CONGENITAL INFANTILE HYPERTROPHIC PYLORIC

STENOSIS IN THE AFRICAN INFANTS

AS SEEN IN

KENYATTA NATIONAL HOSPITAL (K.N.H.)

JANUARY 1973 TO DECEMBER, 1981.

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CANDIDATE

THIS THESIS IS MY ORIGINAL WORK AND HAS NOT BEEN PRESENTED IN ANY OTHER UNIVERSITY.

SUPERVISOR

THIS THESIS HAS BEEN SUBMITTED FOR EXAMINATION WITH MY APPROVAL AS A UNIVERSITY SUPERVISOR.

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INTRODUCTION

It was generally thought that the incidence of congenital hypertrophic pyloric stenosis in African is low (Bowesmann 1960 (1), Bwibo 1970 (2), Jain 1977 (3), Scragg 1958(4), Swan, T.T. 1961 (5). Those with a long and wide African experience feel that this is not true, Bowesmann (1), Bwibo 1970 (2), Miller 1964 (6). Miller (1964) thinks that the most likely explanation is that the cases are underreported and others are undiagnosed. In the developed countries, it is one of the commonest condition requiring laparotomy in the infants, Dennison . 1974 (7), John 1978 (8), Nelson 1975 (9).

The occurrence in the Africans is variable and scattered over the whole of the continent. In Southern Africa, 2 cases were reported in the Republic of South Africa, Grightnths 1956(10). In Zimbabwe, three other cases were reported by Shephard (10) in 1954. Forbes (11) reported a further three cases in the same country in 1964. In West Africa, two cases were reported by Petcher in 1953 (12). Bowesman writing in 1960 reported seeing six cases in a period of 20 years in the same area. Swan in 1961 collected ten Nigerian infants with pyloric stenosis in four years in, four hospitals. Increasing numbers are seen in Ibadan University Hospital, ten in 1957-59 and twenty one during 1960-62, Davey 1968 (13). The earliest reported case of congenital pyloric stenosis in Eastern
Africa is probably the case referred to by Miller, 1955(14) in Kisumu. In Uganda cases were reported by Boroda, C., 1959 (15), Luder, L., 1955 (16), Hamilton, 1956 (17) and Bwibo recently reported nineteen cases seen at Mulago Hospital in 1970. Menezes 1957 (18) reported a cases in Zanzibar. Jain 1977 recently described ten infants in Ethiopia and Somalia.

In the above series due to small numbers encountered there is no mention of familial incidence though these have been recognised to be familial in occurrence in larger series, Benson (19) in western series. In this African set up the only factor mentioned in relation to onset of symptoms is the sudden onset of symptoms in Ethiopian children following uvulectomy and tonsillectomy by "witch doctors".

The factors that have reduced the operative mortality are:- C. Maingot (20).

1. The recognition that early operation is the treatment of choice. The longer the duration of symptoms and the lower the weight at the time of operation, the higher the mortality. This is a plea for early diagnosis and early operation.

2. The adoption of preoperative treatment of
   (a) Careful preparation of the patient for a period of at least two days before operation. These should not be regarded as emergencies or be operated upon without due preparation.
(b) Bodyfluid loss and chemical deficiency are restored by giving intravenous fluids containing potassium and frequent small nourishing feeds of 5% glucose in water.

(c) In every cases a small nasogastric tube is passed through the nostril into the stomach and the stomach is aspirated and irrigated with normal saline. This should be done routinely till one hour before theatre. The catheter is left in situ during the operation.

(d) Precautions are taken to prevent loss of body heat during the operation.

(e) Premedication. Atropine is given 30 minutes before theatre.

(f) General Anaesthesia is the method of choice although in a few desperately ill infants, local anaesthesia by infiltration method using 0.5% solution of procaine may be employed.

3. Universal acceptance of the Ramstedt operation as the technical procedure of choice.

4. A well-planned scheme of post-operative treatment is essential. In Britain most of the cases are handed over to the physician paediatrician who supervises both the preoperative and post-operative treatment.

These simple measures have seen the mortality rate fall from 75% to less than 1% in the developed countries. Lynn (21), Dennison and also in India, Bhasin (22).
This dissertation presents the clinical features of 95 cases of proven pyloric stenosis as seen at K.N.H. in a period covering 9 years extending from January 1973 to December 1981 inclusive.

