A CLINICAL STUDY OF
CONGENITAL HYPERTROPHIC PYLORIC STENOSIS:
WITH INVESTIGATION OF 128 CASES.

by

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INTRODUCTION.

Congenital hypertrophic pyloric stenosis is the name of a disease which has several synonyms: the chief ones being pyloric stenosis of infants and spastic hypertrophy of the pylorus. By whatever name it may be called a useful definition is that suggested by Parsons (1) "a disease in which symptoms of increasing pyloric obstruction arise in children under three months of age".

It is a disease that has been brought to prominence only during the last half century. But it was recognised over two hundred years ago. In 1717 Patrick Blair reported to the Royal Society an account of the dissection of a child "who was five months old and did not weigh more than five pounds: who had been seized with violent vomiting and stoppage of urine and stool since he was a month old".

It was not until fifty years had elapsed that another case was described in 1767 by George Armstrong. In 1783 Beardsley described the post mortem findings of a case. Subsequently several cases were described by different clinicians but it was not until 1887 that Hirschprung drew attention to the fact that the condition was peculiar to infancy. Upon Finklestein falls the honour of describing a palpable tumour in a living case of congenital hypertrophic pyloric stenosis.
for the first time.

In 1895 John Thomson (1) wrote an excellent article in which he described a case of congenital hypertrophic pyloric stenosis, and offered as probable an explanation of the pathogenesis of the condition as has since been suggested.

In 1913 Rammstedt (1) instituted the operative procedure that is employed to-day. An operation which he modified from that introduced by Fredet a few years previously.

Although the operative technique has changed but little since 1913, the mortality rate has been greatly reduced owing to a sounder understanding of coexisting complications that so often attend this disease.

During the last few years, since Swensgaard introduced Eumydrin therapy in 1935, medical treatment has attracted considerable attention.

Ever since the Eumydrin therapy was introduced, all cases of congenital hypertrophic pyloric stenosis have been treated by this method in Leicester Royal Infirmary. As surgery had held sway until 1935 in that hospital, there appeared to be an excellent opportunity to compare the two methods of treatment under similar conditions. So, as Senior House Physician during the winter six months 1938-39 I have attempted to make a complete clinical study of the 128 cases treated/
treated in that hospital during the ten years 1929-1939. All but 10 of the cases discharged alive during those years have been followed up and reexamined personally. Radiology has been employed in order to complete the "follow up" of these cases.

As this thesis is essentially a clinical study of congenital hypertrophic pyloric stenosis, no attempt has been made to discuss the pathology as a separate entity.

The results of the cases investigated are tabulated at the end of the paper.

I should like to acknowledge here, my thanks to members of the Staff of Leicester Royal Infirmary and my great indebtedness to Dr. Forbes Lawson, Senior Honorary Radiologist, for his assistance in the radiological investigations, to Sister Williams for her enthusiasm in the follow up of the cases, and especially to Dr. Vernon Braithwaite, Senior Honorary Physician, for placing at my disposal the records of the cases investigated.
AETIOLOGY.

Congenital hypertrophic pyloric stenosis is an almost unique condition, in that the age of onset, sex and place in the family occur so uniformly in each case. Let us consider these separately.

Age of onset of symptoms.

There is nearly always a period of satisfactory progress after birth for a few weeks before symptoms commence. In this series of cases the average age of onset was at 3 1/2 weeks. The symptoms never commenced before the seventh day in the earliest case, whilst the latest date of onset was at 7 1/2 weeks. These figures agree with those observed by Sheldon (1) in a similar number of cases.

Sex.

There is a remarkable preponderance of cases that occurs in the male sex.

In the series under review 84% were boys. Similarly Peer found 80% to be boys, when investigating a large number of cases. The preponderance of male children affected makes the sex appear as a secondary diagnostic feature, since pylorospasm, which appears to be a separate condition, occurs more frequently in female infants.

Place in family/
5.

Place in family.

The peculiar likelihood of the first born child to be affected is quite as remarkable as the incidence in male infants. In my series, 79% were first born children. This percentage is rather higher than that obtained by Sheldon (1) in the series already referred to, where 50% were first born children.

The disease may be rarely found in other than first born children: in the cases under review, there were twins (case nos. 50 S.M. and 51 S.M.) affected, who were the fourth children in the family.

Familial incidence.

There appears to be a distinct tendency, though slight, for the disease to develop in more than one member of a family. I found three instances of this: a brother (case no. 26 H.C.) and a sister (case no. 62 A.B.), as well as one of their first cousins (case no. 99 M.C.)

In another family, two brothers (case nos. 62 A.B. and 85 D.B.) developed the disease.

Twin brothers (case nos. 50 S.M. and 51 S.M.) to whom reference has already been made, were also affected.

Tribble reports of the disease occurring in three children of one family, all of whom were operated upon and did well. However, the occurrence in twins/
twins is much less common, as is shown by the fact that Moore in describing a case of twins in 1924, stated that his was the first case to be described. In 1935 Bilderback also reported another case occurring in twins. Cockayne considers that heredity plays a most important part in the aetiology.

**Associated diseases.**

Suggestions have been made from time to time that congenital hypertrophic pyloric stenosis is associated with other diseases or syndromes. Pitfield found that in 10 cases, all the patients had one neurotic parent, and he considers that the disease is a form of hypersuprarenalism. From this he deduces that the condition is due to an hereditary imbalance. I hope to show, when discussing the cases which have been followed up, that there is little evidence of any neurotic tendency being transmitted to children. In my opinion there is no justification for this hypothesis.

Lapage has described four cases of congenital hypertrophic pyloric stenosis occurring in typical mongols. This would appear to be purely an unusual coincidence, since mongolism usually occurs towards the end of the mother's child-bearirig period, while congenital hypertrophic pyloric stenosis usually develops in the first child.

In
In my series of cases none of the patients when followed up, showed any signs of mongolism.

I am of the opinion that there are no diseases which occur in association with congenital hypertrophic pyloric stenosis.

Summary.

The aetiology of congenital hypertrophic pyloric stenosis is still obscure.

It has been verified from the cases under review, that there is considerable regularity in the age at onset, susceptibility of the male sex, and familial incidence in this disease. Also, an attempt has been made to disprove suggestions that this disease is associated with other conditions.
PATHOGENESIS.

This subject has been described at length by many authorities, so it will only be dealt with briefly in order to complete this study of the disease.

There are several theories as to the cause of congenital hypertrophic pyloric stenosis. John Thomson (2) in 1902, suggested that the primary condition is spasm of the pylorus and that hypertrophy is necessarily secondary. He suggested that "it may possibly be a sort of intrauterine developmental neurosis, one of those passing disturbances which sets in when function, interfered with, is in a process of very active development, being acquired but not yet perfect".

He considers that other disorders like Hirschprung's disease and congenital hypertrophy of the bladder have a similar pathogenesis.

The opposite view is held by many authorities, who consider that the hypertrophy of the pyloric canal is the primary factor and that it is congenital in origin.

Tyrrell Gray considers that the closure of the pyloric sphincter is controlled by stimulation of the sympathetic nerve supply and by its hormone (adrenaline). He suggests that the pyloric hypertrophy is associated with hyperadrenalism, the result of excessive sympathetic stimulation. Also he/
he considers that the hyperadrenalism is due partly to apprehension and anxiety of the mother while the child is in utero. This hypothesis offers an explanation of the reason why the condition occurs in first children in such a large percentage of cases.

There is another theory that failure of the stomach to relax is not due to stenosis, but to failure of the sphincter to relax. This theory is held by Levi (1). He considers that pyloric spasm is but an early manifestation of so-called pyloric stenosis. However this view is not held by most paediatricians in this country. The clinical picture of pylorospasm, which will be described later, has many essential features that differ from those of congenital hypertrophic pyloric stenosis. In my opinion they are separate conditions.

Summary.

A brief résumé has been made of the main views as to the pathogenesis of congenital hypertrophic pyloric stenosis.

Antispasmodic drugs, especially eumydrin, have been found to relieve symptoms in cases of congenital hypertrophic pyloric stenosis, where a palpable tumour is still found weeks later. This finding, in my opinion, corroborates the hypothesis advanced by John Thomson (2), that spasm is the primary factor whilst hypertrophy of the musculature of the pyloric canal is secondary.
10.

DIAGNOSIS.

Symptoms.

The onset of the symptoms bears a greater uniformity than in almost any other condition.

Usually the baby is a first child and often of the male sex, as discussed previously. He has been born at full term after a normal pregnancy and labour.

The baby usually progressed satisfactorily for the first few weeks, taking his feeds well and gaining weight. Usually about the third to fourth week sickness commences after feeds, at first perhaps only once or twice a day, but soon, after every meal. Almost invariably the vomiting is ascribed by the mother to unsuitability of the milk and so the infant is weaned onto some other food. For a few days cessation of the symptoms confirm the mother's pre-formed opinion; however vomiting soon recommences, and so the food is again changed. This may continue indefinitely with short periods of improvement, but the symptoms always return with increasing severity, and the baby steadily loses weight.

