ANAEMIA AS SEEN IN CHILDREN ADMITTED TO THE FAEDIATRIC OBSERVATION WARD OF THE KENYATTA NATIONAL HOSPITAL

FLORENCE NANTULYA NAKIMWERO, M.B.CH.B.

A Dissertation submitted in part-fullfilment for the degree of Master of Medicine (Paediatrics) of the University of Nairobi, 1981.



ANAEMIA AS SEEN IN CHILDREN ADMITTED TO THE PAEDIATRIC

OBSERVATION WARD OF THE KENYATA NATIONAL

HOSPITAL

This dissertation is my original work and has not

been presented for a degree to any other univeristy

FLORENCE NANTULYA NAKIMWERO, M.B.CH.B.

This dissertation has been submitted for examination



DR. J.S. MEME, M.B.CH.B., M.Med.

SENIOR LECTURER

DEPARTMENT OF PAEDIATRICS

CO-SUPERVISOR

PROF.E.G. KASILI, M.B.CH.B., M.R.C. PATH., M.D.

ASSOCIATE PROFESSOR IN HAEMATOLOGY

DEPARTMENT OF PATHOLOGY



ACLINOWLEDGEMENTS

I would like to express my sincere appreciation:

To my supervisors, Dr. J.S. Meme and Prof. E.G. Kasili for their tremendous help and guidance throughout the production of this work.

To Mr. Jepha Mudasia of the Haematology laboratory for his special help with processing of the bone marrow aspirates.

To all my colleagues working in the POW at the time who helped in referring the patients to me.

To Ms. Doris Churi of ILRAD for her excellent and patient typing of this work.

To all members of my family and especially my husband, Vinand, without whose help and encouragement it would have been impossible to produce this work.

Lastly, my sincere gratitude goes to World University Service (WUS) who offered me the scholarship which has enabled me to undertake this course.

CONTENTS

PAGE NOS.

List of Figures	A1
List of Tables	A2
Summary	B1
Introduction	1
Materials and Methods	6
Results	10
Discussion	23
Conclusion and Recommendations	41
References	
Appendices	

LIST OF FIGURES

Between Pages

 Age Distribution of the patients.....11 & 12
 Age Distribution of patients from the four major tribes.....11 & 12

LIST OF TABLES

Between Pages

1.	Distribution	of patients	according		
	to tribe	•••••	••••••	10 &	11

111. Pattern of Presentation in Relation

1V. Classification of anaemias seen

V. Features of Patients presenting with

a bleeding problem.....18 & 19

V1. Causes of anaemia in relation to

V11. Distribution of the various types of anaemia amongst the tribes studied 19 & 20

SUMMARY

In this prospective study, the pattern of anaemia seen in children admitted to the paediatric observation ward (POW) of Kenyatta National Hospital was investigated, between November, 1979 and March, 1980. There were 294 (7.3%) cocumented cases of anaemia out of a total of 4,044 admissions to the POW. Of these 75% had come to the hospital primarily with some other disease problem, and clinical presentation varied according to the underlying condition. Luos and Luyias formed 75.5% of all the patients; half of these resided in Nairobi and the rest came from their indigenous areas in Western Kenya. Basically all children belonged to the low social-income group.

Children under 3 years of age were the most affected and formed 62.4% of the series. The main causes of anaemia in the series were haemolysis from malaria and/or sickle cell anaemia (30.2%) and iron deficiency of nutritional origin (26.2%). Both haemolysis and iron deficiency accounted for 12.2% of the cases. Hookworm infestation played a significant role in aggravating iron deficiency, especially in older children. Megaloblastic anaemia was seen infrequently and was also nutritional in origin. Other causes of anaemia included bone marrow failure from various causes, and bleeding tendencies, mainly from primary bleeding diatheses.

B1

Many cases were undiagnosed because of problems involving every sector of attending staff. Management of the patients with severe anaemia was essentially the same, i.e. transfusion and discharge. The problems encountered, and the implications of the present form of patient care of anaemic children in POW are discussed. Recommendations are made regarding areas where improvement can be made to effect better patient care.

INTRODUCTION

Anaemia is defined as a decrease in circulating haemoglobin per unit volume of blood below the level previously established as normal with regard to age, sex and altitude (1). Just like fever, anaemia is a common problem in medical practice which should properly be regarded as a manifestation of some underlying disorder (2). In our paediatric practice at the Kenyatta National Hospital (KNH), like elsewhere, anaemia is a common cause of morbidity and indeed occasionally mortality. Frequently one sees children admitted solely because of anaemia.

The causes of anaemia are many and varied, but can be broadly grouped into the following categories: (i) Failure of red blood cell production, arising from failure of the manufacturing mechanism through affections of the bone marrow. This may be primary, called aplastic anaemia, which may be idiopathic or secondary to some marrow poison. Bone marrow failure may also be secondary to some other underlying disorders, as occurs in chronic liver disease, chronic renal failure, chronic infections or to some metabolic endocrinopathies like hypothyroidism. It may also be secondary to mechanical crowding out of the marrow elements by infiltrations like granulomatous diseases, primary and metastatic bone marrow neoplasms (2,3). (ii) Deficiency of the essential raw materials from which red blood cells are made, namely folic acid, iron, Vitamin B12 and proteins. This group is commonly referred to as nutritional anaemias (4). Those deficiencies may arise from primary inadequate intake or may be secondary to poor absorption, increased loss or needs (5,6).

(iii) Enhanced red blood cell destruction resulting in a shorter than expected erythrocyte survival time. Anaemia ensues when the bone marrow fails to cope with the maintenance of normal red cell mass. This may arise from inherent or acquired cellular deformities of the red cell membrane, snzymes or haemoglobins. It may arise from a non-cellular defect, either immunological or toxin-induced. It may also arise from a hyperactive mopping up system, namely, the reticuloendothelial system and principally the spleen (3,5).

Anaemia is worldwide. The role of any of the many aetiological factors, and the degree of anaemia in various parts of the world is modified by varying environmental factors. For instance, in the tropics, anaemia is a commoner problem than in temperate areas, and multiple aetiological factors interplay to produce a severer disease. The commonest causes in the tropics are deficiency anaemias, but haemolysis due to haemoglobinopathies and malaria feature prominently (7,8,9,10,11,12,13). Even then, the frequency of each cause varies from community to community. It is therefore desirable to define the magnitude and assess the role of different causes of anaemia for each community, is a means of rationalizing the approach to its

clinical management.

Work done in different parts of East Africa, namely, in Zanzibar (14), Uganda (15,16), and Tanzania (17,18,19, 20), has shown anaemia to be very common. Similarly, various workers in Kenya have shown that anaemia is common and its pattern the same as elsewhere, but have noted regional differences in the distribution of various actiological types (7). It was found that iron deficiency anaemia was the principal type encountered at the coastal belt, while in Nairobi and the surrounding highlands, megaloblastic anaemia was the more common (8). Turner (21) however, found that, although iron deficiency anaemia was the commonest at the coast, megaloblastic anaemia did occur at a significantly higher frequency than had been previously noted. Forster (22) in another study went on to show that the incidence of megaloblastic anaemia at the coast had a seasonal variation, an observation that had been made earlier in Nairobi by Foy and Kondi (8). More recently Kasili (23) working at the Western Provincial Hospital, Kakamega, found that haemolysis from sickle cell disease and malaria, followed by iron deficiency anaemia ranked highest in that order in that community. In Nairobi Ferguson et al. (24) working at Kenyatta National Hospital and studying severe anaemia in all age groups found that 46% of his patients had iron deficiency anaemia, 15% had the megaloblastic type while the rest had miscellaneous causes. Nati et al. (25) studying anaemia in pregnancy

at the same venue found that most of the patients had megaloblastic anaemia, while the rest had either iron deficiency or had both types. In all these studies the majority of patients were adults, mostly women in the child-bearing age and anaemia was directly related to pregnancy. Where anaemia in children has been studied on a large scale, malaria, iron and/or folic acid deficiency have been the greatest problems (26,27).

There has been no specific wide-scale study of anaemia in children in this country and therefore the pattern of anaemia in our children can only be speculated. At prosent, in our over-populated practice full investigation is only possible in those children admitted with either very severe anaemia or have some other serious problems as well. Even in these, need for urgent treatment and quick turnover compromises diagnosis. Attempts at followup often fail, and not surprisingly, therefore, the same patients are seen time and again with the same problem but without a definite diagnosis.

It was, therefore, the aim and objective of this work to conduct a large scale study of anaemia in children with reference to

(i) Determining the prevalence of anaemia in children
 admitted to the Paediatric Observation Ward at the Kenyatta
 National Hospital;

(ii) Establishing the pattern, mode of presentation and the role of different causes of anaemia, and thereby

(iii) Assist in arriving at a more rational approach to the clinical management both on an out-patient and in-patient basis at the POW.

(iv) In addition, the availability of such data would make it possible to review changes in pattern of disease due to changing social and economic factors.

.

MATERIALS AND METHODS

Pationts

This study was carried out in the Paediatric Observation Ward (POW), the children's admission ward of the Kenyatta National Hospital. The study was done over a total period of four months, stretching from 1st November to 15th December, 1979, and then from 16th January to 30th March, 1980. The period of study was chosen for logistic purposes.

Of the children admitted to the POW during the study period, the following cases, selected on the basis of the clinical impression of anaemia, namely, pallor, as noted by the admitting registrar, were included in the study.

(i) All those children admitted with anaemia as their main problem.

