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DEPARTMENT OF PAEDIATRICS AND CHILD HEALTH

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TOPIC: A STUDY ON MENTAL RETARDATION IN CHILDREN IN DAR ES SALAAM

A DISSERTATION IN PARTIAL FULFILMENT OF THE REQUIREMENT FOR THE DEGREE OF MASTER OF MEDICINE IN PAEDIATRICS AND CHILD HEALTH

BY

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DECLARATION

I hereby declare that this dissertation has not been submitted for a degree in any other university.

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ABBREVIATIONS

C.A. - Chronological age

gm - Grams

H.A. - Horizontal axis

I.Q. - Intelligence quotient

Kg. - Kilogram

M.A. - Mental age

M.Q. - Mental quotient

N.S. - Not significant

S. - Significant

S.D. - Standard deviation

UNESCO - United Nations Educational, Scientific and Cultural Organisation.

V.A. - Vertical axis

WHO - World Health Organisation

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SUMMARY

A study of mental retardation in children in Dar es Salaam was done from December 1986 to October 1987. Children covered were those attending Uhuru Mchanganyiko Primary School and Mtoni Deaconic Lutheran Centre. Others were those at Young Women Christian Association, Mother Thereza's Home at Mburahati and Kurasini Children's Home. Included also were those admitted or attending clinics in Muhimbili Medical Centre.

The objectiveswere to determine the developmental and clinical patterns of mentally retarded children. It was also aimed at determining the intelligence quotient and deliniating various aetiological factors in the mentally retarded children.

A total of 55 mentally retarded children with their controls matched for age and sex were studied. There were 29 males and 26 females.

The study group was found to be significantly developmentally delayed. Mean developmental age of study group was 42.98 months compared to 123.4 months for the control group. This was significant, $p < 0.001$.

The mentally retarded children had abnormal behaviour and clinical features. These aided in the clinical diagnosis.

The intelligence quotient was determined by psychometric testing. It was low in the study group with a mean of 37.4 compared to 119.6 in the controls.

The aetiological factors seen were basically preventable. The commonest were due to postnatal causes which accounted for 43.6% followed by prenatal causes 21.8%. Convulsions with fever were highest single cause accounting for 41.8% of the cases. Maternal age over 35 years old also was a significant associated factor, $p = 0.45$, sign test.

Birth weight and duration of labour were not statistically significantly associated with mental retardation in this study, $p = 0.41$ and $p = 0.09$ respectively. Birth order was also noted to have no significancy, $p = 0.43$.

1. INTRODUCTION AND REVIEW OF LITERATURE

1.1. DEFINITION

Mental retardation is not a disease or a single entity. It is manifested by slow, incomplete maturation, impaired learning ability and poor social adjustment which happens during developmental period.[1,2,3,4] The commonly used definition is that by the American Academy of Mental Deficiency (AAMD) which states that mental retardation is significant subaverage general intellectual functioning existing concurrently with deficits in adaptive behaviour and manifest during developmental period.[5] This is further explained as a score on the standard tests that would be lower than that obtained by 97 to 98 percent of persons of same age meaning that certain individuals fail to meet standards of indipendence and social responsibility expected of the age and cultural group. There is failure to learn basic academic skills and participate in appropriate social group activities. All these should be observable during childhood. Problems of similar nature manifested only in adulthood would be classified as mental illness, not mental retardation.[5] It should be pointed out that what is being stressed in this definition is adaptive behaviour, intelectual subnormality and developmental period.[5,6]

In the past various synonyms of mental retardation have been used like mental subnormality, mental deficiency, mental handicap,

mental deficit and amentia.[2,6,7,8,9,10,11] However, mental retardation has gained more use in the United States of America than in England where mental subnormality is still maintained.[2,6] Intelligence has always been difficult to define. When grouped together, four acceptable classes of definition have always emmeged namely biological, psychological, operational and developmental.[12] Other basic approaches used to define intelligence have been based on intelligence being ability to learn or being what has been learned and also being the ability to adapt to the environment. It has also been taken as an ability to carry on abstract thinking or regarded on global terms.[13]

Binet and Simon while developing their intelligence scale considered the nature of intelligence as having goal direction to mental process involved. They also considered it to involve the ability to show adaptive solution and selectivity of judgement and critism of choices. Intelligence as defined by Wechsler in 1949 as being aggregate of global capacity of an individual to act purposefully to think rationally and to deal effectively with the environment is still a valid and useful definition.[14] Intelligence is therefore the capacity to comprehand relations, the ability to think and solve problems and to adjust to new solutions.[15]

1.2. HISTORICAL BACKGROUND OF INTELIGENCE QUOTIENT (I.Q)

Mental testing dates back to 1882 when Galton was able to measure individual behaviour after having established the "Anthropological laboratory". However, he was only able to measure functions of sense namely sensory, acuity, hearing, sight and temperature.[12,14,16] In 1890 Catell appeared to have been the first to use the words "mental test". He still tested sensory-motor functions or perceptual tasks and occassionally memory.[12,14,16] In 1892 Kraepelin's tests were set to measure various mental activities in psychiatric patients. This was the first time psychological methods were used in clinical work.[12]

Binet in 1895 designed 30 tests for measurement of intelligence. These included verbal items. Together with a fellow French psychologist, Simon were requested by the ministry of education to devise a simple, practical and objective way of identifying children who were unable to benefit from ordinary elementary education. The testing included performance of different functions at each age level. The performance of every child was scored according to his age level of performance the results of which was mental age (M.A.). The M.A. was then compared with the chronological age (C.A.) so as to determine the overall performance. The results showed some degree of retardation in mental development if the M.A. fell below the C.A. If the M.A. was above the C.A. it indicated that there was acceleration of development. Binet and

Simon revised the first set of tests thereby increasing the number of tests which were grouped into age levels. In 1911 a final Binet-Simon Intelligence Scale was given. Mental age performance remains Binet's most significant contribution. The scale was translated in English and revised by Terman in 1916 at Stanford in United States of America with the English version being known as Stanford-Binet. Later revisions were undertaken by Terman and Merrill.[12] Later on a German psychologist Wilhelm Stern thought it more useful to express the rate of mental development in terms of the ratio between M.A. and C.A. and referred to it as mental quotient (M.Q.).[14] Terman subsquently introduced the concept of intelligence quotient (I.Q.) which is derived by dividing M.A. by C.A. multiplied by one hundred.[12]

$$I.Q. = \frac{M.A.}{C.A.} \times 100$$

Others who contributed to the above developments were Boaz in 1891 who developed immediate memory span and Gilbert in 1893 who developed mental capacity testing.[17] Thus the measure of subaverage general intellectual functioning is I.Q. which is an index of intelligence determined through objects answers to arbitrary chosen questions.[15] There has been more development in establishing more psychometric tests. The commonly used ones are the Stanford-Binet Intelligence Scale and Wechsler Scale for children or the revised versions.[18,19]

