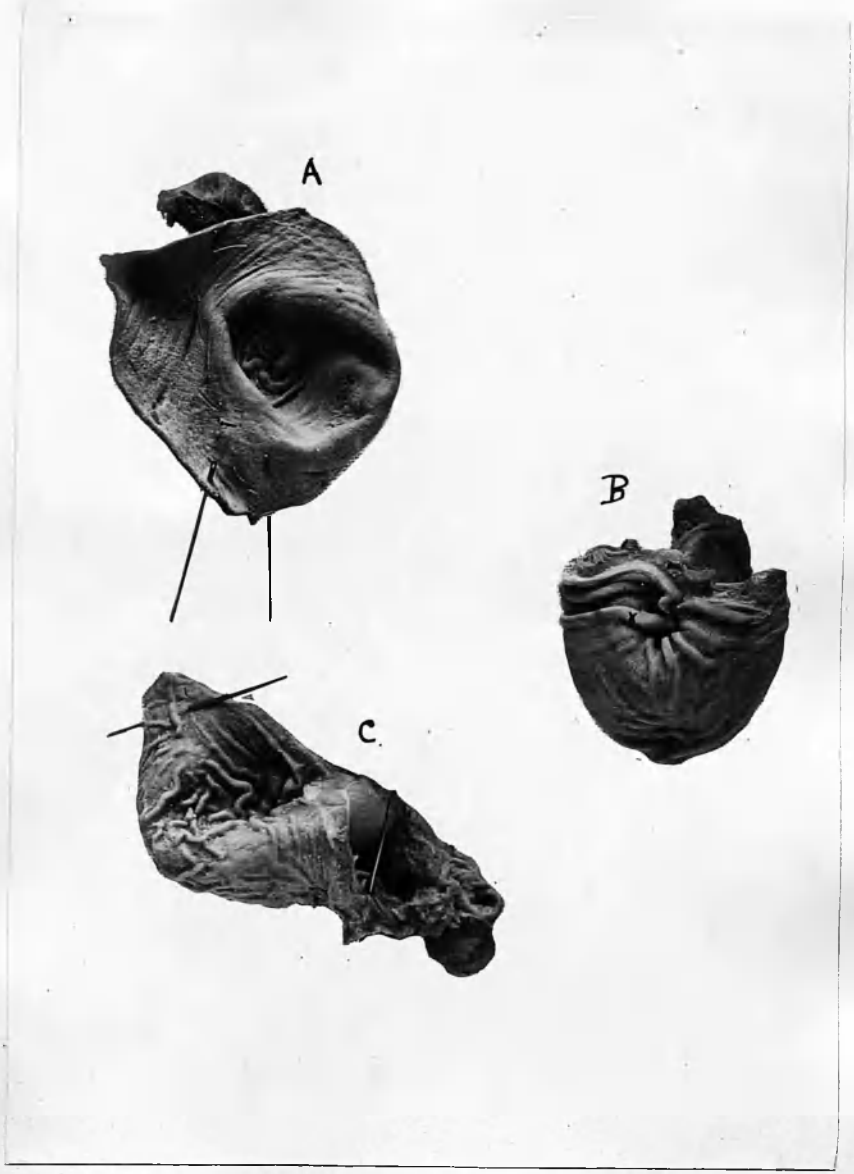