There are certain symptoms which have characteristic features, that may monopolise the clinical picture.

I. Vomiting.

At the onset this may be slight and without any/
any characteristic features, but its projectile character is usually soon exhibited. It is forcible and explosive, and the milk shoots out on to the floor through the mouth and nose. The vomited material is often large in amount, representing more than one meal. However, shortly after copious vomiting the infant appears hungry. Also, apart from the vomiting there are very few of the ordinary signs of dyspepsia. The tongue is clean and there are no sour eructations and no diarrhoea.

Rarely there may be a haematemesis as the first symptom.

The vomited material, on examination, is found to be strongly acid and contains no bile.

Although the projectile character of the vomit is very constant, it is surprisingly often not noticed by the mother.

Leonard Findaly (1) describes a case of congenital hypertrophic pyloric stenosis in which a definite tumour was felt without projectile vomiting being produced on more than two occasions, although visible peristalsis was present and the tumour was large. He does not attempt to elucidate this unusual case.

In the series under review, projectile vomiting was noted in all except one case, that is over 99% cases.

As/
As a result of the severe vomiting important alterations in the blood chemistry occur, which result in the development of varying degrees of alkalosis. This is often shown by slow, shallow respiration with prolonged apnoeic periods. Sometimes symptoms of tetany, as shown by convulsions and hypertonicity, occur. The treatment by replacing the chlorides causes cessation of these symptoms.

II Constipation

is another prominent symptom, but is not quite so constant as vomiting. Even when vomiting has been delayed until the third or fourth week, the constipation may date from birth and is thus often the earlier symptom. In this series of cases it was present in all but three cases, that is in 97.6% cases.

III Wasting.

A slow gain in weight usually takes place until the vomiting commences, then for a short period the weight remains stationary before beginning to drop. When once the weight has begun to decline, the fall - in absence of treatment - is steady without intermission, and is often very alarming.

IV General appearance.

When the disease fully develops, the facies
is almost typical; the face becomes thin and drawn, the expression becomes worried and the brows are creased into transverse furrows.

The photograph below of a case shortly after operation demonstrates the typical facies.
There are two main physical signs on examination.

**Visible peristalsis.**

This is seen as waves of peristalsis taking shape at the left costal margin, and rolling slowly across the epigastrium to fade away in the right side. In a well defined case, the waves are often aptly described as resembling a succession of golf balls slowly passing across the upper abdomen, but in more advanced cases, when the stomach is dilated the waves become broader and less obvious. Occasionally the peristalsis may be noted to be passing in a retrograde manner.

In order to see the peristalsis clearly, it is usually necessary to stimulate the stomach by distending it with a feed, and by "flicking" the abdominal wall. This also applies to the second chief sign, namely a palpable tumour. So, for this reason, examination of these cases is best performed during a feed, but also because the abdomen is relaxed whilst feeding.

See photographs.
Peristaltic wave commencing at left costal margin.

Peristaltic wave passing across epigastrium to the right side.
Visible peristalsis is an almost constant feature of congenital hypertrophic pyloric stenosis. Thompson and Gaisford in a series of 209 cases found visible peristalsis to be present in 100% of cases. However, this sign also occurs in pylorospasm and congenital duodenal stenosis. Thus it is of little value in differential diagnosis.

It was only absent in 4% of the series of cases investigated by me, and all of these babies were operated upon and a tumour was found. The case notes do not mention whether examination was made during feeds or not. This fact is, of course, of extreme importance and reduces the value of the observation.

Palpable tumour.

This term "tumour" is used to describe the greatly hypertrophied portion of the pyloric canal.

In order to palpate the abdomen for the presence or absence of a tumour, a proper technique should be employed. As mentioned above, the examination should be made while the infant is being fed, and the observer must be on the infant's left side, therefore the infant should be fed either from the mother's left breast, or else by a nurse feeding him from his right side. The infant must be disturbed as little as possible; the examiner's hand must be warm and should be on the same level as the infant.
infant, so that he can rest his hand comfortably across the baby's epigastrium. The tumour is often most easily felt by the middle finger, while the index and ring fingers are abducted and press gently against the abdominal wall.

If the tumour is not felt immediately, palpation should be prolonged for 5 to 10 minutes, as the tumour appears to undergo periodic contraction and relaxation. Thus, it may be extremely difficult to palpate and repeated examination during one or two feeds may be necessary. It is usually felt half way between the umbilicus and the costal margin at the outer border of the right rectus muscle, as a hard lump which, in my opinion, somewhat resembles a segment of a finger palpated under a blanket.

In some cases where the tumour has not been palpated, it has been found subsequently at operation to be tucked up beneath a rather large liver; whilst in long standing cases the grossly dilated stomach may push the tumour into the right flank.

In those cases treated subsequently by operation, an analysis of the percentage of cases in which no tumour was found on clinical examination shows remarkable variation in different clinics.

Wallace and Wevill analysed 145 cases treated by operation in the Royal Edinburgh Hospital for Sick Children, and found only 24.1% of the cases had a clinically/
clinically palpable tumour. In Thompson and Gaisford's series of 178 hospital cases, a palpable tumour was found in 76.6% of cases.

In the series of cases investigated by me, it was found that out of the 77 cases treated surgically no tumour was found in 18 cases prior to operation; thus, a palpable tumour was felt in 76.6% of cases. It should be noted that in all the cases of this series a typical tumour was found on operation.

The discrepancy between these figures is difficult to explain, but it probably shows how easily one can imagine clinical findings if examination is made with a preconceived diagnosis.

As has already been seen, prior to the fairly widespread use of medical treatment the diagnosis of congenital hypertrophic pyloric stenosis was often made without a tumour being felt. However, since the installation of medical treatment in many clinics, surgeons often view with scepticism the diagnosis in cases where a tumour has not been clinically palpated. Therein lies the difficulty in assessing the results of medical treatment. If one decides that the essential feature in diagnosis is a palpable tumour, then a large number of cases must fall erroneously into the category of pylorospasm. For this reason aid to the diagnosis by radiology has been sought.
X-RAY EXAMINATION.

There has been much controversy in the current literature concerning this method of investigating cases of congenital hypertrophic pyloric stenosis. A careful study of this subject in all its aspects has been made in order to reach an impartial conclusion as to the true value of radiology in this disease.

Technique of radiological examination.

Dobbs has described a useful routine method which is used in the University Hospital, London.

As soon as the child is seen, 1 dram of barium cream in 1 ounce of milk is given by the mouth and radiograms are taken at intervals up to 8 - 12 hours. Meanwhile isotonic saline to which is added 5% solution dextrose is given subcutaneously in sufficient quantities to relieve dehydration, as indicated by the clinical condition and by the passage of sufficient urine. After the last radiogram, the stomach is washed out with normal saline until the fluid is returned clear. These preliminaries often mean that the infant has little or no food for 6 - 8 hours after admission, but this has not been found to be anything but beneficial. All his series of cases have been treated medically by eumydrin, where it is so essential to combat the dehydration and alkalosis first/
first, before commencing administration of the eumydrin.

This technique has been employed in a modified form in Leicester Royal Infirmary, but only taking films up to 5 - 6 hours.

Friemann-Dahl prefers to examine the child behind the X-ray screen after 8 hours, first having given the child aqueous barium solution (1 in 2). The examiner's hand is held against the abdomen, and the contrast meal is pressed through the narrow lumen of the pyloric canal into the duodenum.

Methods of interpretation of films.

There is considerable diversity of opinion in the method of interpretation of the films amongst those authorities who believe that radiology is of value in diagnosis.

Köhler states that if within an hour the stomach of a newly born child has not allowed any of the contrast barium meal to pass through the pylorus, which he says normally happens at once, then congenital hypertrophic pyloric stenosis is present, even if no corresponding tumour can be felt. He further states that if the greater part of the meal has left the stomach within three hours the condition is purely due to spasm.

Runström, however, considers that the essential feature in the radiological diagnosis is the observation of the lumen of the pyloric canal, which he calls/
calls the "Canalis egestorius" (the most pyloric part of the stomach). This is narrowed down to a lineal lumen 1 - 3m.m. broad often provided with an even arc-like lumen.

The duodenal cap shows normal shape and size. The emptying time is markedly delayed. He emphasises that, proximally, the line bordering the stomach is distinct and there are no peristaltic waves passing beyond this limit. The stomach is more or less dilated and shows peristalsis of stenosis, often with intervals of atony. The lower limit of the stenosis extends to the pylorus. The duodenal cap is normal in shape and size, but it often takes 1 - 2 hours before the cap is filled. Jochims and Friemann-Dahl both hold the same opinion as Runström and lay emphasis on the diagnostic importance of the narrow needle-like lumen of the canal.

