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 spas-:tic 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 prom-:inence 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 1788 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 con-:dition was peculiar to infancy. Upon Finklestein falls the honour of describing a palpable tumour in a living case of congenital hypertrophic pyloric stenosis for/

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 oppor-:tunity 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 reexam-
:ined 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 at-
:tempt 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 $3\frac{1}{2}$ weeks.   The symptoms never commenced before the seventh day in the earliest case, whilst the latest date of onset was at $7\frac{1}{2}$ 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 Feer 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/

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 high-
:er 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 oper-
:ated 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 occur-
:ring 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.   Pit-
:field 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 de-
:duces 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-bearing 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 hyper-:trophic 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 brief-:ly 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 con-:dition 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 hyper-:trophy 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 ex-
planation 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 fail-
:ure 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 pyloro-
:spasm, which will be described later, has many
essential features that differ from those of congeni-
:tal hypertrophic pyloric stenosis.   In my opinion
they are separate conditions.

Summary.

A brief resumé 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 con-
:genital hypertrophic pyloric stenosis, where a pal-
:pable tumour is still found weeks later.   This
finding, in my opinion, corroborates the hypothesis
advanced by John Thomson (2), that spasm is the pri-
:mary factor whilst hypertrophy of the musculature of
the pyloric canal is secondary.

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 character-:istic 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, re-
:presenting 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 eluci-
:date 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 import-
:ant 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 per-
:iods.   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 vomit-
:ing 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 treat-
:ment - is steady without intermission, and is
often very alarming.

IV   General appearance.

When the disease fully develops, the facies

is/

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.   Occasion-:ally 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 pres:ence or absence of a tumour, a proper technique should be employed. As mentioned above, the exam:ination 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 dis:turbed 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, palpa-:tion 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 seg-:ment 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 oper-:ation, 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 surg-
:ically 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 examina-
:tion 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 con-
:clusion 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% solu-
tion 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 re-
:turned 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 congeni-
:tal 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 observa-
:tion 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 posi-
:tion 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 con-
:genital hypertrophic pyloric stenosis, but is prob-
:ably 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 con-
:siderable 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 suffer-
:ing 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 in-
:gestion 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 com-
:paratively little may have entered the duodenum.
Usually/

Usually it was found in cases of congenital hyper-
:trophic 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 con-
:siderable 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 con-
:genital 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 surgi-:cal 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 $3\frac{1}{2}$ - 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 diag-
:nosis 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 diag-
:nosis/

: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 investi-:gated by me, the diagnosis of congenital hyper-:trophic 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 diag-:nosis 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 diag-:nosis 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.

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 con-:stipation is a less conspicuous feature. Examina-:tion 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 differ-:entiation 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 peris-:talsis 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 sten-:osis 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 vora-
:cious 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 ex-
:cluded rapidly if a good clinical history is ob-
:tained.

TREATMENT.

A. Medical.

Although congenital hypertrophic pyloric
stenosis had been recognised as a clinical entity for
many years, it was not until 1913 that really success-
:ful results were obtained when Rammstedt (1) intro-
:duced the operation that is used to-day.

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 con-
:genital hypertrophic pyloric stenosis cured by means
of atropine. He gave 2 drops of 1/1000 solution
atropine sulphate 4 - 5 times a day, usually pre-
:ceded 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 -
14/

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 treat-
:ment 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 sub-
:stituted 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).

$$CH_2 - CH - CH_2$$
$$NCH_3 \quad CH - O - OC - CH - CH_2OH$$
$$CH_2 - CH - CH_2$$

(I)

$$CH_2 - CH - CH_2$$
$$CH_3 - N \overset{NO_3}{\underset{CH_3}{\big\langle}} \quad CH - O - OC - CH - CH_2OH$$
$$CH_2 - CH - CH_2$$
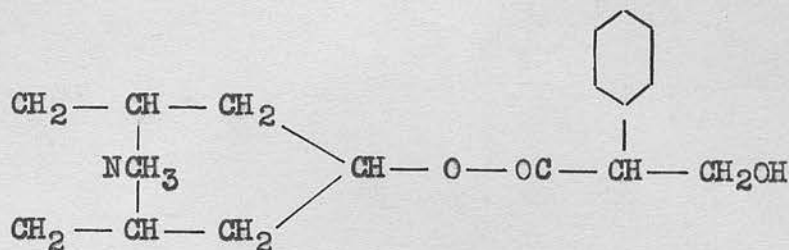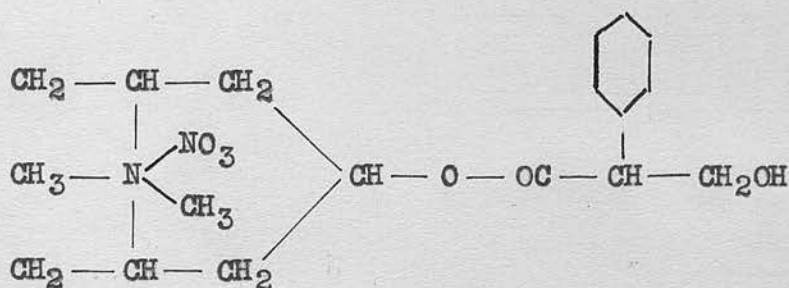
(II)

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 parti-
:culars or references are given.

Technique/

Technique employed in medical treatment with
eumydrin.

The technique used in my series was a modi-
:fication 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 (.9%) were given subcutan-
:eously 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 sal-
:ines, 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 de-
:hydration.   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 mal-
:leolus 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 em-
:ployed.   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 accomodate 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 $2\frac{1}{2}$ oz. per pound body weight.   If dehydra-:tion was marked, supplementary feeds of glucose in water were given together with the saline in-:fusions, 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"Lacidac" was usually employed.   No sugar was added as this production already con-:tains 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 em-
:ployed 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 insti-
:tuted in Leicester Royal Infirmary, separate
glass cubicles were built chiefly for isolation
of these cases.   Also a rule was made that any-
:one 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.

TREATMENT.

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 as-
:cribed 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 hosp-
:ital.)

There were 8 deaths, which will now be des-
:cribed 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 dehydra-
tion.   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 admis-
:sion.

(7) Case no.116. A.A.   Marasmic state on admission.
Died on 3rd day after admis-
:sion.   Eumydrin therapy for
36 hours.

(8) Case no.123. J.H.   Failed to respond to eumydrin
therapy after 24 days treat-
:ment.   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 ex-
:cluded 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 ther-:apy, 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 antispasmod-:ic/

: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)/

(4) Case no. 23. R.W.   Admitted in very dehydrated
state.   Rammstedt operation
performed on same day.
Died a few hours later.

(5) Case no. 24. C.T.   State on admission satisfac-
:tory.   Rammstedt operation
performed.   Developed broncho-
pneumonia.
Died 3 days after admission.

(6) Case no. 25. E.G.   State on admission satisfac-
:tory.   Rammstedt operation
performed.   Developed acute
gastro-enteritis.
Died 9 days after admission.

(7) Case no. 26. H.C.   Admitted in dehydrated state.
Intrafontanelle blood trans-
:fusion given.
Died on 7th day after Ramm-
stedt 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 opera-
:tion/

:tion, 4 days after admission.
Post mortem examination re-
:vealed a large intraperiton-
:eal 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 per-
:foration 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.