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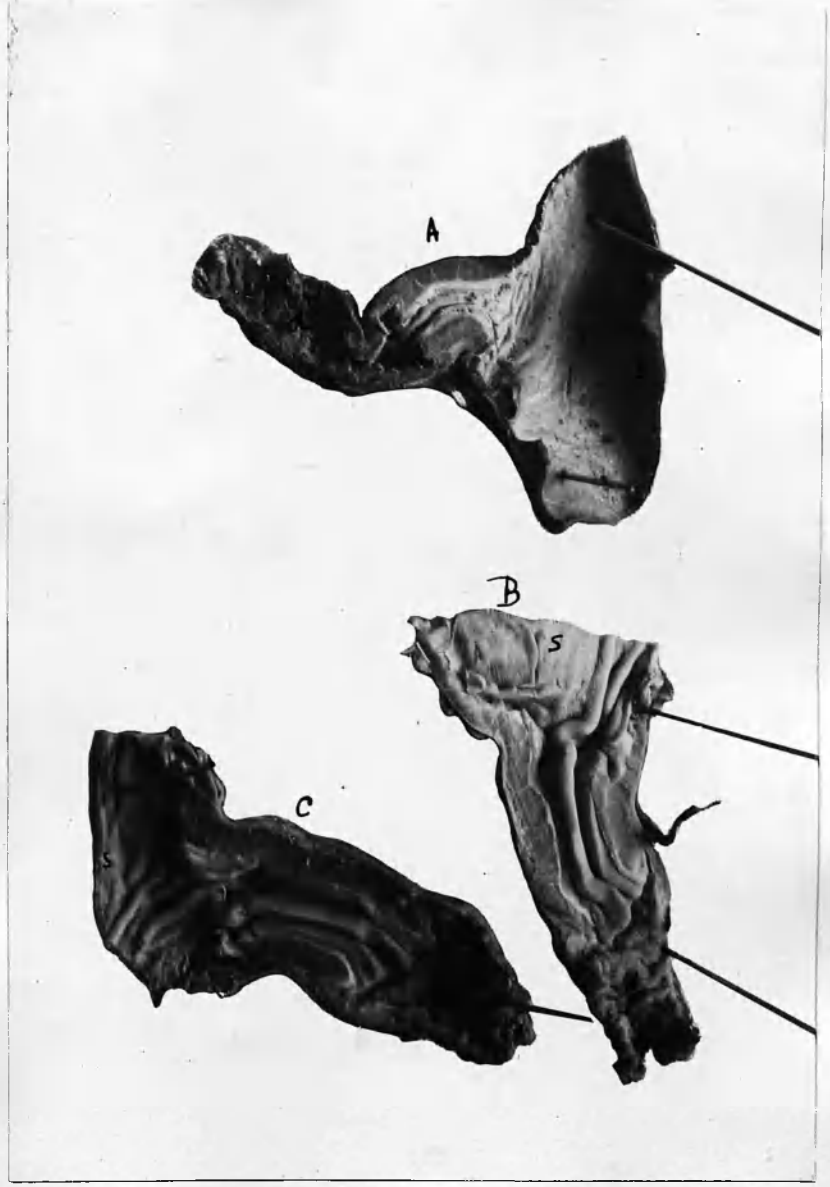

