

MEDICAL PRACTICE

Outside Europe

Paediatric Neurology in Africa: A Ugandan Report

H. G. EGDELL, J. P. STANFIELD

British Medical Journal, 1972, 1, 548-552

Summary

The findings in 138 children attending a neurology clinic in Uganda are presented. In contrast with findings in developed countries, only 25 had an abnormal birth and history dating from birth compared with 63 who had a normal birth and early development with symptoms of *postnatal* onset. The commonest mode of onset in the postnatal period was a catastrophic, feverish illness. Effective and usually easily achieved drug control of epilepsy and hyperkinesis enabled most parents to cope with disabled children. Simple explanation to parents and teachers can reduce the rejection and educational retardation associated with epilepsy.

Primary prevention lies in earlier diagnosis and treatment of cerebral malaria, meningitis, and encephalitis and improved obstetric services. Secondary prevention requires closer follow-up of potentially brain-damaged children and the education of doctors in neurological and behavioural assessment and the more efficient treatment of epilepsy and hyperkinesis.

Introduction

The rapidly developing interest in paediatric neurology has resulted in the publication of texts such as those of Ford¹ and Gamstorp² and journals such as *Developmental Medicine and Child Neurology*. The need for special clinics, however, has

been recognized only slowly, even in the Western World.^{3, 4} Disorders of the nervous system in children in the tropics has received very superficial and sporadic study⁵ despite the fact that children form a large proportion of the population of developing countries. In addition these children are exposed to more frequent environmental hazards, such as birth trauma, acquired infection, malnutrition, hyperthermia, and water and electrolyte disturbances. Late or ineffective treatment of these conditions adds to the toll of brain-damaged children among the survivors while the introduction of effective treatment of formerly killing diseases inevitably results in the survival of an increasing number of damaged individuals. Neurological problems in children in Ibadan, Nigeria, have been described by Elam⁶ and Animashaun⁷ and in New Delhi by Basu.⁸

The Ugandan African child population under 15 years of age amounts to 46.2% of a total of 9½ million. Most of the present patients came from the capital, Kampala—population 330,000.⁹ They were all African (mainly Baganda tribe) with the exception of one Indian child. Most lived in rural or semirural surroundings on a subsistence economy and vegetarian diet, looked after by parents or members of their extended family. Smaller groups of the children seen were more severely poverty-stricken—immigrants from neighbouring countries living on the threshold of malnutrition or new from urban slum dwellings with fathers who had come to the city to earn as much as possible before returning to their rural homes, often in Kenya. Some were referred from other areas in Uganda and neighbouring countries or from hospitals or orphanages in Kampala and elsewhere.

Mulago Hospital is the focus of paediatric care for Kampala and the surrounding rural and semirural area of Buganda district and is a reference centre for the rest of Uganda. The 100 children's beds and the outpatient department are mainly concerned with the resuscitation and treatment of children with acute organic disease. The service is staffed by personnel from both the Uganda Ministry of Health and Makerere University. The nearby dispensary on Mulago Hill screened 80,000 new children for the hospital wards and outpatient department during the period of study from July 1968 to July 1969. Altogether 250-300 new children are seen each day at this dispensary, of

Makerere University, Kampala, Uganda

H. G. EGDELL, M.R.C.P., D.P.M., Senior Lecturer in Psychiatry (Present appointment: Consultant Psychiatrist, Airedale District General Hospital, Eastburn, Keighley, Yorkshire)

J. P. STANFIELD, M.D., M.R.C.P., Reader in Paediatrics and Child Care (Present appointment: Consultant Paediatrician, Medical Research Council Child Nutrition Unit, P.O. Box 6717, Kampala, Uganda)

which some 30-50 are referred to Mulago Hospital paediatric outpatient clinic.

As a comprehensive neurological and psychiatric survey of such a clinic population was not practicable it was decided to create a separate referral clinic to study the presenting problems in this field while providing a limited clinical and teaching service. From the beginning it was felt that such a study of chronically handicapped children, which would include possible care and management problems, would require considerable restriction of numbers and therefore specific criteria for referral.