KNH is situated in the centre of Nairobi City. It is the main referral centre for all the medical and surgical problems from the whole of the Republic of Kenya. It also handles approximately a yearly figure of 100,000 paediatric cases suffering from a multitude of maladies from hospital suburbs. It is also the main centre of medical training for all cadres of medical profession.
MATERIALS AND METHODS

A combined retrospective and prospective study of all the case records with the clinical diagnosis of congenital pyloric stenosis was done. The prospective study was composed of patients seen in the year 1981. For these the case records were studied after the paediatrician or the surgeon had seen and examined the patients so that no personal bias could be introduced. The author also took the opportunity to study the cases by taking history and examining the patients for his own interest. The case records of all the patients were scrutinised with particular reference to:-

(a) Clinical presentation,
(b) The findings on physical examination.
(c) Radiological findings, if any,
(d) The operative findings.

Only those cases with operative diagnosis of congenital pyloric stenosis were included in the final analysis. Some of the patients were admitted, investigated and operated from the general paediatric wards. This pattern is changing and now most of the cases are admitted, investigated and operated in paediatric surgical wards.

A total of 73 cases had operative diagnosis prior to 1981. A further 22 cases were identified during the calendar year ending in December 1981. The clinical data analysis is presented below.
RESULTS

A total of 95 cases were properly documented and had operative proof of the diagnosis. A further 18 cases had been diagnosed as cases of pyloric stenosis. Among these, there was neither radiological evidence nor operative proof of the diagnosis and these were excluded from the study. Another patient was also excluded from the study as the operative details were not consistent with the diagnosis.

Out of the 95 cases seen eleven (10.5%) were referrals from the Provincial Hospitals. These came from Central Province 4, Rift Valley 2, Western Province 3 and Eastern Province 2. It is worth of note that none of the patients had been referred from the Coast Province as there is always a surgeon available.

All the cases reported were Africans and out of all the cases presented, one was from Tanzania mainland.
FIG 1

YEARLY INCIDENCE

1973  74  75  76 ~ 77  78  79  80  81

12
10
8
6
4
2
TRIBAL DISTRIBUTION

The tribal distribution is as shown below. There is as expected in this type of analysis a predominance of the Kikuyu. This does not necessarily mean that they are more susceptible to the condition than other tribes. There are two reasons to explain this. One, they live on the suburbs of KNH in Nairobi and secondly they form the largest tribal division in the population in the Republic followed by Luo and Luhya. On the other hand small tribes are also represented meaning that no tribe is exempted.

TABLE I: TRIBAL DISTRIBUTION

<table>
<thead>
<tr>
<th>TRIBE</th>
<th>NOS.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kikuyu</td>
<td>32</td>
</tr>
<tr>
<td>Luo</td>
<td>12</td>
</tr>
<tr>
<td>Luhya</td>
<td>9</td>
</tr>
<tr>
<td>Meru</td>
<td>6</td>
</tr>
<tr>
<td>Embu</td>
<td>6</td>
</tr>
<tr>
<td>Masai</td>
<td>4</td>
</tr>
<tr>
<td>Kisii</td>
<td>4</td>
</tr>
<tr>
<td>Nandi</td>
<td>4</td>
</tr>
<tr>
<td>Kuria</td>
<td>2</td>
</tr>
<tr>
<td>Kipsigsi</td>
<td>1</td>
</tr>
<tr>
<td>Nyamwezi</td>
<td>1</td>
</tr>
<tr>
<td>Others not indicated</td>
<td>4</td>
</tr>
<tr>
<td>TOTAL</td>
<td>95</td>
</tr>
</tbody>
</table>
AGE

It is known that these cases can present within the first few days of life. Premature infants have been known to present with this condition and have been successively operated on. In this presentation, no prematures were reported. In a recent post mortem examination carried out on all the still births in Nairobi, none was found to have the condition, Njoroge et al (22). The youngest was one and half weeks old. The mean age was found to be 8 weeks. (Fig. II). The majority of the patients were however between 2 weeks and 12 weeks of age (92%). The oldest child in this analysis was 3 years old. This child had been operated upon at Nakuru, the Rift Valley Provincial Hospital when she was an infant. She was apparently cured after the operation. After a period of about 2½ years, at the age of 3 years, she again presented with severe episodes of projectile vomiting. She was subjected to contrast studies at K.N.H., after referral, which confirmed a diagnosis of pyloric stenosis. At operation, the diagnosis was confirmed and a normal Fredet Ramstedt operation was performed. She has not presented again with similar problem. This perhaps may be the first case of recurrence in the African child after most likely an incomplete operation. It should however be borne in mind that recurrence forms a rare but recognised complication of incomplete or inadequate operation. The time interval in this patient between the first and second operation is rather too long.
FIG II

AGE AT PRESENTATION IN WEEKS
SEX

This has been stated to be a predominantly male afflicting condition. It has been found to be true even in this analysis. 75 patients out of 93 patients were males and the rest were females. The other two patients had male gender recorded for sex but were in fact bearing female Kikuyu names (author is Kikuyu). This is a male female ratio of 4:1 which is the same as in the Western countries which gives a ratio of 4:1 (John).