(13)/

(13) Case no. 82. R.P.   State on admission satis-
                            :factory.
                            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 post-operative 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 assist-
ance 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 Wevill 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 satisfac-
:tory 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 pul-:monary 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 in-fection 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 med-:ical 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/

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 in-
:cidence 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 pan-
creatic insufficiency.   He suggests that the dia-
rrhoea is attributable to this pancreatic insuffi-
ciency.   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 follow-
:ing a perforation of the mucous membrane.　This is
probably the most common and most dreaded danger in
the whole operation.
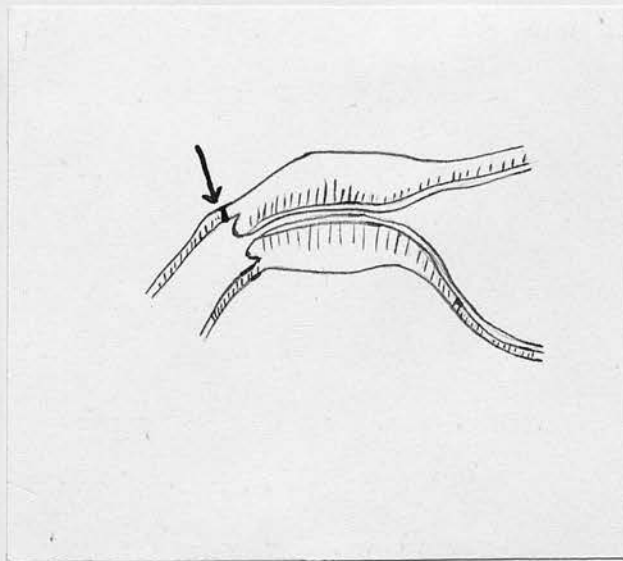
Arrow indicates usual site
of perforation of the mucous membrane.

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 oper-:ation 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/

This tragedy occurred in spite of great care being
taken.   Until recently, this method was extensively
used for giving blood transfusions and saline in-
:fusions, 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 dis-
:continued 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.

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

Medical Treatment/

Medical Treatment.

Year	Paediatrician	No. of cases	Mortality.
1929	Bayer	40	2.5%
1930	Rammstedt	1345	15%
1935	Swensgaard	61	1.6%
1939	Dobbs	20	5%
1939	Findlay (3)	6	0%
1939	Vertue	21	4.8%

To these may be added the series collected by Swensgaard:-

Year	Paediatrician	No. of cases	Mortality.
1921	Einberg & Hamilton	57	3.5%
1922	Ibrahim	52	1.9%
1928	Monrad	228	4.4%
1930	Wolff	98	2.2%
1933	Faxen	126	3.3%

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 remember-:ed 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/

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 it-
:self 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 em-
:ployed to-day.   Thus it is apparent that the diag-
:nosis of many of the medical cases was grossly in-
:accurate; a large number of cases being really
functional in origin, namely, suffering from pyloro-
:spasm.   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.

Clinician	weight at birth		weight on admission		age on admission	No. of weeks between onset of symptoms and admission.	no. of cases	mortality rate
	lbs.	oz.	lbs.	oz.				
Thompson & Gaisford	a. 8 d. 7	4 1	6	15	6 wks. 1 day	-	128	16.4%
Wardill	-		6	8	6 wks.	3	123	23.5%
Lanman & Mahoney	-		-		4 wks.	-	425	6.3%
Hipsley & Tait	-		7		$5\frac{1}{2}$ wks.	$1\frac{1}{2}$	120	4%
Wallace & Wevill	a. 8 d. 8	8 8	6	8	$6\frac{1}{2}$ wks.	$3\frac{1}{2}$	145	24.8%
Own series	a. 6 d. 7	13 1	6	13	$5\frac{1}{2}$ wks.	$1\frac{3}{4}$	128	18.5%

Birth/

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 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 prog-:nosis 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 - $6\frac{1}{2}$ 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 mort-:ality 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 relation-:ship which these figures bear in the different series.
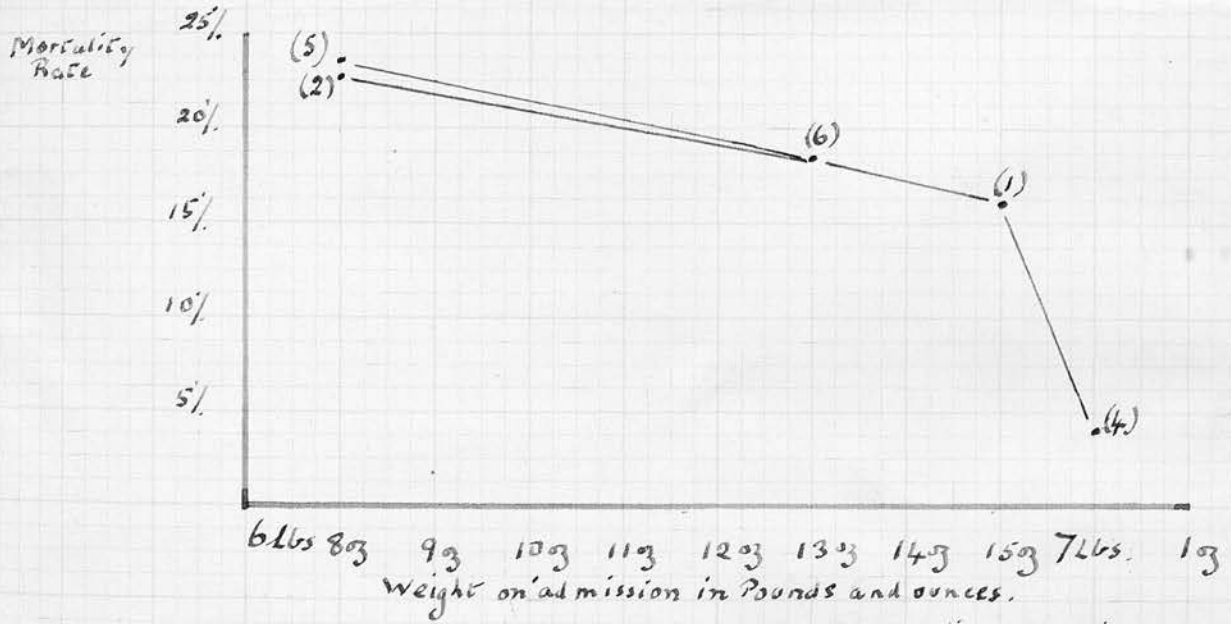
Graph no: I.