However, Bull lays more emphasis on the rate of emptying of the stomach. He says that the position and shape of the stomach are the same as in the normal infant, and despite clinical evidence, the X-ray rarely shows excessive peristalsis.

He has found that no barium passes through the stomach in the first few hours of examination. He further states that in a true case of congenital hypertrophic pyloric stenosis, the barium remains in the stomach indefinitely. A stomach which shows a steady/
steady rate of emptying, although this be in excess of the normal time, should not be diagnosed as congenital hypertrophic pyloric stenosis, but is probably an example of reflex pyloro spasm.

Teall found that "in infantile pyloric stenosis emptying usually commences within the first quarter of an hour after the food has been taken, but proceeds very slowly: almost invariably there is still barium in the stomach 6 - 8 hours later, and often at 24 hours after the meal, the feed has not passed through the pylorus". He has found that in pyloro spasm, on the other hand, one usually finds that none of the barium leaves the stomach during the first hour, or perhaps two hours after the meal has been taken, and then the stomach commences to empty rapidly, and emptying will probably be complete in another hour.

Assessment of value of radiological diagnosis.

As has already been mentioned, there is considerable difference of opinion firstly as to the accuracy of radiological diagnosis, and secondly as to its clinical value in relation to the treatment employed.

Sheldon (3) considers that X-ray examination does confirm the diagnosis, but thinks that this is not justified, as once a tumour has been palpated, the/
the diagnosis is complete; also in a wasted infant any amount of disturbance entailed, increases the exhaustion.

Maizels, in a series of cases, found radiology to be negative in 3 cases and inconclusive in 11 others where the diagnosis was subsequently confirmed at operation.

Leonard Findlay (2) recently submitted to a radiological examination, after a barium meal, a series of 12 examples of this disease, and as controls, 12 infants who appeared quite healthy, or were suffering from vomiting, but in whom no pyloric tumour could be felt.

As some interesting and important conclusions were drawn from these investigations, it is of value to describe them in detail.

He divided the course of investigations into two periods, (1) during the first two hours after ingestion of the meal, (2) from two hours after ingestion onwards.

Results in (1) "In the normal infant, opaque meal begins to leave the stomach immediately and within 40 - 50 minutes considerable amount has entered the small intestine. Occasionally, in the absence of stenosis, food does not leave the stomach immediately, or even at the end of an hour after ingestion comparatively little may have entered the duodenum. Usually/
Usually it was found in cases of congenital hypertrophic pyloric stenosis that the passage of the meal into the small intestine was delayed, but in some cases verified by operation the opaque meal may enter the duodenum as usual at the same rate as in the normal child. Thus from this aspect of the investigation, there was no sharp line of distinction between the cases with or without pyloric stenosis."

(2) In his experience the normal stomach is not usually completely empty until 5 - 6 hours after the ingestion of the opaque meal. In the presence of congenital hypertrophic pyloric stenosis delay in emptying is usually prolonged until 5 - 6 hours after ingestion. He found that variations were so considerable that the overlap deprived this method of investigation of any diagnostic value.

Also, he found that in those cases in which a barium meal was performed three or four weeks after operation the emptying rate, as judged by the passage of the meal into the duodenum and by the complete emptying of the stomach, had returned to normal. But this was not so in the cases treated medically, since even as long as 6 - 8 weeks after symptoms had disappeared, the typical radiological picture of congenital hypertrophic pyloric stenosis persists.

From these last observations he suggests that there/
there are two factors causing the obstruction, (1) spasm of the muscle (which is relieved by medical measures), and (2) narrowing of the pyloric canal from the mass of hypertrophied muscle.

In my series of cases radiology was employed in 58 cases out of the total 128. Of these 18 were treated surgically and 40 medically. Of the surgical cases 3 were re X-rayed recently, and of the medically treated cases 15 were also re X-rayed recently, when the cases were followed up.

Results in 18 cases treated surgically.

In 1 case no delay was noted at all, but in all the rest, varying degrees of delay were observed. In 3 cases marked 6 hour delay in gastric emptying was found. In the rest 3½ - 5 hour residues were found. In only 5 cases was definite narrowing of the pyloric canal seen.

Results in 40 cases treated medically.

In 2 of these cases where a definite tumour was felt there was no delay in gastric emptying. 2 showed a marked 6 hour delay, and the remaining 36 all showed a 4 - 5 hour delay in gastric emptying.

Spasm of the pyloric canal was only noted in 7 of the cases. Several of the others were too indefinite to make conclusions.

Thus, out of the total 58 cases, 3 showed no definite/
definite gastric delay in emptying, while only 3 had a marked 6 hour residue. Also, only 12 cases showed definite narrowing of the pyloric canal.

It must be noted that all the cases in which X-ray findings were indefinite had a palpable tumour.

Conclusions.

I am unable to agree with Sheldon's view that radiology is contraindicated because it is harmful to the child. If a careful technique be employed, in which saline is given as described by Dobbs, then the dehydration can be countered during the time that the series of radiograms are being taken.

However, I entirely agree with Leonard Findlay in that radiology is not justified, since the diagnosis by this means has been shown to be extremely unreliable, and that the method of treatment to be employed should be based upon the clinical findings and upon the views of the clinician.

Summary.

There are two schools of thought with regard to diagnosis, (1) those who consider that congenital hypertrophic pyloric stenosis can be differentiated from pylorospasm by means of radiology, without a tumour being felt, and (2) those who believe that a tumour must be felt in all cases before such a diagnosis/
nosis can be reached.

Now it has been shown quite definitely, that operation has been undertaken and a large "tumour" found without any "tumour" being felt preoperatively. In Wallace and Wevill's series, 79% of the cases had no clinically palpable tumour. It is obvious that if a palpable tumour is the criterion upon which the diagnosis is based, then a large number of cases of congenital hypertrophic pyloric stenosis must be missnamed as pylorospasm. In this way results from medical treatment will be wrongly assessed, while surgeons will still continue to operate upon cases which have no clinically palpable tumour, and so their figures will be more accurate.

Thus, when comparing results of treatment by the two methods one should base the diagnosis upon the same factors. In the series of cases investigated by me, the diagnosis of congenital hypertrophic pyloric stenosis has been confirmed if a tumour is found. However, if no tumour were felt, but a typical history of projectile vomiting and constipation with signs of visible peristalsis were present, then the diagnosis of congenital hypertrophic pyloric stenosis was accepted. This method of diagnosis has been shown to be trustworthy, since every case in which a laparotomy was performed a "tumour" was found. For a few years diagnosis by radiological methods/
methods has been employed as in the medical series, mainly to test their value. That this basis of diagnosis is unsatisfactory is obvious, but I have excluded from this series all cases diagnosed on this basis in which the subsequent course of the illness has made the diagnosis appear doubtful, so that comparison with surgical results should be on an equal basis.

In the entirely medically treated cases, 31 had a palpable tumour. Of the remaining 8 without a palpable tumour 1 subsequently died and the diagnosis was confirmed at post mortem examination.

In conclusion, I consider that a diagnosis of congenital hypertrophic pyloric stenosis should be made if the symptoms and signs enumerated above are present, even if no palpable "tumour" is felt. Radiology, in my opinion, is of little or no value in the differential diagnosis between pylorospasm and congenital hypertrophic pyloric stenosis and should be abandoned.
29.

**DIFFERENTIAL DIAGNOSIS.**

There has been some unnecessary controversy over the differential diagnosis owing to differences in nomenclature. Many continental paediatricians refer to congenital hypertrophic pyloric stenosis as pylorospasm. For this reason, in discussing the differential diagnosis, firstly, the clinical picture of the disease must be defined clearly. This especially refers to pylorospasm.

**Pylorospasm**

is a condition in which the symptoms commence usually rather later than in congenital hypertrophic pyloric stenosis, about the third month. It is commoner in girls than in boys and is less frequently found in the first child. It seldom occurs in breast fed babies. The symptoms are less well marked than in congenital hypertrophic pyloric stenosis. Vomiting may be copious and projectile, but the constipation is a less conspicuous feature. Examination may show a wasted infant with marked visible peristalsis, but a palpable tumour is never found.

Radiology often reveals almost complete gastric stasis for 1 - 3 hours, but the stomach is seldom grossly dilated as in congenital hypertrophic pyloric stenosis. Also, there is no narrowing of the/
the pyloric end of the stomach.

Opinion as to the value of radiology has been discussed previously.

The rapid response to treatment by medical measures quickly reveals the true diagnosis.

Rodrigo says that in a case of habitual vomiting spasmophilia and vagotonia can be excluded, and the symptoms subsided at once when 1 dram of 15% solution Bism: Carbonate in an acasia solution is given immediately before and after feeding. Difficulties in nomenclature occur here again, and the terms "spasmophilia" and "vagotonia" are not defined. One assumes from the context that they are meant to refer to pylorospasm. There do not appear to be any other references to this method being employed. No personal experience of it has been had.

Estella y B. de Castro on the other hand, considers that there are no certain signs for differentiation of the organic and functional forms.