Patients and Methods

A total of 138 children (85 boys, 53 girls) were included in the study—14 aged up to 1 year, 81 aged 1-5 years, and 40 aged 5 years and over; the ages of three children were not known. Eighty of them were from the Baganda tribe.

Criteria for Referral.—All patients were referred to the clinic by doctors, mainly from Mulago Hospital and Mulago Hill dispensary, with a few from upcountry Government and mission hospitals. A letter of guidance in referral had been circulated suggesting obvious neurological syndromes, behaviour disorders, and refractory epilepsy (easily controlled epilepsy was too widespread to be coped with in one clinic). In practice failure of expected development was the most common reason for referral. There were separate clinics for poliomyelitis and malnutrition. Many doctors were so preoccupied with immediately treatable organic disease that they tended to overlook, and hence failed to refer, child neurological problems. The community itself set limitations to referral by its attitudes to Western medicine as a whole and to some conditions—for example, epilepsy. Distance and the expense of travel rendered most upcountry referrals impracticable. Finally, time and limited staff further restricted numbers.

History-taking.—This was limited by lack of past records, children often being accompanied by members of the extended family, and children and parents speaking only indigenous languages. Carefully selected interpreters were used. Difficulties with retrospective data have, however, been reported in much more sophisticated populations and studies.¹⁰ Operational definitions are given in the Appendix.

Results

The age, sex, and tribal distributions of the children did not differ appreciably from the usual pattern of child outpatients attending Mulago Hospital except that boys only slightly exceed girls in the general clinic.

HISTORY AND TIME OF ONSET

The symptom of most common concern to parents or guardians was failure of the child to achieve an expected level of development, in some cases complicated by fits (Table I). Refractory epilepsy formed a further large group. The remaining third were about equally divided between specific failures of achievements—for example, talking or walking—and behaviour disorders.

In 46 (33%) of the children onset dated from the time of birth and in 86 (63%) it occurred postnatally, either after a

TABLE I—Principal Presenting Symptoms

Not achieving expected level of development	40
Fits only	30
Not achieving expected level of development and fits	29
Bad behaviour (excluding transient postepileptic)	9
Not talking or walking	7 each
Not achieving expected level of development and deaf	4
Not talking and fits, not talking and deaf, school refusal, headache, weakness arm, fainting attacks, "confusion," night terrors, stutter	1 or 2 of each

catastrophic acute illness (38 cases) or in an ill-defined manner (48 cases) in which the evolution of the symptoms seemed to have taken place over some time. Some of these histories may represent the gradual expression of the child's inadequacy present in reality from birth. Of the 38 children whose disability occurred suddenly this had followed a feverish illness, often with fits, in 27 (71%). In only a few of these could a clear diagnosis be made retrospectively. In six cases the time of onset of the disorder was not known.

A birth history was available in 115 of the children, though in some it was sketchy. In 33 the birth history was abnormal and most of these children had problems dating from birth (Table II). By contrast a history of no abnormality at birth was

TABLE II—Abnormalities of Delivery or of Baby in First Few Days of Life related to Onset of Symptoms from Birth or Postnatal Onset

	Delivery and Neonatal Period			Total
	Abnormal	Normal	Unknown	
Symptoms dating from birth	25	18	3	46
Postnatal onset	8	63	15	86
Unknown	0	1	5	6
Total	33	82	23	138

associated in most instances with a postnatal onset of symptoms. A history of other possible causes of central nervous system disease was sought. No cases of familial disease were detected, though relatives may have been reluctant to admit family failings. No history of head injury was elicited.

Jaundice in the neonatal period was specifically inquired into in the last 78 patients of this series, and data were available on 55 of these. Only eight gave a history of jaundice in the first week of life—in two it was present at birth and in six it occurred postnatally. In four of these patients the jaundice was transient and considered irrelevant. One of the remainder was jaundiced in association with severe neonatal septicaemia. Three children had a history suggesting neonatal hyperbilirubinaemia with consequent permanent effects—one was premature and deaf, another was deaf, subnormal, and athetoid, and the third was subnormal with severe spastic tetraplegia. In the remaining 47 patients jaundice did not occur.