(ii) All those admitted with any other problem but found to have pallor at the same time and were investigated for it. Children admitted with severe malnutrition without severe pallor were excluded from the study, although they were investigated for anaemia routinely.

For each patient, the following information was obtained and recorded on the proforma (Appendix 1).

(a) Particulars of name, age, sex, tribe, residence, date of admission and discharge, and whether the patient was referred and source of referral.

(b) A physical examination was done, noting the following:

general condition of patient, degree of pallor of the tongue, conjunctivae and nail beds; temperature, nutritional status, presence of lymphadenopathy, jaundice, oedema, or evidence of bleeding tendency, and whether the spleen, liver or any other intra abdominal masses were palpable. The respiratory and cardiovascular systems were examined specifically for congestive cardiac failure. Any other abnormal presentation seen was also noted.

Laboratory Investigations

A: Routine Investigations

(i) A 2 ml sample of venous blood was collected into a sequestrene bottle for the following tests:

(a) Full blood count, using the Coulter Counter Model"S" in the haematology laboratory.

(b) A peripheral blood film stained with May-Grunwald Giemsa was routinely reported and a differential white cell count done.

(c) A sickling test was done by the sodium metabisulphite incubation method, followed by haemoglobin electrophoresis on cellulose acetate paper at pH 8.6.

(d) A reticulocyte count was done from films stained
with brilliant cresyl blue stain, whenever possible.
(ii) Stool specimens were routinely collected and examined
for occult blood, and then for the presence of parasites
using the Formol-Ether concentration method.

B: Other Investigations

All these investigations were done by specialized individual technologists and all were evaluated and reported on by the haematologists.

(a) A bone marrow aspirate was done at the anterior iliac
 crest, except in 2 cases when it was done at the sternum,
 on patients in the following categories:

(i) Those who presented with a bleeding problem;
(ii). Those with significant lymphadenopathy;
(iii) Those referred for recurrent anaemia;
(iv) Those in whom the peripheral film had abnormalities that required further investigation;
(v) Those patients whose history or physical signs pointed

to an underlying disease to have contributed to the causation of anaemia.

The smears were immediately made from the aspirates, fixed, and then routinely stained with May-Grunwald Giemsa as well as for iron using the Prussian blue technique. (b) A platelet count was done and where necessary coagulation screening tests (bleeding time, Prothrombin time, Thrombin time, and Kaolin Cephalin Clotting time as well as specific factor assays when indicated) were done in patients with a suspected bleeding disorder. Further tests for haemolytic screening (namely Direct Coombs, blood grouping, Glucose-6-phosphate dehydrogenase screening, Heinz-body test and osmotic fragility) were done

wherever possible in those instances of unexplained haemolysis.

(c) Other investigations relevant to diagnosis of underlying disease were also done.

All specimens were taken and delivered to the laboratories by either the author or through the routine hospital channels. The results were likewise collected daily by the author either straight from the laboratory or from the relevant areas in POW, via the hospital dispatch system, and were then noted on to the proformas.

RESULTS

Of the 4,044 recorded admissions during the study period, 294 (7.3%) were documented cases of anaemia. Out of these, the author saw and interviewed 244 (83%) cases, at some time during their period of hospitalization. There were 158 (53.7%) males and 136 (46.3%) females, giving a slight male preponderance.

Tribal Distribution and Location of Patients

Tribal distribution is shown in Table 1. Luos and Luyias predominated, making up 75.5% of the total number of patients in the study group.

Of the Luo patients seen, 59.5% stayed in Nairobi or had not been out of Nairobi in the 6 weeks preceding their admission, while the rest (40.5%) had recently been to or had come straight from Nyanza Province, mostly Kisumu and Siaya districts. Of the Luyias 41.7% were residents of Nairobi while 58.3% had arrived from Western Kenya recently, i.e. one or two weeks immediately prior to hospitalization. The majority were from Kakamega and a few from Busia districts. Most of those staying in Nairobi were concentrated in areas like Kibera, Mathare Valley, Eastleigh and Kariobangi although a few cases came from other residential areas of Nairobi. All Kikuyus came from Central Province, mainly in and around Nairobi and were mostly from Limuru, Muguga, Mbotela and Kangemi. Almost all Kambas and Kalenjins came from their respective home

TABLE 1: Distribution of patients according to tribe

Tribe	Luo	Luyi a	Kikuyu	K amb a	Kalenjin	Others	Total
Number of Patients	153	69	38	19	7	8	294
Percentage of Total	52.1	23.4	12.9	6.5	2.4	2.8	100

areas and formed the majority of the referred cases from the provincial hospitals. The other tribes comprised one Embu, one Meru, one Somali and one Kuria from Musoma, Tanzania, as well as 4 of coastal tribes, and the number coming from their respective home areas was similar to that from around Nairobi.

Age distribution

Figure 1 shows the age distribution of the patients studied. About one third (32.7%) of the patients seen were in their first year of life and 62.4% of the patients were within the first three years. There was a smaller peak in numbers at nine to ten years in an otherwise downward trend with advancing age (Appendix II). The youngest patient was one day old delivered at home to a healthy primigravida, and the oldest was thirteen years old. The mean age of the patients was three and a half years.

The age distribution according to tribes of the 4 major tribes is shown in Figure 2 and the distribution of all the tribes is detailed out in Appendix III.

Luos and Luyias feature more prominently in the younger age groups (Appendix III) when compared with the other tribes. In fact 69.9% of the Luo and 65.5% of the Luyias cases appeared within the first three years as compared with 48.6% of the Kikuyus.



Fig 2 Age distribution of patients from the four major tribes



Five of the eight (72.5%) Kikuyu patients seen in the first year of life were in their first three months as compared to 20% of the Luo patients. The other tribes were more or less evenly scattered across the whole age spoctrum.

Clinical Presentation

Referral cases constituted 22.4% of the total number of patients. Of these 6.6% came from the provincial hospitals and the rest from various City Council clinics in and around Nairobi.

The clinical presentation was very varied as this depended on the symptomatology and/or signs of the primary underlying diseases. The features seen are presented in Table II.

Pallor was present in all cases since it formed the clinical basis for the presumptive diagnosis of anaemia. More patients (161) had moderate pallor as compared to those (134) who had severe pallor. The pallor correlated best with eventual haemoglobin levels in those cases of anaemia without other complicating diseases, and it was in fact exeggerated in those with pneumonia and diarrhoea and vomiting.

Fever was the next most common finding. This is a common symptom in paediatric admissions.

Most patients with splenomegaly had malaria whereas those with jaundice and bone pains had sickle cell disease .

TABLE II

Frequency of clinical features seen in the patients.

Clinical Foatures	Mild- moderate form	Marked or severe form	Total
Pallor	161	134	294
Fever	35	12	47
Malnutrition	36	9	45
Respiratory infections	22	15	37
Splenomegaly	32	3	35
Diarrhea+vcmiting	18	10	28
Hepatosplenomegaly	20	6	26
Jaundice	17	3	20
Bone pains	15	3	18
lepatomegaly	12	3	15
leakness	12	2	14
Abdominal swelling	7	3	10
Epistaxis	11	0	11
Convulsions	2	5	7
Cardiac disease	6	0	6
ymphadenopathy	3	3	6
ailure to thrive	4	1	5
iscellaneous	6	2	8

Hepatosplenomegaly in most cases was of undiagnosed cause but was a frequent finding in those with neoplastic diseases.

Epistaxis was minimal in all cases. It was seen in all types of bleeding disorders (congenital and acquired) and in some cases who were eventually proven to have no bleeding diathesis. There were no patients with purpura or petechiae.

Many children had features of mild to moderate malnutrition and a few were severely mainourished, mostly, with kwashiorkor.

Respiratory tract infections, both upper and lower, were seen in 37 cases and included acute coryza, tonsillitis, otitis media and bronchopneumonia.

Diarrhea and vomiting was not infrequent, presenting either alone, or with a febrile illness and in 10 cases was life-threatening.

Other presenting features like failure to thrive, convulsions, cardiac disease, lymphadenopathy and loss of appetite were seen in a few cases. Miscellaneous findings seen on single occasions included haemarthrosis, hydrocephalus, haematuria, haematemesis, malabsorption syndrome, orchitis and a systemic-lupus-like disease which was not confirmed.

Some patients (23.8%) presented with features of anaemia only i.e. fatigue, palpitations, failure to play or malaise. A few of these, especially in the first year of life, were picked up and referred from the child wel-

fare clinics without any symptoms except for the presence of gross pallor. In this group, the only abnormality noted on physical examination was pallor and/ or features of congestive cardiac failure (CCF). Only two patients presented with peripheral pitting oedema that could not be attributed to some other disease. The pattern of presentation of the patients with regard to cardiac failure is summarised in Table III. More patients having anaemia only presented with complicating CCF (23.8%) than those who had anaemia with another disease process (5.6%).

Haematological Parameters

Seven patients had no results at all, two died before samples were collected and the results of five patients got lost. Thirty (10.2%) patients had haomoglobin and packed cell volumes only done because either it was the only investigation requested or it was the only one done by the laboratory at night. In some, results of subsequent investigations were not obtained because the blood samples clotted or could not be traced. Eighteen cases had incomplete haematological data, making it impossible to make a diagnosis or determine the type of anaemia present. In some instances this arose from incomplete requisitions and /or incomplete testing, or inadequate reporting, especially of the peripheral film.