1.3. CLASSIFICATION

Mental retardation classification has met many changes. Previous ones which used the terms idiot, imbecile and moron have fallen out of use.[7,8,9] Classification into four categories has been given. In this classification I.Q. score of 50 - 70 shows mild mental retardation while that of I.Q. 35 - 49 indicates moderate mental retardation. Severe mental retardation is of the I.Q. 20 - 34 and that of below 20 is profound mental retardation.[20]

Classification according to AAMD is similar but with different cut off points. Both Stanford-Binet and Wechsler levels are used. Mild mental retardation for Stanford-Binet Scale is I.Q. 52 - 69 while that of Wechsler is I.Q. 55 - 69, moderate mental retardation is 36 - 51 against that of I.Q. 40 - 54 for Wechsler Scale, severe mental retardation is I.Q. 20 - 35 for Stanford Binet compared to I.Q. 25 - 39 for Wechsler. Profound mental retardation is below I.Q. 19 for Stanford Binet and below I.Q. 24 for Wechsler Scale.[16] Other studies have classified mental retardation into mild and severe only. Those with I.Q. above 50 but up to 70 are mildly mentally retarded and those with I.Q. below 50 are severely mentally retarded.[21,22]

The classification by Hutchison[7] seems to be more functional and is going to be used in this study. The scores are that I.Q. 50 - 70 means mild mental retardation while the I.Q. 25 - 50 is

moderate mental retardation. The I.Q. below 25 is severe mental retardation. However, the overlap will be corrected to I.Q. 50 - 70, I.Q. 26 - 49 and 0 - 25 accordingly.

1.4. EPIDEMIOLOGY

This is the first study of mental retardation with psychometric testing in Tanzania. The prevalence of the problem is therefore not clearly known. However, it was estimated in 1985 that there were approximately 163,000 mildly and 24,000 severely mentally retarded children respectively.[23] Okeahialam in Dar es Salaam while reviewing the handicapped children in 1974 found that out of 208 children, 60 (28.7%) had mental retardation.[10] Studies done in Singapore, China, Kenya and Nigeria have shown that mental retardation is a public health problem in developing countries.[11,24 25,26,27] Given that the causes related to low socioeconomic status like communicable diseases and accidents are on the increase,[8] the prevalence is bound to be high. The situation is made worse by rudimentary obstetric services in the developing countries.

The prevalence of mental retardation in developed countries is variable, ranging from 2 - 3.8 percent.[5,28] The variation may not be real. The differences occur both from the psychometric tests used and cut off points. For example Stanford Binet Intelligence Scale has a standard deviation of $\pm$ 16 while that of

Wechsler Scale is ± 15. This will give a variation in prevalence.[16]

Other variations in prevalence can be ascribed to different ways used in determination of adaptive behaviour in different locales that have different proportions of social classes. Given that mild mental retardation is associated with lower social classes, this will affect prevalence.[5]

Differences in prevalence due to sex have also been reported. Various explanations for this variation are that the male fetuses and neonates are more prone to various types of trauma like premature delivery, brain damage and as they grow are more susceptible to many environmental hazards than females. Psychologically parents treat boys differently from girls.[6]

Noted also has been differences in prevalence due to different ages; the lower age groups have less number of the mentally retarded. The preschool age group has a lower prevalence which increases during the school age and declines in those aged 16 years and above.[6]

Variations due to past and present studies has been noted. The old prevalence rates were lower than the present ones and this has been due to the widening of the knowledge in the field with more elaborate definition of the problem.[6]

The aetiology of mental retardation can be due to prenatal, perinatal and postnatal causes. Prenatal causes are either due

to structural abnormalities of the brain, genetic disorders or infections especially those in the first trimester. Congenital malformations are a common cause of mental retardation. They feature most in developed countries where infectious diseases have been controlled.[29]

Down's Syndrome, first described by John Langdon Down in 1896 is the commonest of the genetic abberation associated with mental retardation. The commonest form of Down's Syndrome is trisomy 21 which is due to non-dysjunction of chromosome 21 followed by translocation of the D/G group and lastly mosaism.[16,30] Trisomy 21 is age dependent.[5,16]

Of the prenatal factors, Down's Syndrome is the commonest cause of mental retardation followed by fragile X syndrome.[31, 32,33.] Fragile X syndrome has got a variety of clinical features which make it easy for clinical diagnosis. These include large protruding ears, prominent forehead, prognathism, delayed speech development, macro-orchidism and mild to moderate mental retardation.[31,34,35]

Infections in the first trimester are dangerous. They lead to structural damage of the brain. The most notorious organisms are viruses like cytomegalovirus, rubella and herpes viruses. Toxoplasmosis also occurs. Their clinical manifestations are similar.[16,36,37,38]

Disorders of metabolism due to enzyme deficiency lead to mental retardation. The enzyme lack can be partial or complete. Enzymes involved can be those for protein, fat or carbohydrate metabolism. They result into different clinical forms of diseases like phenylketonuria, homocystinuria, Gauchers disease, Nieman Pick disease, Hurler's and Hunter's Syndrome.[1,5,16,39]

Maternal chronic alcoholic consumption has been shown to lead to structural damage of the brain of the newborn babies.[5,40,41]

Perinatal causes of mental retardation are mainly related to the management of delivery. Birth asphyxia with subsquent hypoxia is an import ant cause of mental retardation. There is necrosis of the neurons. In addition to mental retafdation, there are motor deficits and seizure disorders.[16,42,43,44,45] Other perinatal causes are those due to hyperbilirubinaemia caused by haemolytic diseases of the newborn. The effect is mainly on the basal ganglia and the defect is associated motor and sensory disorders.[11]

Causes of mental retardation in the postnatal period are due to severe complications of infections. These can be bacterial, viral or protozoal leading to meningitis, encephalitis and cerebral malaria.[11,46]

Tanzania has no existing law which protects the mentally retarded. WHO inacted such a legislation protecting the individuals against cruelity and economic exploitation. Other aspects of legislation involve education and guardianship. Other countries

like Great Britain and United States of America have been constantly reviewing their laws so as to properly cater for the mentally retarded.[2,9,47,48]

2. OBJECTIVES

2.1. Broad objective

The main objective of the study was to determine the clinical and epidemiological pattern of mental retardation as seen in Dar es Salaam.