CLINICAL FINDINGS

Neurological deficit (Table III) showed a pattern similar to that reported in previous studies. Hypotonia was found frequently (Table IV), when the complaint was usually "weakness." Aphasia was diagnosed in six patients, being the only problem with three of them.

Mental subnormality affected half of all the patients seen, and two-thirds of these also had a neurological defect. Thirty-three children had a neurological deficit with clinically normal intelligence.

TABLE III—Neurological Findings and Clinical Subnormality*

Tetraplegia	36	Hypotonia	27
Hemiplegia	10	Tremor	1
Paraplegia	6	Deafness (moderate & severe)	13
Monoplegia	1	Blindness (moderate & severe)	6
Athetosis	7	Aphasia	6
Spasticity	36	Clinically subnormal intelligence	68
Ataxia	3		

* Some children had more than one neurological disorder.

TABLE IV—Reflexes in the 27 children with Hypotonia

Distribution of Hypotonia	Reflexes			Total
	Increased	Decreased	Uncertain	
Generalized	9	10	4	23
Localized	1	2	1	4

Behaviour disorders were found in 41 children (31 boys, 10 girls). In 17 of these the main feature was hyperkinesis, in 9 it was physical violence, and in 15 various features were noted. Twenty-one of the disturbed children suffered from epilepsy, including three of the nine physically violent and eight of the 17 overactive children. The principal cause of these disorders was organic in 32 and non-organic in 9.

Finally, convulsions occurred in 65 patients (in 8 only during the initial illness). The vast majority of these children had convulsions complicated by other conditions, reflecting the bias of the referral criteria of the clinic. These conditions were neurological defects (30 cases), clinical subnormality (29 cases), and behaviour disorder (21 cases). Some patients had more than one additional complication. In the uncomplicated cases four were associated with fever and 15 were not.

Discussion

The preponderance of male over female attendances is in keeping with experience in paediatric neurology clinics in England,¹¹ Scotland,¹² the United States,¹³ and India,⁸ and in child psychiatry generally.^{14 15}

PRESENTING SYMPTOMS

The presenting symptoms were similar to those at European clinics—namely, mental handicap, epilepsy, and motor dysfunction.^{3 16} The large proportion of children presenting with delayed development indicates not only parental concern but also the hope that Western medicine might influence the situation. Learning problems presented relatively rarely. This is understandable against the background of limited school places, the cost of education at both primary and secondary levels, and the intense desire for education in the community as a whole. Any clinical impediments to educability, whether organic or emotional, would transform the already difficult task of education into an impossibility. Learning problems are in this way shelved rather than solved.

Presentations noteworthy by their absence were those behaviour and psychosomatic disorders which trouble the child but not society. Furthermore, many of these problems are assumed to be unresponsive to Western medicine. Nevertheless, some varieties of behaviour problems were thought by parents to have a medical aspect and were a major concern in almost one-third of the cases. This was almost exactly the same proportion as found by Small¹³ in the United States but several times that found by Elam⁶ in Nigeria.

ONSET

Onset of symptoms was postnatal in almost two-thirds of the children and in almost half of these cases was catastrophic, usually associated with pyrexial illness. In addition three-quarters of the children with a postnatal onset of symptoms had a normal birth. When birth history was related to time of onset of symptoms (Table II) two major groups emerged: (a) symptoms from birth with an abnormal birth history (25 patients), and (b) postnatal onset of symptoms with a normal birth history (63 patients). Though an important group dated from birth (with more than half having had an abnormal delivery) the most frequent timing of onset of disability was postnatal with a previously normal history.