A total of 59 (19.4%) bone marrow aspirates were done, two of them at postmortem; 45 of them confirmed the

Presentation	Total	Without CCF	With CCF
With anaemia only	58	39 .	19 (32.8%)
Anaemia + other disease	186	170	16 (8.6%)
Total	244	209	35 (41.4%)

diagnosis but the rest were either qualitatively inadequate or not conclusive. In nine cases, a bone marrow examination was indicated from the peripheral blood findings but it was not done because the patients had either been discharged or had absconded before the blood report was

Degree of Anaemia

The haemoglobin level ranged from 2.0-14.0 gm/dl. One with Hb of 10.2 g/dl, was a 6 month old baby who was brought to hospital with gastro-enteritis and severe dehydration, while the other one with 14.0 g/dl was a 12 year-old boy who had acute haematemesis and was in a state of shock.

There were 19 (6.4%) patients with Hb levels of 3 g/dl or below. This group included some of those cases who died soon after admission without investigation, but those who were investigated had mostly iron deficiency anaemia and many were in congestive cardiac failure (CCF). The Hb level distribution showed that 40.6% of the patients had Hb below 5.0 g/dl, 49.6% had 5-8 g/dl and 7.8% had Hb levels above 8 g/dl (Figure 3 and Appendix 1V). The average haemoglobin level was 5.5 g/dl.

Type of amassia

The type of anaemia was determined from the haematological data obtained from the investigations detailed in the materials and methods. The parameters used were:





Haemoglobin level, packed cell volume, red blood cell count, mean corpuscular volume, mean corpuscular haemoglobin, mean corpuscular haemoglobin concentration, haemoglobin electrophoresis in conjunction with peripheral film report and where available, bone marrow examination and blood slide reports.

Single, as well as multiple, factors were encountered among the causes of anaemia. In 50 cases, no diagnoses were made either because of incomplete basic data or in a few cases, because the results obtained even when detailed, could not point to a diagnosis. The diagnoses made are shown in Table 1V.

There were 87 cases of nutritional anaemia, of which 80.5% were of the iron deficiency type, while 18.4% were dimorphic, i.e. combined features of iron deficiency and megaloblastic anaemia. One patient had megaloblastic anaemia; this was a one-month old baby who had presented with failure to thrive and was anaemic. His coulter indices, as well as peripheral film examination report, showed a megaloblastic (macrocytic) type of anaemia. Unfortunately, no confirmatory bone marrow examination was done as the patient was transfused and sent home before the peripheral blood report was received. Four previous siblings had died in the same family having presented with a similar illness but the mother appeared normal.

Most of the patients with the dimorphic type of anaemia had predominantly iron deficiency but, in four cases, magaloblastosis dominated the picture. All these

TABLE 1V: "Classification of Anaemias seen according to causes".

	Type of Anaemia	Numbers seen	% Age of total
Α.	Nutritional		
	Iron deficiency only	70	
	Dimorphic	16	
	Magaloblastic	1	
•	Total	87	29.6%
В	Haemolytic		
	Malaria cnly	42	
	Sickle Coll Anaomia only	29	
	Undetermined Haemolysis	21	
	Malaria & Sickle Coll Anaemia	4	
	Kalaazar	1	
	Congenital Hereditary Spherocytosis	1	
	Total	98	33.4%
С	Combinations (Nutritional & Haemolytic		
	Iron deficiency + Malaria	22	
	Iron deficiency + Undetermined haemolysis	10	
	Iron deficiency + Sickle Cell Anaemia	2	
	Dimorphic + malaria	1	
	Total	35	12.2%
D	Bone Marrew Depression		
	Infections (Tb + Typhoid)	4	
	Malignancies (AML, ALL, Wilms')	7	
	Kalaazar	2	
	Pure Red Cell aplasia	1	
	Unknown chuse	2	
	Total	16	5.4%

..../

L

I

TABLE 1V (CONT'D)

"Classification of Anaemias seen according to causes"

Type of Anaemia	Numbers seen	% Age of Total
E Bleeding Disorders		
Idiopathic thrombocytopenic		
Purpura	5	
Haemophilia	1	
Christmas Disease	1	
Von-Willebrands Disease	1	
Total	8	2.7%
Undiagnosed		
Incomplete data	45	
Uncertain diagnosis	5	
Total	50	16.9%

had bone marrow examination.

The accual underlying causes of the nutritional deficiencies could not be accurately correlated, but 70% of the patients presenting with malnutrition had iron deficiency anaemia and 19 patients out of 32 who had hookworms in the stool had iron deficiency anaemia. One patient who presented with recurrent haematuria and was iron deficient, had <u>Schistosoma haematobium</u> eggs in the urine.

Ninety-eight patients, being the largest group, had haemolytic anaemia. Of these, 42.4% had malaria, 29.3% sickle cell disease, a few had a combination of both and 24.2% had a haemolytic anaemia of uncertain origin. This latter group included cases suspected of having malaria or sickle cell anaemia but had no definite confirmatory evidence. One case, a 10 year-old Kamba, referred with recurrent anaemia, had congenital hereditary spherocytosis.

Four patients with sickle cell anaemia had evident megaloblastic anaemia and one had a hypoplastic marrow.

Multifactorial actiology was seen in 12.2% of the total patients studied, having both a haemolytic process as well as nutritional deficiency. As shown in Table 1V, malaria and iron deficiency coexisted in 22 cases, iron deficiency and sickle cell anaemia in 2 cases and malaria and a dimorphic anaemia in one case.

Bone warrow failure was seen in sixteen (5.1%) cases. Four were due to chronic infections (Tuberculosis and

Typhoid) and two had kala-azar. One case had congenital pure red cell aplasia. Seven cases in whom anaemia was secondary to malignancies, five had acute leukaemias (myelocytic and lymphocytic) and two had nephroblastoma, all previously undiagnosed. There were two cases of marrow aplasia, the causes of which were not established.

Table V shows the presentation and findings in the patients presenting with a bleeding problem. Eleven patients presented with epistaxis, of which 8 were subsequently proved to have a bleeding diathesis and 3 had no haematological cause for the epistaxis. Among the 8 cases, five had primary bleeding disorders and three were secondary to thrombocytopenia caused by kala-azar and leukeamia (already included in the bone marrow depression group).

A total of 8 primary bleeding disorders were seen and included 5 idiopathic thrombocytopenic purpura (ITP), one haemophilia, one Christmas disease and one Von-Willebrand's disease. ITP was seen in only female patients, 2 Kikuyus of 6 and 10 years, two 5-year old Luos and one 6 year-old Luyia. Two of these patients presented with recurrent anaemia only while the other three came with epistaxis, and none had previously been diagnosed.

The haemophilic boy, already diagnosed, presented with classical features and the Christmas disease patient, a 9-month-old boy with moderately severe disease, presented with prolonged bleeding from a cut lip and had an older brother already diagnosed with the same problem.

TABLE V Peatures of Patients presenting with a

bleeding problem.

the second second

1.

Clinical Presentation	Eventual Diagnosis	Numbers scen
Epistaxis	· ·	11
	Idiopathic Thrombocyatopenic	
	Purpura	3
	Leukemias	3
."	Kalaazar	1
	Von-Willebrand's disease	1
н	No haematological cause	3
Haematemesis	Unknown cause	1
Haematuria	Schistosoma haematobium	1
Haemarthrosis	Haemophilia	1
Prolonged bleeding	Christmas disease	1
TOTAL		15

Causes of anaemia in relationship to age

This is detailed in Table VI. Malaria and then iron dificiency were the leading causes of anaemia in early life, both constituting 47% of the causative factors in the first year of life. Sickle cell anaemia and the other haemolytic disorders were also mostly seen in the younger age groups, contributing 26% of the causes of anaemia in infancy. Malaria and sickle cell anaemia affected mainly infants especially in the first eight months, and contributed less and less among the causes as age advanced. Iron deficiency,however, although mostly seen in infancy, continued to be seen in later childhood. The other major groups, namely bone marrow depression and bleeding disorders were uniformly distributed among all age groups.

Distribution of the various types of anaemia amongst the tribes studied.

A summary of the distribution of different types of anaemia among the different tribes is shown in Table V11. This confirmed what had already been conceived as possible from earlier results. Luos and Luyias, who contribute the bulk of patients, have the highest incidence of malaria and sickle cell anaemia in the series. Kambas and Kikuyus do constitute a relatively higher percentage of the less common bleeding disorders and marrow aplasias. Iron deficiency was a common problem for all tribes but
Type of Anaemia				Age	Rai	nge	In	3	lears				
	0-1	>1-2	>2-3	>3-4	4-5	5-6	>6-7	> 7-8	> 8-9	>9-10	> 10-11	>11	Total
Nutritional Iron deficiency	14	13	Э	6	5	4	5	3	4	4	2	1	70
Dimorphic	- 4	2	1	1	1	1	0	1	0	2	0	3	16
Meraloblastic	1												1
Haemolytic													
Malaria	25	9	5	1	0	2	0	0	0	0	0	0	42
Sickle Cell Anaemia(SCA)	8	7	4	1	4	1	0	0	0	2	2	0	29
Others	9	6	3	6	1	0	0	1	0	0	1	0	27
Combinations	12	9	5	2	4	0	1	2	0	0	Ō	0	35
B.M. depression	1	2	0	1	0	1	2	2	2	2	1	2	16
Bleeding disorders	1	0	0	0	1	2	1	0	1	1	1	0	8
Undiagnosed	21	6	6	5	3	4	0	0	0	3	2	0	50
TOTAL	96	54	33	23	19	15	9	9	7	14	9	6	294

TABLE V1 Causes of Anaemia in Relation to Age

TABLE V11Distribution of the various types of
anaemia amongst the tribes studied.