<table>
<thead>
<tr>
<th>BIRTH ORDER</th>
<th>MALE</th>
<th>FEMALE</th>
<th>TOTAL</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st</td>
<td>32</td>
<td>3</td>
<td>35</td>
<td>37.6</td>
</tr>
<tr>
<td>2nd</td>
<td>12</td>
<td>3</td>
<td>15</td>
<td>17.2</td>
</tr>
<tr>
<td>3rd</td>
<td>10</td>
<td>8</td>
<td>18</td>
<td>19.35</td>
</tr>
<tr>
<td>4th</td>
<td>14</td>
<td>0</td>
<td>14</td>
<td>15.1</td>
</tr>
<tr>
<td>5th</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>3.2</td>
</tr>
<tr>
<td>6th</td>
<td>3</td>
<td>2</td>
<td>5</td>
<td>5.36</td>
</tr>
<tr>
<td>7th</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1.07</td>
</tr>
<tr>
<td>TOTAL</td>
<td>~75</td>
<td>18</td>
<td>93</td>
<td></td>
</tr>
</tbody>
</table>
BIRTH ORDER

In this naner, the first born patients comprised 37.6% of the total. The 2nd born were 17.2% of the total and the 3rd born accounted for 19.35%. This fairly agrees with the other African series but the results are different from those of developed countries where the 1st born accounts for 30% to 60% of the cases. There were also cases in the 4th, 5th and in the 6th position in the family a reflection of bigger African families (Figure III).
FIG III

The graph shows the distribution of birth order among patients. The height of each bar represents the number of patients in each birth order category. The legend indicates that the bars are divided into two categories: total and female. The x-axis represents the birth order: 1st, 2nd, 3rd, 4th, 5th, 6th, and 7th. The y-axis represents the number of patients, ranging from 2 to 36.

- The 1st birth order has the highest number of patients, with a peak at 36.
- The 2nd birth order has a significant number, with a peak at 18.
- The 3rd birth order follows, with a peak at 16.
- The 4th and 5th birth orders have lower numbers, with peaks at 14 and 12, respectively.
- The 6th and 7th birth orders have the lowest numbers, with peaks at 2 and 4, respectively.

The data suggests a trend towards a higher number of patients in the earlier birth orders, with a sharp decrease in the later birth orders.
DURATION OF ILLNESS

This is a point which should be differentiated from the age of the infant when first seen. This is the time during which the infant is having the relevant or suggestive symptoms but the right diagnosis has not yet been made. It is the time which affects the severity of the disease at the time of diagnosis. If it is too long most of them would be dehydrated to variable degrees and will also be suffering from malnutrition. The shortest duration elicited from the patients was 3 days and the longest was that incredible 145 days. The mean duration of illness was 3 weeks.

In some of these cases the mother or the parent reported that the infant had been presented to various health workers earlier but were told that patients were suffering from other kinds of diseases and different treatment offered. As the infants failed to improve, the parents on their own accord, decided to seek for better medical opinion elsewhere and most of them came to K.N.H. Six patients were thus seen at K.N.H. through their own initiative. In this way three patients were apparently symptomatic since birth from the history. It is at this point that the practice of giving symptomatic treatment without proper clinical diagnosis was noted to be particularly common and rather hazardous.

FAMILIAL INCIDENCE

The condition tends to be familial and this was examplified by two cases. One of our patients had his elder brother operated when he was young at this hospital for pyloric stenosis. The father in this particular case was very
reluctant to give consent for operation and this child was lost after the operation. The other case is that of a twin where the other twin died from projectile vomiting and soon thereafter this second twin developed the symptoms which made the parents rush the infant to the hospital.

**SEASONAL INCIDENCE**

This was not possible to work out as most of the case reports did not indicate the dates. In those where the dates were indicated it was found that there are two peaks in the year. This is in July to September and another one from December to February. These are the seasons just or during the long or short rains in the country.

**FIGURE IV**

18
16
14 .
12
10):
8 x
6
4

Jan | Feb | Mar | Apr | May | Jun | July | Aug | Sept | Oct | Nov | Dec | MONTH
BREAST FEEDING OR BOTTLE FEEDING

Formula fed babies have been found to be slightly-predisposed to developing pyloric stenosis and especially those who are on four hourly feeding schedules. Attempts at finding out the difference between those who are breast fed had any lower incidence as compared to those who were on formula feeds was not possible as it became apparent that at one time or another all the infants had been bottle fed as well as breast fed.

SYMPTOMS

Projectile vomiting

The cardinal symptom in this condition is projectile vomiting. This symptom often serves to differentiate this condition from the other common causes of vomiting in the infant as these are not so persistent. It is therefore always necessary to even directly question the parent whether or not the child has had projectile vomiting during the course of illness. This symptom may not be there in the initial stages when the muscular mass of the stomach and pylorus has not hypertrophied. It may also be absent in the child who has been weakened by the illness who will have effortless vomiting.