David Levi (1) considers that pylorospasm is but an early manifestation of so-called pyloric stenosis. However, the general opinion held by most clinicians is that the differential diagnosis from congenital hypertrophic pyloric stenosis is usually possible if a careful history of the case is taken and a methodical examination is made.

Congenital/
Congenital duodenal atresia and stenosis.

The symptoms usually occur within 24 hours of birth, and vomiting is projectile. Visible peristalsis may be present, but is not marked and a "tumour" is never palpable. Bile is found in the vomit.

Radiology is said to be of great value as it demonstrates the site of the obstruction, which is usually in the second part of the duodenum near to the entrance of the common bile duct.

Congenital duodenal stenosis due to constriction of the viscus by abdominal peritoneal bands, may produce a similar clinical picture. Such a case has been described by Higgins, in which radiology was found to be of great value in the diagnosis. Frank also lays emphasis on the value of radiology in such conditions. Cautley had a case of duodenal stenosis, but had no difficulty in differentiating it from congenital hypertrophic pyloric stenosis.

However, all forms of congenital duodenal stenosis are rare, and there is no record of a case being admitted to the Leicester Royal Infirmary during the past ten years.

Severe gastritis.

may cause some difficulties in diagnosis, but constipation is less frequent than diarrhoea in such cases/
cases. The appetite is impaired while it is voracious in congenital hypertrophic pyloric stenosis. A tumour is never found.

Summary.

The differential diagnosis between congenital hypertrophic pyloric stenosis and pylorospasm is usually possible if all the symptoms and signs are carefully assessed. Congenital duodenal atresia or stenosis, and severe gastritis can usually be excluded rapidly if a good clinical history is obtained.
A. Medical.

Although congenital hypertrophic pyloric stenosis had been recognised as a clinical entity for many years, it was not until 1913 that really successful results were obtained when Rammstedt (1) introduced the operation that is used today.

Until 1922 medical treatment was tried chiefly by feeding the infant by means of a duodenal tube combined with gastric lavage to remove decomposing contents of the stomach. This treatment was largely unsuccessful because no attempt was being made to overcome the spasm of the pylorus.

The first advance in medical treatment occurred in 1924, when Johannessen described 7 cases of congenital hypertrophic pyloric stenosis cured by means of atropine. He gave 2 drops of 1/1000 solution of atropine sulphate 4 - 5 times a day, usually preceded by gastric lavage. In 4 cases suspension of the atropine was followed by return of vomiting, banished anew by brief resumption. It was given half an hour before feeds, aiming at opening the pylorus by this means when the food reaches it. The action was found to manifest itself in a few days, but did not reach its maximum until the 10th -
14th day. The atropine had to be given for 9 - 10 weeks. Infants were found to be very tolerant to atropine. The maximum dose employed by Johannessen was .7mgm.

In only 1 case was there serious poisoning, but the child recovered, and was still able to take small doses without marked toxic effects.

Thickened feeds had been tried without much success prior to the introduction of atropine, but combined with atropine therapy considerable success was claimed by both Meagher and Bruce.

However, it was not until 1935, when Elizabeth Swensgaard described the results of treatment with eumydrin, that medical treatment began to be taken up by many paediatricians.

**Pharmacology of eumydrin.**

After atropine was found to be of value as an antispasmodic for cases of congenital hypertrophic pyloric stenosis, numerous substances were tried in which other acids were combined with atropine.

Atropine (I) is an ester formed from the base tropine and acid trophic acid with elimination of water. One of the hydrogen atoms can be substituted by a methyl group, so that there are now two attached to the nitrogen bridge: if the acid in/
in combination at the same time is nitric acid, the result is eumydrin or atropine methyl nitrate (II).

\[
\begin{align*}
\text{CH}_2 &- \text{CH} - \text{CH}_2 \\
\text{NCH}_3 &
\end{align*}
\]

(I)

\[
\begin{align*}
\text{CH}_2 &- \text{CH} - \text{CH}_2 \\
\text{CH}_3 &- \text{N} <\text{NO}_3 > \text{CH}_3 \\
\text{CH}_2 &- \text{CH} - \text{CH}_2
\end{align*}
\]

(II)

Eumydrin was intended as a mydriatic.

Paul Drucker, while on the Staff of the Children's Department of Rigshospital, had found this atropine compound to be non-poisonous, and yet to be particularly efficacious. Swensgaard quotes that it is fifty times less poisonous than atropine, although the efficacious dose is only two or three times larger, but as Vertue points out, no particulars or references are given.
Technique employed in medical treatment with eumydrin.

The technique used in my series was a modification of that introduced by Swensgaard. It is simplest to describe the various aspects of the treatment separately.

Dehydration.

This is mentioned first because it must be the first consideration in treatment.

In almost all cases 3 - 5 oz. of normal isotonic saline solution (0.9%) were given subcutaneously into the mammary region. Full aseptic precautions were naturally employed.

After the saline had been administered, the fluid was gently massaged away.

Sometimes shock was noted after giving the saline, so 10 - 15 min: brandy were given orally in 1 - 2 teaspoonfuls of water.

In some of the severer cases in order to avoid the shock caused by the subcutaneous salines, isotonic saline was administered by the intraperitoneal route. The anterior abdominal wall was pinched up in the fingers, and a needle was inserted about half way between the umbilicus and the symphysis pubis. A "Wasserman" needle was/
was employed. By this route a rather larger quantity of saline could be given, up to 6 oz.

Saline infusions were given from 1 - 3 times a day as indicated by the degree of dehydration. They were continued until the weight of the infant was rising satisfactorily.

Frequently, if the infant was very wasted and anaemic as well as dehydrated, considerable benefit was obtained by giving 2 - 3 oz. whole blood intravenously. For reasons that will be discussed later, the intrafontanelle route was not employed. Cutting down over the internal saphenous vein as it runs over the medial malleolus and then inserting a fine glass cannula was preferred.

Feeding.

Breast milk was given whenever possible. If the mother was unable to come to the hospital several times a day, then a breast pump was employed. All the milk being collected together over twenty four hours and sent to the hospital daily. This was usually most unsatisfactory, as almost invariably secretion of milk dried up after a few days. The only satisfactory method, which is to accommodate the mother in the hospital, was seldom/
seldom feasible.

When breast milk was unavailable, a dried milk half cream was employed. "Cow and Gate" half cream was the standard food used.

The calorific measurement employed was 45 calories per pound body weight.

Sugar was added to the dried milk in order to make up the necessary calories.

The fluid required daily was estimated as being 2 1/2 oz. per pound body weight. If dehydration was marked, supplementary feeds of glucose in water were given together with the saline infusions, in order to raise the fluid intake up to about 4 oz. per pound body weight daily.

Seven feeds were given daily at three hourly intervals. The usual times were 6 a.m., 9 a.m., 12 a.m., 3 p.m., 6 p.m., 9 p.m. and 12 p.m. No feeds were given at 3 a.m.

A lactic acid milk was found to suit some of the more wasted babies better than the half cream dried milk. The "Cow and Gate" production, half cream "Lacidad" was usually employed. No sugar was added as this production already contains sugar.

**Eumydrin administration.**

Eumydrin was usually given half an hour before each
each feed. 80 - 100 min: of a freshly prepared 1/10,000 solution was given, in many cases before each feed, but sometimes only before five of the feeds as suggested by Swensgaard.

In all cases administration of eumydrin was never commenced until satisfactory diuresis had occurred.

Signs of intolerance.

In about 10% cases a pyrexia developed after eumydrin had been given. It was seldom above 100°F., and did not occur until a few days after treatment with eumydrin had been instituted. Whenever this pyrexia occurred, no eumydrin was given before the next feed and thereafter for a few feeds only 20 - 40 min: were given.

In one infant distension of the abdomen was noted. This was considered to be due to the eumydrin causing relaxation of the intestinal musculature. After reducing the dose of eumydrin to 30 min:, the distension disappeared.

Gastric lavage.

Swensgaard disapproves of gastric lavage in these cases. She says "It has been shown that because of the severe vomiting, the body becomes extremely deficient in chloride and the patient/
patient may go into a hypochloraemic coma. In view of this, Seckel disapproves of gastric lavage which makes the body still more deficient in chloride. However the substance used for the gastric lavage is not described, but from the text one would assume that it is sodium bi-carbonate or a similar acting compound.

If isotonic saline solution be employed, the chloride washed out by the gastric lavage can be satisfactorily replaced by leaving a little of the saline solution in the stomach at the end of the operation.

Gastric lavage by this method has been employed frequently as routine at 8 a.m. each day, in my series. It also acts as a gauge to the progress of the case, by estimating the volume of the gastric residue.

General precautions.

One year after eumydrin therapy was instituted in Leicester Royal Infirmary, separate glass cubicles were built chiefly for isolation of these cases. Also a rule was made that anyone entering a cubicle must don a gown and mask.