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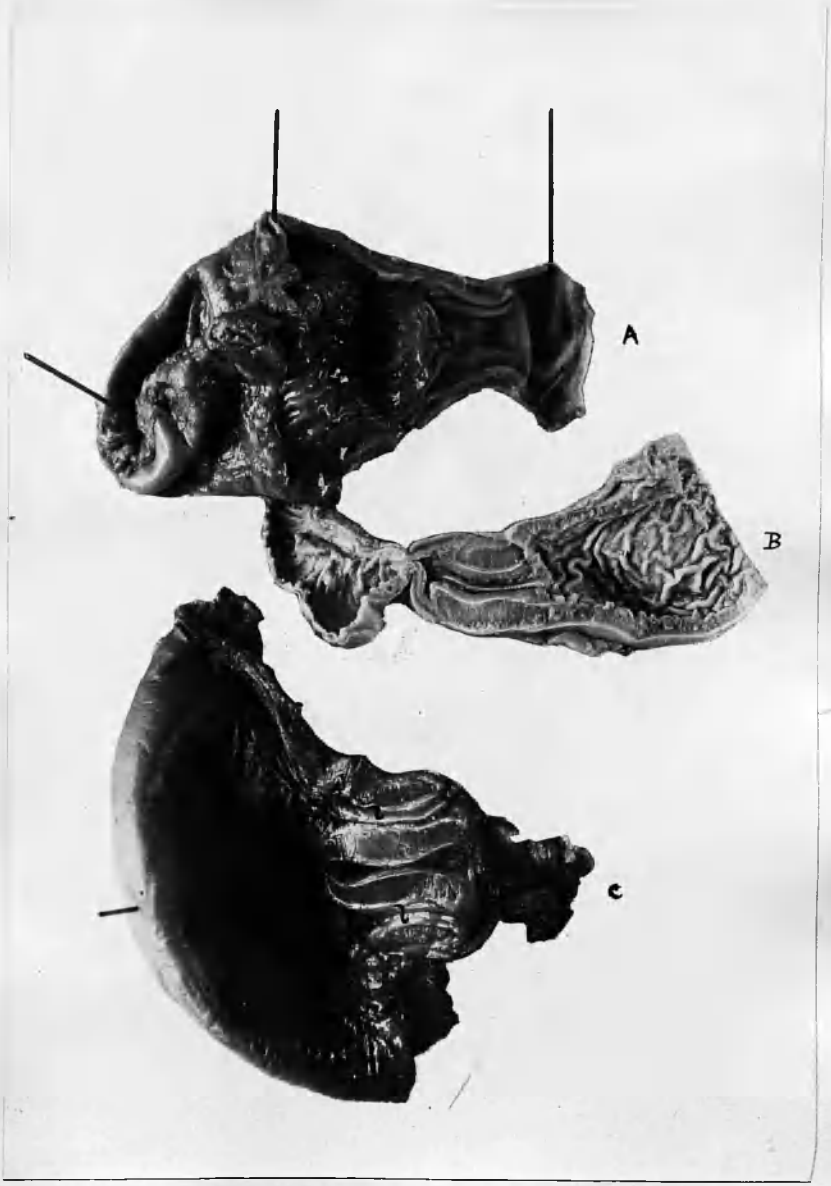
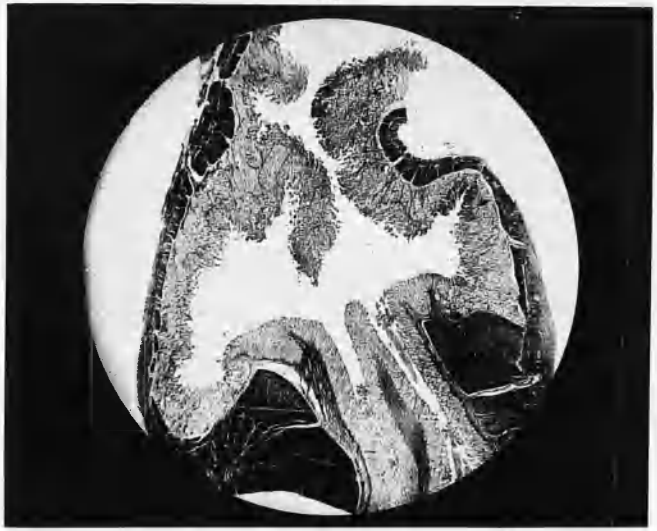


Plate No. VII.

Plate No. VIII.

A



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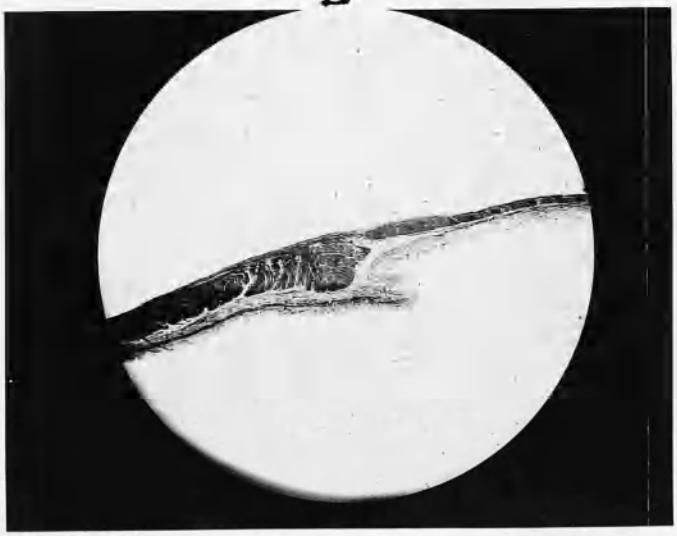