(1) = Thompson & Gaisford's series.
(2) = Wardill's series.
(4) = Hipsley & Tait's series.
(5) = Wallace & Wevill's series.
(6) = "own" 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. no: II.
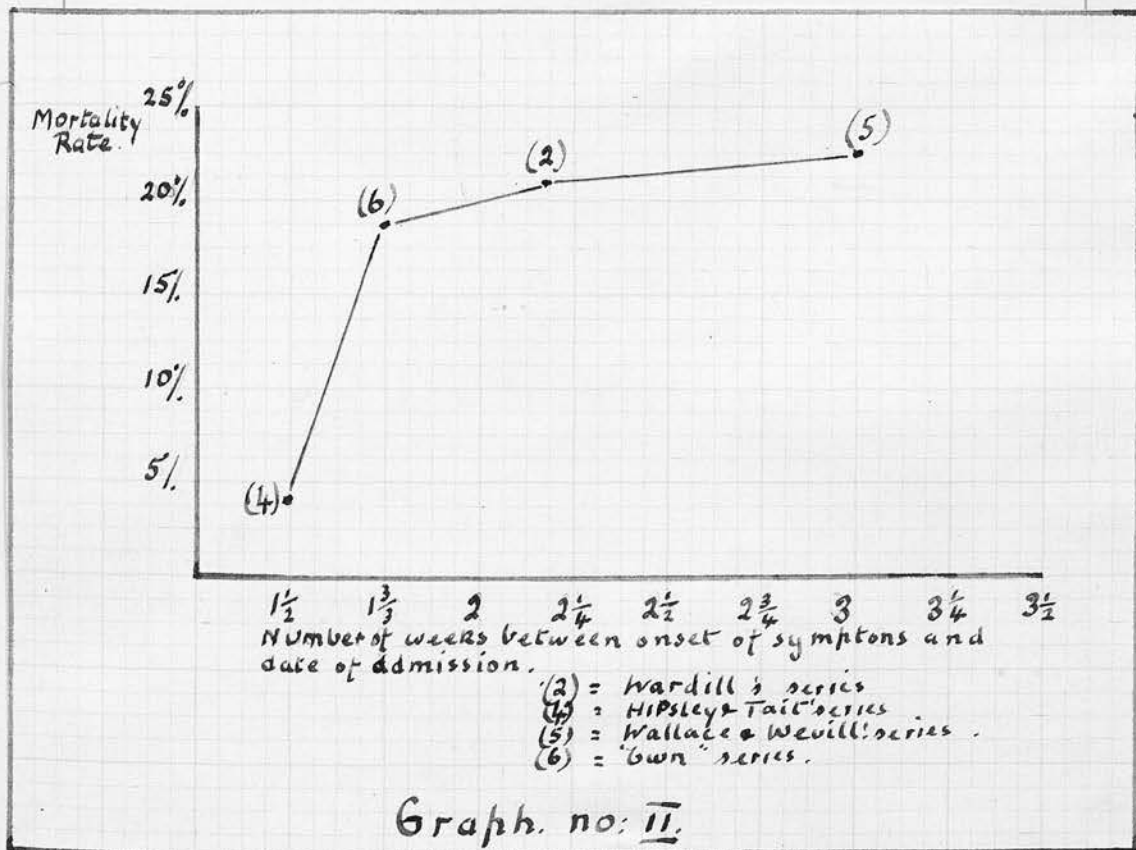
Influence of Types of Feeding Employed.

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 per-:centage of cases breast fed with the mortality rate in a few different series, irrespective of the treat-:ment employed.

Paediatrician	No. of cases	Entirely breast fed	Partly breast fed	Entirely bottle fed	Mortality rate
Swensgaard	179	55%	-	-	5.5%
Jewesbury & Page.	144	55%	35%	10%	12.5%
Own series	128	33.6%	21.9%	44.5%	18.5%

The number of series of cases obtained is too small to draw definite conclusions, but it is inter-:esting to note that my own series had a relatively high percentage of bottle fed babies with also a relatively high mortality rate.

In/

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 relation-
:ship 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 em-
:ployed.

STUDY OF THE PROGRESS OF CASES TREATED
DURING THE LAST TEN YEARS.

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 exam-:ined 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.

4. State of bowels.

Examination.   1.   General appearance of patient.

2.   Mental stability.

3.   Height.

4/

4. Weight.

5. Head circumference.

6. State of abdominal scar.

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 dis-
:charged from hospital.

(1) Case no. 10 R.R.   Died aged 5 years.
                        Cause unknown.

(2) Case no. 50 S.M.   Twins.   Died of lobar
    Case no. 51 S.M.   pneumonia aged 3 years.

General appearance/

General appearance.

All the patients appeared healthy, with the
exception of one child, case no. 20 B.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 examina-
:tion.   These case 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 opera-
:tion.)   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 dis-
:charged from hospital.

General appearance/

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.   How-
:ever, 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/

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 tend-:ency for these patients to develop a nervous dis-:position.

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 hyper-
:trophic 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/

Case no. 16 A.C. aged 8 years

There is contraction of the pyloric end of
the stomach and the beginning of the duo-
:denum appears to take a descending curve,
forming a small residue of barium in its
dependent portion, but no gastric delay
in emptying.
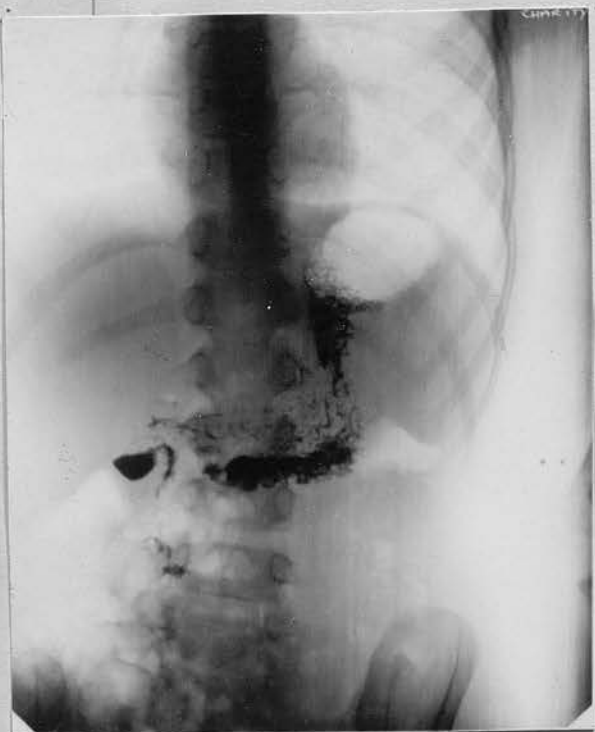
A-P view.
Immediate film.
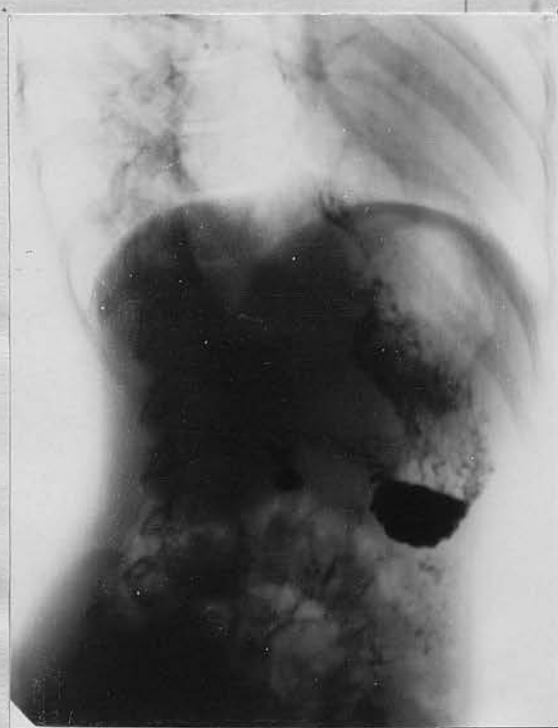
Oblique view.
Immediate film.