Type of Anaemia	TRIBE										
	LUO	LUYIA	KIKUYU	KAMBA	KALENJIN	OTHERS	TOTAL				
Nutritional:											
Iron deficiency	28	21	13	4	2	2	70				
Dimorphic	1 + 8	3	2	3	0	0	17				
Haemolytic:											
Malaria	25	10	3	2	1	1	42				
SCA	20	8	0	0	0	1	29				
Others	16	11	0	0	0	0	27				
Combinations:	24	4	3	1	2	1	35				
B.M. depression:	5	1	4	4	1	1	16				
Bleeding disorders:	2	1	3	1	1	0	8				
Undiagnosed:	24	10	10	4	0	2	50				
TOTAL	153	69	38	19	7	8	294				

relatively more so in the Kikuyus where it contributed 34.2% as compared with 18.3% in the Luos as a cause of anaemia.

OTHER FINDINGS

Eosinophilia: Varying degrees of eosinophilia were observed in 20 (6.8%) cases, the highest count being 24% with an average count of 8%. There was no clear correlation with the presence of parasites in the stools.

<u>Stool examinations</u>: Of the 118 stool examination results received, 66 (55.9%) had no pathogens, 22 (18.6%) had hookworms only, 17 (14.4%) had other pathogenic parasites namely, <u>Ascaris, Trichuris</u> or <u>Strongyloides</u>, and 10 had both types (i.e. hookworm and the other helmiths). Only one patient had occult blood reported positive and he had Ascaris as well.

Of the patients with hookworm in the stools, 60.5% had iron deficiency anaemia and the rest were found in those with other types of anaemia.

PATIENT MANAGEMENT AND FOLLOW-UP

Over 95% of those patients with severe and 55% of those with moderate anaemia were transfused. Blood collection from patients for investigations as well as for grouping and cross-matching at the same time, in those with severe pallor, was routine practice. In most cases blood for transfusion was received before the results of the investigations. The patients were therefore transfused, and in the absence of any other problem they were discharged within 48 hours. The majority therefore were given appointments to return when the haematological and other results would be expected to be ready. Only a few returned and fewer still could get their results traced.

Among those discharged before diagnosis and not seen again were 9 patients of whom 4 had dimorphic anaemia, one ITP, one congenital spherocytosis, one typhoid fever, one schistosomiasis and one had acute myeloid leukaemia. There is no evidence that any of them ever received appropriate treatment.

Twenty five patients were seen with recurrent anaemia. Of these, 6 were repeatedly seen during the study period but only 3 were eventually fully diagnosed. One of them died soon after readmission and the rest were again quickly treated and discharged.

Only 25 patients were successfully followed and subsequently discharged by the author: 35 others requiring specialized follow-up were referred to the haematology clinic. The majority were lost to follow-up.

There were 34 (11.6%) deaths among the 294 cases included in this study, in comparison with the overall figure of 154 (3.8%) deaths in POW from all causes during the study period. Fifteen patients died within minutes to hours of admission, with little or no investigations

done. The remaining deaths had severe anaemia associated with malaria (6), malignancies (6) sickle cell anaemia (2), iron deficiency (2), ITP (1) and uncertain cause (2). A few of these later deaths occurred in patients who had already been admitted to the main wards awaiting transfusions (repeated) and or on definitive treatment for their ailments.

DISCUSSION

The results of this study show that anaemia is indeed still a major problem, and also of similar pattern as previously found. The finding of 294 (7.3%) anaemic children in a period of 4 months among 4404 admissions, only goes to emphasize the gravity of the problem. Moreover, this may only be an underestimate as many unrecorded cases are likely to have been missed and therefore not included in the study, especially those admitted for some other severe disease. Although there are no comparable statistics from previous studies done here, there is no evidence to show that anaemia is on the decline in our set up in spite of social and economic progress. On the contrary, a review of records in the blood transfusion service of Kenyatta National Hospital (KNH) revealed that an average of 17 transfusions were being given to anaemic children in POW in a week, during the study period. This compared with 6 to 7 transfusions weekly in the same type of patients 2 years proviously. This is a big increase even when one takes into account the increase in the admission rate over that period. Furthermore, there has been a significant increase in the number of children who attend the weekly haematology clinic for disease conditions that would invariably present to the POW with anaemia.

Tribal discribution

Kenyatta National Hospital (KNH) serves as a referral hospital, s district hospital for Nairobi, as well as a

provincial Lospital for part of the Central Province. One would therefore, expect the tribal distribution of the attending population to relate closely to that of the tribal distribution in Central Province and neighbouring areas. Indeed, many workers at KNH have found this to be so; with the Kikuyus forming about 60%, Kambas 15%, Luos 10%, Luyias 7% and the rest of the tribes 8% (24, 28, 29, 30. 31). In this study, however, Luos formed 52.1%, Luyias 23.4%, Kikuyus 12.9%, Kambas 6.5% and the rest of the tribes 3.2%. In fact 75.5% of the patients were of Western Kenya origin although about half of them were Nairobi residents. They were mainly children of labourers working in Nairobi and stayed in the areas of the low income groups. Even recent arrivals to Nairobi had their fathers similarly working in Nairobi, and on falling sick they were brought for medical attention in the city. Mati et al (25) studying anaemia in pregnancy found a similar distribution of patients and their migration patterns.

Age distribution

Not many studies have been done on anaemia in children in the tropics. In East Africa most workers who have studied anaemia have had only a few children in their series (7, 8, 17, 18, 19, 21, 23, 24) Ebrahim (26) specifically investigated anaemia in infants and Varnier

(32) looked at anaemia in children very generally. However, from all these studies it was evident that, like elsewhere in the tropics, anaemia of children is commonest and most sovere in infancy and early childhood (27,33). The same pattern was observed in this series, in which about a third (32.8%) of the patients were infants and over half (62.4%) were under 3 years old. Although the age range of the patients seen was from 1 day to 13 years, very few children were seen above 6 years (18.6%), and most were clustered around 9-11 years of age, a finding that was difficult to explain since there was no common factor.

There are many possible explanations for the occurrence of severe anaemia in early childhood. Infants and young children have rapid growth rates, with high demands for nutrients and other growth requirements; hence making them vulnerable to deficiences, some of which could result into anaemia (1,4). These same children are also more susceptible to diseases like gastro-enteritis, malaria and haemoglobinopathies, any of which could give rise to anaemia (27).

Clinical features

Most of the children, especially in the younger age group, never had symptoms relating to anaemia, even when it was the sole problem on admission. Many would never have been seen therefore, if they had not presented to

medical institutions for some other reason. Morley (34) states that the adulthood symptomatology of anaemia is vague and uncommon in children. This is likely to contribute to the fact that about 75% of the patients in this series were admitted for some other problem, and the anaemia discovered incidentally. However, the clinical signs due to anaemia in children are comparable to those of adults because of similar haemodynamic changes. Hence there is no special identification problem on physical examination. It is also not surprising that the patients who came to hospital with anaemia as the only presenting feature had severer forms (average Hb 3.3 gm/dl as compared with 5.5gm/dl for all patients). Again, many more of these patients had complicating congestive cardiac failure (32.8%), in contrast to 8.6% of the other patients with other diceases. Some workers did relate severer physical features with megaloblastic anaemia when compared with iron deficiency of the same degree (8, 35). No features were specific or related to any type of anaemia in this study as most were related to the underlying illness which were very varied. Splenomegaly was most commonly found in those with malaria and sickle cell anaemia. This is a common association especially in the younger age group (36, 37). However, it was also found in many cases with other types of anaemia, some of which could not be explained and was not investigated further. Oedema that could certainly be attributed to anaemia was seen in 2

patients only. This was consistent with earlier findings of Foy and Kondi (7,8), that anaemic patients in this country don't often present with oedema. In contrast, it was observed in Uganda that patients with severe anaemia walk into the wards with puffiness and oedema (15,38,39,40).

Degree of Anaemia

In this study, a haemoglobin level of 10 gm/dl was considered as the level below which anaemia was unquestionably present. This was to allow for differences in 'normals' at different ages. Other workers have used the same haemoglobin level to define presence of anaemia (5.25,41) because the true levels are unknown in the tropics. The WHO however considers Hb level of 14.g/dl and above as normal. Again Hb level of 5g/dl or below was defined as severe anaemia, while moderate anaemia was 5-8g/dl and above this as mild anaemia (25,26,24, 40). From the results of this study then, only two patients were not considered to be anaemic (Hb> 10gm/dl), and 40.5% of the patients were severely anaemic, 49.2% moderately and 7.8% wildly anaemic. The presence of pallor was therefore an accurate indicator for the presence of anaemia. The severity of anaemia could not be as well matched with the degree of pallor because of other influencing factors. Considering that about 75% of the patients in the study came to hospital for enterely different reasons, it is not unreasonable to predict that

the prevalence of anaemia in children in the general population is quite high.

Types and Underlying causes of anaemia

Identification of the underlying actiology of anaemia forms the basis for its management. Therefore, every effort should be made to find the cause in every anaemic patient. Many of the patients in this series were inadequately investigated, and could not be diagnosed for reasons already indicated. In many others treatment came before diagnosis, so that the results obtained later were not utilized in patient management - a deplorable situation because investigation then is a waste of time and resources, which are scarce in our setting.

Classification of the anaemias according to causes was extremely difficult because of the multiplicity of causative factors and the resultant modification in haematological indices considered diagnostic of the various types of anaemia. Several early workers on anaemia in the tropics found this multiplicity in causes very common (7, 21, 36, 42, 43).