The vomitus is strictly nonbilious as any vomitus with bile means that the obstruction is distal to the Ampulla of Vater and therefore not at the pylorus. Where this is present the diagnosis should be reconsidered.
In this analysis, definite history of projectile vomiting was elicited in 94% of the cases at one time during the history of the illness and was persistent in 87% of the cases.

As a consequence of the vomiting, the infants become dehydrated, oliguric and are often constipated as no food or water goes down the gastrointestinal tract. The stool becomes infrequent and hard as the illness progresses. If diarrhoea supervenes, then it means that there is infection and the child has gastroenteritis superimposed on the pyloric stenosis. Diarrhoea was noted in 4 patients and constipation was complained of by 20% of all the cases seen.

**DEHYDRATION**

Persistent vomiting with little or no retention of any feeds leads to dehydration when these cases presented, most of them were dehydrated to some degree or other. In the analysis, 95.7% of the patients were dehydrated on clinical examination. Dehydration was classified on either:-

(a) No dehydration.
(b) Mild dehydration.
(c) Moderate and
(d) severe dehydration depending on the skin turgor, dryness of the mucous membranes and sunken eyes or where palpable, sunken fontanelles.

4.3% of the cases seen were not dehydrated at the time of admission. These had come from other hospitals within the vicinity of the hospital where primary hydration or medical treatment had been given before transfer to (see table III).
There are important facts which come out of this analysis. It was found out that the degree of dehydration had a direct relationship with the final outcome of the case. For those who were not dehydrated on admission, no deaths were reported. For those who were severely dehydrated the mortality in the group was 83.3%*. It should however be noted that the severely dehydrated patients were also having severe acid base imbalances.

**TABLE III**

<table>
<thead>
<tr>
<th>DEGREE OF DEHYDRATION</th>
<th>NUMBER OF CASES</th>
<th>DEATHS</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not dehydrated</td>
<td>4</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mild dehydration</td>
<td>51</td>
<td>5</td>
<td>9.8</td>
</tr>
<tr>
<td>Moderate dehydration</td>
<td>33</td>
<td>1</td>
<td>3.3</td>
</tr>
<tr>
<td>Severe dehydration</td>
<td>6</td>
<td>5</td>
<td>83.3</td>
</tr>
</tbody>
</table>

The other fact which was found was the relationship between dehydration and the duration of hospital stay. As the degree of dehydration worsened so did the length of hospital stay increase. The "most notable thing here is that for those who were not dehydrated, the average hospital stay was 9 days and those who were severely dehydrated stayed longer than two weeks. In comparison, the average hospital stay by other
Daediatric surgical cases is 21.2 days while the overall stay by cases of pyloric stenosis was found to be 40.6 days (see table IV).

**TABLE IV: DEGREE OF DEHYDRATION AND HOSPITAL STAY**

<table>
<thead>
<tr>
<th>DEGREE OF DEHYDRATION</th>
<th>AVERAGE STAY IN THE HOSPITAL EN DAYS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not dehydrated</td>
<td>9</td>
</tr>
<tr>
<td>Mild dehydration</td>
<td>13.1</td>
</tr>
<tr>
<td>Moderate dehydration</td>
<td>14.2</td>
</tr>
<tr>
<td>Severe dehydration</td>
<td>16</td>
</tr>
</tbody>
</table>

**WASTING**

The patients were also noted to be wasted clinically. It was not always possible to obtain the weights on admission and therefore the average weights below or above the general population could not be compared. Most parents did not know or could not remember the birth weights and this data was not put to any useful analysis. However on clinical examination alone, our patients were on the whole wasted. This varied from the "normal weight for age to severely marasmic cases so that the typical picture of a wizen active and hungry infant was presented. In 30% of the cases seen severe malnutrition was present and 5% were marasmic.
PALPABLE ABDOMINAL TUMOURS

The presence of palpable tumour in the upper abdominal half was present or recorded in 71 cases (73.1%) including those palpated after test feeding. It was not recorded in 5(6.4%) and was absent in 19 (20%). These later were confirmed by radiological examination as this is noted to have been done in all the 19 cases.

VISIBLE PERISTALTSIS

Whenever there is an obstruction in the gastro-intestinal tract, the proximal gut tries to overcome the obstruction by waves of strong contractions. This also forms a prominent sign in these cases and in most of these cases visible peristalsis is always present. The waves move from left to right in the upper half of the abdomen. Sometimes it is possible to stimulate a contraction by stroking the upper abdomen. Visible peristalsis was noted or elicited in 80% of the cases. In the rest of the cases no peristalsis was present. The parents sometimes brought the patient to the hospital because of the "moving snake" seen per abdomen.