Particular emphasis was stressed upon nurses that they must roll up their sleeves and thoroughly/
thoroughly wash their hands before bottle feeding each infant.

Since these precautions were undertaken, the incidence of gastro-enteritis has greatly fallen as compared with the incidence during the previous year.
B. Surgical.

Operative technique has changed but little since the original operation was introduced by Rammstedt in 1913, but the mortality rate has greatly fallen during the last few years. This can be ascribed to the fact the surgeons too, have realised that dehydration must be overcome at all costs, even if operation has to be postponed for 2 - 3 days. Donovan lays great emphasis on this fact when he describes the technique employed in his 143 cases where no deaths occurred.

In Leicester Royal Infirmary, cases were treated by different surgeons, so no uniform pre-operative technique can be described. In some of the earlier cases of this series, operation was carried out as an emergency measure, but latterly the modern views of dealing with the dehydration first were adhered to.

In all the cases open ether anaesthesia was employed.

The actual operation employed was the standard Fredet-Rammstedt submucous pyloroplasty. No value can be obtained from describing the technique of this operation here, as it is fully described in all surgical text books.

Post/
Post operatively breast feeding, if possible, was commenced. If breast milk was unobtainable then "Cow and Gate" half cream was used, as described in the medical treatment.
RESULTS OBTAINED FROM MEDICAL TREATMENT.

In this series, 50 cases were treated by eumydrin therapy. These cases consisted of all but 3 cases admitted to Leicester Royal Infirmary since 1935. (The 3 cases treated by operation had been referred to the surgeons before admission to hospital.)

There were 8 deaths, which will now be described separately.

(1) Case no. 72. R.P. Treated with eumydrin for 5 days. No response obtained. Rammstedt operation performed. Died on following day from shock. (No post mortem examination.)

(2) Case no. 78. M.L. Responded well to eumydrin. Was to be discharged home next day, but died suddenly on 58th day after admission. Post mortem revealed collapse of the lower lobe of the right lung.

(3) Case no. 80. R.S. Responded well to eumydrin, but developed gastro-enteritis and died on 10th day after admission.

(4)/
(4) Case no. 81. P.W. Admitted in marasmic state. Never recovered from dehydration. Received eumydrine for 24 hours.

(5) Case no.100. I.H. Responded well to eumydrin. Died suddenly on 28th day. No cause found at post mortem examination.

(6) Case no.101. B.L.H. Responded well to eumydrin. Developed broncho-pneumonia. Died on 6th day after admission.


(8) Case no.123. J.H. Failed to respond to eumydrin therapy after 24 days treatment. Rammstedt operation performed, but died of shock.

One case has not been mentioned, (no. 118. L.G.) as the infant was admitted in a very marasmic state and died in 18 hours after admission. Diagnosis was only made at post mortem examination. No eumydrin had been given. This case has been excluded from both the surgical and medical series.

Summary/
Summary.

The mortality rate was 16%. Two of the deaths occurred within 3 days of admission.

In the cases that responded to eumydrin therapy, there was great variation of the time taken for the symptoms to subside. In most cases this was not dramatic, but very gradual and frequently daily subcutaneous salines had to be administered for a fortnight.

The average duration in hospital was 41 days.

Eumydrin failures.

There were 6 cases that failed to respond to eumydrin treatment: 5 of these were operated upon, 2 of which, as have been described, died. The remaining case was treated with syntropan with success.

Thus, 12% of the cases failed to respond to eumydrin.

SYNTROPAN.

Syntropan or 3 diethylamino-2; 2 dimethyl propanol, is another antispasmodic drug which was produced by the Hoffman la Roche Laboratory and has been claimed by Fromherz to have an equal antispasmodic/
ic action as atropine on the intestine, but its action on the pupil, salivary secretion and vagus is claimed to be more than 1,000 times less than that of atropine.

One baby in my series was treated by this drug after eumydrin had failed to cause cessation of vomiting following a months treatment. He was given 10 mgm. of syntropan suspended in 2 c.c. water orally before each feed. Vomiting practically stopped, and he began to gain weight.

However, since then another case treated elsewhere in Leicester, developed hyperpyrexia and died, so use of syntropan has been discontinued there.
RESULTS OBTAINED FROM SURGICAL TREATMENT.

The number of cases treated surgically in this series, between the years 1929 and 1935, was 73.

There were 13 deaths, which will now be described in detail.

(1) Case no. 2. F.C. Admitted in very dehydrated state. Rammstedt operation performed on the same day. Died a few hours later. Cause of death - shock.

(2) Case no. 4. F.F. Admitted in dehydrated state. Died 7 days after Rammstedt operation from sudden collapse. Post mortem examination - no abnormality found.

(3) Case no. 6. A.C. Admitted in dehydrated state. Died on 5th day after Rammstedt operation. Post mortem examination - only a few fine adhesions around pyloric canal found. Cause of death - shock.
(4) Case no. 23. R.W. Admitted in very dehydrated state. Rammstedt operation performed on same day. Died a few hours later.


(7) Case no. 26. H.C. Admitted in dehydrated state. Intrafontanelle blood transfusion given. Died on 7th day after Rammstedt operation. Post mortem examination showed a large subdural haematoma, having spread from superior sagittal sinus.

(8) Case no. 42. K.T.S. Admitted in satisfactory state. Died on day of Rammstedt operation/
tion, 4 days after admission.
Post mortem examination re-
vealed a large intraperitoneal haemorrhage from a tear of the portal vein.

(9) Case no. 68. G.L. Died 21 days after Rammstedt operation. Had suddenly collapsed.
At post mortem examination no cause was found.

(10) Case no. 69. S.D. Died 7 days after Rammstedt operation.
Post mortem examination re-
vealed collection of pus in the lower sac due to perforation of pyloric mucosa.

(11) Case no. 70. D.B. Died 22 days after Rammstedt operation.
Post mortem examination re-
vealed extensive broncho-
pneumonic consolidation.

(12) Case no. 71. A.L. Died 19 days after Rammstedt operation.
Post mortem examination re-
vealed no obvious cause.
Case no. 82. R.P. State on admission satisfactory.
Died 6 days after Rammstedt operation from gastro-enteritis.

As in the medically treated series, there is one case which has not been included in the above fatal cases. Case no. 41. M.O. was admitted in a marasmic state and as the dehydration was unable to be overcome, no operation was performed.

Summary.
The mortality rate was 17.8%.
There was considerable variation as to the cause of death.
The various causes of death will now be discussed.
CONCLUSIONS DRAWN FROM FATAL CASES UNDER
MEDICAL AND SURGICAL TREATMENT.

Postoperative shock.

Until recent years this accounted for a large percentage of the deaths in surgical cases, when all cases were operated upon as emergencies. Death was due to the dehydration being inadequately corrected.

In the series of cases investigated by me, three out of the first four cases died of postoperative shock. These cases occurred in 1929 and at that time many surgeons operated upon hypertrophic pyloric stenosis as an emergency procedure. Since then, the surgical death rate has fallen largely because the dehydration is first combatted.

In 1921 Gray and Reynolds analysed 50 cases operated upon. There was a death rate of 55%. Of these, immediate collapse accounted for 38% of the deaths, whilst sudden collapse within three days accounted for 33% of the deaths.

In 1924 Poynton, Higgins and Brydson reported an account of 20 cases operated upon, in which three out of the four deaths were due to dehydration.

In 1929 Bayer reported upon the result of treatment of 86 cases between 1919 and 1929, in the Children's/
Children's Clinic of Berlin. Careful investigation revealed that the fatalities were not due to faulty technique, but the condition of the patient was the cause of death. He emphasised that the preoperative treatment is more important than the technique of the operation.

All these cases are reported in order to stress the importance of overcoming dehydration before operation is undertaken. However, a great deal of the responsibility for the appalling state in which many cases are admitted, rests with the family practitioner who must be urged to get expert assistance earlier.

**Sudden collapse.**

Three of the surgical cases died suddenly over a week after the operation, when their progress had been perfectly satisfactory. At postmortem examination no cause for the death could be found.

In one of the cases treated with eumydrin, a similar type of collapse occurred when the child was about to be sent home the next day. There were none of the characteristic signs of eumydrin intolerance, such as pyrexia.

Wallace and Wevil found that 66.6% of the deaths in their series of cases treated at the Royal Hospital/
Hospital for Sick Children, Edinburgh, occurred as a result of sudden collapse and yet no pathological change was found at postmortem examination. As they aptly state the infants "fade away for no apparent reason".

This cause of death appears to be unassociated with the type of treatment employed. No satisfactory explanation has as yet been offered and very little reference is made to it in the text books. Until satisfactory research is carried out, a large number of deaths will occur each year in spite of whatever treatment is undertaken.

**Intercurrent infection.**

**Respiratory infections.**

In the surgical series broncho-pneumonia accounted for two of the deaths, while in the medical series it accounted for one death.

