CONGENITAL HYPERTROPHIC PYLORIC STENOSIS.

A Study of the Pathology, Diagnosis, Treatment, and Prognosis with Reference to Seventeen Personal Cases.

By

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INTRODUCTION and HISTORICAL REVIEW.

The condition of Congenital Hypertrophic Pyloric Stenosis, also commonly called Congenital Pyloric Stenosis, or Pyloric Stenosis in Infants, may be shortly defined as "A disease in which symptoms of increasing pyloric obstruction arise in children under three months of age" (Parsons and Barling).

In our opinion the full title Congenital Hypertrophic Pyloric Stenosis is the most desirable in that it implies the congenital nature of the malady, and emphasises the muscular hypertrophy of the pyloric canal so constantly found post mortem and at operation in these cases.

Although much prominence has been given to Congenital Hypertrophic Pyloric Stenosis within the last decade, it was a condition not unknown to the physicians of the seventeenth and eighteenth centuries. Thus, in 1629, Fabricius Hildanus (1560-1634) a physician of Cologne, Geneva, and Berne, described a case he had seen in 1627, occurring in the small wasted six months old heir of Henry Otho, Esq.; this boy recovered on treatment with thickened feeds, but unfortunately there is no record as to the time of onset of his symptoms.

Three writers of the eighteenth century mention cases presenting symptoms that are suggestive. Blair in 1717, George Armstrong in 1771, and Beardsley in 1778; the latter's case occurring in a male child of
5 years, and there would seem, therefore, to be some doubt as to its being a true Congenital Hypertrophic Pyloric Stenosis.

The year 1841 saw the publication of "A case of schirrhus of the stomach, probably congenital" by Williamson of London, and in 1842 Siemon-Dawosky recorded the first case in Germany. These two authors were the first to examine the pathology of the condition; both attributing the stenosis to a hypertrophy of the submucous layer of the pylorus. Siemon-Dawosky, in addition, remarks upon the projectile nature of the vomiting.

Ianderer, in 1879, uses the term "Congenital Pyloric Stenosis" for the first time, and in 1887 Hirschsprung described the clinical features of two cases in young infants under his own care. In 1896, Finkelstein called attention to the diagnostic significance of a palpable pyloric tumour, and in 1898 John Thomson, of Edinburgh and London, published his well known paper dealing with the pathogenesis and clinical symptoms as they had struck him in his wide and extensive experience. Thus at the beginning of the twentieth century our knowledge of the symptoms, signs and pathogenesis of Congenital Hypertrophic Pyloric Stenosis was fairly complete, and has since been supplemented in detail by such well known authorities as Still, Sauer, and Ibrahim.

The history of the Surgical treatment to relieve symptoms /
3.
symptoms is characterised by the diversity of methods employed and the complicated technique required of the surgeon.

Jejunostomy (Cordua), gastro-enterostomy (Lobker), pyloric divulsion (Loreta), pylorectomy (Stiles) and phloroplasty in its various forms have all enjoyed a period of popularity.

Cordua performed the first operation for Congenital Hypertrophic Pyloric Stenosis when, in 1893, he carried out a jejunostomy with fatal result; a successful gastro-enterostomy was performed, however, by Lobker in 1898, and although the operative mortality was high, this operation was the method of choice for a considerable period.

Loreta’s pyloric divulsion through a gastrectomy was next advocated, but lost favour owing to its high mortality and uncertain beneficial effect.

Pyloroplasty of various types then came into vogue, but was attended by a high mortality, until, in 1907, Fredet instituted a new departure when he performed a successful pyloroplasty, employing a straight incision through the muscular coats of the pyloric canal, exposing the mucous membrane in the wound. He then fashioned the longitudinal wound in the muscle layers into a transverse one, by means of sutures, a somewhat similar procedure to that employed in the modern pharyngoplasty for cleft palate.

To Rammstedt (1912) belongs the credit of devising
vising the modern and highly successful operation generally known as the Fredet-Rammstedt operation. He suggested leaving the wound in the pyloric musculature unclosed with the mucous membrane bulging through it; this procedure obviated the time factor, rendered the operative technique extremely simple, and was so successful, that it soon became the universal method of choice.

In the following Study of Congenital Hypertrophic Pyloric Stenosis we hope to contrast and compare the generally accepted views with what we have personally observed during a period of residence at The Children's Hospital, Sheffield, and the Royal Hospital for Sick Children, Edinburgh.

We wish it further to be understood that, in the cases referred to, a definite diagnosis was established either post mortem or at operation.
Pathology:--

The Pathology and Pathogenesis of Congenital Hypertrophic Pyloric Stenosis has given rise to much speculation and discussion in the past. Pathologists have long been familiar with the morbid and microscopic appearances of the pyloric region of the stomach in this condition, but have failed to reach any definite or unanimous conclusion with regard to the causative factors producing the pathological changes so regularly observed on the post mortem table.

During our period of residence at the Royal Hospital for Sick Children, Edinburgh, we were fortunate in securing five post mortem specimens of the stomach region. Four of these specimens were from male infants who died suffering from Congenital Hypertrophic Pyloric Stenosis, and otherspecimen was from a normal male infant, aged five weeks, who died of septicaemia. We propose describing and contrasting the Morbid and Microscopic appearance found in the normal stomach and pylorus with that found in these cases of true Congenital Hypertrophic Pyloric Stenosis.

Thereafter the various theories as to the Pathogenesis of the condition will be discussed in the light of the pathological and clinical findings.
I. The Macroscopic Appearances in the Normal Stomach and Pylorus.

Male child, aged 5 weeks.

fig. 1. Normal Stomach and Pylorus.
Male child, aged 5 weeks.
(Life Size).

Morbid Appearances.

The stomach in this specimen was a little larger than is usual at this age, but apart from this slight dilatation the specimen was normal in every respect. The Pylorus was 1.8 cm. in length and .9 cm. in diameter.

Smooth,
Smooth, glistening, transparent peritoneum covered the pyloric region and showed the pylorus to have a pink colour similar to that of the stomach, but quite distinct from the rather darker blueish pink colour of the duodenum. When handled with the fingers the pylorus was found to be soft and pliable to the touch; a slight increase in diameter and consistency could be felt at the duodenal end in the vicinity of the pyloric sphincter. On viewing the pyloric sphincter through the divided duodenum it could be seen bulging in a cone like manner into the duodenal lumen, with the opening of the pyloric canal showing as an oval at its apex. The appearance presented being very similar to the cervix uteri and external os, as viewed from the vagina.

On Longitudinal Section of the Pylorus.

The wall of the pylorus was seen to consist of five distinct layers. From without inwards these layers were the peritoneum, the longitudinal muscle layer, the circular muscle layer, the submucous layer, and the mucous coat lining the pyloric canal.

The Peritoneum.

The peritoneal covering of the pylorus was of negligible thickness and stretched continuously from the stomach, over the pylorus, and on to the duodenum beyond. It was observed to be closely adherent to the underlying longitudinal muscle layer.
The Longitudinal Muscle Layer.

The longitudinal muscle fibres of the pylorus arose from the longitudinal muscle fibres originating in the body of the stomach. As these muscle fibres approached the pyloric sphincter a differentiation was noted when viewed with a lens. The more superficial fibres continued onwards to the duodenum without interruption, whereas the deeper fibres bent sharply inwards, seeming to become interwoven with the circular muscle fibres, and together forming the pyloric ring sphincter.

The Circular Muscle Layer.

The circular muscle fibres were of a pinkish white colour. This layer, like the longitudinal, took origin in the body of the stomach from the circular muscle fibres there. These circular muscle fibres, continued from the stomach, formed a cylinder surrounding the pyloric canal. At the duodenal end of the canal the circular muscle fibres interlaced with the deeper fibres of the longitudinal muscle layer, constituting the pyloric ring sphincter. The circular muscle coat ended in the pyloric ring sphincter; the circular muscle coat of the duodenum was composed of quite a separate and new group of muscle fibres, entirely isolated from the circular fibres of the pylorus by a thin, white, glistening fibrous septum which showed as a fine line under a lens.
The Submucous Layer.

This layer was firmly attached to the circular muscle layer, but seemed more firmly attached to the mucosa lining the pyloric canal. The submucous layer was a continuation of the submucosa of the stomach, it stretched throughout the length of the pyloric canal, to be continued on beyond the pyloric sphincter to the duodenum.

The Mucous Coat.

The lining coat of the pyloric canal was a continuation of the gastric mucosa and presented a similar structure. A distinct thickening occurred where the mucosa covered the pyloric ring sphincter, but thereafter it regained its previous thickness and continued onwards to line the proximal duodenum. The mucosa was thrown into small longitudinal rugae in the pyloric canal encroaching slightly upon the lumen, thus a view from the pyloric antrum looking towards the pyloric canal (before longitudinal section) showed the mucosa in stellate outline contained within the cylinder composed of the muscle layers.

II. The Macroscopic Appearances of the Stomach and Pylorus in Congenital Hypertrophic Pyloric Stenosis.

The material at our disposal for purposes of examination consisted of the stomach and pyloric region as it was found post mortem in four infants who had died suffering from Congenital Hypertrophic Pyloric Stenosis.
Stenosis. Two of the cases had died following the Fredet-Rammstedt operation, and the remaining two died before operation had been undertaken. The ages of these infants ranged from three to eight weeks at the time of death. In order to simplify description the Morbid Appearances found in one case only will be described in detail, but the various distinctive features will be illustrated from the other three specimens.

Morbid Appearances.

F.M., aged $\frac{5}{52}$ male child. See fig. II. below.

Stomach and Pylorus in Congenital Hypertrophic Pyloric Stenosis.

F.M. Male Child.

Aged 5 weeks.

(Life Size).

Note:- The dilated stomach.
The barrel shaped well marked pyloric tumour.
The wound of the Rammstedt operation, performed.
The stomach in the specimen was dilated and thick walled; the hypertrophy of the muscular coat being most marked towards the pyloric end of the stomach. The gastric mucosa was congested due to a mild degree of gastritis. The Pylorus was easily recognisable by reason of its "barrel shaped" enlargement constituting the "pyloric tumour". The "tumour" was 2.1 cm in length and 13 mm in diameter. See figs. III. and IV.

**fig. III.** Stomach and Pylorus in Congenital Hypertrophic Pyloric Stenosis.
G.C. Male Child.
Aged 7 weeks.
(Life Size).

**Note:**
The marked pyloric tumour.
The white translucent colour of the pyloric tumour.

Smooth, tense, glistening, transparent peritoneum covered the pyloric region and showed the underlying pylorus.
pylorus to have a white translucent appearance, quite distinct from the pink colour of the stomach and the darker blueish pink colour of the duodenum. When palpated with the fingers the pyloric tumour was found to be hard, tense, and elastic with a cartilaginous like consistency. It was impossible to differentiate the pyloric sphincter by the touch as it was incorporated in the pyloric tumour.

fig. IV. The Stomach and Pylorus in Congenital Hyper-trophic Pyloric Stenosis.
P.S. Male Child.
Aged 3 weeks.
(Life Size)

Note:— Stomach only slightly dilated.
Well marked pyloric tumour.
Wound of Rammstadt operation performed nine days before death.

The pyloric sphincter as viewed from the divided duodenum presented the cervix like appearance already described /
described in the normal specimen. See fig. V.
To ensure completeness and for purposes of comparison we give a table of the length and diameter of the pyloric tumour found in each of the four cases examined.

<table>
<thead>
<tr>
<th>Initial</th>
<th>Sex</th>
<th>Age</th>
<th>Length of Pylorus</th>
<th>Diameter of Pylorus</th>
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<tbody>
<tr>
<td>F.S.</td>
<td>M</td>
<td>3/52</td>
<td>1.7 c.m.</td>
<td>12 mm.</td>
</tr>
<tr>
<td>F.M.</td>
<td>M</td>
<td>5/52</td>
<td>2.1 c.m.</td>
<td>13 mm.</td>
</tr>
<tr>
<td>G.C.</td>
<td>M</td>
<td>7/52</td>
<td>1.8 c.m.</td>
<td>11 mm.</td>
</tr>
<tr>
<td>J.B.</td>
<td>M</td>
<td>8/52</td>
<td>1.7 c.m.</td>
<td>11 mm.</td>
</tr>
</tbody>
</table>

fig. V. /
fig. V. Stomach and Pylorus in Congenital Hypertrophic Pyloric Stenosis.

F.M. Male Child.
Aged 7 weeks.
(Life Size).

Note:-- The dilated stomach.
The cervix like protrusion of the pyloric sphincter as viewed from the reflected duodenum.
The os like opening of the pyloric canal.

On Longitudinal Section.

The wall of the pylorus consisted of five distinct layers similar to those enumerated in the normal specimen.

The Peritoneum.

This was very thin and closely adherent to the underlying longitudinal muscle layer.

The Longitudinal Muscle Layer.

This layer took its origin in the body of the stomach, formed the outer muscular coat of the pylorus, and divided into superficial and deep fibres opposite the pyloric sphincter. The superficial fibres continued uninterrupted to form the longitudinal muscle layer of the duodenum, whereas the deeper fibres bent inwards to join the circular muscle fibres in the formation of the pyloric ring sphincter.

The Circular Muscle Layer.