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/

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.
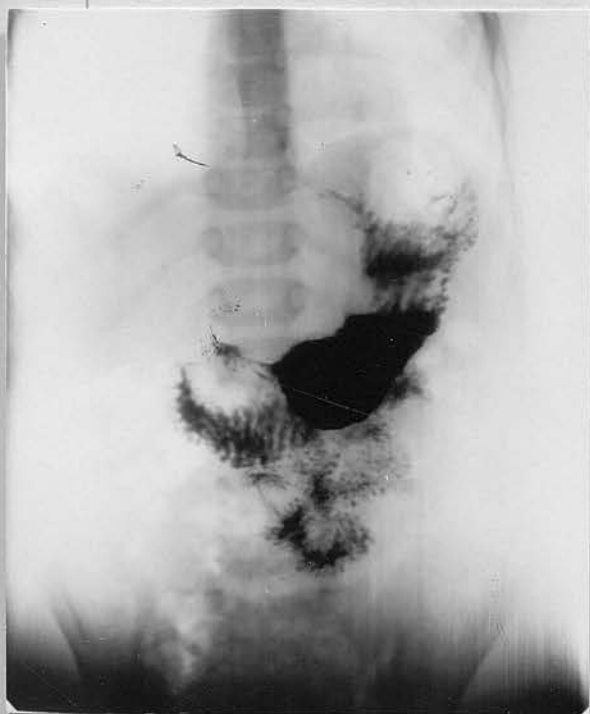
Immediate film,
follow up.

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/

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.
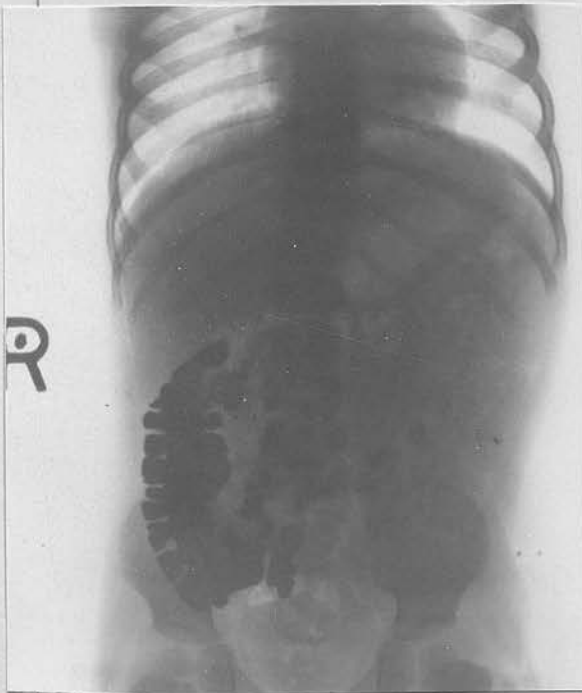
5 hour film,
follow up.
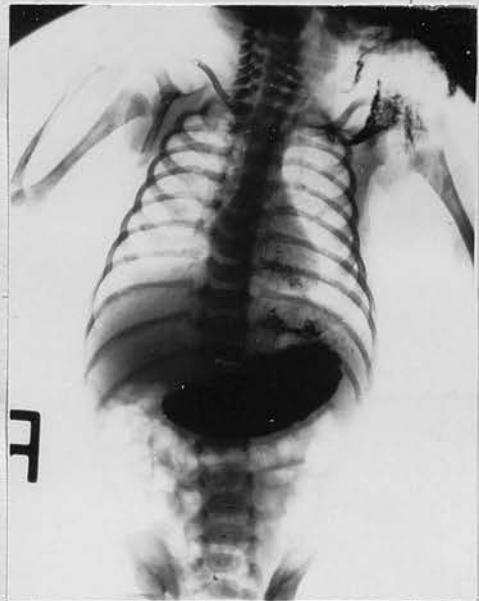
3 hour film.
Baby aged 5½ weeks.
Note definite delay
in emptying.

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.
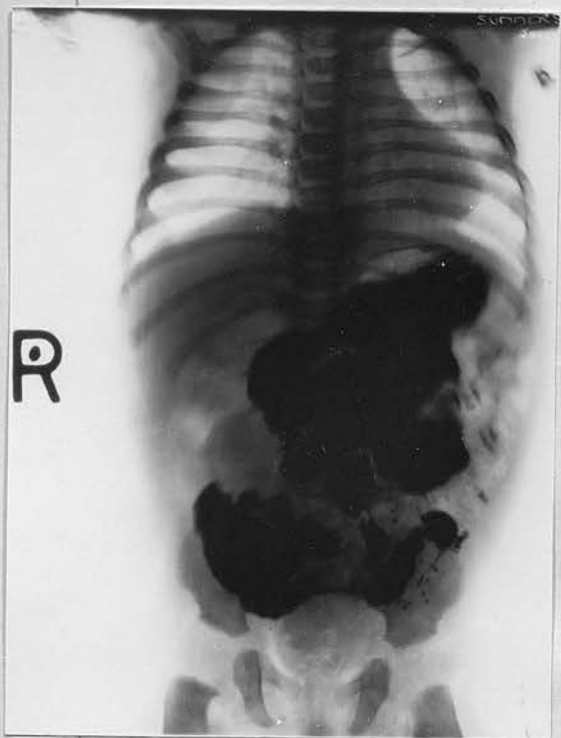
2 hour film,
follow up.

5 hour film,
follow up.
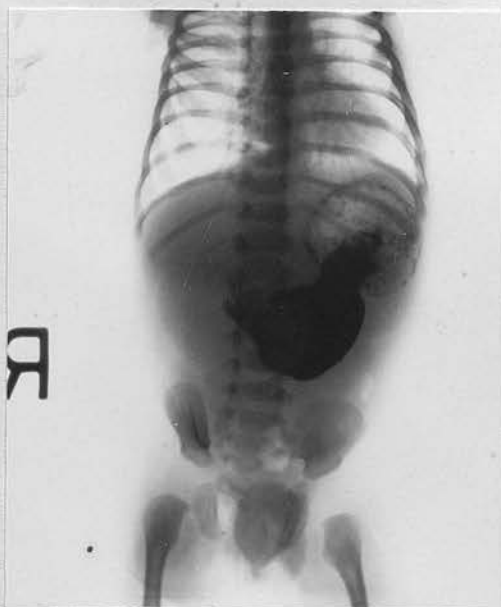
3 hour film as a
baby aged 6 weeks.
Note marked residue.