2.2. Specific objectives

2.2.1. To determine the developmental pattern of mentally retarded children.

2.2.2. To delineate the clinical features of mentally retarded children.

2.2.3. To determine the intelligence quotient of mentally retarded children.

2.2.4. To delineate various aetiological factors in mental retardation.

3. METHODOLOGY

The study was carried out in Dar es Salaam from December 1986 to October 1987. The study group was of children attending Uhuru Mchanganyiko Primary School, Mtoni Lutheran Deaconic Centre and Young Women Christian Association. Others were those at Mother Therezia's Home at Mburahati, Kurasini Children's Home and children admitted or attending clinics in Muhimbili Medical Centre. The criteria for inclusion in the study was delayed development in comparison to previous siblings or age-mates and abnormal behaviour like aggressiveness, destructiveness or passiveness.

The diagnosis of mental retardation was based on clinical grounds. No laboratory investigations were done for genetic disorders, infections or electroencephalography except for one child who had his karyotyping done abroad. Other studies have also been reported on the same clinical basis.[25,26,27] Knowledge of the stage of onset of mental retardation helps in reducing the large number of special investigations.[49]

The C.A. ranged from 2 - 15 years and the consent was obtained from parents or guardians. Every subject was matched for age and sex with a control who was selected from Uhuru Mchanganyiko Primary School and Muhimbili Medical Centre. Age was matched by calibre method whereby at any given age in years, a range of 6 months can include a child above or below that given age in years. Thus age of 4 years can be used to match those who are between $3\frac{1}{2}$ and $4\frac{1}{2}$ years.

All the children were screened developmentally in order to assess the developmental retardation. The assessment was made in four areas according to Gesell.[50] In motor behaviour, the children were assessed for motor development. Parents were interviewed about developmental stages like neck control, sitting, crawling, standing, walking and manipulation of objects. For fine motor adaptive development they were screened for eye-hand corodination and exploration, transfer of objects and drawing of simple diagrams. In the field of language parents were asked to give ages when children had achieved facial expressions, gestures, vocalisation, comprehension and saying meaningful words. They were lastly screened in social behaviour for the of attainment of self feeding, toilet training, play, response to training and emotions.

Physical examination was done in all children. Clinical features were recorded and these in addition to the findings of developmental retardation aided in clinical diagnosis. The features of chromosomal disorders, dysmorphism, cerebral palsy and epilepsy were used. Anthropometric measurements, weight, height and head circumference were taken for all children.

The psychometric test which was used in this study was the Stanford-Binet Intelligence Scale.[18] The tests are set at different age levels from age level of 2 years to superior adult. Every year level has got a number of subtests and at the end of every year level there is an alternative test which is administered when one of the subtests has been spoiled.

Testing was carried on up to an age level when a subtest was missed. The preceding age level to the one that a mistake was made, that is a year level that all the subtests were correctly scored was taken as the basal age. The testing continued till the child missed all the subtests. The age level at which all the subtests were missed was taken as the ceiling age. The basal age in years was recorded. The subsquent scored subtests were given credits in months which varied according to year levels. The credits were added up and then divided by 12 to get the credit in years which were subsquently added to basal age and the sum gave the M.A. The scored M.A. and C.A. were used to compute I.Q. scores from the Pinneau Revised tables of I.Q.[18] The materials used in testing were according to intelligence scale used. These included pictures of various objects and animals, dolls, blocks, beads and toys. Tests for adaptive behaviour were not done in this study.

Information about prenatal, perinatal and postnatal history was obtained by interviewing parents and or examining records when these were available. Prenatal history was obtained from 44 mothers of the study group, there were 11 orphans for whom no records were available. The history included maternal diseases, use of drugs and exposure to irradiation. Others were alcohol ingestion, smoking and maternal age at the time of conception.

Perinatal history included duration of labour, mode of delivery and activity of the child soon after delivery like immediate cry, colour of the baby and whether any rescustative measures were done. Apgar Score was obtained from records whenever possible. Neonatal history included jaundice together with early neonatal convulsions.

History leading to postnatal causes was obtained from parents or guardians. Age at which fever and convulsions, trauma, measles, severe diarrhoea and dehydration occured were recorded. Hospital records were used when they were available for the results of blood slide for malaria parasites and the results of cerebral spinal fluid examination. These disease events were mainly due to cerebral malaria, meningitis or measles. When there were no records available the cause of fever and or convulsions was regarded as unspecified.

Social history of parents was recorded. This included education, occupation and knowledge about the existence of mental retardation in the particular child.

Sign test was used for analysis of birth order, duration of labour, birth weight and advanced maternal age while t test was used for developmental screening and anthropometric measurements.

4. RESULTS

During the period of study, a total of 55 mentally retarded children were examined. Twenty nine were males and 26 were females giving a male/female ratio of 1.1:1.

The mean age was 9.9 years, 63.6% of the study group was made up the age group 9 - 14.

Table I: Age and sex distribution of the study population.

AGE GROUP/ YEARS	STUDY GROUP			CONTROL GROUP		
	MALES	FEMALES	TOTAL	MALES	FEMALES	TOTAL
4	3	0	3	3	0	3
5 - 8	6	4	10	6	5	11
9 - 12	10	14	24	12	13	25
13 - 15	10	8	18	8	8	16
TOTAL	29	26	55	29	26	55

In this study the classification of causes of mental retardation were prenatal 21.8%, perinatal 20.0%, postnatal 43.6% and unknown 14.5%. Postnatal group of causes were the commonest.

Table II: Distribution of causes of mental retardation

CAUSE	FEMALES	MALES	TOTAL	%
Congenital abnormalities	5	7	12	21.84
Birth asphyxia	4	6	10	18.82
Hyperbilirubinaemia	0	1	1	1.82
Cerebral malaria	6	4	10	18.82
Encephalitis	2	1	3	5.46
Meningitis	1	0	1	1.82
Convulsions of unspecified causes	2	7	9	16.38
Trauma	1	0	1	1.82
Unknown	5	3	8	14.54
Total	26	29	55	100.10*

*Rounding errors

The prenatal causes of mental retardation were due to congenital abnormalities. These were Down's Syndrome 7, trisomy 4, 1, neurotubular defect 1, congenital rubella syndrome 1 and congenital abnormalities of unknown causes 2. There were also some prenatal factors such as drugs, alcohol intake and smoking which were noted in the study but had no adverse effects on the offsprings. The children were born normal and mental retardation was due to other causes in the perinatal and postnatal life.