The circular muscle fibres were of a greyish white colour and seemed avascular. A very marked increase in the thickness of this layer was also noted; the increased thickness extended from the pyloric sphincter proximally /
proximally for the whole length of the canal. The hypertrophy continued into the pyloric antrum, and thereafter gradually diminished until, at the incisura angularis of the stomach, the circular muscle had regained its normal thickness. At the duodenal end of the pyloric canal the circular muscle fibres interlaced with the deeper fibres of the longitudinal layer; as far as could be seen there was no hypertrophy of the circular muscle fibres actually in the sphincteric ring itself e.g., there was no marked increase in the size of the pyloric sphincter nor did it protrude itself more than usual into the lumen of the pyloric canal.

fig. VI. Stomach and Pylorus in Congenital Hypertrophic Pyloric Stenosis.
J.B. Male Child.
Aged 8 weeks.
(Life Size).

Note:
Note:--
The thick walled stomach.
The thick hypertrophic circular muscle coat of the pyloric canal.
The pyloric ring sphincter encroaching but slightly upon the lumen of the pyloric canal.

The circular muscle fibres ended in the pyloric ring sphincter; they were observed to be isolated from the circular muscle fibres of the duodenum by a thin, white, glistening fibrous septum similar to that observed in the normal specimen.

The Submucous Layer.

This layer was observed to be a little irregular in its outline and seemed to be a little thicker than it was in the normal specimen.

The Mucous Coats.

The mucosa was thrown into well marked longitudinal rugae. In two places it was neatly folded on itself, and protruded into the lumen of the pyloric canal. See fig VII. below.

fig. VII. Transverse section of Normal and Hypertrophied Pylorus. Reproduction from Thomson & Findlay, page 348.

Note:-- The mucosa protruding into the lumen of the pyloric canal in fig. III.
At the pyloric antrum, just as the mucosa was entering, the pyloric canal proper, it was observed that the entering folds were crowded together and contorted in their outline.

Despite the marked circular muscle hypertrophy and the enlarged rugae of the mucosa a fine probe one and a half millimeters in diameter could be passed along the pyloric canal with ease in all four specimens.

III. The Microscopic Appearances in the Normal Pylorus and in the Pylorus in Congenital Hypertrophic Stenosis.

For the purpose of microscopic examination longitudinal sections were cut of the pyloric antrum, the wall of the pyloric canal, and the pyloric ring sphincter.

The Pyloric Antrum. Examination of the pyloric antrum in the normal specimen and in a specimen of Congenital Hypertrophic Pyloric Stenosis (hereafter referred to as the abnormal) showed a thin ill defined longitudinal muscle layer, a thicker well defined circular muscle layer with the circular muscle fibres gathered in fasciculi, and the submucosa forming the basement membrane for the mucosa of the pyloric antrum. See figs. VIII. and IX.
fig. VIII.
Magnification X13.

The Normal Pyloric Antrum.
Male Child, aged 5 weeks.

fig. IX.
Magnification X13.

The abnormal Pyloric Antrum.
F.M. Male child, aged 5 weeks.

Note:— The somewhat thicker circular muscle layer in the abnormal as compared with the normal.
The ill defined mucous membrane and glands in the abnormal as compared with the normal.

Two points of difference can, we believe, be demonstrated between the two sections. Firstly the circular muscle layer is slightly thicker and better defined in the abnormal than in the normal, and secondly the mucosa in the abnormal is ill defined as compared with that in the normal. We may infer, then, that the hypertrophy of the circular muscle layer of the pyloric canal proper is not sharply circumscribed at the gastric end of the canal, but extends into the pyloric antrum becoming gradually less marked as the body of the stomach is reached. The appearance of the mucous membrane in the abnormal section is suggestive of the condition of subacute gastritis caused by the retention of food in the stomach.

The Wall of the Pyloric Canal.

Longitudinal Sections of the wall of the pyloric canal were made in the normal and abnormal specimens, at a point where the wall appeared to be of the greatest thickness.

All five sections showed the longitudinal muscle layer to be of small thickness as compared with the circular muscle layer; this proportional disparity being particularly marked in the abnormal sections. The circular muscle layer showed the muscle fibres to be grouped together in fasciculi divided from one another by fibrous tissue, which grew out in finger like projections /
projections from the submucous layer. The submucous layer was of variable thickness in the five sections examined; there was no very marked difference between this layer as it appeared in the normal and abnormal sections. The mucous membrane of the pyloric canal appeared to be less well defined in the abnormal specimens than in the normal.* From figs. XI. and XIII. it will be observed that the lumen of the pyloric canal, although encroached upon by the mucous membrane was never completely occluded.

fig. X.
Magnification XI3.

The Normal Wall of the Pyloric Canal.
Male Child, aged 5 weeks.

fig. XI.
Magnification XI3.
21.

The Abnormal Wall of the Pyloric Canal.
E.M. Male Child, aged 5 weeks.

fig. XII.
Magnification X13.

The Abnormal Wall of the Pyloric Canal.
P.S. Male Child, aged 3 weeks.
fig. XIII.
Magnification $XI_3$.

The Abnormal Wall of the Pyloric Canal.
G.C. Male Child, aged 7 weeks.

fig. XIV.
Magnification $XI_3$.

The Abnormal Wall of the Pyloric Canal.
J.B. Male Child, aged 8 weeks.
Note:- The thin longitudinal muscle layer in both the normal and abnormal sections. The very marked increase in thickness of the circular muscle layer in the abnormal sections. The submucosa little, if at all, increased in thickness in the abnormal sections. The mucous membrane flattened and rather ill-defined in the abnormal sections.

We believe that comparison between the section of the normal pyloric wall and that found in the abnormal sections affords a very striking demonstration of two essential differences. Making allowances for disparities in age, it is still very evident that the circular muscle layer is almost twice as thick in the abnormal sections as it is in the normal. Examination of the individual fasciculi of muscle fibres shows this thickening to be due to a hypertrophy of the individual muscle fibres, and we further believe that in some cases (fig. XIII.) the number of individual muscle fibres is increased in each fasciculus.

The mucous membrane affords the other difference between the normal and abnormal sections. In the normal section it has stained well and its outline is easily defined. The abnormal sections, on the other hand, show the mucous membrane to have stained poorly and to be irregularly defined in outline. These differences in the mucous membrane are, we believe, brought about, by pressure when the pyloric musculature goes into spasm.

The Pyloric Ring Sphincter.

Turning now to the region of the pyloric ring sphincter.
sphincter. The normal and abnormal sections of this region demonstrated several points in common; in all the sections the more superficial fibres of the longitudinal muscle layer could be seen passing on to form the longitudinal layer of the duodenal wall. Careful examination of figs XV., XVI. and XVIII. shows the deeper fibres of the longitudinal layer turning inwards to intermingle with the circular muscle fibres in the formation of the pyloric ring sphincter. As the circular muscle fibres approached the pyloric ring sphincter they tended to lose their fascicular arrangement, and blending with the deep fibres of the longitudinal layer formed a rather undifferentiated muscle mass - the pyloric ring sphincter. See figs. XV. and XIX.

A fibrous septum dividing the pyloric ring sphincter from the circular muscle layer of the duodenum could be defined in all the sections; it is particularly well marked, however, in figs. XVI., XVII. and XIX. The submucosa could be observed passing round the pyloric ring sphincter, and continuing uninterrupted into the duodenum. The mucous membrane appeared a little thicker over the pyloric sphincter, and once it had reached the duodenum the more deeply staining Brunner's glands could be observed as seen in figs. XV. and XVII.
fig. XV.
Magnification X10.

The Normal Pyloric Sphincter.
Male Child, aged 5 weeks.

fig. XVI.
Magnification X10.

The Abnormal Pyloric Sphincter.
F.M. Male Child, aged 5 weeks.
fig. XVII.
Magnification X10.

The Abnormal Pyloric Sphincter.
F.S. Male Child, aged 3 weeks.

fig. XVIII.
Magnification X10.

The Abnormal Pyloric Sphincter.
G.C. Male Child, aged 7 weeks.
The Abnormal Pyloric Sphincter.
J.B. Male Child, aged 8 weeks.

We have observed the points in common that were demonstrable in the normal and abnormal pyloric ring sphincter. In our opinion the pyloric ring sphincter is little, if at all, involved in the general circular muscle hypertrophy, which was so easily appreciable on examination of the normal and abnormal sections of the wall of the pyloric canal. No great relative increase in the size of the pyloric sphincter as a whole can be seen in any of the abnormal sections; a glance at fig. XIX will show that the sphincter, in this case, was relatively very small in size as compared with the hypertrophied wall of the pyloric canal. Further, we believe that the loss of fascicular formation of the circular muscle layer as it approaches the pyloric ring /
ring sphincter and blends with the deep fibres of the longitudinal layer is just as well marked in the abnormal as in the normal specimen. If there was a hypertrophy of the circular muscle fibres in the pyloric sphincter itself, we think it likely that there would be little loss of fascicular formation of the circular muscle fibres, and that the longitudinal muscle fibres would undergo fibrous degeneration due to the pressure exerted by the hypertrophied circular muscle fibres. No such appearances have been found in the abnormal sections, and we therefore feel justified in stating that the pyloric ring sphincter is not affected by the general hypertrophy of the circular muscle fibres in the wall of the pyloric canal.

Summary :-

The study of the macroscopic and microscopic appearances of the pylorus in a normal child and in Congenital Hypertrophic Pyloric Stenosis has, we believe, demonstrated several points of importance.

From the practical standpoint of surgical treatment the appearance of the pylorus to the naked eye with its barrel shaped enlargement and altered consistency to the touch, are points of no small importance. When the abdomen has been opened a glance at the pylorus and palpation with the fingers will at once confirm the
the diagnosis of Congenital Hypertrophic Pyloric Stenosis in the surgeon's mind. Longitudinal section of the pylorus illustrates clearly the marked local hypertrophy of the circular muscle layer, encroaching upon, but never completely occluding, the lumen of the pyloric canal. This hypertrophy, while extending into the pyloric antrum at the gastric end of the canal, does not, in our opinion, affect the pyloric ring sphincter, as is evidenced by the discreet appearance of the sphincter in the four specimens we have examined.

Microscopic examination confirms the existence of a hypertrophy of the circular muscle layer of the pyloric canal; this hypertrophy extends in some degree into the pyloric antrum but gradually disappears. At the sphincteric end of the pyloric canal, we believe that the circular muscle hypertrophy ceases as soon as the longitudinal and circular muscle fibres intermingle to form the pyloric ring sphincter. The lumen of the pyloric canal, although diminished by the circular muscle hypertrophy, is not completely occluded thereby, and we have found no appearance to suggest that oedema of the submucosa plays an important role in further diminishing the lumen of the canal, although the projection of the mucosa in longitudinal folds diminishes the lumen to some extent.

IV. /
IV. The Pathogenesis of Congenital Hypertrophic Pyloric Stenosis.

The essential pathology of Congenital Hypertrophic Pyloric Stenosis has been shown to be a hypertrophy or local gigantism of the circular muscle fibres of the pyloric canal. It has further been demonstrated that this muscular hypertrophy is localised to the pyloric canal, and does not occur to any appreciable extent in the pyloric ring sphincter.

The underlying cause of this circular muscle hypertrophy has given rise to much controversy; the points at issue being:-

1. Whether the condition is congenital or not.
2. Whether circular muscle hypertrophy can occur as the result of long continued spasm of the pyloric sphincter.

1. Hirschsprung and Cautley believed that there was a primary congenital circular muscle hypertrophy in the pyloric ring sphincter and canal, and that this was associated with a secondary increased functional activity. The combination of these two factors resulting in pyloric obstruction by occluding the lumen of the pyloric canal. This view is strongly supported by Strachauer who reports having found circular muscle hypertrophy in the pyloric canal of a seven months foetus, and having seen this condition in the still-born on several occasions.

Parsons /
Parsons and Barling make the following observations in support of the primary congenital muscle hyperplasia theory. Firstly, the condition of pyloric spasm has long been recognised as a clinical entity, but they have never seen a case of pyloro-spasm develop into pyloric stenosis, nor can they believe this possible.

Secondly, in the foetus the pyloric canal is even more of a canal than in the infant; the pyloric ring sphincter does not function as a valve, but closure occurs by the contracture of the circular musculature of the pyloric canal. In Congenital Hypertrophic Pyloric Stenosis the whole circular musculature of the pyloric canal is hypertrophied and acts as a sphincter in a manner similar to that of the pyloric canal in the foetus. This similarity between the normal foetal pyloric canal, and that of Congenital Hypertrophic Pyloric Stenosis is strong evidence in favour of a congenital hyperplasia.

Thirdly, additional evidence in favour of a primary congenital hyperplasia has been adduced by Brash, who, in observations in the development of the pylorus, found that in a 20 m.m. cat embryo, the pyloric musculature was so thickened that the mucous coat was crumpled and nearly filled the lumen of the canal.

2. John Thomson, on the other hand, championed the theory that there was a primary spasm, and that this was followed by the circular muscle hypertrophy. He believed
believed that a functional overactivity or spasm was the primary factor, and that this functional overactivity was due to a want of neuro-muscular co-ordination.

The error in neuro-muscular co-ordination, in his opinion, began before birth, when the activity of the stomach pylorus were stimulated by the swallowing of liquor amnii; such an error in co-ordination resulted in an abnormality of function. This abnormality or functional overactivity takes the form of an ill timed, forcible, and prolonged closure of the pyloric sphincter, and it is this spasm that causes the secondary circular muscle hypertrophy to occur.

This view is supported by the observation that the true sphincter muscle of the pylorus does not share in the circular muscle hypertrophy, but remains small and inconspicuous. (Cameron, Illingworth and Dick.) Further the sudden onset of symptoms in some cases and the intermission of symptoms for two or three days at a time are occurrences strongly suggestive of a primary spasm and secondary muscular hypertrophy. Lastly, no relationship between the size of the tumour and the severity of the symptoms has been found to exist in our experience.