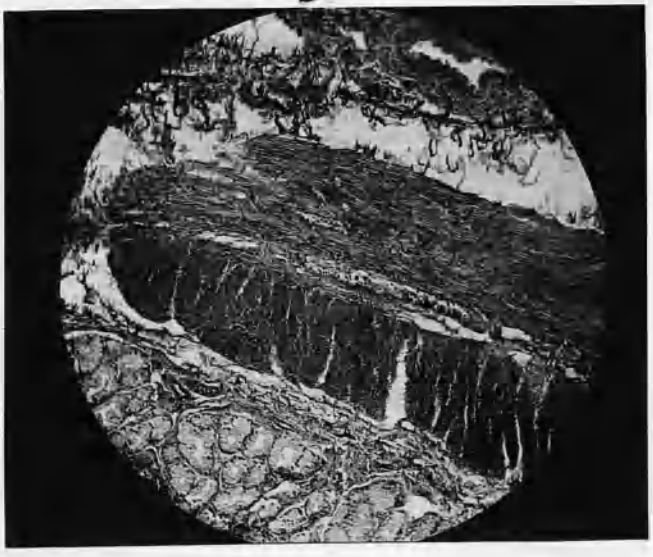
Plate No. IX.

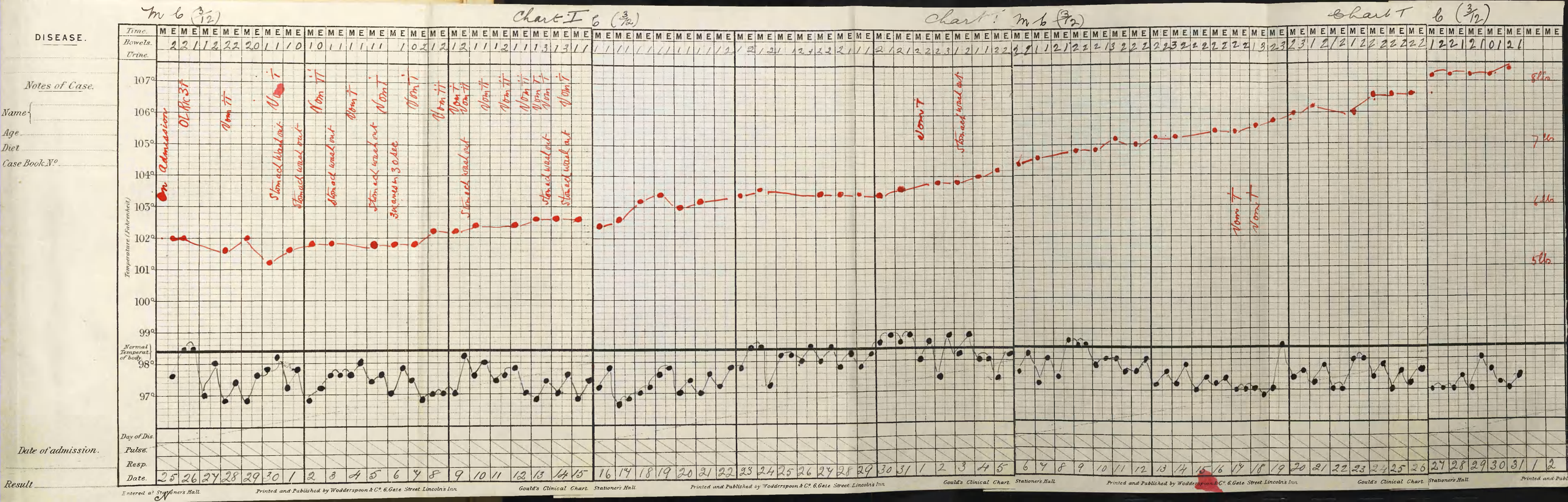
x

Plate No. X.

A

B




DISEASE.
Notes of Case.
Name
Age
Diet
Case Book No.
Date of admission.
Result
Time.
Bowels.
Urine.
Temperature (Fahrenheit)
Normal Temperat. of body
Day of Dis.
Pulse.
Resp.
Date.
On Admission
Vom T
Stomach washed out
Entered at Stationers Hall.
Printed and Published by Wodderspoon & Co. 6, Gate Street Lincolns Inn.
Gould's Clinical Chart. Stationers Hall.