LABORATORY INVESTIGATIONS

The standard investigations done were, complete haemogram in 20 cases. The results ranged from 6.5gm/100 ml. to 16 gm/100 ml. The low haemoglobin of 6.5 gm./100 ml. was noted in a patient with bleeding tendency. Two of the other haemograms were normocytic normochromic anaemia. It was necessary to transfuse 7 cases before operation as the haemogram results were below 10 gm/100 ml. Transfusion was however, not used as a means per se of resuscitation before theatre. The commonest type of anaemia was hypochromic microcytic anaemia from hookworm infestation as found in 3 of the patients.
Urea and electrolytes were estimated in 30 cases. The commonest finding was some degree of metabolic alkalosis associated with hypokalaemia. Definite hypochloremic metabolic alkalosis was present in the severely dehydrated cases.

Other features not commonly found:- These were symptoms and signs which were found on patients due to other conditions.
1. Three patients had otitis media.
2. Six patients had bronchopneumonia.
3. Five patients had oral thrush.
4. Two patients had malaria.

Of special interest were two cases. One of the patients had obstructive jaundice from the age of two weeks. Investigations could not reveal the cause of the jaundice. After operation the jaundice cleared rapidly. In another patient hematemesis and maleana complicated the picture. This was the patient who had a-haemogram of 6.5 gm/100 ml. This patient required several transfusions. At the operation the cause of the bleeding was not found. A normal Ramtedt’s operation was done. The post operative period was also plagued by persistent maleana which finally cleared on its own. Haematemesis in infants is associated with gastritis on the antrum (John).

DIAGNOSIS

The diagnosis was in most cases made on clinical grounds alone. This is based on a triad of projectile vomiting, palpable abdominal mass and visible peristalsis. This combination was present in 62% of the cases. In the
others projectile vomiting and palpable mass in the right upper quadrant was taken as confirmatory of the diagnosis and this was present in 90° of all the cases seen. When there was doubt to the diagnosis, the patients were subjected to either test feeds or radiological diagnosis. Resumption of projectile vomiting and visible peristalsis on test feeding was recorded in a total of 6 patients and no more investigations were done. Where the diagnosis was still in doubt; but index of suspicion was very high, barium meal studies were carried out. In all 38 contrast studies were done. Out of these two studies were suggestive of pyloric stenosis but at operation no evidence of pyloric stenosis was found. In another case the radiological findings were negative but at operation a pyloric stenosis was found. Thus out of 38 contrast studies done, 3 of them were misleading as to the diagnosis. This gives an accuracy rate of approximately 80%, This strengthens the importance of clinical diagnosis.

On the other hand, the contrast studies helped to identify one infant who had a hiatus hernia and another with cardio-oesophageal reflux. Both of these, in addition to pyroloomyotomy, had fundoplication carried out.

TREATMENT

(a) Pre-operative management

This invariably consisted of correction of fluid and electrolyte imbalances as was found at the time of admission. Majority only required rehydration (821). In two patients, this preliminary rehydration was not found to be necessary as it had been done elsewhere.
7 cases were also transfused before being subjected to operation to raise their haemogram which had been found to be below 10 gm./100 ml.

In general Half Strength Darrow's solution was given in all the patients because of its Potassium content. Where this was not available normal saline in Dextrose was used or plain 5% Dextrose in some cases.

**Definitive treatment**

The definitive treatment in all the patients presented in the analysis has been Standard Fredet-Ramstedt-operation. This was performed by the various attending surgeons available in the hospital. There is no case in record where local anaesthesia was used as general anaesthesia was available to all the patients. Consent was refused in one patient only. Most likely this patient died.

The only variation to the operation was the skin incision. Incisions used were the standard right para-median incision, the McBurney type subcostal incision with muscle splitting, and the transverse incision with rectus transection. Otherwise the operative details were the same. Twelve cases had mucosal perforation at operation but this was recognised and sewn up by stitching the perforated mucosa by means of 2.0 atraumatic catgut sutures* There is no report of fatalities from peritonitis as a result of perforated mucosal layer.

Those who were offered medical treatment by definition have not been included in the study. The medical treatment consisted of drops of amyl nitrate before
feeds or atropine where amyl nitrate was not available. Post-operatively, intravenous fluids were continued till the patient could retain oral fluids. Feeding was started at variable post operative periods depending on the progress of the patients and also depending on the surgeon in charge of the cases- This varied from 4 hours to 12 hours with the majority starting at 6 hours post-operatively.

Only one patient was discharged after two days. The others, if they had no other complications were discharged on the 7th day or the 14th post operative day after the stitches were removed. In two of our patients, burst abdomen was recorded. There is a great tendency for this to develop especially where paramedian incisions are used (Maingot).