Furthermore a single actiological factor could cause anaemia by several machanisms. However, effort was made to simplify a complex problem by considering the predominant picture in the results of each case. Woodruff (44,46) reviewing different studies on anaemia in the tropics concluded that haemolysis from malaria

and haemoglobinopathies, as well as anaemia of deficiencies were the commonest. Workers in East Africa have found the same high frequency of malaria and iron deficiency as main causative factors of anaemia in early childhood (17, 18, 26, 27, 23, 40, 43). The present study shows a very similar pattern. The commonest types were haemolytic due to malaria and sickle cell anaemia (30.2%), and iron deficiency anaemia (26.2%) or a combination of both (12.0%); accounting for 68.4% of the total number of the cases. Higher rates of the main causes would be obtained if the percentages were based only on these cases in which the diagnoses were well worked out. The results imply that the pattern of causes of anaemia in this environment has not changed much over the last two decades.

Most of the children afflicted with malaria and sickle cell anaemia were those in the first 2 years of life. Age analysis of the patients according to tribe showed that most of the patients in the first 2-3 years of life were Luos and Luyias. These two ethnic groups come from areas where malaria is endemic and sickle cell trait incidence is high. This is in accordance with what is already known about these two diseases. Malaria only seriously affects the young children in those indigenous in the malaria endemic areas. Similarly sickle cell anaemia is most severe in the first 18 months

of life (36,37). Almost all cases of malaria were infections due to Plasmodium falciparum, the commonest and most severe type, but one case of Plasmodium vivax infection was seen in a child presenting with recurrent fevers and anaemia. Among those who had malaria were nine Kikuyus and one Masai, who had only resided in their respective home areas, where malaria transmission is only seasonal; occurring during and soon after the rain season of March to May (46). Mati et al (25) who conducted their study soon after the rain season found that 100% of their patients with haemolytic anaemia had malaria. The present study, however, was mainly conducted during the dry season, and many of the patients afflicted were residents of Nairobi and the surroundings. This then raises the question as to whether malaria may not be more prevalent in Nairobi, at present, than previously found.

Sickle cell anaemia was seen among Luos and Luyias only, which is in keeping with the already established distribution data on haemoglobinopathies in Kenya (46,47). Folic acid deficiency can complicate and aggravate anaemia of malaria and sickle cell anaemia, especially in subjects with borderline stores of the haematinic. This deficiency was seen in a number of cases who had malaria or sickle cell anaemia with frank megaloblastosis on marrow smears. The number could have been more if a larger group had had marrow aspirations done. Any febrile illness

and in particular malaria, has been found to aggravate haemolysis and thrombosis in those with sickle cell anaomia(48,49). This may have contributed to the large number of sicklers in crises. Malarial prophylaxis in these patients was found to reduce the number of crises, (50); this forms the basis for its routine use in these patients. In the haematology follow up clinic, this is also routinely done, but may not be so widely practised in other clinics due to the present misconception that malaria is uncommon in Nairobi. Other preventive measures against respiratory infections, which are very common in our overcrowded poor urban areas, are not practised in our set up.

A case of congenital spherocytosis, a rare hereditary haemolytic disorder, and one case of hypersplenism in a kalaazar patient with a huge spleen, were seen. This implies that even the rare disorders may not be so rare if properly looked for. However, in many cases of haemolytic anaemia the underlying cause was not confirmed, although one can say that most of these cases were of malaria or sickle cell origin, when all clinical and laboratory information is considered. The majority were young children of Luo and Luyia tribes, who made up most of those fully diagnosed haemolytic anaemias. One must emphasize,however, the importance of doing full haemolytic screening tests in uncertain cases to rule out less common causes, such as G-6-P-D deficiency, which is prevalent in East Africa (51, 52). No such was seen in

this series; neither was any auto-immune haemolytic type encountered.

Deficiency anaemias were seen in 29.2% of the cases, mostly iron deficiency and a few dimorphic cases. Only one case of megaloblastosis was seen in an infant, apart from those cases seen complicating malaria and sickle cell anaemia. On the whole, little megaloblastic anaemia was encountered in this study. Earlier studies at KNH had seen percentages ranging from 15% to 30% of the total causes in adults (8, 24, 25, 53). Many could have had incipient deficiences that could only be diagnosed on bone marrow smears which were not done in all cases. However, in the studies quoted above at KNH, no megaloblastosis was seen in the children included in those studies; and this has also been observed elsewhere (43, 54). Significant megaloblastic anaemia in children has only been described in association with Kwashiorkor, infective processes and sickle cell anaemia (36,54,55,56,57).

The underlying cause of the deficiency anaemias in the tropics has been found to be basically dietary in origin, the commonest being due to absolute low dietary intake. In some instances however, the dictary content of these elements is adequate, but rendered deficient through loss by improper or faulty food preparations. Low animal proteins in diets renders iron absorption from vegetable sources much less absorbable, while diets rich in phytates have been implicated in impairing iron absorption by

intestinal precipitation (4,58,59,60,61,62). These are particularly problems of the low social and income groups who cannot afford rich animal foods, coupled with the frequent diarrhoeal diseases that go with poor sanitation. In this study, many of those afflicted had diarrhea and vomiting, and/or obvious features of malnutrition. The figure would have been higher if all cases with malnutrition were included, as they are usually anaemic to some degree. Looking at the occupations and residences of their parents revealed that most of them were amongst the lowest income group in the city. Their diets consisted mostly of uji or ugali (maize meal) and fresh vegetables. Maize has high phytate content, is a vegetable and its iron and folate content is low. Most younger ones were on inadequate breast milk or diluted infant formulas.

In iron deficiency, the presence of hookworm infestation contribute to worsening of the deficient state, and often precipitate frank anaemia (8, 12, 42,60). The prevalence of hookworm infestation is very high in the warm tropical environment. Workers in East Africa found hookworm infestation very common, especially in the poor sectors (12,63,64,65,66,67). However, the presence of hookworm and iron deficiency in an individual does not always bear a causal effect relationship. The hookworm load and the level of previous iron stores are the determining factors in these situations(8,18,60,68,69). Hookworm infestation has been found to be an important causative factor after the first 18 months (19). In this

study, intensive and detailed investigations on hookworm infestation could not be undertaken. Nevertheless a significant number of children (60%) with hookworm in stools. had iron deficiency anaemia, and most were 2 years old and above. This may have played a role in the severity of anaemia in those patients. It must be pointed out that the method of stool collection is very inadequate, leaving specimens standing for long periods, which may have contributed to the many negative results obtained. Moreover, only 43% of the stool examinations requested had results received. One patient with haematuria as the cause of iron deficiency had <u>Schistosoma haematobium</u> as the underlying cause. This is commonly found in those areas where this parasite is a common infection (42,44).

In megaloblastic anaemia, dietary deficiency is a much commoner cause. Changes in intestinal flora with resultant interference in B_{12} absorption has been implicated (70). Pernicious anaemia is rare in Africans and unknown in childhood (35, 71). The cases in this study can therefore only be attributed to nutritional deficiency associated with high demands. Other dietary factors that have been implicated in the cause of anaemia are Ascorbic acid, Riboflavin, and pyridoxine (55,7A,73). These require sophisticated methods for evaluation and they are not available in our routine set up. The role of protein deficiency in causation of anaemia is still under debate. Some workers have stressed its importance as a cause of

anaemia (57,72,75,76) while others have doubted its significance (8, 19,58). In malnutrition per se, anaemia has been found to be of multiple actiologies, and variously macrocytic, normocytic or hypochromic (55,38,73). In the present study most of the malnourished children included had iron deficiency and a few had the dimorphic type. Combinations of nutritional deficiences and haemolytic processes were seen in 35 (12.2%) cases, again emphasizing the fact of multiple actiological factors occuring, especially among the common causes.

Anaemia of bone marrow failure can arise from numerous causes as was outlined earlier, but these assume much less importance in this environment in the face of the overwhelming causes described above. In this study, a small but significant number of bone marrow failure cases (5.1%) was seen, and in most cases an underlying cause was identified. These included acute leukemias, nephroblastomas, chronic infections, namely typhoid fever, tuberculosis and kalaazar. In 2 cases, an underlying cause was not identified from both the clinical and laboratory data. One case of pure red cell aplasia was also seen.

Tuberculosis and typhoid fever are still frequent illnesses in this set up. The leukemias and nephroblastomas are believed to be infrequent childhood diseases, but are commonly seen at KNH, this being the national referral centre. However, none of these patients had been diagnosed previously, indicating that these diseases are not as

uncommon as previously thought. The patients often present with anaexia, and therefore should be borne in mind in all casos of anaomia. Kalaazar is ondomic in Ukambani aroa of Kenya, where all 3 patients in this study came from. Several mechanisms in the actiology of anaemia in this infection have been implicated (77, 78). Poor iron utilization in the marrow is thought to result in the peripheral hypochromic, microcytic picture often seen in these cases (77, 78,79,80). This picture was seen in all 3 cases in this study, two of them had no stainable iron while the other had plenty on marrow smear. They also had features of bone marrow depression or haemolysis. Pure red cell aplasia, also known as erythrogenesis imperfecta or aregenerative anaemia, is a rare congenital disorder of unknown cause; manifesting with selective erythropoietic hypoplasia, first described by Blackfan and Diamond in 1933 (1). In Africans less than 10 cases have been described, 4 of them at KNH (81,82), and this case is one of those already reported by Kinoti et al (81). Some workers have seen an acquired form of this disorder in children with malnutrition, who are undergoing treatment and are improving. The disorder improved when the children were given riboflavin, implying that this element may be the underlying cause (83).