Maternal diseases were noted in 8 mothers. These included hypertension 1, malaria 2, anaemia of pregnancy 2 and hypertension of pregnancy 3. Two of the mothers admitted to have taken alcohol during the pregnancy on the average of 1 litre per month. The drugs used by the mothers during pregnancy were mainly the routine drugs which were given at the antenatal clinics. In addition to this, Diazepam and Aldomet were given due to hypertension and Imferon for anaemia. There were none who smoked cigarettes and there was no exposure to irradiation.

Perinatal factors which caused mental retardation were birth asphyxia 10 and hyperbilirubinaemia 1 who developed jaundice soon after birth. No exchange blood transfusion was done. Three other children developed jaundice. One developed jaundice 84 hours after birth, the other one 144 hours later while the fourth was after 22 days. These did not suffer the consquences of hyperbilirubinaemia. The perinatal causes associated with mental retardation are shown in Table III.

Table III: Perinatal attributable factors for mental retardation

FACTOR	FEMALES	MALES	TOTAL	DEVELOPED MENTAL RETARDATION	
				n	%
Birth asphyxia	4	6	10	10	100
Hyperbilirubinaemia	2	2	4	1	25
Prolonged duration labour	1	2	3	0	-
Low birth weight	4	4	8	0	-

Birth asphyxia and hyperbilirubinaemia led to mental retardation in 100% and 25% respectively. Low birth weight and prolonged duration of labour were not found to be associated with mental retardation. Those who developed hyperbilirubinaemia or were delivered after prolonged labour or were born with low birth weight but had no complications are included here so as to show factors noted in the study group which were expected to be associated with mental retardation but they did not contribute to mental retardation. These children later developed mental retardation due to other causes. In this study the duration of labour did not significantly contribute to mental retardation, $p = 0.09$. There were 4 males and 4 females in the study group

and 1 in the controls with low birth weight. Birth weight was found to be insignificant in this study in causation of mental retardation, $p = 0.41$.

Birth order showed that 30 in the study group were delivered in the first 3 parities while it was 37 in the control group. It was an insignificant associated factor in the causation of mental retardation, $p = 0.43$. Later maternal age of 35 years and above was noted in 9 mothers and 14 fathers in the study group while there were 4 fathers in the control group. This maternal age was found to be a significant cause of mental retardation in this study, $p = 0.049$.

Postnatal causes which featured most in the study were childhood convulsions. These were preceded by fever. They occured during the developmental period of the brain, leading to neurological complications and thus mental retardation.

There were 23 (41.8%) children in the study group who had convulsions. No convulsions were reported in the control group. Among these, meningitis was 1, cerebral malaria 10, encephalitis 3 and convulsions of unspecified causes 9. The onset of convulsions was before 24 months of age in 17 of these children, 36 months in 4 and 60 months in 2 children showing that the insult was during brain development.

Another postnatal cause which led to mental retardation was head injury which was seen in 1 child. She had motor accident and recovery was later complicated by mental retardation. Both perinatal and postnatal causes are largely preventable cause of mental retardation.

Socioeconomic status has been said to be associated with mental retardation. In this study occupation of parents was examined and showed a cross-section of occupations as they are available in Dar es Salaam.

Table IV: Parental occupation in the study group in comparison to the control group

OCCUPATION	STUDY GROUP				CONTROL GROUP			
	MOTHERS		FATHERS		MOTHERS		FATHERS	
	n	%	n	%	n	%	n	%
Unemployed	21	38.2	0	0	34	61.8	0	0
Self employed	6	10.9	16	29.1	2	3.6	17	30.9
Employed: goverment/parastatal/private	17	30.9	28	50.9	19	34.6	38	69.1
Not questioned	11	20.0	11	20.0	0	0	0	0
Total	55	100.0	55	100.0	55	100.0	55	100.0

Level of education has some bearing on socioeconomic status and knowledge about diseases. Those who received secondary education and above had less numbers of mentally retarded children in comparison to other levels of education.

Table V: Level of education of parents in the study

LEVEL OF EDUCATION	STUDY GROUP				CONTROL GROUP			
	MOTHERS		FATHERS		MOTHERS		FATHERS	
	n	%	n	%	n	%	n	%
None	6	10.9	5	9.1	6	10.9	3	5.5
Adult	1	1.8	1	1.8	0	0	0	0
Primary	27	49.1	20	36.4	41	74.5	24	43.6
Secondary +	10	18.2	18	32.7	8	14.6	28	50.9
Not questioned	11	20.0	11	20.0	0	0	0	0

Knowledge about mental retardation by parents was tested. In the study group 34 (61.8%) parents knew that their children were mentally retarded. Among these, 25 parents had discovered this by the age of 4 years. Ten (18.2%) parents did not know that their children were mentally retarded. Parents who were not questioned were 11. None of the control parents thought their children were mentally retarded.

Abnormal behaviour is found in the mentally retarded children. Sometimes this is the first indication of mental retardation. In this study a few behavioural changes were noted in the study group by parents. None of these were noted in the control group.

Table VI: Abnormal behaviour as seen in the mentally retarded children

Abnormal behaviour	Female	Male	Total
Aggressiveness	8	11	19
Hyperkinetic	9	14	23
Passive	14	10	24
Destructive	11	15	26
Abbusive	10	12	22

The mentally retarded children present with some typical clinical features which aid in the clinical diagnosis. This is more evident for older children. These were not seen in the control group. The following table summaries these clinical features.

Table VII: Clinical features as seen in the mentally retarded children.

Clinical Feature	Female	Male	Total
Drooling of salive	7	9	16
Grinding teeth	8	7	15
Destructiveness	10	12	22
Self preoccupation	11	18	29
Forgetfulness	13	14	27
Hydrocephalus	1	1	2
Microcephalus	13	11	24

Noted also were cerebral palsy 19, chromosomal abnormalities 8 and epilepsy 14 mentally retarded children.

In this study, weight, height and head circumference were studied. The aim was to assess the nutritional status of the children. The results in the following table show that there was significant difference between the study group and the control group. This indicates that the control group was better nourished than the study group.

Table VIII: Weight, height and head circumference measurements

Anthropometric Measurements	$\bar{X} \pm 2SD$		t	p value	Remark
	Case	Control			
Height (cm)	125.7 ± 38.2	133.6 ± 38.2	2.17	$p < 0.05$	s
Weight (kg)	25.6 ± 18.4	30.3 ± 18.6	2.66	$p < 0.05$	s
Head circumference (cm)	50.7 ± 7.2	53.2 ± 3.4	4.65	$p < 0.01$	s

Head circumference is on anthropometric measurement which mainly shows brain growth. It shows either normal head size or abnormal size in the form of microcephalus or hydrocephalus. In this study the abnormal head size was significantly associated with mental retardation, $p < 0.01$.