IG (12 wks) Chart II

DISEASE.

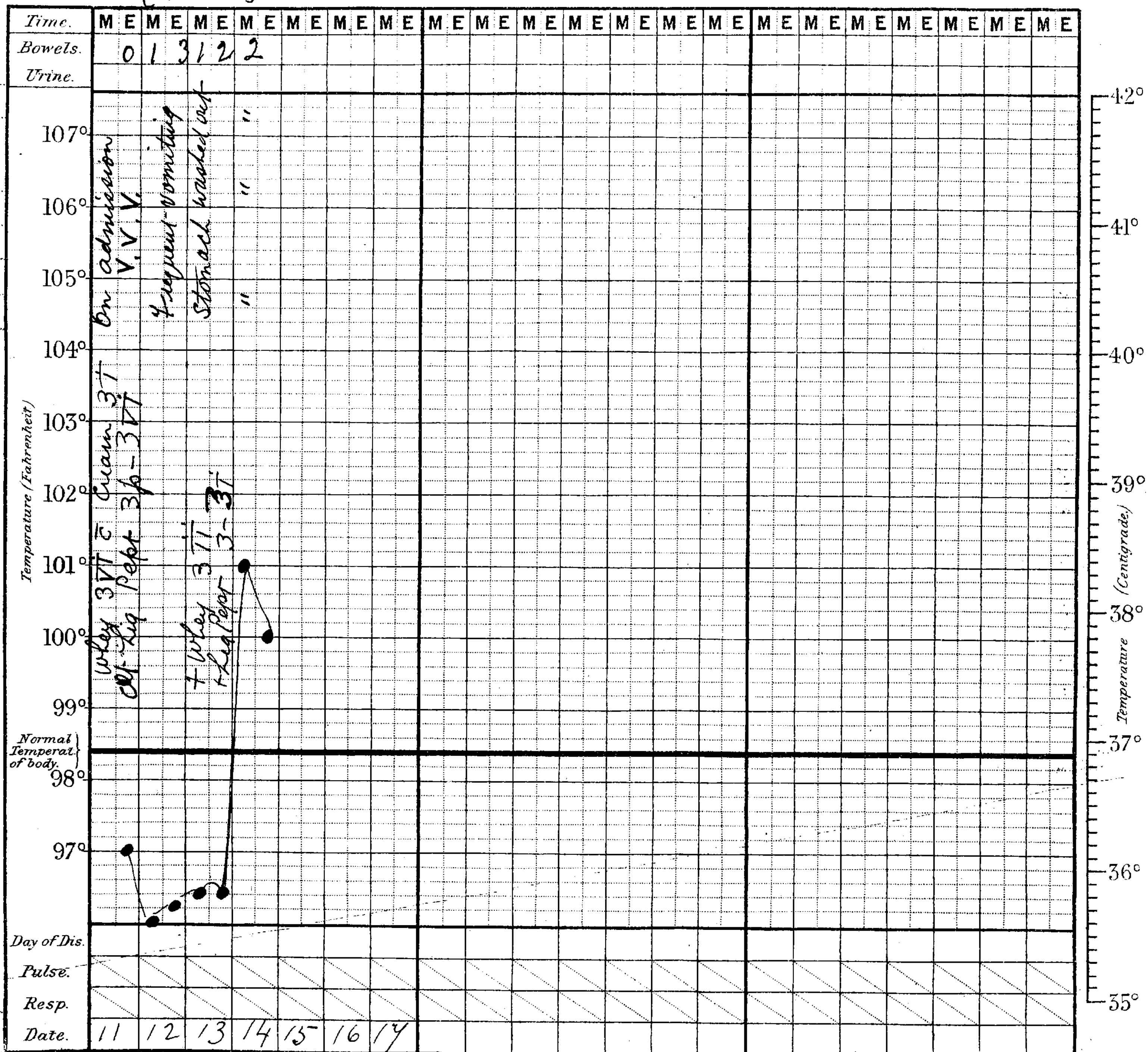
Notes of Case.

Name

Age

Diet

Case Book No.



Date of admission.