This is in line with the previous experience of this clinic described by Stanfield and Martin,¹⁷ who found that acquired postnatal brain lesions were responsible for nearly 40% of the total (209) disabilities. This is in contrast to recent experience in developed countries. Pond¹¹ found that among 58 children with brain damage without epilepsy the onset in 35 was in the first

two years of life, birth events being the largest single cause. Drillien *et al.*,¹⁸ in a series of nearly 400 mentally retarded children, found that acquired postnatal lesions accounted for only 6.4% of the total. Postnatal onset predominated in a clinic in India for crippling diseases but this was due to the fact that 80% of the patients suffered from poliomyelitis.⁸

CATASTROPHIC INFECTIONS

Catastrophic infections either specifically neurotropic, such as cerebral malaria, bacterial meningitis, and viral encephalitis, or general, associated with severe "febrile" convulsions, are the common causes of postnatal damage to the central nervous system in Ugandan children. "Febrile encephalopathy of unknown aetiology" has been suggested as a substitute for "viral encephalitis," as viruses have not often been isolated nor have significant rises in antibody titres been found.^{19 20} The effects of these infections were aggravated by delays in presentation for treatment due to problems of distance and an overloaded medical service. Some of this damage could have been prevented by earlier diagnosis and treatment.

Many case histories suggested that the initial feverish illness was not treated with the seriousness it deserved and antimalarial therapy was given too late or only by mouth, and fever allowed to continue unchecked. This is in keeping with the study by Virmani and Devi²⁰ of the neurological sequelae in children with pyrexia of unknown origin. They found that the ultimate deficits were in proportion to the degree and duration of the acute disturbance of the sensorium or of pyrexia or both, particularly in children less than 1 year of age. The prognostic importance of the control of high fever and prompt restoration of water and electrolyte balance in such patients has previously been emphasized by Lyon *et al.*²¹ It is perhaps only in retrospect that this is clear and it must certainly be viewed against the vast background of feverish illness in children presenting at out-patient departments and rural dispensaries in developing countries.²²

NEONATAL JAUNDICE

Neonatal jaundice is an important cause of brain damage, notably kernicterus and deafness. Only three of the 138 children were thought to have suffered this injury. In contrast Animashaun⁷ reported on a West African paediatric neurology clinic where 97 out of 196 new patients with cerebral palsy seen in one year were thought to be suffering from the effects of kernicterus. Of these children 42 had been given an exchange transfusion at least once. Basu⁸ reported from India the cases of 28 patients with a history of neonatal jaundice among 280 with cerebral palsy. The reasons for this geographical variation in morbidity are not clear.

MENTAL SUBNORMALITY

Milestone achievement and clinical observation are coarse tools for the recognition of subnormality, and its frequency was probably underestimated. Even if psychological testing had been available the problems of interpretation of results of such tests in the absence of normal controls would have been difficult. The adaptation of psychometric tests based on Western norms presents problems as these measure behaviour inevitably influenced by culture.²³ There is a growing need in developing countries for local norms of behaviour response to standard or adapted psychological assessments. One study in Uganda has shown that the response of 20 children aged 2½-9 years with cerebral palsy and brain damage to a wide battery of psychological tests was so poor that scores of any reliability could not be assigned.²⁴

The parents of these severely subnormal and often physically

handicapped children found that their main problems were not education but basic care and the need to control disturbed behaviour. The increasing size of these children provided difficulties in feeding and toilet and in their need to be constantly lifted and carried. On the whole the uncomplicated severely subnormal child is cared for by the extended Ugandan family and the threat of rejection and abandonment arises only where there is either totally unacceptable antisocial behaviour or no family support—for example, the unmarried or deserted mother. This contrasts with Elam's suggestion that the mentally retarded do badly in the community in Nigeria.

Hence the first aim was to encourage these community resources for caring for the mentally handicapped child and to support the families with their problems rather than to relieve them of the child. The latter is at the moment out of the question in Uganda, where institutional care for these children is negligible and already overloaded with abandoned children. It is doubtful whether the development of institutions for mentally handicapped children should be advised for developing countries over and above those required to cope with total abandonment. They might well encourage the community to abdicate responsibilities which are accepted at present and provide an increasing drain on slender resources, for the care of these children is life long.