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.
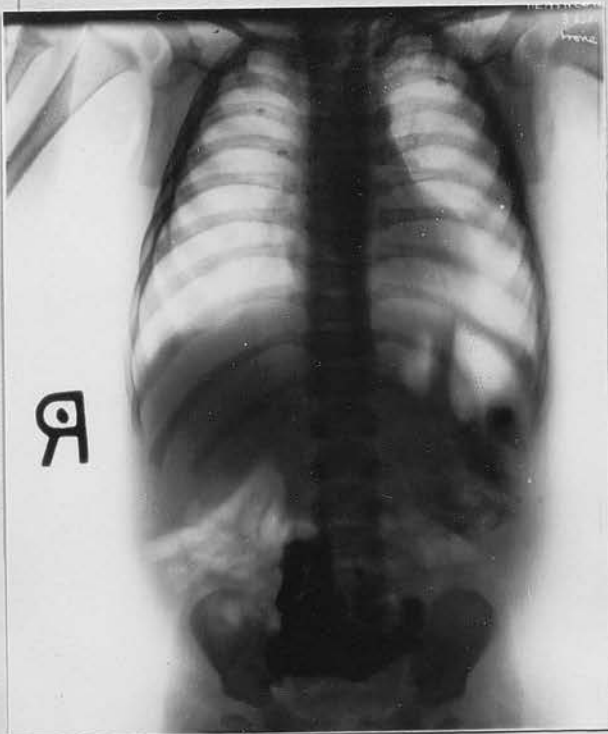
3 hour film,
follow up.

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

Conclusions.

In 40% of the surgically treated cases, deform-
:ities 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 perman-
:ent 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 con-
firm 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 treat-:ment 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 re-
:quired 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 prac-
:tise 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 con-
:siderable 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 im-
:provement 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 co-
:operation with the surgeon is essential for success.

BIBLIOGRAPHY.

ARMSTRONG, George (1767) An essay on the diseases
        most fatal to infants.   Cadell, London.
        p. 60.

BAU, A. (1937) Kinderärztl. Prax. VIII, 277.

BAYER, W. (1929) Dtsch. med. Wschr. LV, 2049.

BEARDSLEY, H. (1788) New Haven J. Meigs. (Reprint
        by Osler, W. (1903) Arch. Pediat. XX,
        355.)

BILDERBACK, J. B.   Quoted by Thompson, W. A. &
        Gaisford, W. F. (1935) Brit. med. J.
        ii, 1037.

BLAIR, Patrick (1717) Phil. Trans. no. 353, 631.

BRUCE, J. W. (1923) South. med. J., Birmingham, Ala.
        XVI, 123.

BULL, H C. H. (1935) X-ray interpretation.   London.
        p. 213.

CAUTLEY, E. (1919) Brit. J. Child. Dis. XVI, 65.

COCKAYNE, E. A. (1938) Arch. Dis. Childh. XIII, 75.

DOBBS, R. H. (1939) Lancet. i, 12.

DONOVAN, E. J. (1937) J. Amer. med. Ass. CIX, 558.

DRUCKER, Paul.   Quoted by Swensgaard, E. (1935)
        Arch. Dis. Childh. X, 443.

ESTELLA Y B. DE CASTRO, J. (1926) Arch. Espan. de
        Pediat., Madrid. X, 129.

FEER/

FEER, -   Quoted by Thompson, W. A. & Gaisford, W. F.

(1935) Brit. med. J. ii, 1037.

FINDLAY, L.

(1) (1937) Arch. Dis. Childh. XII, 399.

(2) (1938) Arch. Dis. Childh. XIII, 145.

(3) (1938) Brit. med. J. i, 538.

FINKELSTEIN, H. (1896) Jahrb. Kinderheilk. XLIII,

105.

FRANK, E. S. (1931) Ned. Maandschr. Geneesk., Leyden.

XVII, 495.

FRIEMANN-DAHL, J. (1935) Acta Radiol., Stockh.

XVI, 333.

FROMHERZ, K. (1937) J. Pharmacol. LX, 1.

GRAY, H. Tyrrel & REYNOLDS, F. N. (1921) Brit. med.

J. ii, 891.

HARRIS, C. & KEYNES, G. (1937) St. Barts. Hosp. Rep.

LXX, 43.

HIGGINS, G. Twistington & PATERSON, D. (1926) Arch.

Dis. Childh. I, 285.

HIPSLEY, P. L. & TAIT, L. G. (1934) Brit. med. J.

ii, 833.

HIRSCHPRUNG, - (1888) Jahrb. Kinderheilk. XXVII, 1.

JEWESBURY, R. C. & PAGE, C. M. (1937) St. Thomas's

Hosp. Rep. 2nd ser. II, 141.

JOCHIMS, J. (1937) Ztschr. Kinderheilk. LVIII, 796.

JOHANNESSEN, A. (1924) Ugeskr. f. Laeger, Copenhagen.

LXXXVI, 113.

KÖHLER/

KÖHLER, A. (1935) Roentgenology. 2nd Engl. ed.
London. p. 524.

LANMAN, T. H. & MAHONEY, P. J. (1933) Surg. Gynec.
Obstet. LVI, 205.

LAPAGE, C. P. (1932) Brit. med. J. ii, 350.

LEVI, D. (1) (1933) Practitioner, CXXX, 67.
(2) (1938) Brit. med. J. i, 486.

MAIZELS, M. (1931) Arch. Dis. Childh. VI, 293.

MEAGHER, J. L. (1926) Brit. med. J. i, 87.

MOORE, L. H. (1924) South. med. J., Birmingham, Ala.
XVII, 187.

OEHLER, J. (1934) Zbl. Chir. LVI, 611.

PARSONS, L. G. & BARLING, S. (1933) Diseases of
infancy and childhood. London.
(1) I, 739.
(2) I, 747.

PITFIELD, R. L. (1925) Med. J. & Rec. CXXII, 148.

POYNTON, F. J., HIGGINS, J. T. & BRYDSON, J. M. (1924)
Lancet, i, 215.

RAMMSTEDT, C. (1) (1913) Zbl. Chir. XL, 3.
(2) (1930) Dtsch. med. Wschr. LVI,
348.

RODRIGO, D. (1926) Arch. Espan. de Pediat., Madrid.
X, 165.

RUNSTRÖM, G. (1937) Acta Radiol., Stockh. XVIII, 524.

SECKEL, H. Quoted by Swensgaard, E. (1935) Arch.
Dis. Childh. X, 443.

SHELDON/

SHELDON, W. (1936) Diseases of infancy and childhood.
2nd ed.   London.   (1) p. 109.
(2) p. 110.
(3) p. 112.

SWENSGAARD, E.   (1935)   Arch. Dis. Childh.   X, 443.

TEALL, C. G.   Parsons, L. G. & Barling, S.   Diseases
of infancy and childhood.   2nd ed.
London.   Vol. II, p. 1702.

THOMPSON, W. A. & GAISFORD, W. F. (1935)   Brit. med.
J. ii, 1037.

THOMSON, J.   (1)   (1895)   Brit. med. J. ii, 711.
(2)   (1902)   Brit. med. J. ii, 678.

THOMSON, J. & FINDLAY, L. (1933)   Clinical study
and treatment of sick children.   5th ed.
London.   p. 344.

TRIBBLE, C. E. (1933)   J. Amer. med. Ass.   CI, 278.

VERTUE, H. St. H. (1939)   Arch. Dis. Childh.   XIV,
173.

WALLACE, H. L. & WEVILL, L. B. (1934)   Brit. med. J.
i, 1154.