Result

Entered at Stationer's Hall. Printed and Published by Wodderspoon & Co. 6, Gate Street, Lincoln's Inn. Gould's Clinical Chart.

G W (3/12) Chart III W (3/12) Chart III

DISEASE.

Notes of Case.

Name

Age

Diet

Case Book No.

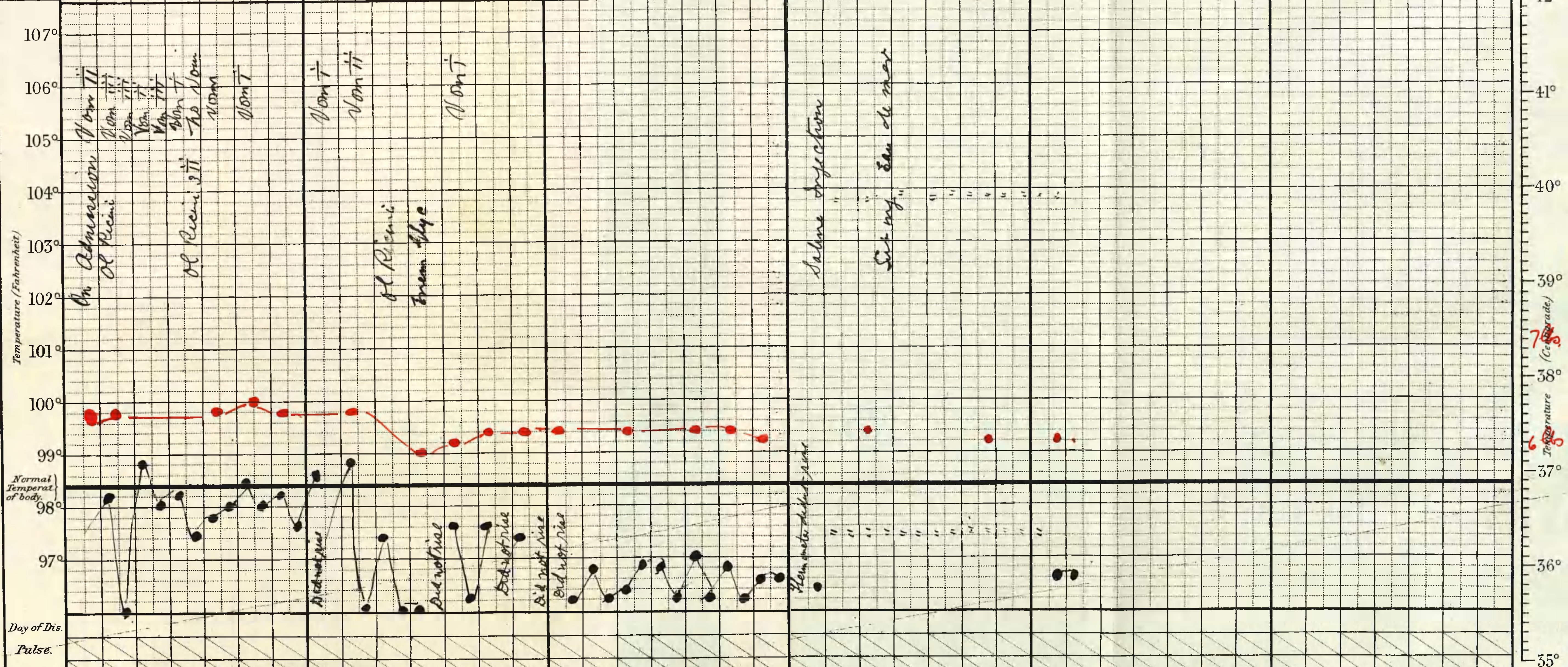
Date of admission.

Result

Time. M E M E M E …

Bowels. 0 0 1 0 0 0 0 1 0 0 1 0 1 0 0 0 0 1 0 1 2 0 1 1 0 1 1 0 1 1 1 1 2 2 2 1 2 2 3 1 2 1 2 0 2 1 2 2 2 2 2 2 0 1 2 2 2

Urine.



Day of Dis.

Pulse.

Resp.