Bleeding disorders may arise from coagulation factor deficiency, capillary fragility or more commonly, from platelet disorders. These disorders may be either congenital or acquired. Early workers had found the congenital

bleeding disorders rare in Africans (84,85). In East Africa, later workers found the disorders, especially haemophilia, not as rare as previously considered (61, 85. 86). Recently, Kasili and Kariithi(89) reviewing hereditary bleeding disorders, and Kariithi (90) reviewing coagulation studies done, both at KNH, found these disorders, especially haemophilia, Christmas and Von-Willebrands' disease to be common. In this study eleven cases of bleeding disorders were seen, of which 8 were primary bleeding diatheses. Of these 8, 5 were Idiopathic Thrombocytopenic Purpura (ITP), one haemophilia, one Christmas disease, and one Von-Willet ands disease. These results differed from those referred to above, but may relate to the age-difference of the patients in each group. ITP was seen in only female patients of 5-6 years of age, and mostly among Kikuyus. This compared well with what has been observed by other workers both here and elsewhere (86, 87,89,91). Most of these cases with ITP presented with epistaxis, and others had anaemia only, although they all gave a history of past episodes of recurrent epistaxis when closely questioned. Therefore, some cases may be missed if the level of suspicion is low and adequate history not taken and therefore relevant investigations not done in those patients presenting with anaemia.

Disgnostic problems encountered

Several factors contributed to the high number of undiagnosed cases in spite of special efforts made to

have all the patients fully investigated during the study period. Many patients with anaemia arrive late in the evening or at night. However, the type of laboratory services available at such a time are inadequate for the provision of information required for diagnosis, while blood can readily be provided for transfusion. Moreover, storage facilities for specimens taken late, on POW are not there. The high turnover of patients in all units of the hospital, and POW in particular, lead to high workload both for the ward and laboratory staff. Therefore, the quickest but often unsatisfactory way to solve the problem is chosen by those concerned. Hence many times, the readily done samples are taken and minimum requisition written, while the least tedious investigations are done in the lakoratory. Collection of specimens and results was noted to be most inefficient. The hospital staff entrusted with this task are not aware of their role in patient care, and they are not even properly supervised. They are not responsible to the wards or laboratories they serve. This results in late or no collection of specimens, as well as the results. On occasions, shortages of proper specimen tottles compromised proper investigations at the relevant times. At times, sequesterene bottles had no anticoagulant, and this led to clotting of blood and wastage of specimens. These are problems likely to be found in many areas where the few available resources are stretched to the limits.

Patient Management

Many workers have emphasized the importance of diagnosis first and then treatment, as the only proper way of managing patients with anaemia. Transfusion and then discharge, without diagnosis as so often done in POW is therefore irrational; whatever the handicaps of the system. Due to poor follow-up, it may prove detrimental to the health of the patient, when some diagnoses requiring prompt, specific therapy are made, but treatment not implemented. This was-seen in some cases in this study, including acute leukemia, typhoid fever, congenital spherocytosis and dimorphic anaemias. None of these patients returned for review to receive treatment. Furthermore, frequent loss of results coupled with poor follow up makes the rationale of discharging patients before diagnosis, and recalling them to be reviewed with results, unjustifiable. Certainly a few of the cases presented to the ward with the same problem as new cases, and treated as such.

On discharge many patients were prescribed haematenics, usually iron, for one to two week periods. This is far below what is needed to correct the anaemia and replenish the stores, in those who require iron. This needs up to at least 10 weeks' adequate treatment (36,39). The same patients would receive Ketrax or Alcopar, the only available drugs against hookworms. The efficacy of these drugs in effectively treating <u>Necator americanus</u>, the commonest species in this environment, has been doubted

(39,42,67,92). Improvement seen in those treated can therefore be only transient. Many of these patients are likely to return with the same condition and contribute to even greater numbers of patients being seen. In many cases, transfusions were not indicated. Another 24-hour stay in hospital to await hagmatological results would facilitate proper management being effected without endangering patients' lives and not costing the hospital much more. Certainly in those with decompesated cardiac function, where life is threatened transfusion is life saving. This is usually in infants, with rapidly progressing anaemias (26,40,39). It is therefore unwarranted to transfuse every anaemic patient indiscriminately. Five cases were adequately investigated but not diagnosed. These represent some obscure anaemias occasionally seen, requiring careful follow up for several months or even years, before the underlying cause can be identified.

Mortality

Deaths occurred more among the anaemic patients than among other patients on the ward. However, those who died after being adequately investigated had fatal primary diseases, and anaemia only hastened their deaths, especially in those where blood was not readily available to correct the anaemia. Where effective specific therapy is available, early diagnosis, and then prompt institution of treatment is desired. Many of those who died however, had no specific diagnoses made, having arrived in the hospital too late to allow full investigation.

Conclusion and Recommendations

Anaemia is a common problem in children admitted to the Kenyatta National Hospital, and constituted 7.3% of the total number of admissions to the POW. Since POW caters for only severely ill patients, then the prevalence of anaemis in the majority of children attending the hospital, and those not in hospital, is not known. Although hospital patient population is the least satisfactory to be used for estimation of general population parameters, the presence of marked anaemia in most of the children seen, who had presented with some other disease, is likely to reflect on high prevalence of anaemia in the general population. One of the recommendations from this study, is a proper population survey, to determine the 'normal' haemoglobin level of the population, and then determine the true prevalence of anaemia, in different parts of the country. More meaningful deductions would then be possible from studies like this one.

The pattern of anaemia seen in this study compared well with earlier studies done in children elsewhere (17,18, 19,23,26,27) and at the KNH (8,24). Haemolysis, mainly from malaria and sickle cell anaemia were the most frequent causes, but nutritional iron deficiency and/or folic acid deficiencies were almost as common. The most probable cause of these nutritional deficiencies is poor dietary intakes. Hookworm infestation is likely to have played a role in precipitating severe anaemia in these

patients having iron deficiency and carrying the parasites. The most affected age group are the infants and those in the second year of life. The problem is concentrated among Luos and Luyias, regardless of whether they reside in Nairobi or their indigenous areas. They all belonged to the very low income groups and this is likely to reflect on their poor diets in essential nutrients, poor sanitation and overcrowding; predisposing them to recurrent infections and infestations. It is the same group of children who make up the majority of marasmic and kwashiorkor admissions in the paediatric wards. It would therefore be worthwhile to increase child welfare clinics in the poorer sectors of the city and rural areas. Children in these clinics could then be supplied with iron and folic acid routinely to boost their stores. Unfortunately it is the non-attenders who usually require these supplimen's most. In older children attending school, good nutrition and possibly fortification of diets in well organised school feeding programmes would be advantageous. This could be incorporated in the present school milk programme. Fortifications have been tried before in some areas and has not been successful (93). In school children, however, periodic deworming and supply of haematinics has been found useful (75, 12) and can easily be done by school authorities with the supervision of health workers. Malaria in Nairobi, even when thought to be uncemmon, was found most prevalent in the Eastland

areas of the city, where most of the patients came from. However, the disease now appears to be much more common than previously considered. Rapid population movement to and from the endemic areas, and the presence of Anopheles mosquitoes in Nairobi has facilitated increased malaria transmission in Nairobi throughout the year. A carefully done survey to define the current prevalence of malaria in Nairobi, would help eradicate the belief among health workers that malaria is uncommon in Nairobi. Then an increase in simple environmental control measures, like removing breeding areas and DDT spraying, would be useful. A more liberal use of anti-malarial drugs in young child clinics and those at special risk, would reduce both the morbidity and possibly mortality.

Causes of anaemia that were previously rare or not seen at all are becoming more common with improvement in diagnostic fascilities. What need be done is to have a higher index of suspicion regarding these 'rare' causes and thoroughly investigating every patient. The numbers would have probably been more if all cases received adequate investigation.

There is need to re-emphasize the need to desist from transfusion of all anaemic patients. It is both unnecessary and expensive. No patient should be transfused if not haemodynamically decompesated or if there is no

evidence that the anaemia is rapidly progressive.

At present there has been great improvement in distribution and keeping of results, within POW, to assist in reviewing patients. What needs urgent attention is specimen collection and delivery to the relevant laboratories as well as the feed back from the laboratory to the wards. Negligence on the part of the staff members who collect and distribute specimens and results, contribute a lot to the delay in and sometimes complete loss of diagnosis. The same kind of attention is needed in reviewing the specimen bottles preparation unit, as often the quantity and quality provided is not sufficient

For those patients requiring urgent transfusions at night, all relevant specimens should be taken, and storage facilities should be provided in POW to keep specimens until the following morning for delivery to the proper laboratories. Patients should be able to await simple haematological results, that are often diagnostic, before treatment and discharge. Follow-up of patients needs reorganization to make it more fruitful. This could be incomporated in the haematology or general paediatric clinic, or in POW on a particular day known to everybody to refer patients to. Such patients should be followed up for at least 2-3 visits over a period of 4 months. It would be beneficial if

haematenics which are cheap drugs, were adequately available to ensure constant supply to those in need.