White has recently brought forward a theory which tries to co-relate spasm and circular muscle hypertrophy. He believes that these infants are hypertonic, and that there is, in addition, an autonomie inco-ordination which leads to contraction of the
the pyloric sphincter, instead of relaxation, when food reaches the pyloric antrum. This continued in-co-ordination increases muscular hypertrophy in a child with rapidly growing muscle. Should this reflex maladjustment right itself early, the child will recover, but should the inco-ordination continue a point is reached when stenosis occurs.

Whichever of the theories is accepted, and we tend to support that of John Thomson, there can be no doubt that spasm is the factor that produces the actual stenosis of the pyloric canal. In none of the post mortem specimens examined was a complete obstruction to the pyloric canal demonstrated. There can be no doubt that as the circular muscle coat increases in bulk the lumen of the pyloric canal is encroached upon, because the peritoneal coat is unyielding in character. This diminution of the lumen is further increased, in some cases, by the rugose oedematous nature of the mucosa. We believe that the muscular hypertrophy in this way causes a relative stenosis of the pyloric canal, but that an element of spasm is essential before the stenosis becomes absolute.

Summary:-

The two main theories as to the basic cause of the circular muscle hypertrophy have been given, and three points in favour of each have been mentioned. In our opinion that held by John Thomson is the more acceptable.
acceptable, although it is impossible to dogmatize upon this subject. Whatever view be accepted, it is important to realise that complete obstruction results from a combination of two errors - the increased bulk of the circular muscle fibres, and the added spasmatic contraction of the hypertrophied muscle.

CLINICAL DIAGNOSIS.

1. Etiology.

Congenital Hypertrophic Pyloric Stenosis shows no racial or climatic predilection, but many writers stress the frequency with which they have encountered a hereditary tendency, citing cases they have seen in several members of one family, or in succeeding generations of a certain stock (John Thomson & Roche).

Still more striking is the incidence in male infants, in the children of so called "nervous" parents, and the relative frequency with which the first born child is affected.

The preponderance of male infants has long been recognised.
recognised, and is remarked upon by Strachauer, Guy Cochran, D. Paterson, Wallace and Wevill and others. From the table below it will be observed that out of a series of 780 cases, collected from various sources, the relative incidence of male to female was five to one. Over our own small series of cases the incidence worked out at fifteen males to females two.

<table>
<thead>
<tr>
<th>Author</th>
<th>No. of Cases</th>
<th>Males</th>
<th>Females</th>
</tr>
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<tbody>
<tr>
<td>Strachauer</td>
<td>48</td>
<td>46</td>
<td>2</td>
</tr>
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</tr>
<tr>
<td>Wallace</td>
<td>145</td>
<td>125</td>
<td>20</td>
</tr>
</tbody>
</table>

Total 780 652 128

No very satisfactory explanation has yet been given for the sex incidence of this condition. Some have stressed the presence of inguinal hernia (much more common in boys, but not universally present in cases of Congenital Hypertrophic Pyloric Stenosis) others again, perhaps with more justification, lay great emphasis on prepucial irritation as a predisposing cause. (Tyrrell Gray).

As regards the "nervous" type of parent, one has frequently been impressed, while taking case histories, by the fact that the parents of these children generally belong to the age period between twenty-five and thirty years; over the seventeen cases studied the average /
average age of the parents has been, fathers twenty-nine years and mothers twenty-six years. In our opinion this age factor is of importance, the older the parents the more intense is their anxiety for their child's welfare.

Lastly there is the high incidence among first born infants; no other disease of infancy presents this peculiarity with such remarkable constancy. Thus Barrington Ward found the first child in the family affected in 57% of his cases; this is borne out by Paterson's figure of 53.5%, and Wallace's figure of 56.6%. Our own percentage incidence is 76.4%.

Three other factors require consideration under this heading.

One may be termed the age of onset, and can be taken as under three months of age; this factor will be more fully discussed under the title "Symptoms and Signs." The remaining two can be conveniently taken together as possible predisposing causes; they are concerned with the obstetric history and feeding of the newly born infant. Careful enquiry into the obstetric history of our cases has shown that an easy labour without instrumental delivery is the rule, and a survey of the birth weight of these infants gives an average of 7 lbs. 8 oz., proving that the cases are healthy and well developed at birth.

The method of early feeding of the child does not materially affect the incidence of the condition;
breast fed infants being as frequently encountered as these artificially fed in our cases. Irregularity in the amount and timing of the feeds, on the other hand plays an important role in determining the frequency and time of onset of the initial symptoms of vomiting.

Summary.

Congenital Hypertrophic Pyloric Stenosis is a condition affecting infants under three months of age, showing a marked predilection for males, the children of older parents, generally the first born child. The obstetric history of these cases is uneventful and the average birth weight is rather above the usual figure. Breast and artificially fed children are equally affected.

II. Symptoms and Signs.

Clinical Diagnosis in Congenital Hypertrophic Pyloric Stenosis is a complex problem. Each individual case must be considered separately, giving due consideration to the relative value of the positive and negative findings. Care and patience in the observation and examination of the case are essential if an accurate diagnosis is to be made.

We propose classifying the Symptoms and Signs under the headings Cardinal and Subsidiary, hoping by this means to simplify description and ensure a true prospective.
Cardinal Symptoms and Signs.

These are four in number and constitute the basis of the diagnosis; rarely do we encounter all four together in any one case, but there are few cases in which three at least cannot be elicited. They may be conveniently summarised as follows:

I. Persistent vomiting of projectile type, occurring shortly after feeding, the vomitus consisting of stomach contents, never bile stained.

II. The presence of visible gastric peristalsis passing from left to right across the epigastrium.

III. The palpation of a pyloric tumour.

IV. Constipation with small hard stools.

The above Cardinal Symptoms and Signs will now be considered in some detail in order to ascertain their frequency of occurrence and true diagnostic value.

I. Persistent vomiting of the projectile type.

Vomiting is the symptom constantly complained of in all cases; a gradual onset is reported in the vast majority, although cases do occur in which the onset of vomiting is acute.

Detailed questioning is usually unnecessary when enquiring with regard to the history of onset and development of this symptom; it is such a striking phenomenon that the lay mind is impressed by its occurrence, and the mother can give a most graphic description /
scription of all that she observed. The typical history is that the child thrived at first, then he began to vomit occasionally after a feed, within a few days the vomiting became more frequent, and was seen to be more forcible in character. Later still the feed was "shot out" over the floor when he vomited, and curds tended to be forced through the nose at these times. This is a very typical history, and the genesis of true projectile vomiting at once supplies a clue with regard to the further investigation of the case from the diagnostic standpoint.

These children are eager for their feeds, tend to take the feed quickly, then - either before the feed is actually finished or within five to ten minutes after finishing a feed - the feed is forcibly regurgitated. The vomit is either found on the floor at the side of the cot, or, if the child is sitting seated on its mother's knee, it may be projected a distance of some two to four feet. Observation during such an attack will reveal the fact that the regurgitation also takes place through the nose, and that instead of crying afterwards as one would expect, the infant tends to fall back exhausted by the effort.

The actual amount and character of the vomitus is most important. Frequently it will be noted that the amount vomited is in excess of the actual feed just given; this, of course, is due to delay in gastric emptying, and gives a valuable indication both /
both of the degree of pyloric obstruction which exists, and the amount of consequent gastric dilation that has occurred. The character of the vomitus affords information which is equally important. The presence of bile in the vomitus indicates obstruction beyond the pyloric sphincter; a bile stained vomit, therefore, is not compatible with an obstruction of the type that we associate with Congenital Hypertrophic Pyloric Stenosis. Careful examination of the vomited material will often reveal the presence of excess mucus, and occasionally a little blood. This finding, in conjunction with an increased amount of vomited material, confirms the existence of delayed gastric emptying, dilatation, and consequent gastritis from irritation.

No mention has yet been made of the time of onset of this symptom, and the factors which influence it. Strachauer classifies the vomiting and its time of incidence as follows:— "Vomiting commencing at the end of the second week of life, becoming alarming by the end of the third week, and definitely projectile in character by the fourth or fifth week. Sauer, on the other hand, states that 11% of his cases vomited from birth, 85% after the first week, and 33% after the third week. In his opinion the most frequent time of onset of vomiting is the end of the second week and the beginning of the third week. Steen and Davison have both reported cases in which vomiting occurred very late, at the age of seven months and eight months respectively; /
respectively; these cases are rarities, however, and need not materially influence our conclusions.

A study of the table below shows that the average age of incidence is toward the end of the second week and the beginning of the third week. In this connection our own average age of onset is three weeks. The earliest age of onset being one week; this early onset was experienced in three cases.

<table>
<thead>
<tr>
<th>Authority</th>
<th>Age at onset of vomiting.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stautsauer</td>
<td>end of second week</td>
</tr>
<tr>
<td>Sauer</td>
<td>end of second week and beginning of third week.</td>
</tr>
<tr>
<td>McLeod</td>
<td>average 3½ weeks.</td>
</tr>
<tr>
<td>Paterson</td>
<td>average 3 weeks (581 cases)</td>
</tr>
<tr>
<td>Wallace</td>
<td>average 3 weeks.</td>
</tr>
<tr>
<td>Thomson &amp; Gaisford</td>
<td>average 2 weeks 4 days.</td>
</tr>
</tbody>
</table>

Turning now to the factors which influence the age of onset. Our study of the pathology in Congenital Hypertrophic Pyloric Stenosis has revealed the existence of marked hypertrophy of the pyloric canal; we have also concluded that, in order that complete obstruction may occur, superadded spasm is necessary.

During the first week of life little food is taken and consequently there is little chance of the "vomiting threshold" being reached. In the second and third weeks, however, the amount of food ingested greatly
greatly increases, gastric retention, with its associated gastritis occurs, the "vomiting threshold" is reached and vomiting occurs. Gastric retention and gastritis increase reflex spasm, and hence the vomiting becomes more forcible as the vicious circle becomes more chronic.

Such an explanation as to the cause and progressive nature of the vomiting will at once make it obvious that the nature of the feeds and the regularity with which they are given are most important factors in determining at what age vomiting is likely to make its first appearance. This irregular feeds or large feeds at long intervals, or irritant feeds will tend to produce gastric dilation and gastritis, and the "vomiting threshold" will therefore be reached at an earlier age. We can conclude, therefore, that "errors" in feeding whether by the breast or bottle, will lead to vomiting at an earlier age than in children that are correctly fed.

II. Visible Gastric Paristalsis.

This sign has been present in all our cases, admittedly it has been more marked in some than others, but careful and patient observation of the child has shown it to be present in some degree in all cases.

Leonard Findlay states that this is the most important single sign on which to base a diagnosis of Congenital Hypertrophic Pyloric Stenosis. Sauer, in an analysis of 113 cases, found it present on 96 occasions.
occasions, and entirely absent in the remaining 17 cases.

In its classic form the peristalsis reveals itself as a tumour, most frequently likened to a "golf-ball" in appearance, which arises from beneath the left costal margin and passes slowly towards the right across the epigastrium, rarely crossing the midline by more than an inch or an inch and a half. The point at which the tumour disappears is determined by the position of the pylorus, and as soon as the peristaltic wave reaches this point a fresh "golf ball" appears under the left costal margin, and commences its progress across the epigastrium from left to right in similar fashion to the first.

The time at which this sign can best be seen varies from case to case. In some infants it occurs during /
during a feed, in others it can be seen between feeds, but more usually it is best seen during the five or ten minutes immediately following a feed. In order that it may be best demonstrated the mother should be seated in a good light, preferably beside a warm fire. The upper abdomen is then exposed and the peristalsis watched for before and during a feed. Should no peristalsis be seen during the feed the mother should now move a little closer to the fire in order that the warm air may play on the child's abdomen; this simple procedure is often remarkably successful in stimulating the appearance of visible peristalsis. In the event of peristalsis still being absent, the skin of the left upper abdomen may be flicked with the finger or the stomach palpated with the hand in order to stimulate peristalsis. There are few cases in which one or other of these simple procedures fails to reveal visible peristalsis; it is not sufficient to examine the child on one occasion only; careful observation should be made before, during, and after each feed, over a period of several days, before all hope of demonstrating this sign is given up.

III. /
III. The Palpation of a Pyloric Tumour.

Contention wages round this sign among all paediatricians, some hold that it can be demonstrated by careful palpation in all cases, others again maintain that by reason of the anatomical position of the pyloric palpation is a physical impossibility in many cases. When palpation of the pyloric tumour is possible, it is felt most usually an inch to the right and just above the umbilicus. The tumour presents to the fingers a feeling of elasticity through the abdominal wall. In size and shape it may be likened to a small walnut.

Turning now to the question of the relative frequency with which this sign can be elicited, we find that opinions vary very considerable on this point. The table below gives figures from the case records of several well known authorities, and shows that there is a wide divergence as regards the results. In our own series a tumour was definitely palpated in seven cases out of a total of seventeen.