Control of epilepsy, hyperkinesia, and outbursts of temper with the use of drugs can make the situation tolerable for the mother and family. Discussion of aetiology (especially with regard to the commonly feared parental responsibility for the illness) and likely prognosis may enable the parent to view the situation more realistically. Children with normal or only slightly reduced intelligence debarred from school because of physical handicap or epilepsy can be rehabilitated, and a letter from the clinic to the headmaster was often enough to enable schooling to begin.

MALNUTRITION

Twelve children were clinically malnourished. In 10 of these this was thought to be largely due to maternal deprivation and neglect as a reaction to the child's physical and mental disabilities. In two children, however, the mental retardation was thought to be the result of a combination of maternal deprivation and malnutrition. Satisfactory nutritional and social rehabilitation was accompanied by improved achievement of milestones and clinical intellectual normality. Malnutrition may be responsible for a retardation of mental development either as a result of the associated deprivation of emotional and learning experience or, possibly, because of direct damage to the cerebral cortex. The extent to which this persists into later life is still uncertain.²⁵

SPEECH AND BEHAVIOUR DISORDERS

Speech delay in the presence of normal hearing and intelligence occurred in this study and also a previous study of the clinic by Stanfield and Martin. An isolated developmental disorder, emotional deprivation, or negativism are possible causes. Behaviour disorders were present in nearly one-third of all the patients and showed an excess of boys over girls (31/10).

Highly selected patients were included in this study and they cannot be said to represent the pattern of problems in the community. Two studies of behaviour disorders in Eastern Africa suggest that the preponderance of brain damage in hospital clinics contrasts with the higher incidence of neurosis in the community.^{26, 27} In over half of these children distress to the parents was caused by overactivity or violence which the referring doctors had been unable or unwilling to treat. The 17 with hyperkinesia of severe degree were distractible, intolerant

of restraint, fearless, destructive, and noisy, and restlessness, especially at night, seriously disrupted family life. The clinical picture was precisely that described in English children by Ounsted.²⁸ A history suggestive of a catastrophic brain insult in most of these children left little doubt about the organic aetiology. Mothers showed considerable tolerance but were under great stress, particularly when there were other children, and some were forced to use mechanical restraints. Fortunately there was a good response to drug treatment—chlorpromazine (up to 100 mg or occasionally 150 mg a day in divided doses) and less often diazepam (up to 60 mg a day) were effective. Occasionally there was a response to amphetamine (up to 30 mg a day) if the others failed. Mothers tolerated the child's drowsiness as a result of chlorpromazine better than the loss of appetite with amphetamine. Satisfactory control of coexisting epilepsy often produced much improved behaviour. Successful treatment of hyperkinesia and epilepsy is not difficult and should be within the expertise of all doctors.

In contrast to paediatric clinics in the West there was an absence of direct complaints of emotional disorders such as anxiety or depression and also of hysterical illness, appetite disorders, constipation, abdominal pains, enuresis, encopresis, sleep problems (except in hyperkinesia), and disciplinary and learning problems. This cannot be due to the clinic being regarded as concerned only with neurological problems as there were also relatively few of these symptoms in a series of children presenting at the nearby mental health clinic during part of the same period.²⁷ This is probably accounted for by the parental view that emotional symptoms are neither important nor a medical matter. Most somatic symptoms of an underlying emotional disorder are treated physically by the doctor or medical assistant (as the parent expects) and are not referred to a paediatrician or psychiatrist. The heavy load of gross and treatable diseases in Ugandan children has forced general practitioners and paediatricians to concentrate on these to the exclusion of psychosomatic and behavioural problems. It would be unfortunate, however, if this attitude were perpetuated indefinitely in the teaching of medical students. Locally trained medical personnel (with notable exceptions) were often unskilled in this important aspect of paediatrics.