1

C 2 Int Co. a a Printer of supply 1804,9100 Court

Anderson and the state of the local

in F , dates of human 1, 511 (1991)

man at all states of

REFERENCES

- Linman, J.W. Haematology physiologic, pathologic and clinical principles. McMillan. N.Y. 1975.
- Lewis, S.M. Practical Haematology. 5th Ed. Churchill-Livingstone, 1975.
- Mengel,C.F. Haematology, Principles and practice.
 Year-Book. Chicago. 1975.
- 4. WHO Group of Expert Report. Wld.Hlth Org.tech. Report Ser, 503;14,1972.
- Joint FAO/WHO group of Expert Report. Wld Hlth.
 Org. tech. Rep. Ser, 452: 121, 1970.
- Woodruff, A.W. In Alimentary and Haematological aspects of Tropical Disease Ed. Woodruff, A.W., Arnold-London p.211 1970.
- Foy, H. Kondi, A. and Hargreaves, A. Anaemia of Africans. Trans. R. Soc. Trop. Med. Hyg. 48:327, 1952.
- Foy, H. and Kondi, A. Anaemias of the Tropics -East Africa. Trans. R. Soc. Trop. Med. Hyg. 52: 46, 1958.
- 9. Woodruff, A.W. and Schofield, F.D. Haemoglobin values among Gambians. Trans. R. Soc. Trop. Med. Hyg. 51:217, 1957.

- Woodruff, A.W. Anaemia in the Tropics. Practitioner
 193: 138, 1964.
- Stott, G. Hookworm Infection and anaemia in Mauritius.
 Trans. R. Soc. Trop. Med. Hyg. 55:20, 1961.
- Watson-Williams. In Alimentary and Haematological aspects of Tropical Disease ed. Woodruff, A.W. p.232. Arnold. London 1970.
- Forsyth, B.M. Anaemia in Zanzibar. Trans. R. Soc. Trop. Med. Hyg. 64:601. 1970.
- Trowell, H.C. Anaemia of Kwashiokor in relation to iron deficiency. Arch. Ds. Child. 12: 193. 1937.
- 16. Brown, R.E., Wilks, D. and Allen, D. Incidence of malaria, splenomegaly and Hookworms in school children in Uganda. E.Afr. Med. J. 47:6.1970.
- Meredith, J.S. and Eyekuze, V.M. Anaemia in children and adults in Dar-es-Salaam. E. Afr. Med. J. 39:290. 1962.
- Blackman, V. Some observations of the severe anaemias in Dar-es-Salaam. E. Afr. Med. J. 39. 235. 1962.
- Rowlands, A.K. Anaemia in Dar-es-Salaam and methods of its investigations. Trans. R. Soc. Trop. Med. Hyg. 60: 143. 1966.

20. Vaughan, J.P., Menu, J.P., Kihama, F. F. Brooke, D. Kiwia, A. and Mohamed S.A. Anaemia at a Coastal area of Tanzania. E. Afr. Med. J. 50: 86, 1973.

- 21. Turmer, P.P. Megaloblaștic anaemia in Africans at coast Provincial Hospital. Trans. R. Soc. Trop. Med. Hyg. 57:34,1963.
 - 22. Forster, R.M. The seasonal incidence of megaloblastic anaemia at Mombasa. E. Afr. Med. J. 45: 673. 1968.
 - 23. Kasili, E.G. Anaemia in patient population at a Provincial Hospital in Western Kenya. E. Afr. Med. J. 57:373. 1980.
 - Fieguson, J.C., MacKay, N. and Watson, W.C. Anaemia in Nairobi Region. E. Afr. Med. J. 45:663, 1968.
 - 25. Mati, J.K.G., Habany, A. and Gebbie, D.A.M. Importance of anaemia of pregnancy in Nairobi and the role of malaria in the aetiology of megaloblastic anaemia. J. Trop. Med. Hyg. 74: 1, 1971.
 - Ebrenim,G.B. Anaemia in infants. E. Afr. Med. J.
 43: 155, 1966.
 - Wright,S.G. Anaemia in infancy. Tropical Doctor.
 9:3, 1979.

28. Kinuthia, D. Nephrotic syndrome in patients at KNH.

- 29. Kilonzo, B.M. Aplastic anaemia at K.N.H. during 1973-1978. M. Med. Thesis, 1979.
- 30. Muita, A.K. Some observations on Natural History
 of Rheumatic fever as seen in central Kenya.
 M. Med. Thesis, 1980.
- Anahwani, G. Outcome of caesaria sections done at KNH. Maternity. M.Med. Thesis, 1980.
- 32. Vernigr,T. Causes of Anaemia in children admitted to Mulago Hospital. Tropical and Geographical Med. (Amst) 18:287, 1966.
- 33. Akinkugbe, F.M. Iron deficiency in childhood anaemia.
 E. Afr. Med. J. 55: 151, 1978.
- 34. Morley, D. Paediatric priorities in the Developing world p. 284. Butterworths, London, 1973.
 - 35. Trowell, H.C. Pernicious anaemia in Africans in Uganda. Lancet: 2, 761, 1951.
 - 36. Maegraith, B. In clinical Tropical Diseases. Ed. Adams and Maegraith. 6th Ed. Blackwell Scientific Publishers, London, 1976.
- 37. Lehrmann, H. and Huntsman, R.G. In Alimentary and Haematological aspects of Tropical disease.
 Ed. Woodruff, A.W. p. 297. Arnold-London 1970.
- 38. Trowell, H.C. Anaemia in Uganda. E. Afr. Med. J. 14: 286, 1938.

- 39. Knight, R. The management of Hookworm anaemia. E.Afr. Med. J. 45: 746, 1968.
- 40. Bwibo, N.O.. Haemoglobin response following Intra mascular Iron dextran (Inferon) in children with iron deficiency unaemia. E. Afr. Mod. J. 47:254, 1970.
- Rée,G.H. Anaemia in Maldivian population.
 J. Trop. Med. Hyg. 74: 224,1971.
- 42. Foy,H. and Nelson,G.S. Helminths in the actiology of anaemia in the tropics with special reference to hookworm and schistosomes. Expt. Parasit. 14: 240, 1963.
- 43. Luang,L.I. and Viriki,L. Anaemia in children in Malaya. Trans. R. Soc. Trop. Med. Hyg. 60: 53, 1966.
- 44. Woodruff,A.W. Recent work on anaemias in the tropics. Bri. Med. Bull. 28: 92, 1972.
- 45. Foy,H. and Kondi,A. In Health and Disease in Kenya ed. Vogel,L.C. East African literature Bureau. Nairobi, 1974.
- 46. Roberts, H.R. In Health and Disease in Kenya. Ed. Vogel, L.C. E.A.L. B. Nairobi, 1974.
- Kencall, A.G. and Barr, R.D. Haemoglobinopathies in
 Kenya. Trans. R. Soc. Trop. Med. Hyg. 67:770, 1973.

- 49. Basu, A.K. and Woodruff, A.W. Pyrexia and red blood cell destruction in sickle cell disease Lancet 2: 1088, 1963.
- 50. Warley, M.A. Hamilton, P.J.S., Marsaden, P.D. Brown, R.E. Merselis, J.G., Wilks, N. Chemoprophylaxis of Homozygous sicklers with anti-malarials and Longacting Penicillin. Bri. Med. J. 2: 86, 1965.
- 51. Knight, H.R. and Robertson, D.H.H. The prevalence of Erythrocyte G-6-P.D. deficiency among Africans in Uganda. Trans. R. Soc. Trop. Med. Hyg. 57: 95, 1963.
- 52. Marti,H.R. Schoepf,K. and Gsell,O.R. Frequency of HbS and G-6-P.D. deficiency in S. Tanzania. Bri. Med. J. 1: 1476, 1965.
- 53. WierEinga, A. and Korte, R. Anaemia among a group of male prisoners in Kenya and their nutritional status. E. Afr. Med. J. 47:646, 1970.
- 54. Adams, E.B. and Hift, W. Nutritional and other megaloblastic anaemias among Africans and Indians in Durban. Trans. R.Soc. Trop. Med. Hyg. 55: 374, 1961.

- 55. Chopra, J.G. Tantiwongse, P. Everette, L. and Villegas, N. Anaemia in malnutrition. J. Trop. Med. 11, 18, 1965.
- 56. Watson-Williams, J. Folic acid deficiency in sickle cell anaemia. E. Afr. Med. J. 39: 213, 1962.
- 57. Walt, F. Holman, S. and Hendrickbe, R.G. Megaloblastic
 anaemia of infancy in Kwashiokor and other
 diseases. Brit. Med. J. 1: 1199, 1961.
- 58. Patwardhan, V.N. Nutritional anaemias in Tropics E.Afr. Med. J. 39: 200, 1962.
- 59. Foy, H., Kondi, A. and Austin, N.H. Effect of dietary phytates on foecal absorption of Radioactive Ferric chloride. Nature, London, 183:691,1959.
- 60. Foy,H., Kondi,A. and Austin,N.H. Hookworm as a cause of iron deficience anaemia- Radioactive studies. E. Afr. Med. J. 35: 607, 1958.
- Lothe, F., Patet, K.M. and Tozer, R. Megaloblastic anaemia in Uganda. Trans. R. Soc. Trop. Med. Hyg. 63: 393, 1969.
- 62. Martinez-Torres, C. and Layrisse, M. Effect of amino-acids on iron absorption from a staple vegetable food. Blood: 35:669, 1970.