<table>
<thead>
<tr>
<th>Authority</th>
<th>No. of cases</th>
<th>Tumour palpated</th>
<th>Tumour not palpated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Still</td>
<td>248</td>
<td>246</td>
<td>2</td>
</tr>
<tr>
<td>Foynton</td>
<td>55</td>
<td>54</td>
<td>1</td>
</tr>
<tr>
<td>Strauss</td>
<td>221</td>
<td>55</td>
<td>166</td>
</tr>
<tr>
<td>Davison</td>
<td>60</td>
<td>36</td>
<td>22</td>
</tr>
<tr>
<td>Sauer</td>
<td>119</td>
<td>111</td>
<td>8</td>
</tr>
<tr>
<td>Wallace</td>
<td>145</td>
<td>35</td>
<td>110</td>
</tr>
<tr>
<td>Bolling</td>
<td>454</td>
<td>453</td>
<td>1</td>
</tr>
</tbody>
</table>

The reasons for such widely divergent results are not difficult to find if we bear in mind the fact /
fact that the liver occupies from two fifths to three fifths of the whole abdominal cavity in young infants. Thus the pylorus may be completely covered by the lower lobe of the liver, and cannot be palpated for this reason. Again, the thickness of the abdominal wall, and the degree of relaxation at the moment of palpation, are bound to influence the success or otherwise of the examination. Some clinicians advise turning the child on its face on to the warm hand, hoping to bring the pylorus up against the abdominal wall by this manoeuvre. In all cases frequent and patient examination must be made; no case can be said to have been completely investigated unless the pyloric tumour has been sought while the child is asleep, while feeding is in progress, and immediately following gastric lavage. There are clinicians who advocate the administration of a light general anaesthetic to facilitate examination, but this is neither safe, nor certain in the results achieved. Every effort should be made to demonstrate the presence of a palpable pyloric tumour, because it affords, in our opinion, "The" most valuable diagnostic sign in this condition. Care must be taken, however, in the identification of the tumour, such conditions as prolapsed right kidney, congenital cystic kidney, Spigelian Lobe of the liver, and a distended gall bladder may simulate a pyloric tumour. The occurrence of these conditions /
tions, along with the other symptoms and signs of Congenital Hypertrophic Pyloric Stenosis is very unlikely, and therefore, this danger is probably more theoretical than real.

IV. Constipation.

This symptom is present in almost all the cases encountered; in our own experience it was present in 98% of cases on admission to hospital. The severity of the constipation is closely related to the frequency with which vomiting is occurring in each case, and we have found that marked constipation goes hand in hand with projectile vomiting due to the small amount of fluid entering the intestine. The stools are small, well formed, and scanty; the size of stool passed is important, as it affords an index of the amount of nourishment that is passing through the pylorus. Occasionally constipation alternating with diarrhoea may be found. This diarrhoea is of the "starvation type", and consists of mucus mixed with the intestinal secretions; this type of diarrhoea may therefore be anticipated in any case in which little or no nourishment is passing through the pylorus into the intestine over a long period.

B) Subsidiary Symptoms and Signs.

The following symptoms and signs, although not essential for diagnosis, are of considerable value as confirming evidence of the existence of Con-
genital Hypertrophic Pyloric Stenosis. Taken in association with the four Cardinal Symptoms and Signs they represent the clinical picture that might be expected in a classical case of the disease.

I. The facies.

The appearance of the child as it lies in bed is frequently striking and rather characteristic. The face is pale and thin, the brow furrowed, and the eyes bright. The sucking pads are prominent, and when observed over a period of time the infant is usually found sucking a finger or gnawing its fist at intervals, but otherwise he lies quietly in bed.

II. Loss of Weight.

This symptom requires some qualification; a failure to gain in weight, in the absence of fever or other sign of infection, is a more accurate description. It has already been noted that these infants are a little above the average weight at birth, on their admission to hospital, however, they are usually considerably below the average weight for their age. Those looking after them at home are often struck by the fact that no progress is being made, or that the child's skin is loose and hanging in folds, especially over the abdomen and lower extremities. In some cases the loss of weight is so extreme as to constitute true emaciation.. The normally rounded contours of an infant's body are lost due to lack of fatty /
fatty tissue, the brows are prominent and the eyes appear deep set; the skin hangs in superfluous inelastic folds from the lower extremities and abdomen.

III. Dehydration.

This sign varies considerably, and its degree depends upon the severity and frequency of the vomiting which is occurring. An accurate indication of the presence of dehydration can be obtained from the examination of the anterior fontanelle and the tongue in these cases. A dry white coated tongue indicates that there is a lack of fluid and, should the anterior fontanelle also be depressed, measures should at once be instituted to increase the fluid intake, as a state of marked dehydration then exists.

IV. Alkalosis.

Graham and Morris, of Glasgow, have drawn attention to the frequency with which signs of alkalosis can be observed in cases of Congenital Hypertrophic Pyloric Stenosis. They have made clinical observations with coincident blood analysis in these cases, and have reached the following conclusions.

(a) "Pyloric Stenosis in infants is associated with alkalosis, due to loss of the acid radical chlorine from the body; the loss of chlorine is roughly related to the severity of the vomiting."

(b) "The rise in CO₂ in the blood is evidenced by a depression of the breathing (Biot type of respiration /
respiration), which affords a ready method of its clinical recognition."

Routine blood analysis is neither practical nor desirable in all cases, but in view of the above conclusions the clinical observation of a very drowsy child with a shallow type of respiration, and a diminished respiratory rate, or - in severe cases - periods of apnea followed by three or four shallow respirations (Biot respiration), is sufficient to indicate the presence of alkalosis. Cases have been described, however, in which the alkalosis was so profound that the child exhibited hypertonicity of the muscles, carpo-pedal spasm, and a positive Chvostek sign. We have never seen a case of Congenital Hypertrophic Pyloric Stenosis showing these signs of advanced alkalosis, but when present they are sufficiently striking to merit careful attention.

The infrequent occurrence of tetany is no doubt due to CO₂ retention occasioned by the shallow type of respiration (Maizels).

V. Changes in the Upper Abdomen.

Inspection of the upper abdomen may afford confirmatory evidence of pyloric obstruction, and thus aid in diagnosis. In some cases a degree of flattening may be observed in the R- hypogastrium due to the failure of food to pass into the duodenum and upper /
upper ileum. Much more frequent, however, is the observation of a distinct fulness in the epigastrium, due to dilatation of the stomach and hypertrophy of its musculature.

VI. X-Ray Appearances.

The routine administration of a barium meal and the taking of x-ray photographs at intervals thereafter has fallen into disfavour in this country. There are two objections to this type of investigation. In the first place the barium meal upsets the child considerably, and secondly the results obtained tend to be confusing rather than helpful. The most that can be expected from such an examination is the demonstration of a dilated stomach and a long delayed gastric emptying; this is illustrated in the reproductions in figs. XXI., XXII., and XXIII. which show somewhat dilated stomachs and delayed emptying after a period of four hours.

[Images of x-ray reproductions are shown: fig. XXI., fig. XXII., fig. XXIII.]
figs XXI. XXII. & XXIII.

X-Ray Appearances of the stomach in Congenital Hypertrophic Pyloric Stenosis.

fig. XXI. P.S. aged 3 weeks.
fig. XXII. W.L. aged 7 weeks.
fig. XXIII. D.B. aged 8 weeks.

Note: - Retention of barium in the stomach after four hours.
Dilatation of the stomach well marked in fig. XXII.

Many American authors, and most notably Strauss, stress the importance of examining these cases under the x-ray screen after a barium meal; they claim that by this means a positive diagnosis can be made in almost every case and Strauss, moreover, undertakes to differentiate between true Stenosis and Spasm by this means.

"A small amount of barium sulphate is added to the breast milk which the infant is given while he is being observed under the fluoroscope. The rhythmic, snake like, peristaltic contractions seen in the pylorus, independent of the contractions of the rest of the stomach, are absolutely pathognomic of Congenital Pyloric Stenosis." (Strauss).

VII. The Urine.

This is small in amount, of high specific gravity, and usually alkaline, p. H. 6-7.

Should there be an element of alkalosis present the urine may, however, be acid, p.H. 4-5, due to the retention of the bicarbonate in the blood and tissues to compensate for the chloride ion lost dur-
during vomiting.

When vomiting is very severe the urine is frequently free of chloride, as demonstrated by the absence of a white precipitate when AgNO₃ is added to the urine already acidified with nitric acid.

Summary.

The importance and clinical significance of the Four Cardinal Symptoms and Signs have been described, and an attempt made to elaborate how they may be elicited by careful and repeated clinical observation. Seven Subsidiary Symptoms and Signs are enumerated which, while not comprehensive, are in our opinion the most helpful, as confirmatory findings, making diagnosis more certain.

Here, as in all disease of early infancy, the key to Diagnostic Success lies in painstaking examination, and careful continuous clinical observation of the patient.

Differential Diagnosis.

There are two other conditions which should be considered before a positive diagnosis of Congenital Hypertrophic Pyloric Stenosis is made.

The first is Pyloric Spasm or Pylorospasm; a condition which presents features very similar to Congenital /
genital Hypertrophic Pyloric Stenosis. So much akin are the manifestations of these two conditions that a most detailed analysis of the symptoms and signs is necessary, before an opinion can be given with any degree of accuracy.

The second can be termed Duodenal Obstruction, and should afford little difficulty in differentiation, if a careful clinical examination is made and an accurate history is obtained.

Pyloric Spasm has received extensive study both in America and France within recent years, and the following points of difference between the two conditions have been noted.

I. Pyloric Spasm is much more common in female than in male infants.

II. No predeliction for the first born child has been found.

III. Children with Pyloric Spasm tend to vomit from birth or within the first week of life; the vomiting occurs regularly after every feed, and is projectile in type from its commencement.

IV. Gastric peristalsis, while demonstrable, is seldom so well marked as in Congenital Hypertrophic Pyloric Stenosis, and is often accompanied by spasms of colicky pain which cause the child to scream.

V. No pyloric tumour can be palpated.

VI. /
VI. Constipation is invariably present, but the stool is usually larger in bulk.

VII. The infant with Pyloric Spasm is restless and irritable in bed, and frequently inhibits signs of muscular hypertonia. Such muscular hypertonia can be best demonstrated by lifting the infant by the wrists when the elbows will remain partially flexed; the knees similarly, fail to extend fully when the infant is lifted by the ankles.

VIII. As previously mentioned some writers claim that the appearances of the stomach and pylorus under the fluorescent screen during the ingestion of a barium meal are suggestive. Thus a case in which rhythmic pyloric contraction coincident with gastric contraction is seen would suggest Spasm rather than true Congenital Stenosis in which the pyloric contractions are independent of the gastric contractions.

IX. During the routine x-ray examination of cases of Pyloric Spasm an enlargement of the thymus gland has been demonstrated with remarkable regularity. Thus Rubin, in 1928, reported the co-existence of an enlarged thymus gland along with Pyloric Spasm in thirteen consecutive cases, whereas he found an enlarged thymus in only one case out of seven in Congenital Hyper trophic Pyloric Stenosis.

X./
X. It has been found that Spasm of the Pylorus responds readily to treatment with anti-spasmodic drugs, atropine sulphate being the most commonly used. Ward writes in this connection: "If, on the administration of atropine sulphate and on a thick diet, the child improves, the case is one of Pylorospasm; if no improvement follows these measures, or if the symptoms continue to grow worse despite them, a diagnosis of organic obstruction is made."

B) Duodenal Obstruction may occasionally simulate the Signs and Symptoms of Congenital Hypertrophic Pyloric Stenosis, but we would lay stress on the fact that some additional symptoms or signs are usually present in these cases, which, if given their proper value, make erroneous diagnosis unlikely. On the other hand, cases of Duodenal Obstruction do occur in which differentiation is extremely difficult, unless due weight be given to the absence of positive signs of Congenital Hypertrophic Pyloric Stenosis.

To make these points clear the following cases are referred to:

I. Case reported by H.H. Greenwood.

J.V.C. aged 3/12th years.

History: First born male child, breast fed throughout. Attacks of acute colicky pain when aged two and a half months; pain relieved by vomiting partially digested milk and some greenish bile.
On Examination.

No visible gastric peristalsis.

Edge of liver 2" below the R-costaI margin.

Attached loosely to the liver margin, but much more mobile, was a tense hard swelling about the size of a walnut, close under the thin abdominal wall.

Laparotomy Findings.

Gall bladder enormously and tightly distended.

The antrum of the gall bladder lay across the second part of the duodenum compressing it against the spine.

Comment:-

The occurrence of acute colicky pain and the bile stained vomitus, taken in conjunction with a "tumour" attached to the liver margin, all pointed to some other condition than Congenital Hypertrophic Pyloric Stenosis. This interpretation of the findings was confirmed by the absence of visible gastric peristalsis. There were, then, in this case additional symptoms and signs which would lead to the consideration of Duodenal Obstruction as the cause of the symptoms.

II. Four cases reported by E.F. Burt and R.M. Tyson.

These cases were aged one day, three weeks, four weeks, and two days respectively, when vomiting commenced.

The vomiting was projectile in type and never bile stained.
Peristalsis was seen in two cases, and absent in the first and third.

In no case was a pyloric tumour palpated, and constipation is not remarked upon.

Laparotomy.

In all four cases the obstruction was caused by primary transduodenal bands; these bands were of congenital origin and stretched from the under surface of the liver and gall bladder to the pylorus and duodenum. They were similar in type to the adhesions and bands which Morris, in 1905, called "Cobwebs in the attic of the abdomen."

Comment:-

These four cases were typified by the absence of positive symptoms and signs of Congenital Hypertrophic Pyloric Stenosis; this would seem to have been the only factor by which they could have been differentiated from Duodenal Obstruction.

The findings at laparotomy constitute a rare cause of duodenal obstruction, and the absence of bile from the vomitus is especially difficult to explain.