Case no. and initials	Year of case	Age symptoms commenced (weeks)	Age admitted (weeks)	Place in family	Sex	Weight at birth (lbs.)	Weight on admission (lbs.)	Type of feeding	Symptoms P.V.	C.	Signs on examination Dehydr.	P.	T.	Cases X-rayed	Treatment. Surg.	Cases with tumour at O.P.	Med.	Result. alive or dead	Days in hosp.	Cases follow-ed up	Ht. in ins.	Wt. in Lbs.	Cases re X-rayed
1.J.B.	1929	1½	2	1	M	7	6	Bot.	+	+	+	+	+	−	+	+		A	20	−			−
2.F.C.	1929	3½	5	4	M	8	6,13	Bot	+	+	++	+	+	−	+	+		D	1				
3.R.B.	1929	11	12	1	M	7,4	6,12	Br.	+	+	+	+	+	−	+	+		A	14	+	50½	64	−
4.F.F.	1929	4	4	1	M	8,6	7,2	Bot.	+	+	+	+	+	−	+	+		D	7				
5.H.R.	1929	5	6	1	M	8,4	7,0	Bot.	+	.+	+	+	+	−	+	+		A	10	+	51	67	−
6.A.C.	1929	3	6	1	M	7,6	6,2	Bot.	+	+	+	+	+	−	+	+		D	5				
7.T.G.	1929	6	10	1	M	6,12	6,14	Bot.	+	+	−	+	+	−	+	+		A	24	+	50	65	−
8.J.F.	1929	5	7	1	M	8,2	7,4	Br.	+	+	−	+	+	−	+	+		A	14	+	52½	60	−
9.B.J.P.	1930	2½	3	1	M	7,0	6,0	Bot.	+	+	−	+	+	−	+	+		A	11	+	53	65	+
10.R.B.	1930	5	10	1	M	7,2	6,13	Bot.	+	+	+	+	+	−	+	+		A	10	+	Died	aged 5 years.	
11.J.R.	1930	3	6	1	M	7,7	7,0	Br.	+	+	+	+	+	−	+	+		A	11	+	50	62	+
12.D.J.	1930	1	3	1	M	7,10	6,12	Bot.	+	+	+	+	+	−	+	+		A	34	+	51	60	+
13.P.S.	1930	2	12	1	M	8,0	7,10	Bot.	+	+	+	+	+	−	+	+		A	32	+	55½	63½	+
14.M.B.	1931	5	6	1	M	9,0	7,4	Br.	+	+	−	+	+	−	+	+		A	10	−			−
15.D.A.N.	1931	3	4	1	M	7,4	6,5	Bot.	+	+	−	+	−	−	+	+		A	12	+	45	51	+
16.A.C.	1931	4	5	1	M	7,3	6,6	Br.-Bot.	+	+	+	+	−	−	+	+		A	27	+	51	49¼	+
17.M.P.W.	1931	3	6	1	M	8,7	7,2	Br.-Bot.	+	+	−	+	+	−	+	+		A	11	+	51	63½	−
18.B.H.	1931	4	7	1	F	8,2	6,3	Br.	+	+	+	+	−	−	+	+		A	24	+	49	47½	+
19.F.W.	1931	3	5	1	F	6,13	5,12	Bot.	+	+	++	+	−	−	+	+		A	19	−			−
20.B.J.B.	1931	8	16	1	M	7,0	6,10	Br.	+	+	+	+	+	−	+	+		A	24	+	50	66	+
21.T.K.	1931	1½	2	2	F	8,0	7,4	Br.	+	+	+	+	+	−	+	+		A	23	+	47½	46½	+
22.D.J.	1931	3½	5	1	M	8,1	7,0	Bot.	+	+		+	+	−	+	+		A	12	−			−
23.R.W.	1932	2	2½	1	M	7,6	6,7	Bot.	+	+	++	+	+	−	+	+		D	1				
24.C.T.	1932	1½	2	3	M	7,5	6,13	Bot.	+	+	+	+	+	−	+	+		D	3				
25.E.G.	1932	3	4	1	F	7,6	6,5	Br.-Bot.	+	+	+	+	+	+	+	+		D	9				
26.H.C.	1932	5	10	1	M	7,4	6,10	Bot.	+	+	+	+	+		+	+		D	1				
27.T.F.	1932	1½	13	1	M	7,9	6,9	Br.-Bot.	+	+	+	+	−	+	+	+		A	22	+	48½	52¾	

Case no. and initials	Year of case	Age symptoms commenced (weeks)	Age admitted (weeks)	Place in family	Sex	Weight at birth (lbs.)	Weight on admission (lbs.)	Type of feeding	Symptoms P.V.	C.	Signs on examination Dehydr.	P.	T.	Cases X-rayed	Treatment. Surg.	Cases with tumour at O.P.	Med.	Result alive or dead	Days in hosp.	Cases follow-ed up	Ht. in ins.	Wt. in lbs.	Cases re X-rayed
28.C.C.	1932	3	4	2	M	7,0	6,14	Br.	+	+	+	+	-	-	+	+		A	14	+	43	46¾	+
29.J.S.	1932	2½	4	1	M	7,3	6,8	Br.	+	+	+	+	+	-	+	+		A	11	+	43½	45	-
30.J.R.L.	1932	4	5	1	M	7,6	7,1	Bot.	+	+	+	+	+	-	+	+		A	13	-			-
31.G.L.	1932	4	5	2	M	7,14	6,15	Bot.	+	+	+	+	-	-	+	+		A	17	+	48½	52	
32.B.P.	1932	5	8	1	F	8,1	6,10	Br.	+	+	+	+	+	-	+	+		A	13	+	44	50	
33.D.B.	1933	3	8	1	M	9,0	7,9	Bot.	+	+	+	+	-	+	+	+		A	21	+	43	41¾	+
34.D.M.	1933	1	7	1	M	8,8	7,5	Br.	+	+	+	+	+	-	+	+		A	11	+	43	40	+
35.P.S.	1933	7	10	1	M	6,0	7,2	Br.	+	+	+	+	+	-	+	+		A	18	-			-
36.J.S.	1933	2	6	2	M	9,12	7,4	Br-Bot.	+	+	+	+	+	-	+	+		A	16	+	45	42	
37.W.C.	1933	2	3	1	M	6,12	6,4	Bot.	+	+	+	+	-	-	+	+		A	24	+	42	44	
38.C.M.	1933	5	6	1	M	7,9	7,1	Bot.	+	+	+	+	+	-	+	+		A	11	+	43	48¼	+
39.I.T.	1933	6	7	1	M	7,2	7,0	Bot.	+	+	+	+	+	-	+	+		A	11	-			-
40.P.S.	1933	7	10	1	M	6,0	6,2	Bot.	+	+	+	+	+	-	+	+		A	18	-			-
41.M.O.	1934	2	5	1	F	6,12	6,9	Bot.	+	+	++	+	+	-	No operation performed			D	2				-
42.K.T.S.	1934	1	6	3	M	8,2	7,5	Bot.	+	+	+	+	+	-	+	+		D	4				-
43.H.S.	1934	6	12	1	M	9,0	8,3	Br.	+	+	+	+	+	+	+	+		A	18	+	34½	40	
44.P.C.	1934	9	11	1	F	8,4	7,5	Br.	+	+	+	+	+	+	+	+		A	14	+	40½	38½	-
45.A.S.	1934½	5	11	1	M	8,0	7,3	Br.	-	+	+	+	+	-	+	+		A	18	+	39½	34¼	-
46.J.H.	1934	3	7	1	M	7,0	6,8	Bot.	+	+	+	+	-	-	+	+		A	16	+	39	39	-
47.R.C.	1934	4	6	1	M	7,5	6,5	Br.	+	+	+	+	+	-	+	+		A	13	-			-
48.J.T.	1934	4	5	3	M	9,0	7,8	Br.	+	+	+	+	+	-	+	+		A	11	+	38	37	
49.R.B.	1934	2	3	2	M	6,14	6,9	Br.	+	+	+	+	+	-	+	+		A	16	+	42½	39½	+
50.S.M.	1934	2	4	5	M	6,4	6,9	Bot.	+	+	+	-	-	-	+	+		A	16	+			-
51.S.M.	1934	2	4	5	M	9,4	7,13	Bot.	+	+	+	+	-	-	+	+		A	16	+			
52.A.R.	1934	7	8	2	M	8,2	7,6	Br.	+	+	-	+	+	-	+	+		A	13	-			-
53.J.W.	1934	1	5	1	M	8,4	7,7	Bot.	+	+	-	+	+	-	+	+		A	14	+	41½	40	+
54.K.M.	1934	4	8	1	M	6,4	6,4	Bot.	+	+	-	+	+	-	+	+		A	12	+	43	42	+