**Complications**

The post operative complications noted here as shown in order of their frequency:-

1. Bronchopneumonia (7).
2. Diarrhoea (4).
3. Post operative persistent vomiting (4)
4. Wound dehiscence (2).
5. (Incomplete operation)(1).
6. Bleeding disorder (1).
7. Oral thrush (1).
8. Incisional hernia (1).
(d) **Mortality**

The case mortality in this presentation varied from 0% per year to 28.5% per year (see table II). The overall yearly mortality has been found to be 11.6°*. The causes of death are shown in the same table where this is recorded.

(e) **Summary of presentation**

Congenital pyloric stenosis is fairly common in Kenya.

A total of 95 cases of confirmed congenital pyloric stenosis have been analysed and the clinical features presented.

The condition as it occurs does not seem to differ in its clinical aspects from the disease as it occurs in the developed countries.

The management of the case is standard and does not seem to differ from other countries except emphasis is on operative treatment.

Earlier on in this hospital, medical treatment was being offered to the patients where the diagnosis was suspected. These patients have been excluded from this study. However, medical treatment is in vogue in some countries especially in some parts of America and the Scandinavian countries, Benyl (24), Corner et al(25) Mellin et al (26), Wallergan (27).
DISCUSSION

The incidence of congenital pyloric stenosis has been estimated to be between 1.5 to 5 per a thousand live births Benson 1957 (28), Beryl,1955 (24), Laron, 1957 (29) in the Caucasians in the developed world. In the Negro population of America this has been estimated to be 1 in 2073 live births and this is probably much lower than what it is. The true incidence of the disease has yet to be worked out among the African patients. The condition is prevalent in the continent except that there are no experienced personnel to look for it in some countries. It is fairly common in some places (Jain, Swan). As this paper tried to show or has shown, it is not rare in Kenya (see the yearly incidence diagram). As the medical care improves and reaches the remote corners of Africa, the true incidence will emerge.

The clinical picture of congenital pyloric stenosis is characteristic and the diagnosis is based on a triad of symptoms and signs (Swan). These are projectile non bilious vomiting, palpable pyloric "tumour" and visible peristalsis. The fact that this is a clinical diagnosis can not be over emphasised. Where projectile vomiting or persistent vomiting is present, the clinician is duty bound to rule out pyloric stenosis. Patience in taking a careful history will lead to the diagnosis. As to the finding the pyloric tumour, there are few hints for the uninitiated. The palpable tumour in the pyloric stenosis is the hard hyper-
trophied pylorus. This should not be mistaken for the antrum which is also palpable but the antrum is soft and will disappear if the infant vomits or the stomach is aspirated. The antrum is more to the left of the right rectus muscle and is most prominent just before the infant vomits. The pyloric mass on the other hand is to be palpated at the lateral border of the right rectus muscle just below the liver and the costal margin. It is hard and is not palpated after the stomach is empty. It is the size of the examiners’ termal phalanx of the thumb or as is often described the size of an "olive". It is slightly mobile up and down but not from the left to the right or vice versa. The other sign is the visible peristalsis. This is an important sign and one should also spend time looking for it. The child should be adequately exposed and adequate light provided. When the above three signs are present in an infant of the right age, the diagnosis can hardly be missed. The tumour in most cases is the cornerstone of the diagnosis as has ably been shown by Benson, Benson(28), Lynn (21), Martin (30). These people found its presence in 60$ - 80$ of the patients. This was present in 73.6% of this analysis.

The aetiology and the pathology of the condition is still not very clear. It is not known why for example, a condition which is assumed to be congenital does not often present at birth, but only after a few weeks.
Preclinical radiological examination of 1000 infants in Sweden led to discovery of predictable 5 cases after a period of 5 cases after a period of 5 weeks of following (Wallergan 27). These infants were completely symptom free until they developed projectile vomiting and later hard typical pyloric tumour with typical radiological findings. The pathogenesis is also not clear. It is theorised that the circular muscles of the pylorus become hypertrophied secondary to the spasm of the sphincter itself (Wallergan, Morton (31), Kissame (32). The Myenteric plexus at the pylorus has also been said to be abnormal but the evidence has not been found to be consistent by some investigators (Morton, Kissame). It appears that some people think that environmental factors play a definite role in the pathogenesis of this disease. There is however enough evidence to show that familial factors play a role. There is therefore interplay of both factors. The seasonal incidence has been noted in this series although, I have not come across another reference to this. The only possible explanation is that an environmental factor, either in form of an infective agent or dietary change taken by the mother during the following season of plenty and therefore of greater variety in the dietary items trigger the mechanism which sets up the events leading to hypertrophy of the circular pyloric muscles.
The presence of jaundice has been noted before in association with the pyloric stenosis (Arias (33), Nakai (34), Gryosboski (35). This was noted in one of our patients. The theory is that the presence of the tumour causes pressure on the hepatic artery. This causes back pressure into the hepatocytes and the activity of gluculonyl transferase is interfered with. Hematemesis is also associated and is explained by the presence of gastritis in the antrum (Laron).