III. Case seen in the Sick Children's Hospital, Sheffield.

C.M.W. age 3/52.

Male child, first born in the family. Breast fed throughout.

Vomiting commenced on the third day of life and was /
was intermittent at first. By the end of the second week the vomiting was definitely projectile in type, occurred after almost every meal, the vomitus being slightly bile stained. The bowels were at first constipated, but later the stools became frequent, loose and green.

On Examination.

A wasted dehydrated infant.

Slight gastric paristalsis seen on three occasions following a feed.

No pyloric tumour palpated.

A barium meal revealed gastric retention after four hours, and a small residue of barium in the stomach after ten hours.

Laparotomy.

Right paramedian incision under local anaesthesia. Stomach grossly dilated; no free fluid in the abdomen. Slight thickening of the pylorus, but no definite tumour present. Rammstedt's operation performed.

Progress:

The bile stained vomiting continued after operation, and the child became still more dehydrated, Death occurred two days after operation.

Post Mortem Report.

General appearance:

A very wasted male child.

Recent clean laparotomy wound.

Peritoneum. No peritonitis.

Stomach. Dilated.
Recent Rammstedt operation wound in the pyloric region.

**Intestines.**

The duodenum was dilated as far as the flexure. The Caecum and appendix were found lying immediately over the duodeno-jejunal flexure. The Caecum and terminal ileum were twisted on themselves four or five times, and this volvulus was pressing on the terminal duodenum. The remainder of the small intestine was very dark in colour and congested, but there was no evidence of actual gangrene.

**Conclusion.**

Volvulus of caecum and lower ileum causing obstruction of the duodeno-jejunal flexure.

**Comment.**

The diagnosis was missed in this case, because the significance of the slightly bile stained vomit was disregarded. On the other hand, no pyloric tumour could be felt, and it is surprising that the volvulus was not palpated when these examinations were made.

At the operation there was no free fluid in the abdomen nor was there any difficulty in delivering the pylorus into the small incision employed. It was only when vomiting continued after operation, and the child's condition became grave, that duodenal obstruction was thought of as the cause of the initial symptoms.
SUMMARY :-

The major points of difference between Pyloric Spasm and Congenital Hypertrophic Pyloric Stenosis have been enumerated. With the exception of the radiological differentiation, the other features can be easily elicited from a careful history and a simple analysis of the clinical signs.

The rare condition of Duodenal Obstruction whether due to the pressure of a hollow viscus, congenital adhesions, or volvulus has been described and illustrated by cases. The possibility of such an occurrence must always be considered when bile appears in the vomit.
The treatment of Congenital Hypertrophic Pyloric Stenosis is Medical and Surgical.

The view most commonly held in this country at the present time is that the infant should be admitted, in the first place, to a medical ward, where treatment is inaugurated to ameliorate symptoms and improve the general condition of the child. Such treatment should not be persevered with too long unless marked improvement is taking place, as soon as the physician feels that no further progress is being achieved a surgeon should be asked to operate, otherwise the infant will be so debilitated that surgical operation holds small prospect of success. Following operation the child should again return to the medical wards for the equally important medical care following operation.

This view advocates initial treatment as a "feeding case" and, in the event of non success, the immediate employment of surgery as an "incident" in treatment, at a time when the child is in the optimum condition to ensure surgical success. Such a scheme of treatment is usually applicable to these cases, considerable variation is bound to occur, however, depending upon the state of the infant when first seen, and the length of time that has elapsed since the onset of vomiting.

Medical treatment alone has been carried out with
with considerable success within recent years both in America and in Denmark. In these countries boundless time and thought are devoted to these infants, and with the help of a large staff working continuously day and night medical treatment has effected a permanent cure in many cases.

Occasionally cases are seen in which the infant's condition is so grave as to constitute an "abdominal emergency" from the first. In such cases the minimum of time is given to preparing the child for the surgeon; the only hope of recovery lying in the remarkable rapidity with which the general condition improves following operation.

I. We propose dealing with the General Medical Treatment of a case of Congenital Hypertrophic Pyloric Stenosis and then giving the indications for surgical operation.

II. A brief survey of the relative advantages and disadvantages of Medical Treatment Alone will be made.

III. Lastly the Preoperative Preparation, actual Operative Technique, and Post-Operative Treatment of a case will be described.

I. General Medical Treatment.

The aim of treatment is to decrease or stop vomiting, and improve the general condition of the infant. If the child is breast fed every effort should be made to continue breast feeding; the mother being urged to live near the hospital, or arrangements being made /
made to evacuate the breasts with a pump several times in the day.

I. The ideal place to nurse a case of Congenital Hypertrophic Pyloric Stenosis is an isolation ward, here the child can lie quietly, undisturbed by other children, and far removed from the possibility of infection which is ever present in a general ward. This method is employed in the Children's Hospital, Sheffield, with marked success.

II. Equally important is the nursing staff; the nursing of a "Pyloric Baby" requires skill, patience, and experience on the part of the nurse, if possible, she should devote her whole time and attention to the one case, and thus be able to give adequate time to the feeding and observation of her little patient.

III. Coming now to the question of feeding, no matter what type of feed is being given the cardinal rule should be small feeds at short intervals.

Most physicians advocate gastric lavage before the administration of a feed; this procedure removes any curds retained from the last feed and excess mucus present from gastritis, and this minimises the chance of vomiting following the subsequent feed. Many babies do not tolerate lavage well and become exhausted if it is persisted with, in the majority of cases, however, it will be found beneficial if used with discretion.

In the event of the child being breast fed every effort must be made to continue with this. The infant should /
should be put to the breast for a period of from 10 to 15 minutes every 2 hours, but it is probably better to withdraw the milk from the mother with a breast pump, and then administer it with a spoon to the child. In this way the amount given to each feed can be more accurately controlled according to the child's age and weight.

If artificial feeding is adopted there is a choice of feeds. Initially a mixture of either, milk and water half in half with sodium citrate gr. i. to the ounce or, lactic acid skim milk and water in the proportions of one to a half should be tried. Dextrimaltose gr. to the ounce may be added to either of these feeds. In the event of vomiting persisting changes in the proportion of milk to water should be tried, and a slight lengthening of the time between each feed up to three hours instituted. Should vomiting still continue recourse may be taken to the administration of thickened cereal feeds. This method of feeding is strongly advocated by Sauer, who advises the following method of preparation and administration of the feed:

**Ingredients:**
- Skimmed milk 9 oz.
- Water 12 oz.
- Farina 6 tablespoonfuls
- Dextrimaltose 3 tablespoonfuls.

**Preparation:**
Mix together and boil for one or two hours in a double boiler.

**Administration:**

The /
Administration:–

The thick feed should be scraped off a spoon on to the back of the tongue with a spatula. Feeds of two to eight tablespoonfuls should be given six times in the day. Wollstein advocates that the feed be given through a modified Hygeia nipple, the thickened feed being "basted" in with a spoon. Sauer had great success with this thick feed in 26 out of 35 cases, and L. Porter also reports success in ten cases so treated.

In our experience the feed advocated by Sauer is too thick, but good results have been seen repeatedly, using Benger's Food 1 oz. to a pint of milk or water mixture for thickening; this has the additional advantage of easy administration. The explanation why thickened feeds often effect an improvement is given by Hotz as follows:– "The stomach wall has the power of surrounding and grasping its food content; this is termed peristolic function (Stiller). The peristolic activity is not well developed in the newborn infant, but is best brought out when food of sufficiently thick consistency is taken. At such times the gastric content is subject to the combined muscular peristalsis and peristolsis. The latter tends to retain the food and the former to propel it."

Davison has suggested a further method of trying to overcome persistent vomiting, although we have not seen this method employed it seems worthy of trial in difficult cases. He writes:– "The fact that in many
many cases of pyloric stenosis a palpable tumour can only be palpated immediately after a peristaltic wave would seem to indicate that the hypertrophied pyloris is relaxed between gastric contractions. Because of this fact feeding and refeeding is frequently successful. If food is administered persistently, eventually some of it may pass the hypertrophied pylorus during one of its periods of partial relaxation, and in this way the patient's nutrition may be maintained."

IV. Certain antispasmodic drugs are frequently used in conjunction with feeding methods in an effort to reduce the vomiting by relaxing pyloric spasm. Atropine sulphate, Tincture of Belladonna, Luminal, and more recently Eumydrin have all been used sometimes with marked success, and at other times with equally marked failure.

Atrophine Sulphate.

This drug can be administered orally or by hypodermic injection; whichever method is employed the importance of using a freshly prepared solution should be borne in mind. For oral administration the initial dose is one drop of 1 in 1,000 solution (.05 mgm.) half an hour before a feed; this can be increased by one drop at every alternate feed up to the point at which vomiting ceases, or a dry tongue, dilated pupils, and physiological flushing appear, indicating toxic effect. We have not seen this method of treatment employed /
employed, but Haas, Ibrahim, and A. Johannessen all report its beneficial effect.

Burt and Tyson employed atropine sulphate \( \frac{1}{1000} \) gr. hypodermically quarter of an hour before a feed; this method has obvious disadvantages if employed over a long period of time.

**Tincture of Belladonna.**

This drug is administered orally half an hour before a feed in doses of one drop, increasing by one drop with each feed until vomiting ceases or physiological effects similar to those of atropine poisoning make their appearance. It was used in the treatment of three cases, subsequently operated upon in the Sick Children's Hospital, Sheffield. In these cases it failed to stop vomiting, but in two of the cases vomiting was reduced from after every feed, to two or three times in twenty four hours.

**Luminal.**

Sodium Luminal has been tried by some clinicians in doses of \( \frac{1}{8} \) to \( \frac{1}{6} \) of a grain, half an hour before feeds, in the belief that it depresses the vomiting centre and damps down reflex spasm. Its use is not without danger, however, especially if alkalosis is suspected, as depression of the respiratory centre is also liable to occur.

**Eumydrin.**

E. Svensgaard of Rigs hospital, Copenhagen, pub-
lished a series of cases in 1935, treated by this new drug. Eumydrin is Atropinemethylnitrate and is fifty times less poisonous than atropine, although the efficacious dose is only two or three times larger. A 1 in 10,000 fresh solution is used in doses of 5 or 2.5 cc., three or four times a day, half an hour before a feed. At first 5 cc. was employed, equalling .5 mgm of the drug, but more recently good effect has been observed with 2.5 cc. or .25 mgm of the drug. Toxic effects are indicated by a redness of the skin and some slight pyrexia; these have only been noted in cases where treatment was commenced before dehydration had been overcome.

This method of treatment was employed in Copenhagen in 61 cases, without operation, with a mortality of 1.6%. The drug has been employed in the Royal Hospital for Sick Children, Edinburgh, with good results. One case so treated indicates the value of this new treatment under certain circumstances.

C.S. a female child aged 2/12 years. The history was that she was bottle fed and had vomited persistently for two weeks.

The birth weight was 3½ lbs and her weight on admission was 6½ lbs.

She had been markedly constipated with small hard stools for two weeks before admission.

On examination visible gastric paristalsis was seen on several occasions, but there was some doubt as
as to whether the pyloric "tumour" could be palpated.

**Treatment:**

The little patient had been treated by her own doctor with feeds thickened with Sister Laura's food, and drop doses of atropine sulphate without any appreciable improvement.

On admission she was given three hourly feeds of a milk and water mixture equal parts, with Eumydrin 5 cc (.5 mgm) half an hour before each feed; this brought about a marked improvement. The weight, after about one month in hospital, was 7½ lbs. and vomiting occurred occasionally, but seldom more than once in the day. At this point, when she was three months old, it was thought advisable to employ surgical aid, and accordingly operation was arranged for. On the morning of operation, however, the temperature had suddenly risen to 103.4°F, the respirations were 80 per minute, and rales could be heard all over the chest. An acute bronchitis had developed and surgery was therefore deemed impossible. Eumydrin treatment was continued, and once the acute infection of the chest had cleared up the vomiting practically ceased, and the child continued to gain weight.

**Comment:**

This case recovered without surgical aid on Eumydrin treatment alone. Atropine sulphate had previously been used without good effect; the results from Eumydrin were much more satisfactory. The Eumydrin undoubtedly
undoubtedly carried this child through a very difficult and dangerous period when surgical aid was impossible, because of the presence of an acute infection. The age of the child (2/12) when admitted may, however, have been an important factor in this case as it is well known that the spontaneous cure of Congenital Hypertrophic Pyloric Stenosis frequently occurs during the age period three to four months. On the other hand, the facts that no pyloric tumour was ever definitely palpated and that the patient was a girl might be taken as indicative of Pylorospasm rather than true Hypertrophic Stenosis.

V. Dehydration must be combated from the outset in these infants, probably the best means of replacing the fluid loss due to vomiting is by the administration of subcutaneous or intravenous saline. Rectal salines are seldom retained well by young infants, and only tend to induce diarrhoea. The subcutaneous saline may consist of Normal Saline (.9 per cent Na Cl), Ringer's solution (Na Cl .7 per cent, K Cl .01 per cent, and Ca Cl₂ .02 per cent), or 6 per cent anhydrous glucose saline, and can be given twice in the day as a routine or in proportion to the frequency and severity of the vomiting. The amount given at a time varies from 60 to 150 cc; the best rule in this connection being to give an amount that the tissues seem capable of absorbing without becoming indurated and waterlogged.