The high incidence of broncho-pneumonia in the surgical series can probably be ascribed to the use of general anaesthesia during the operation. Open ether anaesthesia was used in all cases. In the Edinburgh series by Wallace and Wevill none of the deaths were due to this cause. This is probably due to the use of local anaesthesia in every case, reducing/
reducing the chance of irritation of the bronchial mucosa.

However, Donovan has used general anaesthesia in all his cases and has had no incidence of pulmonary complications. This is contrary to the findings of most surgeons in Great Britain.

In the case of the medically treated patients broncho-pneumonia is a less common complication, but when it does occur it arises probably from the lowered resistance of the patient to droplet infection present in the ward. This will be referred to later.

**Gastro-enteritis.**

In the surgical series gastro-enteritis accounted for two deaths, while in the medically treated cases it only accounted for one death.

The higher incidence in the surgically treated patients would appear very unexpected if the cases were truly comparable. All the surgical cases were treated in an open ward, while after 1926, the medical cases were treated entirely in cubicles, with full aseptic precautions, no nurse being allowed in the cubicle without gown and mask.

The only medical death occurred before these precautions were undertaken.
The incidence of gastro-enteritis, apart from fatal cases, has been greatly reduced since this precaution was taken.

In the Edinburgh series reported by Wallace and Wevill, enteritis was responsible for 25% of the deaths. This shows the high incidence that may occur in spite of careful post-operative treatment.

However, Jewesbury and Page found a low incidence of gastro-enteritis when they reviewed 303 cases that had been treated surgically in St. Thomas's Hospital in an open ward. Of the total 33 deaths only 2 were due to gastro-enteritis, that is only 6%.

Gray however, analysed 50 cases operated upon, and found that diarrhoea was present in 28% of the fatal cases. He considers that congenital hypertrophic pyloric stenosis is partly due to hyper-adrenalism and that this is associated with pancreatic insufficiency. He suggests that the diarrhoea is attributable to this pancreatic insufficiency. However, no confirmation of this hypothesis is obtainable.

Conclusions.

The incidence of gastro-enteritis in the surgically treated cases has been very high in the past, but the risk of this complication is much greater in medically treated cases, which are treated under similar conditions, owing to the more prolonged stay/
stay in hospital, and to the more gradual improvement from the dangerously wasted state in which these infants are often admitted.

From my own series the conclusion drawn is that the incidence of gastro-enteritis can be greatly reduced if vigilant asepsis is adhered to during the whole period of hospitalization. One of the chief causes of necessitating prolonged hospitalisation has been the development of diarrhoea in the eumydrin series. Most surgeons consider this to be one of the main disadvantages of medical treatment. This will be discussed more fully later.

**Surgical errors.**

Two of the deaths in the surgical series occurred as a result of errors in technique.

One case, as has been described, died following a perforation of the mucous membrane. This is probably the most common and most dreaded danger in the whole operation.

Arrow indicates usual site of perforation of the mucous membrane.
The other death was due to a slight tear of the portal vein. This is an uncommon complication.

In the Edinburgh series only 2.8% of the deaths were due to peritonitis, while in the series investigated by Jewesbury and Page, 9% of the deaths were due to this cause.

The Rammstedt operation is a difficult operation to perform with constantly good results. In teaching hospitals these operations are usually performed by surgeons who are specialised in the surgery of children, with the result that constant practise in this specialised form of surgery is maintained. In provincial towns there is naturally less tendency to specialisation, and so there is less opportunity for developing quite such high grades of efficiency in one particular branch of surgery. Rammstedt (2) himself has emphasised the importance of experience in this operation.

This subject will also be discussed later, when considering the advantages and disadvantages of surgical and medical treatment.

Death due to blood transfusion.

One death in the series of cases under review was due to a haematoma forming as a result of puncturing right through the superior sagittal sinus.
This tragedy occurred in spite of great care being taken. Until recently, this method was extensively used for giving blood transfusions and saline infusions, but during the last few years it has been less and less frequently employed as its dangers have been more fully realised. In the Leicester Royal Infirmary this method has been entirely discontinued in the Children's Hospital for three years. The more laborious method of using the internal saphenous vein as it crosses in front of the internal malleolus is preferred.

This cause of death is mentioned here, as it may occur equally in medically or surgically treated cases, but can be so easily avoided if that method is discontinued.
**COMPARISON OF MORTALITY RATE OF SURGICAL AND MEDICAL CASES.**

Mortality rate figures produced by various authorities are often quoted in arguments as to the relative merits of surgical and medical treatment. It is of interest to study a large number of figures produced from different series, in order to attempt to draw conclusions as to their value in deciding upon the line of treatment.

### Surgical Treatment.

<table>
<thead>
<tr>
<th>Year</th>
<th>Paediatrician</th>
<th>No. of cases</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1921</td>
<td>Tyrrell Gray</td>
<td>38</td>
<td>55%</td>
</tr>
<tr>
<td>1924</td>
<td>Poynton, Higgins &amp; Brydson</td>
<td>20</td>
<td>20%</td>
</tr>
<tr>
<td>1929</td>
<td>Bayer</td>
<td>46</td>
<td>26.3%</td>
</tr>
<tr>
<td>1930</td>
<td>Rammstedt (2)</td>
<td>497</td>
<td>22.5%</td>
</tr>
<tr>
<td>1933</td>
<td>Lamman &amp; Mahoney</td>
<td>425</td>
<td>6.6%</td>
</tr>
<tr>
<td>1934</td>
<td>Hipsley &amp; Tait</td>
<td>120</td>
<td>4%</td>
</tr>
<tr>
<td>1934</td>
<td>Wallace &amp; Wevill</td>
<td>145</td>
<td>24.8%</td>
</tr>
<tr>
<td>1935</td>
<td>Oehler</td>
<td>42</td>
<td>14%</td>
</tr>
<tr>
<td>1935</td>
<td>Thompson &amp; Gaisford</td>
<td>178</td>
<td>16.4%</td>
</tr>
<tr>
<td>1937</td>
<td>Donovan</td>
<td>143</td>
<td>30%</td>
</tr>
<tr>
<td>1937</td>
<td>Harris</td>
<td>37</td>
<td>2.8%</td>
</tr>
<tr>
<td>1937</td>
<td>Jewesbury &amp; Page</td>
<td>303</td>
<td>12.5%</td>
</tr>
</tbody>
</table>

**Medical Treatment**/
Medical Treatment.

<table>
<thead>
<tr>
<th>Year</th>
<th>Paediatrician</th>
<th>No. of cases</th>
<th>Mortality.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1929</td>
<td>Bayer</td>
<td>40</td>
<td>2.5%</td>
</tr>
<tr>
<td>1930</td>
<td>Rammstedt</td>
<td>1345</td>
<td>15%</td>
</tr>
<tr>
<td>1935</td>
<td>Swensgaard</td>
<td>61</td>
<td>1.6%</td>
</tr>
<tr>
<td>1939</td>
<td>Dobbs</td>
<td>20</td>
<td>5%</td>
</tr>
<tr>
<td>1939</td>
<td>Findlay (3)</td>
<td>6</td>
<td>0%</td>
</tr>
<tr>
<td>1939</td>
<td>Vertue</td>
<td>21</td>
<td>4.8%</td>
</tr>
</tbody>
</table>

To these may be added the series collected by Swensgaard:

<table>
<thead>
<tr>
<th>Year</th>
<th>Paediatrician</th>
<th>No. of cases</th>
<th>Mortality.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1921</td>
<td>Einberg &amp; Hamilton</td>
<td>57</td>
<td>3.5%</td>
</tr>
<tr>
<td>1922</td>
<td>Ibrahim</td>
<td>52</td>
<td>1.9%</td>
</tr>
<tr>
<td>1928</td>
<td>Monrad</td>
<td>228</td>
<td>4.4%</td>
</tr>
<tr>
<td>1930</td>
<td>Wolff</td>
<td>98</td>
<td>2.2%</td>
</tr>
<tr>
<td>1933</td>
<td>Faxen</td>
<td>126</td>
<td>3.3%</td>
</tr>
</tbody>
</table>

Discussion.

On examining the above series of cases one is struck with the great variation in the surgical mortality rate. Although the mortality rate in the last two surgical series is high, it must be remembered that they extend over a longer period of years than some of the earlier series. Taking this into consideration, a considerable fall in the mortality rate during the last few years is evident.
Another interesting but distressing feature is the relatively high mortality rate of British series as compared with Continental and American series. With all due modesty, it can be claimed that the high mortality rate cannot be accounted for by any relative inferiority in the treatment of patients in this country. Thus, the cause must be that either our patients are admitted in a more wasted and dehydrated state, or that the disease itself is severer.