Developmental screening was done in both the cases and controls. It showed that there was a significant difference in the later developmental milestones whereas the difference was insignificant in the early milestones.

Table IX: Developmental screening in months

Milestones	$\bar{X} \pm 2$ S.D.		t	p value	Remark
	Case	Control			
Smilling	2.4 ± 9.6	1.6 ± 1.22	1.28	$p > 0.05$	NS
Neck control	5.54 ± 16.36	3.31 ± 0.94	2	$p > 0.05$	NS
Sitting unsupported	8.8 ± 21.8	5.7 ± 1.12	2.1	$p < 0.05$	S
Pulling to sitting from supine	8.6 ± 24.4	6.1 ± 0.60	1.5	$p > 0.05$	NS
Transfer of objects	4.1 ± 17.50	5.9 ± 3.0	1.5	$p > 0.05$	NS
Babbling	7.9 ± 27.4	5.9 ± 1.7	1.1	$p > 0.1$	NS
Mouthing of objects	4 ± 11.0	6.87 ± 2.0	3.78	$p < 0.01$	S
Bearing weight on both legs	11 ± 26	8 ± 0.54	1.76	$p > 0.05$	NS
Standing holding on furniture	14.7 ± 31.2	8.9 ± 2.24	1.3	$p > 0.2$	NS
Waving bye bye	6.1 ± 21.0	10.2 ± 2.16	2.86	$p < 0.01$	S
Saying words with meaning	27.4 ± 65.4	12.0 ± 1.4	3.54	$p < 0.01$	S
Standing alone	19.4 ± 35.6	12.6 ± 2.16	2.83	$p < 0.05$	S
Walking	23.8 ± 39.0	14.7 ± 3.6	3.4	$p < 0.001$	S
Taking off shoes	33.8 ± 72.1	20.4 ± 7.6	2.73	$p < 0.01$	S
Feeding self	33.81 ± 58.0	22.8 ± 5.4	2.82	$p < 0.01$	S
Walking backwards	8.8 ± 38.4	26 ± 9.4	6.75	$p < 0.001$	S
Dry at night	34.3 ± 62.6	29.1 ± 6.8	1.2	$p > 0.2$	NS
Obeying four simple comands	41.8 ± 73.4	26.5 ± 5.96	3.1	$p < 0.01$	S
Walking on tip toes.	4.36 ± 31.8	27.75 ± 9.6	10.43	$p < 0.001$	S
Unbuttoning front buttons	55.1 ± 96.2	35.5 ± 21.74	2.95	$p < 0.01$	S
Singing songs	49.3 ± 88.6	34.8 ± 23.14	2.35	$p < 0.05$	S
Going to toilet without help	52.1 ± 81.6	34 ± 16.6	3.12	$p < 0.01$	S

Developmental age assessment showed that there was considerable delay in overall development of the mentally retarded children in comparison to the control group. Mean developmental age for the study group was 42.9 months while that of the control group was 123.4 months. This was found to be significant, $p < 0.001$.

The children underwent psychometric testing. The study group scored low I.Q. levels compared to the control group. The average I.Q. for the study group was 37.6 while that of control group was 119.4.

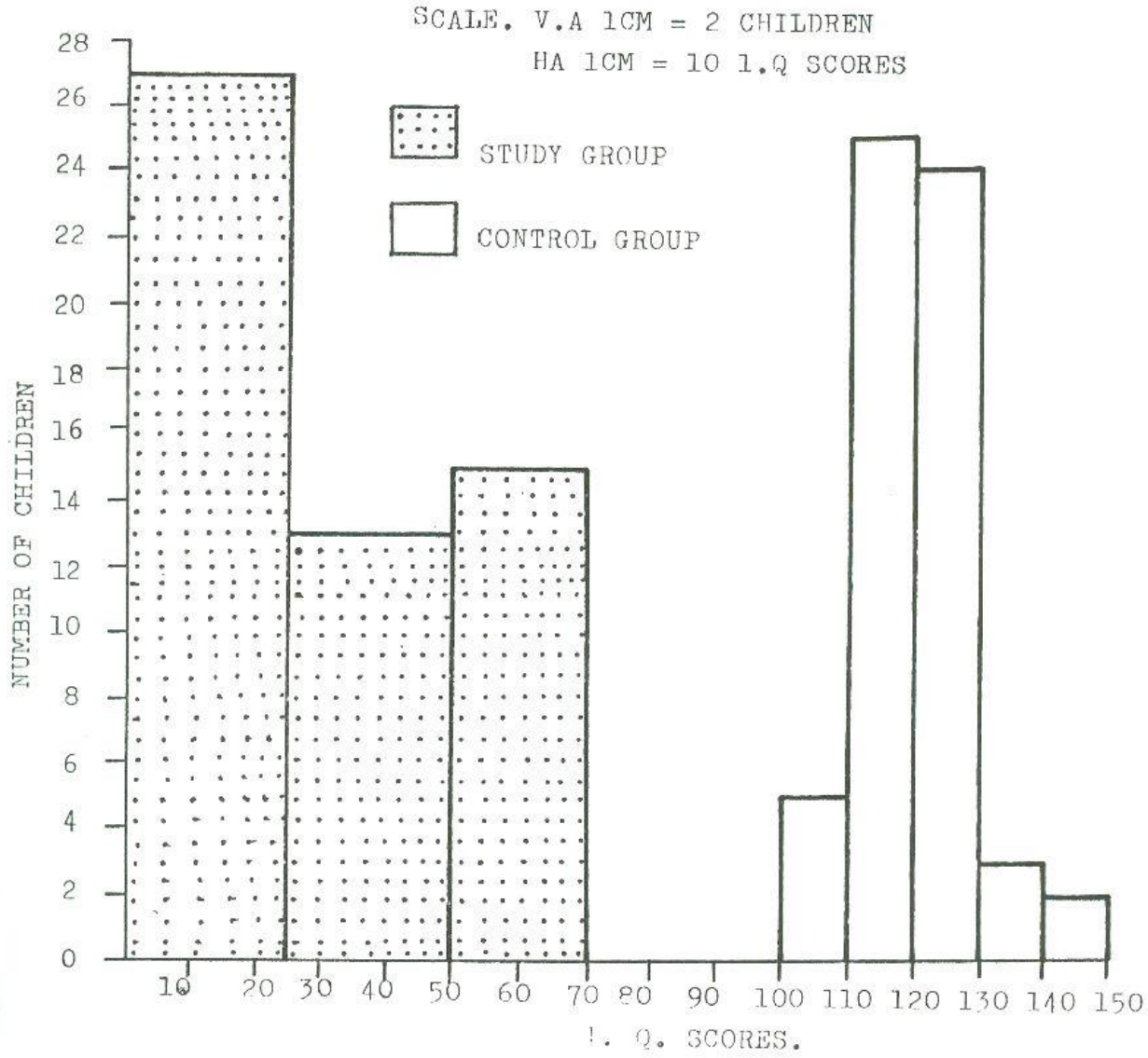
Table X: I.Q. scores after psychometric testing

I.Q. Score	Study group	Control group
0 - 25	27	0
26 - 49	13	0
50 - 70	15	0
100 - 109	0	5
110 - 119	0	23
120 - 129	0	22
130 - 139	0	3
140 - 149	0	2
Total	55	55

There were 27 severely mentally retarded, 13 moderately mentally retarded and 15 mildly mentally retarded children.