In this analysis reference has been made of the familial incidence of pyloric stenosis. This is not a very strong point but there is enough evidence that there is a definite role of genetics here. Where one of the parents is affected, the chances of an offspring developing pyloric stenosis are 6.9% Carter (36). If the infant is a female and she begets children, the chances that one of them will be affected are four times compared to the father if he were affected, Benson, Carter, Spitz (37). When one of the twins is affected, the chances are twenty times more for the other child to be affected compared to the normal (Spitz, Cammeron (38). In the laterature there are two sets of triplets who have been reported. In these reports it appears the chances are 100% & of all
the individuals of the triplets to be affected if one of them is affected Spitz, Leon (39).

As the diagnosis is mainly clinical, the role of radiology needs to be made clear. The x-ray, plain or contrast, are taken where the diagnosis is clearly in doubt and this is an axillary, though, very important investigative tool. In this series 38 patients were subjected to radiological examination. This is by any standard a very high figure, where more experience has been gathered only in 25% of the cases are found to need radiological examination, Benson, Benson, Lynn, Martin. The x-ray when taken is very useful with an accuracy of 99% in good hands. The plain film shows dilated, air filled stomach. The contrast film on the other hand shows a dilated stomach, a narrowed pyloric canal and a region of hypertrophied pylorus and delay in emptying of the stomach. Whatever contrast material is used, it must be aspirated at once after the examination is over. There are definite risk associated with the barium sulphate when this is used, as the child may inhale the contrast and in the process develop aspiration pneumonia or worst get asphixiated. If this has to be used a diluted solution should be used or a water soluble gastrograftin be used instead.
Our patients on the whole generally present late for proper management although they might have presented themselves earlier elsewhere-. There is a group which delay in seeking medical advice and there is a group which prefers itself early but the right diagnosis is not made. In both groups health education is the answer but the target groups are different. The first group - the target group - is the patient’s parents and relatives. To these the message which should be conveyed is that medical treatment and at that surgical treatment is the only cure to save their patient from certain death (Jain and Scragg). The other target - is the health worker who are at the periphery (and also in the bigger centres). The message here is that projectile vomiting especially non bilious with a palpable upper abdominal tumour is very likely a surgical problem which should be referred to a centre where a surgeon is available. A common practice here is, which should be discouraged, is that of giving symptomatic treatment without a proper diagnosis (Swann). It might be too late by the time the proper diagnosis is made.

As a result of late diagnosis, the disease is often presented at an advanced state and as infants come in when they are dehydrated and at times severely so (Bwibo, Jain, Scragg), they also suffer some degree of acid base imbalances especially metabolic alkalosis and hypochloremia (Clark 40) as a result of prolonged vomiting of acid-rich gastric juices. As this has to be corrected, i.e., the dehydration and acid base imbalance
Our patient's stay in the hospital is longer than in the western series. Also the degree of dehydration has a direct bearing on the total length of the hospital stay (table I). This assumes that all the required apparatus and equipment and manpower is available. One should bear in mind that with patience and forebearing one can orally rehydrate these infants after the stomach is initially aspirated of all the milk curds in it (Benson, Benson, Lynn and Martin).

Pyloric stenosis, in an infant or in adult is not an emergency. One has all the time to correct all the present deficiencies before one can take the child to theatre. Proper rehydration and correction of the acid base imbalance is the mainstay of the treatment and it shows that one really understands the mechanism at hand to have all these corrected. If this is not carried out properly, the opportunity may not present itself again and it may be too late for the patients. Diagram V shows the causes of death as recorded in the case files. It is obvious that most of the cases died from acid base imbalances and this could have been avoided if stepwise logical management was followed up right from the beginning. The technical aspect of the operation is not difficult but the management calls for proper understanding of the pathophysiology of disease for proper and safe outcome of the management. This cannot be over emphasised enough.
## TABLE