The medical series of cases show a strange mortality rate. Some of the early series, produced long before eumydrin therapy was introduced in 1935, have a surprisingly low mortality rate. One may draw a probably fallacious conclusion that medical treatment, before eumydrin was introduced, was as good if not better than any form of treatment employed to-day. Thus it is apparent that the diagnosis of many of the medical cases was grossly inaccurate; a large number of cases being really functional in origin, namely, suffering from pylorospasm. Vertue describes his cases very clearly, showing excellent results obtained from eumydrin therapy, but unfortunately he does not mention his basis for diagnosis, which greatly reduces their value in this discussion.

Conclusions/
GENERAL FACTORS AFFECTING THE MORTALITY RATE
IN CONGENITAL HYPERTROPHIC PYLORIC STENOSIS.

There are several factors which greatly affect the mortality. These will now be considered separately. For the sake of comparison between my series and those obtained in other hospitals, certain facts are tabulated below.

<table>
<thead>
<tr>
<th>Clinician</th>
<th>weight at birth</th>
<th>weight on admission</th>
<th>age on admission</th>
<th>No. of weeks between onset of symptoms and admission</th>
<th>no. of cases</th>
<th>mortality rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thompson &amp; Gaisford</td>
<td>a. 8 4</td>
<td>6 15</td>
<td>6 wks.</td>
<td>-</td>
<td>128</td>
<td>16.4%</td>
</tr>
<tr>
<td></td>
<td>d. 7 1</td>
<td>6 15</td>
<td>1 day</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wardill</td>
<td>-</td>
<td>6 8</td>
<td>6 wks.</td>
<td>3</td>
<td>123</td>
<td>23.5%</td>
</tr>
<tr>
<td>Lamman &amp; Mahoney</td>
<td>-</td>
<td>-</td>
<td>4 wks.</td>
<td>-</td>
<td>425</td>
<td>6.3%</td>
</tr>
<tr>
<td>Hipsley &amp; Tait</td>
<td>-</td>
<td>7</td>
<td>5 1/2 wks.</td>
<td>1 1/2</td>
<td>120</td>
<td>4%</td>
</tr>
<tr>
<td>Wallace &amp; Wevill</td>
<td>a. 8 8</td>
<td>6 8</td>
<td>6 1/2 wks.</td>
<td>3 1/2</td>
<td>145</td>
<td>24.8%</td>
</tr>
<tr>
<td></td>
<td>d. 8 8</td>
<td>6 8</td>
<td>6 1/2 wks.</td>
<td>3 1/2</td>
<td>145</td>
<td>24.8%</td>
</tr>
<tr>
<td>Own series</td>
<td>a. 6 13</td>
<td>6 13</td>
<td>5 1/2 wks.</td>
<td>1 1/2</td>
<td>128</td>
<td>18.5%</td>
</tr>
<tr>
<td></td>
<td>d. 7 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Birth weight.

One would expect that the birth weight of those patients who died would have been lower than that of the survivors, but it has been found in the series of cases investigated by me, that the birth weight of those that died was 4 oz. higher than those that survived. This unexpected finding has also been noted in Wallace and Wevill's series of cases. Thus the birth weight appears to play no part in the prognosis of this disease.

Weight on admission.

In my series the average weight on admission was 6 lbs. 13 oz. This is about the average weight that was found in most statistics.

It will be noted that Wallace and Wevill found their average weight on admission to be lower — 6½ lbs., with a correspondingly higher mortality rate of 24.8%. While Hipsley and Tait, with a higher average admission weight of 7 lbs. had a lower mortality rate of 4%. Thompson and Gaisford's and Wardill's series both fit into this ratio.

A graph of the mortality rate relative to the weight on admission, demonstrates the close relationship which these figures bear in the different series.
Graph no. 1.

Mortality Rate

25% 20% 15% 10% 5%

6 lbs 8 oz 9 lbs 10 oz 11 lbs 12 lbs 13 lbs 14 lbs 15 lbs 16 lbs 17 lbs 18 lbs

Weight on admission in pounds and ounces.

1. Thompson's data 6 series.
2. Wardill's series.
3. Hsipsley's Tait 6 series.
5. "Bion" series.

(1) Thompson's data 6 series.
(2) Wardill's series.
(3) Hsipsley's Tait 6 series.
(4) Wallace's review series.
(5) "Bion" series.
Number of weeks between onset of symptoms and date of admission.

One finds that this also bears a distinct ratio to the mortality rate, as is demonstrated from the graph below.

These facts are very important because they show how essential it is that the general practitioner should recognise the condition early, and so allow treatment to be undertaken while the patient is still in good condition.

[Graph showing relationship between number of weeks and mortality rate]
Influence of Types of Feeding Employed.

One of the few subjects on which paediatricians agree is the preference for breast feeding of infants. This applies to cases of congenital hypertrophic pyloric stenosis as much as to healthy infants. To quote Parsons, "if the child is seen early in the course of the disease and is breast fed, the post-operative mortality should not exceed 5%".

Here it is of interest to compare the percentage of cases breast fed with the mortality rate in a few different series, irrespective of the treatment employed.

<table>
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<tr>
<th>Paediatrician</th>
<th>No. of cases</th>
<th>Entirely breast fed</th>
<th>Partly breast fed</th>
<th>Entirely bottle fed</th>
<th>Mortality rate</th>
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<td>179</td>
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<td>Jewesbury &amp; Page.</td>
<td>144</td>
<td>55%</td>
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<tr>
<td>Own series</td>
<td>128</td>
<td>33.6%</td>
<td>21.9%</td>
<td>44.5%</td>
<td>18.5%</td>
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</table>

The number of series of cases obtained is too small to draw definite conclusions, but it is interesting to note that my own series had a relatively high percentage of bottle fed babies with also a relatively high mortality rate.
In Leicestershire, bottle feeding unfortunately has a very high incidence as compared with other parts of Great Britain. This is probably partly due to the high standard of living in that county, where consideration of the price of artificial foods is not an important factor, and partly due to the mother's desire to return to work as early as possible after the confinement.

The results from investigations entirely agree with the general medical opinion that the incidence of bottle feeding in infants bears a direct relationship to the mortality rate in congenital hypertrophic pyloric stenosis, as in healthy children.

**Summary.**

Various factors have been shown to greatly affect the mortality rate, irrespective of the method of treatment employed. If the mortality rate is to be reduced, more attention must be paid to these factors and rather less to the actual treatment employed.
Many clinicians are apt to leave behind, after an apparent cure, symptoms that may easily be missed unless a large number of cases are followed up.

Number of cases followed up.

94 cases have been followed up.

24 of the original 128 cases admitted, died in hospital. Out of the 104 cases discharged alive, 10 were unable to be traced.

3 cases had since died. 91 cases were examined by myself during the investigation.

Barium meal examinations were performed on 36 of these cases.

Notes were made of the following symptoms and signs:-

1. If the mother considered the child to be unduly nervous.
2. State of appetite.
3. Presence or absence of vomiting after discharge from hospital.

Examination.

1. General appearance of patient.
2. Mental stability.
3. Height.
4. Weight.
5. Head circumference.
7. Presence or absence of abdominal tenderness.

In order to simplify the assessment of the results of the investigations, they are grouped under three headings.

1. General appearance.
2. Mental state.
3. Alimentary system.

A. Surgical Cases.

56 cases were followed up, all of whom had had a Rammstedt operation performed.

19 of these cases have had barium meal examinations.

Ages ranged from 3 - 10 years.

3 of these patients have died since being discharged from hospital.

(1) Case no. 10 R.R. Died aged 5 years. Cause unknown.
(2) Case no. 50 S.M. Twins. Died of lobar pneumonia aged 3 years.

General appearance/
General appearance.

All the patients appeared healthy, with the exception of one child, case no. 20 E.J.B., who had typical symptoms and signs of Sydenham's chorea.

Mental state.

8 cases had a history of being "highly strung". Confirmation of these symptoms were noted at examination. These cases were all males, and 5 were only children.

Alimentary system.

3 patients had had poor appetites since being discharged from hospital. 2 of these (case no. 34 D.M. and case no. 54 K.M.) were among the group of nervous patients.

1 patient, case no. 12 D.B., had a well marked post operative hernia. (Case notes revealed that the wound had become septic immediately after operation.) Otherwise all the scars were satisfactory.

B. Medical Cases.

35 cases were followed up.
17 of these cases had barium meal examinations.
Ages ranged from 6 months - 3 years.
None of the cases have died since being discharged from hospital.
General appearance.

All the patients looked particularly healthy. The average height and weight was above the normal for the respective ages.

Mental state.

Undue nervousness was noticed in only one case - no. 87. B.R. aged 3.

Summary.

The general health of these cases was excellent, in fact better than one would expect in a similar number of control cases. This was particularly noticeable in the eumydrin series who were, of course, nearly all still babies.

A nervous temperament was present in 8 out of the 56 surgically treated cases. At first sight, this may appear as being rather higher than would be expected in a similar series of controls. However, consideration must be taken of the fact that the parents of these children would tend to be over careful of their health after such a serious illness. This surely, will account for the nervousness without attributing it to the actual condition from which the children had suffered. The particularly high weight found in most of the cases conforms with this view.
The scars of the operation have left weakness of the abdominal wall in only one case, and this can be attributed to postoperative sepsis. Thus the operation, per se, cannot be regarded as affecting the prognosis, with regard to the development of ventral hernias.