In the event of dehydration being very marked at the /
the outset it can be more rapidly overcome by the iv administration of any of the previously mentioned solutions. The amount given by the iv route should depend on the weight of the infant; 25 to 30 cc of normal saline or Ringer's solution per pound body weight and 20 to 25 cc of glucose saline per pound body weight being the usual proportion. The fluid, heated to body temp., should be run in slowly by the gravity method through a metal needle inserted into the longitudinal sinus at the posterior angle of the anterior fontanelle.

By the introduction of fluid the infant's general condition is rapidly improved, colour returns to the face, the breathing becomes stronger, and the pulse is better sustained.

VI. Obstinate Constipation can be relieved by the gentle administration of a soap and water enema every second day as required. In some cases it may be necessary to augment this by a saline purge; these are best avoided if at all possible, because they tend to irritate the empty intestines and cause diarrhoea.

VIII. The hygiene of the mouth and nose require careful attention if projectile vomiting is occurring; the nose should be washed out with warm normal saline or water after every vomit in order to clear it of small clots of curd which lodge in the nasal passages and cause irritation.

IX. /
IX. A most careful watch should be kept for a slight rise in temperature or undue irritability on the part of the child. These signs at once suggest the advisability of an auroscopic examination, as otitis media is liable to occur following projectile vomiting which forces organisms or foreign material up the Eustachian tube.

X. Lastly the child's weight should be taken each day or every alternate day. In this way an accurate record of progress can be kept, and the efficacy of treatment evaluated.

Indications for Surgical Operation:

The determination of the correct time at which to employ surgical aid in Congenital Hypertrophic Pyloric Stenosis is not an easy matter, but there are certain factors and signs which serve as a guide in this respect, and provide definite indications for surgical operation. Consideration of the child's age may lead the physician to delay operation longer than would ordinarily be justified. Thus it is justifiable to persist with medical treatment if the child is at or past the age of three months, when spontaneous recovery can be reasonably expected to occur within a short time. Again the desirability of the continuance of breast feeding makes early operation essential in some cases. If the mother can only report at the hospital for a short time, or if her milk shows signs of failing, then operation is immediately indicated to ensure the
the supply of breast milk during convalescence. The persistence of frequent projectile vomiting after seven to ten days of careful medical treatment should lead to the consideration of surgery, and if, in addition, the weight is not increasing then operation is indicated without delay. A steady loss in weight, even associated with a decrease in the frequency of vomiting, is an indication for operation before the child's condition becomes worse; Cochran recommends operation if the child has lost more than 20% of the birth weight. Lastly, the child that is amaciated, collapsed, and dehydrated on admission must be treated as a surgical emergency. Only a few hours are given to the restoration of body fluids, and then operation is performed in the belief that the only hope of recovery in this type of case lies in the very rapid, and often miraculous improvement that follows operation.

In conclusion we briefly enumerate the indications for operation thus: -

1. Operate on the breast fed infant to ensure the continuance of breast milk during convalescence.
2. Operate when vomiting persist, and the weight is stationary.
3. Operate when the weight is decreasing.
4. Operate on the amaciated, dehydrated, collapsed infant.

Summary
Summary:-

Breast feeding must be continued if at all possible once the child has been admitted to hospital; should the mother be unable to attend the hospital for the purpose of suckling her child the importance of trying to keep her breasts active until the child can go home, after operation, should be emphasised to her.

Gastric lavage in conjunction with regular feeds - in proportions and at times found suitable by trial - is the basis of medical treatment. These measures will usually be found to considerably decrease the frequency with which vomiting occurs. Should little or no improvement be achieved then a trial can be made of the thickened feed. In the event of continued non-success recourse can be taken to the administration of one of the anti-spasmodic drugs. Eumydrin is, in our limited experience, a much more effective and less dangerous drug than any of the older remedies.

Dehydration must be combated from the outset, if well marked when the child is first seen the intravenous administration of fluid is indicated, followed later by subcutaneous salines at intervals. We are well aware that subcutaneous salines tend to be painful during administration, but their continued use over a long period is less dangerous than frequent puncture of the longitudinal sinus through the anterior fontenelle.

The time at which surgical operation is to be undertaken /
undertaken is a matter for the judgment of the physician in the light of his previous experience of these cases. Early operation is the safest choice, and delay — once a definite diagnosis has been arrived at — is fraught with considerable danger.

II. The Advantages and Disadvantages of Medical Treatment Alone.

A comparison of the mortality rate in medically treated cases, and those that are operated on as an "incident" in treatment shows conclusively that surgery raises the mortality rate. In 1930, Rammstedt published interesting figures in this connection, collected from sixty different clinics. The total number of cases was 1,342, and of these 1,342 were treated medically with a mortality of 16%. On the other hand, the 497 cases treated by surgical operation showed a mortality of 22.5%.

Taking the figures quoted in Svengaard's paper in December 1935 thus:

<table>
<thead>
<tr>
<th>Authority</th>
<th>Medical Treatment</th>
<th>Mortality per cent.</th>
<th>Surgical treatment</th>
<th>Mortality per cent.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bayer</td>
<td>40</td>
<td>2.5</td>
<td>46</td>
<td>26</td>
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<tr>
<td>Birk</td>
<td>56</td>
<td>18</td>
<td>20</td>
<td>0</td>
</tr>
<tr>
<td>Behrmann</td>
<td>61</td>
<td>18.6</td>
<td>50</td>
<td>28</td>
</tr>
<tr>
<td>Munting</td>
<td>66</td>
<td>16.6</td>
<td>58</td>
<td>12</td>
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<tr>
<td>Wiedhopf</td>
<td>35</td>
<td>5.7</td>
<td>15</td>
<td>13.3</td>
</tr>
<tr>
<td>Willi</td>
<td>87</td>
<td>5.7</td>
<td>37</td>
<td>24.3</td>
</tr>
<tr>
<td>Hochstein</td>
<td>92</td>
<td>18.4</td>
<td>110</td>
<td>3.4</td>
</tr>
</tbody>
</table>

We find the total number of cases treated by these seven authorities was 773, of these 437 were treated medically /
medically with a mortality of 7.2%, whereas 336 cases surgically treated show a mortality of 15.2%.

This lower mortality figure is the only advantage which medical treatment alone has over surgical treatment, and in our opinion it is far outweighed by the very real disadvantages now to be described.

The disadvantages of medical treatment alone are largely economic; such treatment is lengthy, costly, and not without danger. Medical treatment involves a very long stay in hospital.

The average stay in hospital of a case treated surgically was 19 days in our series of cases; compare this with the average of 53 days for medical treatment quoted by Davison, or the still longer period of 77 days quoted by Svengaard. Medical treatment is, then, at a definite disadvantage as regards the economic factor of length of time spent in hospital. Again medical treatment is costly; extra nurses are required to carry out the detailed nursing regimen so necessary for success, and should the baby be breast fed the mother may require financial assistance to enable her to attend the hospital. Further, medical treatment carried on for a long period of time may suddenly prove unsuccessful; the child is then left to face operation in a grave state of malnutrition. Lastly, and not least important, is the fact that every extra day in hospital is continuing the danger of enteritis developing. No matter what precautions are /
are taken a young infant nursed for a long period in a general hospital, where there are bound to be cases of enteritis in the wards from time to time, is ever open to this risk. We have been fortunate in Edinburgh in this respect, largely due to very efficient nursing. In Sheffield, on the other hand, two deaths from enteritis had occurred in "Pyloric Babies", shortly before the custom of nursing the child in an isolation ward was adopted.

Summary:-

Medical Treatment Alone has been very successful as regards the lower mortality prevailing in the Scandinavian hospitals. Such results can only be achieved by long continued patient nursing, an exacting attention to detail, and a highly specialised nursing staff working day and night.

In our opinion the difference between the Medical and Surgical mortalities is not sufficiently great to justify the increased cost, exposure to the risk of infection, and exacting nursing regimen entailed if Medical Treatment Alone is adopted as the ideal for all cases.

III. The Surgical Treatment of Congenital Hypertrophic Pyloric Stenosis.

To ensure clarity of description we propose dividing this subject into three parts.
I. The Preoperative Preparation of a Case.
II. /
II. The Actual Operative Technique.

III. The Post Operative Treatment.

(a) The Preoperative Preparation of a Case.

Preoperative treatment is aimed at combating these conditions which make operation dangerous; in Congenital Hypertrophic Pyloric Stenosis such conditions as dehydration, alkalosis, starvation, and anaemia, all tend to greatly increase the operative risk. The combating of these risks is, then, the first purpose of the Preoperative Preparation. In the second place, everything must be done to make the operation easy for the surgeon, and to minimise the possible risks arising out of the actual technique of the operation.

Dehydration can be combated by the administration of normal saline, 6% glucose saline, or Ringer's solution. In our experience the subcutaneous infusion of from 60 to 120 cc of normal saline, half an hour before going to the operating theatre, is all that is required. If the child be very dehydrated, however, the intravenous administration of 6% anhydrous glucose, 20, to 25 cc. per pound of body weight, is probably a better method of restoring the fluid loss.

On the other hand, if vomiting has been very persistent and there is reason to fear the onset of acute alkalosis 5% calcium chloride, 1/2 cc. per pound body weight, should be given along with normal saline, by the intravenous route, before operation.
Emaciation and anaemia can be best overcome by the simultaneous administration of a blood transfusion and 10 to 20% glucose saline. Four of the cases in our series had a blood transfusion of 15 cc of citrated blood, per pound body weight, along with 15 to 25 cc. of 10% glucose saline before operation. The transfusion was given within three hours of operation, directly into the longitudinal sinus, and in all cases a beneficial effect was noted. From the cases observed we would strongly urge the adoption of preoperative blood transfusion as a routine measure, in the belief that such a procedure would materially lower the operative mortality, by reducing post-operative shock and increasing the child's resistance.

Turning now to those measures that aim at making the operation easy for the surgeon. The stomach should be washed out with normal saline half an hour before the operation; this is a most important matter and should never be omitted. By this means the dilated stomach is emptied, and thus becomes much easier to handle once the abdomen has been opened. Should accidental perforation of the stomach or proximal duodenum occur during operation, the resultant soiling of the peritoneum will be less dangerous if the stomach has been previously washed out. Again there is a distinct danger of the child vomiting during operation under local anaesthesia, if the stomach has not been washed out and respiratory obstruction is liable to occur,
with disastrous results. Lastly, when the operation is complete, stagnant food and mucus will pass into the easily irritated intestine or a plug of mucus may block the pyloric canal. To avoid such undesirable occurrences a careful gastric lavage before operation is essential.

It has been our custom to administer Luminal half a grain, and atropine sulphate 1/250 gr., three quarters of an hour before the operation. The Luminal ensures that the child will be quiet and drowsy by the time he reaches the operating theatre; this greatly facilitates the injection of local anaesthetic, and prevents spasmodic contractions of the abdominal muscles with resultant protrusion of the viscera at a later stage in the operation. The atropine sulphate diminishes the secretions during operation, and the child is also adequately prepared for a general anaesthetic should it be found necessary. Just before being carried to the operating theatre the infant is tied to a padded "Pyloric board" or a wooden crucifix with the arms stretched out at right angles to the trunk. This ensures the minimum of movement during operation, and allows of an adequate exposure of the upper abdomen, without removing warm clothing from the arms, thorax, and lower trunk.

(B) Operative Technique.

The classical Fredet-Rammstedt operation was performed on all the cases in this series, the average time /
time taken to the operation from start to finish being from ten to fifteen minutes. To ensure such speedy and expeditious work it is essential to have an adequate and well organised theatre staff; the theatre sister must have the instruments, sutures etc., prepared before the child arrives, and should be fully conversant with the steps in the operation. It is her duty to have the operating theatre at a temperature of from 75 to 80°C, thus keeping the child warm during operation and minimizing surgical shock.

Anaesthesia.

The injection of local anaesthesia has been found most satisfactory in our cases; on only two occasions was it found necessary to administer a light gas and oxygen anaesthetic towards the end of the operation. This was rendered necessary by spasmodic contractions of the abdominal muscles which tended to cause omental herniation, and made closure of the peritoneum difficult.

Novocain 2% was the local anaesthetic used, with three to four drops of 1 in 1,000 adrenalin hydrochloride added to ensure haemostasis in the operative field.

The anaesthetic was administered by means of a long fine needle introduced under the skin of the abdomen over the right rectus muscle, midway between the umbilicus and the costal margin. Working upwards and downwards from this point the sheath and contents were infiltrated with the anaesthetic. The skin and superficial planes were first anaesthetised and then the deeper /
deeper structures, special care being exercised to adequately anaesthetise the posterior rectus sheath and underlying peritoneum. In this way an area as illustrated in fig. XXIV. was rendered insensative. We have found that some means of distracting the child’s attention during the administration of the local anaesthetic and subsequent opening of the abdomen is an advantage. A small feed of sterile glucose water is given for this purpose, and the child focuses its attention on this while the injection is given and the abdomen opened. As soon as the peritoneum has been incised a number fourteen rubber catheter is passed down the oesophagus to withdraw the fluid and air from the stomach before it is handled by the surgeon.

Actual Operation:-

The abdomen is opened by a right paramedian incision two to two and a half inches in length, commencing at a point quarter of an inch below the costal margin. The employment of this high incision ensures that the lower margin of the liver will prevent omental and visceral herniation once the peritoneum has been incised. All bleeding points should be secured with artery forceps, and haemastasis effected with the coagulating diathermy current. When the peritoneum is incised the lower lobe of the liver usually presents in the wound; this should be gently pushed upwards and to the right exposing the pyloric end of the stomach.
fig. XXIV. The Fredet-Rammstedt Operation. 
Reproduction after Bevan.