When these results are presented in a histogram, the study group shows skewed distribution on the right while the control group has normal distribution (see Figure 1).

FIGURE 1. FREQUENCY DISTRIBUTION OF I.Q. SCORES OF STUDY AND CONTROL GROUPS DEPICTED IN HISTORAM



There were 14 mentally retarded children who had earlier attended normal schools. Their performance in school was reported by teachers as having been below normal. When these were compared with the performance of 43 children in the control group who were at school it was found out that all the 14 were below normal. In the control group there was 1 child below normal, 36 in the average group and 6 were above average performance. This further indicates mental retardation.

5. DISCUSSION

Mental retardation is of world wide public health importance. It is the most common associated handicap of different notable neurological syndromes which include cerebral palsy, hydrocephalus, epilepsy, severe visual failure or deafness.[22,51] With these handicaps they therefore need help and protection from the community.[2,9,47,48] In order to help them, their existence must be known. In Tanzania, it was estimated in 1985 that there were 163,000 mildly and 24,000 severely mentally retarded children.[23]

This study included 55 mentally retarded children and controls matched for age and sex. There were 29 males and 26 females. Other studies have shown male preponderance.[22,51] This study does not show the true male preponderance probably due to the fact that children who were seen were mostly at social service centers such as schools or hospitals.

The male preponderance has been due to the fact that the males are more susceptable to both birth trauma and trauma due to physical environment.[6] The X linked mental retardation which is due to inheritable site near the tip of the long arm of the X-chromosome has added to more male preponderance. As chromosomal analysis was not done, the types of chromosomal abberations could not be known.

Classification of causes of mental retardation were prenatal 21.84%, perinatal 20.02%, postnatal 43.6% and unknown were 14.54%; the commonest being postnatal group. Findings in Nigeria showed that prenatal were 29.3%, perinatal 8.6%, postnatal 9.5% and unknown, being the commonest were 52.6%.[27] A study done in Israel showed that the commonest group of causes was prenatal 43%, others were perinatal 12%, postnatal 17% and unknown were 28%.[49] The finding of more postnatal causes in this study was due to a large number of attacks of fever and convulsions. Almost all of these attacks were associated with severe neurological factors. Among the 11 who were not questioned, 3 had a clinical diagnosis of Down's Syndrome. The low number of unknown in the study compared to the Nigerian study was that detailed history and physical examination was done to establish the cause of mental retardation. There were low causes of prenatal group because no laboratory investigations were done as opposed to the findings in the Israel where these were carried out.

Prenatal group of causes noted were Down's Syndrome 7, trisomy 4, 1 congenital rubella syndrome 1, neurotubular defect 1, and abnormalities due to unknown causes 2. This figure is lower than what was expected in a large population of Dar es Salaam which was estimated to be 1.5 million people in 1967. Children with congenital abnormalities are mainly kept indoors for fear of being run over by vehicles, destruction and above all there

is a social stigma in the families with these children. Karyotyping and other chemical investigations confirm the diagnosis. As this was not done, the figure should be low. Studies done in China[24] and Israel[49] employed these investigations and had more congenital abnormalities.

Factors leading to prenatal group of causes which were considered in the present study were maternal age at the time of conception, alcohol ingestion, maternal diseases and drugs taken during pregnancy and infections.

Young maternal age is associated with perinatal birth complications. These are seen mainly in the first and second parity with subsquent decrease in the third and fourth parieties rising again in the fifth parity.[53] Birth order was studied here and found not be a significant factor in causation of mental retardation in the study, $p = 0.43$. This might be explained by the fact that there were few mothers within the age group less than 19 years who were only 6 in the study group and 4 in the controls. The advanced maternal age of 35 years and above has been considered as being at high risk of chromosomal abberations especially trisomy 21 leading to nondysjunction. This leads to Down's Syndrome.[16] In the study there were 9 mothers in the study group who were above the age of 35 years and none in the control. This was found to be a significant factor in causation of mental retardation $p = 0.049$. If there were more mothers

with the age of 35 years and above in the study may be the figure might have been high.

There were 2 mothers who took alcohol at a rate of about 1 litre per month. This number is small and further than that the offsprings of these mothers had no adverse effects. Studies done elsehere have shown that alcohol intake during pregnancy leads to structural damage to the brain.[42,43] The effects are thought to be due to the ethanol toxicity because it crosses freely the placental barrier.

There was 1 mentally retarded child who had signs of congenital rubella syndrome. This shows an underestimate of the problems of intrauterine infection. With the high rate of infectious diseases, this should have been high. Those intrauterine infections lead to severe congenital abnormalities. These are associated with other congenital abnormalities whose severity leads to early childhood deaths thus eliminating these children early before they can be seen in community. These abnormalities due to intrauterine infections are severe if they occur in the first trimester. Studies done elsewhere have shown that the commonest organisms are viruses namely cytomegalovirus, rubella virus and herpes virus together with toxoplasmosis.[36,37,38]

Maternal diseases seen in the study together with drugs used were not associated with mental retardation. This can be explained by the fact that these were not serious enough to

lead to any adverse effects. Moreover the drugs used were those routinely given at the maternal and child health clinics which were chloroquine tablets and ferrous sulphate. There were no mothers who smoked cigarrettes and mone were exposed to irradiation.

There were 8 mentally retarded children in the study who were born with birth weight of less than 2500 gm in the study group and 1 in the control group. This given about 8.2% in this study that constitutes low birth weight. This figure is less than that is usually observed of 10 - 15% of low birth weight. This low number of those with low birth weight which is that below 2500 gm either due to premature birth of less than 37th week of gestation age or birth weight of less than 10th centile for gestational age was reflected by there being a large number of unknown birth weight. There were 29 of unknown birth weight in the study group and 14 in the control group.