96/

Case no. and initials	Year of case	Age symptoms commenced (weeks)	Age admitted (weeks)	Place in family	Sex	Weight at birth (lbs.)	Weight on admission (lbs.)	Type of feeding	Symptoms P.V.	Symptoms C.	Dehydr.	P.	T.	Cases X-rayed	Surg.	Treatment Cases with tumour at O.P.	Med.	Result alive or dead	Days in hosp.	Cases followed up	Ht. in ins.	Wt. in lbs.	Cases re X-rayed
55.K.H.	1934	3	4	1	M	7,4	7,2	Br.	+	+	+	-	-	-	+	+		A	15	+	42	36	+
56.M.B.	1935	4	11	1	M	6,8	6,4	Br.-Bot.	+	+	+	+	+	-	+	+		A	29	+	40½	46	-
57.F.F.	1935	6	9	1	M	7,2	7,4	Br.	+	+	+	+	+	+	+	+		A	12	+	39	46	+
58.D.B.	1935	3	8	1	M	7,3	6,13	Bot.	+	+	+	+	+	+	+	+		A	21	+	40	42½	-
59.J.W.	1935	7	8	1	M	8	7,4	Br.	+	+	+	+	+	+	+	+		A	20	-			-
60.P.S.	1935	3	4	1	M	7,6	6,10	Br.-Bot.	+	+	+	-	-	+	+	+		A	22	-			
61.D.W.	1935	2	3	1	M	7,8	7,0	Br.	+	+	+	+	+	+	+	+		A	21	+	36½	36	-
62.A.B.	1935	2½	3	1	M	7,9	6,12	Br.-Bot.	-	+	+	-	-	+	+	+		A	23	+	35	34,7	-
63.S.W.	1935	1	8	1	F	7,1	7,2	Bot.	+	+	+	+	-	-	+	+		A	31	+	40½	39¼	-
64.B.J.	1935	2	3	1	F	7,5	6,11	Bot.	+	+	+	+	+	+	+	+		A	14	+	40	36	-
65.G.B.	1935	4	5	2	M	7,11	6,6	Br.-Bot.	+	+	+	+	+	+	+	+		A	17	+	37½	33½	-
66.C.E.C.	1935	5	10	1	M	8,0	7,9	Br.-Bot.	+	+	+	+	+	+	+	+		A	19	+	36	34	-
67.A.L.	1935	3	4	1	M	6,3	5,3	Bot.	+	+	+	+	-	-	+	+		A	14	+	37	35	-
68.G.L.	1935	1½	2	1	M	7,5	6,12	Br.	+	+	+	+	+	-	+	+		D	23				
69.S.D.	1935	3½	4	1	M	6,8	6,4	Bot.	+	+	+	+	+	+	+	+		D	8				
70.D.B.	1935	3	4	1*	M	7,6	7,0	Br.-Bot.	+	+	+	+	+	-	+	+		D	24				
71.A.L.	1935	7	9	1	M	7,0	7,0	Br.-Bot.	+	+	+	+	-	-	+	+		D	20				
72.R.P.	1936	7½	8	1	M	5,6	6,2	Br.-Bot.	+	+	+	+	+	-	+	+	Eum.	D	2				
73.C.W.	1936	5	8	1	F	7,5	7,4	Br.-Bot.	+	+	+	+	-	+			Eum.	A	78	+	36	33	-
74.J.L.	1936	4	7	1	F	6,4	6,6	Br.-Bot.	+	+	+	+	-	+			Eum.	A	69				
75.P.H.	1936	5	11	2	F	7,0	7,2	Br.-Bot.	+	+	+	+	+	-			Eum.	A	80	+	23	25	+
76.S.P.	1936	6	8	1	F	7,0	6,12	Br.	+	+	+	+	+	+			Eum.	A	58	+	38	32,5	-
77.K.H.	1936	3	4	1	M	7,8	7,1	Br.	+	+	+	+	+	+			Eum.	A	33	+	33	29	+
78.M.L.	1936	4	5	1	M	8,10	6,18	Br.-Bot.	+	+	+	+	+	+			Eum.	D	58				
79.B.F.	1936	4½	6	2	F	7,4	6,9	Br.-Bot.	+	+	+	-	+	+	-		Eum.	A	30	-			
80.R.S.	1936	4½	5	1	M	8	7,1	Br.	+	+	+	+	+	+	-		Eum.	D	10				