EPILEPSY

Epilepsy is an important and common problem in all paediatric and general outpatient clinics in developing countries. The community attitude towards epilepsy is of profound importance. Many people believe that it is not amenable to Western medical treatment and almost all fear it as being infectious.²⁹ This frequently led to the child being withdrawn from school, as teachers feared infection and parents felt that it was not worth spending money on school fees for a child with a "spoilt brain." At home the child was rejected and restricted to his own eating utensils and separate sleeping accommodation. Treatment with phenobarbitone was very satisfactory. Occasionally phenytoin or primidone was used but only when absolutely essential, as it was difficult for patients in outlying areas to obtain further supplies. The child could usually be reinstated in school by writing to head teachers. This simple procedure is important, as many adults have been seen in whom one or two fits as a child were enough to end their school life in a country where education is not compulsory.

Future of Paediatric Neurology in Africa

Paediatric neurology is bound to grow in importance as needs are recognized and more effectively met. Ideally specialists would be required, but the shortage of paediatricians, neurologists, and psychiatrists in developing countries make this an unlikely achievement. One person with a special interest and

experience in this field should be in every teaching hospital to fulfil teaching and research needs and should be primarily a paediatrician with an interest in child development and psychiatry. Norms of development and developmental test responses should be devised.

A team approach enabling adequate paediatric, psychiatric, speech, hearing, and physiotherapy assessment is desirable in dealing with many of these complicated clinical problems. The present clinic functioned without such a comprehensive team or special facilities, and developing countries cannot delay care and research until such ideal conditions are available. Doctors and medical assistants will need to be able to recognize and treat more effectively increasing numbers of children presenting with chronic neurological, emotional, behavioural, and learning problems. This will necessitate modifications in attitudes and training.

Facilities for inpatient assessment and care are not easy to provide. Paediatric wards expose these children to infection and are not geared to coping with the grossly overactive and destructive child. Our practice of admitting a handful of such children to an adult neurosis and early psychosis ward worked surprisingly well, mainly owing to the availability of nursing skills in observation and management. Thus there is a greater need for trained nursing staff than for elaborate facilities and that inpatient care can be planned (in the first instance) with relatively simple physical provisions. The Ugandan Ministry of Health is to be congratulated on its pioneering of a child psychiatry ward which will be particularly concerned with paediatric neurological conditions.

The general public and, in particular, teachers need to learn far more about the true nature of such conditions as mental retardation, epilepsy, and severe physical handicap so that children afflicted by these may not be further handicapped by social and educational ostracism.

Prevention of brain damage in deprivational and behaviour disorders is very important. Improved obstetric care and the earlier recognition of the fetus "at risk," early diagnosis and treatment of acute infections, immunization and malaria prophylaxis, as well as the prevention of malnutrition and emotional deprivation are all necessary in order to reduce the amount of disability with which this area of paediatrics is concerned.

We are indebted to Professor G. Allen German for his encouragement of the clinic and his valuable guidance and assistance in the preparation of the paper. Professor Bengt Hagberg, Dr. John Wilson, and Dr. Janet Goodall have made useful criticisms. Acknowledgement for their contributions to the clinic are due to Miss B. Bremner, physiotherapist, Mrs. S. Karim, speech therapist, and the interpreters Samuel Kiwanuka and Michael Balingenera. We are also grateful to the Chief Medical Officer, Ministry of Health, Uganda.

Requests for reprints should be addressed to Dr. H. G. Egdell.

Appendix

Postnatal onset refers to the onset of symptoms after the first month of life, particularly if the preceding development was normal.

Normal birth history refers to a history taken direct from the mother in the absence of (a) prolonged labour with many hours of strong pains, especially when compared with the birth of the patient's siblings; (b) precipitate labour with an extremely rapid or unexpected delivery; (c) delay in crying; (d) poor sucking during the first week; (e) forceps, vacuum, or caesarian delivery; (f) twitching or fits in the first week.

Mental subnormality was based on a combination of (a) delay in milestone achievement—namely, sitting, walking, speech (single words or short sentences); (b) parental impression of less than expected social achievement; (c) total clinical impression at interview and examination; (d) school report—only occasionally available.