<table>
<thead>
<tr>
<th>YEAR OF STUDY</th>
<th>NO. OF CASES SEEN</th>
<th>DEATHS</th>
<th>%</th>
<th>POSSIBLE EXPLANATION FOR CAUSE OF DEATH OF EACH CASE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1973</td>
<td>5</td>
<td>1</td>
<td>20</td>
<td>Operated as an emergency. Developed B. Pneumonia. Severely dehydrated.</td>
</tr>
<tr>
<td>1974</td>
<td>10</td>
<td>2</td>
<td>20</td>
<td>Post operative vomiting. Died of electrolyte imbalance Operated as an emergency and developed electrolyte imbalance K+ above 6.2 mg.</td>
</tr>
<tr>
<td>1975</td>
<td>7</td>
<td>2</td>
<td>28.5</td>
<td>Patient was dehydrated before theatre and did not fully recover Developed electrolyte imbalance operated as an emergency.</td>
</tr>
<tr>
<td>1976</td>
<td>13</td>
<td>2</td>
<td>15.3</td>
<td>Initial treated medically. Died while barium study was being done.</td>
</tr>
<tr>
<td>1977</td>
<td>12</td>
<td>1</td>
<td>8.3</td>
<td>Initially treated medically. Died while barium study was being done.</td>
</tr>
<tr>
<td>1978</td>
<td>12</td>
<td>1</td>
<td>8.3</td>
<td>Post operative dehydration. Severe hyperkalaemia. K+ 6.4 mg.</td>
</tr>
<tr>
<td>1979</td>
<td>6</td>
<td>0</td>
<td>-</td>
<td>.</td>
</tr>
<tr>
<td>1980</td>
<td>8</td>
<td>0</td>
<td>-</td>
<td>.</td>
</tr>
<tr>
<td>1981</td>
<td>22</td>
<td>2</td>
<td>9</td>
<td>Severe dehydration and electrolyte imbalance as a result of post operative vomiting. Electrolyte imbalance K+ above 6.8 mg.</td>
</tr>
<tr>
<td>TOTAL</td>
<td>95</td>
<td>11</td>
<td>11.6</td>
<td>.</td>
</tr>
</tbody>
</table>
At some time back, either due to the teaching one obtained, or due to lack of faith on surgery or purely for personal reasons, patients were offered medical treatment in the form of methyl scopolamine nitrate or atropine. This cannot be condemned as some people have a lot of faith in this treatment (Beryl) but it cannot be recommended. There is of course the occasional death in medical treatment. Medical treatment entails intensive nursing attention, it takes a long time, and when this fails, there is often resort to surgery. This is not suitable method in our setup here. It is almost criminal to my mind to offer medical treatment where the diagnosis is definite and even proved by radiology. The role of medical treatment in this country can be in that place where there is no surgeon and no theatre or while awaiting transfer to a better equipped hospital. It is a great pleasure to see that this habit is no longer practised in our hospital.

Our mortality pattern is the saddest chapter in this dissertation and Diagram V is almost self explanatory. The figures are too high compared with the mortality figures of less than 1$ in the developed countries. But this is no worse than the other African series in our present time (Bwibo, Jain, Swan). However two factors stand out clearly, that the severely dehydrated patients end up poorly than the other patients. Of the 6 severely dehydrated patients, 5 of them died. This a 83$ case mortality. The other
important point is that the practise is changing for the better. Since 1977 when the paediatric Surgical Unit was established, or it established itself properly, the mortality figures has shown a dramatic improvement (see table VI)

TABLE VI : THE EFFECT OF PAEDIATRIC SURGICAL UNIT

<table>
<thead>
<tr>
<th>PERIOD OF STUDY</th>
<th>NUMBER OF CASES</th>
<th>DEATHS</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1973 - 1977</td>
<td>37</td>
<td>8</td>
<td>21.6</td>
</tr>
<tr>
<td>1977 - 1981</td>
<td>58</td>
<td>3</td>
<td>5.17</td>
</tr>
</tbody>
</table>

Before 1977 the average mortality per year was 21.61. After 1977 the mortality is now at average of 5.171, a quarter of what it was before. This trend is assumed to be going to continue.

When one reflects on the mortality figures for pyloric / stenosis in the western countries and compares with ours, one may see a solution to our problems. Man power in form of medical worker, availability at the lowest district level should be increased and improved and diversified. The doctor at this level must be able to handle most of these and other paediatric emergencies which occur at this age group.
CONCLUSIONS

The myth that pyloric stenosis is rare should be discarded in this part of the country. Understanding of the basic patho-physiology and the mechanisms involved in pyloric stenosis may help to improve the management of the case. Specialised care seem to be improving what used to be a gloomy picture in terms of mortality.
RECOMMENDATIONS

In order to achieve the health for all by year 2000 a lot of groundwork has to be done.

The population at large must be taught to have faith in the medical profession and to trust them where surgical management of some cases is the only treatment.

The medical profession must also be taught not to treat cases symptomatically without a proper diagnosis. Where one is not sure one should ask for opinion and the earlier this is done the better. Awareness of some diseases should be made basic knowledge to all cadres in the medical profession.

In order to improve the medical care at the rural level medical personnel must be available at that level. It is to be noted that these people must have equipment and facilities at this level to be able to function.
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