1/

Case no. and initials	Year of case	Age symptoms commenced (weeks)	Age admitted (weeks)	Place in family	Sex	Weight at birth (lbs.)	Weight on admission (lbs.)	Type of feeding	Symptoms P.V.	C.	De hydr.	P.	T.	Cases X-rayed	Surg.	Cases with tumour at O.P.	Med.	Result alive or dead	Days in hosp.	Cases follow-ed up	Ht. in ins.	Wt. in lbs.	Cases re X-rayed
81.P.N.	1936	2	9	1	M	6,8	6,12	Bot.	+	+	+	+	+	+	-		Eum.	D	3				
82.R.P.	1936	1½	2	2	M	5,0	5,4	Bot.	+	+	+	+	+	+	+	+	-	D	7				
83.J.H.	1936	5½	7	1	M	6,8	6,12	Br.-Bot.	+	+	+	+	+	+	-		Eum.	A	61	+	34	28	+
84.T.C.	1936	3	5	3	M	9,0	7,6	Bot.	+	-	+	+	+	+	+	+	-	A	57	+	35	33,11	+
85.D.B.	1936	3	4	2	M	8,0	7,3	Bot.	+	+	-	+	-	+	+		-	A	18	+	35	35	-
86.R.K.	1936	4	6	1	M	7,9	7,6	Br.-Bot.	+	+	+	+	+	-	+		-	A	61	+	28	32	+
87.B.R.	1936	7½	8½	1	M	8,0	6,7	Bot.	+	+	+	+	+	+	-		Eum.	A	55	+	35	26	+
88.A.W.	1936	1	5	2	M	7,2	6,9	Bot.	+	+	+	+	-	+	-		Eum.	A	54	+	34	27	-
89.M.C.	1936	4	6	2	F	9,9	8,1	Br.	+	+	+	+	+	+	-		Eum.	A	20	+	33	28	-
90.J.Y.	1936	3	5	1	M	7,8	6,12	Br.	+	+	+	+	+	+	-		Eum.	A	28	+	22	27	-
91.M.W.	1936	3	5	1	M	8,0	6,13	Bot.	+	+	+	+	+	-	+	+	Eum.	A	21	+	33	28	-
92.K.T.	1936	4	6	2	M	7,4	7,4	Bot.	+	+	+	+	+	-	+	+	Eum.	A	10	+	34	29	+
93.T.H.	1936	3½	6	1	M	7,4	7,2	Bot.	+	+	+	+	+	-	-		Eum.	A	17	+	28	31	-
94.B.B.	1936	1½	2	1	F	7,0	6,6	Br.	+	+	+	+	+	-	-		Eum.	A	17	+	29	31	-
95.K.C.	1936	3	12	1	M	5,2	6,13	Bot.	+	+	+	+	+	-	-		Eum.	A	63	+	26	30	-
96.P.R.	1936	4	7	1	M	6,12	6,14	Bot.	+	+	+	+	+	+	-		Eum.	A	28	+	30	26	-
97.J.G.S.	1936	2	7	1	M	8,0	6,13	Br.-Bot.	+	+	+	+	+	-	-		Eum.	A	21	+	25	29	-
98.B.H.	1937	4	6	1	M	7,0	6,7	Bot.	+	+	+	+	-	+	-		Eum.	A	14	+	26	28	-
99.M.C.	1937	6	7	1	M	3,12	4,0	Bot.	+	+	+	+	+	+	-		Eum.	A	32	+	23	25,6	-
100.I.H.	1937	1½	6	1	M	6,8	7,0	Br.	+	+	+	+	+	+	-		Eum.	D	28				
101.B.L.H.	1937	2	4	1	M	5,4	6,4	Bot.	+	+	+	+	+	+	-		Eum.	D	6				
102.F.U.	1937	1½	3	1	F	8,8	8,10	Br.	+	-	+	+	+	+	-		Eum.	A	30	+	30	24,7	-
103.A.C.	1937	1	2½	1	M	7,0	6,15	Br.	+	-	+	+	+	+	-		Eum.	A	30	+	-	21	+
104.R.P.	1937	4	7	1	M	7,15	7,10	Br.-Bot.	+	+	+	+	+	+	-		Eum.	A	23	+	23	21	+
105.C.C.	1937	2½	4	1	M	8,0	7,6	Br.	+	+	+	+	+	+	-		Eum.	A	80	+	32	31	+
106.D.M.T.	1937	3	5½	1	M	7,12	6,15	Br.-Bot.	+	+	+	+	+	+	-		Eum.	A	77	+	27	30½	+
107.T.J.	1937	1	3	1	M	9,0	7,11	Br.	+	+	+	+	+	+	+	+	Eum.	A	23	+	-	28	-

108/

Case no. and initials	Year of case	Age symptoms commenced (weeks)	Age admitted (weeks)	Place in family	Sex	Weight at birth (lbs.)	Weight on admission (lbs.)	Type of feeding	Symptoms		Signs on examination			Cases X-rayed	Treatment	Cases with tumour at O.P.		Result alive or dead	Days in hosp.	Cases followed up	Ht. in ins.	Wt. in lbs.	Cases re X-rayed
									P.V.	C.	Dehydr.	P.	T.		Surg.		Med.						
108.M.A.	1937	3	5	1	M	7,8	7,4	Br.	+	+	+	+	-	+	-		Eum.	A	64	+	22½	30	+
109.D.C.E.	1937	3	8	1	M	8,15	8,0	Br.-Bot.	+	+	+	+	-	+	-		Eum.	A	65	+	36	33½	+
110.R.M.	1938	3	10	1	M	6,6,	7,0	Br.-Bot.	+	+	+	+	+	+	-		Eum. & Syntr.	A	59	+	-	21,9	+
111.R.S.	1938	5	6	1	M	6,10	6,4	Br.	+	+	+	+	+	+	-		Eum.	A	30	+		17	+
112.B.H.	1938	5	7	1	M	8,0	9,2	Br.	+	+	+	+	+	+	-		Eum.	A	30	+		22	+
113.D.L.	1938	5	11	1	M	7,14	7,2	Br.	+	+	+	+	+	+	-		Eum.	A	35	+		21	+
114.R.T.	1938	5	8	1	M	7,0	6,12	Br.-Bot.	+	+	+	+	+	+	-		Eum.	A	49	+		19½	+
115.A.S.	1938	3½	5	1	M	7,0	5,8	Bot.	+	+	+	+	+	+	-		Eum.	A	42	+		14	+
116.A.A.	1938	3	6	2	M	6,8	5,12	Bot.	+	+	+	+	+	-	-		Eum.	D	3				
117.E.A.	1938	2½	3	1	F	7,4	6,13	Bot.	+	+	+	+	+	-	-		Eum.	A	42	+		13	-
118.L.G.	1938	3	5	2	M	7,0	6,12	Bot.	+	+	+	+	-	-	-		Eum.	D	1				
119.W.B.	1938	7½	8	1	F	7,4	5,11	Br.	+	+	+	+	+	-	-		Eum.	A	49	+		11	-
120.K.R.	1938	2	14	2	M	7,4	7,6	Br.-Bot.	+	+	+	+	+	-	-		Eum.	A	20	+			-
121.G.T.	1938	2½	3	1	M	7,8	7,1	Bot.	+	+	+	+	+	+	-		Eum.	A	24	+			-
122.D.B.	1938	1	2	1	M	7,0	6,0	Bot.	+	+	+	+	-	+	-		Eum.	A	10	+			-
123.J.H.	1938	2	3	1	M	6,7	5,8	Br.	+	+	+	+	+	+	+		Eum.	D	24				
124.I.B.	1938	4	5	1	M	8,0	7,8	Br.	+	+	+	+	+	+	-		Eum.	A	27	+			-
125.P.N.	1938	5½	7	2	M	7,0	6,4	Br.	+	+	+	+	+	+	-		Eum.	A	14	+			-
126.M.H.	1938	4	6	5	M	8,0	6,0	Br.	+	+	+	+	+	+	-		Eum.	A	73	+			-
127.B.G.	1938	3	7	2	M	7,4	7,0	Br.-Bot.	+	+	+	+	-	+	-		Eum.	D	35				
128.D.S.	1938	3½	4	1	M	6,12	6,8	Br.	+	+	+	+	+	+	-		Eum.	A	50	+			-

Br. = Breast fed.
Bot. = Bottle fed.
P.V. = Projectile vomiting.
C. = Constipation.

P. = Palpable tumour.
T. = Tumour.
Eum. = Eumydrin.
Syntr. = Syntropan.