Note:—

A. (i) The shaded area of anaesthesia. 
(ii) The high incision.

B. (i) The lower lobe of the liver presenting in the wound. 
(ii) The pyloric end of the stomach exposed by pushing the liver upwards and to the right.

With the pyloric end of the stomach as a guide the pylorus can now be delivered with the fingers, and should be rotated so that its upper posterior surface looks directly forwards. Holding the tumour between the forefinger and thumb of the left hand it is then incised longitudinally from end to end through the bloodless area above the level of the pyloric vein. The incision should be half to three quarters of an inch in length, and is made with the special sharp bladed Tyrrell Grey knife. It is carried down to, but not into, the sub-mucosa, which is easily recognised by /
by its yellowish white glistening surface. See fig. XXVI. C. At the duodenal end, the incision should commence at a point half a centimeter proximal to the transverse vein of Mayo, and should be extended through the longitudinal and circular muscle fibres of the pylorus well on to the pyloric antrum. The very greatest care is required while making the incision at the duodenal end of the tumour, because the muscle coats and duodenal mucous membrane are thin and perforation may easily occur. Should such perforation take place it may be recognised by the escape of bile stained bubbles, and can be closed by the procedure illustrated in fig. XXV, as advocated by Lamson.

Fig. XXV. Lamson method of closure in the event of accidental perforation of the mucosa.

The separation of the divided longitudinal and circular muscle fibres is now completed by the insertion into the incision of a pair of blunt forceps, with the blades closed, by gradually opening the blades the muscle edges can be pushed apart so that the submucosa bulges in the wound. Care must be taken to ensure that /
that every circular muscle fibre is divided as otherwise the submucosa will be constricted at one point, and the obstruction to the pyloric canal will remain. See fig. XXVI. D & E.

fig. XXVI. The Fedet-Ramnstedt Operation. Reproduction after Bevan.

Note:-

C. (i) The pylorus gripped between the forefinger and thumb of the left hand. (2) The line of incision.

D. (i) The method of separation of the divided muscle fibres with forceps.

E. (i) The sub-mucosa of the pyloric canal bulging in the wound.

There is little likelihood of bleeding from the field of operation, but should this ensue every effort must be made to stop it. Such haemorrhage can be best controlled by undercutting the vessel with a small round needle and fine catgut; such procedures as the implantation of a tag of omentum or a shred of muscle over the bleeding point are usually unnecessary, and add to the time taken in the operation.

The pylorus is now returned to the abdomen, and the wound closed in layers. The closure of the wound in the /
the abdominal wall is best achieved by means of three "through and through" silkworm-gut sutures which are inserted before the peritoneum is closed. The peritoneum and muscle layers are closed by means of continuous catgut sutures, or silk can be used for the peritoneum if preferred. The skin edges are approximated with fine interrupted silkwork-gut sutures, and finally the "through and through" sutures - already inserted - are loosely tied, so as to ensure adequate apposition of the cut surfaces during healing.

(C) Post Operative Treatment.

On its return from the operating theatre the child is wrapped in a warm blanket, and put in its cot under an electrically heated shock cage. Every effort must be made to keep the child warm at this period as post operative shock is often well marked. The oral administration of fluid is best commenced two to four hours after the operation. A definite scheme such as that here detailed has proved satisfactory in our experience, although some physicians believe in hourly feeds during the first twelve hours following operation.

I. Two hours after operation give one drachm of sterile water or sterile glucose water (1/2 a drachm to 1 ounce) by the mouth with a spoon.

II. Increase the amount of fluid by one drachm every two hours, until-at the end of twelve hours-one ounce of fluid is being given.

III. Fourteen hours after operation a test feed is given.
given. This should consist of the mixture used before operation either milk and water mixture equal parts with sodium citrate or lactic acid skim milk and water equal parts. The test feed should be one ounce in quantity and is given slowly. If it is well retained on the first occasion, it should be continued at two hourly intervals until the end of the first twenty four hours.

IV. During the following twenty four hours the feeds can be gradually increased by half an ounce until three ounce feeds are being given every three hours.

Thereafter the amount of the feed can be adjusted in proportion to the child's age and weight.

In the case of breast fed infants, we have found that the best results are obtained by putting the child to the breast twenty four hours after operation.

It may be necessary to further augment the fluid intake of the child within the first twenty four hours after operation; this can be done by the administration of subcutaneous glucose saline twice in the day. Vomiting may occur following operation but this is uncommon. In our series only three cases vomited after operation, and that was on the second and third days. McLeod states that he has found vomiting after operation occurring most commonly either during the first three days, or from the ninth to the fourteenth day. We have not seen a case that vomited so late after operation. Breast fed infants can be allowed home four
four or five days after the operation, with an adhesive dressing over the wound; they should return to have the sutures removed on the tenth day. The artificially fed child has the sutures removed on the eighth day, and in our experience has been ready for discharge home fourteen to twenty-one days after the operation.

Summary.

The importance of making every effort in pre-operative preparation cannot be overstressed. A few hours devoted to the restoration of body fluids, and the administration of a blood transfusion before operation, frequently weigh the balance in favour of the child's life following the operation.

The actual technique of the operation is simple, and is attended with little danger if adequate care is exercised when making the incision through the muscle layers of the pylorus. Gentle handling of the viscera by the surgeon, and the close co-operation of the theatre sister are the essential factors which go far to minimise shock by excluding trauma and diminishing the time taken to the operation.

Post operative treatment is largely a question of careful nursing; keeping the infant warm, restoring body fluids, and ensuring sleep are the three ideals aimed at to ensure the best chance of recovery.
Post Operative Complications:-

We propose describing the principal complications commonly met with following operation and giving the reasons for these. It will be evident that most of these complications can be prevented by adequate pre-operative preparation and care during the actual performance of the operation.

I. Heamorrhage.

This takes the form of a steady oozing from the exposed surface of the pyloric mucosa or a frank arterial bleeding from the vessels in the divided muscle layers. The capillary oozing from the mucosa is due to the fact that the coagulation time of the blood in these infants is delayed; haemorrhage from the divided muscles, on the other hand, is reactionary in type. Every precaution should be taken at the operation to ensure complete haemostasis. As already described haemorrhage from the divided muscle edges can usually be controlled by undercutting the vessel with a fine cat-gut stitch. Should bleeding persist, or the oozing of blood from the exposed mucosa be noted, the more elaborate muscle or omental graft must be employed to ensure haemostasis before the pylorus is returned to the abdomen.

II. Peritonitis:-

This is caused by the accidental perforation of the
the mucosa of the stomach or duodenum in the course of the pyloroplasty, and the subsequent escape of food or secretions into the peritoneal cavity. Such an occurrence should never take place if adequate care is taken when making the incision and muscle separation at the duodenal end of the tumour. Once the muscle separation is completed, inspection should be made for the escape of bile stained bubbles which are a sure indication of perforation.

Should such a perforation be discovered or suspected it must there and then be repaired by the Lamson method already illustrated in fig. XXV.

III. Shock.

Post-operative shock is probably the most common complication of operation in Congenital Hypertrophic Pyloric Stenosis. Dehydration, emaciation, and anaemia existent before operation undoubtedly play a large part in producing this shock, but we would stress the fact that a preliminary blood transfusion, keeping the child warm, and handling the viscera gently during operation will greatly minimise the degree of shock produced.

IV. Herniation of Abdominal contents.

This is the only complication that we have actually experienced following operation on our series of cases. The factors that make its occurrence likely are an extreme degree of emaciation which renders healing very slow, the employment of inadequate through and through "stay /
"stay sutures", the presence of a low grade infection, or a haematosa in the wound. Any one of these factors may be present alone, but they are more frequently encountered together in view of their close inter-relationship.

Omental herniation occurred in a male child, aged five weeks, who was under our care in the Royal Hospital for Sick Children, Edinburgh. The child vomited on two occasions after operation, and developed loose green stools three days after the operation. On the seventh day, when the dressing was removed from the wound on the anterior abdominal wall, it was found that a piece of omentum had herniated between the sutures, and had become congested, swollen, and oedematous. The child was at once taken to the operating theatre, the wound was opened, and the omentum was returned to the abdomen. During this procedure it became evident that the omentum had pushed its way through a small gap left in the peritoneum during the previous closure. On this second occasion great care was taken in closing the muscle layers, and the child aided by a blood transfusion, made an uninterrupted recovery thereafter.

Comment. The cause of the herniation in this case was slow healing of the wound in conjunction with an imperfect closure of the peritoneum at operation, which had allowed the initial escape of the omentum. There was /
was no haematoma or infection of the wound, and three through and through "stay" sutures had been employed.

V. The Presence of Projectile Vomiting.

This is uncommon, but may occur within the first three days following the operation for the following reasons. In the first place, a few transverse muscle fibres may have been left undivided when the submucosa of the pyloric canal was exposed at operation. These tend to form a residual constricting band, and in conjunction with the oedema of the tissues following incision in this area cause a complete occlusion of the pyloric canal. Secondly, it is essential to extend the incision well on to the pyloric antrum as otherwise symptoms of vomiting will reappear. Gaisford reports a case in which a second operation was necessary, twenty-three days after the first, in order to rectify such an error in operative technique. Thirdly, in cases where pre-operative gastric lavage have not been employed there is a very real danger that a plug of mucous or curd may lodge in the pyloric canal and form a complete obstruction.

VI. Enteritis.

This is one of the most fatal complications met with and must be carefully guarded against by careful feeding and general management. The most probable reasons for its frequent occurrence are as follows:

(a) In many cases little or no food has been passing /
passing into the bowel before operation; following operation food begins to reach the bowel regularly. Frequently the bowel is found to be unused and unprepared for its normal function and diarrhoea will ensue.

(b) Again, by reason of its previous lack of function, the bowel is rendered more susceptible to irritation, and has little immunity to the organisms of infective diarrhoea. Hence when food arrives in the intestine after operation it sets up irritation and causes infection more easily than would be the case had the bowel been functioning normally from birth.

(c) Lastly, the amount of the feed may be too rapidly increased following operation. The bowel is not able to absorb all the food it receives, fermentation in the bowel occurs, and enteritis ensues from this irritation.

VII. Hyperpyrexia.

A rise in temperature following operation may be met with; this is possibly due to the shock of operation disturbing the heat regulating centre which is very susceptible to stimulus in an already dehydrated exhausted infant. Again, there is reason to believe that the passage of food into the small intestine after a period of starvation causes a rise in temperature. In our series of cases the highest temperature recorded following operation was 101°F, on the other hand, Ramsay has reported a case in which the temperature rose to 108°F before death occurred.

VIII. /
VIII. Otitis Media.

The lowered general resistance following operation may lead to this complication. Projectile vomiting forces organisms from the naso-pharynx and small clots of curd up the Eustachian tube; should the child be unable to resist this invasion an acute otitis media will result. We have already stressed the importance of immediately examining the ears with an auroscope if the child develops a temperature or becomes fretful for no apparent cause.

Summary.

Eight post-operative complications have been enumerated, and their underlying cause and prevention have been discussed.

Shock following operation is, in our opinion, by far the most common complication; its degree in all cases depending upon the condition of the child before operation and the time taken to perform the pyloroplasty. We believe that the employment of local anaesthesia in these cases does not increase the post-operative shock.

Enteritis is the most fatal of all the complications following operation; we have been fortunate in avoiding it in our series of cases thanks to the high standard of nursing our cases have received, and the short interval elapsing between diagnosis and operation.

Of the other complications haemorrhage, peritonitis, herniation of viscera, and post-operative projectile vomiting are all grave occurrences which manifest themselves /
selves from time to time. Adequate care on the part of the surgeon in the performance of the pyloroplasty and subsequent closure of the abdomen will, however, greatly reduce the incidence of these complications.
Prognosis:

The parents of a child with Congenital Hypertrophic Pyloric Stenosis which is treated medically with surgical operation as an incident in treatment demand an opinion on two points. They wish to know the chances of survival of the operative risk, and they want an assurance that, should the child survive operation, it will be cured of its symptoms. The prognosis with regard to the operative risk should always be very guarded, as the mortality is high, and the factors influencing it are complex. On the other hand, an assurance can be given that, once the operative risk is passed successfully, a complete cure will eventuate, and that no further disability need be feared in later life.

We have already stated that the operative mortality is high; this is borne out by the figures below collected from the statistics quoted by several well known authorities on the treatment of this condition by the Fredet-Rammstedt method.

<table>
<thead>
<tr>
<th>Authority</th>
<th>Number of cases</th>
<th>Mortality %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Borchard</td>
<td>301</td>
<td>16.3</td>
</tr>
<tr>
<td>Grey &amp; Reynolds</td>
<td>38</td>
<td>55</td>
</tr>
<tr>
<td>Findlay</td>
<td>18</td>
<td>66</td>
</tr>
<tr>
<td>Parsons</td>
<td>70</td>
<td>45.8</td>
</tr>
<tr>
<td>Sauer</td>
<td>120</td>
<td>13.2</td>
</tr>
<tr>
<td>Page</td>
<td>90</td>
<td>12.2</td>
</tr>
<tr>
<td>Paterson</td>
<td>591</td>
<td>30</td>
</tr>
<tr>
<td>Lanman &amp; Mahoney</td>
<td>150</td>
<td>2</td>
</tr>
<tr>
<td>Wallace &amp; Wavill</td>
<td>145</td>
<td>24.8</td>
</tr>
<tr>
<td>Thomson &amp; Gaisford</td>
<td>178</td>
<td>16.4</td>
</tr>
<tr>
<td>Rammstedt</td>
<td>497</td>
<td>22.5</td>
</tr>
</tbody>
</table>

Of the 14 cases operated upon in our series, three died, giving a mortality of 21%. This figure compares favourably with the results obtained by the authorities.
authorities already quoted, although the total number of cases is small.