Low birth weight has got several causes. Maternal diseases may lead to nutritional deficiencies in the mother and concurrently poor food stores to the growing fetus. Chronic diseases like tuberculosis, hypertension and other cardiovascular diseases are known to lead to placental insuficiency and the fetus will undergo intrauterine malnutrition.[54] As there were few mentally retarded children and the maternal diseases noted were not severe enough in this study, birth weight was found to be insignificant factor in causation of mental retardation, $p = 0.41$.

Birth asphyxia caused mental retardation in 10 children. Other studies have also reported cases of mental retardation due to birth asphyxia.[24,46] These children were reported at birth to have been generally inactive with a weak cry and some developed cyanosis and convulsions. Their development has been associated with severe motor as well as mental retardation.

Delivery of an asphyxiated baby is mainly due to mismanaged labour and delivery. Together with poor management of labour, its duration is equally important. Too short a labour period is just as dangerous as that which is prolonged over 16 hours. A newborn baby should be assessed for birth asphyxia using Apgar Score. A score of 0 - 2 is severe birth asphyxia, 3 - 4 moderate birth asphyxia and 5 - 6 mild type. A score of 7 - 10 is normal. These scores are assessed in the 1st and 5th minute. No records were available for Apgar Score. Duration of labour has an important bearing on birth asphyxia. When it is very short this constitutes precipitate labour and when it lasts over 16 hours this is defined as prolonged labour. Both extremes are dangerous. It was found not to be a contributory factor to birth asphyxia in this study, $p = 0.09$.

The newborn baby should have his weight taken and gestational age assessed. These are then used to group the newborn in the appropriate weight and gestational age group.[55] This will help in identifying the low birth weight children. These are at risk

and should receive more attention. As these are likely to be inactive and cyanotic, they later develop pathological process in the brain.[43,44]

Hyperbilirubinaemia occured in 4 children in the study group and none in the control group. It was in 1 child who developed jaundice soon after birth that neurological deficit and mental retardation was later noted. The other three developed jaundice 84 hours, 144 hours and 22 days after birth respectively. These recovered uneventifully only to encounter other causes of mental retardation in early childhood. Other studies have found more cases of hyperbilirubinaemia.[11,24,25,27,46] The cause of neonatal jaundice in this study was not known but could have been possible that for the one who developed jaundice soon after birth might have had haemolytic disease of the newborn. The 4th child who developed jaundice in the 3rd week was said to have had fever too indicating that this might have been due to septicaemia. No formal treatment was given for neonatal jaundice.

Knowledge about hyperbilirubinaemia had led to improvement of quality of life in the newborn. Formation of bilirubin first leads to indirect bilirubin which is water insoluble but fat soluble. In the liver it is conjugated becoming direct bilirubin and water soluble. Causes of neonatal jaundice are many but those due to prematurity and haemolytic disease of the newborn like Rhesus and ABO incompatibility and glucose 6 phosphate

dehydrogenase deficiency lead to indirect bilirubin. This bilirubin is deposited in basal ganglia with subsquent neurological deficits. Development of exchange blood transfusion has greatly reduced the risks of brain damage.[56] It has also been shown that careful use of phototherapy in jaundice due to prematurity can greatly reduce use of exchange blood transfusion with reduction in mental retardation.[57]

Childhood convulsions were the highest single cause of mental retardation 23 (41.8%). There were none in the control group. This makes it difficult to make a conclusion about the risk of having convulsions and developing mental retardation as a complication. This is because recovery after febrile convulsions is not complicated by neurological deficit. All of these convulsions were preceded by high fever and were generalised, tonic-clonic with loss of consciousness of variable durations. There were 10 due to cerebral malaria, meningitis 1, encephalitis following measles 3 and unspecified causes of convulsions 9. This big number of unspecified causes was due to unavailability of records. Many of these children were severely mentally retarded with some motor and seizures disorders. Cerebral palsy was prominent. This partially explains the high number of severely retarded children in the study. These convulsions occurred early in life and by the age of 4 years it had already occurred in

17 (73.9%) of those who had convulsions. This is the period of maximal brain development but there is continued brain maturation up to adolescence.

The effects of convulsions on developing brain are compounded by many other factors. The sick child is not able to eat, running into a high risk of ending up in hypoglycaemia. There is delay in seeking medical treatment while administering local herbs which do more harm than good. The overall effect is hypoxic - ischaemic effects which cause great brain damage. The organisms involved are bacteria, viruses and protozoa. More or less the same sequence of events are seen in a child who gets severe bouts of diarrhoea and severe dehydration. There is hypovolaemic shock, hypoxia and ischaemia resulting into brain damage.

There was 1 mentally retarded child who had sustained head injury following motor accident. This was most probably severe concusion and she only exhibited mental retardation without other neurological disorders. In a follow up of 36 children who had severe brain injury, 17 were found to have mental retardation. Other neurological deficits noted were hemiperesis and epilepsy.[58] Study done in Dar es Salaam on childhood accidents did not however reveal any neurological deficits.[59] Considering the perinatal and postnatal group of causes in this study, they are those which are mainly of preventable nature.

A review of occupation of the parents showed a normal trend as this was available in Dar es Salaam. Every parent was considered individually. There were more unemployed mothers in the control group 34 as opposed to 21 in the study group. One would have expected the reverse because mothers in the study group needed more time to care for their mentally retarded children. There was a slight difference in the employed group for fathers; 28 in study group and 38 in the control group while it was almost equal for mothers. Again this doesn't reflect the need for parents to devot more time for caring the handicapped children.

Considering education, one would have expected to find more of none and adult education level parents having a high number of mentally retarded children. This is the group which knows little about diseases and correct treatment. Instead there were 13 parents compaired to 75 parents in the study group who were in none and adult education and primary levels of education respectively. There was no much difference in the secondary and higher education for study and control groups. This doesn't reflect the expected result of the study group being less educated than the control group. Nevertheless, the difference in education is only expected to have an impact on causes which are easily preventable like the perinatal and postnatal group of causes. Prenatal group of causes are expected to affect the

educated as well as the uneducated unless there is a high degree of suspicion like an affected previous sibling whereby prenatal diagnosis can be done and necessary measures taken which are either to conserve or terminate pregnancy. Inspite of this, there were 34 (61.8%) parents of the study group who recognised that their children had mental retardation. Recognition was in the first 4 years in 25 (73.2%).