The use of the above criteria gives only a rough guide and may overlook borderline subnormality while including some children of normal intelligence with physical disabilities.

Behaviour disorder was any behaviour thought to be outside the normal range in the opinion of either the parent or the examiner. The tolerance of either is important. Behaviour considered part of the classical picture of mental subnormality, speech delay, or neurological defect was not recorded under behaviour disorder.

References

- 1 Ford, F. R., *Diseases of the Nervous System in Infancy. Childhood and Adolescence*, 5th edn. Springfield, Thomas, 1966.
- 2 Gamstorp, I., *Paediatric Neurology*, London, Butterworths, 1970.
- 3 Hagberg, B., Ingram, T. T. S., and Mac Keith, R., *Lancet*, 1970, 1, 940.
- 4 Wilson, J., personal communication, 1971.
- 5 Spillane, J. D., *Proceedings of the Royal Society of Medicine*, 1969, 62, 403.
- 6 Elam, H. P., *Developmental Medicine and Child Neurology*, 1967, 9, 784.
- 7 Animashaun, A., "Cerebral palsy in African children. Aetiological considerations." Paper read to 1st International Conference on Tropical Paediatrics, Ibadan, Nigeria, 1970.
- 8 Basu, B., *Journal of the Indian Medical Association*, 1967, 49, 477.
- 9 1969 Population Census of Uganda, Preliminary Report. Entebbe, Uganda, Government Statistician, 1971.
- 10 Kennedy, C., and Ramirez, L. S., in *Brain Damage in Children*, ed. H. G. Birch, p. 13. Baltimore, Williams and Wilkins, 1964.
- 11 Pond, D. A., *British Medical Journal*, 1961, 2, 1377, 1454.
- 12 Ingram, T. T. S., *Paediatric Aspects of Cerebral Palsy*, pp. 49, 73, 436. Edinburgh, Livingstone, 1964.
- 13 Small, J. G., *Archives of General Psychiatry*, 1962, 7, 120.
- 14 Rutter, M., and Graham, P., *Proceedings of the Royal Society of Medicine*, 1966, 59, 382.
- 15 Shaw, C. R., *Psychiatric Disorders of Childhood*, p. 64. New York, Appleton, 1966.
- 16 Wilson, J., *British Medical Journal*, 1969, 4, 152.
- 17 Stanfield, J. P., and Martin, J. A. M., "Delay and Disability in a Group of African Children." Awaiting publication.
- 18 Drillin, C. M., Jameson, S., and Wilkinson, E. M., *Archives of Diseases of Childhood*, 1966, 41, 528.
- 19 Patel, C. C., "Acute febrile encephalopathy in Mulago Hospital." Paper presented to East African Association of Physicians, Nairobi, Kenya, 1968.
- 20 Virmani, V., and Devi, M. G., *Indian Journal of Medical Sciences*, 1969, 23, 132.
- 21 Lyon, G., Dodge, P. R., and Adams, D. R., *Brain*, 1961, 84, 680.
- 22 Stanfield, J. P., *British Medical Journal*, 1969, 1, 761.
- 23 Anastasi, A., *Psychological Testing*, 3rd edn, p. 241. New York, Macmillan, 1968.
- 24 Haskell, S. H., and Anderson, E. M., *Developmental Medicine and Child Neurology*, 1970, 12, 198.
- 25 Cravioto, J., and de Licardie, E. R., in *Malnutrition, Learning and Behaviour*, ed. N. S. Scrimshaw and J. E. Gordon, p. 252. Cambridge, Massachusetts Institute of Technology Press, 1968.
- 26 Cederblad, M., *Acta Psychiatrica Scandinavica*, 1968, Suppl. No. 200.
- 27 Egdell, H. G., *Psychopathologie Africaine*, 1970, 6, 71.
- 28 Ounsted, C., *Lancet*, 1955, 2, 203.
- 29 Orley, J., *Culture and Mental Illness*. Nairobi, Kenya, East African Publishing House, 1970.