It has long been recognised that the operative mortality is very high in hospital cases, and strikingly low in the privately treated case. Thus Paterson treated 581 cases in hospital with a mortality of 30%, and treated 24 cases privately without a single death. Barling's figures in this connection are also striking; of the 75 cases he treated in hospital 48% died, whereas all of the 15 cases treated privately recovered. This difference in the mortality rate among hospital and private cases is remarked upon by Herzfeld and Wallace in their paper on "Prognosis in Congenital Stenosis of the Pylorus", and we quote the following extract from their paper explaining why this difference exists, and stressing the factors which make the prognosis more gloomy in the hospital case:

How does the hospital case differ from the private case? At birth there is probably no difference at all, both infants apparently being strong and healthy, and not infrequently above the average birth weight. It is after the characteristic symptoms have commenced that the difference manifests itself. The mother of the infant which is ultimately brought to hospital is frequently harassed and preoccupied, and the fact that her baby is vomiting occasionally may not at first give rise to any serious concern. Eventually, perhaps on the prompting of a neighbour, she will decide that her breast /
breast milk is not agreeing with the child, and various feeds are tried in quick succession in a vain attempt to find something "which suits baby's stomach". The medical profession is not altogether blameless in this respect, since it is not always the interfering but well-meaning neighbour who advises the mother to wean her infant. Finally, after several weeks have elapsed, during which time the infant has been going rapidly downhill, he is brought to hospital in a state of extreme inanition, and operation is fraught with serious risks. It is this long delay which is so disastrous in a high percentage of hospital cases, since it means not only that at operation the child's resistance is at its lowest ebb, after weeks of starvation, but also that thereafter he is irrevocably deprived of his mother's milk which has long since dried up. It is little wonder that the mortality rate in such cases is high.

The well-to-do mother has more leisure and can usually observe her infant more closely, and, perhaps, though not always, more intelligently. Further, she is apt to be far more exacting in her demands on her medical attendant; he is likely to be summoned at the first sign of trouble, and she does not hesitate to demand a second opinion regarding her infant if she is not completely satisfied. This attitude reacts favourably on the infant, since it means that the pyloric obstruction will probably have been diagnosed and treated /
treated at an earlier stage than has that of the hospital infant, the child will not be so starved and dehydrated when it comes to operation, and, furthermore, the mother's milk will still be available at the critical post-operative period - a point of extreme importance. We believe that the mother is the best custodian of her infant in sickness or in health, and the very fact of removing an infant from its mother and placing it in the strange, somewhat impersonal atmosphere of a hospital, may do much more harm than is generally realised.

Here, then, are factors which may serve to guide us in giving an opinion regarding the immediate prognosis in any particular case. We must ascertain the duration and severity of symptoms, we must note the infant's general condition, and we must determine whether breast-feeding is going to be possible after operation. If these crucial questions have to be answered unfavourably, prognosis must be very guarded, since the mortality-rate may be 20 per cent. or even higher. If, on the other hand, the answers are favourable, we are justified in giving a favourable prognosis since, in our experience, the mortality-rate in this type of case is extremely low. In the experience of one of us (G.H.) over a number of years, the mortality-rate in hospital cases of pyloric stenosis is rather more than 20 per cent., whereas no private case has been lost, and we suggest that this remarkable difference may be explained to a large extent by the factors outlined above.
The conclusions drawn from this paper are borne out by Darling, who analysed ninety cases we had treated with a view to finding out what factors influenced prognosis. Fifteen of his cases were privately treated without a death; the average time of operation after the onset of symptoms was thirteen days. Seventy five of his cases were treated in hospital with a mortality of 48%. Those cases operated upon up to eighteen days after the onset of symptoms survived, those cases operated upon twenty five days or later, after the onset of symptoms, all died. He concludes from this analysis that if all cases were operated on within fourteen days of the onset of symptoms the mortality rate would be less than 5%.

**Summary:**

The prognosis with regard to operation depends upon the duration and severity of the symptoms, the child's general condition, and the mode of feeding, when it is first seen. Should the symptoms be of short duration and the infant be breast-fed a very much better prognosis can be given than would otherwise be the case. An assurance can always be given that, should the child survive the operative risk and the complications of convalescence, a permanent recovery will ensue.
**LIST OF CASES.**

**Operation 14**

**Died before operation 3.**

**Total 17.**

<table>
<thead>
<tr>
<th>Date</th>
<th>Initials</th>
<th>Sex</th>
<th>Place in Family</th>
<th>Birth Wt. lbs. Oz.</th>
<th>Age at onset of symptoms in weeks</th>
<th>Age on admission in weeks</th>
<th>Projectile vomiting</th>
<th>Peristalsis</th>
<th>Palpable Tumour</th>
<th>Constipation</th>
<th>Age at Op. in weeks</th>
<th>Wt. at Operation lbs. oz.</th>
<th>Result</th>
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<tbody>
<tr>
<td>30: x:34</td>
<td>S.H.</td>
<td>F.</td>
<td>1st child</td>
<td>7 8</td>
<td>2</td>
<td>6</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>6</td>
<td>6 8</td>
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<tr>
<td>8: XI:34</td>
<td>P.J.B.</td>
<td>M.</td>
<td>1st child</td>
<td>8 1</td>
<td>3</td>
<td>8</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>7</td>
<td>7 0</td>
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<td>28: XI:34</td>
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<td>M.</td>
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<td>10 10</td>
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<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>2: X:35</td>
<td>W.L.</td>
<td>M.</td>
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<td>4</td>
<td>5</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>7: X:35</td>
<td>G.H.</td>
<td>M.</td>
<td>1st child</td>
<td>7 12</td>
<td>4</td>
<td>5</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>7 0</td>
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<tr>
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<td>3</td>
<td>+</td>
<td>+</td>
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<td>+</td>
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<td>3</td>
<td>6 3</td>
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<tr>
<td>3: XI:35</td>
<td>N.McK.</td>
<td>M.</td>
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<td>1</td>
<td>6</td>
<td>+</td>
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<td>+</td>
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<td>7 4</td>
<td>3</td>
<td>5</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>7</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>6 14</td>
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<td>M.</td>
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<td>4</td>
<td>+</td>
<td>+</td>
<td>+</td>
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<td>6</td>
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<td>5 8</td>
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<td>7</td>
<td>+</td>
<td>+</td>
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<tr>
<td>3, III:36</td>
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<td>M.</td>
<td>1st child</td>
<td>6 8</td>
<td>2</td>
<td>3</td>
<td>+</td>
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<td>13: V:36</td>
<td>J.B.</td>
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<td>1st child</td>
<td>6 8</td>
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<td>8</td>
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<td>+</td>
<td>+</td>
<td>-</td>
<td>+</td>
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</tr>
</tbody>
</table>
Conclusion :-

Our study of Congenital Hypertrophic Pyloric Stenosis has shown it to be a condition associated with pathological changes of a constant and well defined nature. A marked hypertrophy of the pylorus, caused by local gigantism of the circular muscle fibres constituting the wall of the pyloric canal, is the most striking feature of the pathology. We believe that this circular muscle hypertrophy affects only the pyloric antrum and canal, and does not occur in the pyloric ring sphincter. Such hypertrophy of the circular muscle layer, combined with the protrusion of the mucosa into the lumen of the pyloric canal, constitutes a relative obstruction. Our observations have, however, convinced us that an element of spasm is essential, if complete obstruction of the pyloric canal is to occur. The pathological appearances and clinical features of this condition have led us to support the view held by John Thomson in regard to the pathogenesis of Congenital Hyperthrophic Pyloric Stenosis; no one has yet been able to explain why there is a want of co-ordination between the sympathetic and parasympathetic fibres supplying this area, but no doubt the explanation will emerge as our knowledge of the function and control of the sympathetic ganglia increases. There is an impression amongst doctors that Congenital Hypertrophic Pyloric Stenosis is a rare condition, and that the clinical diagnosis requires the skill, perception, and acumen of a paediatric specialist. It is true /
true that careful observation and repeated examination may be necessary before the four cardinal signs and symptoms are ascertained, but when present their observation and significance should at once suggest the diagnosis to the family doctor. Elaborate clinical methods of investigation are neither necessary nor desirable in order to make an accurate diagnosis; the occurrence of forceful vomiting in a first born, male infant, under three months of age, is highly suggestive of the condition and if, in addition, visible gastric peristalsis can be seen and constipation exists the diagnosis is almost certain. The palpation of a pyloric tumour is, in our experience, the least commonly demonstrable sign; we believe that failure to elicit this sign is seldom due to lack of perception on the part of the examiner, but rather to the anatomical position of the tumour beneath the lower border of the liver. Consideration of the subsidiary symptoms and signs, while not essential for clinical diagnosis, will undoubtedly help to make the diagnosis more certain in the difficult case. Recent observations on the existence and manifestations of alkalosis in this condition constitute an important advance in our knowledge, by facilitating diagnosis, and awakening a new consciousness of the risk of surgery without adequate preoperative preparation.

The differential diagnosis of Hypertrophic Stenosis from Pyloric Spasm and Duodenal Obstruction should not present any great difficulty if the different sex incidence,
incidence, earlier onset, and tendency to hypertonicity in the former, and the presence of bile in the vomitus in the latter are borne in mind by the physician.

Turning now to the question of treatment in Congenital Hypertrophic Pyloric Stenosis, we are of the opinion that the results achieved do not justify the adoption of medical measures alone in the treatment of this condition. The optimum chance of recovery for the infant lies in the closest co-operation between the physician and the surgeon; the initial stages of treatment should be medical, so that while the general condition of the patient is being improved coincident observations can be made which will ensure the speedy formation of a definite diagnosis in the physician's mind. The determination of the psychological moment at which to operate is a matter which the physician must decide for himself, aided by his past experience of these cases; early operation, as soon as a definite diagnosis has been made and the infant's condition improved, undoubtedly holds the best promise of a successful cure. We would strongly urge the desirability of with continuation/breast feeding in these cases if at all possible; early operation to ensure the continuance of breast feeding during convalescence is a procedure always attended with the best results. From our limited experience we have formed the opinion that Bumydrin is an antispasmodic drug well worthy of a trial, in the event of careful feeding combined with gastric lavage proving unsuccessful in diminishing the frequency of projectile vomiting.
The classical Fredet-Rammstedt operation has now become the standard method of surgical treatment in this condition, and has proved to be the simplest and most efficacious procedure yet devised. The ultimate success of this operation depends in large measure upon careful pre-operative preparation, which aims at improving the general condition and resistance of the patient, while also rendering the operation simple in execution and minimising the serious risk of post-operative complications. The adequate administration of fluid before operation is of considerable importance, not only does this procedure tend to improve the general condition of the infant and counteract operative shock, but it also allays the very real danger of alkalosis before operation which has recently been stressed by Maizels and Morris. We believe that blood transfusion holds pride of place in the pre-operative measures adopted to compensate for the anaemic, emaciated, dehydrated state of many of these infants before operation; the routine adoption of such treatment would, we believe, very substantially reduce the operative mortality by putting the infant in the best position to withstand the shock of operation. Preliminary gastric lavage and the administration of a mild hypnotic greatly facilitate the safe and speedy execution of the operation, by rendering the stomach easy to handle, and reducing movement to the minimum. In the presence of these favourable conditions the operative technique .
technique is simple in execution and the hazards few, if the operator is fully alive to the danger of perforating the duodenum or closing the abdomen before satisfactory haemostasis has been secured in the operative field. Once completed the success or failure of the operation depends in large measure upon the subsequent nursing of the infant. Small frequent feeds preferably of breast milk, the supplementary administration of fluid, and the maximum of sleep in warm surroundings will go far to ensure a speedy and successful recovery.

Post-operative complications should be of infrequent occurrence in the hands of an experienced surgeon, who has made a careful inspection of the pylorus before returning it to the abdomen, and has repaired the abdominal wall to the best of his ability when closing the wound. A high standard of nursing and the short time spent in hospital probably explain the fact that we have not experienced the dread results of enteritis after operation; this risk can, we believe, be still further reduced by nursing these cases in an isolation ward.

Despite the very satisfactory results of surgical treatment in Congenital Hypertrophic Pyloric Stenosis the prognosis should always be guarded. At the present time the lives of many infants suffering from this condition are needlessly lost owing to long delay in diagnosis, foolhardy experiments with medical treatment, and a lack of appreciation of the correct time at
at which to operate. The very low mortality amongst private cases emphasises the fact that early diagnosis and prompt treatment affects the mortality very favourably, and for this reason we hope that in the future every family doctor will be alive to the possibility of Congenital Hypertrophic Pyloric Stenosis, whenever he is called to see an infant with persistent vomiting, visible gastric peristalsis, and progressive constipation, and that he will act immediately to safeguard the best interests of his patient.

In conclusion we wish to express our appreciation and acknowledge the kindness of Mr W. J. Lytle, F.R.C.S. and Miss G. Herzfeld, F.R.C.S.E., in allowing us to publish these cases treated in the wards under their care, and to Dr R.L. Saunders, Department of Anatomy, University of Edinburgh, for his help in the preparation and microphotography of the pathological material used in this paper.
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