Conclusions.

Once a patient suffering from congenital hypertrophic pyloric stenosis, has been satisfactorily treated by whatever method, then the prognosis is as good as that of any healthy child.

Also, in my opinion, there is no undue tendency for these patients to develop a nervous disposition.

These conclusions confirm the opinion expressed by both Bau and Oehler, who followed up 72 and 36 cases respectively, which had been treated surgically, and found all the patients in perfect health.
Radiological Results.

Several series of cases of congenital hypertrophic pyloric stenosis have been followed up in different hospitals, but little attention has been paid to the radiological examination of the stomach. For this reason, I thought it might be of interest to perform barium meal examinations upon as many cases as possible. Unfortunately, only 36 out of the total 91 cases were able to report at the hospital for this investigation.

Results in surgical cases.

In 8 out of the 19 cases investigated, no abnormality was found.

In the remaining 11 cases, abnormalities were found. These are reported in detail below, with the aid of photographs of the X-ray films.

Case no. 11 J.R. aged 8 years.

Some degree of contraction of the pyloric end of the stomach, suggesting seaming of the wall itself.

No gastric delay in emptying.

Case no. 13 P.S. aged 8 years.

Irregularity and contraction at the pyloric end of the stomach.

No gastric delay in emptying.
Case no. 16 A.C. aged 8 years

There is contraction of the pyloric end of the stomach and the beginning of the duodenum appears to take a descending curve, forming a small residue of barium in its dependent portion, but no gastric delay in emptying.

A-P view.
Immediate film.

Oblique view.
Immediate film.
Case no. 18 B.H. aged 9 years.
The pylorus was very stiff and contracted and at first, filling of the duodenal cap was difficult to obtain, but later it appeared to fill satisfactorily and was normal in appearance.

Case no. 21 T.K. aged 8 years.
Slight residue at the end of 6 hours. Definite contraction of the pylorus, though the duodenal cap was fairly easily filled.

Case no. 28 C.C. aged 6 years.
Definite contraction of the pyloric end of the stomach.
No delay in gastric emptying.

Case no. 33 D.B. aged 5 years.
Slight irregularity of the pyloric end of the stomach.
No delay in gastric emptying.
Case no. 34 D.M. aged 5 years.

The pyloric end of the stomach was narrowed.

No delay in gastric emptying.

Immediate film

6 hour film
Case no. 53 J.W. aged 4 years.

There is definite contraction at the pyloric end of the stomach. Also slight delay in gastric emptying, seen by slight 6 hour residue. Compare with films taken as a baby prior to operation.

Immediate film, follow up.  
6 hour film, follow up.  
6 hour film as a baby prior to operation. Note marked 6 hour delay.
Case no. 54 K.M. aged 5 years.

There is deformity of the pyloric end of the stomach suggesting adhesions.
No delay in emptying.

Case no. 55 K.H. aged 4 years.

Contraction of the pyloric end of the stomach with difficulty in filling of the duodenal cap, but no delay in gastric emptying.

Results in medical cases.

Abnormality was found in 4 out of the 17 cases investigated. These also are reported in detail below, with the aid of photographs of the X-ray films.
Case no. 105 C.C. aged 1 year and 7 months.

There is definite contraction at the pyloric end of the stomach, though the duodenal cap fills quite well. Gastric emptying is definitely slow. Compare with film taken before treatment was commenced.

3 hour film, follow up.

3 hour film as a baby aged 4 weeks. Note marked delay in gastric emptying.
Case no. 106  D.T. aged 1 year 7 months.

There is slight contraction at the pyloric end of the stomach, but there is no delay in emptying.
Compare with film taken before treatment commenced.

5 hour film, follow up.

3 hour film.
Baby aged 5+ weeks.
Note definite delay in emptying.
Case no. 111  K.S. aged 7 months.

The pyloric end of the stomach seems contracted, and the stomach rather dilated, but there is no delay in emptying. Compare with film before treatment commenced.

2 hour film, follow up.

5 hour film, follow up.

3 hour film as a baby aged 6 weeks. Note marked residue.
Case no. 112 B.H. aged 8 months.

There appears to be a little contraction still present at the pyloric end of the stomach, but there is no gastric delay in emptying.

Compare with film taken before treatment commenced.

3 hour film, follow up.

3 hour film as a baby aged 7 weeks. Note marked residue.
Conclusions.

In 40% of the surgically treated cases, deformities were found in the pyloric end of the stomach during these investigations. This might be expected after any operation in which adhesions are apt to subsequently form. However in only one case was slight delay in gastric emptying present. Thus, in my opinion, the Rammstedt operation leaves no permanent dysfunction of the alimentary tract.

In the eumydrin series, abnormalities were found in only 4 cases in these investigations. In only one of these cases was delay in gastric emptying still present. This child was 1 year and 7 months, which shows that the pyloric canal may take a long time to return to normal.

Finally these radiological investigations confirm the conclusions arrived at by the clinical investigations, that after successful treatment of a case of congenital hypertrophic pyloric stenosis, the outlook is as good as that of a healthy infant.
ADVANTAGES AND DISADVANTAGES OF SURGICAL AND MEDICAL TREATMENT.

Certain factors, which will now be discussed, must influence one in deciding upon the method of treatment to be employed.

Length of time in hospital.

The comparatively long period of hospitalisation found necessary in the treatment by eumydrin therapy, has been considered to be the chief disadvantage of this method. Swensgaard's cases had an average period of 77 days, while in my series, although it had been reduced to 41 days, this was 13 days longer than that of the surgical cases.

The considerable difference in the period of hospitalisation has led to an increase in the liability to intercurrent infection in the medical series, especially acute gastro-enteritis.

Expense of treatment.

The long period of hospitalisation of those treated by eumydrin increases the expense of treatment of each patient. Also the necessity for isolation of these cases in cubicles, raises the cost considerably.

Feeding/
Feeding difficulties.

I have attempted to show previously, that breast feeding plays an important part in reducing the mortality of these cases. If the baby is left in hospital for only a few days, the risk of causing cessation of breast feeding is small. It is very difficult indeed, however, to retain the supply of breast milk over several weeks, if most of the milk has to be drawn off by a breast pump.

Surgical skill required.

The one formidable disadvantage of surgical treatment is the high degree of technical skill required to perform the Fredet-Rammstedt operation. This is easily obtained in the large cities where surgeons specialise in child surgery, but in the smaller provincial towns the expert skill is often lacking. Thus the geographical situation of a practice is apt to play a part in influencing a physician as to the method of treatment to be employed. However the greater nursing skill required in eumydrin therapy must be kept in mind when considering the type of hospital available.

Eumydrin failures.

Surgery to-day offers a fairly low mortality rate/
rate. Parsons (2) considers that the mortality rate, with good post operative treatment, should not exceed 5%. Eumydrin, although in some cases produces an equally low mortality rate, does fail completely to effect a cure in a number of cases, 12% in the series investigated by me. The possibility of this failure of eumydrin therapy, must be always kept in mind by its adherants.
CONCLUSIONS AS TO THE IDEAL FORM OF TREATMENT.

The chief disadvantage of eumydrin therapy has been the prolonged stay in hospital. Dobbs and Vertue have treated cases as out-patients with considerable success. If this continues to be found satisfactory, the chief disadvantage of eumydrin therapy will be removed.

In my opinion, eumydrin therapy should be tried in all cases for from one week to ten days, according to the degree of dehydration on admission. If improvement is not obtained by that time, a Fredet-Rammstedt operation should be performed without delay. The decision of changing the treatment should be based on three factors:

1. The extent of the vomiting and constipation.
2. The extent of the gastric residue.
3. Whether the patient has gained weight or not.

The most difficult decision to make is exactly how long to continue the eumydrin therapy, only a slight guide has been offered, as each case must be gauged separately on the physician's judgement. However, if surgery is employed, the post operative treatment of the patient should be carried out under the physician primarily, although of course, full cooperation with the surgeon is essential for success.
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(1) I, 739.
(2) I, 747.


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<th>Age admitted (weeks)</th>
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<th>Sex</th>
<th>Weight at birth (lbs.)</th>
<th>Weight on admission (lbs.)</th>
<th>Type of feeding</th>
<th>Signs on examination</th>
<th>Condition</th>
<th>Cases X-rayed</th>
<th>Treatment</th>
<th>Result alive or dead</th>
<th>Days in hosp.</th>
<th>Cases followed up</th>
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<td>Result</td>
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Br. = Breast fed.
P.V. = Projectile vomiting.
P. = Palpable tumour.
T. = Tumour.
F. = Constipation.
Sym. = Symptomatic.
Syntr. = Syntropan.
Eum. = Eumydrin.
X-rayed = X-rayed.