Date. 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3

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Chart IV. Chart IV Mr Pringle's Cases

DISEASE.

Notes of Case.

Name { John Gordon

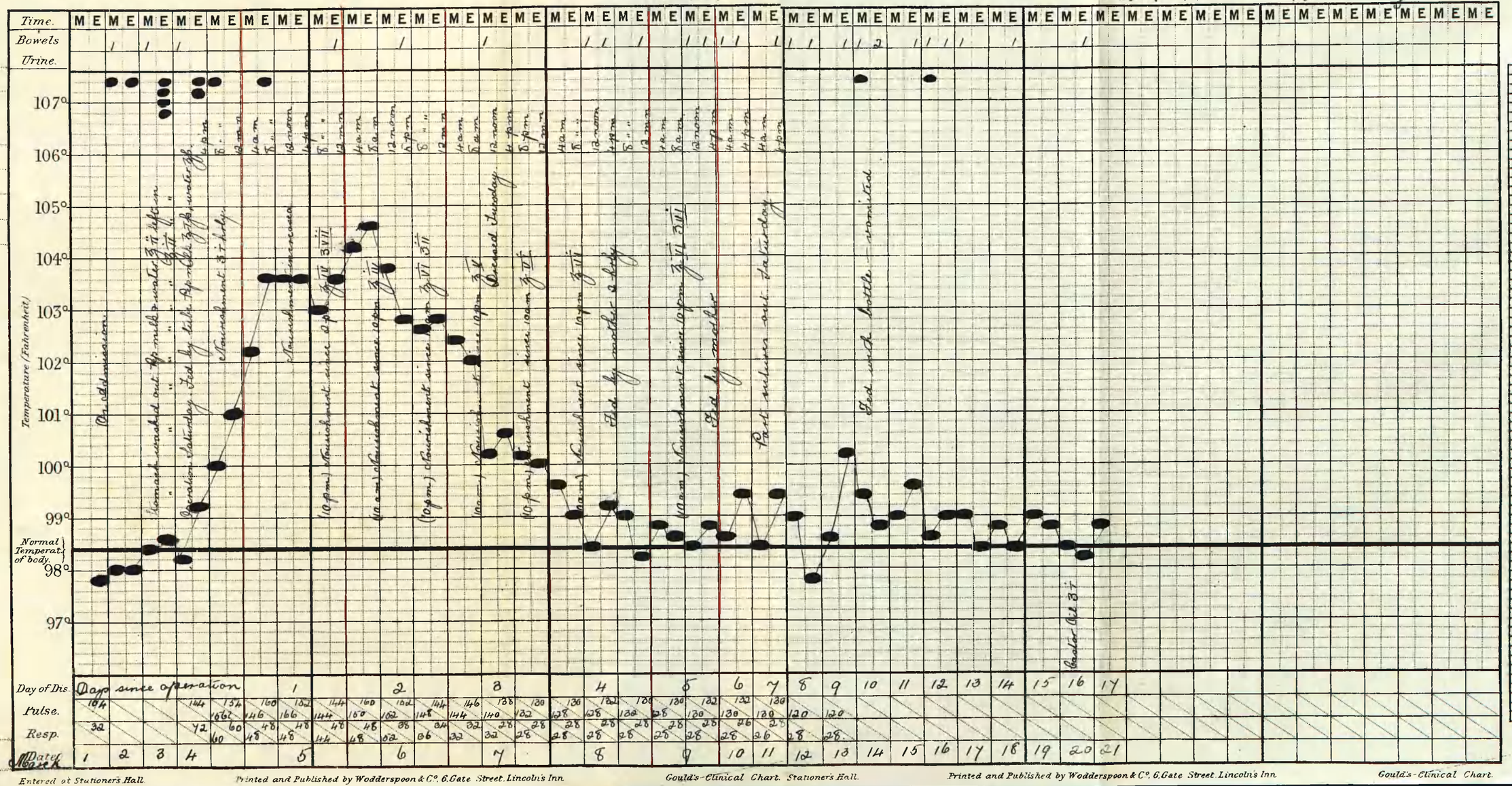
Age 7 weeks.

Diet

Case Book N°

Date of admission. 1. 8. 11.