63. Sturrock, R.F., Hookworm studies in Tanzania Investigations at Tanga. E. Afr. Med. J.
 41:520, 1964.

- 64. Sturrock, R.F. Hookworm studies in Tanzania –
 Investigations at Hombolo Dodoma. E. Afr.
 Med. J. 43: 315, 1966.
- 65. Sturrock, R.F. Hookworm studies in Uganda. Investigations at Teboke, Lango District. E. Afr. Med. J. 43: 430, 1966.
- 66. Miller,L. Studies on the incidence of hookworm infection in E. Africa. E. Afr.Med. J. 47:354, 1970.
- 67. Pamba, H.O. Hookworm and Ascaris infections in Nyanza Province, Kenya. E. Afr. Med. J. 57:891, 1980.
- 68. Foy, H. and Kondi, A. Relation of hookworm load and species to gastro-intestinal blood loss and genesis of iron deficiency anaemia. Trans. R. Soc. Trop. Med. Hyg. 55:26, 1961.
- 69. Sturrock, R.F. Observations on the association of hookworms and malaria with hypochromic anaemia in 4 Rural Communities in E. Africa. E. Afr. Med. J. 43: 602, 1966.
- 70. Foy, H., Kondi, A. and Manson-Bahr, P.C. Penicillin in the megaloblastic anaemia of Africans.
Effect on serum B_{12} levels and absorption of radioactive B_{12} . Lancet 2: 693, 1955.

- 71. Mugola, E.N. Two cases of Pernicious anaemia among Africans. E. Afr. Med. J. 45:669, 1963.
- 72. Alle: ,D.M. and Dean,F.A.R. Anaemia of Kwashiokor in Uganda. Trans. R. Soc. Trop. Med. Hyg. 59:326,1965.
- 73. Brotte-Stewart, B. Anaemia of adult scurvy. Quart. J. Med. 22:309, 1953.
- 74. Foy,H. and Kondi,A. Hypochromic anaemia associated with Pyridoxine and nicotinic acid deficiency. Blood. 13:1084, 1958.
- 75. Latham, M.C. Malnutrition as a cause of anaemias in children. E. Afr. Med. J. 37:418, 1960.
- 76. Woodruff, A.W. The natural history of anaemia associated with protein malnutrition. Bri. Med. J. 1: 1297, 1955.
- 77. Knight, R., Woodruff, A.W. and Pettit, L.E. The mechanism of anaemia in Kala-azar A study of 2 patients. Trans. R. Soc. Trop. Med. Hyg. 61: 701, 1967.
- 78. Woodruff, A.W., Topley, E. and Knight, R. The anaemia of Kalaazar. Brit. J. Haem. 22:319, 1972.

55

56

- 80. Manguyu, F.W. The clinical and haematological
 response to treatment of children with kalaazar.
 M. Med. Thesis, 1980.
- 81. Kinoti,S.N., Bwibo, N.O. and Kasili,E.G. Congenital
 pure red cell aplasia case report. E. Afr.
 Med. J. 55:530, 1978.
- 82. Guillozet, N. and Mbantankhu, J.F. Congenital
 Hypoplastic red cell anaemia of childwood.
 E. Afr. Med. J. 54:345, 1977.
- 83. Foy, H., Kondi, A. and MacDougal, L. Pure red blood cell aplasia in marasmus and kwashiokor treated with Riboflavin. Brit. Med. J. 1:937, 1961.
- 84. Gelfand, M. A clinical study of severe anaemia admitted to medical wards of Harare Hospital -Rhodesia. J. Trop. Med. Hyg. 71:118, 1968.
- Griffiths, S.B. and Lipschitz, R. Anaemia among S.
 African Blacks. S. Afr. Med. J. 23: 720, 1949.
- 86. Forbes, C.D., MacKay, N. and Khan, A.A. Christmas disease and Haemophilia in Kenya. Trans. R. Soc. Trop. Med. Hyg. 60:777, 1966.
- Lothe, F. Haemophilia in Uganda. Trans. R. Soc. Trop.
 Med. Hyg. 62: 359, 1968.

- 88. Taylor, J.R. Ahluwalia, N.S., Morrison, C.J. Kaviti, J.N. and Cadwell, C.L. Cryoprecipitate in the management of haemophilia in Kenya. E. Afr. Med. 46:121, 1969.
- 89. Kasili,E.G. and Kariithi,M.W. Hereditary bleeding disorders as seen at the KNH, Nairobi. Tropical Doctor 9: 76, 1979.
- 90. Kariithi, M.W. Coagulation studies done at KNH. Med. Com. 2: 135, 1980.
- 91. Choi,S.I. and McClure,P.D. Idiopathic thrombocytopenia Purpura in childhood. Year Book of Paediatrics, p.295. 1969.
- 92. Okello,G.B.A. Treatment of Ankylostomiasis Current treatment. E. Afr. Med. J. 57: 298, 1980.
- 93. Foy, H. and Kondi, A. Incidence, aetiology, treatment and prophylaxis of anaemia in Seychelles. Ann. Trop. Med. & Parasit. 55: 175, 1961.

57

AP:	PEN	DIX	1
-----	-----	-----	---

ANAEMIA AS SEEN IN CHILDREN ADMITTED TO POW OF THE KNH

	No
NAME	
TRIBE	RESIDENCE
DATES OF ADMISSION	.DATE OF DISCHARGE
REFERAL?	

Presenting complaints (a	specifify)	DURATION
•••••••••••••••••••••••••	• • • • • • • • • • • • • • • • • • • •	•••••
Related		•••••
•••••••••••••••••••••••••	••••••	•••••
Unrelated		••••
• • • • • • • • • • • • • • • • • • • •		
Past History (specify)	•••••	
••••	••••••	••••
Previous Admissions + Ca	use	
		••••
Previous Treatments (+ D	uration)	
Drugs (specify)	••	
Transfusion (No)		• • • • • • • • • • • • •
Operations		•••••
Social/Family (h/o) (Re	levant)	• • • • • • • • • • • • • • • • • • • •
	•••••••	• • • • • • • • • • • • •
Diet	••••••	•••••
	••••••	
Siblings Health		

CLINICAL ASSESSMENT:

Gen	eral condit:	ion (spec	ify)			
Pal	lor	'	Тетр		edema	••••
Nai	ls	• • • • • • • • • • •	Tongue	N	odes	••••
cvs	: In Failure	B	Lesion		•••••	
Che	st S:	igns (spe	cify)		• • • • • • • • • • • • •	••••
PA.	Liver (size	e)		Ascit	es	• • • •
	Spleen (si	ze)		Other	•••••	••••
OTH	ER FINDINGS					
CLI	NICAL DIAGNO	OSIS OR PI	RESENTATI	ON		
INV	ESTIGATIONS	DONE :				
<u>A</u>	ROUTINE :	(1)	(2)	(3)	(4)	
	НЪ	• • • • • • • •				• • • •
	PCV	••••	• • • • • • • • •	• • • • • • • • • •		• • • •
	MCHC	• • • • • • • •		• • • • • • • • • • •	••••	• • • •
	WBC Tota	1		• • • • • • • • • • •		• • • •
	Differenti	ial			• • • • • • • • • • • • •	
	Film repor	rt		•••••	••••	
	Reticulocy	te count.		• • • • • • • • • • •	•••••	• • • •
	Malarial p	oarasi tes	••••			• • • •
	Stool		••••			• • • •
B	SPECIAL (OPI	TIONAL)				
	Urea/Elect	troytes:		Hb.	Electrophores	sis
	BONE MARC	W				
	Serum prot	teins	(1)	(2) .	(3)	
	G-6-P.D		•••••			• • •
	Coombs Tes	st	•••••	CX	Ray	
	Urinalysis	8	••••	LF	Гв	•••
	FINAL DIAC	NOSIS				

MANAGEMENT

.

TRANSFUSION:;	PACKED/WHOLE	(Nos)
(on)		
	AMOUNT	•••••
HAEMATENICS	•••••••••••••••••••••••••••••••••••••••	•••••
SPECIFIC (for Pathology)	•••••
ANY OTHER (specify)	•••••••••••••••••••••••••••••••••••••••	•••••
FOLLOW UP		

Date

Hb

RETICS

WBC/PLTS

COMMENT

APPENDIX II

Distribution of Patients According to Age

Age	0-1	>1 -2	>2 -3	>3 -4	>4 -5	>5 -6	>6 -7	>7 -8	>8 -9	>9 -10	>10 -11	>11 -12	>12 -13	Total
Number of Patients	96	54	33	23	19	15	9	9	7	14	9	4	2	294
Percentage	32.7	18.4	11.2	7.8	6.1	5.1	3.1	3.1	2.4	4.8	3.1	1.4	0.7	100

APPENDIX III

.

Age Distribution of Patients According to Tribe.

	AGE - RANGE IN YEARS												
Tribe	0-1	>1-2	>2-3	>3-4	> 4-5	> 5-6	> 6-7	>7-8	>8-9	> 9-10	> 10-11	> 11	Total
Luo	57	31	21	13	9	5	1	3	0	5	6	2	153
Luyia	24	17	4	4	6	4	4	1	1	2	2	0	69
Kikuyu	8	4	6	3	3	4	2	2	2	2	0	2	38
Kamba	3	1	1	3	0	0	2	2	3	3	0	1	19
Kalenjin	2	0	1	0	0	0	0	1	1	1	1	0	7
Others	2	1	0	0	1	2	0	0	0	1	0	1	8
Total	96-	54	. 33	23	19	15	9	9	7	14	9	6	294

APPENDIX 1V

٠

Levels of Haemoglobin

Haemoglobin level (gm%)	≤ 3.0	3.1-4	4.1-5	5.1-6	6.1-7	7.1-8	8.1-9	9.1-10	≥ 10	Void	Total
No. of Patients with	19	36	64	58	52	36	13	8	2	6	294
Percentage	6.5	12.2	21.8	19.7	17.7	12.2	4.4	2.7	0.7	2.0	100