The abnormal and clinical features as shown in tables VI and VII were only noted in the study group and none in the control. It should therefore not be taken for granted that their presence in a child means presence of mental retardation. Most of these findings were voluntereed by the parents indicating much of their concern on their children. Destructiveness, agressiveness and hyperkinetism were worysome to the parents as often times brought the parents into quarels with neighbours after destruction of properties. Some of the children were at one stage of development locked up indoors for their control and safety. Epilepsy with its social stigma was nevertheless easily given as a problem in the mentally retarded. Teeth grinding and drooling of saliva were seen in more of the younger age groups than the older groups. Both the abnormal behaviour and clinical features aided in the clinical diagnosis of mental retardation. Some basis of clinical diagnosis has been used in other studies.[25,26,27,46]

Nutritional assessment was done in the study with the aim of comparing the study group and control group. This was not meant to relate the cause and effect of nutrition because the children were already mentally retarded and the present nutritional status just showed whether there was adequate feeding or not. The anthropometric measurements of height, weight and head circumference were matched and were all found to be significantly reduced in the study group; $p < 0.05$ for height in cm, $p < 0.05$ for weight in kg and $p < 0.01$ for head circumference in cm. There was notable microcephalus in the study group. These results indicated that food intake was low in the mentally retarded children. Defective eating was at least expected in a few with cerebral palsy due to defective chewing.

There are situations when malnutrition is thought to cause mental retardation. It is however, a complex issue since factors like ignorance and poverty operate both for malnutrition and mental retardation.[4] It is also not known whether the relationship between malnutrition in infancy and resultant intelectual function is due to malnutrition or deprivation.[60] What is precisely known are autopsy findings for those born small for gestation age who have shown decreased weight of brain in comparison to others.[61] Therefore what matters most is the nutritional status at the onset of mental retardation.

Growth is equally contraversial in the mentally retarded children. This is because in normal population physical size does not correlate with intelligence. The comparison between the mentally retarded and normal children should be in children of the same age group, hence the matched controls for age and sex in this study. The significant findings in this study were therefore for the reduced growth of the mentally retarded children. When they are matched like this any major difference should be explained and any factors interfering with growth should be sought in the mentally retarded.[62] It is with this that short stature should be cautionsly considered because it has same results as intelligence only depending on the nature of brain damage.[63] Those with fragile X syndrome present with more or less good nutritional status with big head.

Developmental milestones were assessed. There was difficulty in remembering when a child aged say 15 years attained neck control, sitting and any such early milestones. There were a few who had not attained a given milestone during the time of interview and this was taken as having not yet developed. The results showed that the mentally retarded were grossly delayed in all spheres, namely gross motor, fine motor, speech and social adaptive. It showed that the early milestones were not significantly affected; neck control was insignificantly delayed, $p > 0.05$ while walking was significantly delayed, $p < 0.001$. Fine manipulations

like transfer of objects, dressing and undressing together with speech were significantly delayed (see table IX). The insignificant results in the early milestones might be due to the fact that some of the mentally retarded children had been growing normally in early infancy. This can be best explained by the fact that the commonest causes of mental retardation in the study were in the postnatal group. Another possible explanation could be difficulty in remembering fine details of early milestones. Overall developmental assessment in ages in months was done by taking into consideration of what the child could do at the present age and this was found to be low in the mentally retarded. They were significantly retarded in comparison to control group ($p < 0.001$).

Psychometric testing was one of the difficult part of the study. It needed patience on the part of the examiner and the subject. The time taken was on the average of 1 - $1\frac{1}{2}$ hours and it was at times difficult to keep the child sitting and answering questions for such a long time. Thanks to the colourful toys and objects which kept the subjects involved in the test. Language barrier was experienced in 1 child whose testing had to be done through the interpretor. The results were interesting for both the study group and the controls. The study group never scored above I.Q.70 while the control group never scored below I.Q. 100 which is taken as the mean I.Q. score. It is from this that standard deviations (S.D.) are based. The S.D. for the Stanford

Binet test is ± 16. By taking 2 S.D. gives us 100 ± 32 which is 68 as the lowest score and 132 highest score. Scores below 68 were taken as mental retardation while scores of 132 were as normal with the highest score being I.Q. 149. The I.Q. scores in the study group showed that these were of skewed distribution to the right. This shows that there were more severely retarded in the study. The control group I.Q scores were normally distributed.

The finding of more severely mentally retarded children in the study just shows how the postnatal causes which were the commonest can severely retard the children mentally. The trend of other findings are that the mildly retarded children have always been the majority. The average I.Q. score for the mentally retarded was 37.6 while that of the control was 119.4. When these results were related to school performance, it was shown that those mentally retarded children who had previously attended normal schools were below average in comparison with the control group.

Many of the psychometric tests have been developed in developed world and are said to have a cultural bias. The need for Africa to have her own tests for the handicapped has been stated.[20] Other countries like India[14] and China[26] have standardised these psychometric tests to fit their cultural set up. Recently Sri Lanka has designed a chart for assessment of the mentally retarded.[64]

Psychometric testing has been used for school placements. The need may be from the teachers, parents or on clinical grounds. Educational background of the mentally retarded dates back to 1799 with modifications in between.[65,66,67] The educational situation for mentally retarded children in Tanzania has remained undefined for a long time. It has just been recently promising after the establishment of special education unit in the Ministry of Education. The establishment in 1980 of the society whose aims include seeking to initiate, establish, promote and co-ordinate programmes for education and training of children with cerebral palsy and mental retardation was also timely.[68]

Currently UNESCO based in Nairobi, Kenya, is running a subregional project for Special Education in Eastern and Southern Africa. With this, there have been many contributions towards education for the mentally retarded under the auspsies of UNESCO.[69,70,71]

Tanzania through the society has established schools for the mentally retarded. There are 4 in Dar es Salaam and 1 each in Tabora, Dodoma and Morogoro and Mtwara regions. Others are underway in Dar es Salaam and Tanga regions. This seems to be the beginning. May be the mentally retarded may sometime find joy in Tanzania.

The limitations of the study were that the study was done in Dar es Salaam and the sample size was small. Children seen

were mainly those of school age and many were attending special schools or treatment. It was not possible to study those who were at home due to short period of study and lack of transport. Poor memory and record keeping was also noted in the study. Psychometric testing used a foreign test and developmental assessment was based on foreign standards. No investigations were done in the study so diagnosis was based on clinical grounds.

6. CONCLUSIONS

It is recommended that:-

6.1. A minicensus be made to determine the extent of the problem in Tanzania.

6.2. Our own local developmental standards should be developed.

6.3. Prevention of the causes of mental retardation should be strengthened.

6.4. Foreign psychometric tests should be standardised for use in Tanzania.

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