Result



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Dr Cowans Case Chart V.

DISEASE.

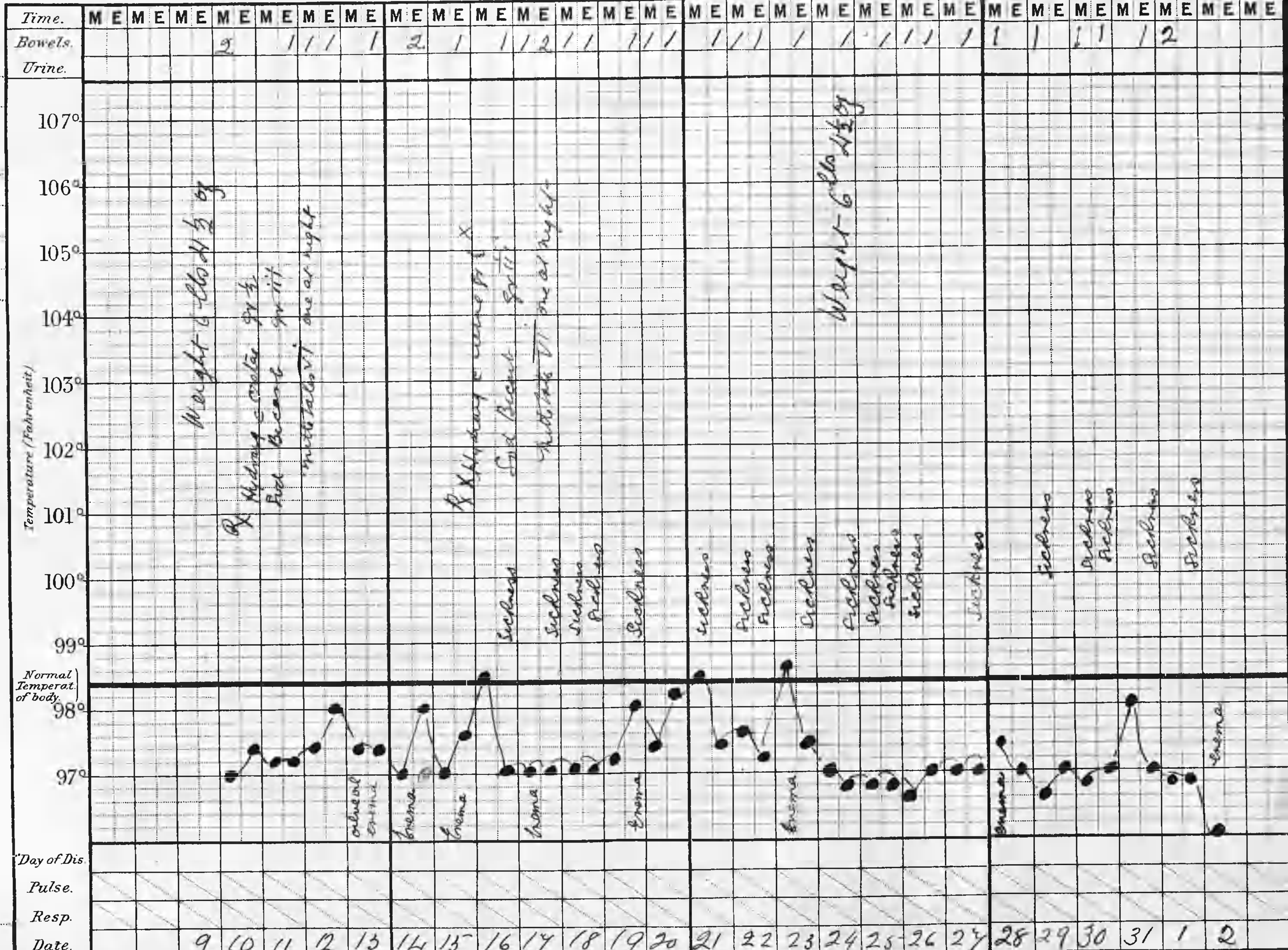
Notes of Case.

Name

Age

Diet

Case Book No.



Date of admission.

Result